WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 13, Issue 20, 445-449. Review Article ISSN 2277–7105

# THE REVIEW ON DISEASE GASTRITIS

**Shraddha Chaudhari***

Chhatrapati Sambhajinagar, Maharashtra, India.

Article Received on
02 September 2024,

Revised on 23 Sept. 2024,
Accepted on 13 October 2024

DOI: 10.20959/wjpr202420-34275

***Corresponding Author**
**Shraddha Chaudhari**
Chhatrapati Sambhajinagar,
Maharashtra, India.

## ABSTRACT

There is evidence concerning the participation of reactive oxygen species in the etiology and physiopathology of human diseases, such as neurodegenerative disorders, inflammation, viral infections, autoimmune pathologies, and digestive system disorders such as gastrointestinal inflammation and gastric ulcer. The stomach is the main organ of exposure and absorption of nutrients, toxic food contaminants and therapeutic drugs. It is also exposed to secreted metabolites and the metabolic products coming from the intestinal bacteria. The alteration of the integrity and/or function of the intestinal epithelium produce a negative impact on the rest of the organism. This review was designed to analyze the existing data from in vitro and in vivo animal and human studies.

**KEYWORDS:** inflammation, nutrients, neurodegenerative disorders, gastric ulcer.

## RESULT

**DISEASE:** GASTRITIS

Gastritis represents an inflammation of the stomach lining in response to injury. It is either acute or chronic, and has many underlying causes which can be diagnosed and classified histologically where endoscopic appearances such as redness are often misleading. Gastritis is seldom if ever symptomatic, but usually have important clinical sequelae, principally duodenal and gastric ulceration, gastric adenocarcinoma and primary gastric lymphoma. The three most important causes of gastritis are categorized as Helicobacter pyloriinfection, prolonged use of aspirin, non-steroidal anti inflammatory drugs (NSAIDs) and autoimmunity.

## DISCUSSION

Types 1. Acute gastritis 2. Chronic gastritis

**Acute Gastritis**

Acute gastritis, is usually a diffuse and intense mucosal alteration, mostly is characterized by a sudden onset of symptoms and rapid resolution after the underlying aetiological mechanisms or agents (either chemical or physical) have been corrected. The patients can present with an acute gastroenteritis-like illness, or other symptoms which may be overshadowed by their general physical condition.

**Pathophysiology:** Acute gastritis has a number of causes, including certain drugs; alcohol; bile; ischemia; bacterial, viral, and fungal infections; acute stress (shock); radiation; allergy food poisoning; and direct trauma. The common mechanism of injury is an imbalance between the aggressive and the defensive factors that maintain the integrity of the gastric mucosal lining.

**Chronic Gastritis**

Chronic gastritis is caused mainly by Helicobacter pylori infection, and nonatrophic gastritis progresses to atrophic gastritis for a long period. It is characterized by the presence of chronic inflammatory infiltrate in the gastric mucosa. Chronic gastritis can be classified on the base of the underlying etiologic agent (eg, Helicobacter pylori, bile reflux, nonsteroidal anti-inflammatory drugs autoimmunity, allergic response) and the histopathologic pattern, which may suggest the etiologic agent and clinical course (eg, H pylori –associated multifocal atrophic gastritis).

Pathophysiology The pathophysiology of chronic gastritis complicating a systemic disease, such as hepatic cirrhosis, uremia, or another infection, is described in the relevant disease. The pathogenesis of the most common forms of gastritis is described as follows. The host response to H. pylori and bacterial products is composed of T- and B-cell lymphocytes, denoting chronic gastritis, followed by infiltration of the lamina propria and gastric epithelium by polymorphonuclear leukocytes that eventually phagocytize the bacteria.

**DIAGNOSIS**

**1. Endoscopy**

Wireless capsule endoscopy (WCE) might move through human body and captures the small bowel and captures the video and require the analysis of all frames of video due to which the diagnosis of gastrointestinal infections by the physician is a tedious task. This tiresome assignment has fuelled the researcher’s efforts to present an automated technique for

gastrointestinal infections detection. The segmentation of stomach infections is a challenging task because the lesion region having low contrast and irregular shape and size. To handle this challenging task, in this research work a new deep semantic segmentation model is suggested for 3D-segmentation of the different types of stomach infections. Similarity among the different types of stomach lesions accurate classification is a difficult task, which is addressed in this reported research by extracting deep features from global input images using a pretrained ResNet-50 model. Furthermore, the latest advances in the estimation of uncertainty and model interpretability in the classification of different types of stomach.

**2. Tests for H. pylori**

Your doctor may recommend tests — such as a stool test or breath test — to determine whether you have the bacterium H. pylori. Which type of test you undergo depends on your situation. For the breath test, you drink a small glass of clear, tasteless liquid that contains radioactive carbon. H. pylori bacteria break down the test liquid in your stomach. Later, you blow into a bag, which is then sealed. If you're infected with H. pylori, your breath sample will contain the radioactive carbon.

**3. X-ray of your upper digestive system**

Sometimes called a barium swallow or upper gastrointestinal series, this series of X-rays creates images of your esophagus, stomach and small intestine to look for anything unusual. To make an ulcer more visible, you may swallow a white, metallic liquid (containing barium) that coats your digestive tract.

**Treatment**

Treatment of gastritis depends on the specific cause. Acute gastritis caused by nonsteroidal anti-inflammatory drugs or alcohol may be relieved by stopping use of those substances.

**Medications used to treat gastritis include**

**1. Antibiotic medications to kill H. pylori**

For H. pylori in your digestive tract, your doctor may recommend a combination of antibiotics, such as clarithromycin (Biaxin XL) and amoxicillin (Amoxil, Augmentin, others) or metronidazole (Flagyl), to kill the bacterium. Be sure to take the full antibiotic prescription, usually for 7 to 14 days, along with medication to block acid production. Once treated, your doctor will retest you for H. pylori to be sure it has been destroyed.

2. Medications that block acid production and promote healing Proton pump inhibitors reduce acid by blocking the action of the parts of cells that produce acid. These drugs include the prescription and over-the-counter medications omeprazole (Prilosec), lansoprazole (Prevacid), rabeprazole (Aciphex), pantoprazole (Protonix) and others. Long-term use of proton pump inhibitors, particularly at high doses, may increase your risk of hip, wrist and spine fractures. Ask your doctor whether a calcium supplement may reduce this risk.

3. Medications to reduce acid production Acid blockers — also called histamine blockers — reduce the amount of acid released into your digestive tract, which relieves gastritis pain and encourages healing. Available by prescription or over the counter, acid blockers include famotidine, cimetidine and nizatidine.

4. Medications that neutralize stomach acid Your doctor may include an antacid in your drug regimen. Antacids neutralize existing stomach acid and can provide rapid pain relief. Side effects can include constipation or diarrhoea, depending on the main ingredients. These help with immediate symptom relief but are generally not used as a primary treatment. Proton pump inhibitors and acid blockers are more effective.

## CONCLUSION

The link of oxidative stress and gastritis ulcer is well organized. Oral intake of the combination of this herbal ingredient can induce reduction in bacterial colony forming unit in acetic acid induce gastric ulcer, inflammation. Herbal medicines are effective in treating gastritis with fewer side effects and lower recurrence rates and anti-H.pylori therapy could improve outcomes of patient with gastric ulcer, gastritis.

## REFERENCES

1. Agra M. F. SilvaK. N. BasilioI. J. L. D. Freitas P. F. Barbosa-FilhoJ. M. 2008 Survey of medicinal plants used in the region northeast of Brazil. Brazilian Journal of Pharmacognosy, 1834725080010-2695X.
2. Alarcon laLastra. C.BarrancoM. D.MartinM. J.Herrerías J. MotilvaV. 2002 Extra-virgin olive oil enriched diets reduce indomethacin-induced gastric oxidative damage in rats. Dig Dis Sci, 4712278327901573-2568
3. Albert-Puleo M. 1980 Fennel and anise as estrogenic agents. J. Ethnopharmacol., 243373440378-8741

4. Alem M. Alem N. Cohen H. England T. Hamedi N. Moussazadeh M. Roth J. A. Shen G. Q. 2002 Diagnostic value of detection of IgM antibodies to Helicobacter pylori. Exp Mol Pathol., 72177830531-5522.
5. Jiang Y., Turgeon D.K., Wright B.D., Sidahmed E., Ruffin M.T., Brenner D.E., Sen A., Zick S.M. Effect of ginger root on cyclooxygenase-1 and 15-hydroxyprostaglandin dehydrogenase expression in colonic mucosa of humans at normal and increased risk for colorectal cancer. Eur. J. Cancer Prev, 2013; 22: 455–460. doi: 10.1097/CEJ.0b013e32835c829b.
6. Moher D., Liberati A., Tetzlaff J., Altman D.G. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. BMJ, 2009; 339: b2535. doi: 10.1136/bmj.b2535.
7. Higgins J.P.T., Altman D.G., Gøtzsche P.C., Jüni P., Moher D., Oxman A.D., Savović J., Schulz K.F., Weeks L., Sterne J.A.C. The cochrane collaboration's tool for assessing risk of bias in randomised trials. BMJ, 2011; 343: d5928. doi: 10.1136/bmj.d5928.