TEXTBOOK OF DIET IN DISEASES

Bushaira V

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FOREWORD

Nutrition is a cornerstone of health and well-being, playing a pivotal role in preventing and managing diseases. "A Textbook of Diet in Diseases", authored by the distinguished faculty of the Home Science Department at Korambayil Ahamed Haji Memorial Unity Women's College, Manjeri offers a comprehensive exploration of how dietary choices influence health outcomes. This book serves as an essential guide for anyone seeking to understand the complex relationship between nutrition and disease, from students and healthcare professionals to individuals committed to improving their health through informed dietary decisions. The faculty at Korambayil Ahamed Haji Memorial Unity Women's College brings together a rich blend of academic expertise and practical experience in nutrition, dietetics, and health sciences. Their dedication to research, teaching, and community service has provided them with deep insights into both the theoretical and practical aspects of nutrition. This book is a culmination of their extensive knowledge and commitment to sharing valuable information with a wider audience.

"A Textbook of Diet in Diseases" addresses a wide array of topics, including metabolic disorders, cardiovascular diseases, cancer, gastrointestinal conditions, and kidney diseases. Each chapter is meticulously designed to offer a blend of theoretical knowledge, practical advice, and evidence-based research. This approach ensures that the book is a valuable resource for both academic study and personal application.

As you delve into the pages of "A Textbook of Diet in Diseases," you will gain a deeper understanding of the profound impact of nutrition on health. The insights and knowledge shared in this book will empower you to make informed dietary choices that can lead to healthier lives and communities.

I extend my gratitude to the faculty of the Home Science Department at Korambayil Ahamed Haji Memorial Unity Women's College for their invaluable contributions to this work. I hope that " A Textbook of Diet in Diseases " will serve as a trusted resource in your pursuit of better health and well-being.

Sincerely,

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PREFACE

In the realm of healthcare, nutrition plays a pivotal role in the prevention, management, and treatment of various diseases. It is a guide that delves into the intricate relationship between diet and disease. As dietary habits continue to evolve in the face of globalization and changing lifestyles, it becomes imperative to understand the role of diet in the management of chronic and acute diseases.

Nutrition plays a critical role in the prevention, management, and treatment of various diseases, and understanding this relationship is essential for healthcare professionals and patients alike. This is a comprehensive resource designed to explore the intricate connection between diet and disease. This book aims to serve as a vital tool for those seeking to understand and apply dietary principles to enhance health outcomes and manage diseases effectively.

This textbook covers a wide spectrum of diseases, offering detailed dietary guidelines and recommendations for each. The chapters are organized to address febrile conditions, cardiovascular diseases, gastrointestinal issues, renal diseases, Diabetes Mellitus, and more. Each chapter is authored by experts, ensuring that the information is accurate, evidence-based, and up-to-date. In addition to disease-specific dietary advice, the book delves into the fundamental principles of nutrition, the physiological effects of various nutrients, and the psychological aspects of dietary behavior.

I would like to extend my deepest gratitude to the contributing authors and experts whose dedication and expertise have made this book possible. Their collective efforts have resulted in a comprehensive resource that will undoubtedly serve as an invaluable tool in the field of dietetics and nutrition.

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TYPHOID

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INTRODUCTION

Typhoid fever is an infection that causes fever, diarrhoea, and a rash. It is most commonly caused by bacteria called Salmonella typhi (S typhi). Typhoid fever is rare in places where few people carry the bacteria. It also is rare where water is treated to kill germs and where human waste disposal is managed. One example of where typhoid fever is rare is the United States. Places with the highest number of cases or with regular outbreaks are in Africa and South Asia. It is a serious health threat, especially for children, in places where it is more common. Food and water with the bacteria in it cause typhoid fever. Close contact with a person who is carrying the salmonella bacteria also can cause typhoid fever. Symptoms include: High fever, Headache, Stomach pain, Constipation or diarrhea.

RISK FACTORS

Common risk factors in the development of typhoid fever are:

Travel to endemic areas

Poor hygiene habits

Poor sanitation conditions

Proximity to flying insects feeding on feces

Contact with someone who recently suffered from typhoid fever

Recent use of antibiotics

Achlorhydria

Immunosuppressive illnesses such as AIDS

Crowded housing

Consumption of raw fruits and vegetables contaminated with sewage

Prolonged illness

Being a health care worker

Being a clinical microbiologist who handles salmonella typhi

Childhood

Pathophysiology

Typhoid fever is caused by the bacterium Salmonella enterica serotype Typhi. The pathophysiology involves several key steps:

Ingestion of contaminated food or water: The bacteria are typically transmitted through the fecal-oral route, often due to contaminated food or water.

Invasion of the intestinal mucosa: After ingestion, the bacteria invade the small intestine‘s epithelial cells, where they multiply and spread to regional lymph nodes.

3. Systemic dissemination: The bacteria can enter the bloodstream from the lymphatic system, leading to bacteremia. This allows them to spread to various organs and tissues throughout the body, including the liver, spleen, bone marrow, and gallbladder.

4. Symptom onset: As the bacteria proliferate in various organs and tissues, the patient experiences systemic symptoms such as fever, headache, malaise, and gastrointestinal symptoms like abdominal pain, constipation, or diarrhea.

Immune response: The host‘s immune system mounts an inflammatory response to combat the infection, leading to the release of cytokines and other inflammatory mediators. This contributes to the characteristic symptoms of typhoid fever.

Formation of granulomas: In some cases, the bacteria can form granulomas in the liver, spleen, and bone marrow, contributing to the persistence of infection and potentially leading to chronic carriage of the bacteria.

Complications: Without prompt treatment, typhoid fever can lead to severe complications such as intestinal perforation, gastrointestinal bleeding, septicemia, and encephalopathy.

Overall, the pathophysiology of typhoid fever involves a complex interplay between the host immune response and the virulence factors of the Salmonella Typhi bacterium.

SYMPTOMS

The symptoms of typhoid fever typically develop gradually over the course of one to three weeks after exposure to the bacteria. Common symptoms include:

Prolonged fever: Sustained high fever, often reaching 103-104°F (39-40°C), is a hallmark symptom of typhoid fever.

Headache: Persistent headache, often severe, is common.

Malaise: Feelings of general discomfort, weakness, and fatigue are typical.

Abdominal pain: Belly pain and tenderness, often concentrated in the lower right quadrant, may occur.

Constipation or diarrhea: Diarrhea is more common early in the illness, but constipation may develop as the disease progresses.

Rash: Some patients develop a flat, rose-coloured rash on their trunk, known as ―rose spots.‖

Enlarged spleen and liver: Abdominal examination may reveal an enlarged spleen (splenomegaly) and liver (hepatomegaly).

Relative bradycardia: A slower than expected heart rate compared to the fever level is a characteristic feature of typhoid fever.

These symptoms can vary in severity and may overlap with other illnesses, so a definitive diagnosis often requires laboratory testing. Without treatment, complications such as intestinal perforation, gastrointestinal bleeding, and systemic infection can occur. Early diagnosis and appropriate antibiotic treatment are essential to prevent serious complications.

COMPLICATIONS

Complications of typhoid fever can be severe and may occur when the illness is not treated promptly or effectively. Some of the potential complications include:

Intestinal Hemorrhage: Bleeding can occur in the intestines due to the ulceration of the Peyer's patches, a type of lymphoid tissue in the small intestine. This can lead to sudden and severe bleeding.

Intestinal Perforation: The same ulcers that can cause bleeding can also perforate, creating a hole in the intestine. This allows intestinal contents to leak into the abdominal cavity, leading to peritonitis (inflammation of the abdominal lining) and sepsis, which are life-threatening conditions requiring emergency surgery.

Encephalopathy: Confusion, delirium, and alterations in consciousness can occur, possibly due to inflammation and the systemic effects of the infection.

Myocarditis: Inflammation of the heart muscle, which can affect the heart‘s ability to pump blood and can lead to heart failure.

Pneumonia: The bacteria can infect the lungs, leading to pneumonia, which complicates the course of the illness.

Hepatitis: Inflammation of the liver, presenting as jaundice (yellowing of the skin and eyes), can occur, indicating liver involvement.

Bone and joint infections: The bacteria can infect bones and joints, leading to conditions like osteomyelitis (bone infection) and arthritis.

Kidney or Bladder Infections: The urinary system can become infected, leading to conditions such as pyelonephritis or cystitis.

Cholecystitis: The gallbladder can become inflamed, potentially serving as a chronic reservoir for the bacteria.

Chronic Carrier State: Some individuals may become asymptomatic carriers of Salmonella Typhi after recovering from the acute illness. They can shed the bacteria in their feces, posing a risk of spreading the disease to others.

These complications underscore the importance of early diagnosis and prompt, appropriate treatment of typhoid fever to prevent severe disease and transmission.

DIAGNOSIS

Diagnosis of typhoid fever typically involves a combination of clinical evaluation, laboratory tests, and sometimes imaging studies. Here are some common methods used for diagnosis:

Clinical Evaluation: A healthcare provider will assess the patient‘s symptoms, medical history, and recent travel or exposure to regions where typhoid fever is prevalent.

Physical Examination: The healthcare provider may perform a physical examination to check for signs such as fever, abdominal tenderness, enlarged spleen or liver, and the presence of a rash.

Blood Cultures: Blood cultures are the primary diagnostic test for typhoid fever. Samples of blood are collected and incubated to see if Salmonella Typhi bacteria grow. Multiple blood cultures may be necessary, as the bacteria can be intermittently shed into the bloodstream.

Stool Cultures: In some cases, stool cultures may be performed to detect Salmonella Typhi bacteria. This is especially useful in individuals with prolonged or chronic symptoms.

Serologic Tests: Serologic tests, such as the Widal test, detect antibodies produced by the immune system in response to Salmonella Typhi infection. However, these tests have limitations and may not be reliable for diagnosing acute infection, particularly in regions where typhoid is endemic.

Imaging Studies: Imaging tests like abdominal ultrasound or CT scan may be performed to evaluate for complications such as intestinal perforation or to assess the extent of involvement of organs like the liver and spleen.

Bone Marrow Culture: In certain cases, bone marrow culture may be performed if there is suspicion of typhoid fever and blood cultures are negative. Bone marrow has a higher yield for detecting the bacteria.

PCR Tests: Polymerase chain reaction (PCR) tests can detect Salmonella Typhi DNA in blood, stool, or other samples. These tests may offer rapid and sensitive detection, especially in the early stages of infection.

A combination of these diagnostic methods, along with clinical judgment, is often necessary for an accurate diagnosis of typhoid fever. Early diagnosis is crucial to initiate appropriate treatment and prevent complications.

PREVENTION

Prevention of typhoid fever involves a combination of vaccination, safe food and water practices, and proper sanitation and hygiene measures. Here are the key strategies:

1. Vaccination: There are two vaccines available for typhoid fever:

a. Capsular Polysaccharide Vaccine: Administered as a single injection, it is recommended for people aged two years and older. Its protection lasts for about 2 to 3 years.

b. Live Attenuated Oral Ty21a Vaccine: Given in four doses, it is recommended for people aged six years and older. Its protection lasts for about 5 to 7 years.

Vaccination is especially recommended for travelers to areas where typhoid fever is common, individuals in close contact with a typhoid carrier, and people living in endemic areas.

2. Safe Food and Water Practices:

Since typhoid is transmitted through contaminated food and water, it‘s crucial to:

- Drink only boiled or bottled water, or water treated with a reliable purification method.
- Avoid raw fruits and vegetables unless you can peel them yourself.
- Avoid foods and beverages from street vendors.
- Ensure food is thoroughly cooked and hot when served.

3. Hand Hygiene: Regular and thorough hand washing with soap and water is essential, especially:

- Before eating or preparing food.
- After using the toilet.
- After handling items that could be contaminated.

Sanitation and Sewage Disposal: Proper disposal of sewage and avoiding contamination of water sources help reduce the risk of typhoid spread in communities.

4. Education: Educating communities about the modes of transmission, symptoms, and prevention measures can help reduce the incidence of typhoid fever.

Antibiotic Stewardship: Judicious use of antibiotics can help prevent the development of antibiotic-resistant strains of Salmonella Typhi.

Carrier Identification and Treatment: Identifying and treating individuals who are chronic carriers of Salmonella Typhi can help prevent the spread of the disease.

Implementing these preventive measures can significantly reduce the risk of typhoid fever infections. Vaccination is particularly important for individuals traveling to or living in areas where typhoid fever is prevalent.

DIETARY RECOMMENDATION

High-Calorie Diet

A high-calorie diet is suggested for typhoid fever to provide the patients with needed energy and also avert weight loss as a result of poor appetite and fever. Some of the food sources abundant in calories include bread, rice, cereals, boiled potatoes and starchy vegetables and bananas.

Food Dense in Carbohydrates

It is vital to have high carbohydrate foods that are light, soft and bland and easy to digest for patients down with typhoid fever. Include cereal porridge, boiled rice, poached eggs, and steamed foods like idly, idiyappam and apple sauces to boosts up your strength.

Protein-Rich Foods

Dairy products like skim milk, curd and yogurt help to meet the demands of protein and also speed the recovery process and support the immune system. Skim milk products and eggs are preferred source of protein than red meat. Vegetarians can include boiled legumes and paneer that are good sources of protein. Also

Comforting Foods

Soothing foods like soups and broth are beneficial in combating fatigue and boosting energy. Some of the options of comforting foods are clear vegetable soup, carrot soup, clear dal soup and clear chicken soup.

Hydration

Adequate hydration is essential to maintain the fluid balance and prevent dehydration caused due to diarrhea and fever. It is essential to supplement the body with enough fluids and drink plenty of water, include tender coconut water, fresh fruit juices, buttermilk and also have fruits with high water content. Drink water which is boiled and filtered well.

Foods to Avoid

Restrict foods that are high in fibre content, as these foods can upset the digestive system. It is best to avoid vegetables like cabbage, cauliflower and bell peppers as they can result in bloating and flatulence.

Spicy foods, deep-fried foods and processed foods are to be avoided to keep infection at bay.

Ghee, butter, rich creamy desserts and sweets are to be avoided

Preventive Measures to be Followed

Cook food thoroughly, avoid fast foods, foods from street vendors and food stored at room temperature.

Avoid eating raw and unpeeled vegetables and fruits and always wash the fruits and vegetables before you eat.

Drink boiled and filtered water.

Wash hands thoroughly with soap and water before cooking, having food, before and after using the toilets.

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TUBERCULOSIS

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INTRODUCTION

Tuberculosis (TB) is an infectious disease caused by the bacterium Mycobacterium tuberculosis. It primarily affects the lungs but can also affect other organs. TB spreads through the air when an infected person coughs, sneezes, or talks, releasing tiny droplets containing the bacteria. The disease can be latent, where the bacteria remain dormant in the body without causing symptoms, or active, where symptoms such as coughing, chest pain, weight loss, and fatigue may occur. Without proper treatment, active TB can be fatal.

Treatment typically involves a combination of antibiotics taken over several months. However, drug-resistant strains of TB pose a significant challenge to global efforts to control the disease.

TB remains a major global health concern, particularly in low- and middle-income countries. Efforts to combat TB include vaccination, early detection, and treatment, as well as addressing social determinants of health and improving access to healthcare services.

RISK FACTORS

1. Weakened Immune System: Individuals with weakened immune systems due to conditions such as HIV/AIDS, malnutrition, diabetes, or certain medications (e.g., corticosteroids, chemotherapy) are at higher risk of TB infection and progression to active disease.
2. Close Contact with Active TB Cases: Being in close contact with someone who has active TB increases the risk of infection. This is especially true in crowded or poorly ventilated settings, such as households, healthcare facilities, and prisons.

3. Healthcare Settings: Healthcare workers and individuals receiving healthcare services, particularly in facilities with inadequate infection control measures, may face an increased risk of TB transmission.
4. Living Conditions: Overcrowded living conditions, poor ventilation, and inadequate access to healthcare contribute to the spread of TB, especially in densely populated urban areas and impoverished communities.
5. Substance Abuse: Substance abuse, including alcohol and drug use, can weaken the immune system and increase susceptibility to TB infection and disease progression.
6. Tobacco Use: Smoking tobacco increases the risk of TB infection and progression to active disease. It also worsens TB-related outcomes and increases the risk of TB-related death.
7. Age: Infants, young children, and the elderly are at higher risk of developing TB disease due to their weaker immune systems.
8. Migration and Travel: Traveling or migrating from areas with high TB prevalence to areas with low prevalence can increase the risk of TB transmission and disease acquisition.
9. Health Disparities: Socioeconomic factors, including poverty, lack of access to healthcare, and inadequate nutrition, contribute to disparities in TB incidence and outcomes.
10. Drug Resistance: Exposure to inadequate or incomplete TB treatment can lead to the development of drug-resistant TB strains, which are more difficult to treat and pose a greater public health threat.

CLINICAL MANIFESTATIONS

Tuberculosis (TB) can manifest with a variety of clinical symptoms and signs, depending on factors such as the site of infection and the immune status of the individual. The most common form of TB is pulmonary TB, which affects the lungs.

Clinical manifestations of pulmonary TB include:

- Cough: Persistent cough lasting more than two weeks, sometimes with the production of sputum, which may be bloody.

- Chest Pain: Pain or discomfort in the chest, especially during breathing or coughing.
- Fever: Low-grade fever, usually in the late afternoon or evening.
- Fatigue: Generalized weakness, fatigue, and malaise.
- Weight Loss: Unintentional weight loss and loss of appetite.
- Night Sweats: Profuse sweating, particularly at night.
- Shortness of Breath: Difficulty breathing, especially with exertion or as the disease progresses.
- Haemoptysis: Coughing up blood or blood-stained sputum.
- In addition to pulmonary TB, the disease can affect other organs, leading to extrapulmonary TB. Clinical manifestations of extrapulmonary TB depend on the site of infection and may include:
- Lymph Nodes: Enlarged lymph nodes, especially in the neck (cervical lymphadenopathy).
- Central Nervous System: Headaches, confusion, seizures, and focal neurological deficits in cases of tuberculous meningitis or brain tuberculoma.
- Bone and Joints: Joint pain, swelling, and restricted movement due to tuberculous arthritis.
- Genitourinary System: Painful urination, haematuria, and flank pain due to renal involvement.
- Gastrointestinal Tract: Abdominal pain, diarrhoea, and gastrointestinal bleeding due to gastrointestinal TB.

PATHOPHYSIOLOGY OF TUBERCULOSIS

The pathophysiology of tuberculosis (TB) involves a complex interaction between the Mycobacterium tuberculosis bacterium and the host immune response.

- Infection and Transmission: TB is transmitted through the inhalation of droplets containing Mycobacterium tuberculosis. Once inhaled, the bacteria reach the alveoli of the lungs, where they are phagocytosed by alveolar macrophages.

- Granuloma Formation: In most cases, the host immune response contains the infection by forming granulomas, which are organized collections of immune cells, primarily macrophages and T lymphocytes, surrounding the infected macrophages. Granulomas serve to limit bacterial growth and prevent dissemination.
- Tubercle Formation: Within the granuloma, infected macrophages and other immune cells release cytokines and chemokines, recruiting additional immune cells to the site of infection. Over time, the granuloma may develop into a tubercle, characterized by central caseous necrosis surrounded by a fibrous capsule.
- Bacterial Persistence: Despite the immune response, some bacteria within the granuloma can persist in a dormant or latent state, allowing the infection to remain latent for years without causing symptoms.
- Reactivation: In some individuals, particularly those with weakened immune systems, the latent infection can reactivate, leading to the development of active TB disease. Reactivation may occur due to factors such as HIV infection, malnutrition, aging, or immunosuppressive medications.
- Disease Progression: In active TB disease, the bacteria multiply within the granulomas and may disseminate to other organs, leading to a range of clinical manifestations depending on the site of infection.
- Host Immune Response: The host immune response plays a critical role in determining the outcome of TB infection. Factors such as the balance between pro-inflammatory and anti-inflammatory cytokines, the activation of specific immune cells (e.g., CD4+ T cells, CD8+ T cells), and the formation of tissue-destructive granulomas influence disease progression and severity.

SYMPTOMS OF TUBERCULOSIS

The symptoms of tuberculosis (TB) can vary depending on whether the infection is active or latent, as well as the site of infection within the body. Here are the common symptoms associated with active TB:

- Cough: Persistent cough that lasts for more than two weeks, sometimes producing sputum or blood.

- Fever: Low-grade fever, particularly in the late afternoon or evening.
- Night Sweats: Profuse sweating, especially during sleep.
- Fatigue: Generalized weakness, tiredness, and malaise.
- Weight Loss: Unintentional weight loss and loss of appetite.
- Chills: Episodes of chills or shivering.
- Chest Pain: Pain or discomfort in the chest, especially during breathing or coughing.
- Shortness of Breath: Difficulty breathing, especially with exertion or as the disease progresses.
- Haemoptysis: Coughing up blood or blood-stained sputum.

In addition to these respiratory symptoms, TB can also affect other organs in the body, leading to extrapulmonary TB. The symptoms of extrapulmonary TB depend on the site of infection and may include:

- Enlarged lymph nodes, especially in the neck, armpits, or groin.
- Headaches, confusion, seizures, and focal neurological deficits in cases of tuberculous meningitis.
- Joint pain, swelling, and restricted movement due to tuberculous arthritis.
- Painful urination, haematuria, and flank pain due to renal involvement.
- Abdominal pain, diarrhoea, and gastrointestinal bleeding due to gastrointestinal TB.

COMPLICATIONS

Tuberculosis (TB) can lead to various complications, particularly if left untreated or inadequately treated. Some of the common complications associated with TB include:

1. Disseminated TB: TB can spread from the lungs to other parts of the body, leading to disseminated or extrapulmonary TB. This can affect organs such as the brain, spine, kidneys, and bones, causing serious illness and complications.

2. Miliary TB: Miliary TB is a severe form of disseminated TB where the bacteria spread through the bloodstream, causing tiny TB lesions to form in multiple organs throughout

the body, resembling millet seeds. This condition can be life-threatening if not promptly diagnosed and treated.

3. Tuberculous Meningitis: TB can infect the membranes covering the brain and spinal cord, leading to tuberculous meningitis. This condition can cause symptoms such as severe headaches, confusion, seizures, and coma, and it can be fatal if not treated promptly.

4. Spinal Tuberculosis (Pott‘s Disease): TB can infect the spine, leading to spinal tuberculosis or Pott‘s disease. This can cause back pain, spinal deformities, paralysis, and neurological complications if the infection affects the spinal cord.

5. Drug-Resistant TB: Inadequate or incomplete treatment of TB can lead to the development of drug-resistant strains of the bacteria, such as multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB). These forms of TB are more difficult to treat and require prolonged and costly treatment regimens, often with less effective and more toxic drugs.

6. HIV Coinfection: TB and HIV frequently coexist, as HIV weakens the immune system, increasing the risk of TB infection and progression to active disease. TB can also accelerate the progression of HIV/AIDS and worsen clinical outcomes in HIV-infected individuals.

7. Respiratory Complications: In severe cases of pulmonary TB, respiratory complications such as pneumothorax (collapsed lung), bronchiectasis, and respiratory failure can occur, particularly if cavities develop in the lungs.

8. Nutritional Deficiencies: TB can lead to malnutrition and weight loss due to decreased appetite, metabolic changes, and increased energy expenditure. Malnutrition can worsen the immune response and increase the risk of complications.

DIAGNOSIS

The diagnosis of tuberculosis (TB) involves a combination of clinical evaluation, imaging studies, laboratory tests, and microbiological confirmation. Here are the common methods used for diagnosing TB:

1. Medical History and Physical Examination: Healthcare providers assess the patient‘s medical history, including symptoms, risk factors, and exposure to TB. A physical examination may reveal signs such as abnormal lung sounds or lymph node enlargement.

2. Tuberculin Skin Test (TST) or Mantoux Test: The TST involves injecting a small amount of purified protein derivative (PPD) tuberculin into the skin of the forearm. A positive reaction, indicated by a raised, hardened area at the injection site within 48 to 72 hours, suggests TB infection. However, TST cannot distinguish between latent TB infection and active TB disease.

3. Interferon-Gamma Release Assays (IGRAs): IGRAs, such as the QuantiFERON-TB Gold test and the T-SPOT.TB test, measure the release of interferon-gamma by T cells in response to TB-specific antigens. These tests are more specific than TST and do not cross-react with Bacillus Calmette-Guérin (BCG) vaccination or most non-TB mycobacterial infections.

4. Chest X-ray: Chest X-ray is used to detect abnormalities in the lungs suggestive of TB, such as infiltrates, cavities, or pleural effusions. However, chest X-ray findings alone cannot confirm the diagnosis of TB.

5. Sputum Smear Microscopy: Microscopic examination of sputum samples for the presence of acid-fast bacilli (AFB) using Ziehl-Neelsen or fluorescent stains is a rapid and inexpensive method for diagnosing pulmonary TB. However, it has limited sensitivity, particularly in patients with paucibacillary disease.

6. Sputum Culture: Mycobacterial culture of sputum samples is the gold standard for diagnosing TB and identifying drug-resistant strains. Culture allows for the isolation and identification of Mycobacterium tuberculosis, as well as susceptibility testing to guide appropriate treatment.

7. Nucleic Acid Amplification Tests (NAATs): NAATs, such as the Xpert MTB/RIF assay, detect TB-specific nucleic acid sequences (e.g., DNA or RNA) in sputum samples with high sensitivity and specificity. These tests can also detect rifampicin resistance, a marker for multidrug-resistant TB.

8. Other Imaging Studies: Computed tomography (CT) scans or magnetic resonance imaging (MRI) may be used to evaluate extrapulmonary TB or to assess complications such as lymphadenopathy, pleural effusion, or central nervous system involvement.

MANAGEMENT:

The management of tuberculosis (TB) involves a combination of pharmacological treatment, patient education, infection control measures, and supportive care. Here are the key components of TB management:

1. Drug Therapy: Pharmacological treatment of TB typically consists of a combination of antibiotics taken over several months. The most common drugs used for TB treatment include isoniazid, rifampicin, pyrazinamide, and ethambutol. Treatment regimens vary depending on factors such as the site of infection, drug susceptibility testing results, and the presence of drug-resistant TB.

2. Directly Observed Therapy (DOT): DOT involves observing patients taking their TB medications to ensure adherence to treatment and reduce the risk of drug resistance. This may be implemented through healthcare facilities, community health workers, or family members.

3. Patient Education and Counselling: Patient education plays a crucial role in TB management, including information about the importance of medication adherence, potential side effects of treatment, infection control measures, and the need for follow-up appointments.

4. Infection Control Measures: Preventing the transmission of TB is essential to reduce the spread of the disease. Infection control measures may include ensuring adequate ventilation in healthcare facilities, using personal protective equipment (e.g., masks), isolating infectious patients, and screening and treating close contacts.

5. Monitoring and Follow-Up: Regular monitoring of patients on TB treatment is necessary to assess treatment response, monitor for adverse effects, and ensure medication adherence. Follow-up appointments allow healthcare providers to adjust treatment regimens as needed and address any concerns or complications.

6. Nutritional Support: Malnutrition is common among individuals with TB and can worsen clinical outcomes. Nutritional support, including dietary counselling and supplementation, if necessary, can help improve treatment outcomes and promote recovery.

7. Management of Complications: Complications of TB, such as drug toxicity, drug interactions, and immune reconstitution inflammatory syndrome (IRIS), may require specific management strategies tailored to the individual patient‘s needs.

8. Contact Tracing: Identifying and screening individuals who have been in close contact with TB patients is essential to detect new cases early and prevent further transmission of the disease.

9. Treatment of Latent TB Infection: Individuals with latent TB infection may benefit from treatment to prevent progression to active TB disease. Treatment options may include isoniazid monotherapy or combination regimens such as rifampicin plus isoniazid.

DIETARY GUIDELINES FOR THE MANAGEMENT OF TUBERCULOSIS

Dietary guidelines play a crucial role in the management of tuberculosis (TB). Consuming the right foods can help boost the immune system, aid recovery and prevent further complications. Here are some dietary guidelines to consider:

1. Consume Protein-Rich Foods

Protein is crucial for the growth, repair and maintenance of tissues in the body, and TB patients require more protein to aid recovery. Protein-rich foods like eggs, lean meat, poultry, fish, beans, lentils and nuts are excellent sources of protein. Consuming protein-rich foods can help prevent muscle loss, promote healing and strengthen the immune system.

2. Consume High-Calorie Foods

TB patients often require extra calories to help aid recovery. Consuming high-calorie foods can provide the necessary energy. Some high-calorie foods that can be included in the Tuberculosis diet are avocados, cheese, nuts, peanut butter, whole milk, yoghurt, dried fruits, dark chocolate and granola bars. It's important to consume these foods in moderation and not rely on them exclusively for calorie intake.

3. Consume Micronutrients

Micronutrients are essential nutrients that are required in small amounts for overall health and well-being. These include vitamins and minerals, which play a crucial role in maintaining the immune system, aiding recovery and preventing further complications. Consuming foods rich in micronutrients like dark green leafy vegetables, berries, citrus fruits, nuts and seeds can help ensure that the body is getting the necessary nutrients to function properly.

4. Consume Superfoods

Superfoods like turmeric, garlic, ginger and green tea have anti-inflammatory and immune-boosting properties that can aid in the management of TB. Turmeric contains curcumin, which has been found to have antimicrobial and anti-inflammatory effects. Garlic and ginger have immune-boosting properties that can help fight infections, while green tea is rich in antioxidants that can help protect against cell damage.

5. Consume High-Energy Foods

TB patients may experience loss of appetite, nausea and weight loss, which can lead to weakness and fatigue. Consuming high-energy foods like smoothies, soups and stews can provide the necessary nutrients in an easily digestible form. These foods can be rich in carbohydrates, protein and healthy fats, providing the necessary energy to aid recovery and prevent further complications.

6. Focus on Good Carbs

Good carbs are an essential part of a healthy diet, especially for TB patients. Good carbs are complex carbohydrates that are rich in fibre and nutrients, such as whole-grain bread, pasta and

brown rice. These foods provide the body with sustained energy and help stabilise blood sugar levels, preventing spikes and crashes that can lead to fatigue and weakness.

7. Invest in Good Fats

Investing in good fats is important for TB patients as they need healthy sources of energy to aid in recovery. Good fats, such as those found in avocados, nuts, seeds and olive oil, are rich in essential fatty acids that can help improve heart health, brain function and immune system function. These fats can also help regulate inflammation in the body, which can help TB patients reduce the risk of developing complications.

8. Ensure Taking Ample Vegetables and Minerals

Vegetables are a great source of vitamins and minerals that can help boost the immune system and aid recovery in TB patients. Dark green leafy vegetables, such as spinach and kale, are rich in vitamins A, C and K, as well as iron and calcium. Other vegetables like carrots, sweet potatoes and bell peppers are high in antioxidants and fibre, which can help prevent cell damage and improve digestive health.

9. Foods Rich in B-Complex Vitamins

B-complex vitamins are important for maintaining good health, especially in the context of tuberculosis management. Foods rich in B-complex vitamins include leafy green vegetables, whole grains, nuts and seeds, legumes, dairy products, meat, fish and poultry. Consuming a balanced diet with these foods can help support the immune system and promote overall health during TB treatment.

10. Foods Rich in Zinc

Zinc is an essential mineral that plays a crucial role in maintaining a healthy immune system, wound healing and growth and development. Some foods that are rich in zinc include oysters, beef, pork, chicken, nuts, beans, whole grains and dairy products. Adequate intake of zinc is particularly important for individuals with TB, as zinc deficiency can impair immune function and increase susceptibility to infection

Food items to limit or avoid in Tuberculosis

Processed Foods: sugars and Processed foods such as canned foods, packaged snacks and sugary cereals should be avoided as they are often high in unhealthy fats, sugars and artificial additives.

High-Fat Meats: Limit the intake of high-fat meats such as red meat, sausage and bacon, as these foods can be difficult to digest and can worsen symptoms of TB.

Fried Foods: Fried foods are high in unhealthy fats and can increase inflammation in the body, making it harder to fight off TB infection.

Refined Grains: Foods made with refined grains such as white bread, pasta and rice should be limited as they are low in fibre and can cause blood sugar spikes.

Sugary Drinks: Beverages like soda, sports drinks, and energy drinks should be avoided as they are high in sugar and can weaken the immune system.

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MALARIA

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INTRODUCTION

Malaria is a life-threatening disease caused by Plasmodium parasites, transmitted to humans through the bites of infected female Anopheles mosquitoes. The parasites multiply in the liver and then infect red blood cells, leading to symptoms such as fever, chills, and flu-like illness. If not promptly treated, malaria can result in severe complications and death. According to the World Health Organization (WHO), malaria remains a significant public health challenge, particularly in sub-Saharan Africa, where the majority of cases and deaths occur. Efforts to control malaria include the use of insecticide-treated bed nets, indoor residual spraying, antimalarial drugs, and ongoing research into vaccines and new treatments.

CLASSIFICATION

Malaria is classified into several types based on the species of Plasmodium parasites that cause the infection. The most common types include:

1. Plasmodium falciparum: This species is responsible for the majority of malaria-related deaths worldwide, particularly in sub-Saharan Africa. It is known for causing severe malaria with complications such as cerebral malaria, severe anemia, and multi-organ failure.

2. Plasmodium vivax: This species is widespread and causes a significant portion of malaria cases outside of Africa. P. vivax can form dormant liver stages (hypnozoites), leading to relapses months or even years after the initial infection.

3. Plasmodium ovale: This species is less common but can cause relapsing malaria similar to P. vivax due to the formation of hypnozoites.

4. Plasmodium malariae: This species typically causes a milder form of malaria with less severe symptoms. However, it can persist in the bloodstream for many years, leading to chronic infections.

5. Plasmodium knowlesi: This species primarily infects macaque monkeys but can also infect humans, particularly in Southeast Asia. It has gained attention as an emerging cause of malaria in humans.

RISK FACTORS

Several risk factors contribute to the transmission and prevalence of malaria:

1. Location: Malaria is most prevalent in tropical and subtropical regions, particularly in sub-Saharan Africa, where transmission is highest. Other regions with high malaria transmission include parts of Asia, Latin America, and the Middle East.

2. Vector Exposure: People living in areas with high mosquito populations, particularly those where Anopheles mosquitoes thrive, are at increased risk of malaria. Factors such as outdoor activities and inadequate housing without proper screening or mosquito control measures increase exposure to mosquito bites.

3. Travel: Traveling to malaria-endemic regions without taking appropriate preventive measures, such as antimalarial medication and mosquito bite prevention strategies, increases the risk of contracting malaria.

4. Immunity: Individuals with limited or no immunity to malaria, such as young children, pregnant women, and individuals from non-endemic areas, are at higher risk of severe malaria if infected.

5. Occupational Exposure: Certain occupations, such as forestry workers, miners, and agricultural workers, may increase the risk of malaria due to outdoor exposure in endemic areas.

6. Climate and Environmental Factors: Climate conditions, such as temperature and rainfall patterns, influence mosquito breeding habitats and malaria transmission. Environmental factors, such as deforestation, irrigation projects, and stagnant water bodies, can create breeding sites for mosquitoes, increasing malaria risk.

7. Socioeconomic Factors: Poverty, limited access to healthcare, inadequate housing, and lack of resources for vector control contribute to higher malaria burden in disadvantaged communities.

8. Drug Resistance**: Resistance to antimalarial drugs, particularly in Plasmodium falciparum, can limit treatment options and increase the risk of treatment failure.

CLINICAL MANIFESTATIONS

The clinical manifestations of malaria vary depending on the species of Plasmodium involved, the level of immunity of the infected individual, and other factors. Common symptoms include:

1. Fever: Fever is a hallmark symptom of malaria, often occurring in cycles corresponding to the replication cycles of the parasite.

2. Chills: Patients with malaria commonly experience chills, which may accompany the onset of fever.

3. Headache: Headaches are frequent and can be severe in malaria patients.

4. Muscle aches: Muscle aches and fatigue are common symptoms of malaria.

5. Nausea and vomiting: Gastrointestinal symptoms such as nausea and vomiting can occur, especially in severe cases.

6. Anemia: Malaria parasites destroy red blood cells, leading to anemia, which can cause fatigue and weakness.

7. Enlarged spleen: In some cases, the spleen may become enlarged due to the body's immune response to the infection.

8. Jaundice: Severe malaria can lead to jaundice, characterized by yellowing of the skin and eyes due to liver dysfunction.

9. Severe complications: In severe cases, malaria can lead to complications such as cerebral malaria (impaired consciousness, seizures), severe anemia, acute respiratory distress syndrome (ARDS), renal failure, and multi-organ failure, which can be life-threatening if not promptly treated.

SYMPTOMS

It's important to note that the clinical presentation of malaria can vary widely, and some individuals, particularly those with partial immunity, may experience milder or atypical symptoms.

The symptoms of malaria typically include:

1. Fever
2. Chills
3. Sweating
4. Headaches
5. Nausea and vomiting
6. Muscle and joint pain
7. Fatigue
8. Loss of appetite
9. Abdominal pain
10. Diarrhea
11. Cough
12. Enlarged spleen
13. Jaundice (yellowing of the skin and eyes)

In severe cases, malaria can lead to complications such as cerebral malaria (impaired consciousness, seizures), severe anemia, acute respiratory distress syndrome (ARDS), renal failure, and multi-organ failure.

COMPLICATIONS

Complications of malaria can be severe and potentially life-threatening. Some of the complications include:

1. Cerebral malaria: A serious complication characterized by impaired consciousness, seizures, and neurological deficits. It can lead to coma and death if not treated promptly.

2. Severe anemia: Malaria parasites destroy red blood cells, leading to anemia, which can result in fatigue, weakness, and organ damage.

3. Acute respiratory distress syndrome (ARDS): Severe malaria can cause fluid accumulation in the lungs, leading to difficulty breathing and respiratory failure.

4. Renal failure: Malaria-induced complications can impair kidney function, leading to renal failure and the accumulation of toxins in the body.

5. Multi-organ failure: Severe malaria can affect multiple organs, including the liver, kidneys, and brain, leading to organ failure and death.

6. Hypoglycemia: Malaria can cause low blood sugar levels, especially in children, leading to weakness, confusion, and coma.

7. Pregnancy complications: Malaria infection during pregnancy can increase the risk of maternal anemia, low birth weight, premature birth, and stillbirth.

DIAGNOSIS

The diagnosis of malaria typically involves clinical evaluation combined with laboratory testing. Common diagnostic methods include:

1. Microscopic examination of blood smears: This is considered the gold standard for malaria diagnosis. A trained technician examines a stained blood smear under a microscope to identify the presence of malaria parasites and determine the species. This method allows for accurate quantification of parasite density and detection of low-level parasitaemia.

2. Rapid diagnostic tests (RDTs): RDTs are simple immunochromatographic tests that detect specific malaria antigens (such as histidine-rich protein 2 or Plasmodium lactate dehydrogenase)

in a blood sample. RDTs provide rapid results (usually within 15-30 minutes) and are particularly useful in settings with limited access to laboratory facilities or trained personnel.

3. Molecular diagnostic tests: Polymerase chain reaction (PCR) tests can detect and identify malaria parasites' genetic material with high sensitivity and specificity. PCR is especially valuable for detecting low-level parasitaemia, differentiating between species, and confirming cases in patients with atypical symptoms or in areas with low transmission.

4. Clinical assessment: In areas where laboratory testing is not readily available, healthcare providers may diagnose malaria based on clinical symptoms and epidemiological factors, such as travel history to malaria-endemic regions.

MANAGEMENT

The management of malaria involves prompt diagnosis and appropriate treatment to alleviate symptoms, clear the parasite from the bloodstream, and prevent complications. The World Health Organization (WHO) recommends the following strategies for malaria management:

1. Prompt diagnosis: Malaria should be diagnosed promptly using laboratory tests such as microscopy, rapid diagnostic tests (RDTs), or molecular diagnostic tests (PCR). Early diagnosis allows for timely initiation of treatment.

2. Antimalarial treatment: The choice of antimalarial treatment depends on factors such as the species of Plasmodium, the severity of the infection, the patient's age, pregnancy status, and drug resistance patterns in the region. Commonly used antimalarial medications include artemisinin-based combination therapies (ACTs) such as artemether-lumefantrine, artesunate-amodiaquine, and dihydroartemisinic-piperaquine. Other drugs such as chloroquine, quinine, and atovaquone-proguanil may be used in specific circumstances.

3. Treatment of severe malaria: Severe malaria requires hospitalization and parenteral antimalarial therapy with intravenous (IV) artesunate or quinine, followed by oral therapy to complete the treatment course. Supportive care, including management of complications such as hypoglycemia, anemia, and respiratory distress, is essential.

4. Prevention of complications: Prompt and effective treatment of malaria helps prevent complications such as cerebral malaria, severe anemia, and multi-organ failure.

5. Vector control: In addition to treatment, vector control measures such as the use of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and environmental management are crucial for reducing malaria transmission and preventing new infections.

6. Chemoprevention: In certain populations, such as pregnant women and travelers to malaria-endemic areas, chemoprevention with antimalarial medications may be recommended to prevent malaria infection.

7. Monitoring and surveillance: Regular monitoring of malaria cases, drug resistance patterns, and vector control interventions is essential for guiding malaria control efforts and adjusting treatment policies as needed.

DIETARY RECOMMENDATIONS

Energy

Simple sources of carbohydrates that do not need much digestion and provide instant energy are at the top of the list. With low appetite and bouts of fever, you need to refuel the body fast. Glucose water, electoral, sugarcane juice, coconut water, sherbets of rose or khus, orange squash are instant energy drinks that replenish fluids and provide energy. Toffees, chocolates, ice creams are other items that can be added to the list. Chilled foods also help reduce nausea.

Proteins

Proteins are a must have as our immune system feeds on proteins, and foods like dals, chicken and eggs are some of the right options to add to your diet. Dals can be made into a soup and flavoured with lemon; cakes are a good way to consume eggs when appetite is low and taste buds need to be cajoled. Milk, lassi and yogurt add not just proteins but valuable minerals. Yogurt also adds probiotics, which help overcome gastric symptoms and improve taste and digestion.

Vitamins

Vitamins from fruits, fresh fruit juices help not just the immune system but also are good sources of energy and electrolytes. Citrus fruits help fight the infection with Vitamin C; red and yellow fruits help add Vitamin A, which is a crucial nutrient for our immune system and a potent antioxidant. Fruits like guava add both Vitamin C and Iron to help build up RBC'S.

Nuts And Seeds

Nuts and seeds are small powerhouses of several healthy nutrients along with healthy fats and proteins. Soaked almonds, or crushed almond in milk, give a lot in small amounts. They are also rich in phytonutrients, which help fight the antioxidant stress caused by the infection

Herbs And Spices

Ajwain-infused water will help keep the digestion healthy. Ajwain being a strong carminative, it well prevents bloating and gas. Saunf is another digestion booster. Turmeric, as we all know, has anti-inflammatory and antioxidant abilities. It also enhances the immune system.

- Keep Yourself Hydrated

Clean drinking water up to 3-3.5 litres a day to keep yourself hydrated even if you have a fever. Dehydration can put you at risk of further deterioration of your condition. So, stay hydrated!

Foods to Avoid:

High Fibre Food

Very high fibre foods may not sit well in your tummy, so give it a break. Whole grains, whole dals and vegetables with tough fibres may cause gas and indigestion.

Spicy Food and Sauces

Avoid very spicy and rich sauces. The chatpata food may sound good but it could trigger of a reaction because of the medication you are taking.

Junk Food

This one is a no brainer! Junk food is a big no when you are suffering from Malaria. Having junk food may even worsen your condition. Junk food is not healthy for a normal body, and during illness, it may wreak havoc.

Avoid Caffeine

Excessive intake of coffee, tea, or any strong caffeine drink may disturb your digestive system.

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DENGUE FEVER

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INTRODUCTION

Dengue fever, caused by the flavivirus transmitted through Aedes mosquitoes, is a significant global health concern. Characterized by flu-like symptoms, severe cases can lead to hemorrhagic fever or shock. According to the World Health Organization (WHO), an estimated 390 million dengue infections occur annually. The impact on tropical and subtropical regions underscores the urgency for effective prevention and control strategies.

RISK FACTORS

Various risk factors contribute to the transmission and severity of dengue fever. These include:

1. Mosquito Vector Density: High Aedes mosquito population increases the risk of dengue transmission.

2. Urbanization: Rapid urban growth often leads to inadequate sanitation and water management, creating breeding grounds for mosquitoes.

3. Climate Conditions: Warm and humid climates favor mosquito breeding, facilitating the spread of dengue in tropical and subtropical regions.

4. Previous Dengue Infection: Individuals previously infected with one dengue serotype may face increased risk of severe illness upon subsequent infections with a different serotype.

5. Age and Immune Status: Children and individuals with weakened immune systems are at higher risk of severe dengue.

6. Travel to Endemic Areas: Visiting regions where dengue is prevalent increases the risk of contracting the virus.

7. Lack of Effective Mosquito Control: Inadequate mosquito control measures contribute to increased transmission.

CLINICAL MANIFESTATIONS

Clinical manifestations of dengue fever encompass a range of symptoms. Reference to the World Health Organization (WHO) guidelines provides a comprehensive overview. Common features include abrupt onset of high fever, severe headaches, joint and muscle pain, rash, and mild bleeding manifestations such as petechiae or gum bleeding (WHO, Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Control, Timely recognition and medical intervention are critical for effective management.

Dengue fever presents a spectrum of clinical manifestations, including:

1. Sudden Onset of High Fever: Abrupt onset of a high fever is a common early symptom.

2. Severe Headache and Pain Behind the Eyes: Intense headaches and eye pain, often referred to as "breakbone fever."

3. Joint and Muscle Pain: Severe joint and muscle pain, leading to the term "bone-breaking fever."

4. Rash: A rash may appear a few days after the onset of fever, usually on the arms, legs, and face.

5. Mild Bleeding: Petechiae (small red or purple spots), nosebleeds, or gum bleeding can occur.

6. Nausea and Vomiting: Gastrointestinal symptoms, including nausea and vomiting, may be present.

7. Fatigue: Prolonged fatigue and weakness can persist after the fever resolves.

SYMPTOMS

Dengue fever symptoms, as outlined by the World Health Organization (WHO), include sudden onset of high fever, severe headaches, joint and muscle pain, skin rash, mild bleeding manifestations (petechiae, nosebleeds, or gum bleeding), and symptoms such as nausea and

vomiting (WHO, Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Awareness of these symptoms is essential for early detection and appropriate medical care.

COMPLICATIONS

Dengue fever can lead to severe complications, and referencing the World Health Organization (WHO) provides insights into these potential outcomes. Complications may include severe plasma leakage, severe bleeding, or organ impairment. Severe dengue, also known as dengue hemorrhagic fever or dengue shock syndrome, can be fatal if not promptly and adequately treated (WHO, Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Control. Seeking medical attention for proper diagnosis and management is crucial, especially when warning signs of severe dengue manifest.

- Severe Plasma Leakage: Leading to shock and fluid accumulation in the abdomen and chest.
- Severe Bleeding: Resulting in hemorrhage, often seen as nosebleeds, gum bleeding, or internal bleeding.
- Organ Impairment: In severe cases, organs such as the liver, heart, and lungs may be affected.

DIAGNOSIS

The diagnosis of dengue fever typically involves a combination of clinical assessment and laboratory testing. The World Health Organization (WHO) provides guidelines for the diagnosis of dengue, emphasizing the importance of considering both clinical and epidemiological factors. Laboratory tests, such as the detection of viral RNA or NS1 antigen, are commonly used for confirmation. Serological tests, like IgM and IgG antibody assays, also play a role in diagnosis (WHO, Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Control, 2009). Consulting these guidelines and relevant health authorities ensures accurate and up-to-date information on dengue fever diagnosis.

MANAGEMENT

The World Health Organization (WHO) provides comprehensive guidelines for the management of dengue fever. The key components include fluid replacement, pain control with acetaminophen (avoiding NSAIDs), rest, and careful monitoring of vital signs and haematocrit levels. In severe cases with warning signs or complications, hospitalization for intensive care may be necessary (WHO, Dengue: Guidelines for Diagnosis, Treatment, Prevention, and Control, consulting these guidelines and collaborating with healthcare professionals ensures optimal care for individuals with dengue fever.

DIETARY GUIDELINES

Low **platelet counts**, severe weakness and dehydration are all possible outcomes of dengue fever, and the diet regimen should be planned cautiously to avert that. A well-planned diet is essential for the speedy recovery of patients.

Good Hydration

Maintaining hydration is essential to prevent **dehydration**, mainly if you are having vomiting or diarrhoea. These symptoms can cause the body to deplete fluids. Water, clear soup, tender coconut water and electrolyte drinks can help to correct fluid imbalances. However, avoid sugary drinks and beverages, which may lead to further dehydration.

Eat Small Meals

Eating small frequent meals is preferred rather than having large meals. This can help ensure that the body gets the right amount of nutrients without putting excess strain on the gastrointestinal system.

Add Nutrient-dense Foods

Body can trigger the immune system by adding foods abundant in vitamins and minerals to the diet regimen. Fruits, veggies, legumes, and whole grains are all impressive sources of vital nutrition.

Limit High-fat And High-fibre Rich Foods

It is ideal to refrain from foods that are loaded with fat or fibre. These foods can be difficult to digest and could exert a lot of pressure on the digestive system.

Foods to Eat and Drink During Dengue

Power-up with Vitamin C Foods

Vitamin C is one of the essential nutrients that should be part of your diet during dengue. Attributed with potent anti-viral and antioxidative traits, vitamin C helps in building a robust immune system. In addition, it helps in absorbing another powerful nutrient iron from the intestine. Some of the vitamin C-rich foods you should incorporate include oranges, lemon, papaya, guava etc., and vegetables like green leafy vegetables, capsicum, cauliflower, broccoli etc.

Papaya extract is an impressive source of digestive enzymes such as papain and chymopapain. These enzymes are known to promote digestion, ease bloating and treat other digestive problems. Also, fresh papaya leaf juice helps in treating dengue by improving platelet counts.

Add Iron Rich Foods

The platelet counts drop during dengue fever. Your system requires a good amount of iron to maintain your blood haemoglobin levels and produce platelets. Platelets are vital in the blood clotting process of the system and hence it is important to prevent blood loss, which is common during dengue. Thus, having iron-rich foods like lean meat, beans and legumes, green leafy vegetables and dates may help in delivering iron. This will help in boosting platelet counts and speed up the recovery process.

Load Vitamin K Foods

Vitamin K is another essential nutrient that assists in improving the platelet count. In addition, it also helps in blood clotting, which is beneficial in managing dengue fever. Try including foods

rich in vitamin K like sprouts, broccoli, cabbage, spinach, kale, kiwi, avocado, and lean meat to recuperate well.

Calorie Dense Foods

Your body becomes weak and exhausted when infected by the dengue virus. Include plenty of energy-dense foods such as rice, milk, and potato, that would help you regain lost strength and convalescence well.

Adequate Fluids

Water is quintessential for the system, particularly when you are recovering from dengue fever. Ensure to drink plenty of fluids including water, tender coconut water, clear soup, and rice porridge as they are packed with electrolytes (like potassium, calcium, sodium and magnesium). Fluids will maintain your electrolyte balance and keep you well-hydrated.

Foods to Avoid

- Caffeine

  Caffeine is a diuretic that causes the system to lose water via urine. Thus, it is vital to restrict caffeinated beverages when suffering from dengue fever, as the body needs to stay hydrated.
- **Spicy Foods**

  Spicy Foods can irritate the stomach and damage the intestinal wall due to excess acid secretion. It is ideal to have a soft and bland diet and avoid species.
- **Fatty Foods**

  The digestion of food is usually slow with dengue fever, making it hard for the stomach to break down fatty foods. So, it is essential to restrict foods like fatty meat cuts, butter, and deep-fried foods. This will reduce the strain on your intestine and use that energy to help you regain strength.

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CHIKUNGUNYA

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INTRODUCTION

Chikungunya is a viral illness transmitted to humans by infected mosquitoes, primarily Aedes aegypti and Aedes albopictus. It is characterized by symptoms such as fever, severe joint pain, muscle pain, headache, fatigue, and rash. Although rarely fatal, the joint pain can be debilitating and persist for months. Outbreaks have been reported in Africa, Asia, Europe, and the Americas. Preventive measures include reducing mosquito breeding sites and using insect repellents.

Incubation period

CHIK virus causes an acute febrile illness with an incubation period of 3-7 days (can be 2- 12 days,). Viraemia persists for up to 5 days from the onset of symptoms. Fever and arthralgia are the hallmark of Chikungunya fever.

Clinical Features: Clinical presentation of Chikungunya is divided in to three phases. In Chikungunya mostly symptoms have an abrupt onset with high grade fever, single or multiple joint pains, skin rashes, headache and myalgia. Clinical presentation of Chikungunya usually follows 3 phases which are as follows: a) Acute phase: Less than 3 weeks b) Sub-acute phase: > 3 weeks to 3 months c) Chronic phase: > 3 months Clinical presentation may be mild, moderate or severe and most of the symptoms subside within 3 weeks from the onset of symptoms. Some of the symptoms may persist for 3 months and even more. Usually 10 – 15 % of the patient those who present with severe Chikungunya progress to Sub-acute or chronic phase.

RISK FACTORS

Geographical Location:

Living in or traveling to regions where chikungunya is prevalent increases the risk of exposure to the virus.

Mosquito Exposure:

Spending time in areas with high mosquito activity, especially during peak biting times (dawn and dusk), increases the risk of mosquito bites and potential transmission of the chikungunya virus.

Lack of Mosquito Control Measures:

Inadequate mosquito control measures in residential areas, such as standing water accumulation and insufficient use of insecticides, can lead to higher mosquito populations and greater risk of chikungunya transmission.

Outdoor Activities:

Engaging in outdoor activities without proper protective measures, such as wearing long-sleeved clothing and using mosquito repellent, increases the risk of mosquito bites and chikungunya infection.

Immune Status:

Individuals with weakened immune systems, such as the elderly, young children, pregnant women, and those with certain medical conditions, may be at higher risk of developing severe symptoms if infected with the chikungunya virus.

CLINICAL MANIFESTATION OF CHIKUNGUNYA

Fever: Chikungunya infection often begins with a sudden onset of high fever, usually above 39°C (102°F).

Joint Pain: Severe and debilitating joint pain, particularly in the wrists, ankles, knees, and fingers, is a hallmark symptom of chikungunya. The joint pain can be persistent and may last for weeks to months.

Muscle Pain: Patients with chikungunya commonly experience intense muscle pain, which can be severe and affect mobility.

Headache: Headaches are common during the acute phase of chikungunya infection and may be accompanied by other symptoms such as photophobia (sensitivity to light) and phonophobia (sensitivity to sound).

Rash: A rash may develop in some individuals, typically appearing 2 to 5 days after the onset of fever. The rash is usually maculopapular and may involve the trunk, limbs, and face.

Other Symptoms: Other symptoms of chikungunya infection may include fatigue, nausea, vomiting, conjunctivitis, and swollen lymph nodes.

PATHOPHYSIOLOGY OF CHIKUNGUNYA

The pathophysiology of chikungunya involves the interaction between the chikungunya virus (CHIKV) and the human immune system, leading to the clinical manifestations of the disease.

Transmission: Chikungunya virus is primarily transmitted to humans through the bite of infected Aedes mosquitoes, particularly Aedes aegypti and Aedes albopictus.

Viral Replication: After the mosquito bite, the chikungunya virus enters the human bloodstream and infects various cell types, including skin fibroblasts, muscle cells, and immune cells.

Immune Response: The innate immune response is activated upon recognition of the viral components, leading to the production of proinflammatory cytokines and chemokines. This early immune response helps to control viral replication and recruit immune cells to the site of infection.

Adaptive Immune Response: The adaptive immune system, including both cellular and humoral immunity, plays a crucial role in clearing the virus from the body. T lymphocytes (CD4+ and CD8+) target infected cells, while B lymphocytes produce antibodies against the virus.

Inflammatory Response: Excessive production of proinflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), contributes to the systemic inflammation observed in chikungunya infection. This inflammatory cascade is responsible for many of the clinical symptoms, including fever, joint pain, and muscle pain.

Joint Involvement: The exact mechanism underlying the joint pain in chikungunya is not fully understood but is believed to involve both viral replication in joint tissues and immune-mediated inflammation. The virus may directly infect synovial cells and cause joint inflammation, leading to arthralgia and arthritis.

Persistent Symptoms: In some cases, chikungunya infection can lead to persistent joint pain and other chronic symptoms, which may be due to ongoing viral replication, immune dysregulation, or autoimmune mechanisms.

SYMPTOMS OF CHIKUNGUNYA

Symptoms of chikungunya usually develop between three and seven days after an infected mosquito bites you, although some people have symptoms as early as two days or as long as 12 days after a mosquito bite.

Fever and joint pain are the most common symptoms of chikungunya virus. The intensity of symptoms can vary depending on the person. Many people feeling crippling joint pain. The fever usually begins suddenly. Some people can have such mild symptoms that they mistake the virus for another illness or don‘t visit a healthcare provider.

Other symptoms could include:

Headache.

Muscle pain.

Swelling in your joints.

Rash.

Fatigue.

Nausea.

Most people experience symptoms for about one week and go on to make a full recovery. Although some people have chronic joint pain after recovery.

DIAGNOSIS OF CHIKUNGUNYA

Laboratory diagnosis of Chikungunya fever

As the clinical manifestations of Chikungunya fever resemble those of dengue and other fevers caused by arthropod borne viruses of the Alphavirus genus, laboratory diagnosis is critical to establish the cause of diagnosis and initiate specific public health response.

Types of Laboratory tests available and specimens required: Laboratory criteria include a decreased lymphocyte count consistent with viremia. However, a definitive laboratory diagnosis can be accomplished through three main laboratory tests: virus isolation, serological test and molecular technique of Polymerase Chain Reaction (PCR). Specimen is usually blood or serum but in neurological cases with meningoencephalitis feature, CSF (cerebrospinal fluid) may also be sent.

Virus isolation Virus isolation provides the most definitive diagnosis, but takes one to two weeks for completion and must be carried out in biosafety level III laboratories to reduce the risk of viral transmission. The technique involves exposing specific cell lines to samples from whole blood and identifying chikungunya virus-specific responses. The isolation process is time consuming and the degree of success is dependent on a number of complicating factors, for example, time of collection, transportation, maintenance of cold chain, storage and processing of samples.

Serological diagnosis Serological diagnosis requires a larger amount of blood than the other methods, and uses an ELISA assay to measure chikungunya-specific IgM levels in the blood

serum. Chikungunya antibody tests are generally appropriate after the first week of symptom onset and onward. Serum obtained from 10-15 ml of whole blood is required. An acute phase serum must be collected immediately after the onset of illness and the convalescent phase serum 10-14 days later. The blood specimen is transported at 4° Celsius and not frozen for immediate transfer to the laboratory. Only if the testing cannot be done immediately, the serum specimen should be separated and then stored and shipped frozen. ELISA test is quite specific with very little cross reactivity with related alphaviruses. Serologic diagnosis can be made by demonstration of four-fold rise in antibody titre in acute and convalescent sera or by demonstrating IgM antibodies specific for CHIK virus. A commonly used test is the Immunoglobulin M Antibody (IgM) capture enzyme-linked immunosorbent assay (MAC-ELISA). Results of MAC-ELISA can be available within same day.

RT-PCR Reverse Transcriptase, (RT) PCR technique using nested primer pairs is used to amplify several Chikungunya-specific genes from whole blood, generating thousands to millions of copies of the genes in order to identify them. The Chikungunya virus reverse transcriptase (RT)-PCR assay is appropriate in the early days of symptom onset, since CHIKV RNA can be detected during the acute phase of illness (≤8 days after symptom onset). RT-PCR can also be used to quantify the viral load in the blood. Using RT-PCR, diagnostic results can be available in one to two days.

The technique is used for diagnosing CHIK virus using nested primer pairs amplifying specific components of three structur0al gene regions, Capsid (C), Envelope E-2 and part of Envelope E1. A specimen for PCR is exactly similar to the one for virus isolation i.e. heparinized whole blood.

COMPLICATIONS OF CHIKUNGUNYA

Persistent Joint Pain: Chronic joint pain and arthritis can persist for months to years after the acute phase of the illness, significantly affecting quality of life for some individuals.

Neurological Complications: Rare but severe neurological complications such as encephalitis, meningitis, and Guillain-Barré syndrome (GBS) have been reported in association with chikungunya infection.

Cardiovascular Complications: Chikungunya infection has been associated with cardiovascular manifestations such as myocarditis and pericarditis, although these are relatively uncommon.

Ophthalmic Complications: Chikungunya virus can cause ocular complications such as uveitis (inflammation of the uvea), retinitis, and optic neuritis.

Miscarriage and Congenital Infection: Pregnant women infected with chikungunya virus may be at risk of miscarriage, preterm birth, and vertical transmission of the virus to the fetus, leading to congenital chikungunya syndrome.

Exacerbation of Underlying Conditions: Chikungunya infection can exacerbate underlying medical conditions, such as diabetes, cardiovascular disease, and respiratory illnesses, particularly in older adults and individuals with compromised immune systems.

MANAGEMENT

Symptomatic Treatment: Medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen (paracetamol), and analgesics are commonly used to alleviate fever, joint pain, and muscle pain. However, caution should be exercised when using NSAIDs, especially in patients with a history of gastrointestinal ulcers or bleeding disorders.

Rest and Hydration: Adequate rest and hydration are essential for managing chikungunya symptoms. Patients are advised to rest and drink plenty of fluids to prevent dehydration, especially during episodes of fever and sweating.

Physical Therapy: Physical therapy and gentle exercises may help alleviate joint pain and improve mobility during the recovery phase. However, excessive or strenuous physical activity should be avoided to prevent exacerbating joint inflammation.

Monitoring for Complications: Healthcare providers should monitor patients closely for potential complications, such as neurological manifestations, cardiovascular complications, and exacerbation of underlying medical conditions. Prompt intervention may be necessary if complications arise.

Preventive Measures: Preventive measures to reduce mosquito bites and prevent further transmission of chikungunya virus include the use of insect repellents, wearing protective

clothing (long sleeves, pants), and implementing mosquito control measures, such as eliminating breeding sites around the home.

Education and Public Health Measures: Public health education initiatives aimed at raising awareness about chikungunya prevention, symptoms, and management are crucial for community engagement and early detection of cases. Surveillance and vector control efforts are also essential for preventing and controlling chikungunya outbreaks.

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OBESITY

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INTRODUCTION

Assessing where we stand in 2017 is an interesting exercise, now that we are several decades into the US obesity epidemic. On the one hand, there are several areas of progress, including increased awareness, increased political and social will to address obesity, new treatment options, and greatly expanded evidence base for prevention and intervention strategies.

The International Obesity Task Force (IOTF) was established in 1994 to address the increase in the worldwide prevalence of obesity. The goals of the IOTF are to 1) raise awareness in the population and among governments that obesity is a serious medical condition, 2) develop policy recommendations for a coherent and effective global approach to the management and prevention of obesity, and 3) implement appropriate strategies to manage and prevent obesity on a population basis worldwide. To assess the global prevalence of obesity in children and adolescents, the IOTF convened a workshop on childhood obesity to determine the most appropriate measurement to assess obesity in populations of children and adolescents around the world. At the workshop, a variety of issues related to this problem were considered—including the best measure of fatness, the effect of application of a variety of existing standards on the prevalence of obesity in the same population, and the role of factors such as visceral adiposity and natural history in the definition of obesity. This article and those that follow represent the information presented at the workshop. The workshop concluded that the body mass index (BMI; in kg/m2) offered a reasonable measure with which to assess fatness in children and adolescents and that the standards used to identify overweight and obesity in children and adolescents should agree with the standards used to identify grade 1 and grade 2 overweight (BMI of 25 and 30, respectively) in adults.

The prevalence of child and adolescent obesity In the United States increased dramatically between 1970 and 2000, and there are few indications that the rates of childhood obesity are decreasing. Obesity is associated with myriad medical, psychological, and neurocognitive abnormalities that impact children‘s health and quality of life. Genotypic variation is important in determining the susceptibility of individual children to undue gains in adiposity; however, the rapid increase in pediatric obesity prevalence suggests that changes to children‘s environments and/or to their learned behaviors may dramatically affect body weight regulation. This paper presents an overview of the epidemiology, consequences, and etiopathogenesis of pediatric obesity, serving as a general introduction to the subsequent papers in this Special Issue that address aspects of childhood obesity and cognition in detail.

The prevalence of obesity has increased substantially in the past in almost all countries of the world, and a further increase is expected for the future. Besides the well-established effects on type 2 diabetes and cardiovascular disease, there is convincing evidence today that obesity also increases the risk of several types of cancer, including colorectal cancer, postmenopausal breast cancer, endometrial cancer, renal cell carcinoma, esophageal adenocarcinoma, pancreatic cancer, and liver cancer. Obesity probably also increases the risk of ovarian cancer, advanced prostate cancer, gallbladder cancer, and gastric cardia cancer. For some cancer types, there is also some evidence that weight gain during adulthood increases cancer risk, e.g., colorectal cancer, postmenopausal breast cancer, endometrial cancer, and liver cancer. However, for most cancers, it is an open question as to whether vulnerability to weight gain in relation to cancer risk depends on specific life periods. There are a number of plausible mechanisms that may explain the relationship between obesity and cancer risk, including pathways related to insulin resistance, inflammation, and sex hormones. For most cancers, there is only limited evidence that weight loss in adulthood decreases cancer risk, which is primarily due to the limited long-term success of weight loss strategies among obese individuals. There is limited evidence suggesting that obesity may also be associated with poor prognosis among patients with colorectal cancer, breast cancer, endometrial cancer, ovarian cancer, and pancreatic cancer. Taken together, these findings support efforts to prevent weight gain on an individual level as well as on a population level. Whether and to what extent overweight or obese cancer patients benefit from weight loss strategies is unclear and needs to be addressed in future studies.

The World Health Organisation recommends exclusive breastfeeding until 6 months of age and continued breastfeeding until 2 years of age or beyond. Appropriate complementary foods should be introduced in a timely fashion, beginning when the infant is 6 months old. In developing countries, early or inappropriate complementary feeding may lead to malnutrition and poor growth, but in countries such as the United Kingdom and United States of America, where obesity is a greater public health concern than malnutrition, the relationship to growth is unclear. We conducted a systematic review of the literature that investigated the relationship between the timing of the introduction of complementary feeding and overweight or obesity during childhood. Electronic databases were searched from inception until 30 September 2012 using specified keywords. Following the application of strict inclusion/exclusion criteria, 23 studies were identified and reviewed by two independent reviewers. Data were extracted and aspects of quality were assessed using an adapted Newcastle–Ottawa scale. Twenty-one of the studies considered the relationship between the time at which complementary foods were introduced and childhood body mass index (BMI), of which five found that introducing complementary foods at< 3 months (two studies), 4 months (2 studies) or 20 weeks (one study) was associated with a higher BMI in childhood. Seven of the studies considered the association between complementary feeding and body composition but only one study reported an increase in the percentage of body fat among children given complementary foods before 15 weeks of age. We conclude that there is no clear association between the timing of the introduction of complementary foods and childhood overweight or obesity, but some evidence suggests that very early introduction (at or before 4 months), rather than at 4–6 months or> 6 months, may increase the risk of childhood overweight.

Overweight and obesity occur when *excess fat accumulation* (globally, regionally, and in organs as ectopic lipids) *increases risk for adverse health outcomes*. Like other chronic diseases, this definition does not require manifestation of an obesity-related complication, simply that the risk for one is increased. This allows for implementation of weight management strategies targeting treatment and prevention of these related conditions. It is important to point out that thresholds of excess adiposity can occur at different body weights and fat distributions depending on the person or population being referenced.

Overweight is a condition of excessive fat deposits.

Obesity is a chronic complex disease defined by excessive fat deposits that can impair health. Obesity can lead to increased risk of type 2 diabetes and heart disease, it can affect bone health and reproduction, it increases the risk of certain cancers. Obesity influences the quality of living, such as sleeping or moving.

The diagnosis of overweight and obesity is made by measuring people‘s weight and height and by calculating the body mass index (BMI): weight (kg)/height^2 (m^2). The body mass index is a surrogate marker of fatness and additional measurements, such as the waist circumference, can help the diagnosis of obesity.

The BMI categories for defining obesity vary by age and gender in infants, children and adolescents.

Adults

For adults, WHO defines overweight and obesity as follows:

overweight is a BMI greater than or equal to 25; and

obesity is a BMI greater than or equal to 30.

For children, age needs to be considered when defining overweight and obesity.

Children under 5 years of age

For children under 5 years of age:

overweight is weight-for-height greater than 2 standard deviations above WHO Child Growth Standards median; and

obesity is weight-for-height greater than 3 standard deviations above the WHO Child Growth Standards median.

CLASSIFICATION AND TYPES OF OBESITY

Childhood obesity, defined as having a body mass index (BMI) that exceeds the 95th percentile, is a growing epidemic with devastating health, academic, and social-emotional implications. Childhood obesity is also associated with a wide range of psychosocial problems including low self-esteem, poor self-image, peer victimization, externalizing behaviors, body dissatisfaction, poor interpersonal relationships, increased suicidal behaviors, and stigmatization. The intent of this special issue is to highlight the devastating effects of childhood obesity and promote a knowledge-based surrounding evidence-based treatment, programs, and policies that may influence effective weight management in children. In sum, the contributions of this issue underscore the significance of the burgeoning epidemic of childhood obesity. Reducing the prevalence of childhood obesity is a public health challenge, and schools have the potential to play a powerful role in diminishing this serious health crisis.

Childhood obesity is a modern epidemic situation and is the most frequent eating disorder. The importance of childhood obesity has driven many researchers to investigate and study its etiology, consequences, and interventions for managing it. Purpose: To review the factors which contribute to the development of childhood obesity, its consequences, and the interventions for managing the problem. Results: It follows from the review that childhood obesity is associated with the presence of genetic, behavioral, and environmental factors, while its consequences are connected with cardiovascular, gastrointestinal, and respiratory diseases. Certain psychosocial and endocrine disorders are also observed. Management of the problem can be achieved with targeted interventions in the context of the family, school and community environment. Conclusions: Childhood obesity is directly connected with the adoption of a sedentary lifestyle, a change in healthy eating habits, and reduced physical activity. Apart from the serious risks it causes to children, this problem considerably affects the cost of healthcare and social security systems.

Childhood obesity is a serious global health problem. It begins early in life during the preschool years, and, for many, the obesity is carried forward into later childhood and adult life. Once established, obesity is difficult to reverse and is associated with poorer health outcomes in the

short and long term. Tackling obesity in childhood is important to reduce life-long risk and protect health.

Adult obesity is a prevalent health issue characterized by an excessive accumulation of body fat, often assessed using body mass index (BMI). A BMI of 30 or higher indicates obesity. This condition is associated with various health risks, including type 2 diabetes, heart disease, stroke, certain cancers, and mental health disorders. Causes of adult obesity include genetic predisposition, unhealthy dietary habits, sedentary lifestyle, and socioeconomic factors. Management involves lifestyle changes, behavioral therapy, medication, and, in severe cases, bariatric surgery.

Prevalence: Obesity rates have been steadily increasing worldwide over the past few decades. According to the World Health Organization (WHO), the global prevalence of obesity nearly tripled between 1975 and 2016. In 2016, more than 1.9 billion adults were overweight, and of these, over 650 million were obese.

Health Implications: Obesity significantly increases the risk of developing various chronic health conditions, including:

- Type 2 diabetes: Obesity is a major risk factor for insulin resistance and type 2 diabetes.

- Cardiovascular diseases: Obesity is associated with hypertension, dyslipidemia, coronary artery disease, and heart failure.

- Stroke: Obesity increases the risk of ischemic and hemorrhagic stroke.

- Certain cancers: Obesity is linked to an increased risk of cancers such as breast, colon, prostate, and endometrial cancer.

- Respiratory disorders: Obesity can lead to sleep apnoea, asthma, and decreased lung function.

- Musculoskeletal disorders: Obesity contributes to osteoarthritis, back pain, and reduced mobility.

- Mental health issues: Obesity is associated with depression, anxiety, and low self-esteem.

Causes and Risk Factors: The development of obesity is influenced by a combination of genetic, environmental, behavioral, and socioeconomic factors. These include:

- Genetic predisposition: Certain genetic variations can predispose individuals to obesity.

- Unhealthy diet: Consumption of high-calorie, high-fat, and processed foods contribute to weight gain.

- Sedentary lifestyle: Lack of physical activity and prolonged sitting increase the risk of obesity.

- Socioeconomic factors: Factors such as lower income, limited access to healthy foods, and neighborhood environments that discourage physical activity can contribute to obesity.

Management and Treatment: The management of obesity involves a multifaceted approach, including:

- Lifestyle modifications: Dietary changes, regular physical activity, and behavioral therapy are key components of obesity management.

- Pharmacotherapy: Medications may be prescribed to aid weight loss in conjunction with lifestyle changes.

- Bariatric surgery: Surgical interventions such as gastric bypass or sleeve gastrectomy may be recommended for individuals with severe obesity who have not achieved weight loss with other methods.

Public Health Interventions: Governments, healthcare organizations, and public health agencies implement various strategies to address the obesity epidemic, including:

- Promoting healthy eating habits and physical activity through public education campaigns.

- Implementing policies to improve access to nutritious foods and safe recreational spaces.

- Collaborating with communities, schools, workplaces, and healthcare providers to support obesity prevention and management efforts.

Classification of obesity with respect to morbidity

Obesity is defined as an excess accumulation of adipose tissue in the body. Since its etiology and pathogenesis are quite heterogenous, classification of disease types by specific purposes is essential for proper clinical management. Although classification by the etiology given in Table I is useful for diagnosing the primary disease inducing obesity, it would be more meaningful to classify simple obesity into subgroups and determine pathological features of each disease type, since simple obesities without specific identifiable cause are much more often encountered in daily medical care.

From this viewpoint, classification by morphological types of fat cells or by sites of adipose tissue accumulation has been carried out as shown in Parts II and III of Table I, and features of each disease type have been reviewed. This paper explains the above in detail, and describes a method of classification that incorporates the concept of visceral fats in comparison with conventional methods. As for the accumulation sites, visceral fat can be analyzed by a newly developed method.

Classification by Cytological Features of Adipose Tissue

This is to classify obesities by cellularity of the adipose tissue biopsied, i.e., by the size and number of fat cells (1). The following classifications are employed because metabolic disorders accompanying obesity are related to the size of fat cells and because of the relationship between the proliferative capacity of fat cells and the onset of obesity.

Hyperplastic Type. Fat cells are normal in size, but are increased in number. Fat cells are said to actively proliferate from the embryonal period (the last trimester of pregnancy) to 1 year after birth, and again during puberty. This type is, therefore, often seen among obese people with onset in youth. Since fat cells are not large, metabolic disorders rarely occur. The treatment for weight reduction is not so effective.

The initial step in evaluation of obesity is calculation of BMI. To measure BMI, one begins by weighing the patient in underclothes and no shoes. Height is measured without shoes. BMI is calculated by dividing weight (in kilograms) by square height (in meters). When measuring

weight in pounds and height in inches, the weight is divided by the square height and the quotient is multiplied by 703, as BMI is always reported and interpreted in kilograms per square meter. Most clinicians have an available BMI table (Figure 1) that casily

RISK FACTORS/ AETIOLOGY:

1. Genetics: Genetic predisposition plays a significant role in obesity. Studies have identified numerous genes associated with obesity risk, including FTO, MC4R, and others.

2. Dietary Habits: High consumption of energy-dense foods, sugary beverages, and processed foods contributes to obesity.

3. Sedentary Lifestyle: Lack of physical activity or sedentary behavior increases the risk of weight gain and obesity.

4. Psychological Factors: Emotional eating, stress, depression, and poor coping mechanisms can lead to overeating and weight gain.

5. Environmental Factors: Environmental factors such as easy access to unhealthy food options, urbanization, and food marketing influence dietary choices and contribute to obesity.

6. Medical Conditions: Certain medical conditions like hypothyroidism, Cushing's syndrome, and polycystic ovary syndrome (PCOS) can predispose individuals to obesity.

7. Medications: Some medications, such as certain antidepressants, antipsychotics, corticosteroids, and antiepileptic drugs, may cause weight gain as a side effect.

8. Social and Economic Factors: Socioeconomic status, education level, and cultural factors can impact access to healthy foods, opportunities for physical activity, and healthcare services, influencing obesity risk.

9. Energy Imbalance: Obesity often results from a prolonged energy imbalance, where caloric intake exceeds energy expenditure, leading to the accumulation of excess body fat.

10. Neuroendocrine Regulation: Dysregulation of hormones involved in appetite regulation, such as leptin, ghrelin, and insulin, can disrupt energy balance and contribute to obesity.

11. Metabolic Factors: Impaired metabolism, insulin resistance, and alterations in lipid metabolism can promote weight gain and obesity.

12. Gut Microbiota: The composition of gut microbiota influences energy extraction from food, inflammation, and metabolic processes, potentially contributing to obesity.

13. Epigenetics: Environmental factors can influence gene expression through epigenetic mechanisms, impacting adipogenesis, appetite regulation, and metabolism, and contributing to obesity risk.

14. Inflammatory Pathways: Chronic low-grade inflammation, often associated with obesity, can disrupt metabolic homeostasis and contribute to insulin resistance and weight gain.

CLINICAL MANIFESTATIONS/ PATHOPHYSIOLOGY

Obesity arises from a persistent imbalance between calorie intake and expenditure, fueled by excessive consumption of high-fat, high-calorie foods and compounded by sedentary lifestyles. Adipose tissue, acting as both an energy reservoir and an endocrine organ, undergoes structural changes in obesity, releasing inflammatory molecules that contribute to systemic inflammation. Insulin resistance, a hallmark of obesity, hampers the body's responsiveness to insulin, leading to hyperinsulinemia and metabolic irregularities. Hormonal imbalances, including leptin resistance and reduced adiponectin levels, disrupt appetite regulation and exacerbate inflammation and insulin sensitivity. Alterations in gut microbiota composition further perpetuate obesity-related metabolic disturbances. While genetic predisposition plays a significant role, environmental factors such as diet, physical activity, socioeconomic status, and cultural influences also contribute to obesity risk. The central nervous system, particularly the hypothalamus, orchestrates appetite, energy utilization, and metabolism, with dysregulation of these pathways implicated in the development and persistence of obesity.

Obesity presents with evident physical manifestations, including a notable increase in body weight, often characterized by a BMI exceeding 30 kg/m^2. Additionally, individuals may exhibit an increased waist circumference, indicative of abdominal obesity and heightened risk for metabolic complications. Skin changes such as stretch marks (striae), skin tags, and darkened patches (acanthosis nigricans) in skin folds are common. Breathlessness frequently accompanies

obesity due to the excess weight impacting lung capacity and function. Furthermore, fatigue is prevalent, largely attributed to decreased mobility and reduced levels of physical activity.

Obesity is intricately linked with metabolic disturbances, notably increasing the risk of type 2 diabetes mellitus, driven by insulin resistance. Hyperlipidemia, characterized by elevated cholesterol and triglyceride levels, heightens the susceptibility to cardiovascular diseases. Hypertension often coexists with obesity, arising from structural and functional alterations in blood vessels. Metabolic syndrome, comprising insulin resistance, hypertension, and abnormal lipid levels, is prevalent among individuals with obesity. Non-alcoholic fatty liver disease (NAFLD), characterized by liver fat accumulation, is strongly associated with obesity and insulin resistance.

Obesity substantially elevates the risk of cardiovascular complications, including coronary artery disease attributed to atherosclerosis and heart failure resulting from structural and functional changes in the heart. Additionally, the likelihood of stroke is heightened due to obesity's association with cerebrovascular disease.

Obesity exerts considerable strain on the musculoskeletal system, leading to conditions such as osteoarthritis, as excess weight places undue stress on weight-bearing joints, contributing to degeneration and pain. Chronic back pain is prevalent among individuals with obesity, stemming from mechanical stress on the spine. Decreased mobility and impaired physical function further exacerbate musculoskeletal issues.

Beyond physical health implications, obesity engenders psychological and social challenges. Depressive symptoms and anxiety disorders are more prevalent among individuals with obesity, reflecting the psychosocial burden of the condition. Low self-esteem often arises due to negative body image perceptions and societal stigma surrounding obesity. Additionally, obese individuals may encounter social discrimination and bias in various spheres of life, including employment opportunities and access to healthcare services.

SYMPTOMS

Symptoms of obesity can vary from person to person, but commonly include:

1. Increased Body Weight: Obesity is defined by having a body mass index (BMI) of 30 or higher. This is calculated by dividing weight in kilograms by height in meters squared.

2. Fatigue: Carrying excess weight can lead to fatigue and reduced energy levels due to increased strain on the body.

3. Breathlessness: Obesity can cause difficulty in breathing, especially during physical activity, due to the extra weight pressing on the chest and lungs.

4. Joint Pain: Excess weight can put pressure on joints, leading to pain and stiffness, particularly in the knees, hips, and lower back.

5. Sleep Problems: Obesity is associated with sleep apnoea, a condition where breathing repeatedly stops and starts during sleep. It can also cause snoring and disrupted sleep patterns.

6. Skin Issues: Obesity can lead to skin problems such as chafing, rashes, and infections in skin folds due to moisture and friction.

7. High Blood Pressure: Obesity increases the risk of developing hypertension, which can lead to heart disease, stroke, and other complications.

8. Type 2 Diabetes: Obesity is a major risk factor for developing type 2 diabetes, characterized by high blood sugar levels due to insulin resistance.

9. Heart-Disease: Obesity contributes to the development of cardiovascular diseases, including coronary artery disease, heart attack, and stroke.

10. Psychological Effects: Obesity can negatively impact mental health, leading to depression, low self-esteem, and body image issues.

These symptoms may vary in severity depending on individual factors such as genetics, lifestyle, and overall health status.

COMPLICATIONS

1. Cardiovascular Complications:

• Hypertension: Obesity is a major risk factor for hypertension, a condition characterised by elevated blood pressure, which increases the risk of heart disease, stroke, and kidney disease.

• Coronary Artery Disease (CAD): Obesity contributes to the development of CAD by promoting atherosclerosis, leading to reduced blood flow to the heart and an increased risk of heart attacks and angina.

• Heart Failure: The increased workload on the heart in obese individuals can lead to heart failure, a condition where the heart is unable to pump blood effectively to meet the body‘s needs.

2. Metabolic Complications:

• Type 2 Diabetes Mellitus: Obesity is strongly associated with insulin resistance and impaired glucose metabolism, leading to the development of type 2 diabetes, a chronic condition characterised by high blood sugar levels.

• Dyslipidemia: Obesity often results in abnormal lipid profiles, including elevated triglycerides, LDL cholesterol, and decreased HDL cholesterol levels, contributing to cardiovascular disease risk.

3. Respiratory Complications:

• Obstructive Sleep Apnea (OSA): Obesity is a primary risk factor for OSA, a sleep disorder characterised by repeated episodes of upper airway obstruction during sleep, leading to fragmented sleep and daytime fatigue.

• Asthma: Obesity increases the risk of asthma and worsens asthma control, possibly due to mechanical factors and systemic inflammation associated with obesity.

4. Gastrointestinal Complications:

• Gastro oesophageal Reflux Disease (GERD): Obesity predisposes individuals to GERD, a chronic condition where stomach acid refluxes into the oesophagus, causing heartburn and potential complications such as oesophageal ulcers and strictures.

• Non-alcoholic Fatty Liver Disease (NAFLD): Obesity is a significant risk factor for NAFLD, a spectrum of liver disorders ranging from simple steatosis (fatty liver) to non-alcoholic steatohepatitis (NASH) and cirrhosis.

5. Musculoskeletal Complications:

• Osteoarthritis: Obesity increases the risk of osteoarthritis by placing excessive mechanical stress on weight-bearing joints, such as the knees and hips, leading to cartilage degeneration and joint pain.

• Low Back Pain: Obesity is associated with an increased risk of chronic low back pain, possibly due to mechanical factors, inflammation, and altered biomechanics.

6. Cancer Risk:

Obesity is linked to an increased risk of various cancers, including breast, colorectal, endometrial, kidney, pancreatic, and oesophageal cancers. Adipose tissue produces hormones and cytokines that can promote tumour growth and progression.

7. Psychological Complications:

• Obesity is associated with psychological issues such as depression, anxiety, and poor body image, which can adversely affect mental well-being and quality of life.

DIAGNOSIS

Diagnosing obesity is a multifaceted process that involves assessing several factors to determine an individual‘s overall health and risk factors. The primary tool used in diagnosis is the body mass index (BMI), calculated using an individual‘s height and weight. A BMI of 30 or above is generally considered indicative of obesity. However, healthcare providers also take into account waist circumference, as excess abdominal fat can increase health risks.

Furthermore, a comprehensive medical history is crucial in diagnosing obesity, as certain underlying conditions or medications may contribute to weight gain. Physical examinations may also be conducted to evaluate for signs of obesity-related health issues such as hypertension, dyslipidemia, or insulin resistance.

Laboratory tests may be ordered to assess for metabolic abnormalities commonly associated with obesity, such as elevated blood glucose levels, dyslipidemia, or liver function abnormalities.

In some cases, additional diagnostic imaging studies like dual-energy X-ray absorptiometry (DEXA) scans or magnetic resonance imaging (MRI) may be used to accurately measure body fat distribution and assess for visceral adiposity.

Ultimately, a thorough and individualised approach is necessary for diagnosing obesity, considering both quantitative measurements and qualitative assessments of overall health.

Diagnosing obesity involves a comprehensive evaluation of various factors beyond just body weight. Key elements include:

1. Body Mass Index (BMI): BMI is calculated using an individual‘s height and weight and is a primary tool for diagnosing obesity. A BMI of 30 or higher generally indicates obesity.
2. Waist Circumference: Excess abdominal fat, indicated by a waist circumference of 35 inches (88 cm) or more in women and 40 inches (102 cm) or more in men, increases the risk of obesity-related health issues.
3. Medical History: A thorough review of medical history helps identify underlying conditions or medications that may contribute to weight gain or obesity-related complications.
4. Physical Examination: Healthcare providers conduct physical exams to assess for signs of obesity-related health problems such as hypertension, dyslipidemia, or insulin resistance.
5. Laboratory Tests: Blood tests may be performed to assess for metabolic abnormalities associated with obesity, including elevated blood glucose levels, dyslipidemia, or liver function abnormalities.
6. Imaging Studies: Advanced imaging techniques such as dual-energy X-ray absorptiometry (DEXA) scans or magnetic resonance imaging (MRI) may be used to accurately measure body fat distribution and assess for visceral adiposity.

An individualised approach considering both quantitative measurements and qualitative assessments of overall health is essential for an accurate diagnosis of obesity.

MANAGEMENT

1. Lifestyle Modifications:

• Dietary interventions: Focus on portion control, nutrient-dense foods, and calorie restriction. Emphasise whole grains, fruits, vegetables, and lean proteins.

• Physical activity: Encourage regular exercise, aiming for at least 150 minutes of moderate-intensity aerobic activity per week.

• Behavioural therapy: Addressing psychological factors contributing to overeating or sedentary behaviour.

2. Pharmacotherapy:

• Prescription medications: Orlistat, Phentermine/Topiramate, Lira glutide, etc., may be prescribed under medical supervision to aid weight loss.

• Considerations: Assess risks and benefits, potential side effects, and patient suitability for pharmacotherapy.

3. Bariatric Surgery:

• Indicated for individuals with severe obesity (BMI ≥ 40) or BMI ≥ 35 with significant co-morbidities.

• Types of surgery: Gastric bypass, sleeve gastrectomy, adjustable gastric banding, etc.

• Postoperative care and long-term follow-up: Dietary modifications, vitamin supplementation, and monitoring for complications.

4. Multidisciplinary Approach:

• Collaboration between healthcare professionals: Physicians, dieticians, psychologists, exercise specialists, and surgeons.

• Individualised treatment plans: Tailored to the patient‘s needs, preferences, and medical history.

• Long-term support: Ongoing monitoring, counselling, and reinforcement of healthy behaviours.

5. Prevention Strategies:

• Public health initiatives: Promote healthy eating habits, physical activity, and community-based programs.

• Education: Raise awareness about the risks of obesity and the importance of early intervention.

• Policy changes: Implementing regulations on food labelling, advertising, and availability of unhealthy foods in schools and public spaces.

6. Behaviour oral Interventions:

• Cognitive-behavioural therapy (CBT): Helps individuals identify and modify unhealthy thought patterns and behaviour ors related to eating and physical activity.

• Mindfulness-based interventions: Techniques such as mindful eating and meditation can promote self-awareness and better management of cravings and emotional eating.

• Motivational interviewing: Counselling approach that helps individuals explore their ambivalence towards behaviour change and develop intrinsic motivation for adopting healthier habits.

7. Nutritional Counselling:

• Individualised meal planning: Dietitian can create personalised meal plans based on dietary preferences, cultural considerations, and nutritional needs.

• Education on portion control: Teaching strategies for managing portion sizes and recognising hunger and fullness cues.

• Monitoring dietary intake: Keeping food journals or using mobile apps to track food consumption and identify areas for improvement.

8. Physical Activity Recommendations:

• Variety of activities: Encourage a mix of aerobic exercise, strength training, and flexibility exercises to improve overall fitness and metabolism.

• Gradual progression: Start with achievable goals and gradually increase intensity and duration to avoid injury and burnout.

• Incorporating lifestyle activities: Encourage activities like walking or cycling for transportation and taking the stairs instead of the elevator.

9. Support Groups and Peer Counselling:

• Group sessions: Provide opportunities for individuals to share experiences, challenges, and successes with others facing similar struggles.

• Online communities: Virtual support groups and forums can offer ongoing support and accountability, especially for individuals with limited access to in-person resources.

• Peer mentoring: Pairing individuals who have successfully managed their weight with those who are just starting their journey can provide valuable guidance and encouragement.

10. Health Monitoring and Follow-Up:

• Regular medical assessments: Monitor weight, blood pressure, blood glucose levels, and lipid profiles to track progress and identify any emerging health concerns.

• Adjusting treatment plans: Periodically review and modify treatment strategies based on individual response, changes in health status, and evolving goals.

• Addressing relapse: Recognise that setbacks are common and provide support and guidance to help individuals get back on track without judgment.

11. Cultural Sensitivity and Health Equity:

• Recognise cultural differences in attitudes towards food, body image, and physical activity and adapt interventions accordingly.

• Address socioeconomic barriers to healthy living, such as limited access to affordable nutritious foods and safe recreational spaces.

• Advocate for policies and programs that promote health equity and address the social determinants of health contributing to obesity disparities.

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UNDERWEIGHT

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INTRODUCTION

Underweight is body weight that is too low for a normal healthy adult or child. It is also known by various other names such as wasting, emaciation, thinness, stunting, etc., and is caused by multiple factors especially lack of adequate nutrients in the body. This type of unhealthy weight is not much publicized in developed wealthy countries because it is not very common except under extreme circumstances like some sick geriatric populations, disabled people, chronic diseases sufferers, the homeless people, refugees, and people afflicted by wars and natural disasters. The discussion about underweight in developed countries seems to be drowned by the chorus on concerns for the obesity epidemic and its consequences as well as the risks of excessive consumption of calories. However underweight is a front burner issue in some developing poor countries where it is a recognized perennial problem that has led to negative health consequences and sometimes death of preschool children, pregnant and lactating mothers as well as very sick, disabled or aged people. Public health professionals as well as nutritionists, social workers, clinicians and educators need to be skilled in recognizing underweight and its implications on health and wellness. Such skills are necessary in order to effectively counsel consumers & patients on food choices and weight management or to give appropriate referrals to affected individuals to enable them get expert help on maintaining healthy weight.

RISK FACTORS

Poor Selection of Food: Poor selection of food along with irregular eating habits may be responsible for insufficient food intake and hence calorie intake. It nlay be due to ignorance or a lack of purchasing power of the family.

Physical Activity and Psychological Factor: Individuals who are tense, nervous and extremely active and who do not rest sufficiently tend to expend more energythan what they are able to eat. This can cause undernutrition.

Mother's Health Status; Poor nutritional status of the girl child coupled with under nutrition during pregnancy results in LBW infant being born. These children born are at a disadvantage right from infancy and may fail to reach optimum weight in adulthood.

Pathologic Conditions: Illness can affect weight status in a number of ways. For example, fevers and infections, increase the demand for energy, which if not met because of poor appetite, lead to loss of weight. Food intake may be severely limited by nausea, vomiting or diarrhoea in gastrointestinal disturbances. Metabolic rate may be greatly increased in hyperthyroidism resulting in underweight. Drug therapy may also alter taste or reduce appetite, leading to weight loss.

Genetic Predisposition: The weight of an individual is inherited basically from his biological mother. In the event of the biological mother being thin, there is 75% likelihood of the individual being thin also.

CLINICAL MANIFESTATIONS OF UNDERWEIGHT

1. Body Mass Index (BMI)

- BMI below 18.5 kg/m² is considered underweight according to the World Health Organization (WHO) classification.

2. Physical Examination

- Visible Ribcage and Bones: Prominence of bones such as ribs, collarbones, and hip bones due to decreased subcutaneous fat.

- Muscle Wasting: Loss of muscle mass, particularly noticeable in extremities and cheeks.

- Sunken Eyes and Cheeks: Hollowed appearance around the eyes and cheeks due to loss of facial fat.

-Thin or Sparse Hair: Hair may appear thin, dull, and brittle due to nutritional deficiencies.

-Dry, Pale Skin: Skin may be dry, flaky, and pale due to inadequate nutrient intake.

-Lack of Subcutaneous Fat**: Reduced padding under the skin, leading to a gaunt or emaciated appearance.

-Decreased Skin Turgor: Skin may lack elasticity and rebound slowly when pinched, indicating dehydration or malnutrition.

3. Laboratory Findings:

- Low Serum Albumin: Decreased levels of albumin, a protein synthesized by the liver, indicating malnutrition.

- Electrolyte Imbalance: Abnormal levels of electrolytes such as potassium, sodium, and calcium due to poor nutrition and fluid balance.

- Anemia: Low red blood cell counts or hemoglobin levels, often caused by deficiencies in iron, vitamin B12, or folate.

- Hypoglycemia: Low blood sugar levels due to inadequate intake of carbohydrates or impaired glucose metabolism.

- Low Serum Cholesterol: Reduced levels of cholesterol, especially HDL (good cholesterol), due to poor dietary fat intake.

- Thyroid Dysfunction: Underweight individuals may experience alterations in thyroid hormone levels, leading to symptoms such as fatigue and cold intolerance.

4. Cardiovascular Examination:

- Bradycardia: Decreased heart rate, often below 60 beats per minute, due to decreased metabolic demands.

- Orthostatic Hypotension**: Drop in blood pressure upon standing, indicating dehydration or volume depletion.

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PATHOPHYSIOLOGY OF UNDERWEIGHT

1. Energy Imbalance:

- Underweight individuals typically have an energy imbalance, where energy expenditure exceeds energy intake. This can result from various factors such as increased metabolic rate, inadequate caloric intake, or excessive physical activity without sufficient calorie consumption.

2. Malnutrition:

- Prolonged inadequate intake of essential nutrients, including proteins, carbohydrates, fats, vitamins, and minerals, can lead to malnutrition in underweight individuals. Malnutrition contributes to impaired cellular function, tissue breakdown, and compromised immune function.

3. Metabolic Adaptations:

- The body undergoes metabolic adaptations in response to chronic energy deficiency. This includes a decrease in basal metabolic rate (BMR), reduced thermogenesis, and alterations in hormone levels such as thyroid hormones and cortisol, which collectively promote energy conservation and weight loss.

4. Muscle Wasting:

- In conditions of prolonged undernutrition, the body may break down muscle tissue to meet energy demands, leading to muscle wasting or atrophy. This process, known as catabolism, results in a loss of lean body mass and contributes to weakness and fatigue in underweight individuals.

5. Fat Depletion:

- Subcutaneous and visceral fat stores are depleted in underweight individuals, leading to a reduction in adipose tissue mass. This decreases the body's ability to store energy, regulate temperature, and provide cushioning and protection for organs.

6. Impaired Immune Function:

- Malnutrition and underweight status compromise immune function, making individuals more susceptible to infections and delaying wound healing. Nutrient deficiencies, particularly in

vitamins A, C, D, and E, as well as zinc and iron, can impair immune responses and increase susceptibility to infectious diseases.

7. Endocrine Dysfunction:

- Underweight individuals may experience hormonal imbalances, including disruptions in the hypothalamic-pituitary-adrenal (HPA) axis and the hypothalamic-pituitary-gonadal (HPG) axis. These hormonal changes can affect appetite regulation, metabolism, and reproductive function, contributing to weight loss and related symptoms.

SYMPTOMS OF BEING UNDERWEIGHT

1. Physical Symptoms:

- Low Body Weight: Body weight significantly below the normal range for height and age.

- Weakness and Fatigue: Feeling constantly tired and lacking energy.

- Brittle Hair and Nails: Hair becomes thin and brittle, and nails may become weak and prone to breakage.

- Dry Skin: Skin may become dry and flaky due to lack of essential nutrients.

- Poor Muscle Tone: Muscles may appear underdeveloped and lack tone.

- Feeling Cold: Underweight individuals may feel cold more easily due to lack of insulation from body fat.

2. Psychological Symptoms:

- Poor Self-Esteem: Negative body image and low self-esteem due to perceived inadequacy in appearance.

- Depression and Anxiety: Underweight individuals may experience symptoms of depression and anxiety related to body image issues and social pressures.

- Social Withdrawal: Avoidance of social situations due to feeling self-conscious about body weight.

3. Digestive Symptoms:

- Loss of Appetite: Underweight individuals may have a reduced desire to eat, leading to further weight loss.

- Digestive Issues: Problems such as constipation, indigestion, and bloating may occur due to inadequate intake of nutrients and fiber.

- Malabsorption: Difficulty absorbing nutrients from food, leading to deficiencies despite adequate intake.

4. Menstrual Irregularities:

Women may experience irregular or absent menstrual cycles due to hormonal imbalances caused by undernutrition.

COMPLICATIONS

Osteoporosis

An underweight person, mostly women, has an increased risk of osteoporosis where the bones become brittle and tend to break.

Skin, hair, or teeth problems

An individual person who doesn't get enough nutrients in their diet can have physical weakening symptoms like thinning skin, hair loss, dry skin, or poor dental health.

Getting sick frequently

If a person who is underweight doesn't get enough energy in their diet, they are prone to get infections in their body which makes them sick again and again. And they may suffer illness which lasts longer in the period.

Feeling Tired

Certain foods provide Calories, which can be taken by the person. A person who does not intake calories cannot keep up the healthy weight and can feel fatigued. Not getting enough calories to keep up a healthy weight can make an individual feel fatigued.

Anemia

An underweight person is more likely to possess less blood count than the standard blood count and a person may feel dizzy, headache, and tired.

Irregular periods

Underweight women often tend to possess irregular periods, or sometimes even menstruation stops. This forms an irregular period in the adolescent person. Irregular periods are the main cause of infertility.

Premature Births

A pregnant woman who is underweight is at a higher risk of pre-term labour as underweight women might not get enough nutrients the baby may need. Hence leading to premature births.

Slow Growth

The Younger generation needs nutrients to develop healthy bones to avoid slow growth. Being underweight can stop the correct growth of the person and the person may not develop as expected. Being underweight has an increased risk for mortality compared to people with a mean BMI.

MANAGEMENT

Energy: The total calorie intake should be 500 to 1000 Kcal in excess of the daily needs in order to result a gain in weight by half to one kilogram in a week. Thus, if you need 2000 Kcal for your normal activity, you require 2500-3000 Kcal per day for weight gain. We can also compute the energy requirements on the basis of ideal body weight (as discussed in subsection 9.5.1 of this unit). The patient may be given 30-35 Kcal per Kg ideal body weight per day. The calories should be increased gradually over a period of one or two weeks to avoid digestive disturbances.

Proteins: Proteins are required for tissue building, as well as, to take care of the daily wear and tear. Underweight individuals generally have depleted lean body mass and poor reserves of amino acids/blood proteins. Thus, the patient may benefit by consuming around 1.2 g per kg body weight of proteins per day. A combination of both animal and plant protein should be

incorporated but emphasis should be laid on the inclusion of easy to digest forms of protein such as half boiled egg, steamed boiled/sauteed flesh food etc.

Fats: We know that fats are concentrated source of energy (lg = 9 Kcals). Fats are capable of increasing the energy value of the diet without adding much bulk to it. Add extra fat gradually, a sudden increase in fatty foods like butter, cream and oil may produce diarrhoea. About 30% of calories should come from unsaturated sources of fat.

Carbohydrates: Liberal amounts of easy to digest carbohydrates should be included in the diet. The intake of dietary fibre should be minimized so as to prepare meals which are nutrient dense and have a small volume. Include more of high calorie vegetable like potatoes, Colocasia and yam instead of raddish, cucumber, leafy vegetables which are low in the carbohydrate content. All cereals provide high calories at low cost and should provide about 55-65% of total kilocalories.

Vitamins and Minerals: If the diet provides good amounts of fresh fruits and vegetables, vitamin or mineral supplements are usually not required. However, if the patient indicates clinical signs of a severe nutritional deficiency, it may be imperative to use supplements or employ other essential medical measures.

Fluids: Take fluids only after a meal instead of with or before meals so that food intake is not reduced. High calorie nourishing beverages such as milk shakes, egg nog should be preferred over low nutrient beverages such as cold-drinks, barley water, plain soda etc.

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CARDIOVASCULAR DISEASES

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INTRODUCTION

Cardiovascular disease is a class of diseases that involve the cardiovascular system. They include diseases of the coronary arteries that supply the heart muscle with oxygen and nutrients; diseases of arteries such as the carotid artery that provide blood flow to the brain; and diseases of the peripheral arteries that carry blood throughout the body. Worldwide, cardiovascular disease is the leading cause of death, causing about a third of all deaths each year. Cardiovascular diseases (CVDs) are a group of disorders affecting the heart and blood vessels, and they are the leading cause of death globally, taking an estimated 17.9 million lives each year. This means that more people die from CVDs than from any other cause, including cancer and respiratory diseases. Coronary heart disease (CHD) is the most common type of CVD, and it is caused by the buildup of plaque in the arteries that supply blood to the heart. This plaque buildup can narrow the arteries, reducing blood flow to the heart and eventually leading to a heart attack.

RISK FACTORS

The factors that play a role in causing or increasing the risk of getting cardiovascular disease are called risk factors. Figure 1 illustrates these risk factors. As you may have noticed in the figure, these factors are classified as modifiable and non-modifiable risk factors. Modifiable risk factors are those which we have control over. For example, obesity, smoking, high blood pressure, high cholesterol, physical inactivity etc. By themselves, they are major risk factors, which increase our risk of developing CHD. Positive healthy living, smokefree air, good nutrition, regular physical activity, and supportive living and working environments can go a long way in preventing CHD. Non-modifiable risk factors are those that we have no control over such as hereditary, age, gender etc.

1. Family history: People who already have the disease in their family are more prone to getting heart disease. Genetic factors greatly influence the risk of developing premature cardiovascular diseases.

2. Obesity: As you know obesity or excessive weight is the primary cause of cardiovascular disease. It is an independent risk factor for heart disease. Obesity is generally associated with elevated triglyceride, elevated low-density lipids, increased blood pressure and impaired glucose tolerance. Weight reduction improves these abnormalities. Keeping the body mass index within the normal range (18.5-24.9) can be helpful in retarding the onset of CAD. Remember we read about BMI in unit 9. Further, android form of obesity makes us more prone to heart diseases as compared to the gynoid form of obesity. Thus, abdominal fat is considered more harmful than fat on the hips as you may recall studying in Unit 9. This can be measured by waist/hip ratio (WHR). Normal WHR is 0.85 for females and 1.0 for males.

3. Hypertension or high blood pressure: It is also one of the risk factors of cardiovascular disease and is frequently accompanied by hyperlipidemia (excess lipids in the blood). Increased coronary artery wall tension is believed to accelerate the atherosclerotic process by stimulating arterial smooth muscle cell hyperplasia and hypertrophy with resultant fibromuscular thickening.

4. Diabetes: Sustained hyperglycemia is associated with tissue damage and cardiomyopathies. Control of blood glucose levels is important to prevent heart disease.

5. Age: Earlier men less than 55 years were more prone but now heart disease has caught up with a younger age-group of 30 years also. In fact, autopsy studies have indicated that the process of atherosclerosis can begin as early as at two years of age and that the sites of blockage may get predetermined in the womb of hyper-cholesterolemic women.

6. Smoking and tobacco: Cigarette smoking and tobacco is a major independent risk factor for myocardial infarction and cardiac failure. Coronary artery disease has been seen in 80% of smokers. Inhaling nicotine, carbon monoxide and various other pollutants narrow the coronary arteries thus reducing the blood flow to the heart muscle. It deserves special attention in the prevention of cardiovascular disease.

7. Alcohol: Excessive amount of alcohol is also a risk factor. Alcohol shows a positive relationship between the amount of alcohol consumed and blood pressure levels, hence it is best to avoid it or take it in moderation.

8. Lack of physical activity: Sedentary and un-exercised people are more prone to CVD.

9. Syndrome X is a cluster of conditions such as central abdominal obesity, diabetes, dyslipidemia or hypertension with elevated triglycerides, decreased HDL and blood sugar abnormalities–all harmful for cardiovascular disease.

10. Plasma fibrinogen and Lipoprotein(a): Plasma fibrinogen is closely associated with blockage in the arteries due to blood clot formation. Serum lipoprotein (a) which is a genetically inherited mutant of plasminogen, is a discriminant marker of early, asymptomatic atherosclerotic plaques in the carotid arteries and aorta of hypercholesterolemic individuals.

11. Psychological, social, cultural and religious factors indirectly influence the risk of cardiovascular diseases by their effects on kind of food and quantity of food consumed, cigarette and alcohol consumed. Highly competitive job stress and physical exercise, people who are impatient, workaholic, can cause greater harmful effects on the heart and its vessels.

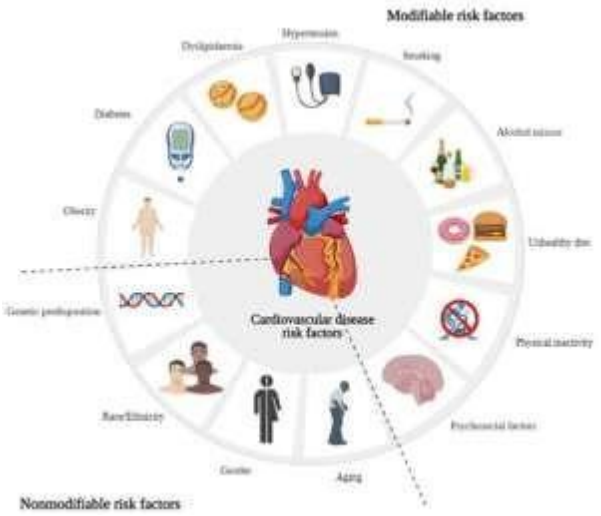

Figure 1: Risk factors of cardiovascular diseases

(Source: https://www.researchgate.net/figure/Cardiovascular-disease-risk-factors-Created-with BioRendercom_fig1_352260450)

Precursors of Cardiovascular Disease

There are two very common conditions that are precursors to virtually all cases of cardiovascular disease: hypertension (hypertension) and atherosclerosis (hardening of blood wall). Both conditions affect the arteries and their ability to maintain normal blood flow.

HYPERTENSION

Hypertension is a chronic medical condition in which the blood pressure in the arteries is persistently elevated, as defined in Table 1. Hypertension usually does not cause symptoms, so more than half of the people with high blood pressure are unaware of their condition. Hypertension is typically diagnosed when blood pressure is routinely measured during a medical visit for some other health problem.

Table 1: Classification of Blood Pressure (in adults)

Category	Systolic (mm Hg)	Diastolic (mm Hg)
Normal blood pressure	90-119	60-79
Prehypertension	120-139	80-89
Hypertension	140 or higher	90 or higher

High blood pressure is classified as either primary or secondary high blood pressure. At least 90% of cases are primary high blood pressure, which is caused by some combination of genetic and lifestyle factors. Numerous genes have been identified as having small effects on blood pressure. Lifestyle factors that increase the risk of high blood pressure include excess dietary salt and alcohol consumption in addition to the risk factors for cardiovascular disease stated above. Secondary high blood pressure, which makes up the remaining 10% of cases of hypertension, is attributable to chronic kidney disease or an endocrine disorder such as Cushing‘s disease.

Treating hypertension is important for reducing the risk of all types of cardiovascular disease, especially stroke. These and other complications of persistent high blood pressure are shown in Figure 2.

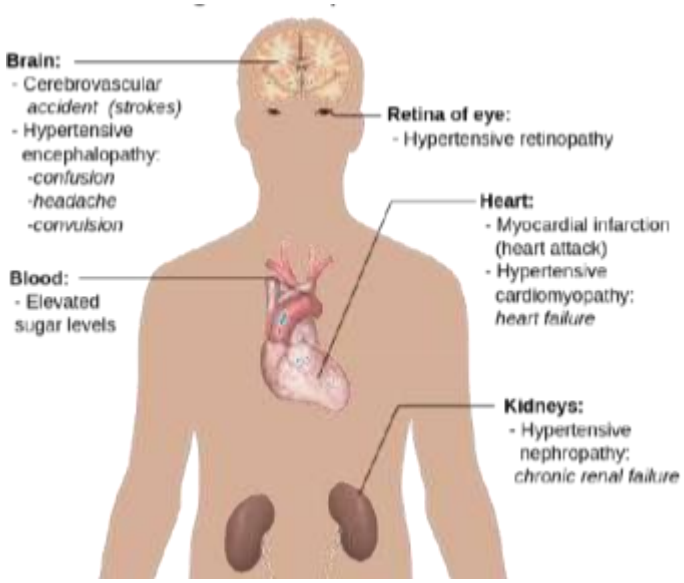

Figure 2 : If high blood pressure is not brought under control, it can eventually cause hypertensive retinopathy, myocardial infarction, heart failure, nephropathy, strokes, confusion, headache, convulsion, and elevated sugar levels. Lifestyle changes, such as reducing salt intake and adopting a healthier diet may be all that is needed to lower blood pressure to the normal range. In many cases, however, medications are also required.

(Source:https://bio.libretexts.org/Bookshelves/Human_Biology/Human_Biology_(Wakim_and_Grewal)/17%3A_Cardiovascular_System/17.7%3A_Cardiovascular_Disease)

DIETARY MANAGEMENT

Energy: Calorie requirement should be based on the concept of maintaining an ideal body weight. Excess calories through fats and carbohydrates have to be reduced so that weight is maintained. Proteins: A normal protein intake is recommended. Protein should contribute 15-20% of the total energy needs. Excess non-vegetarian foods especially red meat and egg yolks could be avoided as it has greater proportion of saturated fatty acids. Fats: The fats incorporated in the diet should be rich in unsaturated fatty acids and should not provide more than 20% of the total energy (refer dietary management of dyslipidemia for details). Carbohydrates: About 60-65% energy should be provided from carbohydrates which are polysaccharides (complex carbohydrates) rather than simple sugars (monosaccharides and disaccharides). Let us now learn about the most important aspect of dietary management, i.e. the intake of minerals and electrolytes, which are closely associated with the maintenance of blood volume. Hypervolemia as we know would cause greater pressure on the arteries. Minerals and Electrolytes: Minerals and electrolytes of clinical significance include calcium, sodium and potassium.

Calcium (Ca): Adequate calcium intake is an essential part of the treatment, and this could be ensured through intakes of milk and milk products and green vegetables as well as adequate cereals and pulse intakes.

Sodium: Studies have shown that sodium restriction along with weight reduction is effective in controlling mild to moderate hypertension along with diuretics recommended. Sodium is restricted to 1-2 g/day which equates to 2.5-5 g of salt for a day. The conversion factor for this is: grams of sodium x 2.5 = grams of salt. 1 teaspoon of salt contains about 2300 mg of sodium. However, it is wise to keep salt at a lower level (2.5 g) as the rest of the sodium can be accounted in a day‘s diet.

Mild Sodium restriction: 2-3 g sodium (2000-3000 mg). Salt may be used lightly in cooking, but no salt at the table is allowed. There is no restriction on naturally occurring fresh foods, but processed foods should be avoided.

– Moderate Sodium restriction: 1 g sodium (1000 mg). In addition to the above restrictions, some control in naturally occurring fresh foods and no salt in cooking is added. Canned

vegetables and baked products are avoided. Meat and milk products are used in moderate amounts.

– Strict Sodium restriction: 0.5 g sodium (500 mg). Apart from the restrictions stated above, meat, milk and eggs are allowed in small portions.

– Severe Sodium restriction: 0.25 g sodium (250 mg). This level is too restrictive and nutritionally inadequate and unrealistic to be used practically. In this, restricted quantities of meat and eggs are used only occasionally. Table 11.6 presents details on low sodium foods.

Potassium: Increasing the potassium content in the diet lowers the blood pressure and improves hypertension. This could be done by increasing fruits and vegetables in the diet, which are rich in both potassium and fibre content. Fluids: Fluid restriction is necessary only if oedema is present. Dehydration may be observed in some patients on diuretics. Thus, normal amount of fluids especially in the form of plain drinking water can be taken. Thus, remember the following points while chalking out a patient care plan for hypertensives.

– Lifestyle changes: Avoiding smoking, use of tobacco, and excess alcohol intake. Physical activity like walking, 4 times a week for 40 minutes, is beneficial.

– Medications: Diuretics, calcium channel blockers and others should be consumed regularly.

– Nutritious balanced diet: The diet of a hypertensive should be nutritious.

It should be low in calories (if required) and fat with a normal protein content. It should be low in sodium but rich in potassium, calcium, magnesium and fibre. Currently the DASH diets are recommended. These are rich in fruits and vegetables, non-fat dairy products and low in total as well as saturated fats.

Atherosclerosis

Atherosclerosis is a condition in which artery walls thicken and stiffen because of the buildup of plaques inside the arteries. Plaques consist of white blood cells, cholesterol, and other fats. Typically, there is also a proliferation of smooth muscle cells that make the plaque fibrous as well as fatty. Over time, the plaques may harden with the addition of calcium crystals. This

reduces the elasticity of the artery walls. As plaques increase in size, the artery walls dilate to compensate so blood flow is not affected. Eventually, however, the lumen of the arteries is likely to become so narrowed by plaque buildup that blood flow is reduced or even blocked entirely. Figure 3 illustrates the formation of a plaque in a coronary artery.

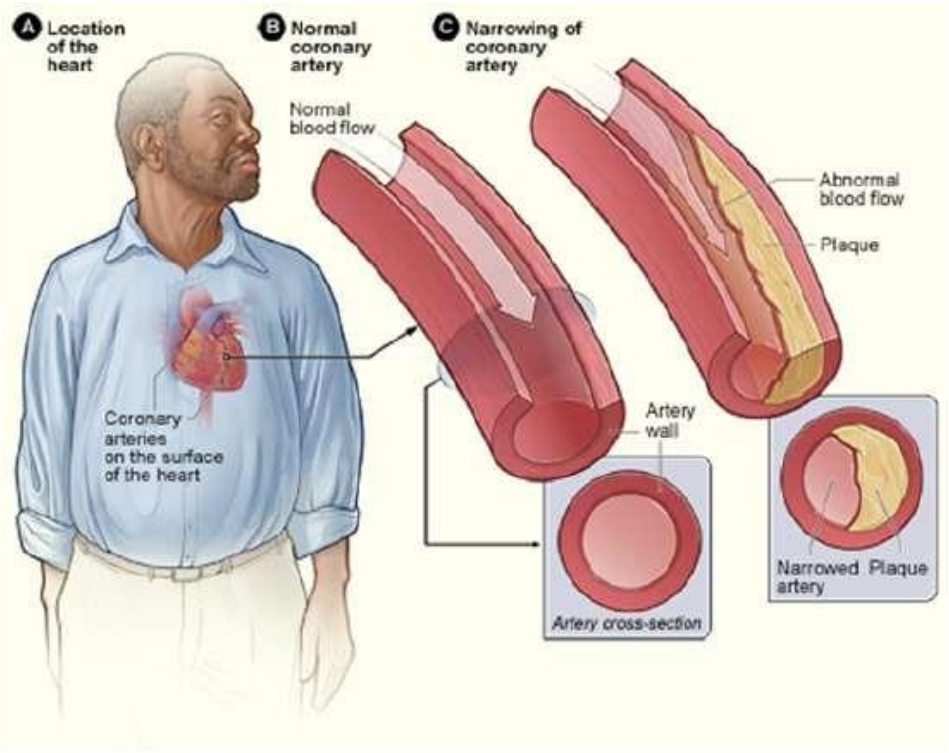

Figure 3: Plaque accumulation in a coronary artery may reduce blood flow to cardiac muscle cells.

(Source:https://bio.libretexts.org/Bookshelves/Human Biology/Human Biology).

In most people, plaques start to form in arteries during childhood and progress throughout life. Individuals may develop just a few plagues or dozens of them. Plaques typically remain asymptomatic for decades. Signs and symptoms appear only after there is severe narrowing (stenosis) or complete blockage of arteries. As plaques increase in size and interfere with blood flow, they commonly lead to the formation of blood clots. These may plug arteries at the site of

the plaque or travel elsewhere in the circulation. Sometimes plaques rupture or become detached from an arterial wall and become lodged in a smaller, downstream artery. Blockage of arteries by plaques or clots may cause a heart attack, stroke, or other potentially life-threatening cardiovascular events. If blood flow to the kidneys is affected, it may lead to chronic kidney disease.

The process in which plaques form is not yet fully understood, but it is thought that it begins when low-density lipoproteins (LDLs) accumulate inside endothelial cells in artery walls, causing inflammation. The inflammation attracts white blood cells that start to form a plaque. Continued inflammation and a cascade of other immune responses cause the plaque to keep growing. Risk factors for the development of atherosclerosis include hypertension, high cholesterol (especially LDL cholesterol), diabetes, and smoking. The chance of developing atherosclerosis also increases with age, male sex, and a family history of cardiovascular disease.

Treatment of atherosclerosis often includes both lifestyle changes and medications to lower cholesterol, control blood pressure, and reduce the risk of blood clot formation. In extreme cases or when other treatments are inadequate, surgery may be recommended. Surgery may involve the placement of stents in arteries to keep them open and improve blood flow or the use of grafts to divert blood flow around blocked arteries.

Role of Carbohydrates: It is important to know about these carbohydrates, as they all differ in their digestive properties. The rate of absorption is variable. Monosaccharides get absorbed the fastest and polysaccharides get absorbed the slowest. This is because polysaccharides contain more fibre. The latter are good for many disorders like intestinal diseases, diabetes, and even cardiac problems. Fibre is beneficial for cardiovascular disease and is found as water-insoluble and water-soluble type.

Soluble fibers like pectins, gums and mucilage‘s have shown reduction in cholesterol levels. Intake of about 20-40 g of soluble fibre has proven to be beneficial. As you can see, legumes, oats, whole grains, fruits (apples, pears, and citrus fruits), and vegetables along with psyllium (isabgol) are a rich source of soluble fibre. Soyabeans are a good source of fibre and soya proteins have estrogenic effect, which causes lipid lowering. A recent analysis of 38 completed trials showed the beneficial effects of soya protein to be in the amount of 47g/day.

Proteins: While the quantity of protein does not impose any significant impact on the serum lipoproteins, it is the quality of protein, which may be of significance. Patients should be advised to consume plant origin proteins over those of animal origin. This is because plant origin foods, which are good sources of protein, are generally rich sources of dietary fibre, have low amounts of saturated fat and are devoid of cholesterol. Egg white and lean meats (meat without fat) should be the preferred options in case of animal foods.

Vitamins: Antioxidants and flavonoids, natural vitamin E, vitamins C and Aare nutrients (vitamins) that scavenge cell-damaging free radicals and act as antioxidants as you may recall studying earlier in Unit 7. It is important to know this because damage through free radicals is quite pronounced among patients with Syndrome X –a risk factor for cardiovascular disease. Vitamin A is present in good amounts in green and yellow fruits and vegetables and lycopene in tomatoes and anthocyanin in grapes and berries. Vitamin E rich foods include corn (0.09 mg), almonds (25.86 mg), sunflower seeds (23.1 mg), spinach (1.29 mg) and soyabean (1.29 mg). Hence, vitamins (E, C and A) containing foods, bright yellow fruits and vegetables like papaya, orange, mango, strawberry, tomato, carrots and green leafy vegetables like methi and spinach, cabbage, red wines, tea and soyabean are excellent foods because of their antioxidant properties.

Minerals: The three most important minerals are chromium, zinc and magnesium. These minerals play a critical role in maintaining proper insulin function. Deficiency of these minerals increases the risk of Syndrome X – a risk factor for cardiovascular disease. Excess sodium intake and lack of potassium have been seen to play an important role in hypertension. Low intakes of calcium can also be a risk for cardiac disorder. Sodium added to food or sodium-rich foods need to be restricted in cardiovascular diseases.

Antioxidants and Flavanoids: You must have already read about different antioxidants present in our foods. The body makes use of a great variety of antioxidants and free radical scavengers for different purposes and to protect tissues with different needs. Vitamins A, C and E have important antioxidant functions as you have already studied above. The B vitamins, although not technically antioxidants, often act as a co-factor with antioxidants. Flavonoids naturally occur in fruits, vegetables, tea and wine.

CORONARY ARTERY DISEASES

Coronary artery diseases are a group of diseases that result from atherosclerosis of coronary arteries. Treatment of the diseases mainly involves treating underlying atherosclerosis. Some of the common coronary artery diseases are given below.

Rheumatic Heart Disease (RHD)

It is a very common cause of cardiovascular disorder in children and adolescents in India. This disease involves damage to the entire heart and its membranes. It is a complication of rheumatic fever (resulting from an untreated Streptococcus throat injection) and usually occurs after attacks of rheumatic fever. Rheumatic fever can damage the heart valves. If the heart valves are damaged, they will fail to open and close properly. When this damage is permanent, the condition is called Rheumatic Heart Disease.

Symptoms

Symptoms generally appear after 1 to 6 weeks of the fever and sometimes the infection may have been too mild to have been recognized. The symptoms are fever, fatigue, shortness of breath, fainting, palpitation and chest pain. Swollen, tender, red, painful nodules or small protuberances may appear. There could be red, raised, lattice-like rash and uncontrolled movements of arms, legs and facial muscles.

Complications

Inflammation of lining of heart (pericarditis), anaemia, heart enlargement, valve deformities (mitral and tricuspid valves), embolism, arrythmia, abdominal pain, fever, arthritis etc.

Dietary management

The diet should be nutritious and without restrictions except in the patient with congestive heart failure, whose fluid and sodium intake should be restricted. Potassium supplementation may be necessary because of the mineralocorticoid effect of corticosteroid and the diuretics (if used).

Valvular Heart Disease

If any of the valves of the heart (aortic, mitral, pulmonary and tricuspid valves) are damaged, it results in valvular heart disease. The basic reason for this disease is the damage to heart valves due to age.

The functions of these valves are to ensure that blood is flowing at the right speed in the right direction. In valvular heart disease conditions, the valves of the heart become very thin and hard that changes the rate and speed of blood flow in the system. Sometimes they may be completely closed.

Symptoms

Symptoms of this disease are very sudden. This disease advances slowly and heart adjusts to it, and it becomes very difficult to find the symptoms. General symptoms are almost similar to rheumatic heart disease. Symptoms are Giddiness, Excess fatigue, Palpitations, Chest pain.

Treatment

Long term antibiotic therapy, Medications which prevent clotting, Balloon dilatation etc.

Inflammatory Heart Disease

Inflammatory heart disease is caused due to inflammation of the pericardium. Causes of this disease include bacterial or fungal infection, Heart attack and myocarditis. Due to radiation therapy to the chest use of medications that suppress the immune system, due to diseases such as cancer, leukaemia, tuberculosis, kidney failure etc.

Symptoms

Severe chest pain, Difficulty in breathing when lying down, Dry cough, Anxiety, Excess fatigue

Treatment

The main goal of the treatment is to suppress acute inflammatory process, Eradication of streptococcal infection, prevent the further occurrence of disease, and protect heart against damaging effects of carditis.

Ischemic Heart Disease

Ischemic heart disease is a wide range of heart diseases caused by the decreased supply of oxygen to the myocardium i.e. the muscle of the heart. It is also known as coronary artery disease.

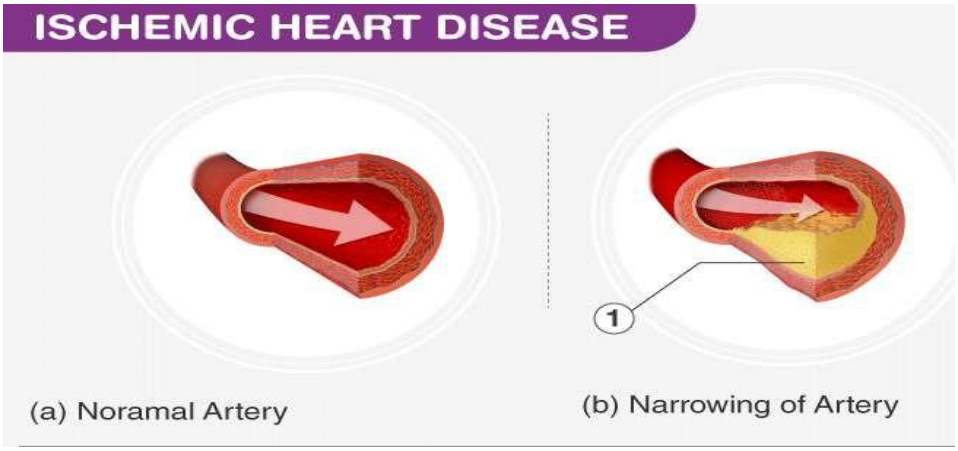

Figure 4. Lipid deposit of plaque

Source : https://www.natural-health-news.com/myocardial-ischemia-causes-symptoms diagnoses-and-treatment)

Symptoms

Chest pain, especially after physical exertion, Dizziness or fainting, Heart palpitations, which may feel like your heart fluttering or skipping beats, Shortness of breath, Swelling in your feet or ankles.

Causes of Ischemic Heart Disease

Risk factors include smoking, diabetes mellitus, and cholesterol levels, Genetic and hereditary factors may also cause this disease, Hypertension, Stress is also a risk factor.

Causes myocardial ischemia

Causes of myocardial ischemia include:

Coronary artery disease. This is a buildup of plaque and cholesterol inside your coronary arteries, which supply blood to your heart muscle. The buildup narrows your artery so much that the oxygen-rich blood your heart needs can‘t get through, and your heart muscle becomes starved for oxygen. This causes ischemia and angina. Atherosclerotic plaque causes 70% of fatal heart attacks.

Blood clot. When plaque that forms in your narrow coronary artery breaks apart, it can attract a blood clot. When a blood clot settles in a coronary artery that‘s already narrow, it can cause a blockage (thrombosis).

Coronary artery spasm. This happens when the coronary arteries spasm, which temporarily reduces or cuts off blood supply to your heart.

Cocaine use.

Coronary artery dissection. This rare condition can keep blood from getting to your heart.

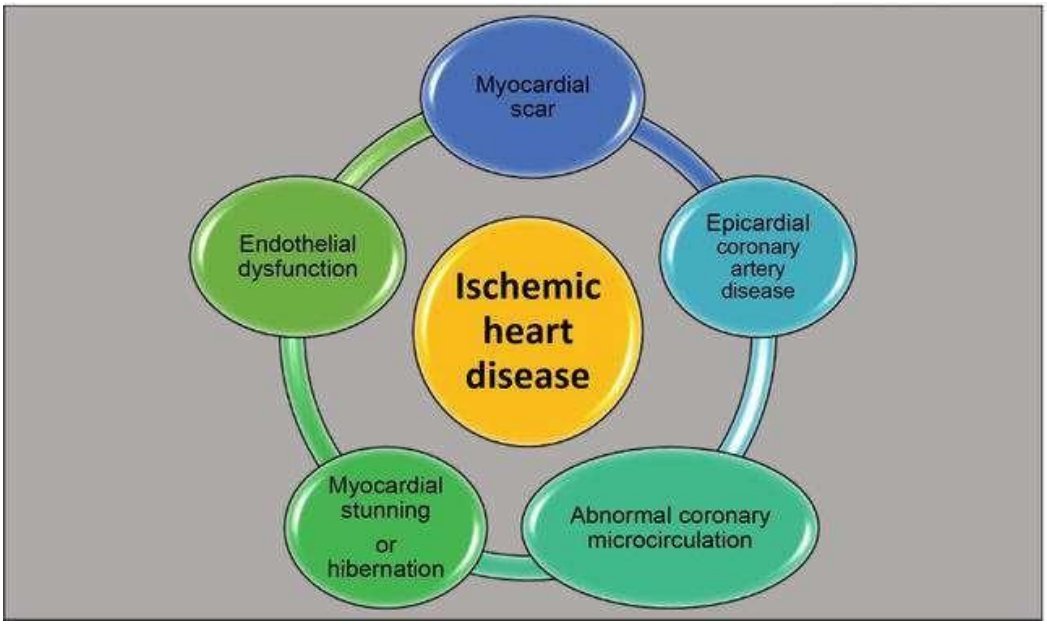

Figure 5. Ischemic heart disease features in the etiology of heart failure.

(Source : https://www.intechopen.com/chapters/77100)

Treatment

Treatment of Ischemic heart disease includes medications like organic nitrates help to relax the non-specific smooth muscles. Beta-blockers help to reduce cardiac work and increase oxygen consumption. Medication like aspirin reduces the risk of angina. Calcium channel blockers reduce the total coronary flow by blocking beta receptors.

Dietary management of valvular heart disease and ishemic heart disease

Limit salt intake: Eating salty foods increases blood pressure, increases pressure on blood vessels, thereby increasing the burden on the heart leading to heart failure. Patients with valvular heart disease need to reduce the burden on the heart, maintain a stable blood pressure The recommended amount of salt in patients with valvular heart disease is 2-4g per day, so the dishes should be processed lighter, should not be eaten. foods containing a lot of salt such as fish sauce and seafood.

Avoid stimulants such as tea, coffee, alcohol, tobacco. Using these stimulants increases the risk of heart attack and stroke.

Smoking: Smoking increases the risk of atherosclerosis leading to coronary heart disease, stroke, and high blood pressure. People with heart valve disease need to quit smoking to reduce the risk of atherosclerosis and reduce the burden on the heart.

Tea and coffee: Tea and coffee contain caffeine, theobromine, theophylline, L-theanine, which stimulates the sympathetic nervous system, which destabilizes physiological activities such as palpitations, palpitations, and tachycardia. Patients with valvular regurgitation regularly use coffee and tea for a long time, which can lead to heart failure and anxiety disorders. In addition, tea and coffee can lead to increased blood pressure, slow blood transport, and make heart valve disease worse.

Drinking a lot of alcohol and beer: Alcohol causes many harmful effects on organs such as the liver, pancreas, kidneys, brain and heart. People with valvular heart disease should avoid consuming alcoholic beverages as they can cause arrhythmias and worsen symptoms. Limit soluble fats such as animal fats, cheese milk, coconut oil, palm oil, canned foods, fried foods, processed meats like sausages, bacon,.Substances This soluble fat causes hypercholesterolemia,

increases the risk of atherosclerosis, burdens the blood vessels and heart, and increases the risk of myocardial infarction and stroke. When the blood vessels are atherosclerotic, the heart has to work harder to meet the blood supply for the whole body. Therefore, the disease condition in people with heart valve regurgitation will become serious. Do not use foods containing refined carbohydrates

Refined carbohydrates including white bread, sugar and sweeteners, people with heart valve regurgitation should not use these foods because the processing process has been done. remove healthy ingredients such as fiber, minerals, fatty acids, and other natural ingredients. In addition, in the processing process, bad ingredients such as trans-fat and sugar are added to the process, which adversely affect the cardiovascular system. Do not drink soft drinks, carbonated drinks: Soft drinks and carbonated drinks contain very high sugar content, making it easy to gain weight quickly, increasing the burden on the heart.

Angina Pectoris

Angina pectoris, or just angina, is temporary chest pain or discomfort caused by decreased blood flow to the heart muscle. Because of the decreased flow of blood, there is not enough oxygen to the heart muscle resulting in chest pain. Coronary artery disease, which can result in narrowing of the coronary arteries that carry blood and oxygen to the heart muscle, is one of the most common causes of angina. While angina is not a heart attack, it does signal an increased risk for a heart attack. Seek immediate medical attention if you experience any chest pain or discomfort.

There are two main types of angina—stable and unstable. Stable angina, the most common type, develops during physical activity and usually lasts a short time (approximately five minutes or less) if the physical activity has ended. Unstable angina is less common and usually occurs during periods of rest.

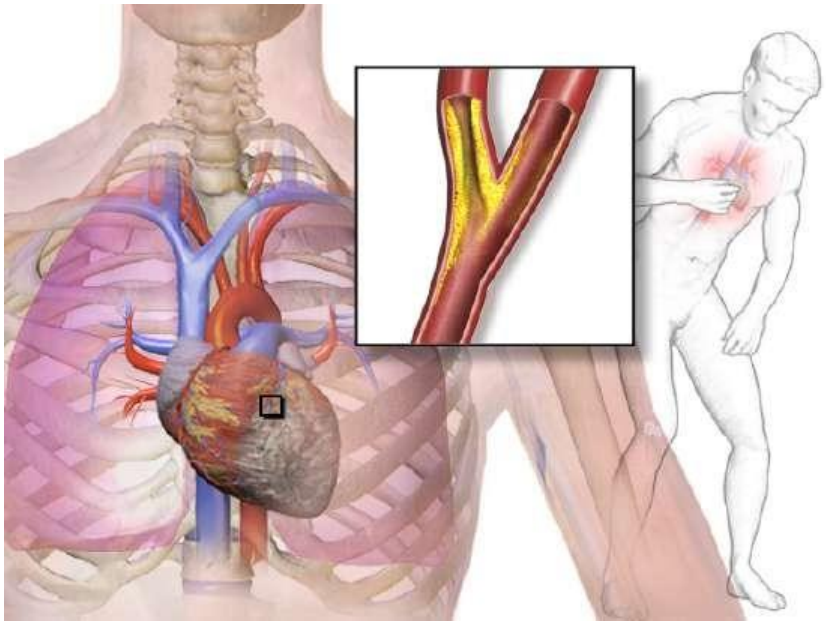

Figure 5: Angina is a pain in the chest due to reduced blood flow in coronary arteries so the heart muscle does not receive adequate oxygen.

(Source: https://byjus.com/biology/angina-pectoris)

Symptoms

Chest pain or discomfort, such as tightening of the chest, Discomfort in the jaw, neck, arms, upper abdomen, shoulder or back, Fatigue, Sweating, Nausea. Dizziness

There are many risk factors associated with angina including, but not limited to, high blood pressure, diabetes, obesity, family history, tobacco use, stress and age.

Diagnosis

Electrocardiogram (ECG): This test records the electrical activity of the heart, which is used to diagnose heart abnormalities such as arrhythmias or to show ischemia (lack of oxygen and blood) to the heart.

Stress test without imaging: This heart-monitoring test is used to help evaluate how well the heart performs with activity. During a stress test, you will usually be asked to perform physical exercise, like walking on a treadmill. An ECG is recorded during the period of exercise. The ECG is assessed by your doctor to see if your heart reached an appropriate heart rate and if there

were any changes to suggest decreased blood flow to your heart. If you are unable to perform exercise, medications that mimic the heart's response to exercise may be used.

Blood tests: The tests can identify certain enzymes such as troponin that leak into the blood after your heart has suffered severe angina or a heart attack. Blood tests can also identify elevated cholesterol, LDL and triglycerides that place you at higher risk for coronary artery disease and therefore angina.

Treatment

Chest x-ray*:* This noninvasive imaging test helps your doctor rule out other sources of chest pain such as pneumonia. Imaging with x-rays involves exposing the chest to a small dose of radiation to produce pictures of the chest and heart.

CT of the chest*:* Chest CT is a more sensitive test than chest x-ray that can identify other causes of chest pain such as aortic disease or **blood clots** in the blood vessels of the lungs. This imaging test combines special x-ray equipment with sophisticated computers to produce multiple images of the chest and heart.

Coronary computed tomography (CT) angiography: This exam evaluates the coronary arteries (blood vessels that supply blood and oxygen to the heart) to determine the extent of narrowing of the arteries due to plaque without the need for an invasive catheter feed through the arteries into the heart.

Magnetic resonance (MR) imaging*:* The primary purpose of this exam is to determine whether there is good blood flow to the heart muscle. If there are areas with decreased blood flow, this could indicate plaque with blood vessel narrowing. This blood flow evaluation may be done twice during the exam with the use of a contrast material. The first time may be performed after the administration of a pharmaceutical, which stresses the heart like exercise. The second time will be at rest. Performing the evaluation both with stress and rest helps determine if the decreased blood flow only occurs with exercise. This exam can also assess function of the heart and determine if there is any scar in the heart muscle. MRI machines use a powerful magnetic field, radio waves and a computer to produce detailed images.

Catheter angiography: In this invasive imaging test, a thin, long plastic tube, called a catheter, is inserted into an artery in your groin or hand using a needle. The catheter is guided with a wire into the coronary arteries and is used to inject contrast material directly into the coronary arteries to determine whether there is any narrowing of the blood vessels. Images of the contrast material in the blood vessels are captured using x-rays. Narrowed portions of the vessels can be reopened using either a balloon or stents.

Echocardiogram: During this test, a transducer that produces high-frequency sound waves is used to create moving images of the heart. The motion of the walls of the heart is evaluated. If there is decreased motion within a portion of the wall of the heart, this could indicate decreased blood flow from narrowing of the coronary artery. Imaging can also be performed with a pharmaceutical agent stressing the heart to detect decreased motion in a portion of the heart muscle with stress.

Single Photon Emission Computed Tomography (SPECT): This stress test with imaging is performed with a nuclear medicine tracer. During an imaging stress test, the patient is usually asked to perform some kind of physical exercise like walking on a treadmill. If the patient is unable to perform exercise for any reason, drugs that mimic the heart's response to exercise may be used. A radioactive tracer will be injected into the blood during the peak of exercise and images of the heart will be taken. The radioactive tracer flows with the blood and will show whether there is an area of the heart with decreased blood flow.

Diet for preventing angina pectoris.

Obviously, a healthy diet goes a long way in preventing angina pectoris. The following basic guidelines need to be followed with meticulous care:- Avoid fatty foods to the maximum possible extent. This includes fried food, milk products such as butter and cheese, full cream milk, oils, etc. Fermented milk products are good for people with angina. This includes curds. Use only vegetable oils for cooking. This includes sunflower, olive, groundnut and rapeseed oils. Avoid salt in the diet. Do not consume foods that are too salty. In meats, red meats such as mutton, beef and pork must be avoided. White meats such as poultry and fish are beneficial. Fishes with high body oil content must be preferred. This includes sardines, tunas, mackerels, salmons, herrings, etc. Canned fish must be strictly avoided. There should at least two to

three fish consumptions per week. Carbohydrates should form the major part of the food. This includes cereals, wheat, rice, bread, potatoes and pasta. It is found that a little bit of alcohol is actually beneficial for angina, but excess is harmful. The safe limit of alcohol is as follows:- For men: 21 units per week, and not more than 4 units on any one day .For women: 14 units per week, and not more than 3 units on any one day.

Prevention of angina pectories

Many of the risk factors for angina can be tackled by lifestyle changes.

• Eat a varied and healthy diet with plenty of leafy vegetables. Avoid sugary foods and saturated fats found in meat and full-fat dairy products.

• Stop smoking. -To provide advice about stop-smoking.

• Lose weight if you are overweight.

• Exercise more: aim for a half-hour walk each day.

• If you have diabetes or high blood pressure, maintain treatment for these conditions.

Ayurvedic herbs

Ayurveda is a treasure-house of remedies for angina pectoris. There is a long list of herbs that have been used since ancient times for the treatment of the condition. The following is a list of these herbs with their actions on the human body:- Guggul is an age-old remedy used by Ayurvedic exponents for treating angina pectoris and its complications. Guggul is in fact a mixture of several substances that have been extracted from the Commiphora mukul plant. This medicine is effective in treating atherosclerosis, which is a leading cause of angina. This is because of guggulsterone, which is a compound found in the guggul plant.

Myocardial Infarction

A myocardial infarction (MI), commonly known as a heart attack, occurs when blood flow stops to a part of the heart causing damage to the heart muscle and the death of myocardial cells. MI usually occurs because of complete blockage of a coronary artery, often due to a blood clot or the

rupture of a plaque (Figure 6). An MI typically causes chest pain and pressure, among other possible symptoms, but at least one-quarter of MIs do not cause any symptoms.

In the worst case, an MI may cause sudden death. Even if the patient survives, MI often causes permanent damage to the heart. This puts the heart at risk of heart arrhythmias, heart failure, and cardiac arrest.

Heart arrhythmias are abnormal heart rhythms, which are potentially life-threatening. Heart arrhythmias often can be interrupted with a cardiac defibrillator, which delivers an electrical shock to the heart, in effect ―rebooting‖ it.

Heart failure occurs when the pumping action of the heart is impaired, so tissues do not get adequate oxygen. This is a chronic condition that tends to get worse over time, although it can be managed with medications.

Cardiac arrest occurs when the heart no longer pumps blood or pumps blood so poorly that vital organs can no longer function. This is a medical emergency requiring immediate intervention.

Dietary management

Proper and careful treatment of the underlying cause (usually dyslipidemia, advanced atherosclerosis or sever chronic hypertension) is imperative to prevent the occurrence of any acute ischemic event namely myocardial infarction/stroke. Dietary management is the key component in preventing the progression of underlying disease condition. The most vital objectives of dietary and lifestyle management include:

To maintain ideal weight for age

To lower blood pressure through drugs and diet control

To avoid exertion and unnecessary stress

To follow a prudent diet /DASH diet The nutrient requirement here are the same as discussed earlier, however, to sum up it can be said that we need to restrict calories from total fats (particularly saturated fats) and from simple carbohydrates; avoid glandular meats (brain, liver, kidneys etc.); use less salt in cooking and avoid salt sprinklers.

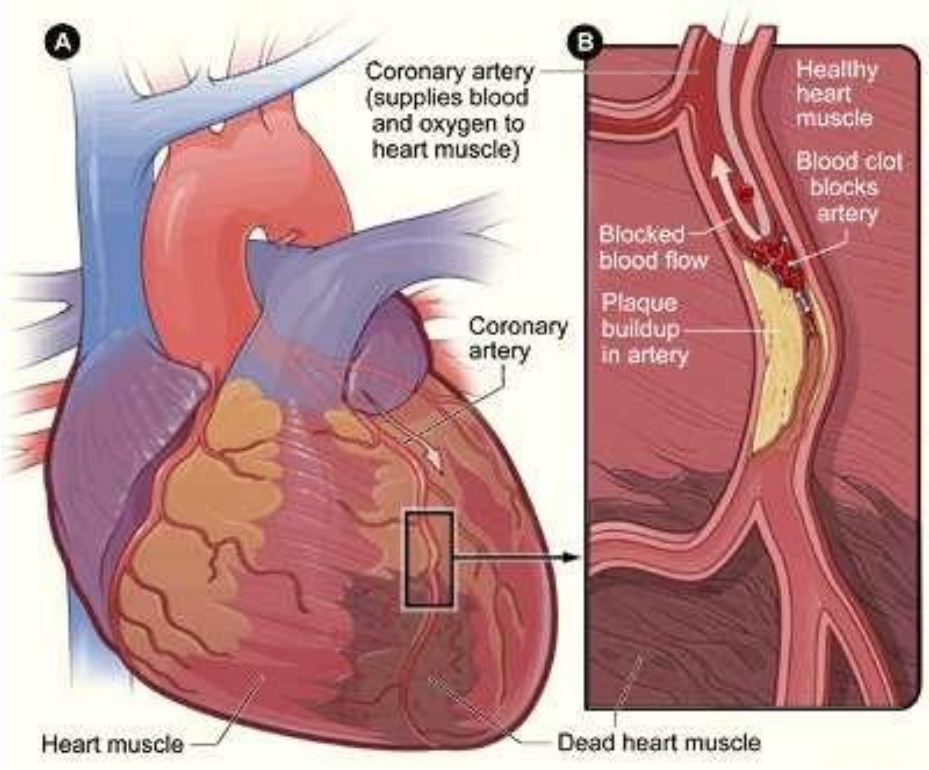

Figure 6 : A myocardial infarction occurs when cardiac muscle cells die due to blockage of a coronary artery.

(Source:https://medicine.umich.edu/dept/cardiac-surgery/patient-information/adult-cardiac-surgery/adult-conditions-treatments/coronary-artery-disease-cad)

Dietary Management

Patients who suffer from an attack of myocardial infarction are hospitalized and are usually kept under strict medical supervision. During the initial 24 to 48 hours most patients are on intravenous support and if needed on an artificial ventilator to meet their oxygen requirements. Initially oral food intake is restricted and not recommended as the ailing heart cannot support the oxygen and absorption of food. Oral food intake is resumed based on several cardiac function tests which help in assessing the residual functional capacity of the heart after injury. Low fat soft diet is generally recommended and during the initial stages foods may be introduced in very small quantities every hour or after every two hours. Observation of the patient during consumption of food and at least till ½ to 1 hour after eating is essential to rule out the elicitation of angina pectoris or another attack of myocardial infarction.

Energy: As mentioned above, patients who have recently suffered from an attack of myocardial infarction are hospitalized in the intensive cardiac care unit wherein their movement is strictly restricted, and they generally are advised not to socialize a lot. Thus, the energy expenditure on physical activity is very low or negligible. The diet should therefore provide enough calories to meet the basal requirements, hence a low-calorie diet is recommended. Other benefits of providing a low calorie diet include: reduction in the adipose tissue mass particularly among obese patients and hence reduced oxygen requirements of the body (tissues); reduction in the requirement of oxygen associated with ingestion, digestion and assimilation of food. The energy intake may initially begin with 800 Kcal which can be slowly progressed to a 1200 Kcal diet till the patient is discharged. Thereafter, the patient‘s energy intake should be governed on the maintenance of body weight which is preferably 1 to 2 kg below IBW.

Protein, Carbohydrates and Fat : The protein intake generally remains the same as per the RDI i.e. 1.0 gm protein per kg body weight per day. Adequate amount of proteins are necessary to promote regeneration of the necrotic tissues in the myocardium. As we had mentioned earlier emphasis should be laid on plant proteins and low-fat animal products (skimmed milk, low-fat paneer, chicken, fish and other marine foods). The majority of MI patients are also hyperlipidemic and have elevated serum triglyceride levels. In such cases, the calorie contribution from fat should not be above 20% and the dietary cholesterol intake should remain below 200 mg per day.

Carbohydrates should provide 60% of the total energy. However, emphasis should be laid on the inclusion of easy-to-digest simple carbohydrates, which are low in fibre. Low fibre cereals, roots and tubers should be served in a soft well-cooked/ blended form (purees etc.). Vitamins and Minerals: The requirement of vitamins and minerals is largely governed by the existing nutritional status and the clinical parameters of the patient. Mild to moderate sodium restriction is generally recommended if the patient is hypertensive or is at risk of developing oedema due to congestive cardiac failure. Inclusion of low fibre, low sodium fruits and vegetables can help in providing good amounts of iron and B-group vitamins particularly folic acid and vitamin B12.

other Considerations

They are initially kept on intravenous fluids to maintain a desirable blood volume and also to supply some amount of calories. As the condition improves, the patients may either be put on enteral tube feeding (intubated with ventilator to supply oxygen) or introduced small sips of full-fluids after every 1 to 2 hours. The diet gradually progressed to a semi-soft and then a soft one. The patients are closely observed when on a soft to normal diet, a few days before discharge. Thus, great care must be taken regarding the consistency and quantity of food being served to the patient. The patient should be advised to eat slowly and adhere to a small frequent meal pattern even after discharge. Rest after meals should be advocated and the patient should avoid all forms of activity after meals. If the patient is overweight/ obese and needs to be operated on, a low calorie diet to aid in weight reduction is a must. As a dietician you must be vigilant if the patient is also a diabetic as his insulin requirements may fluctuate drastically during the post MI period. Proper dietary counseling must be provided, particularly if congestive cardiac failure is present. In our next section, we shall learn about the causes, symptoms, treatment and management of congestive cardiac failure–a decompensated heart disease that frequently develops among patients of MI after several years of rehabilitation.

Stroke

A stroke, also known as a cerebrovascular accident or brain attack, occurs when blocked or broken arteries in the brain result in the death of brain cells. There are two main types of strokes: ischemic stroke and hemorrhagic stroke. Ischemic stroke is illustrated in Figure 7

An ischemic stroke occurs when an embolus (blood clot) breaks off from a plaque or forms in the heart because of arrhythmia and travels to the brain where it becomes lodged in an artery. This blocks blood flow to the part of the brain that is served by arteries downstream from the blockage. Lack of oxygen causes the death of brain cells. Treatment with a clot-busting drug within a few hours of the stroke may prevent permanent damage. Almost 90 percent of strokes are ischemic strokes.

A hemorrhagic stroke occurs when an artery in the brain ruptures and causes bleeding in the brain. This deprives downstream tissues of adequate blood flow and also puts pressure on brain tissue. Both factors can lead to the death of brain cells. Surgery to temporarily open the cranium

may be required to relieve the pressure. Only about 10 percent of strokes are hemorrhagic strokes, but they are more likely to be fatal than ischemic strokes.

In both types of strokes, the part of the brain that is damaged loses is the ability to function normally. Signs and symptoms of stroke may include an inability to move, feel, or see on one side of the body; problems understanding speech or difficulty speaking; memory problems; confusion; and dizziness. Hemorrhagic strokes may also cause a severe headache. The symptoms of a stroke usually occur within seconds or minutes of the brain injury. Depending on the severity of the stroke and how quickly treatment is provided, the symptoms may be temporary or permanent. If the symptoms of a stroke go away on their own in less than an hour or two, the stroke is called a transient ischemic attack. Stroke is the leading cause of disability in the United States, but rehabilitation with physical, occupational, speech, or other types of therapy may significantly improve functioning.

The main risk factor for stroke is high blood pressure. Therefore, keeping blood pressure within the normal range, whether with lifestyle changes or medications, is the best way to reduce the risk of stroke. Another possible cause of stroke is the use of illicit drugs such as amphetamines or cocaine. Having had a stroke in the past greatly increases one‘s risk of future strokes. Men are also more likely than women to have strokes.

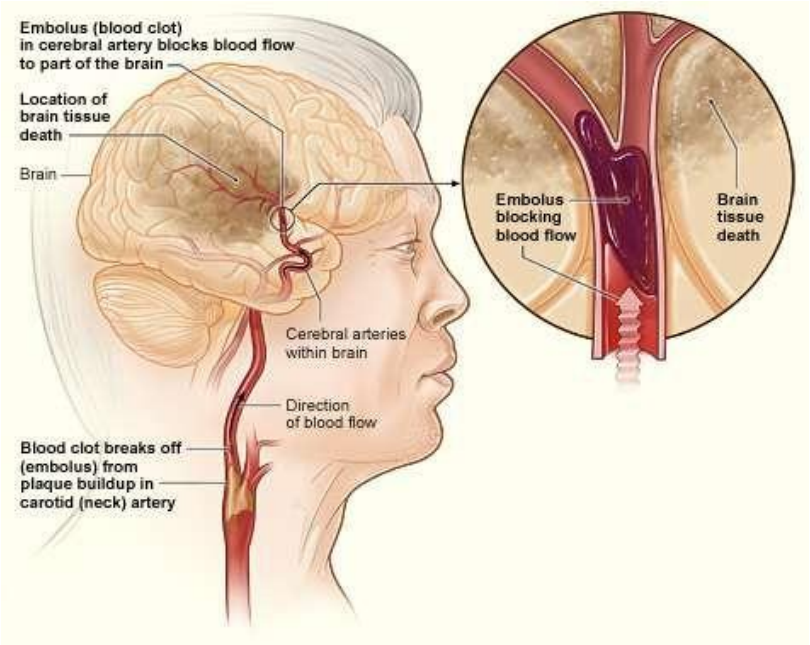

Figure 7 : In an ischemic stroke, brain cells die due to a blocked artery in the brain. The blockage happens when a blood clot breaks from a buildup in the carotid artery and blocks blood flow to part of the brain. As a result, the brain tissue dies. The traveling clot is called embolus.

(Source: https://www.lecturio.com/concepts/ischemic-stroke)

Peripheral Artery Disease

Peripheral artery disease (PAD) is the narrowing of the arteries other than those that supply the heart or brain due to atherosclerosis. Figure 8 shows how the PAD occurs. PAD most commonly affects the legs, but other arteries may also be involved. The classic symptom is leg pain when walking, which usually resolves with rest. This symptom is known as intermittent claudication. Other symptoms may include skin ulcers, bluish skin, cold skin, or poor nail and hair growth in the affected leg(s). However, up to half of all cases of PAD do not have any symptoms.

The main risk factor for PAD is smoking. Other risk factors include diabetes, high blood pressure, and high blood cholesterol. The underlying mechanism is usually atherosclerosis. PAD is typically diagnosed when blood pressure readings taken at the ankle are lower than blood pressure readings taken at the upper arm. It is important to diagnose PAD and treat underlying atherosclerosis because people with this disorder have a four to five times higher risk of myocardial infarction or stroke. Surgery to expand the affected arteries or to graft vessels in order to bypass blockages may be recommended in some cases.

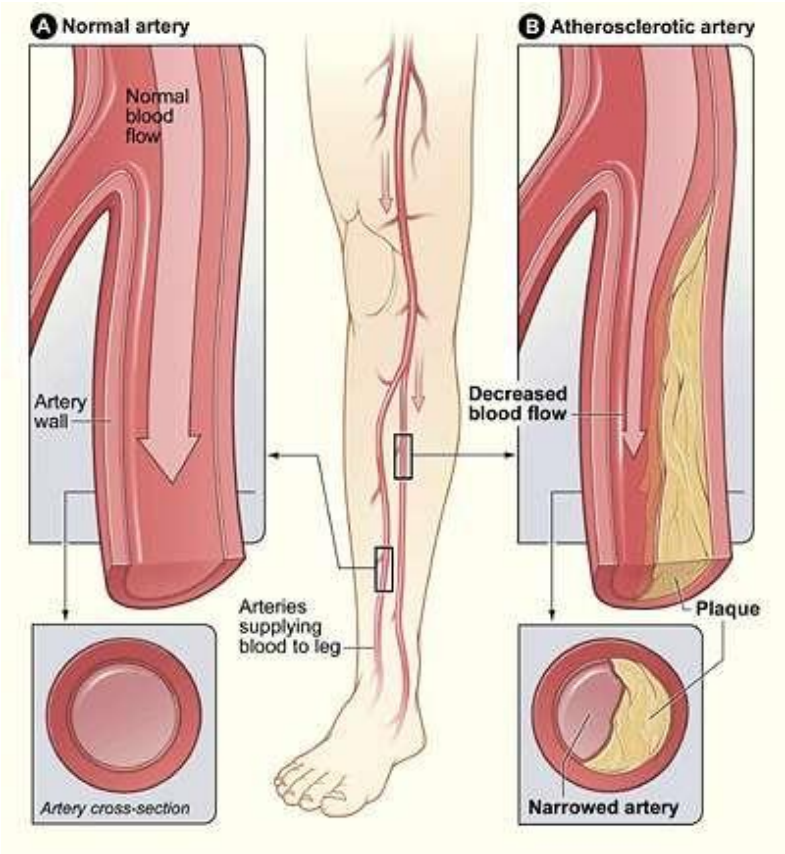

Figure 8 : Peripheral artery disease (PAD) develops when plaque accumulates in the wall of the leg artery. PAD typically causes pain and other symptoms because of decreased blood flow in the leg or other areas of the body served by peripheral arteries.

(Source: https://columbiasurgery.org/news/2016/04/01/peripheral-arterial-disease-pad-may-be-one-most-common-health-problems-you-ve-never)

Dietary management

Mediterranean diet Mediterranean Diet has been associated with lower rates of PAD and improvement of claudication symptoms. Mediterranean diet is characterized by generous amounts of olive oil as the main culinary fat and is characterized by high consumption of plant-derived foods (fruit, vegetables, legumes, nuts and seeds, and whole grain cereals); frequent, but moderate intake of - mainly red - wine with meals; moderate consumption of seafood and dairy products (especially yogurt and cheese, but not whole milk, butter or cream), poultry and eggs; and low consumption of sweet desserts, red and processed meats . The studies showed a

significant relative risk reduction in type2 diabetes mellitus (40%), PAD (64%) incidence and other cardiovascular complications (30%) in patients following a Mediterranean diet. Olive oil consumption seems to be superior to nut oil in terms of cardiovascular outcomes. Dietary advice can be helpful for the prevention of PAD in diabetics, even in populations traditionally accustomed to Mediterranean dietary habits. High adherence to this dietary pattern was not only associated with low prevalence of obesity and hypertension, but also decreased the odds of concentric left ventricle hypertrophy. The Mediterranean diet was proved to increase ventricular-lar filling, which in turn increased end-diastolic filling, left ventricular volumes, stroke volume, and ejection fraction. Data from the studies shows that high adherence to the prudent dietary pattern was associated with lower odds of abdominal obesity, abnormal glucose concentration, and metabolic syndrome. This finding is of clinical impact in primary prevention for cardiovascular events.

Conclusion

This chapter discussed etiological factors, metabolic alterations, clinical manifestations, and dietary management of the disease of heart. Cardiovascular diseases, hypertension, atherosclerosis, myocardial infarction, congestive cardiac failure and RHD are a group of cardiac diseases that are discussed briefly in this chapter. Finally, it focused on the various dietary recommendations proposed by WHO for the prevention of heart diseases, as well as dietary guidelines of the American Heart Association (AHP).

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GASTROINTESTINAL DISEASES

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PEPTIC ULCER

Ulceration in the inner lining of the stomach or upper part of the small intestine is called peptic ulcer. The sore is a result of inflammation caused by the bacteria H. pylori or due to erosion from acids present in the stomach. Stomach pain is the most common symptom experienced by people suffering from peptic ulcer amongst others like bleeding, bloating, and rarely perforation.

Peptic ulcer disease (PUD) is characterized by discontinuation in the inner lining of the gastrointestinal (GI) tract because of gastric acid secretion or pepsin. It extends into the muscularis propria layer of the gastric epithelium. It usually occurs in the stomach and proximal duodenum. It may involve the lower esophagus, distal duodenum, or jejunum. Epigastric pain usually occurs within 15-30 minutes following a meal in patients with a gastric ulcer; on the other hand, the pain with a duodenal ulcer tends to occur 2-3 hours after a meal. Today, testing for Helicobacter pylori is recommended in all patients with peptic ulcer disease. Endoscopy may be required in some patients to confirm the diagnosis, especially in those patients with sinister symptoms. Today, most patients can be managed with a proton pump inhibitor (PPI) based triple-drug therapy.

Peptic ulcers are generally classified into two types:

Gastric ulcers – Ulcers present inside the stomach.

Duodenal ulcers – Ulcers that develop in the duodenum, that is the upper part of the small intestine.

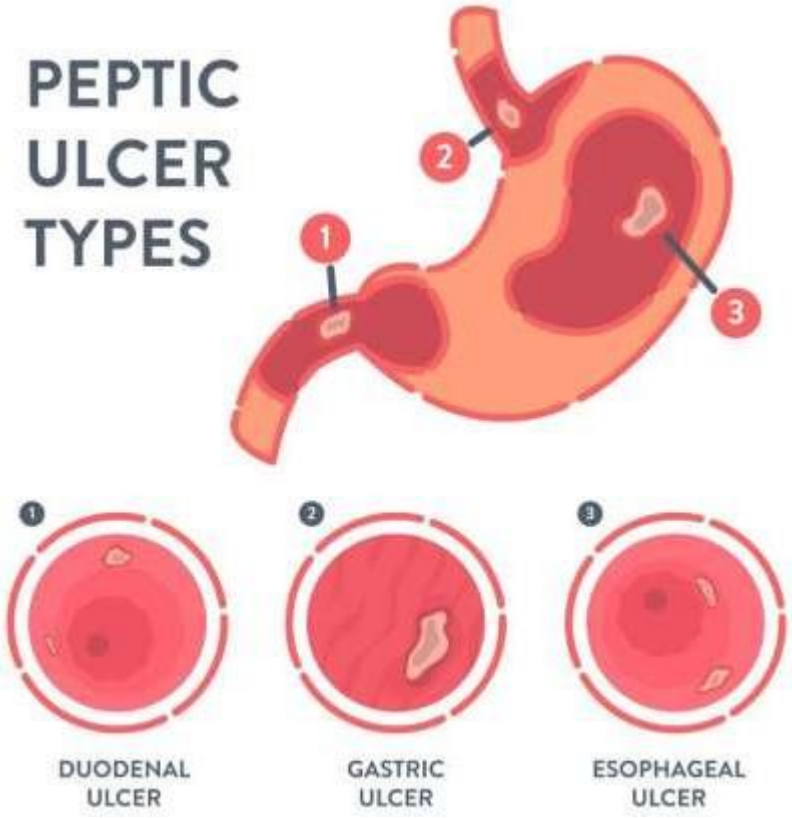
Peptic ulcers are open sores that can be found in the:

Stomach lining

First part of the small intestine (the duodenum)

These painful sores develop when the acidic digestive fluids in the stomach eat away the protective lining of the organs, thus forming ulcers.

Left untreated, peptic ulcers can lead to serious complications including bleeding in the stomach and perforation (tear) of the stomach wall.



https://www.gleneagles.com.sg/

CAUSES

Normally, the digestive tract is covered with a protective mucous layer, that prevents the stomach acid from destroying the inner stomach lining. Conditions that cause an increase in the stomach acids and decreases the amount of mucus results in inflammation in the stomach. The excess acid destroys the mucus layer and eats away the stomach lining.

The common causes are as follows:

Helicobacter pylori (H. pylori) infection – More than half of the population has H. pylori living in the mucous layer of the stomach. It is harmless in most people, but in a few individuals, it increases the stomach acids causing inflammation and ulcer formation. The exact mode of transmission is still not known, but it is believed to spread through direct contact and contaminated food and water.

Indiscriminate use of painkillers – Frequent use of analgesics of the nonsteroidal anti-inflammatory drug (NSAIDs) group like Aspirin, Ibuprofen, Naproxen, and Ketoprofen can irritate or inflame the lining of the stomach.

Other Causes: Smoking, consuming too much alcohol, radiation therapy, too much stress, eating spicy food, family history of peptic ulcer, and stomach cancer can increase the risk of developing a peptic ulcer.

Some rare causes of peptic ulcers are:

Ulcer may develop after being very ill due to any infection or disease.

Due to medications such as steroids.

Ulcer may develop after surgery.

Ulcers can also develop due to Zollinger-Ellison syndrome which causes the formation of acid-producing cell tumors in the digestive tract. They cause severe damage to the stomach tissues, and these tumors can be cancerous or non-cancerous.

SYMPTOMS

Burning type of abdominal pain is the most common symptom of peptic ulcers. This pain radiates from the navel to the chest and varies from being mild to severe. Small ulcers do not produce any pain in the initial stages. The symptoms include:

Feeling bloated.

Belchings.

Heartburn.

Nausea.

Vomiting.

Stools become black and tarry because of the presence of blood in them.

Loss of appetite.

Unexplained weight loss.

Chest pain.

Indigestion.

COMPLICATIONS

It is essential to detect and treat ulcers as soon as possible because untreated ulcers can cause serious health complications like:

Bleeding from the ulcers can cause anemia and other emergency conditions. Blood transfusion might be required if there is a severe loss of blood. Signs of bleeding include blood in vomit, black stools, light headedness, and dizziness.

Sometimes, peptic ulcer can perforate the wall of the stomach. This puts you at risk of serious infections.

The scar tissue formation over an ulcer can obstruct the passage of food through the digestive tract.

All the above conditions are serious, so seek medical attention if you experience any of the following symptoms:

Sharp and sudden abdominal pain.

Fainting.

Profuse sweating.

Confusion.

Rigid abdomen.

RISK FACTORS

People with the following adverse habits are at risk of developing peptic ulcers.

Indiscriminate use of NSAIDs (painkillers).

Smoking.

Alcoholism.

Untreated stress.

Eating a lot of spicy food.

DIAGNOSIS

If the person experiences any of the above symptoms, they should consult a doctor. The doctor will ask about the symptoms and history, and if the doctor suspects a peptic ulcer, the person will have to undergo the following tests:

Upper GI (Gastrointestinal) Endoscopy – This test is not done for all suspected cases of ulcers but is done for people with a higher risk of stomach cancer, bleeding or symptoms not responding to usual medications. Here, the doctor inserts a long tube with a camera attached on one side (endoscope) through your mouth and throat, into the stomach. This will help the doctor

to see the stomach and small intestine lining, and to visualize the ulcer. They can also collect tissue samples for testing.

Upper GI Series – Upper GI radiography is a real-time fluoroscopy technique that involves taking series of X-rays of the esophagus, stomach, and small intestine after the patient drinks a thick liquid called barium (barium swallow). This liquid coats the digestive tract, making the ulcer more visible.

Tests for Helicobacter pylori – The presence of H.pylori bacteria is tested in the biopsies from stomach (taken during endoscopy), blood, stool, or breath. Endoscopic biopsies are the gold standard for detection of H.pylori in stomach tissue. The breath test is the most accurate of them all. In the breath test, the patient is asked to drink or eat something that contains radioactive carbon. H.pylori breaks down the radioactive carbon that is released as carbon dioxide, the presence of which is tested in the breath.

TREATMENT

The treatment depends on the cause. The following treatments are used:

Medications:

Antibiotics are given to kill H.pyori in the stomach. The combination of antibiotics includes Amoxicillin, Clarithromycin, Metronidazole, Tinidazole, Tetracycline, and Levofloxacin.

Proton pump inhibitors (PPIs) are used to reduce the acid production in the stomach. Examples of PPIs are Omeprazole, Rabeprazole, Pantoprazole, Lansoprazole, and Esomeprazole.

Histamine (H2) blockers are used to block the cells that produce acids in the stomach. These medicines include Ranitidine, Famotidine, Cimetidine, and Nizatidine.

Antacids help in neutralizing the stomach acid and help in relieving the pain.

In a few cases, cytoprotective agents are given to protect the tissues in the lining of the stomach. Sucralfate and Misoprostol are the examples.

Home Remedies

Consume a diet rich in vitamins, minerals, and proteins. Eat fruits, vegetables, and whole grains.

PREVENTION

A few alterations in your lifestyle and habits can reduce the risk of you developing peptic ulcers. The lifestyle changes and habits include:

Reduce the consumption of alcoholic beverages.

Do not consume alcohol when on any medication.

Wash your hands frequently to avoid infections.

Limit the use of painkillers.

For many people treatments aim to target the underlying cause which helps in curing the ulcer disease. Ulcers can reoccur if the underlying cause is not completely cured or the person continues to smoke or use NSAIDs (nonsteroidal anti-inflammatory drugs).

CONSTIPATION

Constipation is a condition in which a person has uncomfortable or infrequent bowel movements. Generally, a person is considered to be constipated when bowel movements result in passage of small amounts of hard, dry stool, usually fewer than three times a week. However, normal stool elimination may consist of having a bowel movement three times a day or three times a week; it depends on the person.

About 4 million people in the United States have frequent constipation. Constipation is the most common gastrointestinal complaint, resulting in 2.5 million doctor visits annually.

CAUSES

Hard, dry stools are the result of the colon absorbing too much water. Normally, as food moves through the colon (also known as the large intestine) the colon absorbs water while forming stool (waste products). Muscle contractions then push the stool toward the rectum, and, by the time the stool reaches the rectum, most of the water has been absorbed, making the stool solid.

When the colon‘s muscle contractions are slow or sluggish, the stool moves through the colon too slowly, resulting in too much water being absorbed. Some of the most common causes of constipation include the following:

Medications

Lack of exercise

Not enough liquids

Not enough fibre in the diet

Irritable bowel syndrome

Ignoring the urge to have a bowel movement

Changes in habits or lifestyle, such as travel, pregnancy, and old age

Problems with intestinal function

Abuse of laxatives

SYMPTOMS

The following are the most common symptoms of constipation. However, each individual may experience symptoms differently. Symptoms may include:

Difficult and painful bowel movements

Bowel movements fewer than three times a week

Feeling bloated or uncomfortable

Feeling sluggish

Abdominal pain

The symptoms of constipation may resemble other medical conditions or problems. Always consult your doctor for a diagnosis.

DIAGNOSIS

The tests performed by a doctor will depend on the duration and severity of the constipation, since most persons experience constipation at one time or another. The doctor will also take into

account the patient‘s age, and whether there is blood in the stool, recent changes in bowel habits, or weight loss.

Diagnosing constipation may include:

Medical history. The doctor will ask for a description of the constipation, including duration of symptoms, frequency of bowel movements, and other information to help determine the cause of the constipation.

Physical examination. A physical examination may also include a digital rectal examination (DRE), in which the doctor inserts a gloved, lubricated finger into the rectum to evaluate the tone of the muscle that closes off the anus. This examination also helps detect tenderness, obstruction, blood, amount and caliber of stool, and if enlargement of the rectum is present.

Other diagnostic tests may include:

Abdominal X-ray

Lower GI (gastrointestinal) series (also called barium enema). A lower GI series is a procedure that examines the rectum, the large intestine, and the lower part of the small intestine. A fluid called barium (a metallic, chemical, chalky, liquid used to coat the inside of organs so that they will show up on an X-ray) is given into the rectum as an enema. An X-ray of the abdomen shows strictures (narrowed areas), obstructions (blockages), and other problems.

Colonoscopy. Colonoscopy is a procedure that allows the doctor to view the entire length of the large intestine, and can often help identify abnormal growths, inflamed tissue, ulcers, and bleeding. It involves inserting a colonoscope, a long, flexible, lighted tube, in through the rectum up into the colon. The colonoscope allows the doctor to see the lining of the colon, remove tissue for further examination, and possibly treat some problems that are discovered.

Sigmoidoscopy. A sigmoidoscopy is a diagnostic procedure that allows the doctor to examine the inside of a portion of the large intestine, and is helpful in identifying the causes of diarrhea, abdominal pain, constipation, abnormal growths, and bleeding. A short, flexible, lighted tube,

called a sigmoidoscope, is inserted into the intestine through the rectum. The scope blows air into the intestine to inflate it and make viewing the inside easier.

Colorectal transit study. This test shows how well food moves through the colon. The patient swallows‘ capsules containing small markers which are visible on X-ray. The patient follows a high-fiber diet during the course of the test, and the movement of the markers through the colon is monitored with abdominal X-rays taken several times three to seven days after the capsule is swallowed.

Anorectal function tests. These tests diagnose constipation caused by an abnormal functioning of the anus or rectum.

TREATMENT

Specific treatment for constipation will be determined by your doctor based on:

Your age, overall health, and medical history

Extent of the condition

Your tolerance for specific medications, procedures, or therapies

Expectations for the course of this condition

Your opinion or preference

Most often, constipation can be treated through dietary and lifestyle changes, which relieve symptoms and help prevent the condition.

Treatment may include:

Diet modifications. A diet with 20 to 35 grams of fiber daily helps in the formation of soft, bulky stool. While adding foods such as beans, whole grains, bran cereals, fresh fruits and vegetables is helpful in adding fiber to the diet. Limiting foods such as ice cream, cheeses, meats, and processed foods, which contain little or no fiber can also be helpful.

Laxatives. Laxatives may be prescribed after diet and lifestyle changes have failed to be effective.

Eliminating or changing medication

Biofeedback. Biofeedback is used to treat chronic constipation caused by anorectal dysfunction. This treatment retrains the muscles that control release of bowel movements.

Lifestyle changes, such as increased water and juice intake, regular exercise, and allowing enough time for daily bowel movements can be helpful.

Complications

Constipation can cause complications, such as hemorrhoids, which occur by straining to have a bowel movement, or anal fissures (tears in the skin around the anus) which occur when hard stool stretches the sphincter muscle. This can result in rectal bleeding.

Sometimes, straining also causes rectal prolapse, where a small amount of intestinal lining pushes out from the anal opening. Constipation may also cause fecal impaction, which occurs mostly in children and older adults. The hard stool packs the intestine and rectum so tightly that the normal pushing action of the colon is not enough to expel the stool.

DIARRHEA

Diarrhea is a common condition that varies in severity and etiology. The evaluation of diarrhea varies depending on duration, severity, and presence of certain concurrent symptoms. Treatment also varies, though rehydration therapy is an important aspect of the management of any patient with diarrhea. This activity reviews the evaluation and treatment of diarrhea and stresses the role of the interprofessional team in caring for patients with this condition.

Acute diarrhea is described as the acute onset of three or more loose or watery stools a day lasting for 14 days or less. However, chronic or persistent diarrhea is labelled when an episode lasts beyond 14 days. Infection commonly causes acute diarrhea. Non-infectious etiologies become more common as the duration of diarrhea becomes chronic. This distinction is important because treatment and management are based on the duration and specific etiology. Rehydration therapy is an important aspect of the management of any patient with diarrhea.

ETIOLOGY

Diarrhea is categorized into acute or chronic and infectious or non-infectious based on the duration and type of symptoms. Acute diarrhea is defined as an episode lasting less than two weeks. Infection most commonly causes acute diarrhea. Most cases are the result of a viral infection, and the course is self-limited. Chronic diarrhea is defined as a duration lasting longer than two weeks and tends to be non-infectious. Common causes include malabsorption, inflammatory bowel disease, and medication side effects. Following are some important considerations to be made while diagnosing and managing diarrhea as the identification of the etiological agent is very important:

Stool characteristics vary between different causes, such as consistency, color, volume, and frequency

Presence or absence of associated intestinal symptoms, such as nausea/vomiting, fever, and abdominal pain

Exposure to child daycare where commonly encountered pathogens are rotavirus, astrovirus, calicivirus; Shigella, Campylobacter, Giardia, and Cryptosporidium species

History of the ingestion of infected food, such as raw or contaminated foods

History of water exposure from swimming pools, camping, or marine environment

Travel history is crucial as common pathogens affect certain regions; enterotoxigenic Escherichia coli is the predominant pathogen

Animal exposure has been historically linked with diarrhea, such as young dogs/cats: Campylobacter; turtles: Salmonella

Predisposing factors such as hospitalization, antibiotic use, immunosuppression

EPIDEMIOLOGY

Norovirus is associated with approximately one-fifth of all infectious diarrhea cases, with similar prevalence in both children and adults, and is estimated to cause over 200,000 deaths annually in developing countries. Historically, rotavirus was the most common cause of severe disease in

young children globally. Rotavirus vaccination programs have decreased the prevalence of diarrhea cases associated with rotavirus.

In developing regions, an average of three episodes of diarrhea per child per year is reported in children less than 5 years old. However, certain other areas report six to eight episodes per year per child. In these circumstances, malnutrition plays an additional role in the development of diarrhea.

A common cause of chronic diarrhea includes inflammatory bowel disease, Crohn disease, and ulcerative colitis. In Europe, the incidence of ulcerative colitis and Crohn disease has increased overall from 6.0 per 100,000 person-years in ulcerative colitis and 1.0 per 100,000 person-years in Crohn disease in 1962 to 9.8 per 100,000 person-years and 6.3 per 100,000 person-years in 2010, respectively.

A study conducted by Lübbert et al observed the occurrence of Clostridium difficile related infection in Germany to be 83 cases/100,000 population in 2012. The chance of recurrence escalated with each relapse in these cases.

In the United States, before specific antirotavirus immunization was introduced in 2006 a cumulative occurrence of one hospitalization for the cases of diarrhea per 23-27 children by the age of 5 years was noted. Moreover, over 50,000 hospitalizations were noted. Basing it on these facts, rotavirus was found to be responsible for 4-5% of all childhood hospitalizations costing nearly 1 billion US dollars.

SYMPTOMS

The main symptom of diarrhea is passing loose, watery stools three or more times a day.

People with diarrhea may also have one or more of the following symptoms:

An urgent need to use the bathroom

Cramping

Loss of control of bowel movements

Nausea

Pain in the abdomen

People with diarrhea caused by some infections may also have one or more of the following symptoms:

Bloody stools

Fever and chills

Light-headedness and dizziness

Vomiting

Diarrhea may cause dehydration and malabsorption.

PATHOPHYSIOLOGY

Diarrhea is the result of reduced water absorption by the bowel or increased water secretion. A majority of acute diarrheal cases are due to infectious etiology. Chronic diarrhea is commonly categorized into three groups; watery, fatty (malabsorption), or infectious. Another way of classifying the pathophysiology of diarrhea is into secretory and osmotic forms of diarrhea.

Lactose intolerance is a type of watery diarrhea that causes increased water secretion into the intestinal lumen.Patients typically have symptoms of bloating and flatulence along with watery diarrhea. Lactose is broken down in the intestine by the enzyme lactase. The byproducts are readily absorbed by the epithelial cells. When lactase is decreased or absent, lactose cannot be absorbed, and it remains in the gut lumen. Lactose is osmotically-active, and it retains and attracts water leading to watery diarrhea.

Common causes of fatty diarrhea include celiac disease and chronic pancreatitis. The pancreas releases enzymes that are necessary for the breakdown of food. Enzymes are released from the pancreas and aid in the digestion of fats, carbohydrates, and proteins. Once broken down, the products are available for uptake in the gut. Patients with chronic pancreatitis have insufficient enzyme release leading to malabsorption. Symptoms often include upper abdominal pain, flatulence, and foul-smelling, bulky pale stools due to malabsorption of fats.

In the secretory form of diarrhea, bacterial and viral infections are the common causes. In this instance, the watery stool is the result of injury to the gut epithelium. Epithelial cells line the intestinal tract and facilitate the absorption of water, electrolytes, and other solutes. Infectious etiologies cause damage to the epithelial cells which leads to increased intestinal permeability. The damaged epithelial cells are unable to absorb water from the intestinal lumen leading to loose stool.

COMPLICATIONS

Diarrhea can lead to several complications, some of which can be serious if not addressed promptly. Key complications include:

Dehydration: The most common and potentially serious complication, dehydration occurs when the body loses more fluids and electrolytes than it takes in. Symptoms include dry mouth, excessive thirst, reduced urine output, and dizziness.

Electrolyte Imbalance: Along with water, diarrhea causes loss of essential electrolytes like sodium, potassium, and chloride, which are crucial for normal body functions. Imbalances can lead to symptoms such as muscle cramps, fatigue, and in severe cases, heart problems.

Malnutrition: Chronic diarrhea can lead to poor absorption of nutrients, causing malnutrition. This can be particularly concerning in children, affecting their growth and development.

Weight Loss: Persistent diarrhea can result in significant weight loss due to the loss of fluids, electrolytes, and nutrients.

Skin Irritation: Frequent bowel movements can cause irritation and rashes around the anal area.

Secondary Infections: Continuous diarrhea can damage the intestinal lining, making it more susceptible to infections.

Impact on Quality of Life: Chronic diarrhea can significantly impact daily activities and overall quality of life due to discomfort, urgency, and potential embarrassment.

Acidosis: Severe dehydration and electrolyte imbalance can lead to metabolic acidosis, where the body produces too much acid or when the kidneys are not removing enough acid from the body.

TREATMENT/MANAGEMENT

Important aspect of diarrhea management is replenishing fluid and electrolyte loss. Patients should be encouraged to drink diluted fruit juice, Pedialyte or Gatorade. In more severe cases of diarrhea, IV fluid rehydration may become necessary. Eating foods that are lower in fiber may aid in making stool firmer. A bland 'BRAT' diet including bananas, toast, oatmeal, white rice, applesauce and soup/broth is well tolerated and may improve symptoms. Anti-diarrheal therapy with anti-secretory or anti-motility agents may be started to reduce the frequency of stools. However, they should be avoided in adults with bloody diarrhea or high fever because they can worsen severe intestinal infections. Empiric antibiotic therapy with an oral fluoroquinolone can be considered in patients with more severe symptoms. Probiotic supplementation has been shown to reduce the severity and duration of symptoms and should be encouraged in patients with acute diarrhea.

The treatment of chronic diarrhea is specific to the etiology. The first step is to categorize diarrhea into watery, fatty or inflammatory. Once categorized, an algorithm can be used to determine the next step in management. Most cases require additional fecal studies, lab work or imaging. More invasive procedures like colonoscopy or upper endoscopy may be required.

In 2003 the recommendations were put forward by the Center for Disease Control (CDC) for the treatment of acute diarrhea in children on both the outpatient and inpatient basis including indications for referral.

Indications for referral and further medical evaluation of children include the following:

Under 3 months old

Weighs less than 8 kg (17.6 lbs)

History of premature birth, chronic illnesses, or concurrent medical conditions

Fever of 38ºC (100.4 F) or higher in children less than 3 months old or 39ºC (102.2 F) or higher in children between 3 and 36 months of age

Grossly bloody stool

High-output diarrhea

Persistent vomiting

Signs of dehydration, such as sunken eyes, decreased tear film, dry mucous membranes, and oliguria/anuria

Mental status alterations

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LIVER DISEASES

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Introduction

Liver is a dual organ having both secretory and excretory functions. It is the largest gland in the body. Liver is the second largest organ in human body, more than 5,000 separate bodily functions including helping blood to clot, cleansing the blood of toxins to converting food into nutrients to control hormone levels, fighting infections and illness, regenerating back after injury and metabolizing cholesterol, glucose, iron and controlling their levels. Most people never give their liver a thought until something goes wrong, yet, liver diseases on rise, affecting one in ten. Liver diseases can be inherited or caused by a variety of factors that damage the liver. In fact, there are many types of liver diseases that can be caused by a virus, damage from drugs or chemicals, obesity, diabetes or an attack from own immune system, when the condition is left untreated, it can become life threatening and can permanently damage the liver or the bile duct. This damage leads to malignancy and cause liver cancer.

Liver Diseases

Numerous liver diseases are accompanied by jaundice caused by augmented levels of bilirubin in the body. Bilirubin is the result of degradation of hemoglobin of dead red blood cells which are normally removed by the liver and excreted via bile. In hepatitis, inflammation of the liver, is caused by different viruses, but also some toxic substances, autoimmune diseases and inherited conditions; Liver cirrhosis is the formation of fibrous tissue in the liver to kill hepatocytes, respectively. Liver cell death can be caused by viral hepatitis, alcohol poisoning or other toxic substances; Hemochromatosis is an inherited disorder that causes iron accumulation in the body, leading to long-term liver damage; Benign tumors such as adenoma, "angioma" focal nodular hyperplasia. Liver cancer as the primary tumor or carcinoma cholangiocarcinoma

or metastasis of cancer to other parts of the digestive system; Wilson disease is an inherited disorder that causes copper accumulation in the body; Primary sclerosing cholangitis, an inflammatory autoimmune disease carries bile; Primary biliary cirrhosis, autoimmune disease of minute rage ducts; Budd-Chiari syndrome or hepatic vein obstruction; Gilbert's syndrome, a genetic disorder of bilirubin metabolism. There are also many pediatric liver diseases. Proper liver function can be verified by a number of specialized clinical studies, which measure the presence or absence of typical enzymes, metabolites or substances associated with the regular activities.

Agents Responsible for Liver Damage

- **Dietary Deficiencies**

Fatty changes in the liver, in kwashiorkor may be attributed to a low protein intake and reduced capacity to produce lipo proteins. The liver is of often grossly enlarged in children with kwashiorkor, containing 30-50 per cent, fat by weight, in the form of triglyceride. The intrahepatic accumulation of fat results due to excess delivery of fatty acids to the liver and or enhancing the lipogenesis. This is combined with the impaired lipid transport from the liver secondary to Apo protein deficiency. When protein is supplied fat clears from the liver within a short time. Fatty changes in the liver are common whenever there is a high proportion of fat in the metabolic mixture, for example, in uncontrolled diabetes, in starvation, in some cases of obesity, and when too much carbohydrate has been infused during intravenous feeding. In these cases, as in kwashiorkor, the changes are easily reversible and not followed by fibrosis

- **Infective Agents**

Virus can cause infection and damage the liver. Types A, B, C, D, E and G can cause hepatitis. Type E can be deadly during pregnancy. Hepatitis A and E virus is excreted in the stools and spread by the faecal-oral route. The patient suffers from jaundice and the liver is enlarged and tender. Recovery from a typical infection is complete and not followed by fibrosis and cirrhosis. Hepatitis B, C, D and G virus produce a disease

known as homologous serum jaundice which arises after transfusion of blood or blood products obtained from a donor who is a carrier. Improperly sterilised needles can also cause this hepatitis infection. Most patients recover. In 10 per cent of people the disease may reach a chronic stage. Types B and D can cause extreme jaundice.

- **Toxic Agents**

Alcohol: In malnourished patients consumption of alcohol produces acute liver damage and jaundice that resembles viral hepatitis. Alcohol is also known to have direct action on the lipid metabolism in the liver by enhancing fatty acid synthesis, decreasing fatty acid oxidation and producing specific stimulation to triglyceride formation. Lipids accumulate, because of the increased direct channelling of ethanol as a substrate to acetate via acetaldehyde with the subsequent conversion of acetyl CoA to fatty acids. The increased NADH/NAD ratio overwhelms the cell‘s ability to maintain redox homeostasis and contributes to this conversion. About half the increase in liver size in heavy drinkers is explained by the excess fat.

Drugs and Chemicals: Every drug that is consumed reaches the liver and its chemical nature is altered before being eliminated from the body. Drugs like paracetamol (fever) INH (TB) and oestrogen (contraceptive) may damage the liver. Workers in chemical industry like organic hydrocarbons may develop liver damage. Toxicity due to liver damage has also been observed with several commercially available herbal preparations. Excess store of iron, copper, galactose and glycogen may accumulate in the liver and in time lead to cirrhosis.

In Born Errors of Metabolism: Children with hereditary tyrosinaemia, galactosemia and hereditary fructose intolerance may develop liver damage.

1. Hepatitis

Hepatitis is an inflammation of the liver that is caused by a variety of infectious viruses and noninfectious agents leading to a range of health problems, some of which can be fatal. There are five main strains of the hepatitis virus, referred to as types A, B, C, D and E. While they all cause liver disease, they differ in important ways including modes of transmission, severity of the illness, geographical distribution and prevention methods. In particular, types B and C lead to chronic disease in hundreds of millions of people and together are the most common cause of liver cirrhosis, liver cancer and viral hepatitis-related deaths. An estimated 354 million people worldwide live with hepatitis B or C, and for most, testing and treatment remain beyond reach. Some types of hepatitis are preventable through vaccination.

1.1 Hepatitis A

Hepatitis A is an inflammation of the liver caused by the hepatitis A virus (HAV). The virus is primarily spread when an uninfected (and unvaccinated) person ingests food or water that is contaminated with the faeces of an infected person. The disease is closely associated with unsafe water or food, inadequate sanitation, poor personal hygiene and oral-anal sex. The incubation period of hepatitis A is usually 14–28 days. Symptoms of hepatitis A range from mild to severe and can include fever, malaise, loss of appetite, diarrhoea, nausea, abdominal discomfort, dark-coloured urine and jaundice (a yellowing of the eyes and skin). Not everyone who is infected will have all the symptoms (WHO).

1.2 Hepatitis B

Hepatitis B is a major global health problem. Hepatitis B is an irritation and swelling (inflammation) of the liver due to infection with the hepatitis B virus (HBV). Hepatitis B spread by contact with the blood or body fluids (such as semen, vaginal fluids, and saliva) of a person who has the virus. Symptoms of hepatitis B may not appear for up to 6 months after the time of infection. Early symptoms include loss of appetite, fatigue, low fever, muscle and joint aches, nausea and vomiting, jaundice, dark urine right upper quadrant pain and hepatomegaly. Hepatitis

B can be prevented with a safe and effective vaccine. The vaccine is usually given soon after birth with boosters a few weeks later. It offers nearly 100% protections against the virus.

1.3 Hepatitis C

Hepatitis C is a viral disease that leads to swelling (inflammation) of the liver. Hepatitis C infection is caused by the hepatitis C virus (HCV). Hepatitis C spreads by contact with the blood of someone who has hepatitis C. The following symptoms may occur with hepatitis C infection like pain in the right upper abdomen, abdominal swelling due to fluid (ascites), clay-colored or pale stools, dark urine, fatigue, fever, itching, jaundice, loss of appetite, nausea and vomiting. Globally, an estimated 50 million people have chronic hepatitis C virus infection, with about 1.0 million new infections occurring per year. WHO estimated that in 2022, approximately 242 000 people died from hepatitis C, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer). Direct-acting antiviral medicines (DAAs) can cure more than 95% of persons with hepatitis C infection, but access to diagnosis and treatment is low. There is currently no effective vaccine against hepatitis C.

1.4 Hepatitis D (Delta Agent)

Delta agent is a type of virus called hepatitis D. It causes symptoms only in people who also have a hepatitis B infection. Hepatitis D virus (HDV) is found only in people who carry the hepatitis B virus. HDV may make a recent (acute) hepatitis B infection or an existing long-term (chronic) hepatitis B liver disease which is worse. It can even cause symptoms in people who carry hepatitis B virus but who never had symptoms. Risk factors include abusing intravenous (IV) or injection drugs, being infected while pregnant (the mother can pass the virus to the baby), carrying the hepatitis B virus, men having sexual intercourse with other men, receiving many blood transfusions. Symptoms may include abdominal pain, dark colored urine, fatigue, jaundice, joint pain, loss of appetite, nausea and vomiting. HDV-HBV co-infection is considered the most severe form of chronic viral hepatitis due to more rapid progression towards hepatocellular carcinoma and liver-related death. Vaccination against hepatitis B is the only method to prevent HDV infection.

1.5 Hepatitis E

Hepatitis E is inflammation of the liver caused by the hepatitis E virus (HEV). The virus has at least 4 different types: genotypes 1, 2, 3 and 4. Genotypes 1 and 2 have been found only in humans. Genotypes 3 and 4 circulate in several animals including pigs, wild boars and deer without causing any disease, and occasionally infect humans.The virus is shed in the stools of infected persons and enters the human body through the intestine. It is transmitted mainly through contaminated drinking water. The infection is usually self-limiting and resolves within 2–6 weeks. Occasionally a serious disease known as fulminant hepatitis (acute liver failure) develops, which can be fatal.

2. Fatty Liver Disease

An increased build-up of fat in the liver. Major risk factors include obesity and type 2 diabetes, though it's also associated with excessive alcohol consumption. It usually causes no symptoms. When symptoms occur, they include fatigue, weight loss and abdominal pain. Treatment involves reducing the risk factors such as obesity through a diet and exercise programme. It is generally a benign condition, but in a minority of patients, it can progress to liver failure (cirrhosis). More than 10 million cases per year (India). There are two types of fatty liver disease. These two types can manifest alone, or they can overlap:

- Alcoholic fatty liver disease, which is caused by heavy alcohol consumption
- Non-Alcoholic fatty liver disease, which is caused by other factors.

Without management, both types of fatty liver disease can cause liver damage, leading to cirrhosis and liver failure.

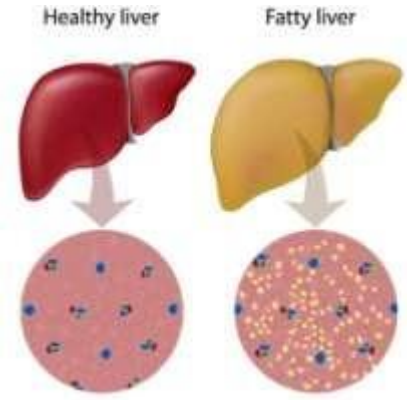

Figure 1: Fatty Liver

3. Alcohol-Related Liver Disease

Alcohol can damage or destroy liver cells. The liver breaks down alcohol so it can be removed from body. Liver can become injured or seriously damaged if drink more alcohol than it can process. There are three main types of alcohol-related liver disease: fatty liver disease, alcoholic hepatitis and alcoholic cirrhosis. Many heavy drinkers will progress from fatty liver disease to alcoholic hepatitis to alcoholic cirrhosis over time. However, some heavy drinkers may develop cirrhosis without having alcoholic hepatitis first. Others may have alcoholic hepatitis but never have symptoms. (O'Shea RS et al., 2010 and Choi G et al., 2012).

4. Auto Immune Conditions

Autoimmune conditions involve immune system mistakenly attacking healthy cells in body.

4.1 Autoimmune Hepatitis

Autoimmune hepatitis is a liver disease that happens when the body's immune system attacks the liver. This can cause swelling, irritation and damage to the liver. The exact cause of autoimmune hepatitis is unclear, but genetic and environmental factors appear to interact over time to trigger the disease. Untreated autoimmune hepatitis can lead to scarring of the liver, called cirrhosis. It can also eventually lead to liver failure. When diagnosed and treated early, however, autoimmune hepatitis often can be controlled with medicines that suppress the immune

system. A liver transplant may be an option when autoimmune hepatitis doesn't respond to medicines or liver disease becomes advanced. Common initial symptoms may include fatigue, nausea, muscle aches, or weight loss or signs of acute liver inflammation including fever, jaundice, and right upper quadrant abdominal pain. Individuals with autoimmune hepatitis often have no initial symptoms and the disease may be detected by abnormal liver function tests and increased protein levels during routine bloodwork or the observation of an abnormal-looking liver during abdominal surgery.

4.2 Primary Biliary Cirrhosis (PBC)

Primary biliary cholangitis (PBC), formerly known as primary biliary cirrhosis, is a chronic disease of the liver, presumably autoimmune in nature, that leads to progressive cholestasis through intrahepatic bile duct destruction and often end-stage liver disease (see the image below). The name change reflects the fact that cirrhosis occurs only in the late stage and therefore does not correctly identify patients with early-stage disease. PBC is most frequently a disease of women and occurs between the fourth and sixth decades of life.

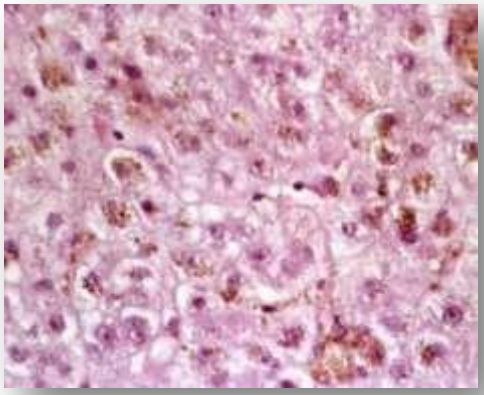

Figure 2: Primary Biliary Cholangitis (Primary Biliary Cirrhosis). This histologic picture is compatible with stage 2 primary biliary cholangitis.

4.3 Primary Sclerosing Cholangitis (PSC)

PSC is a disease that damages and blocks bile ducts inside and outside the liver. Bile is a liquid secreted by liver. Bile ducts are tubes that carry bile out of the liver to the gallbladder and small intestine. In the intestine, bile helps in break down of fat in food. In PSC, inflammation of

the bile ducts leads to scar formation and narrowing of the ducts over time. As scarring increases, the ducts become blocked. As a result, bile builds up in the liver and damages liver cells. Eventually, scar tissue can spread throughout the liver, causing cirrhosis and liver failure. In most people with primary sclerosing cholangitis, the disease progresses slowly. It can eventually lead to liver failure, repeated infections, and tumors of the bile duct or liver. A liver transplant is the only known cure for advanced primary sclerosing cholangitis, but the disease may recur in the transplanted liver in a small number of patients.

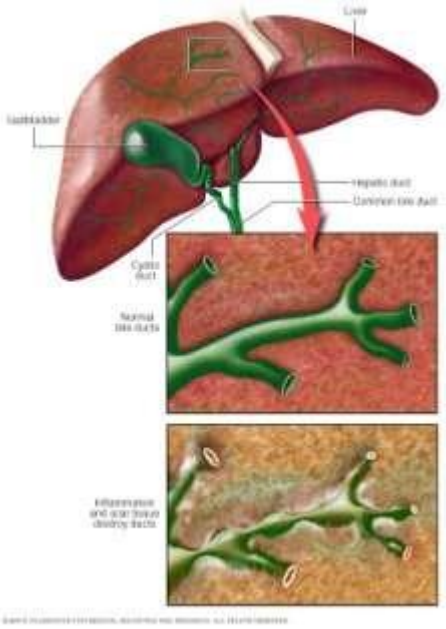

Figure 3**:** Primary Sclerosing Cholangitis (PSC)

5. Genetic Conditions

5.1 Alagille Syndrome

Alagille syndrome is sometimes an autosomal dominant disorder, meaning a person inherits it from one parent who has the disorder. In other cases, a gene mutation develops spontaneously, meaning neither parent carries a copy of the mutated gene. A child who has a

parent with Alagille syndrome has a 50 percent chance of developing the disorder. Most people with Alagille syndrome have a mutation or defect, in the Jagged1 (JAG1) gene. Mutations in the NOTCH2 gene are seen in less than 1 percent of people with Alagille syndrome. Infants with Alagille syndrome may have symptoms of liver disease and poor bile drainage from the liver in the first few weeks. These symptoms can also occur in children and adults with Alagille syndrome. (Kamath BM et al., 2007)

5.2 Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin deficiency (Alpha-1) is a hereditary genetic disorder which may lead to the development of lung and/or liver disease. It is the most common genetic cause of liver disease in children. Adults can also be affected by Alpha-1 and may develop lung conditions such as emphysema as well as liver problems. Fortunately, many persons diagnosed with Alpha-1 never develop any of the associated diseases.

5.3 Wilson’s Disease

Wilson‘s disease, also known as hepatolenticular degeneration and progressive lenticular degeneration, is a rare genetic disorder that causes copper poisoning in the body, especially the liver, brain and eyes. Most people with Wilson's disease are diagnosed between the ages of 5 and 35. But younger and older people can be affected too. Early diagnosis is crucial for stopping the progression of Wilson‘s disease. Treatment may involve taking medication or getting a liver transplant. Delaying or not receiving treatment can cause liver failure, brain damage, or other life threatening conditions.

6. Alcohol-Related Liver Disease

Alcohol can damage or destroy liver cells. The liver breaks down alcohol so it can be removed from body. Liver can become injured or seriously damaged if drink more alcohol than it can process. There are three main types of alcohol-related liver disease: fatty liver disease, alcoholic hepatitis and alcoholic cirrhosis. Many heavy drinkers will progress from fatty liver disease to alcoholic hepatitis to alcoholic cirrhosis over time. However, some heavy drinkers may develop cirrhosis without having alcoholic hepatitis first. Others may have alcoholic hepatitis but never have symptoms.

6.1 Fatty Liver

Fatty liver disease is the build up of extra fat in liver cells. It is the earliest stage of alcoholrelated liver disease. There are usually no symptoms. If symptoms do occur, they may include fatigue, weakness, and weight loss. Almost all heavy drinkers have fatty liver disease. However, if they stop drinking, fatty liver disease will usually go away.

6.2 Alcoholic Hepatitis

Alcoholic hepatitis causes the liver to swell and become damaged. Symptoms may include loss of appetite, nausea, vomiting, abdominal pain, fever and jaundice. Up to 35 percent of heavy drinkers develop alcoholic hepatitis. Alcoholic hepatitis can be mild or severe. If it is mild, liver damage may be reversed. If it is severe, it may occur suddenly and quickly lead to serious complications including liver failure and death.

6.3 Alcoholic Cirrhosis

Alcoholic cirrhosis is the scarring of the liver (hard scar tissue replaces soft healthy tissue). It is the most serious type of alcohol-related liver disease. Symptoms of cirrhosis are similar to those of alcoholic hepatitis. Between 10 to 20 percent of heavy drinkers affect by cirrhosis. The

damage from cirrhosis cannot be reversed and can cause liver failure. Stoping alcohol consumption can help to prevent further damage.

Figure 4: In cirrhosis, scar tissue replaces healthy liver tissue.

7. Liver Hemangioma

Liver hemangioma (he-man-jee-O-muh) is a noncancerous (benign) mass that occurs in the liver. Liver hemangioma is made up of a tangle of blood vessels. Most cases of liver hemangioma are discovered during a test or procedure for some other condition. Most people who have a liver hemangioma never experience signs and symptoms and don't need treatment. There's no evidence that an untreated liver hemangioma can lead to liver cancer.

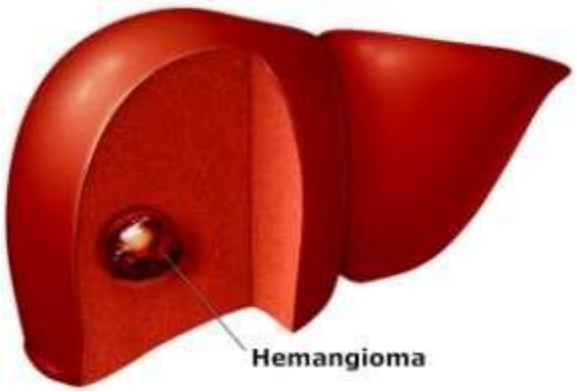

Figure 5: Liver Hemengioma

8. Nonalcoholic Steatohepatitis

Nonalcoholic steatohepatitis or NASH is a common, often ―silent‖ liver disease. It resembles alcoholic liver disease, but occurs in people who drink little or no alcohol. The major feature of NASH is fat in the liver, along with inflammation and damage. Most people with NASH feel well and are not aware that they have a liver problem. Nevertheless, NASH can be severe and can lead to cirrhosis, in which the liver is permanently damaged and scarred and no longer able to work properly. Although having fat in the liver is not normal, by itself it probably causes little harm or permanent damage. If fat is suspected based on blood test results or scans of the liver, this problem is called nonalcoholic fatty liver disease (NAFLD). If liver biopsy is performed in this case reports will show that some people have NASH while others have simple fatty liver.

9. Hepatic Encephalopathy

Hepatic encephalopathy (portosystemic encephalopathy, liver encephalopathy or hepatic coma) is deterioration of brain function that occurs because toxic substances normally removed by the liver build up in the blood and reach the brain. Hepatic encephalopathy may be triggered by bleeding in the digestive tract, an infection, failure to take drugs as prescribed, or another stress in people who have a long-standing (chronic) liver disorder. People become confused, disoriented, drowsy with changes in personality, behavior, and mood.

10. Liver Cancer

Liver cancer originates in liver cells. Symptoms are uncommon in the early stages of liver cancer. Later, symptoms may include weight loss, stomach pain, vomiting and yellowed skin.. If cancer starts elsewhere in the body but spreads to the liver, it‘s called secondary liver cancer. The most common type of liver cancer is hepatocellular carcinoma. It tends to develop as several small spots of cancer in liver, though it can also start as a single tumor. Complications of other liver diseases, especially those that aren‘t treated, may contribute to the development of liver cancer. Treatments vary but may include removal of part of the liver, transplant, chemotherapy and in some cases, radiation.

11. Chronic Liver Failure

Chronic liver disease is a progressive deterioration of liver functions. Liver functions include the production of clotting factors and other proteins, detoxification of harmful products of metabolism, and excretion of bile. This is a continuous process of inflammation, destruction, and regeneration of liver parenchyma leading to fibrosis and cirrhosis. Cirrhosis is a final stage of chronic liver disease that results in disruption of liver architecture, the formation of widespread nodules, vascular reorganization, neo-angiogenesis, and deposition of an extracellular matrix. The underlying mechanism of fibrosis and cirrhosis at a cellular level is the recruitment of stellate cells and fibroblasts that cause fibrosis, while parenchymal regeneration relies on hepatic stem cells.

Test for Liver Diseases

A number of liver function test are available to test the proper function of the liver, (serum proteins, serum albumin, bilirubin (direct and indirect), ALT, AST, GGT, ALP, PT and PTT). Imaging tests such as transient elastography, ultrasound and magnetic resonance imaging can be used to examine the liver tissue and bile ducts. Liver biopsy can be performed to examine liver tissue to distinguish between various conditions; tests such as elastography may reduce the need for biopsy in some situations (Tapper EB, Lok AS, 2017). Liver Biomarkers as well as the related in vitro diagnostic antibodies used for diagnosis being provided.

Nutritional Management in Liver Diseases

According to studies, the liver‘s ability to perform its physiological functions in the body determines the diet of patients with liver diseases. Malnutrition results from the liver‘s inability to metabolize nutrients as a result of chronic liver dysfunctions. The dietary treatment should outline daily calorie targets with a focus on high-quality protein and address any vitamin and micronutrient deficiencies, with a diet high in those nutrients or supplements.

As nutritional issues in patients with advanced liver dysfunctions are multifactorial, treating malnutrition in these patients is difficult. To improve quality of life and prevent medical complications linked to nutrition and improve nutritional status, such patients should have their

nutritional status assessed right away. All stages of chronic liver diseases are associated with the state of protein-energy malnutrition, and patients with chronic liver diseases must consume a typical diet with the addition of supplements as required. For those patients to have a positive long-term outcome, it is critical to conduct an adequate assessment and nutritional therapy, ensuring a proper macronutrient, micronutrient, and vitamin balance. Due to the vital role that the liver plays in controlling nutritional status and energy balance, patients with hepatic diseases are particularly susceptible to developing malnutrition.

Additionally, the presence of chronic liver dysfunctions may cause a decrease in appetite, which may affect the amount of nutrients consumed. Patients who have chronic liver diseases and those who are waiting for a liver transplant are almost always malnourished. Patients with cirrhosis who are malnourished have higher rates of morbidity and mortality. Furthermore, problems and overall survival rates following liver transplants are higher in individuals who are severely malnourished prior to the procedure, despite the crucial role that nutrition plays in the prognosis of persons with cirrhosis, and malnutrition frequently complicates the course of patients with the disease and has complex causes. Despite the crucial role that nutrition plays in the prognosis of those with cirrhosis, the ability to properly manage the patient‘s nutrient needs presents an additional set of challenges due to the catabolic nature of the disease process and the common occurrence of anorexia and other symptoms that lead to a poor oral intake. Malnutrition is a common complication in patients with cirrhosis and has a multifactorial etiology, and, additionally, nutritional condition prior to liver transplantation is one of the most significant factors that affect malnutrition and survival after liver transplantation.

General recommendations for people with severe liver disease include:

- Eat large amounts of carbohydrate foods. Carbohydrates should be the major source of calories in diet.
- Eat a moderate intake of fat, as prescribed by the dietitian. The increased carbohydrates and fat help prevent protein breakdown in the liver.
- Have about 1.2 to 1.5 grams of protein per kilogram of body weight. This means that a 154-pound (70-kilogram) man should eat 84 to 105 grams of protein per day. Look for non-meat

protein sources such as beans, tofu, and dairy products. Take vitamin supplements, especially B-complex vitamins.

- Many people with liver disease are deficient in vitamin D. So should take vitamin D supplements.
- Limit the amount of sodium to 2000 milligrams per day or less to reduce fluid retention.
- Eat fruits and vegetables and lean protein such as legumes, poultry, and fish. Avoid uncooked shellfish due to the risk of hepatitis A.

Conclusion

Liver diseases can be inherited or caused by a variety of factors that damage the liver (virus, drugs or chemicals, obesity, diabetes or an attack from own immune system), when the condition is left untreated, it can become life threatening and can permanently damage the liver or the bile duct. This damage can then become malignant. The liver disease prognosis depends on how quickly the condition was diagnosed and treated. In beginning stages, liver disease usually responds to treatment, but in advanced liver disease, the damage done by fibrosis, cirrhosis and liver failure cannot be reversed. This advanced stage leads to eventual death. While diagnosing liver disease, the condition causing the disease must be treated. If caught early, and are treated correctly, the damage to the liver may heal. In the middle stages of disease, treatment may work to help heal the damage, but as the disease progresses, treatments focus on managing the disease and prolonging the diagnosis.

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DIET IN DIABETES MELLITUS

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INTRODUCTION

Diabetes mellitus (DM) was first recognized as a disease around 3000 years ago by the ancient Egyptians and Indians, illustrating some clinical features very similar to what we now know as diabetes. DM is a combination of two words, ―diabetes‖ Greek word derivative, means siphon - to pass through and the Latin word ―mellitus‖ means honeyed or sweet. In 1776, excess sugar in blood and urine was first confirmed in Great Britain. With the passage of time, a widespread knowledge of diabetes along with detailed etiology and pathogenesis has been achieved.

Diabetes mellitus is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from either insufficient insulin production, ineffective utilization of insulin, or both. This condition poses significant challenges to individuals' health and quality of life, often necessitating ongoing management and lifestyle adjustments, marking diabetes as a non-communicable lifestyle disease and a global public health problem. About 422 million people worldwide have diabetes, the majority living in low-and middle-income countries, and 1.5 million deaths are directly attributed to diabetes each year. Both the number of cases and the prevalence of diabetes have been steadily increasing over the past few decades.

EPIDEMIOLOGY OF DIABETES

The epidemiology of diabetes reveals its significant global burden, with prevalence rates varying across different populations.

Global Burden of Diabetes:

Diabetes mellitus is a major public health concern worldwide, with its prevalence steadily increasing over the past few decades. This rise is primarily attributed to population growth, aging, urbanization, and changes in lifestyle factors such as poor diet and sedentary behavior.

According to the International Diabetes Federation (IDF), approximately 463 million adults (20-79 years old) were living with diabetes globally in 2019. This number is projected to reach 700 million by 2045 if current trends continue. In recent decades, India has witnessed a rapidly exploding epidemic of diabetes. Indeed, India today has the second largest number of people with diabetes in the world. The International Diabetes Federation (IDF) estimates that there are 72.9 million people with diabetes in India in 2017, which is projected to rise to 134.3 million by the year 2045. The prevalence of diabetes in urban India, especially in large metropolitan cities has increased from 2% in the 1970s to over 20% at present and the rural areas are also fast catching up.

Prevalence Rates Across Different Populations:

1. **Regional Variances:** Diabetes prevalence rates vary significantly among different regions of the world. For instance, the Western Pacific region (including China and Japan) and the Middle East have some of the highest prevalence rates, while Africa has comparatively lower rates. However, even within regions, there can be considerable variation.
2. **Urban vs. Rural:** Diabetes rates tend to be higher in urban areas compared to rural areas, primarily due to lifestyle factors such as unhealthy diets, sedentary lifestyles, and higher rates of obesity in urban populations.
3. **Age and Gender:** The prevalence of diabetes increases with age, with older adults being at higher risk. However, there's also an increasing prevalence of type 2 diabetes among children and adolescents, particularly in developed countries. Historically, type 2 diabetes was more common in adults, but changing lifestyle patterns have led to an earlier onset of the disease. Additionally, men generally have slightly higher rates of diabetes compared to women, although this can vary by region and age group.

Efforts to address the global burden of diabetes include public health campaigns promoting healthy lifestyles, initiatives to improve access to healthcare and diabetes management resources, and research aimed at developing more effective prevention and treatment strategies. However, concerted efforts at both individual and population levels are essential to mitigate the impact of diabetes on public health.

CLASSIFICATION OF DIABETES

According to the American Diabetes Association and the World Health Organisation, diabetes can be classified into four main types.

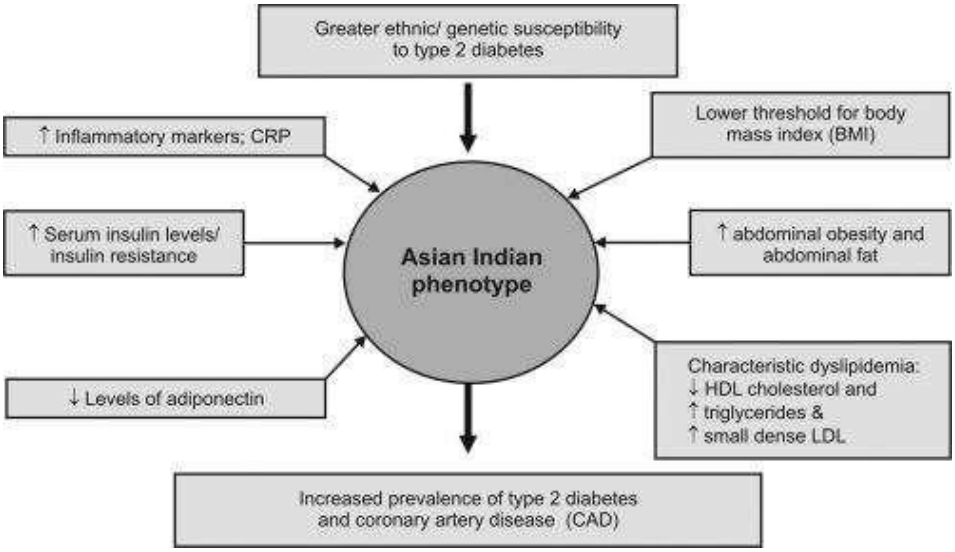
1. Type 1 diabetes
2. Type 2 diabetes
3. Gestational diabetes
4. Other types of diabetes (Monogenic diabetes, pancreatic diabetes, drug-induced diabetes etc.).

These several types of diabetes are, each with its own causes, characteristics, and treatments:

1. **Type 1 Diabetes (T1D):** Type 1 diabetes accounts for 5% to 10% of all cases of diabetes. This results from the autoimmune destruction of the insulin-producing beta cells in the pancreas, leading to an absolute deficiency of insulin. Its risk factors include autoimmune, genetic, and environmental factors. To date, there are no known ways to prevent type 1 diabetes. Type 1 diabetes typically develops during childhood or adolescence but can occur at any age. Individuals with T1D require lifelong insulin therapy for survival.
2. **Type 2 Diabetes (T2D):** Type 2 diabetes accounts for 90% to 95% of all diagnosed diabetes cases. This is characterized by insulin resistance, where the body's cells become less responsive to insulin, combined with inadequate insulin secretion. This form of diabetes generally begins as insulin resistance and, because the body is unable to produce enough insulin to address the resistance, the pancreas may reduce the production of insulin or eventually stop producing it. Type 2 diabetes is strongly associated with lifestyle factors such as obesity, physical inactivity, and poor diet. It often develops in adulthood but is increasingly diagnosed in children and

adolescents due to rising obesity rates. Initially, treatment may involve lifestyle modifications, oral medications, and eventually insulin therapy as the disease progresses. The ―Asian Indian phenotype‖ refers to a peculiar constellation of abnormalities in south Asians, whereby for any given level of body-mass index, they tend to have higher total body fat, visceral fat, insulin resistance and prevalence of diabetes diabetes compared to white Caucasians (Figure 1.1)

Figure 1. The "Asian Indian phenotype"



3. **Gestational Diabetes Mellitus (GDM):** Minority women, women who are obese, women with a family history of diabetes, and women who have had gestational diabetes in a previous pregnancy are at higher risk than other women for developing gestational diabetes. GDM increases the risk of complications for both the mother and baby during pregnancy and childbirth. Strict glycemic control and management of women with gestational diabetes is necessary to prevent birth complications in the developing infant. While blood sugar levels often return to normal after delivery, women who have had GDM have a 20% to 50% increased risk for developing type 2 diabetes later in life.
4. **Other Specific Types:** There are other specific forms of diabetes caused by genetic mutations, diseases of the pancreas, drug-induced factors, or endocrine disorders. Examples include monogenic diabetes syndromes (such as maturity-onset diabetes of the young, MODY),

pancreatic diseases, and diabetes induced by certain medications or hormonal conditions. Other types of diabetes include:

4.1. **Type 3c diabetes**: This form of diabetes happens when your pancreas experiences damage (other than autoimmune damage), which affects its ability to produce insulin. Pancreatitis, pancreatic cancer, cystic fibrosis and hemochromatosis can all lead to pancreas damage that causes diabetes. Having your pancreas removed (pancreatectomy) also results in Type 3c.

4.2. **Latent autoimmune diabetes in adults (LADA)**: Like Type 1 diabetes, LADA also results from an autoimmune reaction, but it develops much more slowly than Type 1. People diagnosed with LADA are usually over the age of 30.

4.3. **Maturity-onset diabetes of the young (MODY)**: MODY, also called monogenic diabetes, happens due to an inherited genetic mutation that affects how your body makes and uses insulin. There are currently over 10 different types of MODY. It affects up to 5% of people with diabetes and commonly runs in families.

4.4. **Neonatal diabetes**: This is a rare form of diabetes that occurs within the first six months of life. It‘s also a form of monogenic diabetes. About 50% of babies with neonatal diabetes have the lifelong form called permanent neonatal diabetes mellitus. For the other half, the condition disappears within a few months from onset, but it can come back later in life. This is called transient neonatal diabetes mellitus.

4.5. **Brittle diabetes**: Brittle diabetes is a form of Type 1 diabetes that‘s marked by frequent and severe episodes of high and low blood sugar levels. This instability often leads to hospitalization. In rare cases, a pancreas transplant may be necessary to permanently treat brittle diabetes.

Prediabetes: In addition to these main types, there's a condition called prediabetes, where blood sugar levels are higher than normal but not yet high enough to be diagnosed as diabetes. Prediabetes is a precursor condition to diabetes in which a person has elevated blood glucose levels but does not meet diagnostic criteria for diabetes. People with prediabetes can have impaired fasting glucose (IFG) or impaired glucose tolerance (IGT), or both. Prediabetes increases the risk of developing type 2 diabetes and cardiovascular disease.

Etiology and Risk Factors of diabetes

Although the pathogenesis of diabetes is complex, a number of factors that increase the risk for the disease have been identified. Risk factors for type 1 diabetes include family history, race (with whites at higher risk than other racial or ethnic groups), and certain viral infections during childhood. Risk factors for type 2 diabetes are more diverse; some are modifiable, and others are not. Non-modifiable risk factors for type 2 diabetes include age, race or ethnicity, family history (genetic predisposition), history of gestational diabetes, and low birth weight.

Modifiable or lifestyle risk factors include increased body mass index (BMI), physical inactivity, poor nutrition, hypertension, smoking, and alcohol use, among others. Increased BMI is consistently shown to be one of the strongest risk factors for development of diabetes. In addition, distribution of body fat, and specifically an increased waist-to-hip ratio, increase a person's risk for diabetes. Consistent findings from various studies show that lower levels of physical activity increase a person's risk for diabetes. Total caloric intake, as well as specific components of diet such as refined carbohydrates and fats, have been linked to diabetes development. Moderate alcohol use may reduce the risk for developing diabetes, but smoking has been shown to be an independent risk factor for diabetes. Psychosocial factors such as depression, increased stress, lower social support, and poor mental health status also are associated with an increased risk for the development of diabetes. Recently, adverse housing conditions were found to be independently associated with the development of self-reported diabetes, although the mechanism by which housing conditions exert their risk is still unknown.

Thus the etiology of diabetes involves a complex interplay of genetic, environmental, and lifestyle factors. Understanding these factors is crucial for identifying individuals at risk and implementing effective prevention strategies.

Genetic Predisposition:

1. **Family History:** Having a family history of diabetes increases the risk of developing the condition. Type 1 diabetes is strongly influenced by genetic factors, with certain human leukocyte antigen (HLA) genotypes being associated with increased susceptibility. Recent exploration of human genomic regions associated with type 1 diabetes has identified candidate genes, gene products, and genetic loci that influence susceptibility to type 1 diabetes. Type 2 diabetes also has a genetic component, with multiple genes contributing to disease risk.
2. **Genetic Mutations:** Certain rare genetic mutations can lead to monogenic forms of diabetes, such as maturity-onset diabetes of the young (MODY) and neonatal diabetes. These mutations affect the function of genes involved in insulin production and secretion.

Environmental Factors:

1. **Obesogenic Environment:** The global rise in obesity rates is a major environmental factor contributing to the increased prevalence of type 2 diabetes. Environmental factors promoting overeating and sedentary behavior, such as easy access to high-calorie foods and reduced opportunities for physical activity, play a significant role. People who are obese, have a family history of diabetes, or belong to high-risk ethnic groups (e.g., African American, Native American, Hispanic, and Pacific Islanders) should be regularly screened for the disorder.
2. **Urbanization:** Urban environments often promote unhealthy lifestyle behaviors, including poor dietary choices and decreased physical activity, which can contribute to the development of diabetes.
3. **Infections:** Exposure to some viral infections (e.g., mumps), trauma (e.g., motor vehicle accident) and environmental factors (e.g., climate, and geographical location) could trigger the onset of type 1 diabetes.

Lifestyle Influences:

1. **Diet:** Poor dietary habits, such as consuming excessive amounts of refined carbohydrates, sugary beverages, and processed foods high in unhealthy fats, contribute to the development of obesity

and insulin resistance, increasing the risk of type 2 diabetes. A diet rich in fruits, vegetables, whole grains, and lean proteins can help reduce diabetes risk.

2. **Physical Activity:** Sedentary lifestyles are strongly associated with an increased risk of type 2 diabetes. Regular physical activity improves insulin sensitivity, helps maintain a healthy weight, and reduces diabetes risk.
3. **Stress:** Chronic stress and poor coping mechanisms can contribute to unhealthy behaviors such as overeating, physical inactivity, and disrupted sleep patterns, all of which increase the risk of diabetes.

Other Risk Factors:

1. **Age:** While gender does not appear to be a significant determinant of type 1 diabetes as incidence rates are generally similar for males and females, the risk of type 1 diabetes increases with age during childhood and adolescence. The risk of diabetes increases with age, particularly for type 2 diabetes. This is partly due to age-related changes in metabolism, decreased physical activity, and increased prevalence of obesity.
2. **Ethnicity:** Certain ethnic groups, including African Americans, Hispanics, Native Americans, and Asian Americans, have a higher risk of developing diabetes compared to Caucasians. This increased risk may be attributed to genetic predisposition, cultural factors, and socioeconomic disparities.
3. **Socioeconomic Status:** Socioeconomic factors such as low income, limited access to healthcare, and inadequate education can contribute to disparities in diabetes prevalence and outcomes. Individuals with lower socioeconomic status are more likely to experience barriers to healthy lifestyle behaviors and diabetes management.
4. **Gestational Diabetes:** Individuals of all ages diagnosed as prediabetic and pregnant mothers diagnosed with gestational diabetes are at high risk for developing type 2 diabetes.

Understanding the multifaceted nature of diabetes risk factors is essential for developing comprehensive prevention and management strategies. Efforts to address diabetes should focus

on promoting healthy lifestyles, improving access to healthcare and education, and addressing social determinants of health to reduce disparities in diabetes prevalence and outcomes.

PHYSIOLOGY OF GLUCOSE METABOLISM

Understanding the physiology of glucose metabolism is crucial for comprehending how the body regulates blood sugar levels in different metabolic states.

1. Normal Glucose Homeostasis:

Glucose Regulation in the Fed State:

- After a meal, blood glucose levels rise due to the absorption of carbohydrates from the diet.
- In response to elevated blood glucose levels, the pancreas releases insulin.
- Insulin promotes the uptake of glucose into cells, particularly muscle, liver, and adipose tissue.
- In muscle and adipose tissue, insulin stimulates glucose uptake by facilitating the translocation of glucose transporter proteins (GLUT4) to the cell membrane.
- In the liver, insulin inhibits gluconeogenesis (the synthesis of glucose from non-carbohydrate sources) and promotes glycogen synthesis, leading to storage of glucose as glycogen.

Glucose Regulation in the Fasting State:

- Between meals or during periods of fasting, blood glucose levels begin to decline.
- In response to decreased blood glucose levels, the pancreas reduces insulin secretion while increasing glucagon secretion.
- Glucagon promotes glycogen breakdown (glycogenolysis) in the liver, releasing glucose into the bloodstream.
- Glucagon also stimulates gluconeogenesis, primarily from amino acids derived from protein breakdown.
- Together, these processes help maintain blood glucose levels within a normal range during fasting.

2. Insulin Action and Secretion:

Role of Pancreatic Beta Cells:

- Insulin is synthesized and secreted by beta cells in the pancreas in response to elevated blood glucose levels.
- Beta cells sense glucose levels through glucose transporters and glucose metabolism pathways.
- Insulin secretion is tightly regulated by glucose and other factors, including incretin hormones released from the gut in response to food intake.

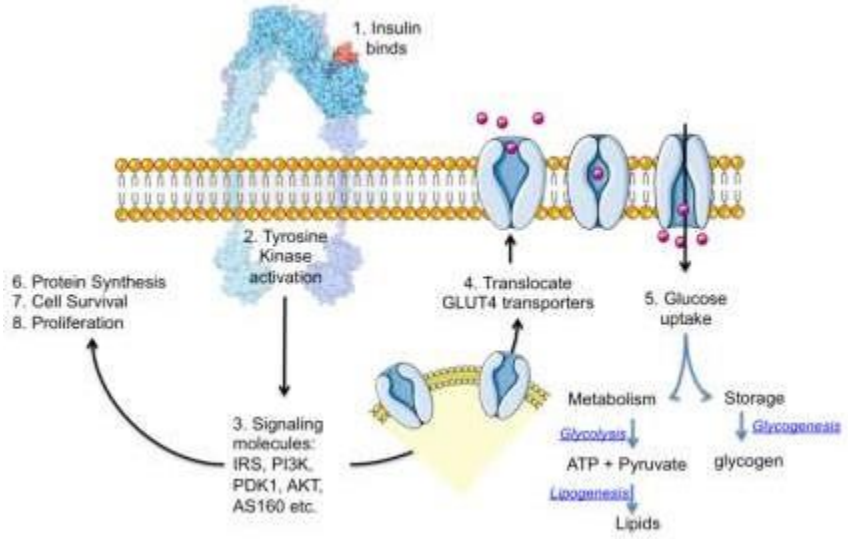
Insulin Signaling Pathways:

- Insulin binds to insulin receptors on target cells, initiating intracellular signaling pathways.
- The insulin receptor activates tyrosine kinase activity, leading to phosphorylation of downstream signaling molecules.
- These signaling cascades ultimately promote glucose uptake, glycogen synthesis, protein synthesis, and lipid storage in insulin-sensitive tissues.

Insulin Resistance: Mechanisms and Implications:

- Insulin resistance occurs when cells become less responsive to insulin's actions, leading to impaired glucose uptake and metabolism.
- It is a key feature of type 2 diabetes and is often associated with obesity, inflammation, and other metabolic abnormalities.
- Insulin resistance results in compensatory hyperinsulinemia as the pancreas secretes more insulin to overcome resistance.
- Over time, persistent insulin resistance can lead to beta cell dysfunction and impaired insulin secretion, further exacerbating hyperglycemia.
- Insulin binds to cell surface receptors to initiate a signaling cascade (Figure 2) that in turn mobilizes glucose transporter proteins to the cell surface to take up glucose. In the absence of the insulin generated signals, these transporter proteins are sequestered in the cell so the cells cannot take up glucose. Exercise activates other signals that can also translocate GLUT4 transporters to the cell surface. This is one of the mechanisms by which exercise increases insulin sensitivity.

Figure 2: Insulin Directed Glucose Uptake



Courtesy: (https://pdb101.rcsb.org/motm/182)

3. Glucagon and Counter Regulatory Hormones:

Role in Glucose Regulation:

- Glucagon is secreted by alpha cells in the pancreas and plays a crucial role in maintaining glucose homeostasis.
- It acts opposite to insulin, promoting glycogen breakdown and gluconeogenesis to increase blood glucose levels during fasting or hypoglycemia.
- Other counter regulatory hormones, such as cortisol, epinephrine, and growth hormone, also contribute to glucose regulation by promoting glycogenolysis and gluconeogenesis.

Interaction with Insulin:

- Glucagon and insulin have reciprocal effects on glucose metabolism, ensuring fine-tuned control of blood glucose levels.
- Insulin inhibits glucagon secretion and glucagon-mediated glucose production, while glucagon antagonizes insulin's actions on glucose uptake and storage.
- The balance between insulin and glucagon secretion is critical for maintaining glucose homeostasis in response to varying metabolic demands and nutrient availability.

Understanding the intricate interplay between insulin, glucagon, and other hormones is essential for maintaining normal glucose metabolism and preventing dysregulation that can lead to metabolic disorders such as diabetes mellitus.

PATHOPHYSIOLOGY OF DIABETES

Type 1 Diabetes Mellitus (T1DM):

1. **Autoimmune Destruction of Beta Cells**: In Type 1 diabetes, the immune system mistakenly targets and attacks the insulin-producing beta cells in the pancreas. This autoimmune response leads to the destruction of these cells, resulting in a significant reduction or complete absence of insulin production.
2. **Insulin Deficiency**: As a consequence of beta cell destruction, there is a severe deficiency in insulin secretion. Insulin is essential for regulating glucose levels in the blood by facilitating the uptake of glucose into cells for energy production or storage. Without sufficient insulin, glucose accumulates in the bloodstream, leading to hyperglycemia.
3. **Pathogenesis and Triggers**: The exact cause of the autoimmune response leading to T1DM is not fully understood. However, genetic predisposition, environmental factors, and potential triggers such as viral infections are believed to play a role in initiating the autoimmune attack on beta cells. Once triggered, the immune system continues to attack and destroy beta cells, progressively worsening insulin deficiency.

Type 2 Diabetes Mellitus (T2DM):

1. **Insulin Resistance and Beta Cell Dysfunction**: Type 2 diabetes is characterized by insulin resistance, where the body's cells become less responsive to the action of insulin. This resistance impairs glucose uptake by cells, leading to elevated blood sugar levels. Additionally, there is a gradual decline in beta cell function and insulin secretion over time, contributing to the progression of hyperglycemia.
2. **Role of Obesity and Adipose Tissue**: Obesity is a major risk factor for the development of insulin resistance and Type 2 diabetes. Adipose tissue, especially visceral fat, releases inflammatory cytokines and adipokines, which interfere with insulin signaling pathways and

promote insulin resistance. Excess adiposity also leads to dyslipidemia and contributes to the development of metabolic syndrome, further exacerbating insulin resistance.

3. **Inflammatory Pathways**: Chronic low-grade inflammation is associated with insulin resistance and beta cell dysfunction in Type 2 diabetes. Adipose tissue inflammation, along with increased levels of pro-inflammatory cytokines, such as TNF-alpha and IL-6, contributes to insulin resistance by impairing insulin signaling pathways in target tissues, such as liver, muscle, and adipose tissue.

Gestational Diabetes Mellitus (GDM):

1. **Pregnancy-Induced Insulin Resistance**: During pregnancy, hormonal changes lead to increased insulin resistance in women, especially in the second and third trimesters. This physiological insulin resistance ensures an adequate supply of glucose to the developing fetus. However, in some women, particularly those with pre-existing insulin resistance or impaired pancreatic function, this insulin resistance becomes exaggerated, leading to the development of gestational diabetes mellitus.
2. **Implications for Mother and Fetus**: Untreated or poorly managed gestational diabetes can result in adverse outcomes for both the mother and the fetus. Maternal complications may include an increased risk of developing Type 2 diabetes later in life, hypertension, and preeclampsia. For the fetus, GDM increases the risk of macrosomia (large birth weight), birth trauma, hypoglycemia after birth, and an increased likelihood of developing obesity and Type 2 diabetes in later life. Therefore, early detection and appropriate management of GDM are crucial to minimizing these risks.

CLINICAL PRESENTATION

The symptoms and signs of diabetes can vary depending on the type and severity of the condition. Common symptoms include:

Hyperglycemia:

The main symptoms of diabetes are described as the three polys - polyuria, polydipsia, and polyphagia.

- **Polyuria** or the need to urinate frequently helps the body remove excess glucose that is filtered from the blood by the kidneys
- **Polydipsia** or increased thirst and fluid intake compensates for the loss of fluids resulting from increased urination
- **Polyphagia** or increased appetite compensates for the loss of glucose and fluids from the body, caused by excessive urination

Individuals with high risk for developing diabetes should be alert to these symptoms and seek medical attention if they notice the above symptoms. Beyond the three "polys", other indicators for diabetes (listed by American Diabetes Association) include:

- Extreme fatigue
- Blurry vision
- Slow-healing sores or frequent infections: High blood sugar levels can impair the immune system's ability to fight infections.
- Weight loss
- Tingling, pain, or numbness in the hands/feet

In more advanced stages of diabetes, the following symptoms and diabetes complications may also be seen

- Fainting and/or dizziness (possibly due to sudden drops in blood glucose levels due to poor management or increased medication)
- Foot ulcers (resulting from inability to attend to foot injuries due to loss of sensation in the extremities. These conditions may lead to gangrene and amputation)
- Peripheral neuropathy (loss of sensation, autonomic dysfunction)
- Retinopathy (blindness, blurred vision, or eye problems)
- Nephropathy (kidney damage)

- Damage to nerves (usually affects the extremities first) and bloods vessels, exposing diabetics to risk of cardiovascular complications (e.g., high blood pressure, heart disease, heart attack, stroke)
- Increased susceptibility to infections (e.g., urinary tract infections, sepsis, gangrene), since high glycemic environment provides a favorable medium for microbes

COMPLICATIONS OF DIABETES

Uncontrolled diabetes can cause metabolic imbalance leading to acute complications that may require immediate medical attention. Hyperglycemia (high levels of glucose in blood) sets the stage for protein glycation, which in turn may lead to chronic conditions requiring constant monitoring and treatment. Individuals with diabetes may also develop high cholesterol and high blood pressure, which require medical management.

Acute Complications

Ketoacidosis

Despite high blood glucose levels, the body cells (muscle and lipid cells) may be starved for glucose due to absence or improper function of insulin. Cells may also starve for glucose if the amount of food intake is low (such as during illness) or if the dosage of insulin is too high. Under these conditions cells start using fats as a source of energy. Liver cells produce ketone bodies from fatty acids. When the glucose levels are low, brain cells can use ketone bodies, but not free fatty acids, for energy. High concentrations of ketones can make the urine acidic and cause fruity-smelling breath. If not managed, this condition can progress to coma (prolonged unconsciousness) and even death.

Hyperosmolar Hyperglycemic Nonketotic Syndrome (HHNS)

High blood glucose level triggers increased urination. If liquids are not replaced, the individual can become severely dehydrated. High blood glucose levels can lead to altered mental states, confusion, seizures, coma, and even death.

Chronic Complications

Complications of diabetes can broadly be categorized into microvascular, macrovascular, and other complications.

1. Microvascular Complications

2. Macrovascular Complications

Microvascular complications

These affect the smaller blood vessels, such as in the eyes (leading to retinopathy), kidneys (leading to nephropathy), and neurons (leading to neuropathy). Individuals with poorly managed blood glucose levels may suffer from one or more of these complications in advanced stages of the disease. Thus, besides monitoring the health of eyes and kidneys, diabetics also require foot care. Interestingly, several large population studies have shown that aggressive management of blood glucose levels (i.e., keeping blood glucose levels within a narrow range) can avoid, or at least delay, the onset of these complications (Nathan et al., 2014). Regular monitoring and management of blood glucose levels is of critical importance in maintaining metabolic balance and avoiding microvascular complications.

1. **Diabetic Retinopathy**: Damage to the blood vessels in the retina, leading to vision problems and potentially blindness if left untreated.
2. **Diabetic Nephropathy**: Kidney damage caused by prolonged high blood sugar levels, which can lead to kidney failure if not managed properly.
3. **Diabetic Neuropathy**: Nerve damage due to diabetes, which can cause numbness, tingling, pain, or weakness, commonly affecting the feet and legs.

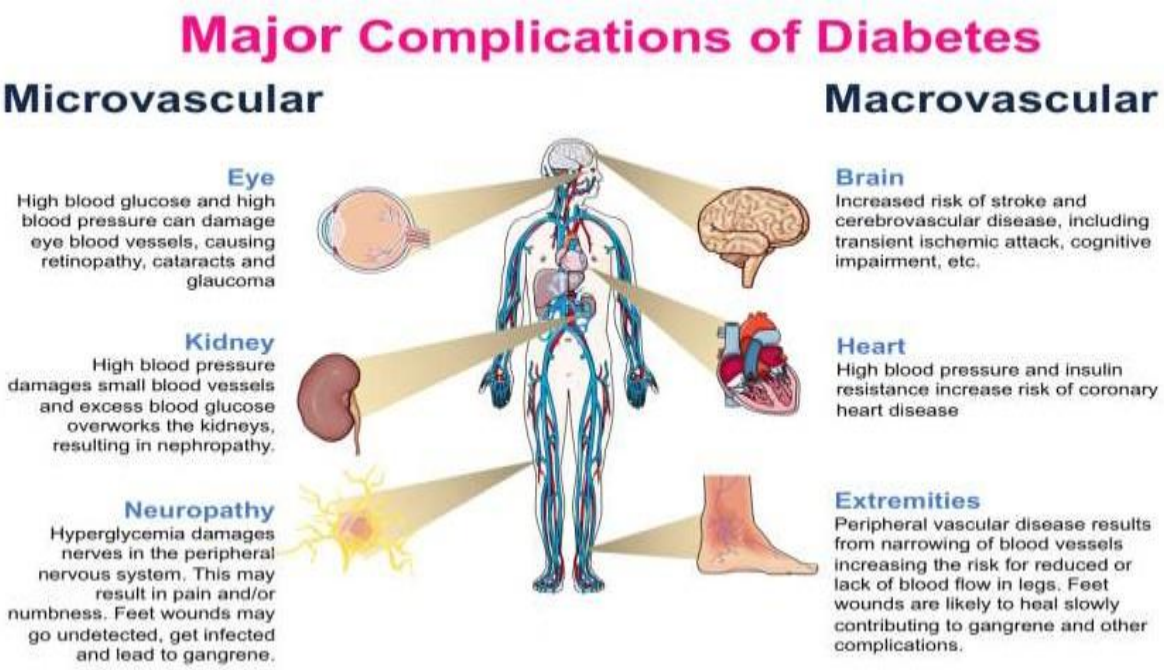
Macrovascular complications

These affect larger blood vessels, such as those supplying the heart, brain, and extremities. The causes of these complications stem from narrowing of blood vessels due to glycation, inflammation, lipid deposition and other factors. Complications resulting from large vessel damage may lead to cardiomyopathy, stroke, rheumatoid arthritis, osteoporosis, and the

degenerative process of aging (Singh et al., 2014). The major concern amongst these complications is myocardial infarction (heart attack). At present, it appears that blood glucose control does not significantly reduce the risks or delay the onset of macrovascular complications. Additional medical management is required.

1. **Cardiovascular Disease**: Diabetes increases the risk of various heart conditions, including coronary artery disease, heart attack, and heart failure.
2. **Peripheral Artery Disease (PAD)**: Narrowing or blockage of blood vessels in the legs, leading to reduced blood flow and potentially causing pain, numbness, or even tissue death (gangrene).
3. **Stroke**: Diabetes is a significant risk factor for stroke, a condition where blood supply to the brain is disrupted, leading to brain damage and potential disability or death.

Figure 3. Complications of Diabetes Mellitus



Parts of the image were adapted from Servier Medical Art.

Protein Glycation

When proteins are exposed to high levels of sugar, for long periods of time, they may covalently attach to proteins by a non-enzymatic process, called glycation (Singh et al., 2014). Note that this process differs from the post-translational glycosylation of proteins in rough endoplasmic reticulum and Golgi, where sugars are attached to proteins systematically, by specific enzymes and carrier proteins. In early stages of glycation, sugars react with free amino groups on the protein to form a Schiff base, which further undergoes rearrangements to form a more stable product called amadori product. Over time, these glycated proteins may be further modified to form advanced glycation products (AGEs). Receptors for AGEs bind to these adducts and initiate an inflammation response, which in turn lead to some of the complications of diabetes.

In poorly managed diabetes, the blood glucose levels may be high and proteins found in the blood, such as albumin, immunoglobulins, and collagen, are often glycated. A glycated product of hemoglobin (an oxygen transporting molecule present in red blood cells), called Hb A1c is used as a measure of overall glucose management in individuals with diabetes.

Diabetes and Foot Care

Diabetic foot infection (DFI) is a common and complex problem in individuals with diabetes. Due to nerve damage and numbness, individuals with diabetes may not feel any pain and foot ulcers may go undetected for long periods. Infections in these ulcers may also go untreated and even when detected may heal slowly (due to peripheral artery disease). In extreme cases, the ulcers and infections may lead to gangrene and require amputation.

Treating foot ulcers in diabetic patients is challenging. Often, multiple microorganisms infect the ulcers, some of which may be resistant to standard antimicrobial treatments. In order to effectively treat the infection, ideally the ulcer microbiome (genomes of all microorganisms found in the ulcer) should be studied. State of the art treatment protocols use molecular techniques such as polymerase chain reaction (PCR) and gene sequencing to identify key components of the ulcer microbiota and design specific treatments. However, such thorough analysis is current

Skin Complications: Diabetes can cause various skin problems, including bacterial and fungal infections, itching, and dry skin.
Dental Complications: Diabetes increases the risk of gum disease (periodontitis) and other oral health issues, which can lead to tooth loss if not treated promptly.

Complications of gestational diabetes

Most women who have gestational diabetes deliver healthy babies. However, untreated or uncontrolled blood sugar levels can cause problems for you and your baby.

Complications in your baby can be caused by gestational diabetes, including:

- **Excess growth.** Extra glucose can cross the placenta. Extra glucose triggers the baby's pancreas to make extra insulin. This can cause your baby to grow too large. It can lead to a difficult birth and sometimes the need for a C-section.
- **Low blood sugar.** Sometimes babies of mothers with gestational diabetes develop low blood sugar (hypoglycemia) shortly after birth. This is because their own insulin production is high.
- **Type 2 diabetes later in life.** Babies of mothers who have gestational diabetes have a higher risk of developing obesity and type 2 diabetes later in life.
- **Death.** Untreated gestational diabetes can lead to a baby's death either before or shortly after birth.

Complications in the mother also can be caused by gestational diabetes, including:

- **Preeclampsia.** Symptoms of this condition include high blood pressure, too much protein in the urine, and swelling in the legs and feet.

- **Gestational diabetes.** If you had gestational diabetes in one pregnancy, you're more likely to have it again with the next pregnancy.

These complications highlight the importance of proper diabetes management, including blood sugar control, regular medical check-ups, and lifestyle modifications to reduce the risk and severity of these complications.

DIAGNOSIS OF DIABETES

Diabetes is diagnosed through various tests that measure blood glucose levels. The diagnostic criteria include:

1. **Fasting Plasma Glucose (FPG) Test:**
    - A blood sample is taken after an overnight fast (typically 8 hours).
    - A fasting plasma glucose level of 126 mg/dL (7.0 mmol/L) or higher on two separate occasions indicates diabetes.

How to Determine Blood (or Plasma) Glucose Levels?

Measuring blood glucose concentrations is critical for diagnosis and monitoring of diabetes. This is usually done using a glucometer with test strips. A drop of blood is placed on the glucose test strip and exposed to a fixed quantity of an enzyme, glucose oxidase (shown in Figure 1), for a finite period of time. Glucose oxidase acts as a biosensor and oxidizes glucose to generate products (gluconic acid and hydrogen peroxide). In older models, the amount of hydrogen produced was measured by a color change reaction to indicate the glucose concentration. In modern glucometers, the hydrogen peroxide interacts with a mediator to transfer electrons to a microcircuitry included in the test strip to give a reading of blood glucose concentration. In order to get consistent glucometer readings, the test strips should be stored away from moisture and extremes of temperature so that the glucose oxidase coated on the test-strip is not denatured and rendered non-functional.

2. **Oral Glucose Tolerance Test (OGTT):**
    - This is an elaboration of the blood glucose test that reveals how the body metabolizes glucose ~2 hours after ingesting glucose. For pregnant mothers with high risk of diabetes, a glucose-screening test (OGTT) is usually performed between 24-28 weeks of pregnancy (or earlier) to identify gestational diabetes and manage the blood glucose levels for the health of mother and baby.
    - After blood is drawn, the subject is asked to drink a liquid, which contains 50, 75, or 100 grams of glucose. Normally the absorption of glucose in the body occurs rapidly, and blood glucose levels rise within 30 to 60 minutes of fluid intake. Blood work is done an hour after drinking the solution and blood glucose measurements are taken at specific intervals.
    - If the blood test shows abnormal blood glucose levels, then blood work must be repeated after three hours.
    - A blood glucose level of 200 mg/dL (11.1 mmol/L) or higher 2 hours after consuming the glucose solution confirms diabetes.
3. **Hemoglobin A1c (HbA1c) Test:**
    - This test measures the average blood glucose level over the past 2-3 months by assessing the percentage of glycated hemoglobin in the blood. Prolonged exposure to high levels of glucose leads to glycation of proteins that are exposed to blood. Red Blood Cells (or RBCs) have a 3-4 month life-span thus measuring the level of glycation of hemoglobin reflects an individual's average blood glucose control for the past 2 to 3 months. High percentage of A1c reflects poor control of blood glucose levels over the past few months.

    - Prolonged exposure to high blood glucose levels leads to glucose attaching itself to proteins including hemoglobin. Since this happens over a period of time and is irreversible - the HbA1c test is not affected by random fluctuations due to temporary alteration in diet, lifestyle, stress or illness of the subject. Maintaining an HbA1c below 5.7% is indicative of normal health. An HbA1c level of 5.7-6.4% signals prediabetes, and any values higher than 6.4% indicates a diabetic condition requiring treatment.

The diagnostic criteria for diabetes and prediabetes are summarized in Table 1.

Table 1. Diagnostic Criteria for Diabetes and Prediabetes*

Blood tests	Diabetics	Pre diabetics		Non-diabetics
		IFG	IGT	
FBS (mg/dl)	≥ 126	110 to 125	<126	<110
$2h Plasma Glucose (mg/dl)	≥ 200	<140	≥140 to <200	<140
HbA1C	≥ 6.5	5.7-6.4		<5.7

$*Venous plasma glucose 2 hours after ingestion of 75 g oral glucose load (OGTT)*

**WHO (2016) & IDF (2017)*

It is important to note that diagnosis should be based on confirmatory testing and not solely on symptoms, as some individuals may be asymptomatic despite having high blood sugar levels. Additionally, diagnosis should consider other factors such as age, risk factors, and comorbidities. Early detection and management of diabetes are essential for preventing complications and improving outcomes.

MANAGEMENT OF DIABETES

Managing diabetes involves a multifaceted approach that encompasses lifestyle modifications, pharmacotherapy, and monitoring/self-care practices. Here's a breakdown of each aspect:

Lifestyle Modifications

The cornerstone of diabetes management is tight glycemic control. The first line of action upon noticing any symptoms of type 2 diabetes should be lifestyle changes, i.e., managing blood glucose levels by regulating diet (both quality and quantity of nutritional intake) and exercise. Through these changes, individuals diagnosed as prediabetic can often check their progression to full-blown disease. Individuals diagnosed with diabetes, who are taking medications can also benefit from these lifestyle changes.

Lifestyle Goals in Diabetes:

- To improve health through optimum nutrition
- To provide energy for reasonable body weight, normal growth and development
- To maintain glycemic control
- To achieve optimum blood lipid levels
- To individualise the diet according to complications and co-morbidities
- Achieve optimal physical activity
- Advise other behavioural changes for: smoking, other tobacco products and alcohol
- Advocate stress management

Medical Nutrition Therapy (MNT) for diabetes mellitus requires application of nutritional and behavioral sciences along with physical activity.

A four-pronged approach is needed:

1. Nutritional assessment which includes metabolic, nutritional and life style parameters
2. Setting goals – practical, achievable and acceptable to the patient—individualized Nutritional Intervention, including nutrition education – individualized meal plans according to family eating patterns
3. Evaluation – to assess if the goals have been achieved and to make necessary changes.

Based on factors like age, sex, physical activity, height, weight, body mass index ICMR.

The diet should be individualized, close to the family pattern, flexible, should have variety and meal timing should be according to the patient‘s daily schedule.

1. Dietary Recommendations:

(i) Energy:

Sufficient to attain or maintain a reasonable body weight for adults, normal growth and development for children and adolescents, to meet the increased needs during pregnancy and lactation and recovery from illness. Daily physical activity and exercise needs to be considered.

Ideal Body Weight (IBW) = (Height in cm – 100) x 0.9.

Approximately, 25 kcals/kg ideal body weight/day can be given to a moderately active patient with diabetes. The change in the daily calorie should be a gradual process, and not more than 500 calories/day. (ii) Energy or Calorie Distribution: (a) Carbohydrates:

Evidence is inconclusive for an ideal amount of carbohydrate intake for people with diabetes.

Therefore, collaborative goals should be developed for individuals with diabetes. 55-60 % of energy from carbohydrates is an ideal recommendation.Carbohydrates should be complex in nature. Although different carbohydrates produce different glycemic responses, from clinical point of view it is important to manage total carbohydrate. It is recommended that carbohydrates from foods high in fi bre e.g. whole grains (unpolished cereals and millets), legumes, peas, beans, oats, barley and some fruits with low glycemic index and glycemic load are consumed. All patients with diabetes should be encouraged to take 6 small meals a day. Food exchange system can be followed to give more variety and individualization to the diet plan.

(a) Fibre:

Fibre recommendation for general population is 40 g/day (2000 Kcals). Traditional Indian diets that include whole grains along with whole pulses like grams, soy, green leafy vegetables and some fruits is the recommendation. Fruits like papaya, guava, apples, pears, oranges, mosambi can be taken in moderation. All fruit juices are best avoided.

(b) Proteins:

Proteins should provide 12-15 % of the total energy intake for people with diabetes – similar to the recommendations for the general population. Proteins from vegetable sources like pulses, soy, grams, peas, low fat milk, low fat curds, fish and lean meats are recommended.

Supplementation of foods like cereal and pulse (4:1 ratio) can improve the protein quality and also gives satiety. For e.g; Idli, dosa, Missi roti, Khichdi, Dhokla, Khandvi etc.

(c) Fats:

Fats should provide 20-30 % of total energy intake for people with diabetes. Evidence is inconclusive for an ideal amount of total fat intake for people with diabetes, therefore, goals should be individualized. Fat quality is as important as the quantity.

Fat quality:

- Saturated fats (SFA) ≤10% energy and 7% in those with raised blood lipid levels
- Polyunsaturated fats (PUFA) 10 % energy, n6: 3-7% energy, n3: >1% energy, n6/n3 ratio 5-10
- Monounsaturated Fatty Acids (MUFA) 10-15% energy + any calories left from the carbohydrate portion
- Trans fats < 1% energy – preferably totally avoided in people with type 2 diabetes, MUFA-rich cooking oil and nuts in moderation may benefit glycemic control and CVD risk factors. This can therefore be recommended as an effective alternative to a lower-fat, higher-carbohydrate eating pattern.Use of MUFA rich oils like mustard, rice bran, peanut (groundnut) and gingelly are good options. Oils rich in n6 PUFA like safflower, sunflower, cotton seed, should be mixed with oils rich in n3 like soy and mustard to maintain N6:N3 ratio between 5-10. Use of mixed oils or alternating of oils is recommended.
- (iii) Salt:Sodium intake recommendations for people with diabetes are the same as that for the general population. Added (iodized) salt should be less than 5 g/day. For persons with hypertension and diabetes, the intake should be reduced to less than 3 g/ day. In hypertensive patients or edematous patients with nephropathy, sodium restriction is required. All preserved and processed foods such as pickles, chutneys, packaged namkeens/savouries, sauces should be restricted.

(iv) Alcohol:

It is best to avoid alcohol, however if used, should be taken in moderation. If alcohol is consumed, it should not be counted as part of the meal plan. However, it should be borne in mind that alcohol does provide calories (7 kcal/ g), which are considered as ―empty calories‖. In the fasting state, alcohol may produce hypoglycaemia. Alcohol can further exacerbate fatty liver, neuropathy, dyslipidaemia, obesity and also worsen blood glucose levels.

Sweeteners:

Nutritive Sweeteners: These include fructose, honey, corn syrup, molasses, fruit juice or fruit juice concentrates dextrose, maltose, mannitol, sorbitol and xylitol. All these are best avoided.

Non-nutritive Sweeteners: Aspartame, acesulfame K, stevia, sucralose and saccharin are currently approved for use. However, they should be used in moderation and are best avoided in pregnancy.

Dietary modifications in the presence of complications of diabetes:

(i) Nephropathy:

(a) Protein: The recommended protein intake for diabetic nephropathy patients is 0.6 g/kg of the ideal body weight plus 24 hour urinary protein loss, if this is significant. However, it is recommended that the protein intake should not be less than 40 g/ day. For patients with increased creatinine, protein restriction should be advised in consultation with the nephrologist.

(b) Sodium: It could vary from 1000 mg to 2000 mg/day depending upon the fl uid status and serum sodium levels.

(c) Potassium: Potassium restriction may be required depending upon the potassium values in the blood and type of diuretic being used.

(ii) Cardiovascular Disease:

Maintaining an optimal body weight and restricting salt. Use of fruits and vegetables should be encouraged, with good quality fats in moderation.

(b) **Dyslipidaemia:** Saturated and trans fats food sources like vanaspati, butter, ghee, margarine, coconut oil, red meats like sausages, ham, bacon, egg yellow, whole milk and its products should be restricted. Use of healthy oils and fi bre rich foods is recommended. Vegetarians can take fl ax

seeds (10 g / day) in their diet as both fi sh and flax seeds are rich in omega-3 fatty acids which is protective for heart disease. Alcohol restriction will bring down the triglycerides. Dietary management should be accompanied with regular physical activity and exercise regimen.

Special situations requiring dietary modification: (i) Sick Days in the event of fever or other illness, the diabetic diet should be modified by changing the consistency and texture of foods to maintain adequate calorie intake. Semi solid foods and fluids or items like thin soups, milk, buttermilk, or fresh lime juice should be encouraged.

Lifestyle Management:

(i) Tobacco:

Smoking and tobacco chewing is totally prohibited

(ii) Stress:

Stress management is essential which could take the form of meditation, yoga, a long outdoor walk, exercise and trying out hobbies like reading, gardening, painting etc. Practice of yoga is our traditional Indian system, which has therapeutic value in controlling our physical and mental health. It should be done under the guidance of an expert.

(iii) Physical Activity and Exercise:

Regular physical activity along with regulated exercise is an essential component of management of type 2 diabetes. Complete evaluation of patients with diabetes should be performed before recommending an exercise program. The exercise programme has to be individualized according to one‘s ability and individual capacity.

Benefits of exercise:

- Improves insulin sensitivity
- reduces the risk of heart disease, high blood pressure, bone diseases, and unhealthy weight gain
- Keeps one flexible and agile
- Helps relieve stress, anxiety and prevents depression
- Increases strength and stamina

- Promotes sound sleep
- Increases metabolic rate and digestion
- Delays the process of aging
- Recommendation is about 150 minutes of aerobic activity or its equivalent /week along with some resistance training at least twice a week and flexibility exercises. People with diabetes need an extra quick acting carbohydrate snack before the exercise and during the exercise, if the exercise period extends the daily-recommended routine.

Diabetes education

Diabetes education means empowering people with diabetes with knowledge and providing tools crucial for making them active partners in the diabetes management team. These include:

- In-depth information about diabetes, its complications and treatment
- Appropriate self care skills
- Appropriate resources for self care
- A positive attitude
- Self monitoring skills

  The compliance of people with diabetes is essential for effective management of diabetes. Education programmes are intended to help people to understand why these actions are so important and thereby increase their motivation for self-management.

Table 2. Targets for Glycemic Control in Diabetes*

Blood sugar	Good	Satisfactory	Poor
FBS (mg/dl)	80 – 110	111 – 125	>125
PPBS (mg/dl)	120 – 140	141 – 180	>180
HbA1C	<6	6 – 7	>7

**ICMR guidelines (2018)*

PHARMACOTHERAPY

Insulin Therapy:

- **Insulin Administration**: Follow prescribed insulin regimen carefully, including timing, dosage, and injection technique.
- **Types of Insulin**: Depending on individual needs, use short-acting, long-acting, or intermediate-acting insulin as prescribed.
- **Blood Sugar Monitoring**: Regularly monitor blood sugar levels to adjust insulin dosage accordingly.

Oral Antidiabetic Agents:

- **Medication Adherence**: Take oral antidiabetic medications as prescribed by healthcare providers.
- **Types of Oral Agents**: Depending on the type and severity of diabetes, oral medications such as metformin, sulfonylureas, or thiazolidinediones may be prescribed.

Injectable Glucagon-Like Peptide-1 (GLP-1) Receptor Agonists:

- **Injection Administration**: Follow healthcare provider's instructions for administering GLP-1 receptor agonists.
- **Benefits**: These medications help regulate blood sugar levels, promote weight loss, and reduce cardiovascular risk.

Monitoring and Self-Care

Blood Glucose Monitoring:

- **Regular Testing**: Monitor blood glucose levels as directed by healthcare providers, especially before and after meals and physical activity.
- **Log Keeping**: Maintain a record of blood sugar readings to track patterns and trends over time.

Hemoglobin A1c Monitoring:

- **Long-term Control**: Hemoglobin A1c provides a measure of average blood sugar levels over the past two to three months. Regular monitoring helps assess long-term diabetes management.

Complication Screening:

- **Regular Check-ups**: Undergo regular screenings for diabetes-related complications, including eye exams, foot exams, kidney function tests, and cardiovascular assessments.

- **Early Detection**: Early detection and management of complications are crucial for preventing or minimizing their impact on health.

By integrating these lifestyle modifications, pharmacotherapy, and monitoring/self-care practices into daily life, individuals with diabetes can effectively manage their condition and reduce the risk of complications. Regular communication with healthcare providers is essential for personalized diabetes management plans and adjustments as needed.

Prevention of Diabetes:

1. **Healthy Lifestyle**: Encouraging individuals to adopt a healthy lifestyle is crucial for preventing type 2 diabetes. This includes maintaining a balanced diet rich in fruits, vegetables, whole grains, and lean proteins, along with regular physical activity.
2. **Weight Management**: Obesity is a significant risk factor for type 2 diabetes. Promoting weight loss through healthy eating and exercise can reduce the risk of developing the disease.
3. **Screening and Early Detection**: Identifying individuals at risk for diabetes through screening programs can facilitate early intervention and prevent the progression to full-blown diabetes. Implementing regular screening programs for individuals at high risk of developing diabetes, such as those with a family history or obesity, can facilitate early detection and intervention. Organizing community-based screening programs in collaboration with healthcare providers and community organizations can increase access to screening services and reach underserved populations.
4. **Clinical Assessment**: Conducting routine clinical assessments, including blood glucose testing and monitoring, can help identify individuals with prediabetes or early-stage diabetes, allowing for timely intervention to prevent progression to full-blown diabetes.
5. **Education and Awareness**: Increasing awareness about the risk factors for diabetes and the importance of preventive measures can empower individuals to make healthier choices and seek appropriate medical care.
6. **Community Interventions**: Implementing community-based programs that promote healthy behaviors, such as healthy eating and regular exercise, can help prevent diabetes on a larger scale.

Research and Innovation:

Advances in Treatment Modalities:

1. **Drug Development**: Continued research into new medications and therapies for diabetes treatment, including insulin analogs, incretin-based therapies, and SGLT2 inhibitors, can improve outcomes for individuals with diabetes.
2. **Gene Therapy**: Investigating gene therapy approaches for diabetes, such as gene editing techniques and stem cell therapies, holds promise for developing novel treatments that target the underlying mechanisms of the disease.

Emerging Technologies and Therapies:

1. **Artificial Pancreas Systems**: Advancements in artificial pancreas technology, which combines continuous glucose monitoring with automated insulin delivery, can provide more precise and personalized management of blood glucose levels.
2. **Implantable Devices**: Research into implantable devices, such as glucose-responsive insulin delivery systems and bioengineered pancreatic tissues, may offer innovative solutions for diabetes management and treatment.
3. **Mobile Health Initiatives**: Leveraging mobile health technologies, such as smartphone apps and telemedicine, for diabetes screening can enhance accessibility and convenience, particularly in remote or rural areas.
4. **Artificial Intelligence (AI)**: AI algorithms can analyze large datasets to identify patterns and predict individuals at risk for diabetes, enabling early intervention and preventive measures.
5. **Technology Integration**: Integration of technology, such as wearable devices and mobile health applications, can facilitate continuous monitoring of glucose levels and lifestyle behaviors, enabling more personalized and real-time interventions for diabetes prevention and management.

Personalized Medicine Approaches:

1. **Precision Medicine**: Integrating genomic data and biomarkers into diabetes management algorithms can enable personalized prevention and treatment approaches tailored to individual patients' genetic profiles and disease characteristics.
2. **Machine Learning and AI**: Leveraging machine learning and artificial intelligence algorithms to analyze large datasets can identify patterns and predict treatment responses, facilitating personalized treatment recommendations for individuals with diabetes.
3. **Behavioral Interventions**: Further research into behavioral interventions, such as cognitive-behavioral therapy and motivational interviewing, can help address the psychological and social factors influencing diabetes prevention and management.
4. **Policy and Public Health Initiatives**: Implementing policies and public health initiatives that promote healthy environments, such as access to nutritious foods and opportunities for physical activity, can support population-wide efforts to prevent diabetes.

By focusing on primary prevention strategies, secondary prevention through early detection and intervention, and ongoing research and innovation, we can work towards reducing the burden of diabetes and improving outcomes for individuals affected by the disease.

In conclusion, diabetes remains a significant health challenge globally, with its prevalence steadily rising. This chronic condition not only affects individuals' quality of life but also imposes a substantial economic burden on healthcare systems worldwide. Effective management of diabetes requires a multifaceted approach, including lifestyle modifications, medication adherence, and regular monitoring of blood glucose levels. Patient education and empowerment play crucial roles in achieving optimal outcomes, emphasizing the importance of self-care practices and early detection of complications. Additionally, ongoing research into novel treatments, technological advancements such as continuous glucose monitoring systems and insulin pumps, and efforts to address social determinants of health offer promising avenues for improving diabetes care and outcomes in the future. By fostering collaboration among healthcare professionals, policymakers, and communities, we can work towards preventing new cases, enhancing access to care, and ultimately reducing the global impact of diabetes.

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KIDNEY DISEASES

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INTRODUCTION

The structural and functional unit of the kidney, the 'nephron,' consists of a renal corpuscle (glomerulus surrounded by a Bowman capsule) and a renal tubule. Each kidney in an adult human contains around 1 million nephrons. A fenestrated endothelium forms the inner glomerular layer, followed by a layer composed of various extracellular proteins forming a meshwork called the glomerular basement membrane (GBM). The outer layer has visceral epithelial cells, podocytes, and mesangial cells. The intricate arrangement provides the basis for continuous plasma volume filtration at the glomerular level.

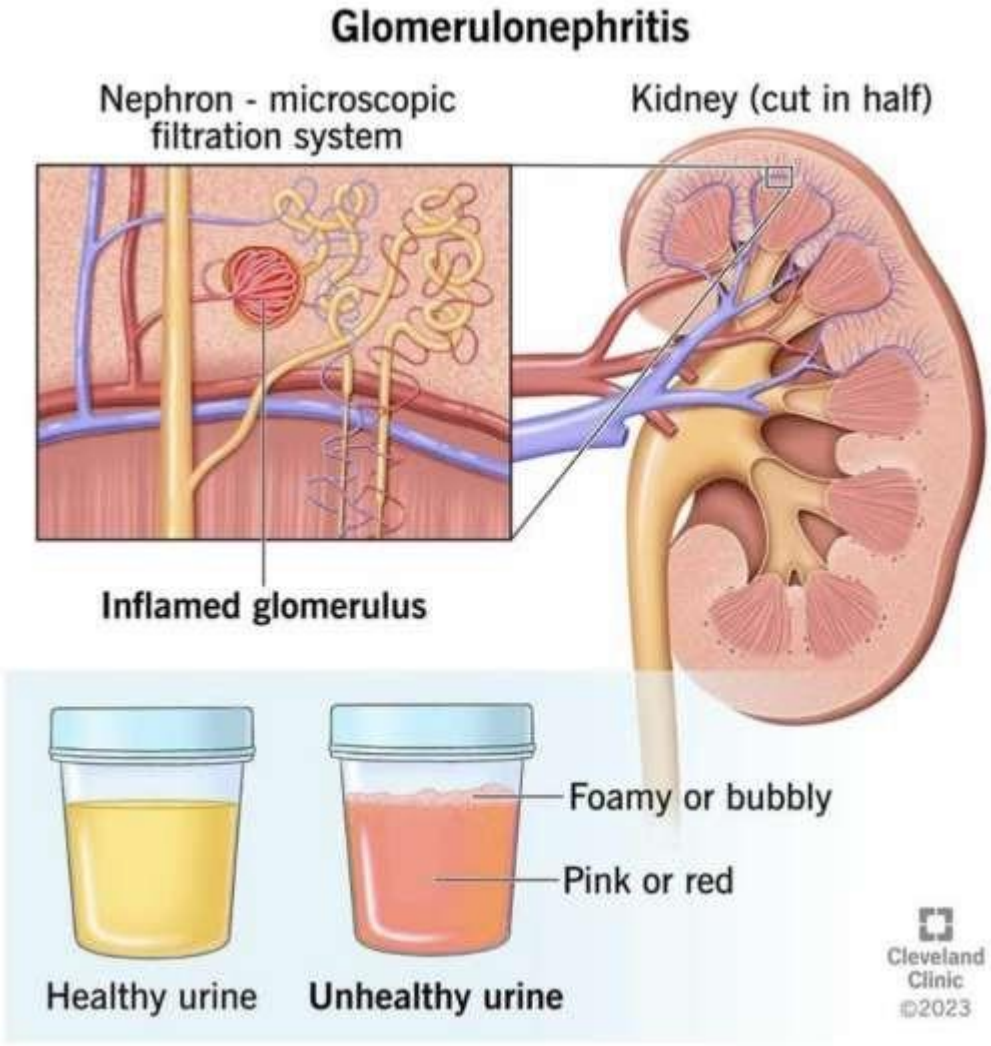
The term "glomerulonephritis" encompasses a subset of renal diseases characterized by immune-mediated damage to the basement membrane, the mesangium, or the capillary endothelium, resulting in hematuria, proteinuria, and azotemia.

Acute forms of glomerulonephritis (GN) can result from either a primary renal cause or a secondary illness that causes renal manifestations. For instance, acute post-streptococcal glomerulonephritis (PSGN) is a typical example of acute glomerulonephritis secondary to a streptococcal infection; similarly, *Staphylococcus aureus* infection can also lead to glomerulonephritis. In recent times, however, the incidence of glomerulonephritis associated with staphylococcal has increased as opposed to the reduction of PSGN in the United States and most developed countries.

Most forms of glomerulonephritis are considered progressive disorders. Without timely therapy, progress to chronic glomerulonephritis (characterized by progressive glomerular damage and tubulointerstitial fibrosis leading to a reduced glomerular filtration rate). This leads to the retention of uremic toxins with subsequent progression into chronic kidney disease (CKD) and end-stage renal disease (ESRD) along with their associated cardiovascular diseases.

Glomerulonephritis is inflammation of the tiny filters in the kidneys (glomeruli). The excess fluid and waste that glomeruli remove from the bloodstream exit the body as urine. Glomerulonephritis can come on suddenly (acute) or gradually (chronic).

Glomerulonephritis occurs on its own or as part of another disease, such as lupus or diabetes. Severe or prolonged inflammation associated with glomerulonephritis can damage the kidneys.



ETIOLOGY

Etiological classification of glomerulonephritis can be made based on clinical presentation, ranging from severe proteinuria (>3.5 g/day) and edema qualifying for the nephrotic syndrome to a nephritic syndrome where haematuria and hypertension are more prominent while proteinuria is less pronounced.

Nephrotic Glomerulonephritis

Minimal change disease

Focal segmental glomerulosclerosis

Membranoproliferative glomerulonephritis

Membranous nephropathy

HIV associated nephropathy

Diabetic nephropathy

Amyloidosis

Nephritic Glomerulonephritis

IgA nephropathy

Henoch Schonlein purpura (HSP)

Post streptococcal glomerulonephritis.

Anti-glomerular basement membrane disease

Rapidly progressive glomerulonephritis

Granulomatosis with polyangiitis

Eosinophilic granulomatosis with polyangiitis

Polyarteritis nodosa

Idiopathic crescentic glomerulonephritis

Goodpasture syndrome

Lupus nephritis

Hepatitis C infection

Membranoproliferative glomerulonephritis (typical presentation is with acute nephritic syndrome, however, sometimes features resembling nephrotic syndrome may occur, additionally)

A more modern and widely accepted way to classify glomerulonephritis is to divide it into five forms of glomerulonephritis based on underlying immune processes. The following is the modern classification of glomerulonephritis, including pathogenic type and the disease entity associated with it:

Immune-complex GN - IgA nephropathy, IgA vasculitis, infection-related GN, lupus nephritis, and fibrillary GN with polyclonal Ig deposits

Pauci-immune GN - PR3-ANCA GN, MPO-ANCA GN, and ANCA-negative GN

Anti-glomerular basement membrane (GBM) GN - Anti-GBM GN

Monoclonal Ig GN - Proliferative GN with monoclonal Ig deposits, monoclonal Ig deposition disease, fibrillary GN with monoclonal Ig deposits, and immunotactoid glomerulopathy

C3 glomerulopathy - C3 glomerulonephritis, dense deposit disease

PATHOPHYSIOLOGY

The underlying pathogenetic mechanism common to all of these different varieties of glomerulonephritis (GN) is immune-mediated, in which both humoral as well as cell-mediated pathways are active. The consequent inflammatory response, in many cases, paves the way for fibrotic events that follow.

The targets of immune-mediated damage vary according to the type of GN. For instance, glomerulonephritis associated with staphylococcus shows IgA and C3 complement deposits.[3]

One of the targets is the glomerular basement membrane itself or some antigen trapped within it, as in post-streptococcal disease.[16] Such antigen-antibody reactions can be systemic, with

glomerulonephritis occurring as one of the components of the disease process, such as in systemic lupus erythematosus (SLE) or IgA nephropathy. On the other hand, in small vessel vasculitis, cell-mediated immune reactions are the main culprit instead of antigen-antibody reactions. Here, T lymphocytes and macrophages flood the glomeruli with resultant damage.

These initiating events activate common inflammatory pathways, i.e., the complement system and coagulation cascade. The generation of pro-inflammatory cytokines and complement products, in turn, results in the proliferation of glomerular cells. Cytokines such as platelet-derived growth factor (PDGF) are also released, ultimately causing glomerulosclerosis. This event is seen in those situations where the antigen is present for longer periods, for example, in hepatitis C viral infection. When the antigen is rapidly cleared, as in post-streptococcal GN, the resolution of inflammation is more likely.

Structural Changes

Structurally, cellular proliferation causes an increase in the cellularity of the glomerular tuft due to the excess of endothelial, mesangial, and epithelial cells. The proliferation may be of two types:

Endocapillary - within the glomerular capillary tufts

Extracapillary - in the Bowman space, including the epithelial cells

In extra-capillary proliferation, parietal epithelial cells proliferate to cause the formation of crescents, characteristic of some forms of rapidly progressive glomerulonephritis.

Thickening of glomerular basement membrane appears as thickened capillary walls on light microscopy. However, on electron microscopy, this may look like a consequence of the thickening of the basement membrane proper, for instance, diabetes or electron-dense deposits either on the epithelial or endothelial side of the basement membrane. There can be various types of electron-dense deposits corresponding to an area of immune complex deposition, such as subendothelial, subepithelial, intramembranous, and mesangial. (See the images below)

Features of irreversible injury include hyalinization or sclerosis that can be focal, diffuse, segmental, or global.

Functional Changes

Functional changes include the following:

Proteinuria

Haematuria

Reduction in creatinine clearance, oliguria, or anuria

Active urine sediments, such as RBCs and RBC casts

This leads to intravascular volume expansion, edema, and systemic hypertension.

SYMPTOMS

Signs and symptoms of glomerulonephritis may vary depending on whether you have the acute or chronic form and the cause. You may notice no symptoms of chronic disease. Your first indication that something is wrong might come from the results of a routine urine test (urinalysis).

Glomerulonephritis signs and symptoms may include:

Pink or cola-coloured urine from red blood cells in your urine (haematuria).

Foamy or bubbly urine due to excess protein in the urine (proteinuria).

High blood pressure (hypertension).

Fluid retention (edema) with swelling evident in your face, hands, feet and abdomen.

Urinating less than usual.

Nausea and vomiting.

Muscle cramps.

Fatigue.

CAUSES

Many conditions can cause glomerulonephritis. Sometimes the disease runs in families and sometimes the cause is unknown. Factors that can lead to inflammation of the glomeruli include the following conditions.

Infections

Infectious diseases can directly or indirectly lead to glomerulonephritis. These infections include:

Post-streptococcal glomerulonephritis. Glomerulonephritis may develop a week or two after recovery from a strep throat infection or, rarely, a skin infection caused by a streptococcal bacterium (impetigo). Inflammation occurs when antibodies to the bacteria build up in the glomeruli. Children are more likely to develop post-streptococcal glomerulonephritis than are adults, and they're also more likely to recover quickly.

Bacterial endocarditis. Bacterial endocarditis is an infection of the inner lining of your heart's chambers and valves. It isn't clear whether the inflammation in the kidneys is the result of immune system activity alone or other factors.

Viral kidney infections. Viral infections of the kidney, such as hepatitis B and hepatitis C, cause inflammation of the glomeruli and other kidney tissues.

HIV. Infection with HIV, the virus that causes AIDS, can lead to glomerulonephritis and progressive kidney damage, even before the onset of AIDS.

Autoimmune diseases

Autoimmune diseases are illnesses caused by the immune system attacking healthy tissues. Autoimmune diseases that may cause glomerulonephritis include:

Lupus. A chronic inflammatory disease, systemic lupus erythematosus can affect many parts of your body, including your skin, joints, kidneys, blood cells, heart and lungs.

Goodpasture's syndrome. In this rare disorder, also known as anti-GBM disease, the immune system creates antibodies to tissues in the lungs and kidneys. It can cause progressive and permanent damage to the kidneys.

IgA nephropathy. Immunoglobulin A (IgA) is an antibody that's a first line of defense against infectious agents. IgA nephropathy occurs when deposits of the antibody accumulate in the glomeruli. The inflammation and subsequent damage may go undetected for a long time. The most common symptom is blood in the urine.

Vasculitis

Vasculitis is inflammation of blood vessels. Types of vasculitis that can cause glomerulonephritis include:

Polyarteritis. This form of vasculitis affects medium and small blood vessels in many parts of your body, including the kidneys, skin, muscles, joints and digestive tract.

Granulomatosis with polyangiitis. This form of vasculitis, formerly known as Wegener's granulomatosis, affects small and medium blood vessels in your lungs, upper airways and kidneys.

Sclerotic conditions

Some diseases or conditions cause scarring of the glomeruli that results in poor and declining kidney function. These include:

High blood pressure. Long-term, poorly managed high blood pressure can cause scarring and inflammation of the glomeruli. Glomerulonephritis inhibits the kidney's role in regulating blood pressure.

Diabetic kidney disease (diabetic nephropathy). High blood sugar levels contribute to scarring of the glomeruli and increase the rate of blood flow through the nephrons.

Focal segmental glomerulosclerosis. In this condition, scarring is scattered among some of the glomeruli. This may be the result of another disease, or it may occur for no known reason.

Other causes

Infrequently, chronic glomerulonephritis runs in families. One inherited form, Alport syndrome, also might impair hearing or vision.

Glomerulonephritis is associated with certain cancers, such as gastric cancer, lung cancer and chronic lymphocytic leukaemia.

COMPLICATIONS

Glomerulonephritis affects the ability of nephrons to filter the bloodstream efficiently. The breakdown in filtering results in:

Accumulation of wastes or toxins in the bloodstream.

Poor regulation of essential minerals and nutrients.

Loss of red blood cells.

Loss of blood proteins.

Possible complications of glomerulonephritis include:

Acute kidney failure. Acute kidney failure is the sudden, rapid decline in kidney function, often associated with an infectious cause of glomerulonephritis. The accumulation of waste and fluids can be life-threatening if not treated promptly with an artificial filtering machine (dialysis). The kidneys often resume typical function after recovery.

Chronic kidney disease. Persistent inflammation results in long-term damage and declining function of the kidneys. Chronic kidney disease is generally defined as kidney damage or decreased function for three or more months. Chronic kidney disease may advance to end-stage kidney disease, which requires either dialysis or a kidney transplant.

High blood pressure. Damage to the glomeruli from inflammation or scarring can lead to increased blood pressure.

Nephrotic syndrome. Nephrotic syndrome is a condition in which there is too much blood protein in urine and too little in the bloodstream. These proteins play a role in regulating fluids

and cholesterol levels. A drop in blood proteins results in high cholesterol, high blood pressure and swelling (edema) of the face, hands, feet and abdomen. In rare instances, nephrotic syndrome may cause a blood clot in a kidney blood vessel.

DIAGNOSIS

Blood

Complete blood count - A decreased haematocrit may suggest a dilutional type of anemia. In the background of an infectious cause, pleocytosis may be apparent.

Serum electrolytes - Potassium levels may be raised in patients with severe renal impairment.

Renal function tests - BUN and creatinine levels are raised, demonstrating a degree of renal impairment. In addition, the glomerular filtration rate (GFR) may be low.

Liver function tests - May point towards the underlying etiology.

Immunoglobulins

C-reactive protein (CRP)

Electrophoresis

Complement (C3, C4 levels) - Differentiation may allow the provider to narrow the differentials. Low complement levels indicate the following diseases: cryoglobulinemia, systemic lupus erythematosus, infective (bacterial) endocarditis, and shunt nephritis. Certain renal disorders may also be considered, such as membranoproliferative GN or post-streptococcal GN. Normal complement levels suggest an underlying abscess, polyarteritis nodosa, Henoch-Schönlein purpura, Goodpasture syndrome, idiopathic rapidly progressive GN, immune complex disease, and immunoglobulin G or immunoglobulin A nephropathy. Chauvet et al. reported that in patients with new-onset nephritis and low C3 levels, anti-factor B autoantibodies might help distinguish new-onset post-streptococcal GN from hypocomplementemic C3 glomerulonephritis.

Autoantibodies, such as antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA), and anti-ds-DNA, anti-glomerular basement membrane (GBM) to rule out collagenopathy as the underlying cause of GN.

Blood culture - Blood culture is indicated when there is a fever, immunosuppression, intravenous drug abuse, indwelling catheters, or shunts.

Ant streptolysin O titer (ASOT) increases in 60 to 80% of cases. The rise begins in one to three weeks, peaks in three to five weeks, and returns to baseline in six months. It is unrelated to the severity, duration, and prognosis of renal disease.

Hepatitis serology - As infectious hepatitis can lead to glomerulonephritis of various types

Urine

The urine is usually dark, and the specific gravity is more than 1.020 with RBCs and RBC casts. The 24-hour urinary protein excretion and creatinine clearance may help establish the degree of renal impairment and proteinuria. The following parameters are usually helpful:

Microscopy, culture, and sensitivity

Bence Jones protein

Albumin to creatinine or protein to creatinine ratio

RBC casts

Imaging

Chest X-ray (helps to see for evidence of pulmonary hemorrhage, if any)

Renal ultrasound (helps in assessing the size and anatomy for biopsy)

Renal Biopsy

The examination of glomerular lesions via a renal biopsy provides the diagnosis of glomerulonephritis by answering the following queries:

Approximate proportion of involved glomeruli (focal vs. diffuse)

Approximate involvement of each glomerulus (segmental vs. global)

Presence of hypercellularity

Any evident sclerosis

Any deposits on immunohistology (immunoglobulins, light chains, complement)

Electron microscopy findings - precise localization of deposits. Exact ultrastructural appearance. Podocyte appearance

Presence of tubulointerstitial inflammation, atrophy, or fibrosis

Evident vessel-related pathology

DIETARY RECOMMENDATIONS

1. Control Protein Intake

Consuming moderate amounts of high-quality protein is important, but excessive protein can strain the kidneys. The recommended amount varies depending on the individual but typically ranges between 0.6 and 0.8 grams of protein per kilogram of body weight. Thus, good sources of high-quality protein include lean meats, poultry, fish, eggs, dairy products, and plant-based sources like legumes and tofu.

2. Limit Sodium (Salt) Intake

Excess sodium can lead to fluid retention and high blood pressure, which can worsen kidney function. But to control your BP you can follow high blood pressure diet plan. Always aim to consume less than 2300 milligrams of sodium per day.

Also, try not to include processed and packaged foods, as they tend to be high in sodium. Instead, choose fresh, whole foods and season meals with herbs, spices, and lemon juice to enhance flavor.

3. Monitor Fluid Intake

In some cases of glomerulonephritis, fluid intake may need to be restricted to manage swelling or fluid buildup. However, your healthcare provider will guide you on the appropriate amount of fluid to consume daily.

4. Control Blood Pressure

High blood pressure can further damage the kidneys. Also, follow a low-sodium diet, maintain a healthy weight, limit alcohol consumption, and engage in regular physical activity to help manage blood pressure.

5. Manage Potassium and Phosphorus Intake

Depending on the severity of glomerulonephritis and kidney function, it may be necessary to restrict potassium and phosphorus intake. Foods high in potassium include bananas, oranges, tomatoes, potatoes, and dairy products. Thus, foods high in phosphorus include dairy products, legumes, nuts, and whole grains. Your healthcare provider will advise you on the appropriate levels for your specific condition.

6. Consume A Balanced Diet

Focus on consuming a variety of fruits, vegetables, whole grains, and healthy fats. These foods provide essential vitamins, minerals, and antioxidants that support overall health.

7. Consider Dietary Modifications

Depending on individual circumstances, additional dietary modifications may be recommended, such as limiting oxalate-rich foods (e.g., spinach, rhubarb) or reducing the intake of certain types of fats. Your healthcare provider or dietitian can provide guidance based on your specific needs.

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CANCER

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INTRODUCTION

Cancer is the disease that we hear about the most and is seen in every part of the world and after cardiovascular disease, cancer is the leading cause of death in world. The public and patients often wonder, ―When will a complete cure for cancer be available‖? The answer to this simple question is very difficult, because cancer is not a single disease, but rather a combination of numerous disorders involving severe cellular deregulations. Unlike some primitive animals, all species including humans are affected by cancer. Cancer is undoubtedly a serious and life-threatening disease, but more often than not it is exaggerated and generalized. Once a cell become cancerous, it divides until a mass of cells forms a tumor. Several diagnostic tests can quickly distinguish malignant or cancerous tumors from harmless ones. As a malignant tumor progresses, cells or groups of cells break off and spread or metastasize throughout the body through the lymphatic system and blood vessels. The only hope for controlling cancer is to learn more about its causes, pathogenesis, and understand cellular physiology at the molecular level. Cancer is always a complex and multifaceted disease that has a significant impact on individuals on worldwide. It is crucial to understand the underlying mechanisms and risk factors associated with cancer in order to develop effective prevention and treatment strategies.

What is Cancer?

Cancer is a group of diseases characterized by uncontrolled growth of abnormal cells in body. These cells can invade and destroy normal tissues, leading to serious health complications. The development of cancer is influenced by a combination of genetic, environmental, and lifestyle factors.

―Cancer may be defined as a complex disease, which is usually a combination of cellular abnormalities developed by mutation of particular gene (Ras protein is muted in many human cancers) or production and function of abnormal proteins‖. (Bisoyi Padmini, 2022). In all major types of cancer, some of the body cells undergo uncontrollable division and spread to surrounding tissues. In other words, we can say that cancer is simply the future of cell cycle control or disease of cell division.

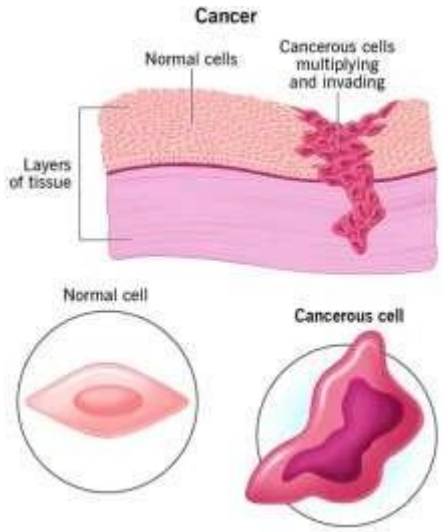


Fig. 1: Normal cell and Cancerous cell

(Source:https://my.clevelandclininc.org/health/cancer)

CLASSIFICATION AND TYPES OF CANCER

Cancer is a complex and multifaceted disease that can affect various parts of the body. There are several ways to classify and categorize cancer, including by the type of tissue or organ where it originates, its behavior, and its genetic makeup. Here are some common classifications and types of cancer:

1. By Tissue or Organ of Origin:

Carcinomas: Cancer that begins in the skin or tissues that line or cover internal organs, such as the lung, breast, prostate, and colon.

Sarcomas: Cancer that develops in the bone, cartilage, fat, muscle, blood vessels, or other connective or supportive tissues.

Leukemias: Cancer that starts in blood-forming tissue, such as the bone marrow, leading to the overproduction of abnormal blood cells.

Lymphomas: Cancer that begins in the immune system cells, specifically in the lymph nodes and tissues of the body's immune system.

Central Nervous System Cancers: Cancer that originates in the tissues of the brain and spinal cord.

2. By Behavior:

Benign Tumors: These tumors are non-cancerous and do not spread to other parts of the body. They are generally localized and have a lower risk of causing serious problems.

Malignant Tumors: These tumors are cancerous and have the ability to invade nearby tissues and spread to other parts of the body, a process known as metastasis.

3. By Genetic Makeup:

Some cancers are classified based on their genetic characteristics, such as specific mutations or gene expression patterns. For example, breast cancer can be categorized into different subtypes based on the presence or absence of certain hormone receptors (e.g., estrogen receptor-positive, HER2-positive).

4. By Staging:

Cancer staging is a way of describing the extent of cancer within the body. It takes into account the size of the primary tumor, whether it has spread to nearby lymph nodes, and whether it has metastasized to distant organs.

5. By Common Types:

Some common types of cancer include breast cancer, lung cancer, prostate cancer, colorectal cancer, skin cancer (melanoma), and ovarian cancer, among others.

Understanding the classification and types of cancer is important for diagnosis, treatment planning, and prognosis. Each type of cancer may require different approaches to treatment, and advancements in personalized medicine are increasingly tailoring therapies to the specific characteristics of a patient's cancer. It's crucial to note that cancer is a constantly evolving field of research, and new discoveries about the molecular and genetic underpinnings of cancer are continually shaping our understanding of the disease. This ongoing research is leading to more targeted and effective treatments for different types of cancer, offering hope for improved outcomes for patients.

Types of tumors:

There are two main types of tumor

1. Benign tumor

2. Malignant tumor or cancer

Benign tumor

A benign tumor is a type of tumor in which the microscopic and macroscopic characteristic appears relatively harmless, this implies that it cannot spread to other sites and will not remain localized. The general development of capsule mass is separated from normal tissue. This capsule is just a rim of compressed connective tissue, derived mainly from the extracellular matrix (ECM) of the host tissue. Usually the patient can be removed surgically and may survive. Benign tumors are also called non-cancerous tumors. However, such tumors can sometimes produce more localized lumps, which may lead to serious diseases.

1.1 Some common types of benign tumors:

In general, the suffix-oma is used to designate a benign tumor. Depending on the origin of cells, there are different types of benign tumors. Such as:

Fibroma or fibroids- Fibroma are benign tumors of mesenchymal origin. These tumors form in the fibrous tissues of the body. For example, uterine fibroids. Such fibroids are noncancerous but can cause pelvic pain, vaginal bleeding, or bladder problems.

Adenomas these are benign tumors of epithelial origin, occur in glands or glandular structures. For example, colorectal adenomas (development of tubular or polyp like structure in the colon).

Papilloma these are warty or finger-like projections that arise from epithelial surfaces. Note: Most of the benign tumors are non-cancerous; however, some of them if not treated earlier can become malignant.

2. Malignant tumor

A tumor is said to be malignant when its microscopic and gross characteristics are considered Aggressive. Such types of tumors can infiltrate, invade, and destroy neighbouring cells and tissues or can spread (metastasize) to distant sites of the body. Malignant tumors are commonly known as Cancer. Generally, malignant tumors are fatal and can cause most cancer death. However, not all cancers are deadly if they are discovered early and are treated successfully.

2.1 Some common types of malignant tumors:

The nomenclature of a malignant tumor is quite the same as a benign tumor, with certain additions. Some of the most common malignant tumors are:

Carcinomas-These are a malignant tumor of epithelial origin. Such tumors are formed in the epithelial cells of the body. For example, hepatocellular carcinoma of the liver, invasive ductal carcinoma (IDC) of the breast, adenocarcinoma of the stomach. Sarcoma these are a malignant tumor of mesenchymal origin. Such tumors are formed in the epithelial connective tissue of the body. For example, osteosarcoma of bones. (Bisoyi Padmini, 2022).

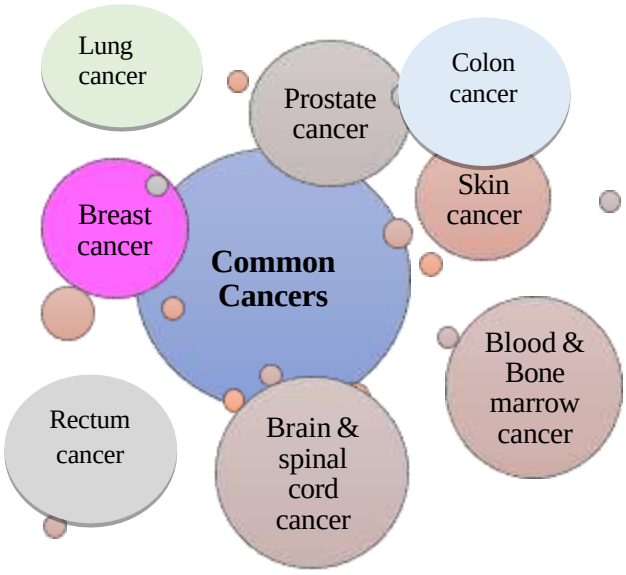


Fig. 2: Common Cancers

RISK FACTORS OF CANCER

Risk factors for cancer can be divided into two main categories:

1. Lifestyle and Behavioral Factors:

These include factors such as smoking, alcohol consumption, poor diet, lack of physical activity, and exposure to harmful chemicals and pollutants.

2. Genetic and Environmental Factors:

These encompass genetic mutations, family history of cancer, exposure to radiation and ultraviolet (UV) light, certain viral infections, and hormonal imbalances.

Lifestyle and Behavioral Factors:

Smoking: Tobacco smoke contains over 7,000 chemicals, many of which are carcinogenic. Smoking is a leading cause of lung, throat, mouth, and bladder cancer.

Alcohol Consumption: Excessive alcohol consumption can increase the risk of developing cancers of the mouth, throat, esophagus, liver, and breast.

Poor Diet: A diet high in processed foods, red meat, and low in fruits and vegetables can contribute to an increased risk of cancer.

Lack of Physical Activity: Sedentary lifestyles are associated with a higher risk of certain cancers, including breast and colon cancer.

Exposure to Harmful Chemicals and Pollutants: Occupational exposure to certain chemicals and pollutants, such as asbestos, benzene, and formaldehyde, can elevate the risk of developing cancer.

Genetic and Environmental Factors:

Genetic Mutations: Inherited genetic mutations, such as BRCA1 and BRCA2 mutations can significantly increase the risk of breast, ovarian, and other cancers.

Family History of Cancer: Individuals with close relatives who have had cancer may have a higher risk of developing the disease themselves.

Exposure to Radiation and UV Light: Prolonged exposure to ionizing radiation (e.g., from medical imaging) and UV light (from the sun or tanning beds) can lead to skin and other types of cancer.

Viral Infections: Certain viruses, such as human papillomavirus (HPV), hepatitis B and C viruses, and Epstein-Barr virus, are linked to an increased risk of specific cancers.

Hormonal Imbalances: Hormonal factors, such as early onset of menstruation, late onset of menopause, and hormone replacement therapy, can influence the risk of breast and endometrial cancer.

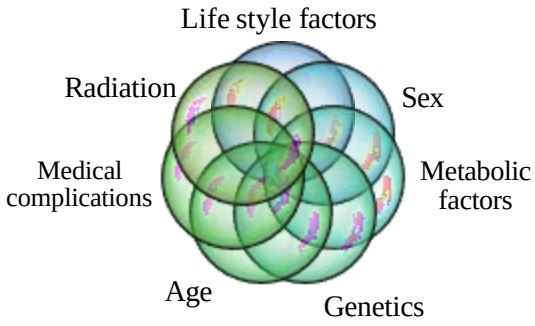


Fig. 3: Risk Factors of Cancer

It's important to note that while these factors can increase the likelihood of developing cancer, they do not guarantee that an individual will develop the disease. Additionally, many cancers arise from a combination of factors, and not all individuals with risk factors will develop cancer. Regular screenings, healthy lifestyle choices, and genetic counseling can play a significant role in cancer prevention and early detection.

PATHOPHYSIOLOGY OF CANCER

The pathophysiology of cancer involves multiple stages and processes that contribute to the development and progression of the disease.

1. Initiation: The process of cancer initiation involves the genetic or epigenetic alterations in normal cells, leading to the transformation of these cells into cancerous cells. These alterations can be caused by various factors such as exposure to carcinogens, genetic mutations, or viral infections.

2. Promotion: Following initiation, the promotion stage involves the proliferation of the initiated cells due to the influence of promoting agents. These agents can include hormones, growth factors, and inflammatory mediators that stimulate the growth and division of cancer cells.

3. Progression: Cancer progression is characterized by the further evolution of cancer cells, leading to the development of invasive and metastatic properties. This stage involves genetic instability, angiogenesis (formation of new blood vessels), and the ability of cancer cells to evade the immune system.

4. Metastasis: Metastasis is the spread of cancer cells from the primary tumor to other parts of the body. This process involves the invasion of cancer cells into blood vessels or lymphatic channels, followed by their transport to distant sites where they can establish secondary tumors.

5. Tumor Microenvironment: The tumor microenvironment plays a crucial role in cancer pathophysiology, as it consists of various components such as stromal cells, immune cells, and extracellular matrix that interact with cancer cells to promote their survival, growth, and invasion.

6. Genomic Instability: Genetic instability is a hallmark of cancer pathophysiology, leading to the accumulation of genetic mutations and alterations that drive the progression of the disease. This instability can be caused by defects in DNA repair mechanisms and exposure to genotoxic agents.

7. Immune Evasion: Cancer cells have the ability to evade the immune system through various mechanisms, such as down regulation of immune recognition molecules, secretion of immunosuppressive factors, and induction of immune tolerance, allowing them to escape immune surveillance and continue growing.

Understanding the pathophysiology of cancer is essential for the development of effective treatment strategies. Targeting the specific molecular and cellular processes involved in cancer pathophysiology has led to the development of targeted therapies, immunotherapies, and personalized medicine approaches that aim to improve patient outcomes.

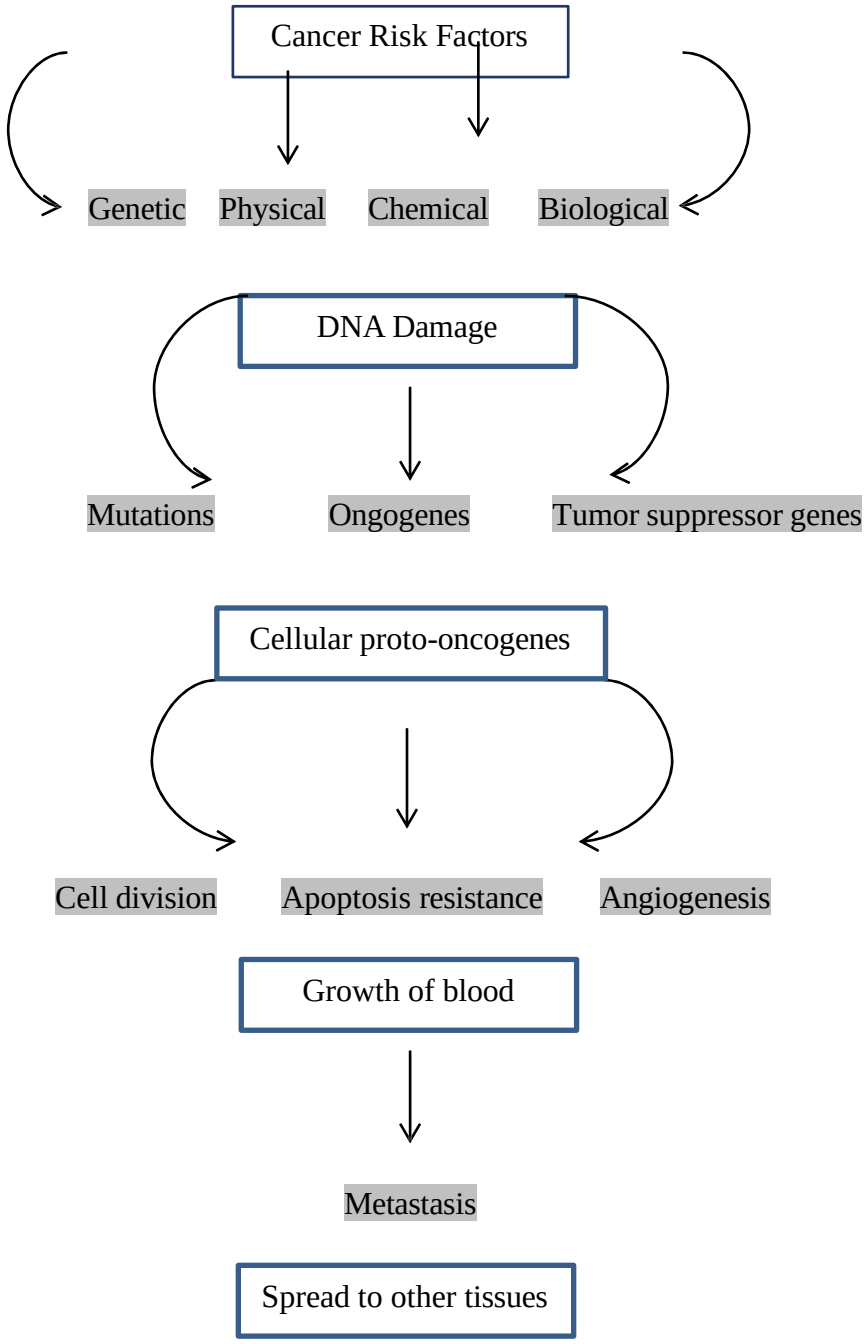


Fig. 4: Pathophysiology of cancer

This flow chart (Fig.4) illustrates the pathophysiology of cancer. The risk factors for cancer, such as genetic, physical, chemical, and biological factors, can lead to DNA damage. This damage can

result in mutations and alterations in cellular proto-oncogenes, oncogenes, and tumor suppressor genes.

These genetic changes can lead to abnormal cell growth, resistance to apoptosis (cell death), and the promotion of angiogenesis (the formation of new blood vessels). The growth of abnormal cells can then lead to the development of a tumor. Furthermore, cancer cells can acquire the ability to metastasize, spreading from the primary tumor to other tissues in the body. This process involves complex interactions between cancer cells and the surrounding microenvironment, ultimately leading to the establishment of secondary tumors in distant organs.

SYMPTOMS OF CANCER

Cancer can present a wide range of symptoms, and they can vary depending on the type of cancer and where it is located in the body. Some common symptoms of cancer can include:

1. Unexplained weight loss

2. Fatigue

3. Persistent cough or hoarseness

4. Changes in bowel or bladder habits

5. Persistent lumps or swollen glands

6. Difficulty swallowing

7. Persistent indigestion or discomfort after eating

8. Changes in mole or wart

9. Unexplained bleeding or bruising

10. Persistent pain

It's important to note that these symptoms can also be caused by conditions other than cancer. However, if you are experiencing any of these symptoms, it's crucial to seek medical attention promptly for a proper diagnosis and appropriate treatment. Early detection and treatment can significantly improve the prognosis for many types of cancer.

Table 1: Symptoms of common cancers

Breast cancer	Blood cancer	Lung cancer	Brain cancer	Skin cancer	Oral cancer
A breast lump or thickening, often without pain. Change in size, shape or appearance of the breast. Dimpling, redness, pitting or other changes in the skin. Change in nipple appearance or the skin surrounding the nipple (areola). Abnormal or bloody fluid from the nipple.	Weight loss that is unexplained. Bruising or bleeding that is unexplained. Lumps or swellings. Shortness of breath (breathlessness) Drenching night sweats. Infections that is persistent, recurrent or severe. Fever (38°C or above) that is unexplained. Rash or itchy skin that is unexplained.	Coughing that gets worse or doesn‘t go away. Chest pain. Shortness of breath. Wheezing. Coughing up blood. Feeling very tired all the time. Weight loss with no known cause.	Headaches that may be more severe in the morning or wake you up at night. Seizures. Difficulty thinking, speaking or understanding language. Personality changes. Weakness or paralysis in one part or one side of your body. Balance problems or dizziness. Vision issues.	A new spot on the skin or. Changes in the size, shape or color of an existing spot. A spot that is itchy or painful. A non-healing sore that bleeds or develops a crust. A red- or skin-colored shiny bump on the top of the skin. A red rough or scaly spot that you can feel.	A lump in your neck. Loose teeth. Swelling or a sore on your lip that won't heal. Difficult or painful swallowing. Changes in speech. Bleeding or numbness in the mouth. White or red patches on the mouth tongue or gums. Unexplained weight loss.

COMPLICATIONS OF CANCER

Complications of cancer can arise due to the disease itself or as a result of cancer treatments. Some common complications include:

1. Pain: Cancer can cause pain due to tumor growth, nerve compression, or treatment side effects. Managing pain is an important aspect of cancer care and can involve medications, physical therapy, or interventional procedures.

2. Fatigue: Many cancer patients experience extreme tiredness and lack of energy, which can be caused by the cancer itself, anemia, or the side effects of treatments such as chemotherapy and radiation therapy.

3. Infection: Cancer and its treatments can weaken the immune system, making patients more susceptible to infections. This can be particularly concerning for individuals undergoing intensive treatments or those with blood-related cancers.

4. Cachexia: This is a wasting syndrome characterized by weight loss, muscle atrophy, fatigue, weakness, and loss of appetite. It can significantly impact a patient's quality of life and prognosis.

5. Psychological and emotional effects: Cancer diagnosis and treatment can lead to anxiety, depression, and other mental health challenges. It's important for patients to have access to psychological support and counseling services.

6. Cognitive dysfunction: Some cancer patients experience "chemo brain" or cancer-related cognitive impairment, which can manifest as problems with memory, attention, and processing speed.

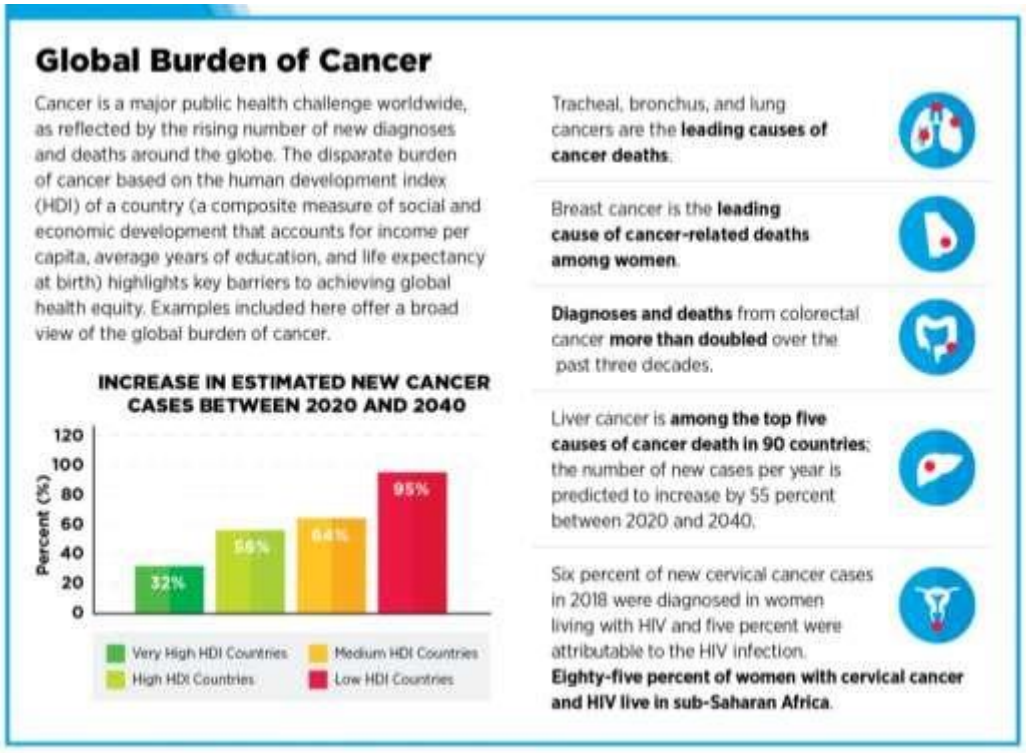
7. Lymphedema: This is a swelling that occurs in the arms or legs due to a blockage in the lymphatic system, often as a result of surgery or radiation therapy for breast or gynecologic cancers.

8. Cardiovascular complications: Certain cancer treatments, such as some chemotherapy drugs and radiation therapy to the chest area, can increase the risk of cardiovascular issues including heart failure, arrhythmias, and coronary artery disease.

9. Bone health issues: Some cancers can spread to the bones, leading to complications such as fractures, bone pain, and hypercalcemia. Additionally, certain cancer treatments can weaken the bones and increase the risk of osteoporosis.

10. Gastrointestinal complications: Cancer in the digestive system can lead to issues such as difficulty swallowing, bowel obstructions, malabsorption, and nutritional deficiencies.

It's important for individuals with cancer to receive comprehensive care that addresses not only the primary disease but also its potential complications. This may involve a multidisciplinary approach with oncologists, pain specialists, nutritionists, physical therapists, mental health professionals, and other healthcare providers working together to support the patient's well-being.



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DIAGNOSIS OF CANCER

Diagnosing cancer involves several methods, and the specific approach depends on the type of cancer suspected. Here are some common diagnostic techniques:

1. Physical Examination: Doctors may conduct a physical examination to check for any unusual lumps or growths.

2. Imaging Tests: Techniques such as X-rays, CT scans, MRIs, and ultrasounds allow doctors to visualize the internal structures of the body and identify any abnormal growths or tumors.

3. Biopsy: This involves removing a sample of tissue from the suspected tumor or abnormal growth. The tissue is then examined under a microscope to check for the presence of cancer cells.

4. Blood Tests: Certain cancers can be detected through blood tests that look for specific markers or substances indicative of cancer.

5. Genetic Testing: In some cases, genetic testing may be used to identify specific genetic mutations that predispose individuals to certain types of cancer.

6. Endoscopy: This involves using a thin, flexible tube with a camera and light at the end to examine the interior of certain organs or tissues, such as the digestive tract.

7. Diagnostic Surgery: Sometimes, surgery may be necessary to obtain a tissue sample for further examination.

Fig. 5: Test to Diagnose Cancer (Source:www.everydayhealth.com/cancer)

It's important to note that the specific diagnostic process will vary depending on the type of cancer suspected and the individual patient's circumstances. Recent years have seen a remarkable progress in the basic, translational and clinical research in cancers. From recent research, molecular markers can assist in early and accurate diagnosis and predict prognosis in Cancers.

Emerging Trends and Technologies in Cancer Diagnosis:

Cancer diagnosis is an area of constant innovation and advancement, with new trends and technologies reshaping the landscape of detection and treatment. As we look to the future, it's clear that several key trends are emerging that have the potential to revolutionize the way we diagnose and treat cancer.

Liquid Biopsies: A Game-Changer in Cancer Diagnosis:

One of the most significant trends in cancer diagnosis is the rise of liquid biopsies. These non-invasive tests analyze blood samples for fragments of tumor DNA, offering a wealth of information about the presence and progression of cancer. Liquid biopsies have the potential to detect cancer at an early stage, monitor treatment effectiveness, and identify the emergence of resistance to therapy. This technology represents a paradigm shift in cancer diagnosis, offering a less invasive and more accessible alternative to traditional tissue biopsies.

Artificial Intelligence and Machine Learning:

Artificial intelligence (AI) and machine learning are rapidly transforming the field of cancer diagnosis. These technologies can analyze vast amounts of medical data to identify patterns and trends that are beyond the scope of human capability. AI algorithms can assist in the interpretation of medical images, such as mammograms and CT scans, leading to more accurate and efficient cancer detection. Furthermore, machine learning models can help predict the risk of cancer development based on individual patient data, enabling personalized and proactive interventions.

Precision Medicine: Tailoring Treatment to Individual Patients:

Another trend shaping the future of cancer diagnosis is the concept of precision medicine. This approach takes into account individual variations in genes, environment, and lifestyle to

customize treatment strategies. By analyzing the genetic makeup of a patient's tumor, healthcare providers can identify targeted therapies that are more likely to be effective and less toxic than traditional treatments. Precision medicine holds the promise of improving patient outcomes and reducing the burden of unnecessary treatments and their associated side effects.

Integration of Multi-Omics Data:

The integration of multi-omics data, including genomics, proteomics, and metabolomics, is becoming increasingly important in cancer diagnosis. By combining information from multiple biological layers, researchers can gain a more comprehensive understanding of the molecular mechanisms driving cancer development and progression. This holistic approach allows for the identification of novel biomarkers and therapeutic targets, as well as the development of more accurate diagnostic tests.

Advancements in Imaging Technologies

In the realm of cancer diagnosis, advancements in imaging technologies are enhancing our ability to visualize and characterize tumors. From high-resolution MRI and PET scans to emerging techniques such as optical coherence tomography (OCT) and photo acoustic imaging, these tools enable earlier and more precise detection of cancerous lesions. Furthermore, the development of molecular imaging agents that specifically target cancer cells is facilitating the visualization of tumors at the molecular level, aiding in diagnosis and treatment planning.

The Role of Wearable Devices and Digital Health Platforms:

Wearable devices and digital health platforms are playing an increasingly important role in cancer diagnosis and monitoring. These technologies allow for continuous and remote tracking of vital signs, biomarkers, and other health-related data, providing a wealth of information that can aid in early detection and personalized treatment. Moreover, the integration of wearable devices with AI algorithms holds the potential to create real-time diagnostic tools that are accessible to a broad population, transcending geographical and socioeconomic barriers.

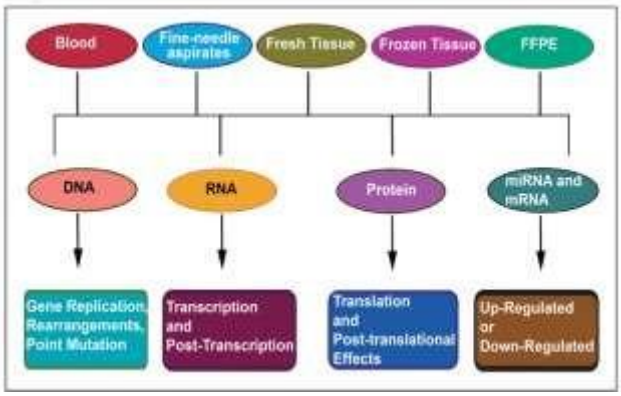

Fig. 6: Molecular Alterations for Cancer Diagnosis (Source: mdpi.com)

MANAGEMENT OF CANCER

Cancer management involves a multi-faceted approach that includes various treatment options and supportive care. The management of cancer depends on the type and stage of the cancer, as well as the overall health and preferences of the patient.

Treatment Options: -

The cancer treatment process can vary depending on the type and stage of cancer, as well as the specific treatment plan recommended by the medical team. However, here‘s a general outline of the cancer treatment process:

Diagnosis:

The first step in managing cancer is an accurate diagnosis. This involves a combination of physical examination, imaging tests (such as X-rays, CT scans, MRI scans), blood tests, and sometimes a biopsy to confirm the presence of cancer and determine its type and stage. Once the type and stage of cancer are determined, the treatment plan can be developed.

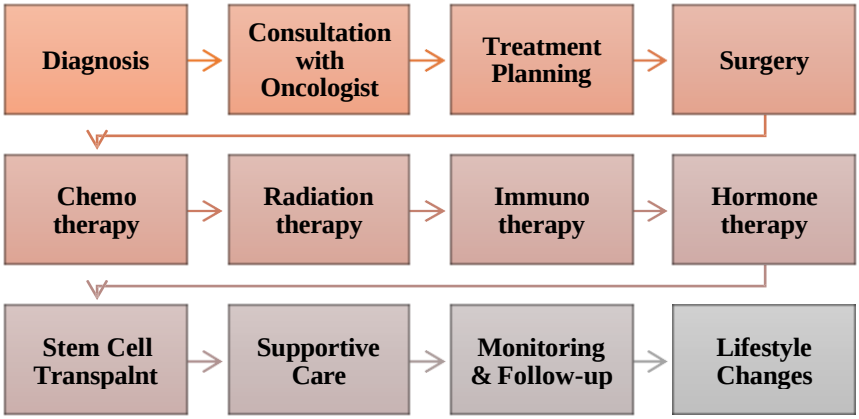

Fig.7: Treatment Process of Cancer

Consultation with Oncologist:

After diagnosis, the patient will meet an oncologist who specializes in cancer treatment. The oncologist will review the diagnosis and discuss treatment options based on the type and stage of cancer.

Treatment Planning:

The oncologist, along with other members of the medical team, will develop a personalized treatment plan for the patient. This plan may include one or a combination of treatment modalities such as surgery, chemotherapy, radiation, immunotherapy, targeted therapy, hormone therapy, or stem cell transplant.

Surgery:

Surgery is often used to remove the cancerous tumor and surrounding tissue. It is most effective for solid tumors that are localized and have not spread to other parts of the body. The extent of the surgery will depend on the size and location of the tumor.

Chemotherapy:

Chemotherapy involves the use of drugs to kill cancer cells or stop them from growing. It can be administered orally or intravenously and is often used in combination with other treatments.

Radiation Therapy:

Radiation therapy uses high-energy rays to kill cancer cells. It can be delivered externally using a machine, or internally through the placement of radioactive materials near the cancer cells.

Immunotherapy and Targeted therapy:

Immunotherapy works by helping the body's immune system recognize and attack cancer cells. It can be used to treat various types of cancer and is often less toxic than traditional treatments. Targeted therapy uses drugs or other substances to identify and attack specific cancer cells without harming normal cells. It is often used in conjunction with other treatments.

Hormone Therapy:

Hormone therapy is used to treat cancers that are hormone-sensitive, such as breast and prostate cancer. It works by blocking the effects of certain hormones or by lowering their levels in the body.

Stem Cell Transplant:

Stem cell transplant, also known as bone marrow transplant, may be used to replace damaged bone marrow with healthy stem cells. It is often used to treat blood cancers such as leukemia and lymphoma.

Supportive Care:

In addition to the specific cancer treatments, supportive care is essential for managing the symptoms and side effects of cancer and its treatment. This may include pain management, nutritional support, psychological support, and rehabilitation services.

Palliative Care

Palliative care focuses on providing relief from the symptoms and stress of a serious illness such as cancer. It is aimed at improving the quality of life for both the patient and their family.

Monitoring and Follow-Up:

After initial treatment, regular monitoring and follow-up are crucial to track the progress of the disease, manage any potential side effects, and address any recurrence of cancer.

Clinical Trials

Participation in clinical trials may be an option for some patients, offering access to new and innovative treatments that are not yet widely available.

Lifestyle Changes:

Patients are often advised to make lifestyle changes such as quitting smoking, improving diet, and increasing physical activity to support their overall health and well-being during and after cancer treatment.

The management of cancer is complex and requires a personalized approach that takes into account the specific characteristics of the disease and the individual patient. It often involves a multidisciplinary team of healthcare professionals working together to provide the best possible care for the patient.

DIET AND CANCER

Diet is a fundamental component of human health, and its influence on cancer risk has garnered significant attention in scientific research. According to the World Cancer Research Fund, approximately 30-35% of cancer cases are linked to dietary factors, highlighting the substantial impact of diet on cancer risk. Several key dietary elements have been identified as influential factors in cancer development, including the consumption of processed meats, high intake of red meat, low intake of fruits and vegetables, and excessive alcohol consumption.

Processed Meats and Red Meat Consumption

Processed meats, such as bacon, sausages, and deli meats, have been associated with an increased risk of colorectal cancer. These products often contain additives and preservatives, such as nitrates and nitrites, which can form carcinogenic compounds in the body. Similarly, a high intake of red meat has been linked to an elevated risk of developing colorectal, pancreatic,

and prostate cancers. The iron and saturated fats present in red meat are thought to contribute to cancer development through various biological mechanisms.

Table 2: Relationship of dietary factors with risk of major cancers

Diet	*Colorectal*	*Breast*	*Prostate*	*Lung*	*Stomach*	*Esophageal*	*Oral*	*Pancreatic*	*Bladder*	*Kidney*	*Endometrial*
Macronutrients/energy balance											
Obesity	↑↑	↑↑			↑[b]	↑[b]		↑		↑	↑↑
GI/GL[c], IGF, height or metabolic syndrome	↑↑	↑	↑					↑		↑	↑
Animal fat			↑								↑
Foods											
Red or processed meat	↑		↑								
Fruits[d]	↓		↓	↓	↓	↓	↓				↓
Vegetables[d]	↓	↓	↓		↓	↓	↓		↓		↓
Nutrients											
Folic acid	↓↓	↓		↓							
Alcohol	↑↑	↑↑				↑↑	↑				
Calcium	↓		↑								
Vitamin D	↓		↓								
β-Carotene *supplements*				↑↑[e]	↓						
Lycopene-containing *foods*			↓	↓	↓						
Vitamin C				↓							
Vitamin E			↓								
Selenium			↓	↓							
Other											
Grilling meat	↑	↑									
Western diet pattern	↑										
High fiber diet	↓										
Salt, preserved foods					↑						
Hot beverages						↑	↑				

[a]Two arrows indicate more consistent evidence. [b]Cancers of the gastric cardia. [c]GI = glycemic index; GL = glycemic load. [d]Evidence for a potential benefit from some components of fruits and vegetables (not necessarily blanket effect). [e]Increased risk limited to smokers

(Source: Marjorie L McCullough *et.al*, Diet and Cancer prevention, 2020)

Fruits, Vegetables, and Cancer Prevention

Conversely, the consumption of fruits and vegetables has been consistently associated with a reduced risk of several types of cancer. These plant-based foods are rich in vitamins, minerals, antioxidants, and dietary fibers, which exert protective effects against cancer development. The phytochemicals present in fruits and vegetables, such as flavonoids and carotenoids, have been shown to inhibit carcinogenesis and suppress tumor growth, highlighting the importance of a diverse and plant-focused diet in cancer prevention.

Alcohol Consumption and Cancer Risk

Excessive alcohol consumption is another dietary factor that has been linked to an increased risk of developing various types of cancer, including liver, breast, and oesophageal cancer. Ethanol, the primary component of alcoholic beverages, is metabolized into acetaldehyde, a known carcinogen that can induce DNA damage and promote tumor formation. Additionally, chronic alcohol consumption can contribute to nutritional deficiencies and impaired immune function, further exacerbating cancer risk.

The link between diet and cancer is a complex and intricate relationship that underscores the significance of dietary choices in cancer prevention and management. While genetic and environmental factors also play pivotal roles in cancer development, the impact of diet should not be underestimated. By adopting a balanced and healthful diet that emphasizes the consumption of fruits, vegetables, and lean protein sources while minimizing the intake of processed and red meats, individuals can proactively reduce their risk of cancer. Furthermore, promoting public awareness of the connection between diet and cancer is essential in fostering informed dietary choices and mitigating the global burden of cancer.

In light of the substantial evidence supporting the influence of diet on cancer risk, it is imperative for individuals, healthcare professionals, and policymakers to prioritize the implementation of comprehensive dietary strategies aimed at reducing the incidence of cancer on a global scale.

Fig. 8: Best anti-cancer foods (Source:http://medium.com/@ppc)

Diet plan for Cancer patient:

A diet plan for cancer should focus on providing the body with essential nutrients, supporting the immune system, and promoting overall health. It's important to note that a healthy diet alone cannot cure cancer, but it can play a supportive role in managing symptoms and side effects of treatment. Here's a general guideline for a cancer-friendly diet:

1. Fruits and Vegetables: Include a variety of colorful fruits and vegetables in your diet as they are rich in vitamins, minerals, and antioxidants. Aim for at least 5 servings per day.

2. Whole Grains: Choose whole grains such as brown rice, quinoa, whole wheat bread, and oats. These foods provide fiber, which is important for digestive health.

3. Protein: Opt for lean sources of protein such as poultry, fish, beans, lentils, and tofu. Protein is essential for tissue repair and immune function.

4. Healthy Fats: Incorporate healthy fats from sources like avocados, nuts, seeds, and olive oil. These fats contain anti-inflammatory properties and support heart health.

5. Hydration: Drink plenty of water to stay hydrated. Dehydration can worsen side effects of treatment and impact overall well-being.

6. Limit Processed Foods: Minimize intake of processed and red meats, sugary snacks, and refined carbohydrates. These foods can contribute to inflammation and may not provide optimal nutrition.

7. Moderate Alcohol: If you drink alcohol, do so in moderation. For cancer patients, it's generally recommended to limit alcohol consumption.

8. Individualized Needs: Every person's nutritional needs are different, especially during cancer treatment. Consult with a registered dietitian who specializes in oncology to create a personalized plan tailored to your specific needs and treatment regimen.

9. Consider Supplements: Some cancer patients may benefit from certain supplements, such as vitamin D or omega-3 fatty acids. However, it's important to discuss supplement use with your

healthcare team to ensure safety and effectiveness, as certain supplements can interfere with treatment.

10. Manage Side Effects: If you're experiencing side effects such as nausea, taste changes, or difficulty swallowing, work with a dietitian to find ways to manage these symptoms through dietary modifications.

It's crucial to remember that a well-balanced diet is just one aspect of cancer care. Always consult with your healthcare provider and oncologist before making any significant changes to your diet, especially during cancer treatment.

Table 3: A Sample meal plan for Cancer patient

	BREAKFAST	LUNCH	SNACK	DINNER
MONDAY	Millet upma	Jowhar roti with mixed vegetable curry	Apple slices with unsweetened peanut butter	Kadhi with steamed rice
TUESDAY	Moong dal dosa (pesarattu) with sambar	Black bean curry with brown rice	Banana smoothie	Ragi wheat phulka with dal banjara
WEDNESDAY	Theplas with methi (fenugreek)	Bajra roti with drumstick curry	Steamed beetroot salad	Kala chana pakora rolls
THURSDAY	Cauliflower parathas with curd	Wheat roti with lentils	Unsalted popcorn	Bottle gourd with mixed millet phulkas
FRIDAY	Oats rava idli	Khichdi with raita	Corn Salad	Carrot thepla with green chutney
SATURDAY	Poha with peanuts	Bamboo rice with moong dal	Freshly made vegetable soup	Mixed vegetable jowhar upma
SUNDAY	Palak puris with peas curry	Sagu masala dosa	Sprouts salad	Cabbage and tomato subzi, with phulkas

(Source: https://onco.com/blog/indian-meal-plan -for-cancer-fighters/amp/)

Lifestyle Changes and Cancer Risk Reduction:

In addition to dietary modification, other lifestyle factors, including physical activity, alcohol consumption, and tobacco use, play a crucial role in cancer risk reduction. Engaging in regular physical activity has been shown to reduce the risk of several types of cancer, including breast, colon, and endometrial cancer. Physical activity not only helps in weight management and hormonal balance but also contributes to enhancing the body's immune function, thereby reducing the risk of cancer development.

Furthermore, limiting alcohol consumption and avoiding tobacco use are integral components of a cancer-preventive lifestyle. Excessive alcohol consumption has been linked to an increased risk of liver, breast, and colorectal cancer, while tobacco use is a well-established risk factor for numerous cancers, including lung, mouth, and throat cancer.

In conclusion, lifestyle changes, particularly dietary modifications, can exert a significant impact on cancer risk reduction. Embracing a diet rich in whole foods, engaging in regular physical activity, and avoiding tobacco and excessive alcohol consumption are essential components of a cancer-preventive lifestyle. While genetic predisposition plays a role in cancer development, the power to influence one's cancer risk through lifestyle choices should not be underestimated. By adopting healthy dietary and lifestyle habits, individuals can empower themselves in reducing their susceptibility to cancer and promoting overall well-being.

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HYPERTHYROIDISM

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INTRODUCTION

The thyroid gland is a butterfly-shaped gland located in the lower front of the neck. The job of the thyroid gland is to make thyroid hormones. Thyroid hormones help the body use energy, stay warm and keep the brain, heart, muscles, and other organs working.

Hyperthyroidism is a common thyroid disorder. "Hyperthyroidism" defines a syndrome associated with excess thyroid hormone production. It is a common misconception that the terms thyrotoxicosis and hyperthyroidism are synonyms. The term "thyrotoxicosis" refers to a state of excess thyroid hormone exposure to tissues. Although hyperthyroidism can lead to thyrotoxicosis and can be used interchangeably. Hyperthyroidism causes many of the body‘s functions to speed up. Hyperthyroidism can influence multiple organ systems, involving the cardiovascular, nervous, gastrointestinal, and hepatic systems.

The most common cause of hyperthyroidism is Graves‘ disease, followed by toxic nodular goitre. Other important causes of thyrotoxicosis include thyroiditis, iodine-induced and drug-induced thyroid dysfunction, and factitious ingestion of excess thyroid hormones. Treatment options for Graves‘ disease include antithyroid drugs, radioactive iodine therapy, and surgery, whereas antithyroid drugs are not generally used long term in toxic nodular goitre, because of the high relapse rate of thyrotoxicosis after discontinuation. β blockers are used in symptomatic thyrotoxicosis, and might be the only treatment needed for thyrotoxicosis not caused by excessive production and release of the thyroid hormones.

The global prevalence of hyperthyroidism in iodine- sufficient countries is estimated at 0.2% to 2.5%.1 The prevalence of overt hyperthyroidism, defined as low thyrotropin (previously thyroid stimulating hormone) levels with elevated triiodothyronine (T3) and/or free thyroxine (FT4), is approximately 0.2% to 1.4%. The prevalence of subclinical hyperthyroidism, defined as low

thyrotropin levels with normal peripheral thyroid hormone levels, is approximately 0.7% to 1.4%.

CLASSIFICATION OR TYPES OF HYPERTHYROIDISM

According to the origin of the abnormality

Hyperthyroidism is classified according to the origin of the lesion into primary, secondary, and tertiary hyperthyroidism.

Primary hyperthyroidism

Grave's disease

Toxic thyroid nodule

Thyroid adenoma

Multinodular goiter

Secondary hyperthyroidism

Pituitary adenoma

Intracranial tumors pressing pituitary gland

Tertiary hyperthyroidism

Intracranial tumors or masses involving hypothalamus

According to iodine uptake

Hyperthyroidism can be classified according to the results of iodine uptake test into high uptake, high or normal uptake, and low uptake.

High uptake

Grave‘s disease

Toxic multinodular goiter

Toxic thyroid adenoma

High or normal uptake

Iodine caused hyperthyroidism

Hashitoxicosis

Germ cell tumors (choriocarcinoma in males and testicular germ cell tumors)

Pituitary TSH producing adenoma

Low uptake

Subacute thyroiditis

Hyperthyroidism due to ectopic thyroid tissue

Factitious thyrotoxicosis

Struma ovaria

Painless thyroiditis

Amiodarone induced thyroiditis-Type 1

Amiodarone induced thyroiditis-Type 2

RISK FACTORS OF HYPERTHYROIDISM

Risk factors for hyperthyroidism include:

• A family history of thyroid disease, particularly Graves‘ disease.

• A personal history of certain chronic illnesses, including pernicious anemia and primary adrenal insufficiency.

• Being female

• Having a personal or family history of autoimmune disease (for example, rheumatoid arthritis, lupus or celiac disease)

• Having a personal or family history of thyroid disease, including thyroid nodules

• Being recently pregnant

• Smoking

• Taking iodine intake or an iodine-containing medication (for example, amiodarone)

• Experiencing trauma to the thyroid gland

• Being deficient in Vitamin D and selenium

• Experiencing psychological stress (for example, divorce or loss of a partner)

Clinical manifestations of hyperthyroidism

Clinical manifestations of hyperthyroidism are largely independent of its cause. However, the disorder that causes hyperthyroidism may have other effects. In particular, Graves' disease, the most common cause of hyperthyroidism, causes unique problems that are not related to the high serum thyroid hormone concentrations. These include Graves' orbitopathy and infiltrative dermopathy (localized or pretibial myxoedema). Most patients with Graves' hyperthyroidism have a diffuse goitre, but so do patients with other, less common causes of hyperthyroidism such as painless thyroiditis and thyroid-stimulating hormone (TSH)-secreting pituitary tumors.

CLASSIC SYMPTOMS

Most patients with overt hyperthyroidism have a dramatic constellation of symptoms. The classic symptoms of hyperthyroidism include heat intolerance, tremor, palpitations, anxiety, weight loss despite a normal or increased appetite, increased frequency of bowel movements, and shortness of breath. Goiter is commonly found on physical examination. Patients with mild hyperthyroidism and older patients often have symptoms that are preferable to one or only a few organ systems.

Specific organ systems

Skin

The skin is warm (and may rarely be erythematous) in hyperthyroidism due to increased blood flow; it is also smooth because of a decrease in the keratin layer. Other changes include:

- Sweating, which increases due to increased calorigenesis; this is often associated with heat intolerance

- Onycholysis (loosening of the nails from the nail bed, Plummer's nails) and softening of the nails

- Hyperpigmentation, which can occur in severe cases; it appears to be mediated by accelerated cortisol metabolism, leading to increased corticotropin (ACTH) secretion

- Pruritus and hives, which are occasional findings, primarily in patients with Graves' hyperthyroidism

- Vitiligo and alopecia areata, which can occur in association with autoimmune disorders

- Thinning of the hair

Eyes

Stare and lid lag occur in all patients with hyperthyroidism. They are due to sympathetic overactivity, possibly mediated by increased alpha-adrenergic receptors in some tissues. Lid lag is evaluated by having the patient follow the examiner's finger as it is moved up and down. The patient has lid lag if sclera can be seen above the iris as the patient looks downward.

The with Graves' disease have orbitopathy. It is characterized by inflammation of the extraocular muscles and orbital fat and connective tissue, which results in proptosis (exophthalmos), impairment of eye muscle function, and periorbital and conjunctival edema. Risk factors for developing Graves' orbitopathy include cigarette smoking, advancing age, and male sex

Patients with orbitopathy may have gritty feeling or pain in their eyes, and they may have diplopia due to extraocular muscle dysfunction. Corneal ulceration can occur as a result of proptosis and lid retraction, and severe proptosis can cause optic neuropathy and even blindness.

Cardiovascular System

Patients with hyperthyroidism have an increase in cardiac output, due both to increased peripheral oxygen needs and increased cardiac contractility. Heart rate is increased, pulse pressure is widened, and peripheral vascular resistance is decreased. Systolic hypertension is common. The left ventricular ejection fraction does not increase appropriately during exercise, suggesting the presence of a true cardiomyopathy. High- or normal-output congestive heart failure can occur in patients with severe hyperthyroidism, and congestive heart failure worsens in patients who already have it.

Atrial fibrillation occurs in 10 to 20 percent of patients with hyperthyroidism, and it is more common in older patients. In one study, 8 percent of all patients and 15 percent of patients between ages 70 to 79 developed atrial fibrillation within 30 days of the diagnosis of hyperthyroidism. Even subclinical hyperthyroidism is associated with an increased rate of atrial ectopy and a threefold increased risk of atrial fibrillation

In 60 percent of hyperthyroid patients with atrial fibrillation, the rhythm converts spontaneously to sinus rhythm when the hyperthyroidism is treated; in one study, all who spontaneously converted did so within four months after becoming euthyroid. Among those who do not convert spontaneously to sinus rhythm and who undergo successful electrical cardioversion, the two-year risk of recurrent atrial fibrillation was 59 percent compared with 83 percent of patients whose atrial fibrillation was not associated with hyperthyroidism.

Anticoagulation should be considered in hyperthyroid patients with atrial fibrillation. In several studies, 10 to 40 percent of patients with hyperthyroidism and atrial fibrillation had an arterial embolus. Left atrial enlargement, which is a risk factor for thrombus formation, is present in approximately 90 percent of hyperthyroid patients with atrial fibrillation and 2 percent of hyperthyroid patients with sinus rhythm. Based on these results, we usually anticoagulated hyperthyroid patients with atrial fibrillation. The role of anticoagulant therapy in patients with hyperthyroidism and atrial fibrillation is reviewed in detail separately.

Other abnormalities, including mitral valve prolapse, mitral regurgitation, and an increase in left ventricular mass index have also been reported.

Metabolic/Endocrine Bone

Thyroid hormone stimulates bone resorption, resulting in increased porosity of cortical bone and reduced volume of trabecular bone. The loss in cortical bone density is greater than that of trabecular bone. Serum alkaline phosphatase and osteocalcin concentrations are high, indicative of increased bone turnover. The increase in bone resorption may lead to an increase in serum calcium concentrations, thereby inhibiting parathyroid hormone secretion and the conversion of calcidiol (25-hydroxyvitamin D) to calcitriol (1,25-dihydroxyvitamin D). In addition, the metabolic clearance rate of calcitriol is increased. These changes can result in impaired calcium absorption and an increase in urinary calcium excretion. The net effect is osteoporosis and an increased fracture risk in patients with chronic hyperthyroidism

Graves' disease may also be associated with thyroid acropachy, with clubbing and periosteal new bone formation in the metacarpal bones or phalanges. Patients with thyroid acropachy commonly present with asymptomatic clubbing, severe ophthalmopathy, and dermopathy; a high percentage are cigarette smokers

Serum lipids

Patients with hyperthyroidism tend to have low serum total and high-density lipoprotein (HDL) cholesterol concentrations and a low total cholesterol/HDL cholesterol ratio. These values increase after treatment

Hyperglycaemia

Although thyroxine (T4) is not a counter regulatory hormone, hyperthyroidism can interfere with glucose metabolism. It is associated with both increased sensitivity of pancreatic beta cells to glucose, resulting in increased insulin secretion, and antagonism to the peripheral action of insulin. The latter effect usually predominates, leading to impaired glucose tolerance in untreated patients

Adrenal function

Interpretation of the cortisol response to corticotropin (ACTH) stimulation testing may be misleading in patients with hyperthyroidism because corticosteroid-binding globulin (CBG)

levels decrease, resulting in lower total serum cortisol concentrations. In one report of 49 hyperthyroid patients undergoing ACTH testing, 35 percent had subnormal total serum cortisol values (<18 mcg/dL), while only 11 percent had a subnormal free cortisol index (ratio of serum total cortisol to CBG).

Respiratory

Dyspnea and dyspnea on exertion may occur for many reasons in hyperthyroidism

● Oxygen consumption and carbon dioxide production increase. These changes result in hypoxemia and hypercapnia, respectively, both of which stimulate ventilation.

● Respiratory muscle weakness is an important cause of dyspnea, and reduced exercise capacity may be largely due to respiratory muscle weakness and decreased lung volume.

● There may be tracheal obstruction from a large goiter.

● Hyperthyroidism may exacerbate underlying asthma.

● Pulmonary arterial systolic pressure is increased

Gastrointestinal

Weight loss is due primarily to increased metabolic rate (hypermetabolism) and secondarily to increased gut motility and the associated hyper defecation and malabsorption; rare patients have steatorrhea. Celiac disease is also more prevalent in patients with Graves' disease. Most patients have hyperphagia, but an occasional patient with mild hyperthyroidism may have sufficient appetite stimulation that weight is gained (more commonly in younger patients). Anorexia may be prominent in older hyperthyroid patients.

Other changes that may occur include:

● Vomiting and abdominal pain, rarely.

● Dysphagia due to goiter.

● Abnormalities in liver function tests, particularly high serum alkaline phosphatase concentrations and, rarely, cholestasis. In a meta-analysis, alanine transaminase (ALT) and

aspartate transaminase (AST) were elevated in 33 and 23 percent of patients presenting with hyperthyroidism, respectively, while alkaline phosphatase was elevated in 44 percent of patients.

Thymic enlargement

In patients with Graves' disease, thymic enlargement due to hyperplasia has been reported. Thymic enlargement is usually discovered incidentally during the evaluation of other symptoms, such as dyspnea. The pathogenetic mechanism is unclear, but it is not a feature of hyperthyroidism due to causes other than Graves' disease. Potential mechanisms include autoimmune mediated (thyroid-stimulating immunoglobulins from patients with Graves' disease bind to the thyroid-stimulating hormone [TSH] receptor in the human thymus and cause thymocyte proliferation) and hyperthyroidism mediated (hyperthyroidism-induced angiogenesis). Causes of thymic enlargement other than Graves' disease should be considered.

In most patients, treatment of Graves' disease with antithyroid medications, radioiodine, or thyroidectomy results in involution of thymic hyperplasia over 4 to 25 months. However, coexisting malignancies have been reported, and therefore, repeat imaging three to four months after initiation of therapy is warranted to ensure regression.

Hematologic

The red blood cell mass is increased in hyperthyroidism, but the plasma volume is increased more, resulting in a normochromic, normocytic anemia. Serum ferritin concentrations may be high.

Graves' hyperthyroidism may be associated with autoimmune hematologic disorders such as immune thrombocytopenia (ITP) and pernicious anemia, and some patients have antineutrophil antibodies. In a meta-analysis, 10 percent of patients presenting with Graves' hyperthyroidism had neutropenia.

Hyperthyroidism may also be prothrombotic. As an example, in a meta-analysis of 51 studies evaluating the effect of thyroid hormone excess (exogenous or endogenous) on the coagulation system, excess thyroid hormone was associated with a rise in prothrombotic factors, including factors VIII, IX, fibrinogen, von Willebrand factor, and plasminogen activator inhibitor-1. Similar findings were noted in patients with either overt or subclinical hyperthyroidism.

Genitourinary

Urinary frequency and nocturia are common in hyperthyroidism, although the mechanism is uncertain. Possible causes include primary polydipsia and hypercalciuria. Enuresis is common in children.

In women, serum sex hormone-binding globulin (SHBG) concentrations are high, which results in high serum estradiolconcentrations and low-normal serum free (unbound) estradiol concentrations, high serum luteinizing hormone (LH) concentrations, a reduced mid-cycle surge in LH secretion, oligomenorrhea, and anovulatory infertility. Amenorrhea can occur in women with severe hyperthyroidism.

In men, the increase in serum SHBG concentrations results in high serum total testosterone concentrations, but serum free (unbound) testosterone concentrations are normal or low. Serum LH concentrations may be slightly high. Extra gonadal conversion of testosterone to estradiol is increased, so that serum estradiol concentrations are high. These changes can cause gynecomastia, reduced libido, and erectile dysfunction. Spermatogenesis is often decreased or abnormal, e.g., more spermatozoa are abnormal or nonmotile.

Neuropsychiatric

Patients with thyrotoxicosis may experience behavioural and personality changes, such as psychosis, agitation, and depression. Less overt manifestations that are more common in less severe thyrotoxicosis include anxiety, restlessness, irritability, and emotional lability. Insomnia is also common. Symptoms often worsen in patients with pre-existing psychiatric disorders.

These behavioural manifestations are accompanied by cognitive impairments, particularly impaired concentration, confusion, poor orientation and immediate recall, amnesia, and constructional difficulties.

GERIATRIC HYPERTHYROIDISM

Hyperthyroidism in older patients may be apathetic, rather than having hyperactivity, tremor, and other symptoms of sympathetic overactivity. However, two-thirds of such patients have symptoms similar to those in younger patients. In cross-sectional studies of patients with hyperthyroidism, older patients had a reduced risk for the presence of several classical symptoms (ie, heat intolerance, tremor, nervousness) but a higher prevalence of weight loss and shortness of breath compared with younger patients. Older patients also had a higher rate of atrial fibrillation and moderate to severe ophthalmopathy

Older patients with Graves' hyperthyroidism are less likely to have a goiter. Toxic multinodular goiter is more common in older patients, although the majority of hyperthyroid patients at any age have Graves' hyperthyroidism. In addition, older patients often have persistent constipation. Tachycardia of 100 beats per minute is absent in 40 percent of older hyperthyroid patients, due primarily to coexistent conduction system disease.

SYMPTOMS OF HYPERTHYROIDISM

Hyperthyroidism has many symptoms that can vary from person to person. It is suggested by several signs and symptoms; however, patients with mild disease usually experience no symptoms. In patients older than 70 years, the typical signs and symptoms also may be absent. In general, the symptoms become more obvious as the degree of hyperthyroidism increases. The symptoms Usually are related to an increase in the metabolic rate of the body.

• Excessive Sweating

• Heat Intolerance

• Increased Bowel Movements

• Tremor (Usually Fine Shaking)

• Nervousness / Agitation

• Rapid Heart Rate / Palpitations

• Insomnia

• Breathlessness

• Irregular or Scant Menstrual Periods

• Fatigue

• Weight Loss

• Muscle Weakness

• Hair Loss

Other symptoms that can occur with this condition:

• Breast development in men

• Clammy skin

• Diarrhoea

• Feeling faint when you raise the hands

• High blood pressure

• Itchy or irritated eyes

• Itchy skin

• Nausea and vomiting

• Protruding eyes (exophthalmos)

• Skin blushing or flushing

• Skin rash on the shins

• Weakness of the hips and shoulders

In older patients, irregular heart rhythms and heart failure can occur. In its most Severe form, untreated hyperthyroidism may result in ―thyroid storm,‖ a condition Involving high blood pressure, fever, and heart failure. Mental changes, such as Confusion and delirium, also may occur.

COMPLICATIONS OF HYPERTHYROIDISM

Hyperthyroidism can lead to the following complications.

Heart problems

Untreated hyperthyroidism can lead to serious complications, mainly related to the heart. When you have hyperthyroidism, the body is, in a way, running on overdrive all the time, and that can greatly affect the heart.

Some possible heart-related complications of uncontrolled hyperthyroidism are:

• Arrhythmia (abnormal heart beat, such as atrial fibrillation)

• Cardiac dilation (increase in the size of the heart cavities, which actually thins the heart muscle) and congestive heart failure.

• Sudden cardiac arrest

• Hypertension (high blood pressure)

• A heart rhythm disorder called atrial fibrillation that increases the risk of stroke.

Brittle bones

Untreated hyperthyroidism can lead to weak, brittle bones. This condition is called osteoporosis. The strength of bones depends, in part, on the amount of calcium and other minerals in them. Too much thyroid hormone makes it hard for the body to get calcium into bones.

Vision problems

Some people with hyperthyroidism develop a problem called thyroid eye disease. It‘s more common in people who smoke. This disorder affects the muscles and other tissues around the eyes.

Symptoms of thyroid eye disease include:

• Bulging eyes.

• Gritty sensation in the eyes.

• Pressure or pain in the eyes.

• Puffy or retracted eyelids.

• Reddened or inflamed eyes.

• Light sensitivity.

• Double vision.

Eye problems that go untreated may cause vision loss.

Discoloured, swollen skin

In rare cases, people with Graves‘ disease develop Graves‘ dermopathy. This causes the skin to change colors and swell, often on the shins and feet.

Thyrotoxic crisis

This rare condition also is called thyroid storm. Hyperthyroidism raises the risk of thyrotoxic crisis. It causes severe, sometimes life-threatening symptoms. It requires emergency medical care. Symptoms may include:

• Fever.

• Fast heartbeat.

• Nausea.

• Vomiting.

• Diarrhea.

• Dehydration.

• Confusion.

• Delirium.

HYPERTHYROIDISM AND PREGNANCY

Uncontrolled hyperthyroidism has many effects. It may lead to preterm birth (before 37 weeks of pregnancy) and low birth weight for the baby. Some studies have shown an increase in pregnancy-induced hypertension (high blood pressure of pregnancy) in women with hyperthyroidism.

A severe, life-threatening form of hyperthyroidism, called thyroid storm, may complicate pregnancy. This is a condition in which there are extremely high levels of thyroid hormone that can cause high fever, dehydration, diarrhoea, rapid and irregular heart rate, shock and death, if not treated.

Improvement of Graves‘ hyperthyroidism-during a woman‘s pregnancy is often associated with a reduction in the titre of maternal serum TRAb concentrations and a change from stimulatory to blocking antibodies. If antibodies do not decline, they will cross the placenta and stimulate the foetal thyroid, evidenced by signs of foetal hyperthyroidism such as tachycardia, intrauterine growth retardation, cardiac failure, and the development of foetal goitre

A deterioration in previously diagnosed thyroid disease is not uncommon during the first trimester of pregnancy and may be due to an increase in the titre of TRAb concentrations or high levels of human chorionic gonadotrophin acting as a thyroid stimulator. Relapse may also be caused by impaired absorption of antithyroid medication secondary to vomiting that is associated with pregnancy or by reluctance to continue medication in the first trimester.

Hyperthyroidism in pregnancy requiring treatment is most often caused by Grave disease, which is estimated to account for 85% to 95% of clinically significant cases of hyperthyroidism. This autoimmune condition is marked by the presence of thyrotropin (TSH)-receptor antibodies (TRAb), which can bind to thyroid receptors and cause their activation, leading to increased production of thyroid hormones.

Hyperthyroidism in pregnancy is treated with medications that inhibit excessive thyroid hormone synthesis. The antithyroid drugs (ATD) most commonly used in the U.S. are thioamides, propylthiouracil (PTU), and methimazole (MMI). Carbimazole is a prodrug

of methimazole that is commonly used outside of North America, with similar efficacy and side effect profile. All ATDs can cross the placenta and affect the foetus.

DIAGNOSIS

Many symptoms of hyperthyroidism are the same as those of other diseases, so hyperthyroidism usually cannot be diagnosed based on symptoms alone. With suspected hyperthyroidism, health care providers take a medical history and perform a thorough physical exam.

If the doctor suspects that you have hyperthyroidism, they may Perform a physical exam to Examine the thyroid for enlargement or nodules. Look for signs of too much thyroid hormone like fast heart rate, shakiness/tremor of the hands, warm and moist skin and

Look for signs of thyroid eye disease like eye redness, swelling, or bulging of the eyes.

Health care providers may then use several blood tests, such as the following, to confirm a diagnosis of hyperthyroidism.

Check the blood for the level of thyroid hormones (TSH, T4 and T3) and thyroid antibody levels (Thyroid stimulating immunoglobulin (TSI), thyrotropin receptor antibodies (TRAB), and/or Thyroid Peroxidase (TPO) antibodies).

TSH test. The ultra-sensitive TSH test is usually the first test a health care provider performs. This test detects even tiny amounts of TSH in the blood and is the most accurate measure of thyroid activity available. The TSH test is especially useful in detecting mild hyperthyroidism. Generally, a TSH reading below normal means a person has hyperthyroidism and a reading above normal means a person has hypothyroidism.

T3 and T_1 test. This test shows the levels of T and T4 in the blood. With hyperthyroidism, the levels of one or both of these hormones in the blood are higher than normal.

Thyroid-stimulating immunoglobulin (TSI) test. This test, also called a thyroid- stimulating antibody test, measures the level of TSI in the blood. Most people with Graves' disease have this antibody, but people whose hyperthyroidism is caused by other conditions do not.

The serum level of thyroid-stimulating immunoglobulins or TSH-receptor antibodies helps distinguish Graves‘ disease from other causes of hyperthyroidism in patients who lack signs pathognomonic of Graves‘ disease and have a contraindication to radioactive iodine uptake and scan.

Radioactive iodine uptake test. The radioactive iodine uptake test measures the amount of iodine the thyroid collects from the bloodstream. Uptake is the percentage of an iodine 123 (I-123) tracer dose taken up by the thyroid gland, ranging from 15% to 25% at 24 hours. The uptake is very low (0% to 2%) in patients with thyroiditis and high in patients with Graves‘ disease, a toxic adenoma, or a toxic multinodular goiter. Measuring the amount of iodine in a person's thyroid helps the health care provider determine what is causing a person's hyperthyroidism. For example, low levels of iodine uptake might be a sign of thyroiditis, whereas high levels could indicate Graves' disease.

Order imaging tests like thyroid ultrasound or nuclear

medicine scans which can help determine why the thyroid is making too much thyroid hormone.

Thyroid scan. A thyroid scan shows how and where iodine is distributed in the thyroid. The images of nodules and other possible irregularities help the health care provider diagnose the cause of a person's hyperthyroidism. The thyroid scan shows the distribution of radiotracer in the gland. A homogeneous distribution indicates Graves disease, but accumulation of I-123 in one area points to a toxic adenoma or in multiple areas to a toxic multinodular goiter.

Ultrasonography is sometimes used as a cost-effective and safe alternative to radioactive iodine uptake and scan. It is the primary imaging modality used during pregnancy, lactation, and in amiodarone-induced thyrotoxicosis.

TREATMENT

There is no single treatment that is best for all patients with hyperthyroidism. The objective of management is to monitor the situation of hyperthyroidism using medical, radioiodine or surgical treatment. Therapeutic objectives for hyperthyroidism are: to normalize the production of thyroid

hormone; minimize symptoms and long-term consequences; provide individualized therapy based on type and severity of disease, patient age and gender, existence of non-thyroidal conditions, and response to previous therapy. Treatment strategies include

antithyroid drugs, radioactive iodine, thyroid surgery, and medications for symptom control.

There are several treatments that may be recommended:

1. BETA BLOCKERS:

Beta blockers are medications that help control the symptoms such as rapid heart rate, shakiness, and nervousness until the thyroid hormone levels have improve. They do not change thyroid hormone levels.

Beta blockers can be started even before you know the cause of hyperthyroidism.

They can be used along with another treatment to control the symptoms until the thyroid hormone blood levels come down.

Examples of these medications may be propranolol, atenolol, and metoprolol.

Multiple of the symptoms of hyperthyroidism such as sweating, anxiety, tremor and palpitations are antecedent by escalated sympathetic activity and can be

controlled promptly by beta blockers. Propranolol in relatively high doses of over 160 mg per day can mildly suppress transformation of T4 to T3. Administering

beta blockers such as atenolol 50 to 100 mg or nadolol 40 to 80 mg once daily can be used to ameliorate medication compliance. In the absence of

contraindications such as asthma, beta blockers are used in the first few weeks of managing hyperthyroidism while awaiting the outcome of antithyroid drugs.

ANTITHYROID DRUGS (ATD)

The consummate commonly used antithyroid drugs are the thioamides, PTU and Methimazole (MMI). PTU and MMI obviate thyroid hormone generation by suppressing the peroxidase

enzyme system of the thyroid gland, thus preventing oxidation of trapped iodide and subsequent incorporation into iodotyrosines and finally iodothyronine (―organification‖); and by suppressing coupling of MIT and DIT to form T4 and T3 and also PTU (but not MMI) also inhibits the peripheral transformation of T4 to T3. MMI and PTU are actively concentrated by the thyroid against a concentration gradient. Their primary effect is to inhibit the intra- and extra-thyroid hormonal synthesis.

ATDs work by decreasing the ability of the thyroid cells to make new thyroid hormones. These drugs are not effective for thyroiditis. They do not cause a permanent reduction in the thyroid hormone levels but keep the levels controlled while you are taking the medication. The dose of the ATD needs to be adjusted to keep the thyroid levels in the normal range.

In some patients, Graves‘ disease gets better while taking ATDs and these drugs can be stopped.

Since toxic nodules do not go into remission from treatment with ATDs, the ATD must be continued to control hyperthyroidism. MMI is usually preferred over PTU because of less side effects, with certain exceptions such as pregnancy.

Extra precautions are needed if these medications are used in pregnancy. If you find out you are pregnant, you should contact the doctor immediately. A change in medications is sometimes necessary.

Side effects

Red skin rash or hives - if this happens, you should contact the doctor immediately to decide on the next steps. Liver injury (rare) – if there yellowing of the eyes or skin, itching, or dark urine you should contact the doctor immediately as you may need to stop the medication. Low white blood cells (rare) – this is called agranulocytosis and can become a life-threatening problem. If develop a fever or severe sore throat while taking this medication, you should seek medical care immediately to get a complete blood count (CBC) checked and not take any more doses of the medication until you are cleared by the doctor to restart the medication

Antithyroid medications and pregnancy.

Because pregnant and breastfeeding women cannot receive radioiodine therapy, they are usually treated with an antithyroid medication instead. However, experts agree that women in their first trimester of pregnancy should not take methimazole due to the rare occurrence of damage to the foetus. Another antithyroid medication, propylthiouracil (PTU), is available for women in this stage of pregnancy or for women who are allergic to or intolerant of methimazole and have no other treatment options.

3. RADIOACTIVE IODINE (RAI):

Radioactive iodine works by destroying the thyroid cells that are making thyroid hormones. This treatment works for Graves‘ disease and toxic nodules but is not effective in thyroiditis.

The RAI treatment is based on how the thyroid gland uses iodine to make thyroid hormone:

Iodine is the main ingredient the thyroid uses to make thyroid hormones. Thyroid cells pull iodine from the blood into the inside of the cell. Overactive thyroid cells pull in more iodine than usual. Therefore, when a small dose of radioactive iodine

is given as a tablet or liquid, it travels to the thyroid gland and is taken into the overactive thyroid cells. Over several weeks to months, the radiation attached to the iodine destroys the overactive thyroid cells. The gland or nodules usually shrink in size and the thyroid hormone levels drop either back to normal or to low levels (hypothyroidism). Therapy is

provided by a single oral dose of radioactive iodine that is absorbed by the thyroid gland and causes organ-specific inflammation. Continuous radiation to thyroid

cells sequences in extensive local destruction and thyroid gland is ablated over a period of six to eighteen weeks.

If you have Graves‘ disease, the doctor will choose a dose of RAI to try to shrink enough of the thyroid gland to cause permanent hypothyroidism. If you have a toxic nodule, the goal is to only destroy the overactive nodule, but leave the rest of the thyroid functioning normally. However, some patients will still develop hypothyroidism. After treatment, the thyroid blood tests are closely monitored every 1-3 months. Radioactive iodine leaves the body through different body

fluids like urine and saliva. Therefore, you will be given precautions for activities like not sharing silverware, cleaning up in the bathroom, and avoiding (or reducing) contact with others, particularly pregnant women and young children. Radioactive iodine should not be used if you are pregnant or breastfeeding. Treatment with radioactive iodine can make Thyroid Eye Disease (TED) get worse. If you have TED, the doctor can help you decide about RAI treatment.

Radioiodine and pregnancy

Although iodine-131 is not known to cause birth defects or infertility, radioiodine therapy is not used in pregnant women or women who are breastfeeding. Radioactive iodine can be harmful to the foetus‘ thyroid and can be passed from mother to child in breast milk. Experts recommend that women wait a year after treatment before becoming pregnant.

4 SURGERIES:

Hyperthyroidism can be cured by surgical removal of the overactive thyroid gland or nodules.

The entire thyroid is removed for Graves‘ disease. Only half of the thyroid may be removed in certain toxic nodules. Hyperthyroidism must be controlled prior to surgery with ATDs, beta blockers, or other medications. If you have Graves‘ disease you may be asked to take a special iodine liquid for 7-10 days before surgery. Thyroid surgery is generally safe in the hands of an experienced thyroid surgeon. It is important to choose an experienced surgeon who performs thyroid surgeries often to decrease the risk of complications.

Complications may include:

damage to the parathyroid glands. These glands are located next to the thyroid gland and control the body‘s calcium levels. If the parathyroid glands are damaged it can cause problems with low calcium levels.

damage to the nerves that control the vocal cords, causing you to have a hoarse voice.

Sometimes surgery may be used to treat

• Pregnant women who cannot tolerate antithyroid medications

• People with large goitres

• People who have cancerous thyroid nodules, though hyperthyroidism does not cause cancer

If the entire thyroid gland is removed, you will become hypothyroid since the body can no longer make thyroid hormone. Thyroid levels are restored to normal by treatment with a daily thyroid hormone replacement. If only part of the thyroid is removed, you will need to follow with the doctor to determine if you need thyroid hormone replacement after surgery.

DIETARY MANAGEMENT OF HYPERTHYROIDISM

Hyperthyroidism happens when there‘s too much thyroid hormone in the body. This condition is also called thyrotoxicosis. An overactive or enlarged thyroid gland may produce more thyroid hormone.

Certain foods can help keep the thyroid healthy and reduce some of the negative effects of this condition. Some minerals, vitamins, and other nutrients are necessary to balance thyroid function.

A low-iodine diet is usually prescribed prior to some treatments for hyperthyroidism. For example, you‘ll need to follow a low-iodine diet before having radiation therapy to remove excess or damaged thyroid cells.

After treatment, it‘s still important to balance iodine in the diet. Other foods help to protect the thyroid and reduce the long-term effects of hyperthyroidism.

Foods to eat if you have hyperthyroidism

Low-iodine foods

The mineral iodine plays a key role in making thyroid hormones. A low-iodine diet helps to reduce thyroid hormones. Add these foods to the daily diet:

• Non-iodized salt

• Decaffeinated coffee or tea (without milk or dairy- or soy-based creamers)

• Egg whites

• Fresh or canned fruit

• Unsalted nuts and nut butters

• Homemade bread or breads made without salt, dairy, and eggs

• Popcorn with non-iodized salt

• Oats

• Potatoes

• Honey

• Maple syrup

Cruciferous vegetables

Cruciferous vegetables and other types may stop the thyroid from using iodine properly. They may be beneficial for hyperthyroidism:

• Bamboo shoots

• Broccoli

• Brussels sprouts

• Cassava

• Cauliflower

• Collard greens

• Kale

• Mustard

• Rutabaga

Vitamins and minerals

Several nutrients are essential for thyroid health and to balance thyroid hormone production.

Iron

Iron is important for many vital bodily functions, including thyroid health. This mineral is needed for blood cells to carry oxygen to every cell in the body. Low levels of iron are linked to hyperthyroidism. Get plenty of iron in the diet with foods such as:

• Dried beans

• Green leafy vegetables

• Lentils

• Nuts

• Poultry, such as chicken and turkey

• Red meat

• Seeds

• Whole grains

Selenium

Selenium-rich foods may help to balance thyroid hormone levels and protect the thyroid from disease. Selenium helps to prevent cell damage and keep the thyroid and other tissues healthy.

Good food sources of selenium include:

• Brazil nuts

• Couscous

• Chia seeds

• Mushrooms

• Tea

• Meat, such as Beef and Lamb

• Rice

• Oat bran

• Poultry, such as Chicken and Turkey

• sunflower seeds

Zinc

Zinc helps you use food for energy. This mineral also helps keep the immune system and thyroid healthy. Food sources of zinc include:

• beef

• chickpeas

• cocoa powder

• cashews

• mushrooms

• pumpkin seeds

• Lamb

Calcium and vitamin D

Hyperthyroidism causes weak and brittle bones. Bone mass may be restored with treatment. Vitamin D and calcium are necessary for building healthy bones.

Calcium-rich foods include:

• spinach

• collard greens

• white beans

• kale

• okra

• calcium-fortified orange juice

• almond milk

• calcium-fortified cereals

Vitamin D is found in these low-iodine foods:

• vitamin D-fortified orange juice

• vitamin D-fortified cereals

• beef liver

• mushrooms

• fatty fish

Healthy fats

Fats that are from whole foods and largely unprocessed may help reduce inflammation. This helps to protect thyroid health and balance thyroid hormones. Nondairy fats are important in a low-iodine diet. These include:

• flaxseed oil

• olive oil

• avocado oil

• coconut oil

• sunflower oil

• safflower oil

• avocado

• unsalted nuts and seeds

Spices

Some spices and herbs have anti-inflammatory properties to help protect and balance thyroid function. Add flavor and a dose of antioxidants to the daily meals with:

• turmeric

• green chilies

• black pepper

Foods to avoid if you have hyperthyroidism

Excess iodine

Eating too many iodine-rich or iodine-fortified foods may lead to hyperthyroidism or worsen it in some cases.

According to the National Institutes of Health (NIH), a teaspoon of iodized salt contains 304 micrograms (mcg) of iodine.

Seafood has the most iodine. Just 1 gram of seaweed contains 23.2 mcg, or .02 milligrams (mg), of iodine.

The recommended daily dose of iodine is about 150mcg (0.15 mg), according to the NIH. A low-iodine diet requires even less.

Avoid the following seafood and seafood additives:

• fish

• seaweed

• prawns

• crabs

• lobster

• sushi

• carrageen

• agar-agar

• algae

• alginate

• nori

• kelp

Avoid other foods high in iodine such as:

• milk and dairy

• cheese

• egg yolks

• iodized salt

• iodized water

• some food colorings

Some medications also contain iodine. These include:

• amiodarone (Nexterone)

• cough syrups

• medical contrast dyes

• herbal or vitamin supplements

Gluten

In some people, gluten may harm the thyroid by causing inflammation. Even if you don‘t have a gluten allergy or intolerance, it may be beneficial to restrict or limit gluten.

Check food labels for gluten-containing ingredients such as:

• wheat

• barley

• brewer‘s yeast

• malt

• rye

• triticale

Soy

While soy doesn‘t contain iodine, it‘s been shown to interfere with some treatments for hyperthyroidism in animals. Avoid or limit foods with soy such as:

• soy milk

• soy sauce

• tofu

• soy-based creamers

Caffeine

Foods and beverages that contain caffeine, such as coffee, tea, soda, and chocolate, can exacerbate the symptoms of hyperthyroidism and lead to increased anxiety, nervousness, irritability, and rapid heart rate.

If caffeine has this effect on you, avoiding or limiting the intake may be a good option. Try replacing caffeinated beverages with natural herbal teas, flavored water, or hot apple cider

Dietary Supplements

Iodine is an essential mineral for the thyroid.However, people with autoimmune thyroid disease may be sensitive to harmful side effects from iodine. Taking iodine drops or eating foods containing large amounts of iodine—such as seaweed, dulse, or kelp— may cause or worsen hyperthyroidism.

Women need more iodine when they are pregnant—about 250 micrograms a day—because the baby gets iodine from the mother‘s diet. In the United States, about 7 percent of pregnant women may notget enough iodine in their diet or through prenatal vitamins. 6 Choosing iodized salt—salt supplemented with iodine—over plain salt and prenatal vitamins containing iodine will ensure this need is met.

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HYPOTHYROIDISM

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INTRODUCTION

Hypothyroidism is a disorder that occurs when the thyroid gland does not make enough thyroid hormone to meet the body's needs. Thyroid hormone regulates metabolism-the way the body uses energy- and affects nearly every organ in the body. Without enough thyroid hormone, many of the body's functions slow down. The thyroid is a 2-inch-long, butterfly-shaped gland weighing less than 1 ounce. Located in the front of the neck below the larynx, or voice box, it has two lobes, one on each side of the windpipe. The thyroid is one of the glands that make up the endocrine system. The glands of the endocrine system produce and store hormones and release them into the bloodstream. The hormones then travel through the body and direct the activity of the body's cells. The thyroid gland makes two thyroid hormones, triiodothyronine (T3) and thyroxine (T4). Thyroid hormones affect metabolism, brain development, breathing, heart and nervous system functions, body temperature, muscle strength, skin dryness, menstrual cycles, weight, and cholesterol levels. Thyroid hormone production is regulated by TSH, which is made by the pituitary gland in the brain. Thyroid hormone production is regulated by thyroid-stimulating hormone (TSH), which is made by the pituitary gland in the brain. When thyroid hormone levels in the blood are low, the pituitary releases more TSH.

When thyroid hormone levels are high, the pituitary responds by dropping TSH production. The thyroid is a vitally important hormonal gland that plays an essential role in metabolism, growth, and maturation of the human body. The thyroid produces and releases into the circulation at least two potent hormones, thyroxine (T4) and triiodothyronine (T3), which influence basal metabolic processes and enhance oxygen consumption in nearly all body tissues. Thyroid hormones also influence growth, temperature regulation, lipid and carbohydrate metabolism, cardiac myocyte activity, reproduction, cognitive functioning, and bone development. Hypothyroidism is the

insufficient production of thyroid hormone. Overt hypothyroidism is present in 0.1-2% of all adults, with 15% of older women meeting the criteria for subclinical hypothyroidism. Worldwide, iodine deficiency is the most common cause of hypothyroidism. Salt is fortified with iodine. Autoimmune thyroiditis or Hashimoto‘s thyroiditis is the most common type of hypothyroidism. In autoimmune thyroiditis there is cell-mediated antibody destruction of the thyroid gland. The second leading cause of hypothyroidism is situations when surgery, medications, or radiation have affected the functioning of the gland. Because the thyroid affects so many different physiologic processes in the body, the clinical signs of hypothyroidism are variable from one individual to another. Some patients. Present with mild symptoms in spite of having low levels of circulating thyroid hormones, while some patients have more significant symptoms despite only mildly abnormal lab testing. A condition in which the thyroid gland doesn‘t produce enough thyroid hormone.

Hypothyroidism‘s deficiency of thyroid hormones can disrupt such things as heart rate, body temperature and all aspects of metabolism. Hypothyroidism is most prevalent in older women. Hypothyroidism is the most common type of thyroid disorder. It means your thyroid gland is not active enough. This tiny gland is found in the front of your neck. Its job is to make thyroid hormone. If the gland is underactive, it may not make enough thyroid hormone. Thyroid hormones control how your body uses energy. They affect almost every organ in your body. When your thyroid doesn‘t make enough of these hormones, parts of your body slow down. Hypothyroidism is a common endocrine disorder characterized by an inadequate production of thyroid hormones by the thyroid gland. This deficiency in thyroid hormone secretion can lead to a wide range of symptoms and complications affecting various systems throughout the body. The thyroid gland, situated in the neck, plays a crucial role in regulating metabolism, energy production, and the function of organs and tissues. When it fails to produce sufficient thyroid hormones—triiodothyronine (T3) and thyroxine (T4)—the body‘s metabolic processes slow down, resulting in a cascade of symptoms. One of the hallmark symptoms of hypothyroidism is fatigue, which can be debilitating and impact daily functioning. Weight gain, despite no significant change in diet or activity level, is another common complaint, often accompanied by fluid retention and bloating. Individuals with hypothyroidism may experience constipation due to

decreased gastrointestinal motility, dry skin, hair loss, and brittle nails as a result of reduced metabolic activity affecting skin and hair follicles.

Hypothyroidism can also affect mood and cognitive function, leading to symptoms such as depression, difficulty concentrating, and memory problems. Sensitivity to cold is frequently reported, reflecting the thyroid hormone‘s role in regulating body temperature. Muscle weakness, joint pain, and stiffness may occur due to decreased metabolism and fluid retention within tissues. In addition to these symptoms, hypothyroidism can have far-reaching effects on cardiovascular health, potentially leading to elevated cholesterol levels, increased risk of atherosclerosis, and ultimately, heart disease if left untreated. Women with hypothyroidism may also experience menstrual irregularities, infertility, and complications during pregnancy. Diagnosis of hypothyroidism typically involves blood tests to measure levels of thyroid-stimulating hormone (TSH) and thyroid hormones T3 and T4. Treatment usually consists of hormone replacement therapy with synthetic thyroxine (levothyroxine) to restore normal thyroid hormone levels and alleviate symptoms. Regular monitoring and adjustment of medication dosage are essential to ensure optimal thyroid function and symptom control. In conclusion, hypothyroidism is a complex disorder with diverse manifestations affecting multiple body systems. Prompt diagnosis and appropriate management are crucial to mitigate symptoms, prevent complications, and improve the overall quality of life for individuals living with this condition.

CLASSIFICATION AND TYPES

Hypothyroidism can be classified into different types based on its aetiology and severity. Common types include:

Primary hypothyroidism: This occurs when the thyroid gland itself is unable to produce sufficient thyroid hormones. Causes can include autoimmune thyroiditis (Hashimoto‘s thyroiditis), thyroid surgery, radioactive iodine therapy, or congenital thyroid disorders.

Secondary hypothyroidism: In this type, the thyroid gland is not directly responsible for the deficiency in thyroid hormones. Instead, it‘s due to a dysfunction in the pituitary gland or

hypothalamus, which affects the production of thyroid-stimulating hormone (TSH) or thyrotropin-releasing hormone (TRH).

Subclinical hypothyroidism: This is a milder form of hypothyroidism where TSH levels are elevated, but free thyroxine (T4) levels are within the normal range. It may not cause obvious symptoms but can progress to overt hypothyroidism over time.

Overt hypothyroidism: This is the classic form of hypothyroidism where both TSH and T4 levels are outside the normal range, and clinical symptoms are present.

Congenital hypothyroidism: This is present from birth and can be due to genetic defects, maternal thyroid disorders during pregnancy, or iodine deficiency. It requires early detection and treatment to prevent developmental issues.

Some additional types and classifications of hypothyroidism:

Postpartum hypothyroidism: Some women may develop hypothyroidism after giving birth, typically within the first year postpartum. This can be due to autoimmune factors, thyroiditis, or iodine deficiency exacerbated during pregnancy.

Iatrogenic hypothyroidism: This refers to hypothyroidism induced by medical treatments or interventions, such as thyroid surgery, radioactive iodine therapy for hyperthyroidism, or certain medications like lithium and amiodarone.

Drug-induced hypothyroidism: Certain medications, including lithium, amiodarone, interferons, and tyrosine kinase inhibitors, can interfere with thyroid function and lead to hypothyroidism as a side effect.

Thyroid hormone resistance syndrome: In this rare genetic disorder, the body‘s tissues are resistant to thyroid hormones, leading to symptoms of hypothyroidism despite normal or elevated thyroid hormone levels in the blood.

Myxoedema coma: This is a severe and life-threatening form of hypothyroidism characterized by extreme symptoms such as unconsciousness, hypothermia, respiratory depression, and low blood pressure. It requires immediate medical attention and intensive care.

Hashitoxicosis: Occasionally, patients with Hashimoto‘s thyroiditis may experience transient hyperthyroidism due to the release of thyroid hormone from inflamed thyroid tissue, followed by eventual hypothyroidism as the disease progresses.

RISK FACTORS AND AETIOLOGY

Hypothyroidism can be caused by various factors, including:

Autoimmune thyroiditis (Hashimoto’s thyroiditis): The most common cause, where the body‘s immune system mistakenly attacks the thyroid gland.

Thyroid surgery or radiation treatment: Surgical removal of the thyroid gland or radiation therapy can lead to hypothyroidism.

Medications: Certain medications, such as lithium, amiodarone, and interferon alpha, can interfere with thyroid function.

Iodine deficiency: Inadequate intake of iodine, a crucial element for thyroid hormone production, can lead to hypothyroidism.

Congenital hypothyroidism: Some babies are born with an underactive thyroid gland due to genetic defects or improper development of the gland.

Pituitary or hypothalamic disorders: Conditions affecting the pituitary gland or hypothalamus, such as pituitary tumors or hypothalamic dysfunction, can disrupt thyroid hormone regulation.

Age and gender: Women, especially those over 60, are more likely to develop hypothyroidism.

Family history: Having a family history of thyroid disorders increases the risk of developing hypothyroidism.

Previous thyroid problems: People who have had thyroid surgery or treatment for hyperthyroidism are at an increased risk of developing hypothyroidism later on.

Pregnancy: Some women develop hypothyroidism during or after pregnancy, known as postpartum thyroiditis.

CLINICAL MANIFESTATIONS:

A lack of vitality, dry skin, aversion to the cold, weight gain, constipation, lethargy, weakness, and weariness. Children‘s growth delay is one possible symptom. Physical symptoms include periorbital puffiness, coarse skin and hair, bradycardia, and slurred or raspy speech. The majority of patients with pituitary failure (secondary hypothyroidism) have either clinical evidence of a pituitary adenoma, such as visual field defects, galactorrhoea, or acromegaloid features, or clinical signs of generalized pituitary insufficiency, such as abnormal menses and decreased libido].

PATHOPHYSIOLOGY:

TSH, which is created and secreted in the anterior pituitary under activation of thyrotropin-releasing hormone produced in the hypothalamus, directly stimulates thyroid gland hormone synthesis. The thyroid glands metabolism is regulated by a negative feedback regulatory system in people with a healthy hypothalamic-pituitary-thyroid axis. TSH levels are controlled by the pituitary gland in response to feedback from free-thyroxine (FT4) and free-triiodothyronine (FT3) levels, which act as biosensors of thyroid hormone levels. TSH secretion is increased when thyroid hormone synthesis declines. The control system has a rather sluggish response time, and it is possible to detect some discrepancy between the levels of TSH and the plasma thyroid hormone concentrations during non-equilibrium periods, which happen at the beginning of hypothyroidism. For three main reasons, measuring TSH is regarded as the primary test for identifying thyroid illness, specifically overt and subclinical hypothyroidism. First, the concentrations of TSH and FT4 have an inverse log-linear relationship. As a result, minor linear FT4 concentration decreases are accompanied by an exponential rise in TSH levels. Second, the primary illness of the thyroid gland accounts for the majority of hypothyroidism patients in clinical practice. Thirdly, TSH immunometric tests have sensitivity and specificity of better than 99%. Finding the FT4 level is the second stage in the thyroid problem screening process. When compared to previously used measurements of total T4 or triiodothyronine, FT4 analysis is significantly less expensive.

SYMPTOMS

The symptoms of hypothyroidism are often subtle. They are not specific (which means they can mimic the symptoms of many other conditions) and are often attributed to aging. Patients with mild hypothyroidism may have no signs or symptoms. The symptom generally become more obvious as the condition worsens and majority of these complaints are related to a metabolic slowing of the body. Common symptoms are:

Fatigue

Depression

Modest weight gain

Cold intolerance

Excessive sleepiness

Dry, course hair

Decreased concentration

Vague aches and pains

Muscle cramps

Increased cholesterol levels

Swelling of the legs

As the disease becomes more severe, there may be puffiness around the eyes, a slowing of the heart rate, a drop in body temperature, and heart failure. In its most profound form, severe hypothyroidism may lead to a life-threatening coma (myxoedema coma). In a severely hypothyroid individual, a myxoedema coma tends to be triggered by severe illness, surgery, stress, traumatic injury. This condition requires hospitalisation and immediate treatment with thyroid hormone given by injection.

Properly diagnosed, hypothyroidism can be easily and completely treated with thyroid hormone replacement. On the other hand, untreated Hypothyroidism can lead to an enlarged heart

myopathy (cardiomyopathy), worsening, heart, failure, and an accumulation of fluid around the lungs (pleural effusion).

COMPLICATIONS:

Hypothyroidism can lead to a number of health disorders if it is left untreated.

Myxoedema coma

Infertility

Miscarriage

Impaired fertility

Difficulty in conceiving

Easy miscarriage

Mental retardation

Cardiac diseases

Lead to increased level of LDL

Psychological problems

Rise of cardiomyopathy

Heart failure

Depression

1) Myxoedema:

Myxoedema is the medical term for extreme hypothyroidism -when the disorder has progressed for a long time with no treatment. Myxoedema is very rare because it‘s highly unlikely that you wouldn‘t recognize the symptoms and seek treatment. This form of hypothyroidism is life threatening.

Myxoedema can eventually slow metabolism to the point where you would fall into a coma. If you experience symptoms of myxoedema, such as extreme fatigue or cold intolerance, seek medical treatment immediately. The key to preventing the complications of hypothyroidism is to understand the disorder‘s symptoms and seek proper medical care. Hypothyroidism is manageable with the right treatment-it doesn‘t have to interfere with your everyday life.

2) Pregnancy and Fertility:

Thyroid hormones have profound effects on reproduction and pregnancy. Thyroid dysfunction is implicated in a broad spectrum of reproductive disorders, ranging from abnormal sexual development to menstrual irregularities and infertility. Hypothyroidism is associated with increased production of TRH, which stimulates pituitary to secrete

TSH and PRL. Hyperprolactinemia adversely affects fertility potential by impairing GnRH pulsatility and thereby ovarian function). Hypothyroid women experience decreased fertility, those who do conceive run a higher risk of various obstetric illness. These include abortion, pregnancy-induced hypertension, placental abruption, and post-partum haemorrhage).

3) Hypothyroidism and Psychological Problems:

Since hypothyroidism usually develops slowly, and the early complaints are frequently minor, vague and diffuse in nature, it is not surprising that the diagnosis is often overlooked. However, the physical changes that accompany the illness are characteristic: dry, rough skin; pale and puffy complexion; Loss of hair; change in voice; decreased appetite, etc.

Psychological symptoms are common and well manifested by the time the patient seeks medical advice. Not infrequently, psychological disturbances are the main complaints that bring hypothyroid patients to the psychiatrist first:

marked slowing of all mental processes

progressive loss of initiative and interest

memory difficulties

thinking is easily muddled

general intellectual deterioration

depression with paranoid flavour

organic psychosis

In severe, untreated cases, dementia may be the ultimate outcome. This underscores the importance of early detection and treatment.

4) Hypothyroidism and heart disease:

Subclinical hypothyroidism (normal serum T4, raised TSH) is usually caused by autoimmune (lymphocytic) thyroiditis, characterized by the presence of antiperoxidase antibodies in the serum,

And may be associated with coronary artery disease. For example, in one postmortem study there was histological evidence of lymphocytic thyroiditis in 20% of men and 50% of women with fatal myocardial infarction and only 10% of men and women who died from other causes. Although hyperlipidaemia is common in overt hypothyroidism this may not explain the putative link between subclinical autoimmune thyroid disease and ischemic heart disease. A meta-analysis of the many studies published between 1976 and 1996 on the effect of thyroxine replacement on lipids in subclinical hypothyroidism showed that restoration of serum TSH to normal reduced total cholesterol by only 0.4 mmol/I, and had little effect on high density lipoprotein (HDL) cholesterol.

5) Renal Complications:

Hypothyroidism frequently lowers the kidney‘s ability to excrete water. As a result, blood levels of sodium may be unusually low, or serum levels of creatinine may be unusually high. Replacing thyroid hormones can fix these complications. But if extremely low hormone levels persist, recovery from these renal disorders can take longer.

6) Nerve Damage:

Hypothyroidism can cause symptoms such as muscle weakness or nerve damage. Those with untreated hypothyroidism may also be more prone to carpal tunnel syndrome.

DIAGNOSIS

A diagnosis of hypothyroidism can be suspected in patients with fatigue, cold intolerance, constipation, and dry, flaky skin. A blood test is needed to confirm the diagnosis.

When hypothyroidism is present, the blood levels of thyroid hormones can be measured directly and are usually decreased. However, in early hypothyroidism, the level of thyroid hormones (T3 and T4) may be normal.

Therefore, the main tool for the detection of hyperthyroidism is the measurement of the TSH, the thyroid stimulating hormone. As mentioned earlier, TSH is secreted by the pituitary gland. If a decrease of thyroid hormone occurs, the pituitary gland reacts by producing more TSH and the blood TSH level increases in an attempt to encourage thyroid hormone production. This increase in TSH can actually precede the fall in thyroid hormones by months or years Thus, the measurement of TSH should be elevated in cases of hypothyroidism.

However, there is one exception. If the decrease in thyroid hormone is actually due to a defect of the pituitary or hypothalamus, then the levels of TSH are abnormally low. This kind of thyroid disease is known as ―secondary‖ or ―tertiary‖ hypothyroidism. A special test, known as the TRH test, can help distinguish if the disease is caused by a defect in the pituitary or the hypothalamus. This test requires an injection of the TRH hormone and is performed by an endocrinologist (hormone specialist).

The blood work mentioned above confirms the diagnosis of hypothyroidism, but does not point to an underlying cause. A combination of the patient‘s clinical history, antibody screening and a thyroid scan can help diagnose the precise underlying thyroid problem more clearly. If a pituitary or hypothalamic cause is suspected, an MRI of the brain and other studies may be warranted. These investigations should be made on a case-by-case basis.

TREATMENT

With the exception of certain conditions, the treatment of hypothyroidism requires life-long therapy. Before synthetic levothyroxine (T4) was available, desiccated thyroid tablets were used. Desiccated thyroid was obtained from animal thyroid glands, which lacked consistency of

potency from batch to batch. Presently, a pure, synthetic T4 is widely available. Therefore, there is no reason to use desiccated thyroid extract.

The average dose of T4 replacement in adult is 1.6 micrograms per kilograms per day. This translates approximately 100 to 150 µg per day.

In young, healthy patients, the full amount of T4 replacement hormone may be started initially.

In patients with pre-existing heart disease, this method of thyroid replacement may aggravate the underlying heart condition in about 20% of cases.

In older patients without known heart disease, starting with a full dose of thyroid replacement may result in uncovering heart disease, resulting in chest pain or a heart attack. For this reason, patients with a history of heart disease or those suspected of being at high risk are started with 25 micrograms or less of replacement hormone, with a gradual increase in the dose at 6-week intervals.

Ideally, synthetic T4 replacement should be taken in the morning, 30 minutes before eating.

Other medications containing iron or antacids should be avoided, because they interfere with absorption. Therapy for hypothyroidism is monitored at approximately six-week intervals until stable. During these visits, a blood sample is checked for TSH to determine if the appropriate amount of thyroid replacement is being given. The goal is to maintain the TSH within normal limits. Depending on the lab used, the absolute values may vary, but in general, a normal TSH range is between 0.5 to 5 Oul U/ml. Once stable, the TSH can be checked yearly. Over-treating hypothyroidism with excessive thyroid medication is potentially harmful and can cause problems with heart palpitations and blood pressure control and can also contribute to osteoporosis.

Every effort should be made to keep the TSH within the normal range.

MANAGEMENT

Hypothyroidism is a condition where the thyroid gland does not produce enough thyroid hormone. Management typically involves thyroid hormone replacement therapy, usually with synthetic levothyroxine. The goal of treatment is to normalize thyroid hormone levels and alleviate symptoms. Here's an overview of the management of hypothyroidism:

Diagnosis: Diagnosis is typically made through blood tests measuring thyroid-stimulating hormone (TSH) and free thyroxine (T4) levels. Elevated TSH and low T4 levels indicate hypothyroidism.

Thyroid Hormone Replacement Therapy: Levothyroxine is the preferred treatment for hypothyroidism. The dosage is usually started low and gradually increased until TSH levels normalize. It‘s important to take levothyroxine consistently, preferably on an empty stomach, and separate from other medications or supplements.

Monitoring: Regular monitoring of thyroid function (TSH and T4 levels) is necessary to adjust medication dosage and ensure adequate hormone replacement.

Lifestyle Modifications: Encouraging patients to maintain a healthy lifestyle, including regular exercise and a balanced diet, can help manage symptoms and support overall well-being.

Education: Patients should be educated about their condition, including the importance of adherence to medication and regular follow-ups with healthcare providers.

Comorbid Conditions: Hypothyroidism is often associated with other conditions such as cardiovascular disease, dyslipidemia, and depression. Management of these comorbidities may also be necessary.

Potential Interactions: Levothyroxine absorption can be affected by certain medications and supplements (e.g., iron, calcium, antacids), so patients should be advised to discuss all medications with their healthcare provider.

Pregnancy: Management of hypothyroidism in pregnant women is crucial, as untreated or poorly controlled hypothyroidism can have adverse effects on both the mother and the developing foetus. Thyroid hormone replacement therapy may need to be adjusted during pregnancy.

Hashimoto‘s Thyroiditis: If hypothyroidism is caused by autoimmune thyroiditis (Hashimoto‘s thyroiditis), management may also involve monitoring for other autoimmune conditions and addressing them as needed.

DIETARY MANAGEMENT

Dietary changes alone cannot cure hypothyroidism, they can support thyroid function and overall health. Some dietary considerations for managing hypothyroidism include:

Iodine: Adequate iodine intake is important for thyroid hormone production. However, excessive iodine intake can exacerbate thyroid dysfunction, so it‘s essential to consume iodine in moderation. Good sources of iodine include iodized salt, seafood, dairy products, and seaweed.

Selenium: Selenium is a trace mineral that plays a role in thyroid hormone metabolism and may help reduce inflammation in the thyroid gland. Food sources of selenium include Brazil nuts, seafood, poultry, eggs, and whole grains.

Limit Goitrogens: Goitrogens are compounds that can interfere with thyroid function by inhibiting iodine uptake or interfering with thyroid hormone synthesis. Foods high in goitrogens include cruciferous vegetables (e.g., broccoli, cabbage, cauliflower), soy products, and millet. Cooking these foods can help reduce their goitrogenic effects.

Balanced Diet: Maintaining a balanced diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats can support overall health and help manage weight, which is important for individuals with hypothyroidism.

Medication Timing: Some foods and supplements can interfere with the absorption of thyroid hormone replacement medication. It's generally recommended to take thyroid medication on an empty stomach, at least 30 minutes to an hour before breakfast, and to avoid consuming calcium, iron, or fibre supplements at the same time.

Hydration: Staying well-hydrated is important for overall health and can support thyroid function. Drinking an adequate amount of water can help maintain proper metabolism and energy levels.

Limit Processed Foods: Processed foods high in refined sugars and unhealthy fats can contribute to inflammation and may negatively impact thyroid function. Opting for whole, nutrient-dense foods is generally a better choice.

PREVENTION

It is possible to avoid developing hypothyroidism, it can be prevented with a proper diet and lifestyle management.

These can help in prevention of hypothyroidism:

Diet rich in iodine, selenium, and zinc

Giving up smoking

Keep stress and anxiety under control by doing regular exercise and yoga

Avoid self-medication

Avoid gluten, processed foods, and fast food

Low exposure to environmental radiation

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