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Available online at: http://www.iajps.com ***Review Article***

MANAGEMENT OF CHRONIC GASTRITIS

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Abstract:

Introduction: *Chronic gastritis is one of the common serious pandemic infections in developing countries with severe sequelae such as peptic ulcer or gastric ulcer, though the prevalence of chronic gastritis has markedly declined in developed population. More than half of the people are suffering from chronic gastritis globally at present. Helicobacter pylori infection in childhood is a major contributing factor of chronic gastritis, which microbial origin is a critical factor for understanding epidemiology and course of the disease. An aggressive inflammation due to gastritis can cause severe destruction of stomach mucosa in years (Atrophic gastritis) and increases the risk of gastric cancer. Thus, prompt early diagnosis and proper measures and treatment is the key to manage chronic gastritis.****The aim of Work:*** *The review aims to understand the course, etiology, and management of chronic gastritis.****Methodology:*** *We conducted this review using a comprehensive search of MEDLINE, PubMed, and EMBASE, January 1985, through February 2017. The following search terms were used: Chronic gastritis, gastric atrophy, Helicobacter pylori, peptic ulcer.****Conclusion:*** *Gastritis has a microbial origin and infectious background; chronic gastritis may occur in long course. With knowledge and epidemiology of H.pylori, it can be managed with well along with improvement in hygiene and socioeconomics. With such improvements risk of acquiring infection lessens and it may eradicate in future decades from medical practice, in most of the developed countries.*

Keywords: *Chronic gastritis, gastric atrophy, Helicobacter pylori, peptic ulcer.*

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INTRODUCTION:

German physician Georg Ernst Stahl was the first person to use the term "gastritis" in 1728 to describe the inflammation of the inner lining of the stomach. For many years gastritis was considered as a histological finding but not disease until the discovery of Helicobacter pylori by Robin Warren and Barry Marshall in 1982 leading to its identification, classification, and description of gastritis. [1]

Chronic gastritis can be classified based on an underlying cause such as:

- H.pylori
- Bile reflux
- Nonsteroidal anti-inflammatory drugs (NSAID)
- Autoimmunity (Allergic response)
- Histopathological pattern

Another classification is based on the endoscopic appearance of gastric mucosa.
Chemical or reactive gastritis is mostly caused by injury to gastric mucosa from reflux of bile and pancreatic secretions into the stomach, but it can also be induced by exogenous substances such as NSAIDS, acetylsalicylic acid, chemotherapeutic agents and alcohol. [2]

These released chemicals cause epithelial damage, ulcers, and erosions that are followed by regenerative hyperplasia, and damage the capillaries with mucosal edema, hemorrhage and increased smooth muscles in lamina propria with less or no inflammation and hence gastropathy or chemical gastropathy is more appropriate term than chemical or reactive gastritis as updated by Sydney classification of gastritis. [3]

In certain circumstances, chronic gastritis is a minor manifestation of disease such as gastritis in the immune-compromised individual. However, H.Pyroli is the primary infection of the stomach and is the most common cause of chronic gastritis, known to infect 50% of the population all over the world.[4]

METHODOLOGY:

• Data Sources and Search terms

We conducted this review using a comprehensive search of MEDLINE, PubMed, and EMBASE, January 1985, through February 2017. The following search terms were used: Chronic gastritis, gastric atrophy, Helicobacter pylori, peptic ulcer

• Data Extraction

Two reviewers have independently reviewed the studies, abstracted data, and disagreements were resolved by consensus. Studies were evaluated for quality and a review protocol was followed throughout.
The study was approved by the ethical board of King Abdulaziz University Hospital

Etiology and Pathophysiology

Both infectious or non-infectious conditions cause chronic gastritis.
Infectious forms include: [5,6]

- H.pylori
- Heilmann infection
- Mycobacteriosis, Histoplasmosis, syphilis
- Parasitic infection- Strongyloides species and Diphyllobothrium latum
- Viral infection- CMV, herpesvirus

Non-infectious forms: [7-9]

- Chemical gastropathy: induced by chronic bile reflux, NSAID, aspirin intake
- Autoimmune gastritis
- Uremic gastropathy
- Wegener granulomatosis
- Lymphocytic gastritis
- Radiation injury to the stomach
- Ischemic gastritis
- Gastritis secondary to drug therapy (NSAIDS and aspirin)

Pathophysiology and potential development of gastric cancer [10]

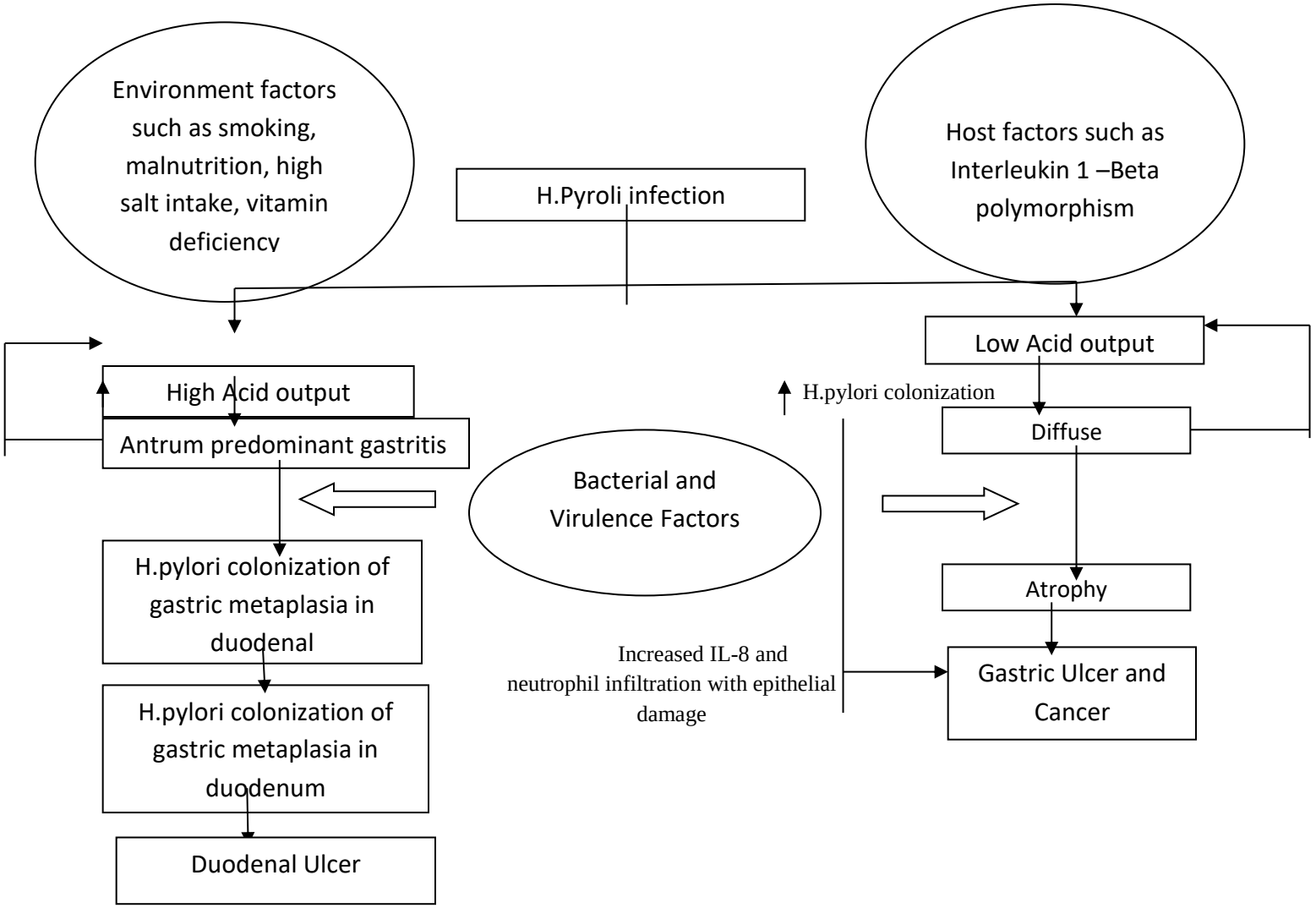

Figure 1: summary of pathophysiology of gastric cancer development by H pylori

DIAGNOSIS AND MANAGEMENT:

History and Physical Examination

Initial assessment of chronic gastritis can be done with detailed history and physical examination. Acute gastritis is usually not detected, but the persistence of H.pyroli in chronic gastritis may present as epigastric pain, nausea, vomiting, anorexia, early satiety or weight loss. Symptoms may develop in the complication of chronic gastritis such as peptic ulcers, mucosa-associated lymphoid tissue (MALT) lymphoma, gastric adenocarcinoma. [11]

Physical examination contributes very little in the assessment of chronic gastritis and management. However, findings related to the complication of chronic gastritis may help in diagnosing. Epigastric tenderness is common finding with atrophic gastritis, and if gastric ulcers are present, then it presents with guaiac-positive stool from occult blood loss. The rest physical findings may result from the development of pernicious anemia and neurologic complication, in case of autoimmune atrophic gastritis. The patient may appear pale due to severe cobalamin deficiency with

slightly icteric skin and eyes. Rapid pulse is present with or without enlargement of the heart Auscultation reveals systolic flow murmur. [11]

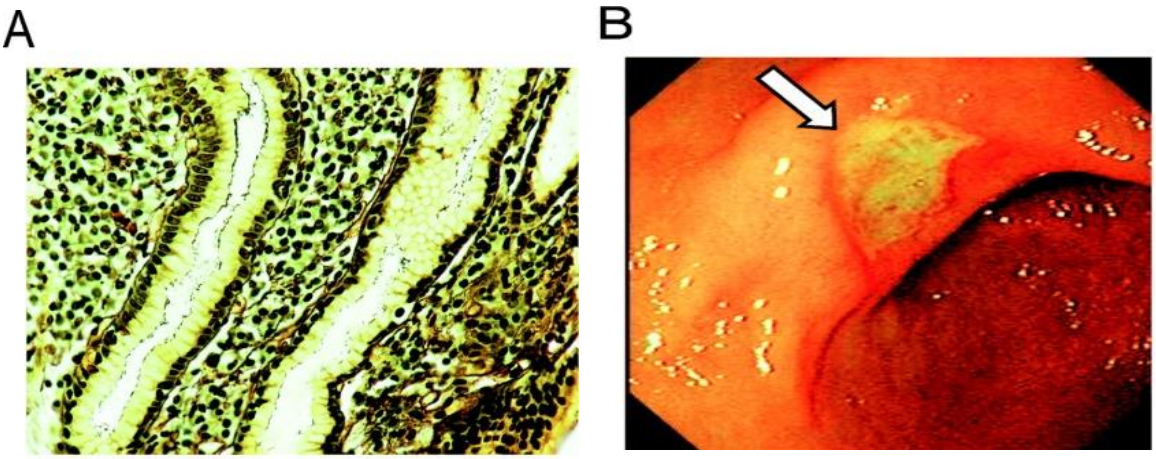

Figure 2: showing (A) Gastric gland colonized with H.pylori shown as dark curved bacilli with the mucosal surface. (B) Endoscopic view of gastric ulcer. [12]

Biopsy and Histological finding

The standard method of determining H.pyroli as an underlying cause of gastritis is histological findings. Minimum two biopsies should be obtained from gastric antrum and corpus and one from incisura. H.pyroli colonization always leads to infiltration of gastric mucosa both in antrum and corpus with neutrophilic and mononuclear cells.

Subjects in whom acid secretion is impaired, have more even distribution of bacteria in antrum and corpus with closer contact with mucosa leading to corpus-predominant pangastritis. Chronic inflammation associated with neutrophilic inflammation, the intensity of which depends on the cytotoxicity of H.pyroli strain.[13-15]

Endoscopy

An alternative to tissue biopsy is magnifying endoscopy for analyzing subepithelial microvascular architecture, as well as the mucosal surface microstructure. Upper gastrointestinal (GI) endoscopy is essential in establishing the diagnosis of gastritis. Although H.pylori infection can be determined on the basis on some unique endoscopic feature such as the presence of antral nodularity but a specific relation between endoscopy and H.pyroli infection, remain controversial. The endoscopic findings of chronic gastritis may include areas of intestinal metaplasia. Other findings such as gastric ulcers and erosions can be seen. [16]

Treatment

Treatment of chronic gastritis aims at specific etiological agent such as H.pyroli although some entities manifested by chronic gastritis do not have a well-established treatment protocol such as lymphocytic gastritis, omeprazole is proven to be successful in treatment. H.pylori infection is not readily cured and is effective with multidrug therapy since monotherapy is associated with antibiotic resistance to metronidazole and clarithromycin. Therapy must include antibiotics to which bacterium is sensitive. Clarithromycin, amoxicillin, metronidazole, tetracycline, and furazolidone is proven to be effective against H.pyroli.

Thus, most commonly used medications are: [11]

Antibiotics

- Amoxicillin
- Clarithromycin
- Tetracycline
- Metronidazole

Proton Pump Inhibitors

It is benzimidazole that inhibits the gastric acid secretion. PPIs do not exhibit anticholinergic or H_2 antagonist activity, but it suppresses acid secretion by specific inhibition of H^+/K^+ adenosine triphosphatase enzyme on the surface of parietal cells.

- Omeprazole
- Lansoprazole
- Rabeprazole
- Pantoprazole
- Esomeprazole-

Gastrointestinal Agent

- Bismuth subsalicylate

Five regimens are approved by the US Food and Drug Administration (FDA). The traditional version includes bismuth-metronidazole-tetracycline (BMT) therapy (commercially available as Helidac). Different combination of clarithromycin has been approved, dual therapies consisting 500mg of clarithromycin thrice a day with either omeprazole or ranitidine or bismuth citrate. Since higher success rate is achieved by adding a third drug to dual therapies, now mostly the triple-drug therapy is recommended which includes proton pump inhibitor (PPI) such as lansoprazole, clarithromycin, and amoxicillin. [11]

Triple therapies with indicated adult dosing (Orally, twice daily) [11]

- Lansoprazole 30mg or omeprazole 20mg or ranitidine bismuth citrate 400mg
- Clarithromycin 500mg
- Amoxicillin 1000mg or metronidazole 500mg.

PrevPac (Orally, twice daily)

- Lansoprazole 30mg
- Clarithromycin 500mg
- Amoxicillin 1000mg

Helidac (4 times a day)

- Bismuth subsalicylate 525mg
- Metronidazole 250mg
- Tetracycline hydrochloride 500mg

Quadruple Therapy with indicated adult dosing (orally) [11]

- PPI (Lansoprazole 30mg or omeprazole 30mg) twice daily
- Tetracycline HCL 500mg 4 times daily
- Bismuth subsalicylate 120mg 4 times daily
- Metronidazole 500mg 3 times daily.

Long-Term Monitoring and Follow-up

Evaluation should be done at least 4 weeks after the beginning of treatment from urea breath test or stool antigen test. Follow-up is individualized depending on the finding of endoscopy. For patients with atrophic gastritis, a minimum of 6 months follow-up is required. [11]

CONCLUSION:

In the past many years, the knowledge on gastritis has enlarged enormously. With the discovery of new form gastritis, new etiopathogeneses, classification, diagnosis, and treatment has been modified and thus there is no specific standardize approach toward management of gastritis. Biopsy sampling, histological assessment, and endoscopy with triple and quadruple drug therapy remain the gold standard for diagnosis and treatment of chronic gastritis.

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