CHAPTER 18

GASTRITIS; TYPES, SYMPTOMS, COMPLICATIONS AND DIETARY MANAGEMENT

Rimshy Sherin C
Postgraduate Student, PG Dept. of Home Science
KAHM Unity Women's College, Manjeri.
Bushaira V (Co- Author)
Assistant Professor Adhoc, PG Dept. of Home Science
KAHM Unity Women's College, Manjeri.

Introduction

Gastritis is one of the commonest problems associated to the stomach and can be considered as the beginning of different complication that may led to peptic ulcers and gastric adenocarcinoma. The term Gastritis is commonly employed for any clinical condition with upper abdominal discomfort like indigestion or dyspepsia in which the specific clinical signs and radiological abnormalities are absent. Gastritis typically presents as inflammation of the mucosal lining of the stomach, which can subsequently lead to the development of ulcers. Gastritis has been classified based on time, course, histological features and anatomical distribution or proposed pathogenic mechanism.

Gastritis and Types

In pathological terms, gastritis is defined as inflammation of the gastric mucosa. It is, however, a term often used loosely for clinical conditions associated with any upper gastrointestinal symptoms without clinical or radiological signs. Gastropathy, on the other hand, refers to a nonspecific

microscopic injury pattern of stomach mucosa, with minimal or no inflammatory cell infiltration. Both gastritis and gastropathy may be clinically asymptomatic (Lorena, E. M. et al., 2018).

Acute gastritis is an acute mucosal inflammatory process, usually of transient nature, while chronic gastritis refers to chronic inflammatory changes which may eventually lead to mucosal atrophy and epithelial metaplasia. Gastritis is a dynamic process, which can vary from acute to chronic (active or inactive), present in different stages of recovery or atrophy and may lead to complications. The inflammation may be diffuse, or affect predominantly the antrum or corpus. The mucosa of the stomach is covered by a thin surface mucus layer serving, in conjunction with bicarbonate secreting surface epithelial cells and local prostaglandin production, as a protective barrier against auto digestion and noxious agents (Evelyn, P. T. et al., 2018).

The chronic gastritis is classified on the basis of their underlying cause (e.g., *H pylori,* bile reflux, non-steroidal anti-inflammatory drugs [NSAIDs], autoimmunity or allergic response) and the histopathology pattern, which may suggest the cause and the likely clinical course (e.g., *H pylori*–associated multifocal atrophic gastritis). Other classifications are based on the endoscopic appearance of the gastric mucosa (e.g., varioliform gastritis). It is important to distinguish between gastritis and gastropathy (in which there is cell damage and regeneration, but minimal inflammation); these entities are frequently included in the differential diagnosis of chronic gastritis (Goh KL et al., 2015).

Chemical or reactive gastritis is caused by injury to the gastric mucosa resulting from reflux of bile and pancreatic secretions into the stomach, but it can also be caused by exogenous substances, including NSAIDs, acetylsalicylic acid, chemotherapeutic agents, and alcohol. These chemicals cause epithelial damage, erosions, and ulcers that are followed by

regenerative hyperplasia detectable as foveolar hyperplasia, and damage to capillaries, with mucosal edema, hemorrhage, and increased smooth muscle in the lamina propria with minimal or no inflammation (Catharina A et al., 2020).

Acute hemorrhagic gastritis

It is characterized by the presence of hyperemic, edematous mucosa, erosions/ulcers and active bleeding, and is usually seen in stress situations (e.g., severe burns), patients in ICU and after ingestion of large doses of aspirin, and other types of NSAID or large quantities of alcohol. *Acute gastritis in Helicobacter pylori infection* the initial phase of Helicobacter infection causes an acute inflammatory reaction and degenerative changes in the surface epithelial cells of the gastric mucosa. Symptoms may include Epigastric pain, a bloated feeling and nausea; these most often resolve within a week. After approximately two week the reaction evolves into an active chronic gastritis (Fang, J. Y et al., 2017).

Acute bacterial or phlegmonous gastritis

This type of gastritis is exceedingly rare and, in many cases,identified at postmortem examination. Predisposing factors include immunosuppression, disability and chronic alcoholism. The most common infective organism is Streptococcus, but Staphylococcus, Haemophilus influenza, Escherichia coli and Proteus spp have also been implicated. These bacteria cause an intense acute inflammatory response with ulceration and abscess formation involving the full thickness of the gastric wall. Patients present with nausea, vomiting, upper abdominal pain and tenderness, usually associated with a neutrophilic leukocytosis. The condition has a high mortality, but patients treated surgically have a better chance of survival (Watari J et al., (2014).

Chronic gastritis

The chronic idiopathic gastritis is caused due to toxins released by the

organisms directly affecting the gastric epithelium and local microcirculation. Gastritis is a dynamic process, which can vary from acute to chronic (active or inactive), present in different stages of recovery or atrophy and may lead to Complications (Kulnigg-Dabsch S et al., (2016)

Lymphocytic gastritis

Lymphocytic gastritis can only be made on histology, but many patients have the endoscopic features of varioliform gastritis with mucosal nodules, chronic persistent erosions and thickened mucosal folds. The disease is characterized by an infiltrate of lymphocytes in the lamina propria with large numbers of lymphocytes among the epithelial cells lining the surface and foveolar. The etiology remains uncertain, although an allergic or autoimmune pathogenesis is proposed. Lymphocytic gastritis may be found in association with coeliac disease (gluten-sensitive enteropathy), Menetrier's disease (hypertrophic gastropathy characterized by a hypertrophic gastric mucosa with convoluted, thickened mucosal folds and protein-losing enteropathy), as an abnormal response to HP infection or NSAID use, or in association with lymphocytic/collagenous colitis (Colpo E et al.,2012).

Collagenous gastritis

This is a rare entity characterized by a thickened sub epithelial band of collagen and chronic inflammatory infiltrate in the gastric mucosa, similar to that seen in collagenous colitis. The disease may be limited to the stomach, then usually in children and young adults presenting with anemia due to gastric bleeding, and demonstrate focal nodularity of the gastric mucosa on endoscopy. It may also be found in adults with collagenous colitis and present with a chronic watery diarrhea(Castellano, VM et al., 2019).

Eosinophilic gastritis

Eosinophilic gastritis and gastroenteritis may affect all age groups and present with failure to thrive (in children), abdominal pain, irritability, gastric dysmotility, vomiting, diarrhea, dysphagia and (in severe cases) protein-

losing enteropathy. Many patients are atopic and have increased serum total IgE and food-specific IgE, as well as blood eosinophilia. This entity is characterized by eosinophil-rich inflammation of all or of the GI tract wall (Blackshaw, AJ et al., 2016)

Gastroscopy may reveal antral mucosal swelling and redness with narrowing of the pylorus and diminished peristalsis. Chemical gastropathy refers to endoscopic and histological changes caused by chemical injury to the gastric mucosa. Improvement after elimination of certain foods from the diet supports an allergic aetiology. Parasites and drug reactions are a much less common cause (Blackshaw, AJ et al., 2016).

Granulomatous gastritis

This group of conditions is characterized by multiple granulomas in the gastric mucosa and has a long list of possible causes. This includes infections such as tuberculosis and histoplasmosis, foreign body reaction directed against postoperative sutures or food trapped in ulcers, tumors such as mucus-producing adenocarcinomas and lymphomas, as well as systemic granulomatous diseases such as sarcoidosis, Crohn's disease and Wegener's granulomatosis (Ko, HM et al., 2014).

PATHOPHYSIOLOGY OF GASTRITIS

H. Pylori-associated gastritis is transmitted via oral-fecal route. H. pylori possess a number of virulence factors which facilitate cell adhesion, cell damage and disruption of tight junctions and evasion from the immune response. In particular, the cytotoxin-associated gene a is considered a potent inducer of inflammation and correlate with gastric cancer development. Another factor influencing H. pylori pathogenic effects is host factors. The host susceptible factors such as polymorphism in genes coding for toll receptors or specific cytokines (Hans-Peter W et al., 2016).

The infection with H. pylori triggers IL-8 which attracts neutrophils

which release oxyradicals leading to cell damages. Lymphocyte infiltration is also noted in H. pylori infection. Chronic gastritis is mostly caused by H. pylori infection and appears either as non- atrophic or atrophic form. These two forms are phenotypes of gastritis at different stages of the same life-long disease (Hans-Peter W et al., 2016).

The pathogenesis of autoimmune gastritis is currently based on two theories. According to the first theory, an immune response against superimposed H. pylori antigen is triggered, antigen cross-reacting with antigens within the proton-pump protein or the intrinsic factor, leading to a cascade of cellular changes and causing damages to the parietal cells and stopping hydrochloric acid secretion and thus these cells gradually become atrophic and not functioning (Hans-Peter W et al., 2016).

The second theory, assume that the autoimmune disorder develops irrespective of H. pylori infection, and it is directed against the proteins of the proton-pump. As per both theories, the autoimmune gastritis is the result of a complex interaction between genetic susceptibility and environmental factors resulting in immunological dysregulation involving sensitized T lymphocytes and autoantibodies directed against parietal cells and the intrinsic factor.

SIGNS, SYMPTOMS AND COMPLICATIONS OF GASTRITIS

The signs and symptoms of gastritis depend on how long the problem has existed. If it occurs suddenly, it is called acute gastritis. In acute phase, superficial inflammation of the stomach causes the classic nausea and pain or discomfort in the upper abdomen. If it develops gradually is called chronic gastritis, and the symptoms might vary from those of acute, with a dull pain in the upper abdomen and a feeling of fullness and loss of appetite after a few bites of food. However, in some cases, people with chronic gastritis could not feel any of these symptoms (Xin, W et al., 2014).

Another type is the reactive or chemical gastritis, which is defined as a foveolar elongation, tortuosity, and hyper cellularity of the gastric surface

epithelium, together with edema, vasodilatation, congestion of gastric lamina propria, and a paucity of inflammatory cells. This type of gastritis has been thought to result from duodenogastric bile reflux or the use of NSAIDs (Oberhuber, G et al., 2017).

As there is obvious evidence that the severity and the extent of gastric atrophy relate to different risk levels of gastric cancer, an international group of gastroenterologists and pathologists, Operative Link on Gastritis Assessment (OLGA), has developed a system of histologically reporting gastritis by combining the semi-quantitative scoring scale of the updated Sydney system with the new definition of gastric atrophy. This system expresses the extent of gastric atrophy in terms of gastritis staging.

Complications of acute gastritis include bleeding from an erosion or ulcer, gastric outlet obstruction due to edema limiting an adequate transfer of food from the stomach to the small intestine, dehydration from vomiting and renal insufficiency as a result of dehydration. Chronic gastritis can lead to a number of complications if the individual does not seek treatment for the condition. Gastritis can be either erosive, which means it leads to the breakdown of the stomach lining alongside any inflammation, or non-erosive, just causing inflammation (Tsukamoto T et al., 2017).

Complications of untreated chronic gastritis include anemia (erosive gastritis can cause chronic bleeding which, in turn, can lead to anemia), atrophic gastritis (chronic inflammation in the stomach can cause the loss of both the stomach lining and glands), peptic ulcers (ulcers can form in the lining of the stomach and duodenum), growths in the stomach lining, the risk of both benign and malignant growths increases in people with gastritis. If *Helicobacter pylori* (*H. pylori*) bacteria cause gastritis, they also increase the risk of a specific form of cancer known as gastric mucosa-associated lymphoid tissue (MALT) lymphoma (Dixon M. F et al., 2014).

DIETARY MANAGEMENT OF GASTRITIS

Prompt medical care is successful in the management of an acute attack of gastritis only if it is accompanied by efficient and judicious nutritional care. During an acute attack, meeting the nutrient requirements is not of prime importance. Depending on the seriousness of the patient food maybe withheld for 24-48 hours. Fluids maybe given intravenously if needed. Liquid foods are given as per patient's tolerance level.

The amount of food and number of feedings are adjusted according to the patient's tolerance, until a full regular diet is achieved. Always follow a progressive diet i.e., liquid to semi solid to solid as when the symptoms improve. The diet should contain less fat and must be bland. Many nutritional deficiencies occur in this disorder especially during chronic gastritis e.g., vitamin B12, iron, and other vitamin deficiencies.

The nutritional treatment must follow general principles of soft diet. The diet should be adequate in calories and nutrients. There must be small feedings at regular intervals. Avoid gastric irritants and highly seasoned foods (onion garlic, chili, caffeine, cola and alcohol). Excess water or other liquids with meals may cause distention. The dietary guidelines are enumerated herewith Energy is give adequate calories through frequent feedings or else proteins would be utilized for energy of repair work. Protein is giving adequate proteins (lg/kg body weight) through skimmed milk, egg, steamed fish, chicken, minced meat etc. (Feinle-Bisset C et al., 2013).

Carbohydrates is simple easy to digest carbohydrates should be included in soft well-cooked form. Thus, semolina, rice, Maida, sago, arrowroot etc. may be included whereas whole cereals and millets should particularly be avoided if gastritis has caused damage to the mucosa. Fiber is eating a diet high in fibre reduces the risk of developing the ulcers and also speeds up the healing process.

Soluble fibres are always included in a soft cooked form. Nutritional

Management of Gastrointestinal should be completely avoided in the diet. While soluble fibre is safer for the patient as Diseases and Disorders compared to insoluble fibre (husk/bran of cereals and pulses, peels of fruits and vegetables (Elseweidy, M. et. al., 2017).

Vitamin B12 supplementation helps to treat pernicious anemia and H. pylori infection. Its sources include fish, dairy products, organ meats, eggs, beef and pork. Vitamin A is a combination of vitamin A, found in many green- and orange-colored fruits and vegetables and antacids is helpful in healing ulcers. Vitamin C is a high dose of vitamin C treatment is effective in treating H. pylori infection, it has been observed that diets high in soluble fiber, carotenoids, and antioxidants reduce the risk of developing gastritis.

Food to be included

Gastritis is an inflammatory condition; an anti-inflammatory diet can help reduce inflammation. Nutritional practices that may help manage inflammation including in the diet certain foods, such as berries, which contain polyphenols such as flavonoids and anthocyanin. Consuming fermentable fiber within lentils and other pulses, choosing carbohydrates with a low glycemic index, such as whole grains, fruits, and vegetables, limiting the intake of saturated fats and aiming for healthier fats such as omega-3 fats within fatty fish, nuts, and seeds (Watari, J. et. al., 2014).

Foods that can play a role in an anti-inflammatory diet include leafy green vegetables, as cabbage, kale, spinach, arugula, oily, such salmon, mackerel, sardines, nuts, including almonds, walnuts, fruits, as strawberries, blueberries, cherries, raspberries ,olive oil ,Garlic, ginger, turmeric, and other spices may also have anti-inflammatory properties. Probiotic compounds may help eliminate *H. pylori* from the gut, can speed up the healing of gastric ulcers (Dinis-Ribeiro, M. et. al., 2012).

Clinical trials found that those who used a probiotic supplement during treatment of *H. pylori* were twice as likely to eliminate the bacterial infection successfully and experienced fewer treatment side effects than those who did

not use a probiotic supplement. Meanwhile, consuming probiotic foods may benefit people with gastritis by boosting their overall gut health. Foods that contain beneficial probiotic bacteria include natural yogurt ,sauerkraut, miso ,kefir ,tempeh, kimchi ,sourdough bread. Additionally found that kimchi helped slow the spread of H. *Pylori* infection in mice. Probiotics are available as supplements, but a person should check with their doctor first to ensure they are safe to use (Kulnigg –Dabsch S et al., 2016).

Foods to be avoided

Dietary factors affect the symptoms include eating too fast, irregular mealtimes, irregular meal sizes, eating in restaurants, eating leftover food. Specific food types that seemed to aggravate symptoms were including sweets, spicy foods, salty foods, meat, barbecue foods, snacks, fried food, and sour foods. Foods that appear to increase the risk of inflammation are red meat, refined carbohydrates, such as white bread and pasta, sugary foods and candies, sodas and sweetened drinks (Kamimura. K. et. al., (2015).

CONCLUSION

Gastritis is the most common illness associated to the stomach and Gastritis is an inflammation of the lining, which causes upper abdominal pain and other possible symptoms like nausea and vomiting. It can have a number of potential causes, including infection with H. pylori, excessive use of NSAIDs and alcohol use. The cause of gastritis can usually be identified with a series of blood tests and/or visual tests like an upper endoscopy. Treatment depends on the underlying cause.

References

1) Blackshaw, A. J., & Levison, D. A. (2016). Eosinophil infiltrates of the gastrointestinal tract. *J Clin Pathol*, *39*, 1–7.

2) Castellano, V. M., Munoz, M, T., & Colina, F. (2019). Collagenous gastrobulbitis and collagenous colitis: case report and review of the literature. *Scand J Gastroenterol*, *34*, 632–8.

3) Catharina, A., Rima, J., Catherine, M., Hollande, W., Angelika, M., & Maria, P. (2020). Helicobacter Pylori Seroprevalence in Six Different Ethnic Groups Living in Amsterdam: The HELIUS Study. *Wiley Helicobacter.*

4) Colpo, E., Ddine, L., Ddine, C., Rodrigues, R., & Kirsten, V. (2012). Factors Associated with Chronic Gastritis in Patients with Presence and Absence of Helicobacter Pylori. *ABCD Arq Bras Cir Dig, 25*(2), 96–100.

5) Dinis-Ribeiro, M., Areia, M., & de Vries, A. (2012). Management of precancerous conditions and lesions in the stomach (MAPS): guideline from the European society of gastrointestinal endoscopy (ESGE), European Helicobacter study group (EHSG), European society of pathology (ESP), and the sociedade Portuguesa de Endoscopic digestive (SPED) Endoscopy, *44*, 74–94.

6) Dixon, M. F., Genta, R. M., & Yardley J. H. (2016). Classification and gradingof gastritis. *The American Journal of Surgical Pathology*, 20(10), 1161–1181.

7) Elseweidy, M. (2017). Brief Review on the Causes, Diagnosis and Therapeutic Treatment of Gastritis Disease. *Alternative and Integrative Medicine*, *6*, 1.

8) Evelyn, P. T., Fernanda, F. M., Mayra, P. D., Luiz, O. M., Marcela, A. P., & Viviane, S. B. (2018). Epidemiological and Clinical-Pathological Aspects of Helicobacter pylori Infection in Brazilian Children and Adults. *Gastroenterology Research & Practice*, 10.

9) Fang, J. Y., Du, Y. Q., & Liu, W. Z. (2017). Consensus opinion on chronic gastritis in China. *Gastroenterology*, *22*(11), 670–687.

10) Feinle-Bisset, C., & Azpiroz, F. (2013). Dietary and lifestyle factors in functional dyspepsia. *Nature Reviews Gastroenterology & Hepatology*, *10*(3), 150–157.

11) Goh, K. L., Chan, W. K., Shiota, S., & Yamaoka, Y. (2015). Epidemiology of Helicobacter pylori infection and public health implications. *Helicobacter*, *1*, 1-9.

12) Hans-Peter, W., & Yang, M. (2016). Different Pathophysiology of Gastritis in East and West? A Western Perspective. *Inflamm Intest Dis*, *1*, 113–122.

13) Kamimura, K., Kobayashi, M., Sato, Y., Aoyagi, Y., & Terai, S. (2015).Collagenous gastritis: Review. *World J Gastrointestinal Endoscopy*, *7*(3), 265-73.

14) Ko, H. M., Morotti, R. A., Yershov, O., & Chehade, M. (2014). Eosinophilic gastritis in children: clinic pathological correlation, disease course and response to therapy. *Am J Gastroenterol, 109*, 1277–85.

15) Kulnigg-Dabsch, S. (2016). Autoimmune gastritis, and dietary management. *166*(13-14), 424-430.

16) Lorena, E. M., Maria, O. M., Simona, M., & Vladut, S. (2018). Ibuprofen, a Potential Cause of Acute Hemorrhagic Gastritis in Children-A Case Report. *The Journal of Critical Care Medicine*, *4*(4), 143–146.

17) Oberhuber, G., Puspok, A., & Oesterreicher, C. (2017). Focally enhanced gastritis: a frequent