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To study the *Helicobacter pylori* infection in peptic ulcer

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Abstract

Helicobacter pylori is a gram negative bacterium which causes chronic gastritis and plays important roles in peptic ulcer disease, gastric carcinoma, and gastric lymphoma. Its major in the human pathogen that produces inflammation of the stomach and etiologically related to the duodenal ulcer, gastric ulcer, gastric cancer and mucosa-lymphoid tissue. It is weakens the protective mucous coating of the stomach and duodenum, thus allowing acid to get through to the sensitive lining beneath. Both the acid and the bacteria irritate the lining and cause a sore, or ulcer. Helicobacter treated with a combination of antibiotics plus a proton pump inhibitor. Peptic ulcer is characterized by discontinuation in the inner lining of the gastrointestinal tract because of the gastric acid secretion or pepsin. It extends into muscularis propria layer of the gastric epithelium. It usually occurs in the stomach and proximal duodenum. Peptic means that cause of the problem is due to the acid. It is most common causes in the peptic ulcers are infection with the bacterium and causes the long-term use of the non-steroidal anti-inflammatory drugs. Gastric ulcer occurs in the stomach. Duodenal ulcer occurs in the first part of the small intestine. The interplay of gastritis phenotype related to the bacterium colonization and resulting acid secretion.

Keywords: Antibiotic; Infection; Treatment; Vaccine; Immune responses.

1. Introduction

Helicobacter pylori is a non spore, gram-negative bacterium with a helical shape curved forms occur and the bacillus also converts to the coccoid morphology under environmental stress. It has multiple flagella at one pole [1-5] and is actively motile. Flagella play important role in motion and adhesion [1]. Helicobacter pylori grow in an atmosphere of 5-15 percent of oxygen, 5-12 percent of carbon dioxide, and 70-90% nitrogen that is [microaerophilic]. Helicobacter pylori can survive in an acid Helicobacter pylori is a type of bacteria that infects mainly in the stomach. It damage the tissue in your stomach and the first part of your small intestine. This can cause redness and soreness [2]. Peptic ulcers are open sores that develop on the inside lining of your stomach and the upper portion of your small intestine. An ulcer in the stomach is a called a gastric ulcer [Figure 1], and an ulcer in the duodenum is called a duodenal ulcer. Most ulcers are caused by bacteria H pylori which are believed to be transmitted from person to person a gastric ulcer, and an ulcer in the duodenum is called a duodenal ulcer. Most ulcers are caused by bacteria H pylori which are believed to be transmitted from person to person [3]

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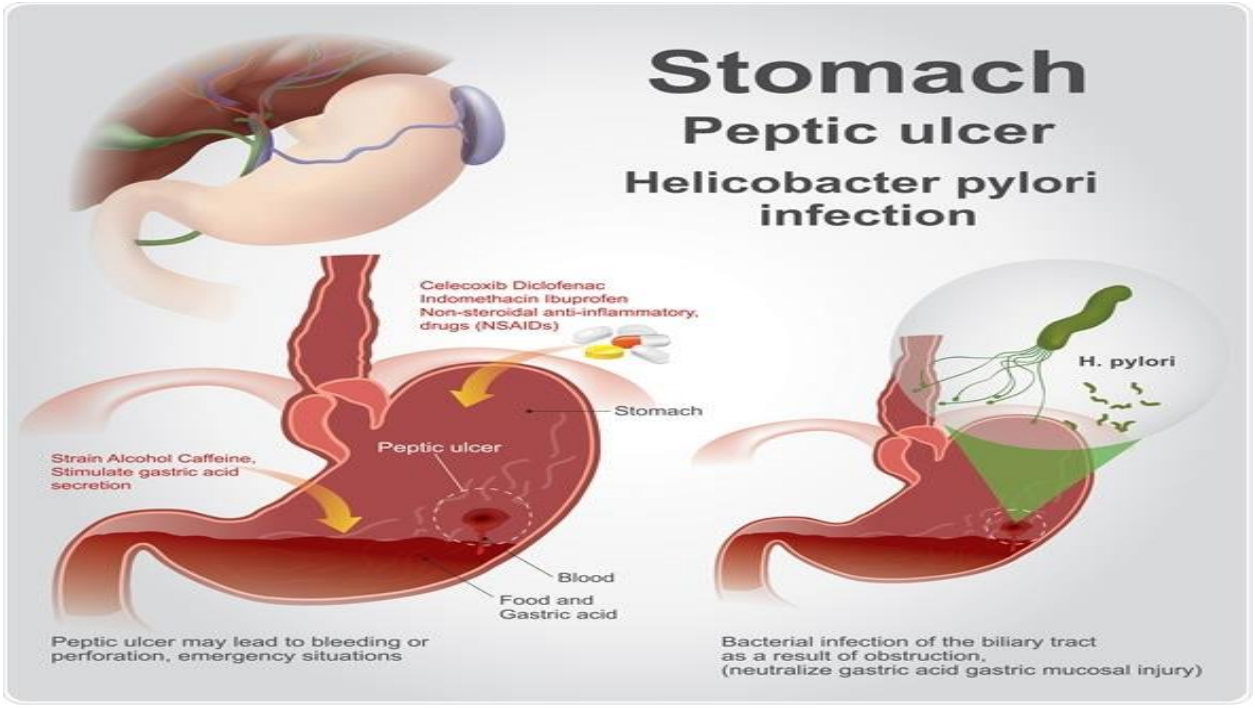

Figure 1 Peptic Ulcer Infection

1.1. General Aspects of the Helicobacter Pylori

In 1979, Robin Warren, an Australian pathologist, performing histological of gastric biopsies, often observed curved microorganisms in the inflammatory cells infiltrated tissue. The organisms were not present inside the gastric mucosa, but in the mucus covering the tissue [4]. Barry Marshall and Robin warren 1982 tried is isolate bacteria organisms from gastric biopsies samples associated with chronic gastritis and gastric ulcers. It over 80 percent of individuals infected with the bacterium are asymptomatic. infected with the bacterium are asymptomatic [5].

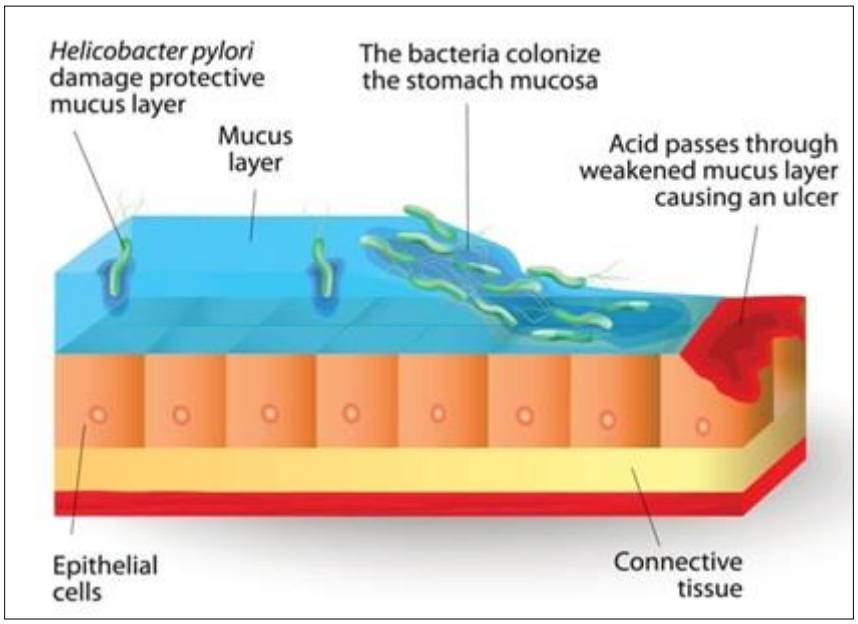

Figure 2 Peptic Ulcer

2. Epidemiology

Helicobacter pylori infection is well known to be the most common human infection worldwide. Approximately 50% of the world's populations are infected and that human beings are the main reservoir [6]. The pattern of the infection is an early childhood acquisition of H pylori [30%-50%] that reaches over 90% [Figure 3] during adulthood in developing countries. Infection in developed countries is less common in young children and reaches up to 60% in older ages [7].

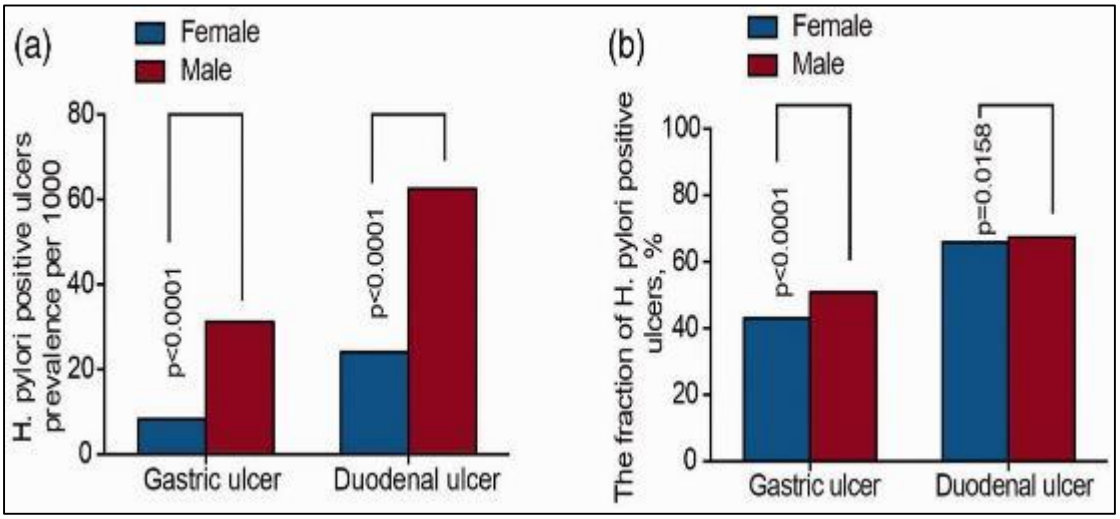

Figure 3 Gastric and Duodenal Ulcer

3. Pathophysiology

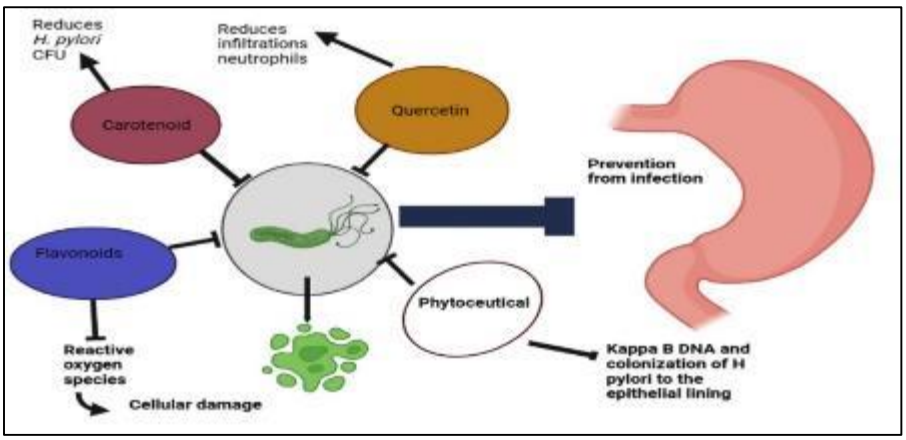

Figure 4 Transient acute Gastritis

Most bacteria are killed in hostile environment of gastric lumen. *H pylori* proliferates in mucus layer over epithelium and is noy cleared by host immuneresponse. Pathophysiology of H pylori infection and its eventual clinical outcome is a complex interaction between the host and the bacterium [8]. *H pylori* survives and grows there because of a variety of virulence factors that contribute to gastric inflammation, after gastric acid production and causes tissue destruction. Flagella- allows penetration of H pylori into gastric mucous layer. Adhesins- mediate binding to host cells.Localized tissue damage mediated by: Mucinases and phospholipases –disruptgastric mucus.Vacuolating cytotoxin –induces vacuolation in epithelial calls that results in epithelial cell damage [9].The ammonium ion as a result of the urease activity, and the production of the phospholipase, which contribute to the formation of a poor quality mucus barrier.Alteration of gastric physiology with enhanced acid production.By stander damage is caused by release of free radicals from the granulocytes.Autoimmunity- autoantibodies are induced by helicobacter that kill the acid –secreating parietal cells. Alteration of the balance of the cell division and apoptosis. Colonization of stomach or duodenum can result in chronic gastritis. Inflammation stimulate more production of gastric acid. This leads to gastric and duodenal ulcers, atrophy and later cancer [10]. Cag A protein was found to contribute to peptic ulcer. Neutrophil-Activating

protein [NAP] recruits neutrophils to gastric mucosa causing inflammation. Free radical production in the gastric lining due to H pylori , increases host call mutation. *H pylori* induces the production of TNF-a and interleukin 8 that leads to host cells mutation [11].

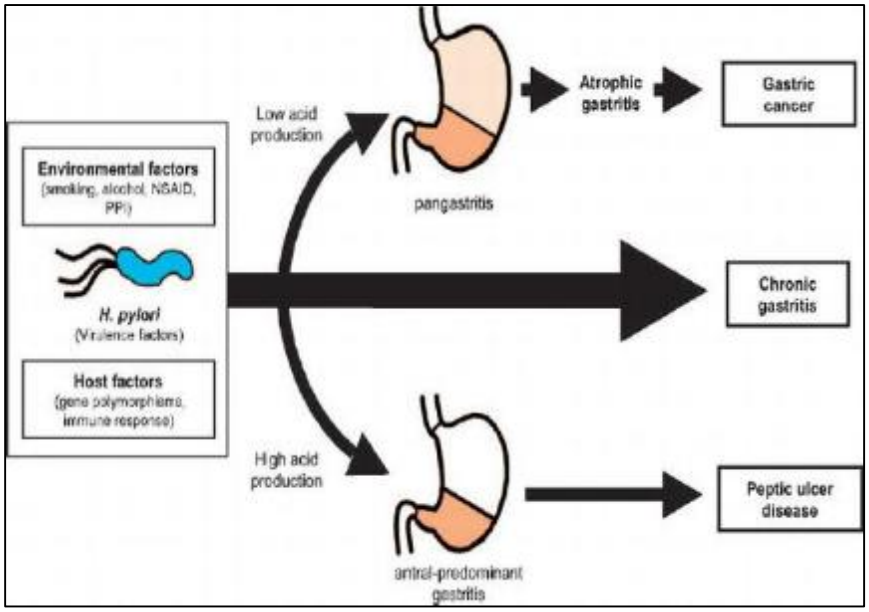

Figure 5 Pangastritis and Antral-Predominant Gastritis

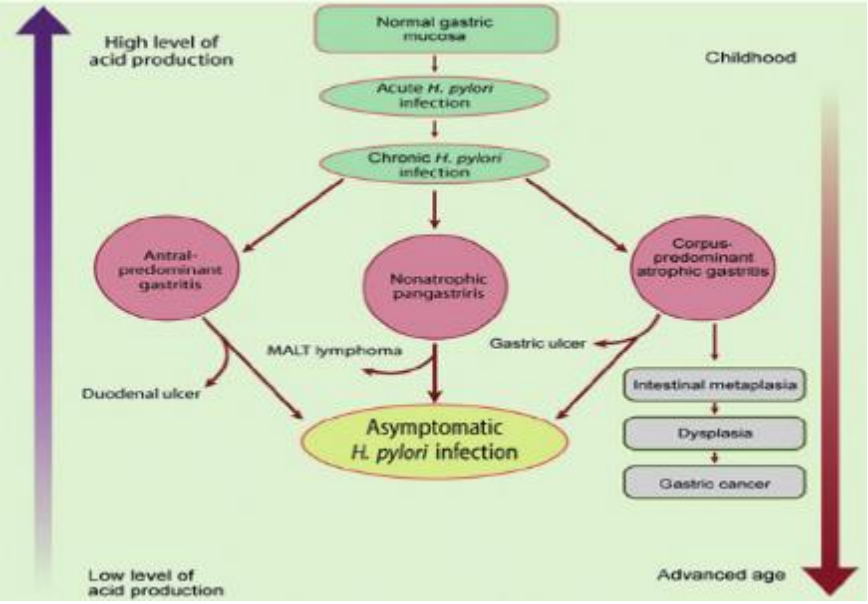

Figure 6 Asymptomatic H. pylori infection

4. Indications [12]

- Peptic ulcer disease.
- Gastric MALT lymphoma.
- Functional dyspepsia after esophagogastroduodenoscopy.
- Idiopathic thrombocytopenic purpura.
- Iron deficiency of unexplained cause [after adequate diagnostic investigation].

In a patient with a history of peptic ulcer disease before the initiation of long –term treatment with acetylsalicylic acid [ASA] or a non steroidal anti – inflammatory drug [NSAID]. Upper gastrointestinal hemorrhage under treatment with ASA or NSAID prophylaxis against gastric carcinoma in a patient risk.

5. Diagnosis [13]

Stool Antigen Test: This IS the most common stool test to detect H. pylori. The test looks for proteins [antigens] associated with H. pylori infection.

Stool Pcr Test: The test can also identify mutations that may be resistant to antibiotics used to treat H. pylori. However, this test is more expensive than a stool antigen test may not be available at all medical centers.

6. Testing Considerations [14]

Radiological Diagnosis: Barium x-ray or upper GI series is a widely used for diagnosis Barium x-ray is difficult to analysis and less sensitive and accurate.

Laboratory test:

- Non invasive urea breath test.
- Patient with refractory or recurrent peptic ulcermay have underlying H pylori infection,histopathology investigation .
- Serologic test for detecting H pylori [levels of IgG and IgA] ELISA test.
- Stool antigen test for non-invasive detecting the presence of H pylori
- Endoscopic diagnosis.
- Rapid urease test.

7. Treatment [15]

Proton pump inhibitor- These drugs stop acid being produced in the stomach. Example-omeprazole, esomeprazole, lansoprazole, pantoprazole.

Histamine blockers-cimetidine, ranitidine.

Bismuth subsalicylate- This drug can be used in the treatment it works by coating the ulcer and protecting from stomach[16]. Antibiotics used to kill H.pylori in the stomach such as: Amoxicillin, Clarithromycin, Levofloxacin, Tetracycline. Two antibiotics are generally recommended reduce the risk of treatment failure and antibiotic resistance.

Figure 7 *H. Pylori* Bacteria

Figure 8 Treatments of Stomach Ulcers

8. Prevention [17]

- There is no vaccine or other specific preventive measure H. Pylori
- Do not eat poorly cooked food.
- Avoid unsanitary areas.
- Wash your hands thoroughly.
- Eat in sanitary places.
- Get adequate nutrition.
- Stop interacting with those infected [18].

9. Symptoms [19-21]

- Pain
- Retching
- Vomiting
- Appetite changes
- Nausea
- Weight loss
- Bloating
- Dark blood in stools

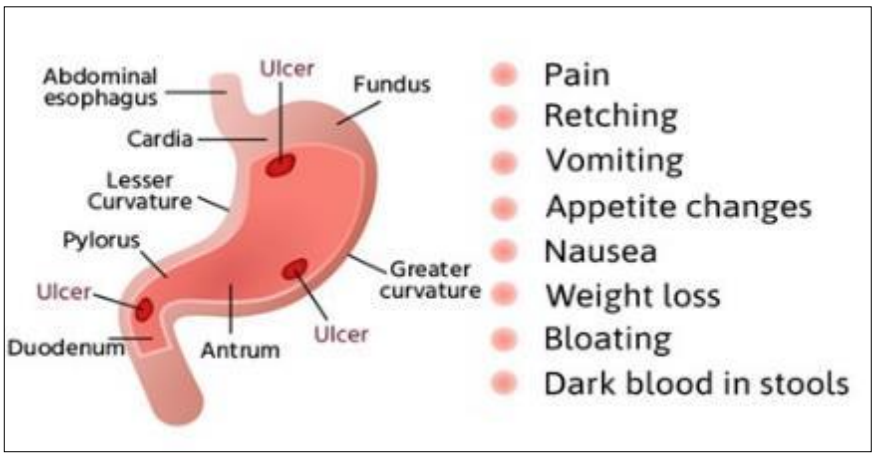

Figure 9 Stomach Ulcer Symptoms

10. Conclusion

Peptic ulcers requiring surgeries are complicated and the patients present as emergency which requires adequate resuscitation.

Delay in presentation, diagnosis and treatment increases morbidity and mortality.

The prevalence of H pylori infection was still high in the chronic dyspeptic patients with peptic ulcer. This may relate to GU, DU, and combined GU and DU. The clinical complications of H pylori infection, such as peptic ulcer disease and gastric ulcer an imbalance in gastric homeostasis.

Compliance with ethical standards

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Disclosure of Conflict of Interest

The authors declare that there is no conflict of interest.

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Reference

[1] Robin George, Bhavana Sree G, Akshaya S, Bhargav C, and Sunil Kumar E. Study on the Prescription Pattern and Antibiogram in the Management of Cellulitis. *Future J. Pharm. Health Sci.* 2023; *3*(2):157–168.

[2] Sonnenberg A. Review article: historic changes of Helicobacter pylori-associated diseases. Aliment Pharmacol Ther. 2013;38:329–342.

[3] Lanas-Gimeno A, Lanas A, et al. Risk of gastrointestinal bleeding during anticoagulant treatment. Expert Opinion on Drug Safety. 2017;16:673–685.

[4] K. Rajini Naidu, B. Jasper Wilson, Md. Sana Safreen Siddiqua Banu, K. Ganga Bhavani, C Madhavi Latha, and Sreenivasulu M. (2023). Review of anaemia in pregnancy. *Future J. Pharm. Health Sci.* 2023; *3*(2):147–156.

[5] Papatheodoridis GV, Archimandritis AJ, et al. Role of Helicobacter pylori eradication in aspirin or non-steroidal anti-inflammatory drug users. World J Gastroenterol. 2005;11:3811–3816.

[6] Grad YH, Lipsitch M, Aiello AE, et al. Secular trends in Helicobacter pylori seroprevalence in adults in the United States: evidence for sustained race/ethnic disparities. Am J Epidemiol. 2012;175:54–59.

[7] Chey WD, Wong BC, et al. Practice Parameters Committee of the American College of G. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. Am J Gastroenterol. 2007;102:1808–1825.

[8] Jhansi Rani M, Veerasamy Hari Bhaskar, and Kothapalli Bannoth Chandra Sekhar. Pharmacognostic and Phytochemical Analysis of Oxalis Latifolia Kunth and their Antiulcer Activity in Albino Rats. *Future J. Pharm. Health Sci.* 2023; *3*(2):193–205.

[9] Huang JQ, Sridhar S, Hunt RH, et al. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet. 2002;359:14–22.

[10] Lanas A, Carrera-Lasfuentes P, Arguedas Y, Garcia S, Bujanda L, Calvet X, et al. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol. 2015;13:906–912. e902.

[11] Somasundaram S, Sigthorsson G, Simpson RJ, Watts J, Jacob M, Tavares IA, et al. Uncoupling of intestinal mitochondrial oxidative phosphorylation and inhibition of cyclooxygenase are required for the development of NSAID-enteropathy in the rat. Aliment Pharmacol Ther. 2000;14:639–650.

[12] Neelima Satrasala, Dwarakanadha Reddy Peram, Chandra Sekhar Kothapalli Bannoth. Nephroprotectivity of Coccinia indica leaves against gentamycin induced nephrotoxicity using in vitro and in vivo models. J. Appl. Pharm. Sci. 2021; 11 (06); 132-139.

[13] Chey WD, Leontiadis GI, Howden CW, Moss SF, et al. ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. Am J Gastroenterol. 2017;112:212–239.

[14] Kanno T, Iijima K, Koike T, Abe Y, Shimada N, Hoshi T, et al. Accommodation in a refugee shelter as a risk factor for peptic ulcer bleeding after the Great East Japan Earthquake: a case-control study of 329 patients. J Gastroenterol. 2015;50:31–40.

[15] Kiss S, Zsikla V, Frank A, Willi N, Cathomas G, et al. Helicobacter-negative gastritis: polymerase chain reaction for Helicobacter DNA is a valuable tool to elucidate the diagnosis. Aliment Pharmacol Ther. 2016;43:924–932.

[16] Sampath Kumar Kalabandi, Battu Karthik Deepak, and Bhoomigani Venkatesh. Clinical Profiling and Therapeutic Outcomes in Diabetic Foot Ulcer. *Int. J. of Clin. Pharm. Med. Sci.*2012;*1*(4), 167–172.

[17] Lanas A, Garcia-Rodriguez LA, Polo-Tomas M, Ponce M, Quintero E, Perez-Aisa MA, et al. The changing face of hospitalisation due to gastrointestinal bleeding and perforation. Aliment Pharmacol Ther. 2011;33:585–591.

[18] Stanley AJ, Dalton HR, Blatchford O, Ashley D, Mowat C, Cahill A, et al. Multicentre comparison of the Glasgow Blatchford and Rockall Scores in the prediction of clinical end-points after upper gastrointestinal haemorrhage. Aliment Pharmacol Ther. 2011;34:470–475.

[19] Praveen Gujjula, Shaganti Gayathri, Potlapalli Sai Sushma, and Pallati Vaishnavi Netha. (2022). Herbal Remedies as a Treatment of Breast Cancer. *Int. J. Exp. Biomed. Res.*, *1*(2), 45–55.

[20] Gomathi Periyasamy, Saraswathi Kaspa, Rathnakar Cherukupally, Srikala Rajala, Mutcherla Pradeep Kumar. Anti-Ulcerogenic Activity Of Novel Pyrazoline Analog 1-(2,4-Dintrophenyl)-3-(4-Hydroxy Phenyl)-5-(2- Furyl)-2-Pyrazoline. Journal of Pharmaceutical Negative Results. 13, 1647-1656.

[21] Jakke Vyshnavi, Yerikala Ramesh, P Venugopalaiah, and Y Prapurna Chandra. (2023). 3D printing technology in pharmaceutical science. *International Journal of Pharmacometrics and Integrated Biosciences*, *8*(2), 5-17. https://doi.org/10.26452/ijpib.v8i4.1504