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OPTIMIZING THERAPY: BENEFITS OF COMBINING OMEPRAZOLE WITH CALCIUM CARBONATE

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Abstract
Omeprazole is a proton pump inhibitor that irreversibly inhibits the gastric H^+/K^+ ATPase pump, suppressing acid secretion. Its effects begin within 1 hour, peak at 3 hours, and last up to 72 hours. It's administered in delayed-release forms to protect it from stomach acid, with 30–40% bioavailability. Omeprazole is metabolized in the liver via CYP2C19 and CYP3A4 and has a plasma half-life of 0.5–1 hour. It's used at 80 mg daily for GERD (up to 8 weeks) and 20 mg twice daily in H. pylori eradication therapy. Calcium carbonate acts as an antacid, neutralizing gastric acid by releasing calcium and carbonate ions, and also serves as a calcium supplement and phosphate binder. It is absorbed in the small intestine, enhanced by food and acidic pH. Doses range from 500–1000 mg for heartburn (max 7 g/day) and 1–2 g/day for supplementation. Long-term omeprazole use reduces stomach acidity, impairing calcium carbonate absorption and potentially affecting bone health. Although not contraindicated, co-administration may reduce calcium bioavailability. Additionally, calcium carbonate may interfere with omeprazole absorption if taken simultaneously. To minimize interaction, dosing should be separated. Omeprazole provides long-term acid suppression, while calcium carbonate offers rapid, short-term relief.

Keywords: Omeprazole, H+/K+ATPase, CYP2C19 & CYP3A4, GERD (gastro oesophageal reflux disease), H-pylori (helicobacterpylori), Calcium carbonate, Erosive esophagitis.

Introduction

Omeprazole

Chemical Name– 6-methoxy-2-[[(4-methoxy-3,5-dimethyl-2-pyridinyl) methyl] sulfinyl]-1Hbenzimidazole

Mechanism of Action

Omeprazole is a proton pump inhibitor.1It inhibits the parietal cell H+/K+ATpase pump in the final step of acid production. Omeprazole suppresses gastric basal & stimulates acid secretion [1]. 2Omeprazole is acid-labile and requires activation in the highly acidic environment of the parietal cell secretory canaliculi. The inhibitory effects of omeprazole occur rapidly with in 1 hour of administration, the maximum effect occurring in 2 hours. The inhibitory effects last for approximately 72 hours after administration [2].

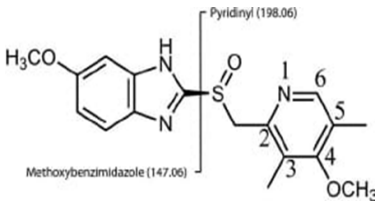


Fig 01: Chemical Structures of Omeprazole

Pharmacokinetics

Absorption

Omeprazole primarily absorbed in the small intestine.

Omeprazole is formulated as enteric coated granules in capsules, tablets, or suspensions) or as IV formulations. It has a bioavailability of 30-40% after first oral dose. Bioavailability increases with repeated once daily dosing. The peak plasma concentration is attained in 0.5 to 3.5 hours.

Distribution

The plasma protein binding is approximately 95 – 96%
The volume of distribution is about 0.3L/Kg [3].

Metabolism

4It is extensively metabolized in the liver by the cytochrome P450 enzyme.

Elimination

- Metabolites are excreted primarily in the urine (about 77 –80%) [4].
- Approximately 20% of metabolites are excreted in the feces via biliary elimination.
- Its plasma half-life is 0.5 – 1 hour.

Administration / Dosing

Gastroesophageal Influx Disease (GERD)

For treating characteristic GERD – 20mg formerly daily for 4remedymay extended to 8 weeks [5].

Gastric Ulcer

For treating gastric ulcer –40mg formerly daily for 4-8 weeks.

Duodenal Ulcer

For H-pylori infection – combination remedyis essential.
"Omeprazole 20mg doubly daily + Amoxicillin 1000mg doubly diurnal + Clarithromycin 500mg doubly daily for 10-14 days".

Hypersecretory Conditions

For hypersecretory complaint – 60mg formerly daily.

Dyspepsia

For dyspepsia condition – 10 to 20mg formerly daily for 2-4 weeks.

Adverse Effects

Omeprazole, while effective in managing acid-related disorders, is associated with several adverse effects like. 7Gastrointestinal disturbances such as nausea, vomiting, abdominal pain, diarrhea and constipation are commonly reported. Prolonged use may lead to hypomagnesemia, manifesting as muscle spasms, seizures, or cardiac arrhythmias. Renal complications including acute interstitial nephritis and chronic kidney disease have also been observed. Cardiovascular risk may be heightened as hypomagnesemia contributes to the development of arrhythmias [6, 7].

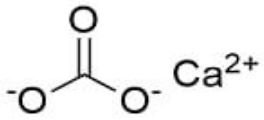
Clinical Uses

Omeprazole is widely prescribed for various acid-related gastrointestinal conditions [8]. It is commonly used in the management of gastroesophageal reflux disease (GERD) and peptic ulcer disease. Additionally, it is beneficial in relieving symptoms of functional dyspepsia and plays an important role in the prevention of nonsteroidal anti-inflammatory drug (NSAID)-induced ulcers. In certain cases, omeprazole is also employed to help prevent pancreatitis following endoscopic retrograde cholangiopancreatography (ERCP) [8].

Calcium Carbonate

Chemical name: Calcium (Ca2+) carbonates (CO32-)

Mechanism of action



Calcium carbonate

Fig 02: Chemical Structure of Calcium Carbonate

Calcium carbonate acts as an antacid by negativing gastric acid in the stomach.Upon reaching the acidic terrain of the stomach, it reacts with hydrochloric acid (HCl), dividing into calcium ions (Ca^{2+}) and carbonate ions (CO_3^{2-}). The carbonate ions bind to free hydrogen ions (H^+), reducing gastric acidity and thereby adding the stomach's pH [9].
10This increase in pH decreases the exertion of pepsin and reduces the dangerous goods of gastric acids and Helicobacter pylori poisons. also, calcium carbonate functions in the small intestine as a phosphate binder and a medicine chelator [10].

Pharmacokinetics

Absorption

- 11 Calcium carbonate is primarily absorbed in the duodenum & proximal jejunum.
- It has a bioavailability generally ranges from 15 to 35 of the ingested cures. Under optimalconditions can be< 10 if taken sated or with low acid.

Distribution

- Absorbed Ca2 enters the blood aqueduct. About 50 of serum calcium is ionized (free, natural active). The keepsake is bound to albumin (40) or perplexed with anion like phosphate, citrate, or sulphate [10].
- Roughly 99 of total body calcium are stored as hydroxyapatite dishes in bones & teeth [11].

Metabolism

- Calcium carbonate itself is not metabolized enzymatically like organic drugs.
- The carbonate ion (CO32-) reacts with stomach acid to form CO2 gas & water.
- Vitamin D activation in the liver and feathersis vital for its physiological operation but isn't direct metabolism of the calcium ion.

Elimination

- About 80 of calcium is excreted urine via the feathers.
- About 20 of calcium is excreted as fecal.

- The unabsorbed salutary/ supplemental calcium and endogenous calcium buried into cattiness/ intestinal authorities(not reabsorbed)
- The minor routes like sweat, hair, nails12(negligible amounts)

Administration/ Dosing

Osteoporosis

*For treating osteoporosis complaint– Grown-ups 19- 50 times & Men 51- 70 times 1000 mg essential calcium per day. Women 51 times & Men 71 times 1200 mg essential calcium per day.

Heart burn, Indigestion, Upset stomach:

*For treating heartburn, indigestion & stomach derangement – 500 mg – 1500 mg (essential calcium) [13].

Hyperphosphatemia

For treating hyperphosphatemia – 1.5 to 3 gm per day.

Order problems (Renal impairment)

For treating order related problems – lozenge must be rigorously determined & covered by a croaker14.

NOTE (specific instructions):

- As a calcium supplement generally taken with food. Calcium immersion is enhanced in the acidic terrain of the stomach when food is present. Taking it with a mess also minimizes implicit stomach derangement.
- As an Antacid Taken as symptoms do, generally 1 hour after reflections and at bed time.
- Don't lie down Remain upright (sitting or standing) for at least 10- 30 min after taking a cure to helpOesophageal vexation or ulceration.

Adverse Effects

Calcium carbonate may produce several adverse effects like.15 gastrointestinal disturbances are common and include constipation, bloating, excess gas, abdominal discomfort, nausea, and vomiting. Excessive intake can lead to hypercalcemia, presenting with loss of appetite, nausea, vomiting, muscle weakness, fatigue, cardiac arrhythmias, and, in severe cases, even coma. Renal complications such as nephrolithiasis (kidney stones) and nephrocalcinosis may alsooccur.In some instances, the milk-alkali syndrome can develop, characterized by hypercalcemia, metabolic alkalosis, and renal impairment [15].

Clinical Uses

Calcium plays a crucial role in maintaining bone health and various physiological functions, and its supplementation is widely used in clinical practice. It is primarily used in the treatment and prevention of calcium insufficiency, as well as in the management of osteoporosis to reduce the risk of fractures. Calcium also serves as an effective antacid remedy for gastrointestinal symptoms and functions as a phosphate binder in chronic kidney disease [16].

Discussion

The combination of omeprazole and calcium carbonate represents a clinically relevant interaction due to their opposing mechanisms of action on gastric acidity. Omeprazole, a proton pump inhibitor, provides potent and long-lasting suppression of gastric acid secretion, making it highly effective in the management of gastroesophageal reflux disease (GERD), peptic ulcers, and hypersecretory conditions. On the other hand, calcium carbonate functions as an antacid that requires gastric acid for dissolution and optimal absorption.

It is also important to recognize that antacids such as calcium carbonate, when taken concomitantly with omeprazole, may transiently increase gastric pH and interfere with omeprazole's absorption, thereby reducing its acid-suppressive efficacy. Although this interaction is less clinically significant compared to the impaired calcium absorption, it underscores the importance of avoiding simultaneous administration.

The combination of omeprazole and calcium carbonate highlights the delicate balance between managing gastrointestinal disorders and preserving bone health. Appropriate patient counselling, judicious supplement selection, individualized dosing strategies, and regular follow-up can help minimize risks while optimizing therapeutic benefits.

When administered together, omeprazole-induced hypochlorhydria may significantly reduce the bioavailability of calcium carbonate, thereby diminishing its therapeutic efficacy as a calcium supplement and potentially predisposing patients to long-term complications such as osteoporosis and fracture risk. This is especially relevant for elderly individuals, postmenopausal women, and patients requiring prolonged PPI therapy who are already at increased risk of bone mineral density loss.

Moreover, calcium carbonate may provide short-term relief from heartburn, but if taken simultaneously with omeprazole, it can interfere with the drug's absorption, reducing its clinical effectiveness. To minimize such interactions, clinicians recommend separating the dosing schedules and considering alternative calcium supplements, such as calcium citrate, which is less dependent on gastric acidity for absorption.

Overall, while both agents play important roles in gastrointestinal and bone health management, careful attention to their pharmacological interactions, dosing strategies, and patient-specific needs is essential to optimize therapeutic outcomes.

Conclusion

Omeprazole and calcium carbonate are both valuable therapeutic agents with distinct clinical roles. Omeprazole provides potent, long-lasting suppression of gastric acid, making it effective in treating GERD, ulcers, and hypersecretory conditions. Calcium carbonate, on the

other hand, acts as both an antacid and a calcium supplement, supporting bone health while offering rapid relief from gastric discomfort. However, when used together, omeprazole-induced reduction in stomach acidity can impair the absorption of calcium carbonate, potentially increasing the risk of deficiencies and bone-related complications during long-term therapy. Therefore, the combination requires careful administration, often by adjusting dosing schedules or considering alternative calcium sources like calcium citrate. Overall, individualized patient care, with attention to drug interactions and nutritional needs, is crucial to ensure both gastrointestinal relief and adequate calcium balance for long-term health.

While omeprazole and calcium carbonate remain integral in the management of gastrointestinal and bone health, their concurrent use demands careful clinical consideration. Long-term proton pump inhibitor therapy, although highly effective for acid-related disorders, should always be weighed against its potential to impair calcium absorption and contribute to skeletal complications. Substituting calcium carbonate with calcium citrate, spacing administration times, and ensuring adequate dietary intake of calcium and vitamin D are practical strategies to reduce risks. Clinicians should also monitor patients on prolonged therapy for bone mineral density loss and adjust treatment plans accordingly. Ultimately, a patient-centred approach-balancing acid suppression with bone preservation-ensures safer and more effective outcomes in both gastrointestinal and metabolic health.

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Conflict of Interest

The authors declare no conflict of interest.

Informed Consent and Ethical Statement

Not Applicable

Author Contribution

All Authors Are Contributed Equally

References

1. Sachs, G., Shin, J. M., & Howden, C. W. (2006). The clinical pharmacology of proton pump inhibitors. Alimentary Pharmacology & Therapeutics, 23(2),2-8.
2. Andersson, T., & Weidolf, L. (2008). Stereoselective disposition of proton pump inhibitors. Clinical Drug Investigation, 28(5),263-279.
3. Robinson, M. (2001). Review article: The pharmacodynamics and pharmacokinetics of proton pump inhibitors—focus on omeprazole. Alimentary Pharmacology & Therapeutics, 15(Suppl 2),2-10.
4. Rost, K. L., & Roots, I. (1994). Accelerated omeprazole metabolism in extensive metabolizers of sparteine. Clinical Pharmacology & Therapeutics, 55(6),630-635.
5. Katz, P. O., Gerson, L. B., & Vela, M. F. (2013). Guidelines for the diagnosis and management of gastroesophageal reflux disease. The American Journal of Gastroenterology, 108(3),308-328.
6. Howden, C. W., & Hunt, R. H. (1990). The relationship between suppression of acidity and gastric ulcer healing rates. Alimentary Pharmacology & Therapeutics, 4(1), 25–33.
7. Metz, D. C. (2000). Diagnosis and treatment of Zollinger–Ellison syndrome in the era of PPIs. Best Practice & Research Clinical Gastroenterology, 15(3), 467–484.
8. Talley, N. J., Vakil, N., & Moayyedi, P. (2005). American Gastroenterological Association technical review on the evaluation of dyspepsia. Gastroenterology, 129(5), 1756–1780.
9. Osman M, Yadlapati R. (2022). Antacids revisited: a review of basic science and clinical utility. Journal of Clinical Gastroenterology, 56(9), 767–774.
10. Gruyaert B, et al. (2021). Onset of acid-neutralizing action of calcium/magnesium carbonate-based antacid using an artificial stomach model.BMC Gastroenterology, 21-64.
11. Wood RJ, Serfaty-Lacrosnière C. (1992). Gastric acidity, calcium absorption, and calcium carbonate supplements. Nutrition Reviews, 50(11):301–306.
12. Sheikh MS, et al. (1989). Reduction of dietary phosphorus absorption by phosphate binders. Journal of Clinical Investigation, 83(1):66–73.
13. Heaney RP, Recker RR, Weaver CM. (1990). Absorbability of calcium sources: the limited role of solubility. Osteoporosis International, 1(2):65–67.
14. Bommer J, et al. Calcium load during $CaCO_3$ administration in healthy individuals; impact on urinary calcium excretion. Nephrol Dial Transplant. 2008;23(9):2861–2866.
15. Penniston KL, et al. (2008). Excess calcium carbonate ingestion and recurrent kidney stones. Urological Research, 36(2):77–81.
16. Beall DP, Scofield RH. (1995). Milk-alkali syndrome associated with calcium carbonate consumption – Report of 7 patients with review of literature. Medicine (Baltimore), 74(2):89–96.