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Omeprazole: A Proton Pump Inhibitor for the Management of Acid-Related Diseases

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Abstract: *Omeprazole (OME) is a common medicine used to treat stomach and digestive problems. While it is effective, using it for a long time may increase the risk of stomach cancer.To better understand this, researchers looked at studies published up to August 2019 using PubMed, Scopus, and ScienceDirect. They reviewed 80 clinical studies, 46 lab (in vitro) studies, and 76 animal (in vivo) studies. The results were mixed. Some studies show that taking OME in high or long-term doses (5 to 40 mg/kg) may cause genetic damage, which could lead to cancer. However, OME also has benefits, such as reducing stomach acid, lowering inflammation, and protecting the stomach lining. These effects happen through changes in body chemicals (like COX-2, interleukins, caspases, and BCL-2), improved blood flow, and less damage from white blood cells (neutrophils). Still, several side effects have been reported, especially in clinical settings. These include:Stomach lining damage (atrophic gastritis). Vitamin B12 (cobalamin) deficiency. Body chemical imbalances (homeostasis disorders). Growth of polyps in the stomach. Liver damage (hepatotoxicity).Cell damage (cytotoxicity). DNA damage (genotoxicity). In summary, while Omeprazole can help treat stomach issues, using it for a long time or without medical advice may lead to serious health risks, including genetic damage and a higher cancer risk. So, it's important to use this drug carefully and under medical supervision.*

Keywords: Cobalamin deficiency, Atrophic gastritis, Hepatotoxicity, Apoptosis markers, Anti-inflammatory effects, Self-medication risks

I. INTRODUCTION

Studies show that common stomach problems and the side effects of some medicines can damage our DNA, which may increase the risk of mutations and cancer. One such medicine is omeprazole (OME), used to reduce stomach acid. While it helps treat acid-related issues, it can also change the balance of bacteria in the gut. This may cause problems like poor nutrient absorption, stomach infections, and damage to the stomach lining. These effects happen because the body reacts to lower acid levels by overworking the stomach lining, which can lead to the loss of stomach glands and a condition called atrophic gastritis .When someone has a Helicobacter pylori infection and is also taking OME, the chances of getting atrophic gastritis increase even more. This condition is linked to a higher risk of abnormal cell growth (dysplasia) and stomach cancer.[1]

Even though the causes may vary, one common factor is the presence of harmful molecules called reactive oxygen and nitrogen species (ROS/RNS), along with cancer-related proteins like CagA from H. pylori bacteria .Taking OME for a long time may also damage DNA. Studies have shown that not just OME, but other similar drugs like esomeprazole, lansoprazole, pantoprazole, and rabeprazole can also cause changes in chromosomes.[2]

Because of these concerns, this review aims to explain the current understanding of how OME works, its side effects, and how it might increase the risk of DNA damage and cancer.peptic disorders (APDs) continue to be a common disorder seen by primary care physicians and gastroenterologists and place a heavy burden on healthcare systems . In 2019, 309,381,599 cases were reported in a survey of 206 countries . APD includes gastroesophageal reflux disease (GERD) and erosive ulcers (including gastric, duodenal, and esophageal). GERD is classified into the following three

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categories: (1) nonerosive reflux disease (NERD), (2) erosive esophagitis (EO), and (3) Barrett's esophagitis. The worldwide prevalence of GERD ranges from 7% to 52%, ranges from 8 to 30% in India, and has a high impact on the quality of life . NERD is more common (∼70%) and 10–30% have EO. [3]

The prevalence of duodenal ulcers also varies greatly in different countries as follows: 2.1% in Sweden, 3% in India, 3.9% in Italy, 5.6% in Northern Saudi Arabia, 7.4% in Bangladesh, and 13.3% in China . Standard treatments for APD include the use of PPIs, adjunctive treatments (histamine H2 receptor antagonists, prokinetics, and alginate), surgery, life-style changes, and dietary considerations.[4]

The World Gastroenterology Organization, Chinese, Korean, and Japanese guidelines recommend the first line of treatment to be a PPI given over 4–6 weeks, but there is no consensus of which type of PPI is more effective . PPIs differ in their pKa, bioavailability, peak plasma levels, route of excretion, recommended doses, and level of efficacy . The choice of which individual PPI drug is more effective and safe is still controversial. In addition, whether the efficacy of PPIs differs for the different APD syndromes is rarely directly compared.[5]

Continuing Education Activity

Omeprazole is a proton-pump inhibitor used to manage and treat several conditions, including uncomplicated heartburn, peptic ulcer disease, gastrointestinal reflux disease, Zollinger-Ellison syndrome, multiple endocrine adenomas, systemic mastocytosis, erosive esophagitis, gastric ulcers, and helicobacter pylori infection. This activity reviews the indications, mechanism of action, administration, dosage, contraindications, interactions, and potential adverse effects of omeprazole therapy. It also highlights other key elements of omeprazole therapy in the clinical setting as it relates to the essential points needed by members of an interprofessional team managing the care of patients with peptic ulcers and other related conditions and sequelae.[6]

Objectives:

- Describe the mechanism of action of omeprazole.
- Identify the administration options for omeprazole.
- Outline potential adverse effects associated with omeprazole therapy.
- Review the importance of improving care coordination amongst interprofessional team members to improve outcomes for patients receiving omeprazole.

II. METHODS

Protocol and Registration

The project and protocol for this meta-analysis followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines [22]. The PRISMA checklist is provided in Supporting Information Table 1. The project and protocol were prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO CRD 415397 (April 7, 2023).[7]

Search Strategy

PubMed, Google Scholar, and the China National Knowledge Infrastructure (CNKI) databases were searched (from database inception to March 30, 2023) to identify prospective RCTs or cross-over trials comparing omeprazole to other types of PPIs or placebo. The search strategy for PubMed was: ((((GERD) AND (omeprazole) AND (randomized controlled trial OR cross-over) AND (efficacy) AND NOT (prokinetics) OR NOT (alginate)))). Secondary searches of grey literature included reference lists, authors, reviews, meeting abstracts websites, and https://clinicaltrials.gov for unpublished trials. There were no language restrictions and articles in languages other than English were translated and reviewed.[8]

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Study Selection

Inclusion criteria included randomized, controlled clinical trials (RCTs) with prospective parallel groups or cross-over design with a minimum of two weeks washout period in children or adult subjects with diagnosis of acid/peptic disorder including either GERD, NERD, OE, upper gastrointestinal (GI) ulcers, GI bleeding, and presence or absence of H. pylori.[9] Included interventions are as follows: omeprazole (oral or IV) for at least 4 weeks (later amended to at least one week) compared to other types of PPIs (esomeprazole (ESO), ilaprazole (ILA), lansoprazole (LAN), pantoprazole (PAN), rabeprazole (RAB), or placebo). Study outcome includes a measure of improvement of APD symptoms and/or ulcer/erosion healing.[10]

Exclusion criteria included nonhuman studies, case reports or case series, early phase 1 (safety) or 2 (mechanism of action, dose ranging, formulation, kinetics) studies, validation of measurement tools for APD, no control group, intervention not well-described, no relevant outcomes provided, not a comparison of interest, reviews, meta-analysis, duplicate reports, presence of other disorders with similar symptoms (organic, metabolic, or drug-induced, chronic cough or asthma, simple laryngitis, Zollinger–Ellison syndrome, primary motility disorder, esophageal stricture, Barrett's esophagus, upper GI malignancy or other severe comorbidity), PPI treatment less than 1 week, only non-PPI comparison group (prokinetics, H2 receptor antagonists, surgery, alginates, or potassium-competitive acid blockers), or did not contain original quantitative data.[11]

Data Extraction

Two reviewers (LV and PM) independently screened titles and abstracts of studies identified by the search strategies. Data from all full-text articles were extracted and reviewed independently by two reviewers using a predesigned data extraction form following the standard methods for systematic reviews and meta-analysis . Any disagreements were discussed until resolved.[12]

The data extracted included PICO data: (1) population (age range and country), (2) intervention (type of PPI or controls used, daily doses, formulation, duration, and follow-up times), (3) comparisons (type of control group either placebo or open and unblinded), (4) outcomes, including improvement in APD symptoms, improvement in symptoms scores (frequency scale for symptoms of GERD (FSSG), dyspepsia symptom scores, heartburn scores, symptom index, etc.) and/or ulcer or erosion healing rates, time to ulcer healing, pH > 4 for 24 hours by treatment end, or percent remaining in remission.[13]

Primary Outcomes

Improvement in APD Symptoms for NERD

This outcome was measured as either "overall improvement/cure" and by improvement of specific APD symptoms (heartburn, pain, and nausea). Other potential outcome measures included frequency remaining in remission, pH > 4 for 24 hours at the end of the treatment period, prepost improvement of esophageal pH, improvements in symptom scores (dyspepsia or heartburn and composite laryngeal score), or other visual analogue scales for symptom severity.[14]

Frequency of Ulcer/Erosion Healing for Erosive Disease (DU/PU or EO)

Erosion/ulcer healing has been defined as epithelium or mucosa healed, no ulcer crater by end of treatment, scarring only, presence or absence of inflammation, ulcer size reduced by >50%, or "total effective rate" (which includes frequency of completely healed (with or without inflammation) and ulcer size reduced >50%) but does not include ulcers or erosions that were only improved or had.[15]

Mechanism of action

Omeprazole, a substituted benzimidazole, is a proton pump inhibitor that binds to and irreversibly blocks H+/K+-ATPase, thereby blocking gastric acid secretion. Omeprazole inhibits gastric acid secretion stimulated by any secretagogue, in contrast to H2-receptor antagonists, which only suppress gastric acid production stimulated by histamine. Because it is a weak base, omeprazole accumulates in the acid compartment of the parietal cell; therefore its

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effect persists after the drug is no longer detectable in blood. Omeprazole is inactive at physiological pH and so does not affect ATPase elsewhere in the body.[16]

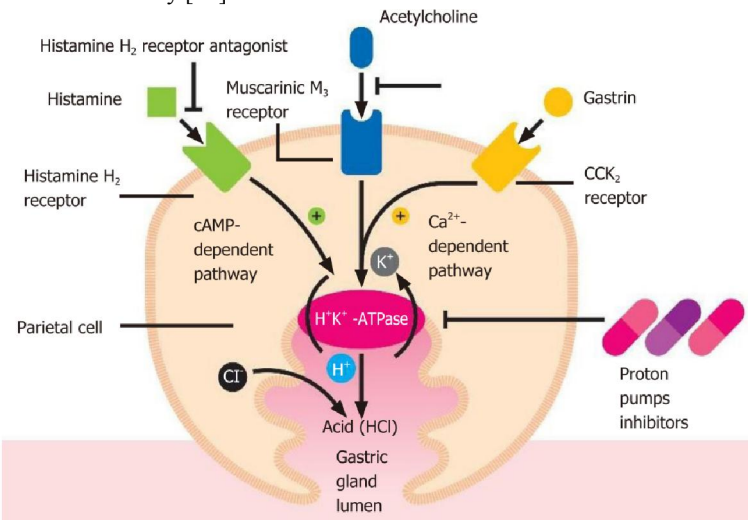

Fig.01: Mechanism of action cellular of Proton Pump inhibitor drug

Omeprazole has a longer duration of action than the H2-antagonists and most recommendations are for once-daily dosing. However, a recent study reported that twice-daily omeprazole maintained gastric lumen pH > 3 throughout a 24-h period, whereas q.24 h dosing did not [17].Whether it is necessary to maintain pH > 3 to achieve effective ulcer healing is not clear. Given the cost and convenience of once-daily dosing, this protocol is likely to remain the most common approach, with more frequent dosing used for refractory cases only.[18]

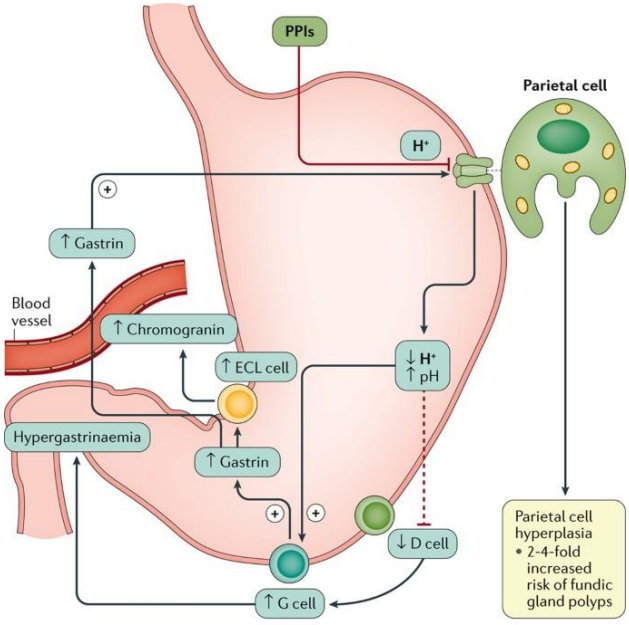

Fig.02: The Regulation of gastric acid secretion and the potential effect of proton pump inhibitors

The antisecretory effects increase with each dose until the drug attains a steady-state inhibition. In dogs, gastric acid output is reduced by about 30% in the first 24 h after an oral dose of 0.7 mg/kg and after five doses gastric acid production is almost completely inhibited.[19]

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Formulations and dose rates

Omeprazole is labeled for equine use in some countries but not, to the authors' knowledge, for small animal use. The drug is rapidly degraded by acid, so is formulated as 20 mg enteric-coated granules in a gelatin capsule. For dogs less than 20 mg, the enteric-coated granules must be repackaged in a gelatin capsule.[20]

Administration

The administration route for omeprazole heavily depends on the diagnosis of a medical condition and the patient's preference. It is available as a delayed-release oral suspension, capsules, tablets, and orally disintegrating tablets.[21]

The recommended oral dose for treating symptomatic gastroesophageal reflux disease absent esophageal lesions is 20 mg daily for up to 4 weeks. However, therapy may extend to 8 weeks if erosive lesions are present.

For H pylori infection, the recommended adult oral regimen is 20 mg plus clarithromycin 500 mg plus amoxicillin 1000 mg plus metronidazole 500 mg, each given twice daily for 14 days. If an ulcer is present on initial diagnosis, then it is recommended to provide an additional 18 days of omeprazole 20 mg once daily. Clinicians should expect antimicrobial resistance; if treatment fails, susceptibility testing should be performed, and the treatment should be adjusted accordingly.[22]

The recommendation for patients with hypersecretory diseases is to start at 60 mg once daily, followed by the individualization of dosage based on the patient's need and clinical response. If the daily dose exceeds 80 mg, they should divide the dosage throughout the day. Long-term treatment with omeprazole is not recommended; eventually, switching to an H2 inhibitor is preferred.[23]

Omeprazole should be ingested 30 to 60 minutes before meals. It may be taken with antacids. When taken twice daily, the first dose should be before breakfast and the second dose before dinner. The capsule and tablet should be swallowed whole, not crushed or chewed. However, it is permissible to open the capsule and mix the contents with one tablespoon of applesauce, soft enough to be swallowed without chewing. The suspension should be left to thicken for two to three minutes, following reconstitution and administration within 30 minutes. Drink with a glass of cool water to ensure the complete swallowing of the pellets.[24]

Omeprazole therapy should be at the lowest dose possible for the shortest duration; physicians have looked into deprescribing proton pump inhibitors if patients are on it long-term. One group recommends deprescribing proton pump inhibitors, meaning reducing the dose, stopping completely, or using "on-demand" dosing in adults who[25]

have completed a minimum of 4 weeks of proton pump inhibitors treatment for heartburn or mild to moderate gastroesophageal reflux disease or esophagitis and who have achieved symptomatic resolution. The clinician should monitor symptoms at four weeks, 12 weeks, and 6 to 12 months. However these recommendations do not apply to patients who have or had Barrett esophagus, severe esophagitis grade C or D, or a documented history of bleeding gastrointestinal ulcers.[26]

Specific Patients Population

Patient with hepatic impairment: As per the manufacturer's label, the bioavailability in patients with hepatic impairment increased by 100 % compared to the intravenous dose due to a decrease in the first-pass effect. Plasma half-life also increased to 3 hours instead of 0.5 to 1 hour. Dose reduction should be considered for patients with hepatic impairment. [27]

Patient with renal impairment: There is no dose adjustment guidance in the manufacturer label for patients with renal impairment or with creatinine clearance ranging between 10 to 62 mL/min/1.73 m2. A slight increase in bioavailability was observed in the patient with renal impairment as a primary route of excretion of omeprazole metabolites; their elimination slowed in proportion to the decreased creatinine clearance.[28]

Pregnant women: Omeprazole crosses the placenta and is pregnancy safety category C.Some PPIs are considered pregnancy category B, with a better safety profile established.

Breastfeeding women: Limited information indicates that maternal omeprazole doses of 20 mg daily would not cause any adverse effects in breastfed infants.[29]

Pediatric patients: The dosage depends on the child's weight for pediatric patients between the ages of 1 and 16.

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Between 5 to 10 kg: 5 mg daily
Between 10 to 20 kg: 10 mg daily
More than 20 kg: 20 mg daily
Geriatric patients: The bioavailability is increased in geriatric patients as the rate of elimination decreases in the geriatric population. There is 76% drug bioavailable when a single dose of 40 mg oral buffered solution is administered to healthy elderly.[30]

Pharmacokinetics

Absorption: Delayed-release Omeprazole capsules contain enteric-coated granules that only release omeprazole once the drug leaves the stomach. Once the drug is released, it is rapidly absorbed. It has an absolute bioavailability of 30% to 40% (20 to 40 mg oral dose), and the peak plasma concentration is attained in 0.5 to 3.5 hours; peak plasma concentration and area under the curve are increased proportionally up to 40 mg. However, due to saturation of the first-pass effect above a 40 mg dose, those increase greater than linear at a dose above 40 mg.[31]
Distribution: The plasma protein binding is approximately 95%.
Metabolism: It is metabolized mainly via the cytochrome P450 system.
Elimination: It is eliminated mainly in urine as an unchanged drug and its metabolites. Its plasma half-life is 0.5 to 1 hour after oral administration in healthy subjects.[32]

Adverse Effects

The following adverse events may occur:

Omeprazole is considered a benign drug; however, the primary adverse effects reported in drug labeling include headache (6.9%), abdominal pain (5.2%), diarrhea (3.7%), nausea (4.0%), vomiting (3.2%), and flatulence (2.7%) in adults. Other than these adverse effects, cough, rash, asthenia, back pain, regurgitation, upper respiratory infection, constipation, and dizziness are also reported in 1 to 2 % of patients.[33]
The major adverse effects in the pediatric population are similar to adults; the most frequent events were reportedly fever and respiratory infections.
Proton pump inhibitor therapy may correlate with an increased risk of Clostridioides difficile (C diff) associated diarrhea.
There are rare reports of hypomagnesemia with prolonged treatment with PPIs.[34]
Avoid concomitant use of omeprazole with St John's wort or rifampin and other CYP450 inducers due to the potential reduction in omeprazole concentration.
There is an increased risk of drug resistance or toxic effects of antiretroviral medicines when used with omeprazole.
Patients on warfarin could experience an increased international normalized ratio resulting in bleeding when used with omeprazole.[35]
Some evidence has shown a diminished antiplatelet activity of clopidogrel due to impaired CYP2C19 function when used in conjunction with 80 mg omeprazole.
According to product labeling, warnings and precautions are advised for patients who develop acute tubulointerstitial nephritis, cyanocobalamin (vitamin B12) deficiency, and cutaneous or systemic lupus erythematosus while on omeprazole.[36]
Long-term and multiple daily dose PPI treatment may have connections with an increased risk for osteoporosis-related fractures of the hip, wrist, or spine. However, newer studies show that long-term PPI use does not correlate with changes in bone mineral density or bone strength that would predispose to increased fracture risk, suggesting this relationship is not causal.[37]

Drug Interactions

Co-administration with amoxicillin:

Combine therapy of omeprazole and amoxicillin may lead to serious fatal hypersensitivity reactions in patients on penicillin therapy. These reactions are more common in patients with a history of hypersensitivity reactions to

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penicillins, cephalosporins, or other allergens. During combined therapy, if such reactions occur, amoxicillin therapy should be discontinued, and appropriate therapy should be implemented.[38]

Co-administration with clarithromycin:

Combine therapy of omeprazole and clarithromycin may increase the plasma levels of omeprazole and clarithromycin.

CYP2C19 Substrates: Omeprazole competitively inhibits CYP2C19, which may increase the plasma concentration of other CYP2C19 substrates like diazepam, proguanil, moclobemide, phenytoin, and warfarin.[39]

Contraindications

Omeprazole is contraindicated in patients with a history of hypersensitivity to the drug or any excipients from the dosage form. Hypersensitivity reactions like anaphylactic shock, angioedema, interstitial nephritis, anaphylaxis, urticaria, and bronchospasm may occur. According to product labeling, omeprazole is contraindicated in patients taking.[40]

Monitoring

The following are indications for monitoring:

When using omeprazole, patients should be monitored for signs and symptoms of gastroesophageal reflux disease and peptic ulcer disease.

Physicians should also monitor for C difficile-associated diarrhea and hypomagnesia when patients are on omeprazole long-term.

Omeprazole is listed in Beers criteria, and care should be taken when used in the geriatric population for more than eight weeks and reassess the need for continuation of treatment.[41]

Monitor INR and prothrombin time in patients using warfarin and omeprazole.

Caution should be exercised when co-administered with CYP2C19 substrates (eg,, clopidogrel, citalopram, cilostazol, phenytoin, diazepam, digoxin).

Toxicity

Omeprazole overdose of up to 2400 mg (120 times higher than the recommended clinical dose) has been reported. The adverse reactions like confusion, drowsiness, tachycardia, blurred vision, headache, dry mouth, nausea, vomiting, diaphoresis, and flushing were seen with overdose. These symptoms were transient, and no serious clinical outcomes were observed with monotherapy. No specific antidote for omeprazole overdosage is known. Dialysis may not be helpful as omeprazole is extensively protein-bound. Symptomatic and supportive care is recommended with an overdose.[42]

Enhancing Healthcare Team Outcomes

Omeprazole was the first proton pump inhibitor discovered in 1979, and it has revolutionized the management of numerous gastrointestinal diseases. Its efficacy compared to new proton pump inhibitors has been studied. One study showed the superiority of esomeprazole for the Japanese population, especially with CYP2C19 polymorphism over omeprazole and other proton pump inhibitors. Studies have shown equivalent efficacy when comparing omeprazole and rabeprazole. Comparative studies with multiple proton pump inhibitors, including omeprazole, have shown greater cost-effectiveness and management of symptoms when using esomeprazole.[43]

Healthcare professionals should avoid empirical prescriptions of omeprazole. Given the number of potential adverse events associated with long-term proton pump inhibitor use, it is recommended that clinicians prescribe the lowest effective dose of omeprazole for the shortest period and monitor the patients to adjust the dose according to their needs. Nursing staff should counsel patients, ensure proper medicine administration, and monitor patients for therapeutic insufficiency. Pharmacists should check for drug-drug interactions, verify the dose, and inform the prescriber of any adverse events. All clinicians, specialists, nursing staff, and pharmacists must collaborate as an interprofessional team and coordinate the treatment plan to enhance patient outcomes using omeprazole.[44]

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Clinical applications

Omeprazole has slightly greater efficacy in promoting ulcer healing in humans than H2-antagonists but is more expensive. Its use in veterinary medicine is usually restricted to refractory ulcers or ulcers associated with gastrinomas or mastocytosis. However, some veterinary gastroenterologists use omeprazole as their first-choice antiulcer drug. Such an approach is supported by the study previously referred to assessing efficacy and duration of gastric acid suppression, by measuring luminal pH, in healthy research dogs; omeprazole and pantoprazole were more effective than either famotidine or ranitidine. Nevertheless, given that the study was in healthy dogs, it is not clear whether the differences noted relate to clinical efficacy.[45]

Excellent long-term clinical outcomes have been reported in humans and dogs with nonresectable gastrinomas treated with omeprazole. Omeprazole has been reported to be useful in dogs in management of severe erosive esophagitis, gastritis or gastric ulcer disease refractory to therapy with H2-receptor antagonists and sucralfate. It has been used successfully to treat severe erosive esophagitis in one cat but had no effect in two similarly affected cats.[46]

One recent study demonstrated that omeprazole has some efficacy in preventing exercise-induced gastritis in racing Alaskan sled dogs. In another study, omeprazole did not reduce mechanically induced gastric ulceration or prevent aspirin-induced gastritis in dogs, although there was a trend that suggested that omeprazole was more effective than cimetidine in this regard. However, a further study demonstrated limited efficacy, in both treating and preventing gastric mucosal lesions, in dogs with acute degenerative disc disease treated with corticosteroids.[47]

III. CONCLUSION

Omeprazole is an effective and safe treatment for acid peptic disorders, including the rapid resolution of GERD symptoms and resolution of erosions and ulcers. Omeprazole was the most cost-effective type of PPI in India. Omeprazole's therapeutic role for patients with acid peptic disorders .Studies on the mechanisms of action of OME are still controversial. As a gastroprotectant agent, it blocks proton pump, activates HSP70 proteins and TGF-β, exerts antioxidant activity, reduces lipid peroxidation, and activates expression of antioxidant defenses, without differentiation of doses and/or concentrations. Additionally, in in vitro and in vivo studies, anti-inflammatory effects of OME have been related to increased gastric flow, increased anti-inflammatory markers (COX-2, IL-10A, and IL-6), and antiapoptotic activity by reducing caspase 3, Bcl-2, mitochondrial calcium, and expression of NTRK2 and GGR1 genes. However, OME adverse effects, especially in vivo, such as changes in bacterial flora, enteric infections, gastric gland destruction, polyp formation, hypomagnesia, hypocalcemia, hyperplasia, intestinal metaplasia, electrolyte deficiency, and immunological component changes, may relate to the consequences of genomic instability. In summary, besides the gastroprotective effects, the adverse effects of OME may be due to its DNA damage capacity by inducing oxidative stress, apoptosis and necrosis, immunological alterations, cell proliferation, autophagy,

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