



CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

<http://doi.org/10.5281/zenodo.1877972>

Available online at: <http://www.iajps.com>

Review Article

**DIFFERENTIAL DIAGNOSIS AND MANAGEMENT
APPROACHES OF GASTRITIS**

¹Mohammed Nasser Ahmed Asiri, ²Abdulrahman Mohammed Aladhadhi, ³Mohammed abdullah mutlaqah, ⁴Khalid Mohammed Hussain Alqahtani, ⁵Hattan Waleed M Alturki, ⁶Mohammed Awad Mohammed AlQahtani, ⁷Musab Abdullah Mohammed dahhas, ⁸Duaa Akram Ali Rajeh, ⁹Zayed Ahmed Eisa Albenawi, ¹⁰Malak Shabab Alotaibi, ¹¹Faisal Hadid Muzil Aljuaid,

¹King Khalid University, ²King Khalid University, ³ King khaild University, ⁴King Khalid University, ⁵Poznan University of Medical Sciences-Poland, ⁶ King Khalid University, ⁷King Khalid University, ⁸Almarefa University, ⁹ King Khalid University, ¹⁰King Saud University, ¹¹Taif University

Abstract:

This article reviews the reasons, diagnostic tools together with gastritis therapy. Causes of acute, chronic and consequence to HP infection are emphasized. Finally, the general examinations for HP, pharmacological medications for gastritis with focus to natural items are outlined additionally. We review the Published literature concerning management approaches of gastritis up to 2018. Search was conducted using electronic databases; Medline, and Embase. Gastritis is a problem in which the stomach lining is inflamed. The term gastritis refers specifically to irregular inflammation in the stomach cellular lining. Nonetheless, gastritis is often incorrectly utilized to define any signs and symptoms of ache or pain in the top abdominal area. Lots of people who have upper stomach symptoms do not have gastritis. One of the most usual root causes of gastritis are H. pylori infections and extended use nonsteroidal anti-inflammatory drugs (NSAIDs). Lots of people with gastritis have no symptoms.

Corresponding author:

Mohammed Nasser Ahmed Asiri,
King Khalid University

QR code



Please cite this article in press Mohammed Nasser Ahmed Asiri et al., *Differential Diagnosis and Management Approaches of Gastritis.*, Indo Am. J. P. Sci, 2018; 05(12).

INTRODUCTION:

Gastritis is an inflammation of the stomach cellular lining, which is relatively usual and can have various reasons. Several sort of agents may lead the stomach right into an irritated statement; in first place, maybe as a result of non-steroidal anti-inflammatory drugs (NSAID) such as aspirin, ibuprofen, naproxen, etc., which are utilized in various treatments to calm down some certain illness, e.g. rheumatoid arthritis; in second place, inflammation could be because of rough substances (alcohol, acids and others) or out of balance diet regimens where the stomach is harmed by its very own gastric acid; in third place, lasting physical and/or mental stress that cause the generation of extreme amounts of stomach acid; in last place, the infection caused by a widely known microorganism, *Helicobacter pylori* (HP) [1]. When stomach inflammation is not treated, mainly in the latter instance, the illness might end in a gastric ulcer or in the most awful situation, in gastric cancer.

The symptoms and signs of gastritis depend upon for how long the trouble has existed. If it occurs instantly is called acute gastritis. In acute stage, superficial inflammation of the stomach triggers the traditional nausea and pain or discomfort in the upper abdomen. If it develops gradually is called chronic gastritis, and the symptoms might differ from those of acute, with a boring pain in the upper abdominal area and a sensation of fullness and loss of appetite after a couple of bites of food [2]. Nonetheless, in many cases, individuals with chronic gastritis might not feel any one of these signs and symptoms. One more type is the reactive or chemical gastritis, which is defined as a foveolar prolongation, tortuosity, and hypercellularity of the gastric surface epithelium, together with edema, vasodilatation, congestion of gastric lamina propria, and a scarceness of inflammatory cells. This kind of gastritis has actually been thought to arise from duodenogastric bile reflux or making use of NSAIDs [2].

Several proofs suggest that *Helicobacter pylori* (H. pylori) infection and non-steroidal anti-inflammatory drug (NSAID) ingestion are significant causative elements. *Helicobacter pylori* is spiral- designed, flagellated, Gram-negative microorganism. It colonizes the stomach of concerning 50 percent of the world populace, especially in the developing countries [3]. It is directly implicated in the dyspepsia, acute and chronic gastritis, peptic ulceration, MALT lymphoma and it is an independent risk factor for gastric adenocarcinoma [3]. It might also be a risk aspect for pancreatic consisting of cancer [4]. H. pylori has been also connected to some extra-gastric diseases including

numerous autoimmune illness. The prevalence of H. pylori infection differs from country to country with huge differences in between developed and developing countries. The epidemiology of H. pylori infection in establishing countries is defined by a quick rate of acquisition of the infection such that around 80 percent of the population is contaminated by the age of 20 due to the fact that the disease is frequently obtained in youth or when young children are present in the home [5]. The prevalence of H. pylori is vice versa pertaining to socioeconomic condition [5]. The significant variable being the condition childhood years, the duration of greatest threat. Attempts to understand the various infection rates in specified groups have focused on distinctions in socioeconomic states defined by occupation, family revenue degree and living conditions. Each of these variables measures a different part of the socioeconomic facility.

This article reviews the reasons, diagnostic tools together with gastritis therapy. Causes of acute, chronic and consequence to HP infection are emphasized. Finally the general examination for HP, pharmacological medications for gastritis with focus to natural items are outlined additionally.

METHODOLOGY:

We review the Published literature concerning management approaches of gastritis up to 2018. Search was conducted using electronic databases; Medline, and Embase. Search strategy through mentioned databases was performed using medical subject headings (MeSH) as following, “*gastritis*”, “*management*”, “*treatment*”. furthermore, bibliographic of found articles were manually search for more relevant studies. Restriction to our search was applied to only English language published studies.

DISCUSSION:**• CAUSES OF GASTRITIS**

Typical causes of gastritis are many like continued intake of alcoholic beverages or long consumption of non-steroidal anti-inflammatory drugs (NSAIDs), Aspirin for Rheumatoids and Osteoarthritis patients, while Stress, chronic bile reflux, autoimmune conditions and HP infection are causal for chronic gastritis [6]. *Helicobacter pylori* (H. pylori) infection triggers most situations of chronic nonerosive gastritis. H. pylori are microorganisms that infect the stomach lining. H. pylori are largely transferred from person to person. In locations with inadequate hygiene, H. pylori might be sent through contaminated food or water. In industrialized

countries like the United States, 20 to 50 percent of the population might be infected with *H. pylori* [7]. Rates of *H. pylori* infection are greater in locations with bad cleanliness and greater population density. Infection rates might be greater than 80 percent in some developing countries [7].

The most typical root cause of erosive gastritis- acute and chronic- is long term use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin and ibuprofen. Various other agents that can trigger erosive gastritis include alcohol, cocaine, and radiation. Traumatic injuries, critical illness, severe burns, and significant surgical treatment can also cause acute erosive gastritis. This type of gastritis is called anxiety gastritis [7].

Much less usual causes of erosive and nonerosive gastritis consist of

- autoimmune ailments in which the immune system assaults healthy and balanced cells in the stomach lining
- some digestive diseases and problems, such as Crohn's disease and destructive anemia
- infections, parasites, fungi, and microorganisms aside from *H. pylori*

• CLINICAL FEATURES AND COMPLICATIONS

Chronic *H. pylori*- connected gastritis per se is asymptomatic however the initial acquisition of the infection causes acute gastritis with hypochlorhydria which might trigger stomach pain, queasiness and vomiting that willpower within a few days [8]. Uncomplicated peptic ulcers commonly cause epigastric pain and much less typically, queasiness, vomiting and weight management, whereas some ulcers (particularly NSAID ulcers) are asymptomatic. The classically explained pain of duodenal ulcer is really felt as an expanding or melting sensation, typically with a relationship to dishes; happening 1-3 hours after meals and/ or in the evening and eased by food. Gastric abscess pain is instead commonly sped up by food. Nonetheless signs are actually very badly prejudiced for ulceration site and even for whether an ulcer is present. Evaluation generally discloses epigastric inflammation however might be normal.

H. pylori ulcers generally heal and regression spontaneously but ulcers of any reason, and especially NSAID- generated ulcers, may cause significant complications. Really hemorrhaging ulcers create anemia, perforation results in peritonism and gastric outlet blockage causes consistent vomiting [8]. The discovery of *H. pylori* for that reason has reinvented the management of peptic ulcers; its obliteration heals *H. pylori*- induced

ulcers and avoids their regression.

• DIAGNOSIS

The most typical diagnostic examination for gastritis is endoscopy with a biopsy of the stomach. The doctor will normally give the patient medicine to lower pain and stress and anxiety prior to beginning the endoscopy treatment. The physician then inserts an endoscope, a slim tube with a tiny camera on the end, with the patient's mouth or nose and right into the stomach. The physician uses the endoscope to analyze the cellular lining of the esophagus, stomach, and very first section of the small intestine. If essential, the medical professional will utilize the endoscope to do a biopsy, which includes accumulating tiny samples of tissue for evaluation with a microscopic lens. Other examinations utilized to determine the cause of gastritis or any issues include the following:

- **Upper gastrointestinal (GI) series.** The patient ingests barium, a fluid comparison material that makes the digestive tract noticeable in an x ray. X-ray pictures may show changes in the stomach cellular lining, such as erosions or ulcers.

- **Blood test.** The doctor might look for anemia, a condition in which the blood's iron-rich compound, hemoglobin, is decreased. Anemia might signify chronic bleeding in the stomach.

- **Stool test.** This examination looks for the presence of blood in the stool, another indication of blood loss in the stomach.

- **Tests for *H. pylori*.**

Test for *H. pylori*

Tests not requiring endoscopy:

Serology: serological tests involve detection of IgG antibodies versus *H. pylori* and the best are very accurate. Nonetheless, precision depends seriously on the accurate serological test used. Serology might continue to be positive for many years after effective elimination of *H. pylori* and therefore is not used for inspecting the treatment success, it is cheap [9]

Urea breath test (UBT): the UBT is an easy, non intrusive examination based on *H. pylori* urease. It is especially helpful for checking the success of treatment. It is likewise more precise than serology and frequently used as a first-line diagnostic examination in position where it is readily available. It needs to be executed at the very least 4 weeks after any bismuth compounds, prescription antibiotics or proton pump preventions have actually been stopped. If not, false-negative results are common. It is cost-effective and readily offered to basic practitioners in the majority of countries.

Stool antigen test: It is more established as alternative to the urea breath examination [10] Like

the last it examines active infection therefore can be used for assessing therapy success given that it is less costly than the UBT.

Test requiring endoscopy:

Biopsy UREASE test: The biopsy is placed in a urea solution or gel with a PH indicator. When *H. pylori* exists, the urea is hydrolysed by its urease, resulting in a color change. Some favorable outcomes may be available within minutes, although originally unfavorable examinations should be maintained for 24 hours to prevent periodic false-negative outcomes. Blood in the upper GIT may likewise occasionally cause a false-negative test. The biopsy urease examination is cheap and extensively offered.

Histology: *H. pylori* infection can be detected properly by histology if special stains are utilized. The distribution of gastritis may provide information on disease threat if biopsies are taken from antrum and corpus. Histology can additionally provide more information, for example on whether gastric atrophy or intestinal tract metaplasia- markers of boosted risk of gastric adenocarcinoma- are present.

Culture: Endoscopic mucosal biopsy samplings can be cultured for *H. pylori*. Although some research studies referred to it as being not helpful as a simply analysis examination as *H. pylori* is not straightforward to grow, and culture is usually wrongly unfavorable. Nevertheless, success rates are high in others, isolates acquired from biopsy of particular people having positive IgM serologically were utilized for induction of gastritis in experimental rats [11]. The serum concentrations of pepsinogen 1 and 11 (Pg1, Pg11), gastrin (G17) and HP anti bodies of IgG class have been used to assess the threat of atrophic gastritis and to distinguish between HP-related and non-HP- relevant gastritis [11].

• **TREATMENT METHODS**

Depending on the causal elements of gastritis especially acute kind due to lengthy intake of non-steroidal anti-inflammatory medications or alcohol, this can soothed by quitting their usage while therapy of the chronic type may require different antibiotics additionally metronidazole.

Gastritis medications

Usually it is recommended to use a mix of antibiotics with metronidazole for 10-14 days [12]. Proton pump inhibitors like omeprazole are likewise suggested to reduce gastric acid production and facilitate quick healing [13]. Randomized medical trial for HP gastritis patients using lansoprazole in combination with clarithromycin, amoxicillin, jinghuaweikang gelatin pearl for 10 days complied with by added 14 days using the gelatin pearl alone showed

symptomatic renovation managing epigastric ache, bloating and belching [14]. Medicines that minimize the amount of acid in the stomach can eliminate symptoms that may come with gastritis and promote healing of the stomach cellular lining. These medicines consist of

- antacids, such as aspirin, sodium bicarbonate, and citric acid (Alka-Seltzer); alumina and magnesia (Maalox); and calcium carbonate and magnesia (Rolaids). Antacids relieve mild heartburn or dyspepsia by neutralizing acid in the stomach. These medications might create adverse effects such as diarrhea or constipation.

- histamine 2 (H2) blockers, such as famotidine (Pepcid AC) and ranitidine (Zantac 75). H2 blockers lower acid generation. They are offered both over the counter and by prescription.

- proton pump inhibitors (PPIs), such as omeprazole (Prilosec, Zegerid), lansoprazole (Prevacid), pantoprazole (Protonix), rabeprazole (Aciphex), esomeprazole (Nexium), and dexlansoprazole (Kapidex). Every one of these medications are readily available by prescription, and some are likewise readily available over the counter. PPIs reduce acid generation better than H2 blockers.

Depending on the cause of the gastritis, added procedures or treatments may be needed. For instance, if gastritis is triggered by prolonged use of NSAIDs, a physician may advise a person to stop taking NSAIDs, minimize the dosage of NSAIDs, or change to another class of medications for pain. PPIs might be made use of to stop anxiety gastritis in seriously unwell patients [14]. Dealing with *H. pylori* infections is essential, even if an individual is not experiencing symptoms from the infection. Neglected *H. pylori* gastritis may cause cancer or the growth of ulcers in the stomach or small intestine. The most common treatment is a three-way therapy that combines a PPI and two antibiotics- typically amoxicillin and clarithromycin- to eliminate the germs. Treatment may additionally include bismuth subsalicylate (Pepto-Bismol) to aid kill bacteria. After treatment, the doctor may make use of a breath or feces examination to see to it the *H. pylori* infection is gone. Treating the infection can be anticipated to cure the gastritis and reduce the danger of various other intestinal diseases associated with gastritis, such as peptic ulcer illness, gastric cancer, and MALT lymphoma.

Natural products as medication for gastritis

Tannins and Flavonoids (Phenolic compounds), mostly found in several medicinal plants have usually certain therapeutic effects. Antiinflammatory, antioxidant, anti-ulcerogenic and wound healing are mostly attributed to these constituents [15].

Antioxidants content of Fresh vegetables and fruits have beneficial effect on GIT mucosa. Curcumin and black seed oil have significant effect on specific mucosal lesions (ulcers) due to their flavonoids content [16]. Many plants like quassin artichoke, quercetin have inhibitory influence on cytokine mediated inflammatory mechanism, ulcer healing and suppression of NO synthesis (iNOS). The quassonoidisobrucein B (isoB), one of the main constituents of *Picrolemmasprucei* has proved to provide protective effect against NSAID -induced gastritis. This was attributed to reduction in IL-1 β , TNF α , prostaglandins additionally leukocyte rolling and migration [17].

Licorice extracts have significant effect on HP gastritis and gastric cancer due to its potent ant oxidative, anti-inflammatory, and antimutagenic actions. The expressions of COX-2, iNOS, VEGF, and IL-8 were increased after *H. pylori* infection, turned to be significantly decreased with s-lico in a dose-dependent manner [18]. *Croton campestris* A. St.-Hill., popularly known as "vela me do campo", is a species native from savannah area of Northeast Brazil and used by traditional communities in folk medicine for a variety of health problems, especially detoxification, inflammation and gastritis. CCRE a traditional Brazilian medicine against gastric disorders have antinuclear activity, mostly due to its stimulation of NO synthesis and activation of endogenous prostaglandin production [19].

CONCLUSION:

Gastritis is a problem in which the stomach lining is inflamed. The term gastritis refers specifically to irregular inflammation in the stomach cellular lining. Nonetheless, gastritis is often incorrectly utilized to define any signs and symptoms of ache or pain in the top abdominal area. Lots of people who have upper stomach symptoms do not have gastritis. One of the most usual root causes of gastritis are *H. pylori* infections and extended use nonsteroidal anti-inflammatory drugs (NSAIDs). Lots of people with gastritis have no symptoms. Those that do have signs might experience dyspepsia- upper stomach discomfort or pain, queasiness, or vomiting. Treating *H. pylori* infection is necessary, even if a person is not experiencing signs and symptoms. Left unattended, *H. pylori* infection may cause peptic ulcer disease or cancer.

REFERENCES:

1. Rugge, M., Fassan, M., Pizzi, M., Pennelli, G., Nitti, D., & Farinati, F. (2011). Operative Link for Gastritis Assessment gastritis staging

- incorporates intestinal metaplasia subtyping. *Hum Pathol*, 42, 1539-1544.
2. Rugge, M., Meggio, A., Pennelli, G., Pisciole, F., Giacomelli, L., De Pretis, G., & Gra-ham, D. Y. (2007). Gastritis staging in clinical practice: the OLGa staging system. *Gut*, 56, 631-636.
3. Bruce, M. G., & Maaros, H. I. (2008). Epidemiology of *Helicobacter pylori* infection. *Helicobacter* 13Suppl , 1, 1-6.
4. Trikudanathan, G., Philip, A., Dasanu, C. A., Baker, W. L., & (2011, . (2011). Association between *Helicobacter pylori* infection and pancreatic cancer. A cumulative metaanalysis. *Jop*, 12, 26-31.
5. Robinson, K., Argent, R. H., Atherton, J. C., & (2007,. (2007). The inflammatory and immune response to *Helicobacter pylori* infection. *Best Pract Res Clin Gastroenterol*, 21, 237-259.
6. Kasper DL, Braunwald E, Fauci AS, Hauser A, Longo DL, et al. (2006) Harrison. *Principios de Medicina Interna*.
7. Lee Y, Liou J, Wu M, Wu C, Lin J. Review: eradication of *Helicobacter pylori* to prevent gastroduodenal diseases: hitting more than one bird with the same stone. *Therapeutic Advances in Gastroenterology*. 2008;1(2):111–120.
8. Fischer, W., Puls, J., Buhrdorf, R., Gebert, B., Odenbreit, S., Haas, R., & (2001, . (2001). Systematic mutagenesis of the *Helicobacter pylori* cag pathogenicity island: essential genes for CagA translocation in host cells and induction of interleukin-8. *Mol Microbiol*, 42, 1337-1348.
9. Majumdar, D., Bebb, J., & (2010, J. A. (2010). *H. pylori* infection and peptic ulcers. *Medicine*, 39, 154-161.
10. Gisbert, J. P., de la Morena, F., Abaira, V., & (2006). Accuracy of monoclonal stool antigen test for the diagnosis of *H. pylori* infection: a systematic review and meta-analysis. *Am J Gastroenterol*, 101, 1921-1930.
11. Elseweidy, M., Taha, M. M., & , N. N. Y. (2010). pattern of Gastritis as manipulated by current state of *H. pylori* infection *Int J of Biology and biomedical engineering* , 4, 1998-4510.
12. Scherübl H, Fischbach W, Glocker E, Malferteiner P (2015) What is new in treating *Helicobacter pylori* infection? *Dtsch Med Wochenschr* 140: 277-280.
13. Sakamoto Y, Shimoyama T, Nakagawa S, Mikami T, Fukuda S (2014) Proton pump inhibitor treatment decreases the incidence of upper gastrointestinal disorders in elderly Japanese patients treated with NSAIDs. *Intern Med* 53: 1107-1111.

14. Wang TT, Zhang YM, Zhang XZ, Cheng H, Hu FL, et al. (2013) Jinghuaweikang gelatin pearls plus proton pump inhibitor-based triple regimen in the treatment of chronic atrophic gastritis with *Helicobacter pylori* infection: a multicenter, randomized, controlled clinical study. *Zhonghua Yi Xue Za Zhi* 93: 3491-3495.
15. Elseweidy MM (2011) "Gastritis and Gastric Cancer - New Insights in Gastroprotection, Diagnosis and Treatments", Role of Natural Antioxidants in Gastritis. INTECH Open Access Publisher.
16. Elseweidy MM, Younis NN, Amin RS, Abdallah FR, Fathy AM, et al. (2008) Effect of some natural products either alone or in combination on gastritis induced in experimental rats. *Dig Dis Sci* 53: 1774-1784.
17. Vieira SM, Silva RL, Lemos HP, Amorim RC, Silva EC, et al. (2014) Gastro-protective effects of isobrucein B, a quassinoid isolated from *Picrolemma sprucei*. *Fitoterapia* 95: 8-15.
18. Park JM, Park SH, Hong KS, Han YM, Jang SH, et al. (2014) Special licorice extracts containing lowered glycyrrhizin and enhanced licochalcone A prevented *Helicobacter pylori*-initiated, salt diet-promoted gastric tumorigenesis. *Helicobacter* 19: 221-236.
19. Júnior FE, de Oliveira DR, Boligon AA, Athayde ML, Kamdem JP, et al. (2014) Protective effects of *Croton campestris* A. St-Hill in different ulcer models in rodents: evidence for the involvement of nitric oxide and prostaglandins. *J Ethnopharmacol* 153: 469-477.