DOI: https://doi.org/10.53350/pjmhs020241878

LETTER TO THE EDITOR

Helicobacter Pylori and Peptic Ulcer Disease

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Dear Editor,

Peptic ulcer disease causes mucosal ulcers primarily in distal stomach and proximal duodenum, presenting as recurring pain in abdomen, bloating, and is often associated with nausea, vomiting and gastrointestinal bleed. The underlying mechanism involves a defect in mucosal protection and an increased gastric acid secretion causing the damage[1]. Helicobacter Pylori, the use of non-steroidal-anti-inflammatory drugs, smoking and alcohol and are some of the common causative agents. H. Pylori affect about 50% of the population worldwide, especially in underdeveloped nations and is linked to chronic gastritis, B cell mucosa-associated lymphoma and gastric carcinoma[2]. While some etiologies can be managed easily by lifestyle change, elimination of H. Pylori remains essential in preventing and treating gastrointestinal diseases.PAC therapy is a standard eradication strategy which uses proton pump inhibitors in conjunction with two antibiotics for up to 7-14 days.

Liu et al., 2024[3] found that despite an initial cure rate of 90%, the rate of failure for PPI-based prescription varies between 29%-40%owing to increased resistance to antibiotics and unstable inhibition of gastric acid by PPIs, hence alternative therapies may be more effective in such cases. PPIs, if used briefly in recommended doses are well endured by the patients with minimal reported adverse effects. However new literature highlights concern over prolonged use of PPIs. Extended PPI use is associated with deficiency of Vitamin B_{12} , calcium and magnesium with increased risk of osteoporotic fractures, kidney issues in elderly and rarely an intense episode of COVID-19 infection[4]. Long term use is also linked to Clostridium Difficile infection and community-acquired Pneumonia. A survey by Alblooshi et al., 2024[5] revealed that only 10% to 15% of American PPI users knew these adverse effects despite widespread use. PPIs are prodrugs which undergo activation in acidic environment causing delayed onset. Owing to instability in acidic environments, PPIs are produced as enteric-soluble preparations, and their efficacy can be effortlessly affected by food making the time of ingestion a challenge.

To overcome the aforementioned drawbacks, an alternate acid-suppressive medication that is equally effective as PPI or perhaps even better is necessary. Potassium-competitive acid blockers, examples include vonoprazan, keverprazan, revaprazan and tegoprazancan act as a possible substitute that functions by competitively blocking the exchange of H+ and K+ through H+/K+ ATPase transporter and suppress stomach acid secretion[1]. Unlike PPIs, P-CAB induces an abrupt rise in intragastric pH, causing H. Pylori to enter a replicative condition and improves bactericidal effect of antibiotics which were unstable in acidic environment. Liu et al., 2024[3] states, the pooled eradication rate of H. Pylori for PAC therapy is inferior at 77.8%, compared to 89.1% achieved with VAC triple therapy along with a better healing of ulcers as well.

People with certain comorbidities maybe more prone to adverse effects of prolonged use of PPIs and P-CAB should be used particularly in those patients. For example post-menopausal women, who are already at higher risk of acquiring osteoporotic fractures, patients receiving diuretics are more likely to develop hypomagnesemia and in patients receiving metformin, as it may increase the risk of Vitamin B_{12} deficiency when paired with PPI use[5]. On the other hand, P-CAB is also effective against refractory NSAID-induced ulcers unmanageable by traditional PPIs[1].

Conflicts of Interests: I have no conflicts of interest to declare.
Funding: I have no funding sources to declare.
Authors` contributions: Muhammad Tahir conceived the idea for the letter, wrote the initial draft, reviewed the literature and provided critical revisions and contributed to the final version of the letter and approved it for submission.

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The Letter to the Editor may be cited as: Tahir MS: Helicobacter Pylori and Peptic Ulcer Disease. Pak J Med Health Sci, 2024;18(7):38.