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ROLE OF PANTOPRAZOLE IN MANAGEMENT OF PEPTIC ULCER DISEASE : A REVIEW

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ABSTRACT :

Peptic ulcer disease (PUD) refers to mucosal erosions equal to or greater than 0.5 cm in the stomach (gastric ulcer) or the first part of the small intestine (duodenal ulcer). It is commonly caused by Helicobacter pylori infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs). This review outlines the current understanding of the epidemiology, pathogenesis, clinical presentation, diagnosis, and management strategies for PUD, along with emerging trends and research.

Keywords : Peptic ulcer disease , NSAIDs , H. Pylori Bacteria.

Introduction:

In conclusion, peptic ulcer disease is a common gastrointestinal condition primarily caused by Helicobacter pylori infection and NSAID use, though genetic and environmental factors also contribute. Its prevalence has declined due to improved hygiene and effective treatments. While most ulcers are linked to known risk factors, idiopathic ulcers still pose diagnostic and therapeutic challenges. Complications such as gastrointestinal bleeding are more likely in individuals taking NSAIDs, aspirin, or certain drug combinations. Emerging evidence also points to psychological stress and other less common causes. Understanding the multifactorial nature of peptic ulcers is crucial for accurate diagnosis, prevention, and management

Causes of peptic ulcer :

Peptic ulcers are mainly caused by H. pylori infection and NSAID use, with other factors like systemic diseases, stress, and smoking contributing to their development and complications. Although many carry H. pylori, only a fraction develop ulcers due to bacterial virulence and host factors. Eradication of H. pylori significantly lowers recurrence, highlighting its critical role

NSAID and peptic ulcer:

NSAIDs are a major cause of peptic ulcers, especially in H. pylori-negative individuals, due to their inhibition of protective prostaglandins and direct mucosal damage. The risk is compounded when H. pylori infection is also present. Long-term NSAID use, particularly in the elderly, increases the likelihood of severe complications like perforation. Preventive measures, including proton pump inhibitors and misoprostol, are effective in reducing ulcer risk and improving patient outcomes. and the importance of early detection, effective treatment, and risk factor management.

Sign and symptoms:

Peptic ulcers commonly present with epigastric pain related to meals, bloating, nausea, and changes in appetite or weight. Alarming signs like hematemesis, melena, or severe pain may indicate bleeding or perforation. Risk increases with NSAID or glucocorticoid use and a history of GERD. For individuals over 45 with persistent symptoms, early evaluation with esophagogastroduodenoscopy is crucial for timely diagnosis and management of potential complications.

Pathophysiology of H. Pylori ulcer :

The pathophysiology of H. pylori-related duodenal ulcers involves more than just acid hypersecretion. An imbalance between acid load and the duodenum's neutralizing capacity is central. H. pylori alters gastric acid regulation through increased acid output, impaired somatostatin-mediated gastrin inhibition, and inflammation-induced cytokine release. Though theories like ammonia's effect on D cells exist, inflammation-driven changes in hormone regulation likely play a key role in ulcer formation.

Duodenal ulcer formation involves impaired bicarbonate secretion, likely due to H. pylori infection, though the exact mechanism remains unclear. Gastric metaplasia, induced by acid exposure, creates a favorable environment for H. pylori colonization, contributing to ulcer development. While increased

acid output correlates with gastric metaplasia, it alone does not explain ulcer formation. These findings suggest that H. pylori plays a complex, multifactorial role in duodenal ulcer pathogenesis beyond mere acid stimulation.

Gastric ulcer:

Gastric ulcers are closely associated with diffuse or corpus-predominant gastritis, reduced acid secretion, and increased cancer risk, differing from the antrum-predominant gastritis seen in duodenal ulcers. H. pylori colonization patterns are influenced by acid levels, host immune responses, and environmental factors. Genetic predispositions, particularly variations in the IL-1 gene cluster, further amplify inflammatory and acid-suppressive responses, increasing the risk of gastric atrophy, ulceration, and malignancy following H. pylori infection.

Treatment:

The treatment of peptic ulcer disease focuses on ulcer healing, preventing complications, and addressing underlying risk factors such as smoking, alcohol, and NSAID use. Effective management includes eradicating H. pylori infection, using proton pump inhibitors (PPIs) for at least four weeks, and adjusting therapy if symptoms persist. For patients on long-term NSAIDs or aspirin, combining these with PPIs or H2 blockers reduces ulcer risk. Severe symptoms should prompt urgent re-evaluation, and for H. pylori-positive ulcers, targeted antibiotic treatment is crucial for successful eradication.

Studies show that triple therapy is more effective than dual therapy for eradicating H. pylori, though recent meta-analyses indicate no significant difference between quadruple and triple therapy. Sequential therapy, involving the sequential addition of antibiotics, has shown better eradication rates and reduced recurrence. While first-line treatments for H. pylori remain consistent worldwide, eradication is particularly beneficial for chronic NSAID users, though it does not fully prevent NSAID-related ulcer disease.

Pantoprazole is a proton pump inhibitor (PPI) used to reduce stomach acid, effectively treating conditions like GERD, erosive esophagitis, and Zollinger-Ellison syndrome. It works by blocking acid production in the stomach. Chemically, it exhibits both acidic and basic properties and is pH-sensitive, degrading more quickly in lower pH conditions. It is highly soluble in water but poorly soluble in non-polar solvents. With a molecular weight of 383.371 g/mol and a formula of $C_{16}H_{15}F_2N_3O_4S$, Pantoprazole remains stable when stored properly between 15 °C and 30°C. Its high boiling point and density further reflect its chemical stability and pharmaceutical suitability.

Pantoprazole is a proton pump inhibitor used to treat conditions like GERD and Zollinger-Ellison syndrome, with FDA approval. It has a bioavailability of 77%, is extensively metabolized in the liver, and primarily excreted through urine. Pantoprazole's serum half-life is about 1–2 hours, and it binds 98% to serum proteins. Administered orally or intravenously, it demonstrates strong acid suppression with a pKa1 of 3.92 and pKa2 of 8.19. Common side effects include headaches and diarrhea, typically mild and temporary. Long-term use may lead to bone fractures, gut infections, or vitamin B12 deficiency, requiring monitoring during extended therapy.

Pantoprazole is a white to off-white crystalline powder and a racemic substituted benzimidazole that covalently binds to the gastric (H^+,K^+)-ATPase enzyme system to inhibit gastric acid secretion. It undergoes extensive hepatic metabolism (primarily CYP2C19-mediated demethylation/sulfation) without accumulation on repeat dosing and has fewer drug interactions than other proton pump inhibitors. Precautions include hypersensitivity (anaphylaxis), masking of gastric malignancy, osteoporosis-related fractures, and vitamin B_{12} deficiency.

Preformulation studies :

The preformulation study revealed the drug as off-white with a crystalline white color, bitter odor, and a melting point of 30°C. Solubility was evaluated in various solvents. The partition coefficient was determined by dissolving the drug in distilled water and n-octanol, shaking, and separating the phases. The aqueous phase was analyzed at 289 nm using UV spectrophotometry. Concentrations were measured using a standard curve, and the partition coefficient was calculated accordingly.

UV Absorption Maximum Determination :

The UV absorption maximum of pantoprazole was found at 292 nm. A calibration curve was prepared using phosphate buffer (pH 6.8), with concentrations ranging from 2–20 µg/mL, showing linear absorbance at 289 nm. Tablet formulation was optimized by direct compression, involving weighing, screening through #40 mesh, manual mixing, blend lubrication with magnesium stearate and talc, and compression using a 16.35 mm × 60 mm capsule-shaped punch for uniform and consistent tablet production.

Preparation and Optimization of formulation :

Optimization was performed using a 3^2 full factorial design, varying the amounts of crospovidone (X1) and sodium starch glycolate (X2) to study their effects on disintegration time and friability. Tablet hardness was evaluated using a Monsanto or Pfizer tester, with results between 3–6 kg/cm², indicating good mechanical strength. Weight variation was assessed on 20 randomly selected tablets, and all batches complied with standard limits, confirming uniformity and quality of the formulations.

Friability test :

The friability test, conducted using a Roche friabilator at 25 rpm for 4 minutes, showed tablet friability below 1%, indicating excellent mechanical resistance. Dissolution studies were performed in two steps using USP type II apparatus at 100 rpm in hydrochloric acid buffer (pH 1.2) and phosphate buffer (pH 6.8), with drug release measured at 280 nm and 265 nm respectively. The disintegration test was carried out using a disintegration apparatus, where tablets were placed in six tubes with discs, and the time for complete disintegration was recorded, ensuring rapid breakdown and effective tablet performance.
The drug was white to off-white, crystalline, with a bitter odor and a melting point of 30°C. It showed good solubility in water and phosphate buffer (pH 6.8). Tablet hardness ranged from 4–6 kg/cm², ensuring good handling. Disintegration time ranged between 5.36 to 10.6 minutes. Weight variation results confirmed uniformity, and friability ranged between 60%–87%. Overall, the formulation exhibited acceptable physical and mechanical properties for tablet development.

Dissolution Test:

In vitro dissolution studies were conducted for all formulations in two stages using a USP type II apparatus at 100 rpm. The dissolution media used were Hydrochloric acid buffer solution (pH 1.2, 900 ml) and Phosphate buffer (pH 6.8, 900 ml), both maintained at 37°C ± 0.5°C. Drug release was analyzed at various time intervals using a UV-visible spectrophotometer, with measurements taken at 280 nm for the acidic buffer and at 265 nm for the phosphate buffer.

RESULT :

The preformulation and evaluation studies successfully identified suitable excipients for the formulation of pantoprazole floating tablets. Through systematic experimentation, optimal formulations with desirable characteristics such as acceptable friability, hardness, disintegration time, and weight variation were achieved, ensuring high bioavailability. The developed dissolution method was validated, and dissolution studies confirmed consistent drug release profiles with a relative standard deviation (RSD) below 2%, demonstrating excellent reproducibility and reliability across batches. These results highlight the effectiveness of the developed formulation and its potential for further development and scale-up.

CONCLUSION :

In the present study, floating tablets of pantoprazole were successfully formulated using the direct compression method. The developed tablets exhibited improved encapsulation efficiency along with satisfactory floating lag time and floating duration. Various evaluation parameters such as friability, hardness, content uniformity, thickness, weight variation, and disintegration time were assessed, and the results were found to be satisfactory. A dissolution method was developed and validated, and dissolution studies conducted on six batches demonstrated consistent reproducibility from batch to batch. Based on the findings of this study, it can be concluded that the floating tablets of pantoprazole sodium enhance gastric residence time and bioavailability, thereby improving therapeutic efficacy.

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CONFLICT OF INTREST :

The author declares that there in no conflict of intrest related to the preparation or publication of this article.