

REVIEW ARTICLE



Comparative Study between H. Pylori Induced and NSAIDs Induced Peptic Ulcer

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Abstract

The use of Non-steroidal anti-inflammatory drugs as well as the infecting agent H Pylori has been attributed as the prominent common etiological factor for peptic ulcers in patients. Distinguishing proof of Helicobacter pylori as the essential etiologic factor in the improvement of peptic ulcer illness and the perception that the frequency of H. pylori increments with age have brought up the issue of a potential synergistic connection between the presence of H. pylori contamination and NSAID use in the improvement of treatment in gastroenterology. Both H.Pylori and NSAIDs have, nevertheless, been appeared to affect the creation rate and the nature of gastric cyclic AMP, the bodily fluid layer, mucosal prostaglandins, blood stream, and platelet-activating factor. Therefore, it is necessary to determine the risk factors such as age and history of peptic ulcers of the patient prior to prescribing. A co-prescription may be important to reduce the risk of peptic ulcers in patients of high risk. Since H.Pylori infection remains the world's most common chronic bacterial infection, it has been suggested that the establishment of a synergistic or additive effect of H.Pylori infection and NSAID use in the development of peptic ulcer is of great clinical importance as eradication of the bacterium is likely to reduce the risk of upper gastrointestinal complications in infected NSAID users. The prevention and overcoming of NSAIDS induced peptic ulcer and H Pylori induced peptic ulcer is embedded in the thorough understanding and assessment of pathophysiology and other underlying causes in each individual patient. There are wide range of studies that emphasize on the various methods of overcoming these conditions as well as understanding the co factors for the risk of ulcer. The main aim of the treatment is to protect the gastric mucosal layer from further eroding away and heal the mucosal ulcer as soon as possible to avoid further complications.

Keywords: H.Pylori, NSAIDs, Peptic ulcer, Comparison

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1 | INTRODUCTION

Both infection with *Helicobacter pylori* and use of nonsteroidal anti-inflammatory drugs (NSAIDs) may result in gastritis and ulcers. *H. pylori* has been identified as a significant etiologic considering the event of ulceration disease; however, its relationship to NSAID-associated toxicity is a less well characterized. Several studies have suggested that NSAID use does not increase susceptibility to *H. pylori*, and therefore the converse has been suggested, namely, that *H. pylori* does not exacerbate NSAID-associated injury. *H. pylori* it may stimulate production of gastric prostaglandins, which can have a task in ulcer healing... More carefully controlled studies may be better able to elucidate the individual and synergistic mechanisms involved in ulceration induced by *H. pylori* and NSAIDs. Distinguishing proof of *Helicobacter pylori* as the essential etiologic factor in the improvement of peptic ulcer illness and the perception that the frequency of *H. pylori* too increments with age have brought up the issue of a potential synergistic connection between the presence of *H. pylori* contamination and NSAID use in the improvement of disease. *H. pylori* is related with a constant, histologic gastritis, utilization of NSAIDs offers ascend to a receptive or synthetic gastritis that is additionally seen with different medications or under the state of bile reflux. This responsive gastritis might be histologically recognized from that brought about by *H. pylori* by the presence of foveolar hyperplasia and muscle strands in the lamina propriety, just as edema and vasodilation. In addition, 0including NSAID clients who were additionally certain for *H. pylori*, it was shown that the sorts of gastritis can emerge from their individual causes autonomously of the presence of the other reason, that the two kinds bring about ulceration, and that there doesn't give off an impression of being worsening of histologic gastritis by NSAIDs (1).

H Pylori Induced Peptic Ulcer

Helicobacter pylori is the major causal factor in ongoing gastritis. Its obtaining prompts a constant, normally deep rooted, irritation of the gastric mucosa, which may step by step advance to decay (with intestinal metaplasia) in a huge extent of contaminated people. This movement is most likely mul-

tifactorial, being impacted by hereditary or natural components notwithstanding *H. pylori* contamination. The pathogenesis of peptic ulcer and gastric malignant growth is firmly connected with *H. pylori* gastritis and its ensuing atrophic sequelae (atrophic gastritis). *H. pylori*-actuated gastritis is a significant danger factor in the multifactorial etiology of these sicknesses. It causes a course of responses that harm the gastric mucosa and epithelium differently. The particular systems included are generally obscure. Some are presumably bacterium-related responses, which are affected by different destructiveness elements, and others are ramifications of the mucosal aggravation and decay. The danger of peptic ulcer and gastric disease in patients with *H. pylori* gastritis can be summed up as follows: I) the danger of both peptic ulcer and gastric disease is low in people with an ordinary stomach; ii) the danger of peptic ulcer is around multiple times higher and the danger of gastric malignancy roughly twice as high in patients with non-atrophic *H. pylori*-positive gastritis as in those with a typical stomach; iii) these dangers are additionally expanded (twofold to triple) when there is antral decay; though iv) within the sight of corpus decay the danger of gastric disease stays high, yet that of peptic ulcer diminishes slowly to zero with expanding seriousness of corpus decay (2).

Nsaids Induced Peptic Ulcers.

A peptic ulcer is an imperfection in the upper gastrointestinal mucosa that stretches out through the muscularis mucosa into more profound layers of the gut divider. There are two significant danger factors for peptic ulcer infection – *Helicobacter pylori* and non-steroidal calming drugs (NSAIDs). NSAIDs including low-portion anti-inflammatory medicine are the absolute most usually utilized medications. They have great viability and a long history of clin-

Supplementary information The online version of this article (<https://doi.org/10.15520/jcmro.v4i03.400>) contains supplementary material, which is available to authorized users.

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ical use, however can cause peptic ulcers, which may have deadly complications. Given inescapable utilization of NSAIDs and headache medicine, the related gastrointestinal poison levels have significant ramifications for the medical care framework (3).

2 | MECHANISM OF ACTION:

The restorative impacts of NSAIDs are interceded by their restraint of prostanoid biosynthesis. (3) Prostanoid subsidiaries emerge from the change of arachidonic corrosive by cyclo-oxygenase (COX) isoenzymes following cell injury. There are two unmistakable isoforms of COX. COX-1 is available in most of cells including endothelial cells, gastrointestinal epithelium and platelets, and capacities consistently. Interestingly COX-2 is available in a couple of tissues and is instigated by irritation. NSAIDs apply their restorative calming and pain relieving impacts by restraining COX-2. The gastric and renal poison levels of the medications are identified with hindrance of the COX-1 isoform.4, 5 there is a range of COX-1 and COX-2 restraint across the class of NSAIDs (3).

Some of the foremost widely used medications are non-steroidal anti-inflammatory products, including low dosage aspirin. it's linked to gastrointestinal damage. It is necessary to work out the chance factors like age and history of peptic ulcers of the patient before prescribing. A co-prescription is also important to scale back the chance of peptic ulcers in patients of high risk. A daily dose of a proton pump inhibitor is that the simplest method of reducing the chance of ulcers induced by non-steroidal anti-inflammatory drugs. A peptic ulcer is a defect in the upper gastrointestinal mucosa, which spreads to deeper layers of the intestinal wall through the muscles. The *Helicobacter pyloric* and non-steroidal anti-inflammatory medications are two main risk factors for peptic ulcer disease (NSAIDs). Some of the most used medications are NSAIDs, including low-dose aspirin. They are safe and have a long history of clinical usage but may cause peptic ulcers, which can be fatal. The related gastrointestinal toxicities have significant consequences for healthcare systems because of the commonly used use of NSAIDs

and aspirin (1). On eradicating the *H.pylori* without conformation become the mainstay of treatment of PUD resulting in the high ulcer-healing rate. Since *H.Pylori* infection remains the world's most common chronic bacterial infection, it has been suggested that the establishment of a synergistic or additive effect of *H.Pylori* infection and NSAID use in the development of peptic ulcer is of great clinical importance as eradication of the bacterium is likely to reduce the risk of upper gastrointestinal complications in infected NSAID users. A well-recognized complication of NSAID use is Peptic ulcer disease. COX-1 inhibition in the gastrointestinal tract contributes to a decrease in the secretion of prostaglandin and its cytoprotecting impact. This thus increases the sensitivity to mucous injury.6 COX-2 inhibition can also be used in mucous lesions. (3) All NSAIDs cause some level of gastrointestinal harmfulness. Huge pooled information from fake treatment controlled preliminaries show that all assessed NSAIDs including COX-2 inhibitors, diclofenac, ibuprofen and naproxen were related with an expanded danger of gastrointestinal injury.9 However, this danger differs between the medications. The overall danger of upper gastrointestinal difficulties for aceclofenac, celecoxib and ibuprofen is low (<2). Diclofenac, meloxicam and ketoprofen are middle (2–4) while naproxen, indomethacin and diflunisal have a higher relative danger (4–5). The most elevated pooled relative danger is related with piroxicam (7.4) and ketorolac (11.5)^{three}.

Medications with more prominent selectivity for COX-2 than COX-1 ought to have less gastrointestinal harmfulness. Enormous pooled information showed that the anticipated outright yearly danger of upper gastrointestinal complexities was lower for COX-2 inhibitors than naproxen and ibuprofen. However, COX-2 inhibitors are related with an expanded danger of cardiovascular occasions. Inhibition of the COX-1 isoform are in connection to gastric and renal toxicities of drugs (4). There is little proof of an expanded danger of cardiovascular confusions with utilization of a low portion of diclofenac. In any case, to stay away from conceivable cardiovascular intricacies the utilization of NSAIDs ought to be at the most minimal conceivable portion and for the briefest time. Once the person stops tak-

ing the drug, NSAID-induced ulcers normally heal. The doctor can suggest taking antacids to neutralize the acid and drugs called H₂-blockers or proton-pump inhibitors to decrease the amount of acid that the stomach produces to assist the healing process and alleviate symptoms in the meantime. Medicines that protect the lining of the stomach aid with healing as well. Bismuth subsalicylate, which coats the entire lining of the stomach, and sucralfate, which sticks to the ulcer and covers it, are examples (5) GI and cardiovascular risk must be balanced if NSAID therapy is needed, and therapy should be administered at the lowest dose possible and for the shortest period of time. NSAID use without gastroprotective co-therapy is deemed acceptable for patients <65 years of age who do not take aspirin and do not have a history of GI. Coxibs or NSAIDs, plus proton pump inhibitor (PPI) or misoprostol, are valid options in patients with GI risk factors but no cardiovascular risk. Coxib plus PPI treatment should be given to patients with a history of ulcer bleeding and should be screened and treated for *Helicobacter pylori* infection. Coxib therapy has greater GI tolerance than conventional NSAIDs, and successful PPI therapy. The key underlying factor in H. pylori-negative ulcers was NSAIDs; several other theories have also been suggested to explain Pylori-negative duodenal ulcer pathogenesis. These includes false negative results due to diagnostic methods, bleeding peptic ulcers, gastric outlet obstruction, perforated peptic ulcers, tobacco use, isolated Hp duodenal colonization, age, PUD history, ethnicity, gastric hypersecretion, genetics, diseases of the duodenal mucosa, *Helicobacter* infection and concomitant disease (5).

The essential problem, which happens after colonization with H. pylori, is ongoing dynamic gastritis. This condition can be seen on the whole H. pylori-positive subjects. The intragastric conveyance and seriousness of this persistent incendiary cycle rely upon an assortment of variables, like qualities of the colonizing strain; have hereditary qualities and invulnerable reaction, diet, and the degree of corrosive creation. H. pylori-actuated ulcer sickness, gastric disease, and lymphoma are largely inconveniences of this ongoing aggravation; ulcer sickness and gastric malignant growth specifically happen in those people and at those locales with the most serious

aggravation (6).

Etiopathogenetic Principles and Peptic Ulcer Disease Classification

Ulceration leads to loss of tissue, breaching the mucosae of muscularis. When ulcers form in the gastroduodenum acid-peptic climate, they are historically called peptic ulcers (PUD). The ulcers in a balanced gastroduodenal mucosa never develop spontaneously. Ulceration is the ultimate result of an imbalance between defensive mucosa-protective factors and aggressive injurious factors. Heavy acid and high proteolytic (pepsin) activity in gastric secretions are the dominant aggressors. The dominant defenders are the phospholipid surfactant layer, which protects the gel of mucus bicarbonate, the layer of mucus bicarbonate protecting the epithelium, the close junctional structures between the epithelial cells, reducing the permeability of protons, and the peptides of epithelial trefoil, leading to injury healing. Acid-peptic hostility was initially considered the overwhelming cause of PUD, backed by Schwartz's groundbreaking work, launching the 'no acid, no ulcer' dictum. This resulted in universal therapy against intragastric acidity, which also interacted with peptic activity at pH >four. The treatment series went from massive doses of antacids to antagonists of the H₂ receptor and eventually to inhibitors of the proton pump (PPIs). The longer the intragastric pH was >3, the faster healing of the ulcer was observed. Unfortunately, after stopping treatment, ulcers frequently recurred, requiring preventive therapy to keep the ulcers cured and to eliminate the need for surgery (vagotomy, partial gastric resection). Later on, the focus gradually moved to the weakening/failure of the protective factors, increasing the vulnerability of the luminal secretions of the gastroduodenal mucosa. Numerous leading harmful mechanisms jeopardize the integrity of the mucosa. These include infections, particularly *Helicobacter pylori*, drug-induced injury, especially acetylsalicylic acid (ASA) and non-steroidal anti-inflammatory drugs (NSAIDs), physicochemical and caustic injury, vascular disorders, perfusion intervention, etc. Currently the leading cause of PUD is H. pylori infection. Pylori-induced PPI is rapidly disappearing within the Western world, in contrast

to drug-induced ulcer disease and what is called idiopathic PUD. Partial prophylaxis of ASA/NSAID-induced ulceration is feasible with PPI maintenance therapy, but novel ways to strengthen the mucosal defense are urgently awaited (7).

Similarity between H Pylori and Nsaids induced peptic ulcer

Helicobacter arch and non-steroidal calming drugs (NSAIDs) are equipped for meddling with different defensive instruments in the gastroduodenal mucosa. Though NSAIDs are perceived for their corrosive animating action, the impact of H pylori on gastric corrosive discharge remains exceptionally theoretical notwithstanding its relationship with hypergastrinaemia. Both H Pylori and NSAIDs have, be that as it may, been appeared to influence the creation rate and the nature of gastric cyclic AMP, the bodily fluid layer, mucosal prostaglandins, blood stream, and platelet-activating factor. Helicobacter pylori and NSAIDs are most likely the commonest known exogenous elements in the aetiology of peptic ulcer infection (8).

SIMILARITIES BETWEEN NSAIDS INDUCED PEPTIC ULCER AND H PYLORI INDUCED PEPTIC ULCER

- Helicobacter and NSAIDs are equipped for meddling with different defensive instrument in the gastroduodenal mucosa.
- Though NSAIDs are perceived for their corrosive animating action, the impact of H Pylori on gastric corrosive discharge remains exceptionally theoretical notwithstanding its relationship with hypergastrinemia
- Both have impact on production and nature of gastric cyclic AMP, the fluid layer, mucosal prostaglandins, blood streams and platelet activating factor.
- H Pylori and NSAIDs are most likely the commonest known exogenous elements in etiology of peptic ulcer infection

FIGURE 1:

The Relationship between Helicobacter Pylori and Nonsteroidal Anti-Inflammatory Drugs

In a forthcoming report by Kim and Graham, there was no critical contrast between the level of H. pylori-positive long haul NSAID clients who created gastroduodenal ulcers (half) and the level of

H. pylori-negative long haul NSAID clients who created ulcers (half). In spite of the fact that serology was the technique for H. pylori examination, on the off chance that anything the utilization of this technique may as a matter of fact have overestimated the H. pylori-positive populations. The lone ongoing investigation that shows a positive connection between H. pylori and expanded danger of NSAID-incited ulcers depends on annihilation of H. pylori utilizing triple treatment: bismuth subcitrate, antibiotic medication, and metronidazole (9).

H. Pylori and Nsaids Both Are Risk Factors for Ulcer Complications

Ulcer entanglements, particularly dying, are the major reason for horribleness and mortality in patients with peptic ulcer infection. A few investigations have tended to the inquiry of whether H. pylori, NSAIDs, or the mix presents a more serious danger for upper GI drains. The majority of these investigations showed that H. pylori and NSAIDs are hazard factors for upper GI seeping, there is still no agreement on whether H. pylori worsens NSAID-related drains. Most investigations have shown that H. pylori and NSAIDs are autonomous danger factors that do not appear to have an added substance or synergistic impact (10).

Two studies have addressed the question of whether H.pylori eradication is beneficial to NSAID users. The amoxicillin and omeprazole together in long-term NSAID users with H. pylori and determined that the presence of H. pylori did not significantly affect the rate of ulcer healing. recession of omeprazole therapy after 6 months and the ulcer healing, there was a quantitatively higher rate (not significant) of ulcer recurrence in an H. pylori-positive group (46%) compared with the H. pylori-negative (27%) or H. pylori- eradicated (31%) groups. The data not only give information about the combination of H. pylori and NSAIDs may be more damaging to the gastric mucosa than NSAIDs alone, but also indirectly suggest that H.pylori eradication may be of benefit in patients requiring NSAIDs (11).

Influence of Nsaids and H Pylori on Gastric Acid Secretion

Increasing basal and maximally stimulated gastric acid were found to be NSAIDs Secretion.They tend

to bypass the H₂ and muscarinic receptors at a locus between the adenylate cyclase activation catalytic subunit and the proton pump and interact with secretagogues. It was also found that the capacity for secretagogue-stimulated acid secretion by non-salicylated NSAIDs was calcium-dependent. In the case of H. pylori, the condition is not so well described. It was hypothesized that H. pylori could increase the parietal cell mass that is characteristic of patients with duodenal ulcers due to its association with hypergastrinemia. The evidence for a rise in the secretion of gastric acid by H. pylori was missing. Hence, there is a consensus that hypochlorhydria is caused by acute exposure to H. pylori. In case of Hypergastrinaemia recent evidence suggests that H. pylori-linked peptic ulcer may not be specifically related to the Parietal cell function, number of antral G cells or operation of urease of the bacterium's urease activity. NSAIDs (indomethacin and ibuprofen specifically) were found to increment basal and maximally invigorated gastric corrosive secretion; they appear to sidestep the H₂ and muscarinic receptors and connect with secretagogues at a locus between the synergist subunit of adenylate cyclase enactment and the proton pump (4). The potentiation of secretagogue animated corrosive discharge by non-salicylate NSAIDs has likewise been discovered to be subject to calcium. The capacity to change the attributes of the gastric bodily fluid layer is basic to the two NSAIDs and H. pylori. Headache medicine and indomethacin were found to hinder bodily fluid emission. Ibuprofen can likewise expand pepsin intervened proteolysis of bodily fluid, decline bodily fluid consistency, and increment the porousness of bodily fluid to hydrogen particle. Indomethacin was appeared to hinder dynamic bicarbonate discharge by the gastric mucosa. It was likewise recommended that NSAIDs could cause disturbance of the gastric mucosal boundary, which thus permits back dispersion of hydrogen particle with its harming results (8)⁸.

3 | NSAIDS-RELATED GI DAMAGE:

From an endoscopic perspective, NSAIDs created a wide scope of injuries incorporating petechial and

disintegrations with minimal clinical outcomes to more genuine dangerous occasions. From a clinical perspective, NSAID-related upper GI unfriendly occasions could be sorted in various kinds relying upon the ramifications for the patients: Symptoms like dyspepsia, sickness, retching, stomach torment and acid reflux are the most successive side GI impacts related with NSAID admission, and can be available in up to 40% of NSAIDs client (12).

Low-Dose Aspirin Related GI Damage:

Low portion ASA clients can likewise create mucosal injury through the entire GI parcel even at exceptionally low dosages. In any case, low portion ASA related mucosal sores grew all the more every now and again in upper GI lot. This mucosal harm incorporates petechiae, ecchymosis, disintegrations and ulcers. Endoscopically controlled examinations have shown that the commonness of disintegrations in gastroduodenal mucosa in low portion ASA clients is about 60%. Low-portion ASA use was liable for somewhere in the range of 8.2% and 12.2%, everything being equal, and passings (12).

H. Pylori Infection In Peptic Ulcer Disease:

H. pylori is perhaps the most well known persistent bacterial contaminations in people, with practically half of the total populace tainted. Albeit most contaminated patients stay asymptomatic, H. pylori contamination inclines to peptic ulcer sickness (PUD), gastric carcinoma and mucosa-related lymphoid tissue lymphoma. PUD can prompt genuine complexities including intense upper GI draining and hole. The mortality from these complexities ranges somewhere in the range of 4% and 30%.¹² Helicobacter pylori and nonsteroidal calming drugs (NSAIDs) cause most peptic ulcer sickness. Since the two elements are exceptionally pervasive, characterizing the exact connection between H. pylori and NSAIDs is significant for hypothetical and functional reasons (13).

How To Overcome Nsaid Induced Peptic Ulcer And H Pylori Induced Peptic Ulcer

The prevention and overcoming of NSAIDs induced peptic ulcer and H. Pylori Induced peptic ulcer is embedded in the thorough understanding and assessment of pathophysiology and other underlying

causes in each individual patient. There are wide range of studies that emphasize on the various methods of overcoming these conditions as well as understanding the co factors for the risk of ulcer. The main aim of the treatment is to protect the gastric protective layer from further eroding away and heal the mucosal ulcer as soon as possible to avoid further complications.

Nsaids Induced Peptic Ulcer

NSAIDS are one of the major drug prescribed for many inflammatory conditions including Rheumatoid Arthritis, Osteoporosis and in most cases in Surgery. Most of the inflammatory conditions requires chronic consumption of NSAIDS, which increases the risk of Peptic ulcer in patients. The first and foremost treatment option for the treatment of NSAIDS induced peptic ulcer is stopping the use of NSAIDS or switching to a lower dose. This is also accompanied by discontinuing other drugs such as Anticoagulants, corticosteroids and bisphosphonates if any. There are surgical and medical treatment available for the treatment of NSAIDS Induced peptic ulcer. The various option include ant secretory drugs such as Proton pump inhibitors, H₂ – receptor blockers, anti-secretory agents, gastro protective agents and Prostaglandin analogs as well as some chemotherapeutic agents such as nitroimidazoles. It is also notable that the Histamine 2 blockers have taken a wide intriguing place over proton pump inhibitors as they have enhanced functionality of greater healing as well as efficacy too. They are also having notable effect in increasing of pH in stomach; they create conditions to act for antibiotics. A prophylactic agent used is Misoprostol, which is a prostaglandin analogue (14).

In case of NSAID- or aspirin-associated peptic ulcers, ulcers heal 85% with 6–8 weeks of PPI therapy. However lesion healing continues but will be delayed with continued NSAIDS use. Anti-secretory therapy is started for hindrance of peptic ulcer in patients on Aspirin. PPIs are much more effective than alternative agents are though PPIs, H₂ blockers, sucralfate, and misoprostol will all be thought of to treat NSAID-associated peptic ulcer (15). Sucralfate medication is effective for treating NSAID-associated small intestine ulcers however not for the

treatment or hindrance of NSAID-associated internal organ ulcers. Additionally to its poor effectiveness, misoprostol is commonly restricted by its aspect side impact profile, which incorporates effects like epithelial duct upset All internal organ ulcers need repeat scrutiny in six to eight weeks for healing update. If a peptic ulceration is not well, biopsies should be taken at time of repeat scrutiny to rule out internal organ cancer. For refractory ulcers, doubling the PPI dose is counseled for 6 to eight weeks, though the proof supporting this is often weak. Moreover, evaluating for false-negative H. pylori testing (via serology), malignancies, infections, Crohn's sickness, vasculitis, higher abdominal actinotherapy, cocaine use, and syndrome ought to be thought of for ulcers that are treated fittingly and haven't well (14).

H-Pylori Induced Peptic Ulcer

The treatment of H pylori infection itself helps in betterment of the disease condition as well as the faster healing of the mucosal ulcer in case of H pylori induced ulcer.

For the treatment of H pylori induced peptic could be a triple program comprising two antibiotics including Clarithromycin and Metronidazole or Amoxicillin and a proton pump inhibitor usually the pantoprazole. The duration of this regimen is recommended for 7 to 14 days (15). The synergistic action of Antibiotic and proton pump inhibitor plays a key role in eradicating the infection.

Multiple treatment regimen for H. Pylori associated peptic ulcer is usually initiated with assessing whether the patient is allergic to the Penicillin. If the patient is found to be having penicillin therapy then rescue therapy like levofloxacin with or without bismuth along with clarithromycin and proton pump inhibitor after culture susceptibility testing is received (14).

The selected antibiotic is needed to be considered for the development of antibiotic resistance. The intense study on the antibiotic resistance should also be studied for better patient outcome as well for reducing the extended hospital stay, increased cost burden as well as for preventing complications in the treatment. In case the Clarithromycin resistance is identified to be greater than 15%, then it is recommended to stop the Clarithromycin based regimen (15). In such cases

of resistance, 14 days of treatment with bismuth containing quadruple therapy with a tetracycline and imidazole along with a proton pump inhibitor is preferred as first line treatment (16).

Quadruple therapy is another option with Bismuth and completely different antibiotics employed if the first line therapy fails. In case of refractory peptic ulcer, a peptic ulcer which is one that does not heal even though 8- 12 weeks of proton pump inhibitor therapy has been initiated, there is suggested surgical treatment (7). Other indication for surgical treatment are Non adherence, complication risk at high stake like gastric cancer as well as unresponsiveness to treatment that is initiated. Vagotomy or partial gastrectomy are the two major options for surgical treatment (15).

The confirmation test of h pylori eradication should be done after 4 weeks of completion of treatment. Second line medical care ought to be prescribed if a primary line program fails or clarithromycin resistance has been developed and reported. Moreover, susceptibleness testing ought to be thought of when two treatment failures or when one treatment failure once examination is performed (for different reasons like follow from viscus ulcer). If culture for H. pylori is not accessible to judge for resistance or when three suggested treatments have unsuccessful, rifabutin-based triple medical care (PPI, rifabutin, and amoxicillin) for ten days are often thought of. If symptoms don't improve when H. pylori demolition, examination ought to be pursued if not already performed. (1) The cost burden should be reduced in patients health care scheme and if tetracycline are costly and less available then doxycycline can be prescribed, though the data have been mixed. (1) In 80-90% of cases these therapeutic interventions fixates eradications completely. Side effects during eradication treatments occur quite rarely (from 15 to 30%) (17–21).

4 | CONCLUSION

Several studies have suggested that NSAID use does not increase susceptibility to H. pylori, and therefore the converse has been suggested, namely, that H.

pylori does not exacerbate NSAID-associated injury. More carefully controlled studies may be better able to elucidate the individual and synergistic mechanisms involved in ulceration induced by H. pylori and NSAIDs. Distinguishing proof of Helicobacter pylori as the essential etiologic factor in the improvement of peptic ulcer illness and the perception that the frequency of H. pylori too increments with age have brought up the issue of a potential synergistic connection between the presence of H. pylori contamination and NSAID use in the improvement of gastropathy. H. pylori is related with a constant, histologic gastritis, utilization of NSAIDs offers ascend to a receptive or synthetic gastritis that is additionally seen with different medications or under the state of bile reflux. This responsive gastritis might be histologically recognized from that brought about by H. pylori by the presence of foveolar hyperplasia and muscle strands in the lamina propria, just as edema and vasodilation. In considers including NSAID clients who were additionally certain for H. pylori, it was shown that the sorts of gastritis can emerge from their individual causes autonomously of the presence of the other reason, that the two kinds bring about ulceration, and that there doesn't give off an impression of being worsening of histologic gastritis by NSAIDs.

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How to cite this article: Jose D.A.M., Surendran D.G., Ajayakumar D.K., Kunjumon D.S., Mathew D.L.A., O D.K., Varghese D.K.K., K M D.A. Comparative Study between H. Pylori Induced and NSAIDs Induced Peptic Ulcer. *Journal of Current Medical Research and Opinion*. 2021;842–850. <https://doi.org/10.15520/jcmro.v4i03.400>