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Safety Profile of Omeprazole: A Narrative Review Of Adverse Drug Reactions and Risk Factors

Amulya Gavara[1], Sridevi Madabattula[1],Monika Devi Maagapu[1] , Revanthi Maila[1] , D Veerandra Kumar[2] , Devanaboyina Narendra[3], Abhinav VKS Grandhi*[4].

1.PharmD Scholar, Doctor of Pharmacy, VJ's College of Pharmacy, Diwancheruvu, Rajahmundry - 533296, Andhra Pradesh, India.

2.Associate Professor, Department of Pharmacy Practice, VJ's College of Pharmacy, Diwancheruvu, Rajahmundry - 533296, Andhra Pradesh, India.

3.Principal and Professor, VJ's College of Pharmacy, Diwancheruvu, Rajahmundry - 533296, Andhra Pradesh, India.

4.Assistant Professor, Department of Pharmacy Practice, VJ's College of Pharmacy, Diwancheruvu, Rajahmundry - 533296, Andhra Pradesh, India.

ABSTRACT

Omeprazole, a widely prescribed proton pump inhibitor (PPI), is a cornerstone therapy for acid-related gastrointestinal disorders, including gastroesophageal reflux disease, peptic ulcer disease, and Zollinger–Ellison syndrome. While generally safe and effective, prolonged or inappropriate use has been linked to a spectrum of adverse drug reactions (ADRs). Common ADRs include headache, abdominal discomfort, nausea, diarrhea, and flatulence, which are usually mild and self-limiting. However, serious complications—such as Clostridioides difficile infection, interstitial nephritis, hypomagnesemia, vitamin B12 deficiency, bone fractures, hepatotoxicity, and severe cutaneous reactions—though rare, pose significant clinical challenges. Risk factors influencing ADR incidence include treatment duration, high dosage, advanced age, polypharmacy, comorbid conditions, and genetic polymorphisms in CYP2C19 metabolism, which alter drug bioavailability and efficacy. Clinical pharmacists play a crucial role in mitigating these risks through therapeutic optimization, monitoring of laboratory parameters, early identification of drug interactions, and patient education. Evidence-based deprescribing strategies and routine safety surveillance are essential to minimize preventable harm while preserving therapeutic benefit. This review highlights the clinical significance of omeprazole-associated ADRs from a pharmacist's perspective, emphasizing the importance of rational prescribing, vigilant monitoring, and interdisciplinary collaboration in ensuring patient safety.

Keywords: Omeprazole, Proton Pump Inhibitors, Adverse Drug Reactions, Clinical Pharmacist, Patient Safety

*Corresponding Author Email: 2402abhinav@gmail.com

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INTRODUCTION

Omeprazole is a proton pump inhibitor (PPI). It is a first-line treatment for acid-related disorders such as gastroesophageal reflux disease (GERD), peptic ulcers, and Zollinger-Ellison syndrome[1]. Its therapeutic effects are achieved by irreversibly blocking the H+/K+ ATPase enzymes in gastric parietal cells, resulting in sustained acid suppression[2]. Since its introduction in the late 1980s, it has become a cornerstone of acid-suppression therapy due to its superior efficacy and longer duration of action compared to histamine H2-receptor antagonists. This drug is formulated as a delayed-release capsule to protect it from stomach acid, ensuring optimal absorption in the small intestine[3,4]. Its metabolism is via CYP2C19, resulting in variable response among individuals, particularly those with gigantic polymorphisms[5]. Omeprazole is a generally well-tolerated drug with common side effects including headache, nausea, and abdominal discomfort. Long-term use has been associated with potential risks such as vitamin B12 deficiency, bone fractures, and increased susceptibility to Clostridioides difficile infections[6]. Omeprazole may interact with medications like clopidogrel, affecting their effectiveness. Current clinical guidelines recommend using the lowest effective dose for the shortest duration to balance benefits and risks. Despite the concerns, omeprazole remains a widely prescribed and cost-effective option for managing gastric acid hypersecretion.[7,8,9].

Drug profile:

Generic: Omeprazole

Brand name: Omez, Ocid, Oskar, Ometab, Omesec, Zolsec, etc., refer to Table 1 [10,11,12].

Table 1: Commonly available Omeprazole brands in India

Brand Name	Manufacturer	Popular Dosage(s)
Ocure-20	Max Life Sciences	20 mg capsules
Ocure-D	Max Life Sciences (combo)	20 mg omeprazole + 10 mg domperidone
Zolsec	Aarpik Pharmaceuticals	Capsules / Tablets
Acichek-20 mg	Abbott Healthcare	20 mg capsule
OATH-20 mg	Aroma LS	20 mg capsule
ESZOL	ABS Remedies	20 mg capsule
Zoom-20 mg	Active Healthcare	20 mg capsule
Opaz	Aglowmed Pharmaceuticals Ltd	Capsule / Tablet
Cozep-20 mg	AHPL Pharma	20 mg capsule
Zolop-20 mg	Albin Healthcare	20 mg capsule
Omez	Dr. Reddy's Laboratories	10 mg / 20 mg / 40 mg capsules/tablets
Ocid	Zydus Cadila	10 mg / 20 mg / 40 mg capsules/tablets
Oskar	Mankind Pharma	10 mg / 20 mg / 40 mg
Ometab	Intas Pharmaceuticals	10 mg / 20 mg / 40 mg
Omesec	Sun Pharma	10 mg / 20 mg / 40 mg

Mechanism of action:

Omeprazole is a substituted benzimidazole and a prodrug that exhibits its pharmacological action following activation in an acidic environment. After oral administration, it is absorbed in the small intestine and reaches the systemic circulation, from where it selectively accumulates in the acidic secretory canaliculi of gastric parietal cells. Within this acidic environment (pH < 4), omeprazole undergoes protonation and is converted to its active sulfenamide form. The active sulfenamide covalently binds to sulfhydryl groups of cysteine residues on the H^+,K^+-ATPase enzyme located on the apical membrane of the parietal cell, leading to irreversible inhibition of the proton pump activity [13].

This inhibition blocks the final step in gastric acid secretion, effectively reducing both basal and stimulated acid production. Since the binding is irreversible, acid secretion can only resume upon the synthesis of new proton pump molecules, which generally requires 18 to 24 hours. This prolonged duration of action supports once-daily dosing for clinical efficacy [13].

The unique mechanism of omeprazole allows it to inhibit acid secretion irrespective of the stimulus, including histamine, gastrin, or acetylcholine. Therefore, omeprazole and other proton pump inhibitors are considered the most potent agents available for the suppression of gastric acid secretion [13].

Indications:

Omeprazole, a proton-pump inhibitor, is approved for a variety of acid-related gastrointestinal conditions, including both FDA-approved and clinically recognized uses,

1. Active duodenal ulcers in adults generally heal within approximately 4 weeks [14].
2. Active benign gastric ulcer in adults [14].
3. Helicobacter pylori eradication therapy, used in combination with antibiotics (e.g. clarithromycin, amoxicillin or metronidazole) to reduce duodenal ulcer recurrence [14,15].
4. Gastroesophageal reflux disease (GERD), including erosive esophagitis, in both adults and pediatric patients [14].
5. Uncomplicated heartburn in adults (OTC use), typically for frequent heartburn occurring ≥ 2 days/week [15].
6. Hypersecretory conditions such as Zollinger-Ellison syndrome, multiple endocrine adenomas, and systemic mastocytosis [14].
7. Prevention of upper gastrointestinal bleeding in critically ill adult patients [15].

Additional clinically utilized indications (non-FDA approved) include:

- Barrett's oesophagus

- Stress ulcer prophylaxis in critical care settings [14,16].

Pharmacokinetics and pharmacodynamics:

Omeprazole is a proton-pump inhibitor that is administered as an enteric-coated oral formulation due to its acid-labile nature. It is a prodrug that becomes activated only under strongly acidic conditions, which occur in the parietal cell canaliculus of the stomach.

Pharmacokinetics:

After oral administration, omeprazole is absorbed in the small intestine, with peak plasma concentrations achieved in approximately 0.5 to 3.5 hours. Bioavailability is approximately 35–40% after a single dose due to extensive first-pass hepatic metabolism, but increases to ~65% with repeated dosing as enzyme saturation occurs [17,18]. The drug is highly protein-bound (~95%) and is extensively metabolized by cytochrome P450 enzymes, primarily CYP2C19 and CYP3A4, in the liver.

The elimination half-life of omeprazole is short (~1 hour), but its pharmacodynamic effect lasts 24–72 hours, due to irreversible inhibition of the H^+,K^+-ATPase (proton pump). The metabolites are excreted primarily via the urine (77%) and to a lesser extent in feces [17,19].

Genetic polymorphisms in CYP2C19 significantly influence omeprazole metabolism. Poor metabolizers (common in Asian populations) may have elevated plasma levels and a more prolonged antisecretory effect, while ultra-rapid metabolizers may exhibit subtherapeutic responses [18,19].

Pharmacodynamics:

Omeprazole acts by irreversibly binding and inactivating the gastric parietal cell H^+,K^+-ATPase enzyme, the final common pathway of acid secretion. This inhibition leads to profound and long-lasting suppression of basal and stimulated gastric acid secretion, regardless of the stimulus (e.g., histamine, acetylcholine, or gastrin) [17,20]. Inhibition begins within 1 hour of dosing, with maximal effect at 2 hours and duration of action lasting up to 72 hours, despite the short systemic half-life [19].

Omeprazole's pharmacodynamic profile makes it highly effective for conditions requiring acid suppression, including gastroesophageal reflux disease, peptic ulcers, Zollinger-Ellison syndrome, and H. pylori eradication therapy. Prolonged use, however, may result in hypergastrinemia, alteration of gut flora, or malabsorption of vitamin B12 and minerals due to chronic acid suppression [17,20].

Dosing: 10/20/40 mg

Formulations:

Omeprazole is highly acid-labile, necessitating specialized pharmaceutical formulations (e.g. enteric coating, buffering agents) to ensure stability, bioavailability, and rapid onset of action.

Enteric-Coated Delayed-Release Formulations

Standard oral products (capsules, tablets, sprinkle granules) comprise enteric-coated pellets or granules that resist disintegration in gastric pH and dissolve in intestinal pH above ~5.5. These are designed to release drug in the duodenum where absorption occurs, yielding ~30–40% bioavailability and peak plasma levels in ~0.5–3.5 hours [21]. Multiple studies confirm their acid resistance and consistent dissolution profiles [21,22].

Extemporaneous and Suspension Formulations

For pediatric or dysphagic patients, compounded oral suspensions using crushed enteric granules or buffered formulations (e.g. with sodium bicarbonate) have been developed. Such formulations vary in stability depending on pH and composition. One comparative study evaluated three compounding strategies crushed commercial granules, crushed granules in sodium bicarbonate-glycerin vehicle, and pure omeprazole in sodium bicarbonate. The buffered suspension significantly improved stability, reducing acid-mediated degradation [22].

Immediate-Release Buffered Formulations

Immediate-release (IR) formulations (e.g. omeprazole with sodium bicarbonate) protect the drug from gastric degradation without enteric coating. Compared to delayed-release forms, these show faster absorption and quicker onset of acid suppression [23].

Formulation Performance Factors

Studies highlight how formulation parameters such as polymer type, coating thickness, and multiparticulate design—influence drug release and stability. Key factors include resistance in acidic media, dissolution timing in buffered media, and consistency under variable gastric conditions [23].

Bioequivalence of Enteric-Coated Products

Multiple pharmacokinetic studies comparing two enteric-coated 20 mg formulations demonstrated bioequivalence in healthy volunteers under fasting and fed conditions: AUC and C_{max} ratios fell within the accepted 0.8–1.25 range [24]

Known/expected side effects (from clinical trials)

In a randomized, double-blind, placebo-controlled pilot study in patients with idiopathic pulmonary fibrosis, adverse events were recorded in 70% of patients receiving omeprazole and 64% in the placebo group. Common adverse events (≥10%) in the omeprazole arm included lower

respiratory tract infection (26%) and abdominal pain, urinary tract infection, vomiting, and cough (~9–13%) [25].

Three participants in the omeprazole arm experienced moderate-to-severe abdominal pain, resulting in treatment discontinuation. No deaths were attributed to omeprazole treatment.

Although not limited to formal clinical trials, a systematic review of safety data up to 2019 revealed that common adverse reactions (1–10% incidence) included headache, diarrhea, abdominal pain, flatulence, back pain, and respiratory tract infections. Post-marketing reports identified serious but rare events such as hypomagnesemia, vitamin B_{12} deficiency, acute interstitial nephritis, bone fractures, and elevated creatinine levels [26].

According to NCBI's LiverTox database, clinically apparent liver injury from omeprazole is very rare—occurring in less than 1 in 100,000 users. These events typically manifest within 1–4 weeks of therapy initiation and resolve rapidly upon drug withdrawal. Associated symptoms may include acute hepatocellular injury, fever, rash, eosinophilia, and on occasion, rhabdomyolysis or Stevens–Johnson syndrome [27]. Refer to Table 2.

Table 2. Common and Clinically Notable Adverse Effects of Omeprazole Reported in Recent Clinical Trials and Safety Reviews (2019–2024)

Side Effects	Frequency / Context
Lower respiratory tract infections	26% in the omeprazole group in the IPF trial
Abdominal pain	9-13% in trial participants
Vomiting, urinary tract infections, cough	Approximately 9%
Headache, diarrhoea, Flatulence, back pain, respiratory infections	1-10% in broader clinical data
Hypomagnesemia, Vitamin B_{12} deficiency, nephritis	Rare post-marketing events
Acute hepatotoxicity	< 1/100000 cases; reversible with withdrawal

Omeprazole, a widely used proton pump inhibitor (PPI), is primarily prescribed for gastroesophageal reflux disease and peptic ulcers. While generally considered safe and well tolerated, it is associated with several adverse drug reactions (ADRs) that practitioners and patients should be aware of.

Common Adverse Reactions:

The most frequently reported ADRs include headache, abdominal pain, nausea, diarrhea, vomiting, flatulence, and constipation [28–31,33,34]. These effects are typically mild and transient.

Serious and Rare Adverse Events: A minority of patients may experience serious complications such as severe diarrhoea (including Clostridioides difficile infections), nephritis, acute tubulointerstitial nephritis, and other kidney-related issues. Serious skin reactions, such as Stevens-Johnson syndrome, toxic epidermal necrolysis, and subacute cutaneous lupus erythematosus, can occur rarely, as can anaphylaxis [28], hematologic disorders, including thrombocytopenia and

agranulocytosis, and liver function abnormalities or, rarely, hepatic failure, have also been reported. Long-term use may increase the risk of bone fractures, vitamin B12 deficiency, and hypomagnesemia [28]. Reduced antiplatelet effectiveness may occur with concurrent clopidogrel use due to CYP2C19 interactions, potentially increasing cardiovascular risk. There is also some evidence of an increased risk of gastric cancer with prolonged therapy.

Considerations; The risk and severity of these ADRs are influenced by treatment duration, dosage, comorbidities, and genetic factors such as CYP2C19 metabolic status. Most adverse effects are rare, but serious reactions should be monitored, particularly in vulnerable or long-term patients [28–37].

Mechanism behind ADRs:

Omeprazole's adverse drug reactions (ADRs) arise from its primary pharmacologic action—potent suppression of gastric acid via inhibition of the H^+/K^+ ATPase in gastric parietal cells—as well as its metabolic and immunologic effects. Below is a concise explanation of the mechanisms responsible for each ADR category:

1.Common, Mild ADRs

Headache, Abdominal Pain, Nausea, Diarrhea, Vomiting, Flatulence, Constipation

These are usually due to changes in gastric acidity and altered gastrointestinal motility. Suppressed acid secretion can affect digestion, lead to bacterial overgrowth, and cause mild GI symptoms. Imbalances in gut flora and slowed gastric emptying are also implicated in these effects [38–42].

2. Serious/Rare Events:

- Severe Diarrhea (e.g., *Clostridioides difficile* infections): By raising gastric pH, omeprazole reduces the natural barrier against ingested pathogens. This allows bacteria like *C. difficile* to proliferate and cause severe colitis .
- Nephritis (Acute Tubulointerstitial Nephritis and Other Renal Effects): These are believed to be immune-mediated, representing hypersensitivity reactions where omeprazole or its metabolites stimulate immune cells, leading to renal interstitial inflammation .
- Serious Skin Reactions (SJS, TEN, Subacute Cutaneous Lupus): These severe dermatological reactions are mostly immune-mediated idiosyncratic responses—possibly involving drug-antigen formation that activates cytotoxic T-cells and initiates widespread skin apoptosis .
- Anaphylaxis and Other Allergic Reactions: These stem from an IgE-mediated immune response to omeprazole or its breakdown products .

- Hematologic Disorders (Thrombocytopenia, Agranulocytosis): The mechanism is likely immune-mediated destruction of blood cells, with omeprazole acting as a hapten or inducing autoimmune reactivity .
- Liver Function Abnormalities/Hepatotoxicity: Omeprazole undergoes hepatic metabolism via CYP450 enzymes. Rarely, toxic metabolites or direct mitochondrial impairment in liver cells may provoke hepatitis or hepatic failure .

3. Long-Term or Drug-Interaction Risks:

- Bone Fracture, Hypomagnesemia, Vitamin B12 Deficiency: Chronic reduction in gastric acid impairs intestinal absorption of minerals (magnesium, calcium) and vitamin B12 (which requires acidic pH for release from food). This predisposes long-term users to bone demineralization, deficiency states, and related sequelae .
- Impaired Clopidogrel Activation (Increased Cardiovascular Risk): Omeprazole is a CYP2C19 inhibitor; clopidogrel, a prodrug, needs this enzyme for activation. Concurrent use leads to reduced clopidogrel efficacy, raising the risk of thrombotic events .
- Gastric Cancer with Prolonged Use: Hypochlorhydria (chronically low stomach acid) may lead to hypergastrinemia, altered gastric microbiota, and chronic inflammation, creating a microenvironment conducive to neoplastic transformation over time .

4. Genetic/Patient-Specific Factors:

CYP2C19 Variants: Individuals genetically predisposed to be poor CYP2C19 metabolizers accumulate higher drug concentrations, increasing the risk and severity of ADRs due to both exaggerated therapeutic and toxic effects [38–48]. Refer Table 3

Table 3: ADR Mechanism

ADR	Mechanism(s)
Common GI symptoms	Gastric acid suppression → altered flora and motility
C. difficile colitis	Loss of acid barrier → bacterial overgrowth
Nephritis	Immune-mediated hypersensitivity
Serious skin reactions (SJS, TEN, lupus)	Immune-mediated cytotoxic responses
Anaphylaxis and allergies	IgE-mediated immune reaction
Hematologic disorders	Immune-mediated destruction of blood cells
Liver injury	Toxic metabolites, mitochondrial impairment
Bone fractures, hypomagnesemia, vitamin B12 deficiency	Impaired absorption due to hypochlorhydria
Reduced clopidogrel efficacy	CYP2C19 inhibition blocking prodrug activation
Gastric cancer (long-term)	Chronic hypochlorhydria → hypergastrinemia, inflammation, microbiota changes
Increased risk in CYP2C19 poor metabolizers	Higher omeprazole levels → increased ADR risk

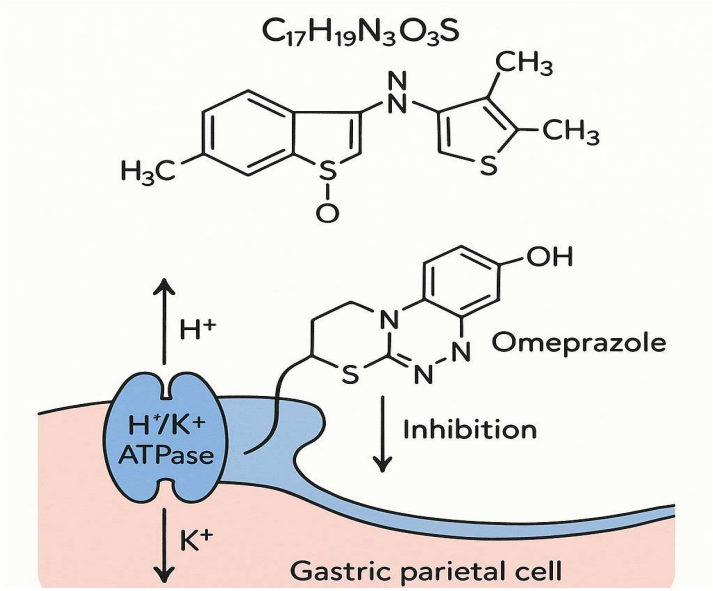

Figure 1: Chemical structure of omeprazole and its mechanism of action in gastric acid suppression

Risk factors:

Although omeprazole is generally well-tolerated, several risk factors predispose patients to a higher incidence and severity of adverse drug reactions (ADRs). These factors span across patient-specific characteristics, genetic polymorphisms, treatment duration, drug interactions, and underlying comorbidities.

Treatment Duration and Dosage

Long-term use of omeprazole, particularly at high doses, significantly increases the risk of nutrient malabsorption, bone fractures, and gastrointestinal infections. Chronic acid suppression leads to hypochlorhydria, which impairs calcium, magnesium, and vitamin B_{12} absorption, predisposing users to fractures and deficiency syndromes [49,50].

Genetic Variability in CYP2C19 Metabolism

Omeprazole is extensively metabolized by the hepatic cytochrome P450 isoenzyme CYP2C19. Individuals who are poor metabolizers (PMs), especially among Asian populations, exhibit elevated plasma concentrations, increasing the likelihood of both therapeutic and adverse effects. Conversely, ultra-rapid metabolizers may exhibit subtherapeutic exposure and increased therapeutic failure [51,52].

Advanced Age and Polypharmacy

Elderly patients are more susceptible to omeprazole-induced complications due to age-related decline in renal and hepatic clearance, and increased likelihood of polypharmacy, which enhances

the risk of drug–drug interactions. For instance, concurrent use of omeprazole and clopidogrel may diminish the latter's antiplatelet activity through CYP2C19 inhibition [53].

Concurrent Medication Use

Drug interactions are particularly important with:

- Clopidogrel – Reduced bioactivation due to CYP2C19 inhibition, potentially increasing cardiovascular risks [53].
- Methotrexate – Delayed clearance due to inhibition of renal transporters, raising toxicity risks [54].
- Warfarin and Phenytoin – Enhanced levels due to metabolic interference, leading to bleeding or CNS toxicity [55].

Comorbid Conditions

Patients with renal insufficiency, hepatic dysfunction, or autoimmune disorders may exhibit heightened sensitivity or atypical ADRs, including interstitial nephritis and hepatotoxicity. Omeprazole-induced nephritis has been frequently reported in individuals with prior renal compromise [56].

Infection Risk

Chronic acid suppression compromises gastric barrier function, promoting bacterial overgrowth (e.g., *Clostridioides difficile*) and community-acquired pneumonia. Individuals on prolonged therapy are particularly vulnerable to such infections [57].

Management and monitoring

Clinical Pharmacist's Perspective

As frontline medication experts, clinical pharmacists are uniquely positioned to oversee the safe and effective use of omeprazole, especially in hospital settings and among long-term users. Their prospective responsibilities include ensuring rational use, minimizing adverse drug reactions (ADRs), optimizing therapeutic outcomes, and enhancing patient education and adherence.

Therapeutic Optimization and Indication Validation

Pharmacists should regularly assess whether the continued use of omeprazole is clinically warranted. Routine audits have shown that up to 50% of long-term PPI use lacks clear indication [58]. Pharmacists should collaborate with prescribers to implement step-down or deprescribing protocols when therapy is no longer justified [59].

Monitoring of Safety Parameters

Long-term PPI use, including omeprazole, is associated with several potential adverse effects. Clinical pharmacists must initiate or recommend periodic monitoring strategies:

- Serum magnesium, vitamin B12, and calcium levels (at least annually for chronic users) [60].
- Renal function tests in patients at risk for interstitial nephritis, especially those with comorbidities [61].
- Bone density assessment in elderly patients or those at risk for fractures [62].

Pharmacists should also monitor for signs of GI infections, especially *Clostridioides difficile*, and educate patients on warning signs such as persistent diarrhea or abdominal pain.

Drug Interaction Surveillance

Pharmacists should actively review patient medication profiles for drugs known to interact with omeprazole, such as:

- Clopidogrel – decreased efficacy due to CYP2C19 inhibition [63].
- Methotrexate – reduced clearance and increased toxicity [54].
- Warfarin or phenytoin – risk of elevated serum levels and toxicity [55].

Pharmacists can recommend alternatives (e.g., pantoprazole with clopidogrel) or coordinate dose adjustments and lab monitoring.

Patient Counseling and Adherence

Pharmacists should educate patients on:

- Taking omeprazole before meals to optimize acid suppression.
- Avoiding unnecessary long-term use, especially in over-the-counter (OTC) settings.
- Reporting side effects such as rash, fatigue, numbness, or unusual bleeding.
- Risk of rebound acid hypersecretion after abrupt discontinuation, and the value of tapering doses [59].

Documentation and Follow-Up

In inpatient settings, pharmacists can ensure that PPI therapy is reviewed at discharge, with clear documentation of indication, duration, and follow-up plans. In outpatient care, pharmacists may schedule routine reviews or flag patients using PPIs >8 weeks without clear indication.

DISCUSSION:

Omeprazole, a widely prescribed proton pump inhibitor (PPI), plays a pivotal role in the management of acid-related gastrointestinal conditions such as GERD, peptic ulcer disease, and Zollinger–Ellison syndrome. Its pharmacological profile—including irreversible inhibition of H^+/K^+-ATPase—translates into potent acid suppression. However, this same mechanism underpins several clinically significant adverse drug reactions (ADRs), particularly when therapy is prolonged or used without appropriate indication.

From a clinical pharmacist's perspective, the spectrum of ADRs linked to omeprazole ranges from commonly reported but mild symptoms (e.g., nausea, headache, diarrhea) to serious and sometimes life-threatening events such as hypomagnesemia, interstitial nephritis, vitamin B12 deficiency, and Clostridioides difficile infection. These effects are well-documented in literature and recent pharmacovigilance reports, highlighting the necessity for continuous patient-specific benefit–risk evaluation.

Key contributing risk factors include long-term use, polypharmacy, and genetic polymorphisms in CYP2C19 metabolism, which alter omeprazole plasma concentrations and influence both efficacy and safety. For instance, poor metabolizers may accumulate toxic levels of the drug, increasing their susceptibility to ADRs, while ultra-rapid metabolizers may experience therapeutic failure. Elderly patients and those with renal or hepatic comorbidities constitute particularly vulnerable subpopulations, further necessitating proactive pharmacist involvement.

Management and monitoring strategies led by pharmacists have demonstrated value in clinical settings. Regular assessment of therapy indication, laboratory monitoring (e.g., magnesium, renal function, vitamin B12), and deprescribing protocols significantly reduce the incidence of ADRs. Pharmacists also play a critical role in detecting potential drug interactions, such as with clopidogrel, where omeprazole-mediated CYP2C19 inhibition can attenuate antiplatelet effects, posing cardiovascular risks.

Despite these concerns, omeprazole remains a cornerstone in acid-suppression therapy due to its efficacy and convenience. The clinical challenge lies not in its use per se but in rationalizing its duration, dose, and indication, all of which fall within the scope of pharmacist-driven stewardship.

In summary, optimizing omeprazole use through clinical pharmacist oversight ensures therapeutic benefit while mitigating preventable harm. Incorporating pharmacists into multidisciplinary care teams enhances pharmacovigilance, improves patient education, and supports evidence-based deprescribing—especially vital as omeprazole continues to be one of the most widely consumed medications globally.

CONCLUSION

From a clinical pharmacist's perspective, the widespread and often long-term use of omeprazole necessitates vigilant therapeutic oversight. While omeprazole remains highly effective in managing acid-related gastrointestinal conditions, its misuse and overprescription have led to a rise in preventable adverse drug reactions, ranging from mild gastrointestinal disturbances to severe complications such as electrolyte imbalances, nutrient deficiencies, and renal injury. Clinical pharmacists are uniquely positioned to address these concerns through prospective medication

review, risk stratification, laboratory monitoring, and patient education. By actively evaluating indications, identifying high-risk patients, and promoting evidence-based deprescribing practices, pharmacists can significantly reduce the incidence of omeprazole-related ADRs. Furthermore, pharmacists serve as critical liaisons in ensuring interdisciplinary collaboration, particularly in managing drug–drug interactions and monitoring long-term therapy outcomes. Their role is central not only to improving the safety and efficacy of omeprazole therapy but also to enhancing overall patient care. Incorporating structured pharmacist-led interventions into standard care protocols will support rational PPI use, minimize long-term risks, and contribute to sustainable, patient-centered pharmacotherapy.

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Conflict of Interest:

The authors declare that there are no conflicts of interest regarding the publication of this article.

Authors Contributions:

A.G. and S.M. conducted the literature review, performed data collection, and contributed to drafting the manuscript. M.D.M. and R.M. assisted with reference compilation, formatting, and preliminary revisions. D.V.K. and D.N. provided critical academic guidance, supervision, and validation of the clinical content. A.V.K.S.G. conceptualized the study, coordinated the review process, finalized the manuscript, and ensured its overall intellectual integrity. All authors read, reviewed, and approved the final version of the manuscript.

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