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Drug-induced kidney injury from Acid-Suppressive Therapy: Mechanisms, clinical outcomes, and role of therapeutic nutrition

Purva Gopal Sharma[1], Payel Roy[2], Souvik Tewari[3], Poulami Biswas[4], Vijayalakshmi P[5*]
[1] Diabetes Educator, Food Science, Technology and Nutrition (FSTN) Exploration Foundation, West Bengal, India
[2] Food Processing and Nutrition Science, IIEST, Shibpur, Howrah, West Bengal, India
[3]Assistant Professor, Department of Food and Nutrition, Swami Vivekananda University, Barrackpore, West Bengal, India
[4] Dietetics and Food Service Management, IGNOU, West Bengal, India
[5] Professor and Head, Department of Pharmacology, JKKN Dental College and Hospital, Affiliated to Tamil Nadu Dr. MGR Medical University, Tamil Nadu, India

Abstract
Acid-suppressive therapies, particularly proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2RAs), are widely prescribed for the management of acid-related gastrointestinal disorders. Despite their clinical efficacy and perceived safety, emerging evidence links long-term and inappropriate use of these agents to drug-induced kidney injury (DIKI). This review explores the mechanisms underlying renal injury associated with acid-suppressive therapy, including acute interstitial nephritis (AIN), acute kidney injury (AKI), and progression to chronic kidney disease (CKD). The immunological, inflammatory, and oxidative pathways implicated in renal damage are discussed. Clinical outcomes, risk factors, and diagnostic challenges are highlighted. Additionally, the role of therapeutic nutrition in preventing and managing renal complications is emphasized, focusing on electrolyte balance, antioxidant intake, and dietary strategies for renal protection. This review underscores the importance of rational drug use, early detection of nephrotoxicity, and integration of nutritional interventions to improve patient outcomes.

Keywords: Proton pump inhibitors, acute interstitial nephritis, chronic kidney disease, nephrotoxicity, therapeutic nutrition, acid-suppressive therapy

Introduction

Acid-suppressive therapy, especially PPIs (e.g., omeprazole, pantoprazole) and H2 receptor antagonists, has revolutionized the treatment of peptic ulcer disease, gastroesophageal reflux disease (GERD), and dyspepsia (Mejia and Kraft, 2009) [12]. However, increasing global use—often without strict indications—has raised concerns regarding long-term adverse effects, particularly renal complications (De Mattos, *et al*., 2000) [3].

Recent epidemiological and clinical studies suggest a strong association between PPIs and kidney disorders, including AIN, AKI, CKD, and end-stage renal disease (ESRD) (Moledina and Perazella, 2016) [16]. Drug-induced kidney injury is now recognized as a significant contributor to morbidity and healthcare burden.

Acid-Suppressive Drugs and Renal Risk

1. Proton Pump Inhibitors (PPIs)

Proton pump inhibitors (PPIs) are the most commonly implicated agents in acid-suppressive therapy-related kidney injury, with accumulating evidence highlighting their nephrotoxic potential (Sanajou and Baydar, 2025) [22]. Clinical and epidemiological studies have demonstrated that PPI use is associated with a significantly increased risk of acute kidney injury (AKI) and acute interstitial nephritis (AIN), with reports indicating up to a two- to threefold higher risk compared to non-users. The pathogenesis is often linked to immune-mediated hypersensitivity reactions and inflammatory damage to the renal interstitium. Moreover, long-term PPI use has been associated with an increased incidence of chronic kidney disease (CKD) and progression to end-stage renal disease (ESRD), even in the absence of preceding acute kidney events. Importantly, the renal risk appears to be both dose- and duration-dependent, with prolonged exposure and higher cumulative doses exacerbating the likelihood of kidney dysfunction. These findings underscore the need for cautious prescribing, regular monitoring of renal function, and limiting PPI use to appropriate clinical indications (Sanajou and Baydar, 2025) [22].

2. Histamine-2 Receptor Antagonists (H2RAs)

Histamine-2 receptor antagonists (H2RAs), such as ranitidine and famotidine, are generally considered to have a safer renal profile compared to proton pump inhibitors (PPIs), with a lower incidence of nephrotoxic effects (Samborska *et al*., 2026) [21]. Unlike PPIs, H2RAs are less frequently associated with acute interstitial nephritis (AIN) or acute kidney injury (AKI), and available evidence suggests no significant association between H2RA use and the development of chronic kidney disease (CKD) (Fan *et al*., 2024) [7]. Their comparatively favorable safety profile may be attributed to a lower propensity to trigger immune-mediated renal inflammation (Solitano *et al*., 2024) [23]. Consequently, in patients at high risk of renal complications—such as the elderly or those with pre-existing kidney disease—H2RAs may be considered a preferable alternative for acid suppression when clinically appropriate (Almabruk *et al*., 2026) [1]. However, cautious use and appropriate dose adjustments are still recommended, particularly in individuals with impaired renal function, to prevent drug accumulation and potential adverse effects.

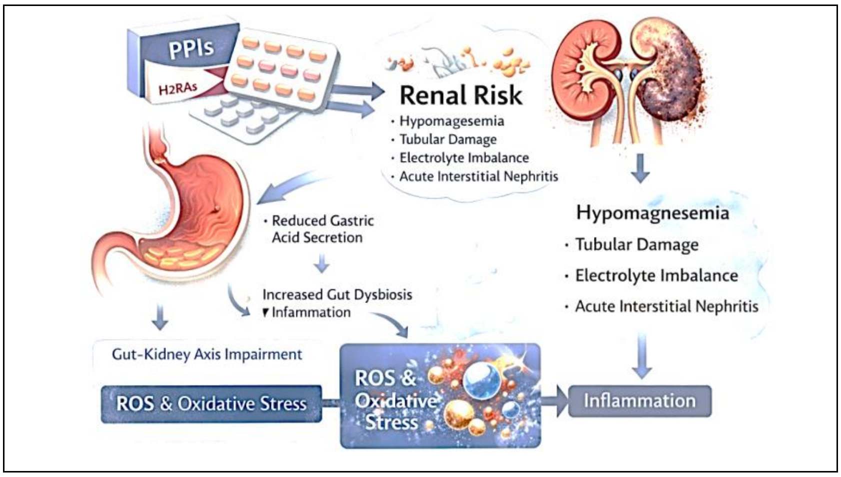

Fig 1: Pathophysiology of Acid-Suppressive Drugs and Associated Renal Risk

Mechanisms of Drug-Induced Kidney Injury

1. Acute Interstitial Nephritis (AIN)

Acute interstitial nephritis (AIN) is the most well-established mechanism underlying proton pump inhibitor (PPI)-induced nephrotoxicity and represents a significant cause of drug-induced kidney injury (Nast, 2017) [17]. It is characterized by inflammatory cell infiltration—primarily lymphocytes, eosinophils, and plasma cells—within the renal interstitium, leading to impaired tubular function and reduced glomerular filtration (Imig and Ryan, 2013) [9]. The condition is typically mediated by an immune hypersensitivity reaction, where the drug or its metabolites act as antigens, triggering T-cell–mediated immune responses and cytokine release (Pichler *et al*., 2011) [20]. Notably, AIN associated with PPIs can occur unpredictably and is not necessarily dependent on dose or duration of therapy, making early detection challenging (Moayyedi and Leontiadis, 2012) [15]. Clinically, it may present with nonspecific symptoms such as fatigue, rash, fever, and elevated serum creatinine levels (Mellstedt, 2007) [13]. AIN accounts for a substantial proportion of drug-induced acute kidney injury (AKI) cases, and if not promptly recognized and managed—primarily through discontinuation of the offending agent—it may progress to chronic kidney damage (Luciano and Perazella, 2018) [11].

2. Immunological Mechanisms

Immunological mechanisms play a central role in the pathogenesis of proton pump inhibitor (PPI)-induced kidney injury, particularly acute interstitial nephritis (AIN). In this process (Miao and Herrmann, 2023) [14], the drug or its metabolites act as haptens, binding to renal proteins and forming antigenic complexes that trigger an immune response. This leads to the activation of T-lymphocytes and the release of pro-inflammatory cytokines, resulting in hypersensitivity reactions within the renal interstitium (Hao *et al*., 2024) [8]. The subsequent inflammatory cascade causes infiltration of immune cells, tissue edema, and tubular damage (Diaz-Ricart *et al*., 2020) [4]. Evidence strongly suggests that PPI-induced AIN is predominantly immune-mediated, explaining its unpredictable occurrence and lack of clear dose-response relationship.

3. Oxidative Stress and Mitochondrial Damage

Oxidative stress and mitochondrial dysfunction are additional key mechanisms contributing to drug-induced kidney injury associated with acid-suppressive therapy (Yu *et al*., 2017) [26]. PPIs have been shown to increase the production of reactive oxygen species (ROS), leading to oxidative damage of renal tubular cells. Excess ROS disrupt mitochondrial integrity and impair energy production, resulting in cellular dysfunction (Song and Gong, 2023) [24]. Mitochondrial injury further promotes apoptosis (programmed cell death) and necrosis of renal cells, exacerbating tubular damage (Padanilam, 2003) [19]. These combined effects significantly contribute to the development and progression of acute kidney injury (AKI), highlighting the importance of oxidative pathways in PPI-related nephrotoxicity.

4. Electrolyte Imbalance and Magnesium Depletion

Electrolyte disturbances, particularly magnesium depletion, represent an important mechanism of kidney injury associated with prolonged proton pump inhibitor (PPI) use (Edinoff *et al*., 2023) [5]. PPIs can impair intestinal magnesium absorption by affecting transport channels such as TRPM6 and TRPM7, leading to hypomagnesemia. Chronic magnesium deficiency disrupts cellular homeostasis and adversely affects renal tubular function, as magnesium plays a critical role in maintaining membrane stability, enzymatic activity, and electrolyte balance. Hypomagnesemia may also contribute to secondary disturbances such as hypocalcemia and hypokalemia, further compromising renal function (Ehrenpreis *et al*., 2022) [6]. Additionally, reduced magnesium levels increase susceptibility to renal injury by promoting inflammation, oxidative stress, and vascular dysfunction. These alterations collectively heighten the risk of kidney damage, particularly in patients on long-term PPI therapy, emphasizing the need for regular monitoring of electrolyte levels and appropriate nutritional or pharmacological correction (Edinoff *et al*., 2023) [5].

5. Tubular Injury and Fibrosis

Tubular injury and subsequent fibrosis represent a critical pathway in the progression of drug-induced kidney damage associated with acid-suppressive therapy (Lewis and Stine, 2013) [10]. Persistent inflammation within the renal interstitium, often initiated by immune-mediated injury or oxidative stress, can lead to structural damage of renal tubules. Over time, this chronic inflammatory state promotes the activation of fibroblasts and excessive deposition of extracellular matrix components, resulting in renal fibrosis. This fibrotic remodeling reduces functional nephron mass and impairs the kidney's ability to maintain normal filtration and homeostasis. Consequently, unresolved or repeated episodes of acute kidney injury (AKI) may transition into chronic kidney disease (CKD), highlighting the long-term impact of sustained tubular damage and the importance of early intervention to prevent irreversible renal impairment (Yeh *et al*., 2024) [25].

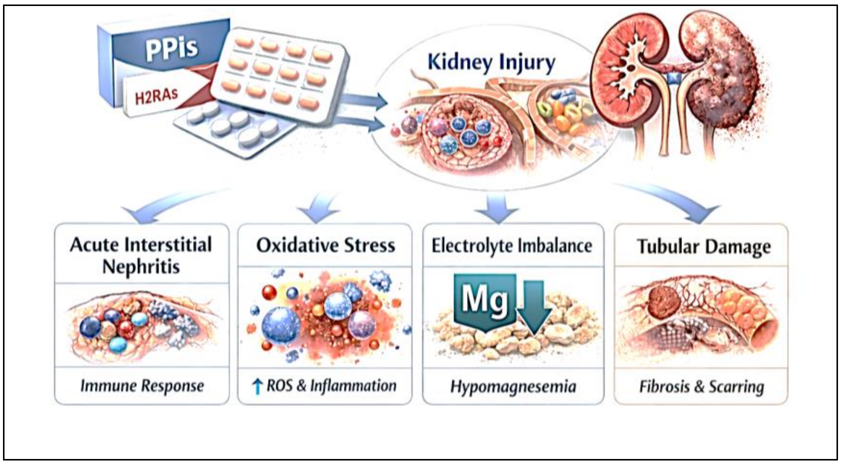

Fig 2: Mechanisms of Drug-Induced Kidney Injury Associated with Acid-Suppressive Therapy (PPIs and H2RAs)

Clinical Outcomes

1. Acute Kidney Injury (AKI)

Acute kidney injury (AKI) is one of the most common clinical outcomes associated with acid-suppressive therapy–induced nephrotoxicity, particularly with proton pump inhibitors. It is characterized by a sudden rise in serum creatinine levels, reflecting an abrupt decline in glomerular filtration rate. Clinically, patients may present with oliguria (reduced urine output), along with nonspecific symptoms such as fatigue, nausea, and malaise (Brown and Goldfarb-Rumyantzev, 2023) [2]. In many cases, AKI is linked to underlying acute interstitial nephritis or tubular injury. Importantly, this condition is often reversible if identified early and managed promptly, primarily through discontinuation of the offending drug and supportive care. However, delayed diagnosis or continued exposure to the causative agent may lead to incomplete recovery and increase the risk of progression to chronic kidney disease (Nochaiwong *et al*., 2018) [18].

2. Chronic Kidney Disease (CKD)

Chronic kidney disease (CKD) is a significant long-term outcome associated with prolonged use of proton pump inhibitors (PPIs), characterized by a gradual and progressive decline in renal function over time. Unlike acute kidney injury, CKD may develop insidiously and, in some cases, occur without any preceding episode of AKI, making early detection challenging. Epidemiological studies have reported a higher incidence of CKD among chronic PPI users, suggesting that sustained exposure may contribute to cumulative renal damage through mechanisms such as subclinical interstitial nephritis, oxidative stress, and fibrosis. This progressive deterioration in kidney function can ultimately impair metabolic and excretory processes, increasing morbidity and healthcare burden.

3. End-Stage Renal Disease (ESRD)

End-stage renal disease (ESRD) represents the most severe and irreversible consequence of drug-induced kidney injury, where kidney function declines to a level insufficient to sustain life without renal replacement therapy. Long-term exposure to PPIs has been associated with an increased risk of ESRD, particularly in individuals with underlying renal impairment or prolonged drug use. ESRD is characterized by extensive nephron loss, severe reduction in glomerular filtration rate, and accumulation of metabolic waste products. Patients with ESRD require lifelong dialysis or kidney transplantation, significantly impacting quality of life and increasing healthcare costs.

4. Prognosis

The prognosis of drug-induced kidney injury from acid-suppressive therapy largely depends on the timing of diagnosis and intervention. Early recognition and prompt discontinuation of the offending agent, particularly PPIs, can lead to substantial recovery of renal function in many patients, especially in cases of acute interstitial nephritis or AKI. However, delayed diagnosis or continued drug exposure may result in irreversible renal damage, progression to CKD, or even ESRD. In certain cases, particularly those involving immune-mediated injury such as AIN, corticosteroid therapy may be required to reduce inflammation and improve renal recovery. Therefore, vigilant monitoring and timely management are essential to improve clinical outcomes.

Risk Factors

Several risk factors increase the likelihood of developing drug-induced kidney injury associated with acid-suppressive therapy, particularly proton pump inhibitors (PPIs). Prolonged use of PPIs is a major contributor, as cumulative exposure enhances the risk of renal damage through chronic inflammatory and oxidative mechanisms. The elderly population is especially vulnerable due to age-related decline in renal function, altered drug metabolism, and higher prevalence of comorbidities. Polypharmacy further elevates the risk, particularly when PPIs are used concomitantly with nephrotoxic agents such as nonsteroidal anti-inflammatory drugs (NSAIDs) and certain antibiotics, which may have synergistic harmful effects on the kidneys. Additionally, individuals with pre-existing renal disease are at greater risk, as their reduced renal reserve makes them more susceptible to further injury. Dehydration and electrolyte imbalances, including hypomagnesemia, can also exacerbate renal stress and impair kidney function, thereby increasing the overall susceptibility to drug-induced nephrotoxicity.

Diagnosis and Monitoring

Accurate diagnosis and continuous monitoring are essential for the early detection and management of drug-induced kidney injury associated with acid-suppressive therapy. Key diagnostic parameters include measurement of serum creatinine and estimation of glomerular filtration rate (eGFR), which help assess renal function and identify any acute or chronic decline. Urinalysis is also important, as findings such as proteinuria and pyuria may indicate underlying renal inflammation or tubular damage. In suspected cases of acute interstitial nephritis (AIN), renal biopsy remains the gold standard for definitive diagnosis, allowing direct visualization of inflammatory changes in the renal interstitium. Additionally, a thorough drug history evaluation is crucial to identify potential nephrotoxic agents, particularly prolonged or inappropriate use of proton pump inhibitors. Early recognition and timely intervention are critical to prevent disease progression and minimize the risk of irreversible kidney damage.

Role of Therapeutic Nutrition in Management

1. Renal-Protective Diet

A renal-protective diet plays a crucial role in the management of drug-induced kidney injury, particularly in preventing progression to chronic kidney disease (CKD) and preserving residual renal function. Dietary protein intake should be moderated (approximately 0.6–0.8 g/kg body weight per day in CKD patients) to reduce nitrogenous waste production and lessen the burden on compromised kidneys. Sodium intake should be restricted to less than 2 g per day to help control blood pressure and minimize fluid retention, both of which are critical in maintaining renal health. Additionally, careful regulation of potassium and phosphorus intake is essential, especially in advanced stages of kidney dysfunction, as impaired renal excretion can lead to hyperkalemia and hyperphosphatemia. Overall, a balanced and individualized renal diet supports metabolic stability, reduces complications, and enhances clinical outcomes in patients with kidney injury.

2. Antioxidant-Rich Diet

An antioxidant-rich diet plays a significant role in mitigating oxidative stress associated with drug-induced kidney injury, particularly in patients exposed to proton pump inhibitors. Increased production of reactive oxygen species (ROS) contributes to renal cellular damage; therefore, the inclusion of antioxidant nutrients is essential for renal protection. Diets rich in fruits and vegetables provide vital antioxidants such as vitamins C and E, which help neutralize free radicals and reduce inflammation. Additionally, polyphenol-rich foods—such as berries, green tea, and other plant-based sources—exhibit strong antioxidant and anti-inflammatory properties that support renal cellular integrity. Regular consumption of these foods can help attenuate oxidative damage, improve overall kidney function, and potentially slow the progression of kidney disease.

3. Magnesium and Electrolyte Management

Magnesium and overall electrolyte balance are critical components in the nutritional management of drug-induced kidney injury, particularly in patients using proton pump inhibitors (PPIs), which are known to impair magnesium absorption. Adequate dietary intake of magnesium through foods such as nuts, seeds, whole grains, and legumes helps maintain normal cellular and renal function. Regular monitoring of serum electrolytes—including magnesium, potassium, and sodium—is essential to detect imbalances early and prevent complications. Preventing hypomagnesemia is especially important, as low magnesium levels can impair renal tubular function, exacerbate oxidative stress, and increase susceptibility to further kidney damage. Therefore, a well-planned diet combined with routine biochemical assessment supports electrolyte homeostasis and contributes to improved renal outcomes.

4. Hydration Therapy

Adequate hydration is a fundamental component in the management of drug-induced kidney injury, as it helps maintain optimal renal perfusion and supports normal kidney function. Sufficient fluid intake ensures proper blood flow to the kidneys, thereby preventing renal hypoperfusion, which can otherwise exacerbate tubular injury and reduce glomerular filtration rate. Hydration also facilitates the dilution and excretion of metabolic waste products and nephrotoxic substances, aiding in toxin elimination from the body. In patients receiving acid-suppressive therapy, especially those at risk of kidney impairment, maintaining appropriate fluid balance is essential to reduce renal stress and prevent further deterioration. However, fluid intake should be individualized based on the patient's clinical condition, particularly in cases of advanced kidney disease or fluid overload.

5. Functional Foods and Nutraceuticals

Functional foods and nutraceuticals offer promising supportive strategies in the management of drug-induced kidney injury by targeting inflammation, oxidative stress, and metabolic imbalances. Omega-3 fatty acids, commonly found in fatty fish, flaxseeds, and walnuts, possess potent anti-inflammatory properties that help reduce renal inflammation and slow disease progression. Probiotics also play a beneficial role by modulating the gut–kidney axis, improving gut microbiota composition, and reducing the production of uremic toxins that can further impair kidney function. Additionally, plant-based diets rich in whole grains, fruits, vegetables, and legumes provide essential nutrients, antioxidants, and fiber that support renal health while reducing acid load and metabolic stress on the kidneys. Together, these dietary approaches contribute to improved renal outcomes and serve as valuable adjuncts in the nutritional management of kidney injury.

6. Nutritional Intervention in AKI and CKD

Nutritional intervention is a cornerstone in the management of both acute kidney injury (AKI) and chronic kidney disease (CKD), requiring individualized planning based on the stage and severity of the condition. Tailored dietary strategies help maintain metabolic balance, prevent malnutrition, and support recovery of renal function. Adequate energy intake, typically ranging from 25–35 kcal/kg body weight per day, is essential to meet metabolic demands and prevent protein catabolism, especially in critically ill patients. Additionally, micronutrient supplementation—including vitamins (such as B-complex and vitamin D) and trace elements—may be necessary to address deficiencies arising from reduced intake, altered metabolism, or dialysis-related losses. A stage-specific and patient-centered nutritional approach plays a vital role in improving clinical outcomes, slowing disease progression, and enhancing overall quality of life.

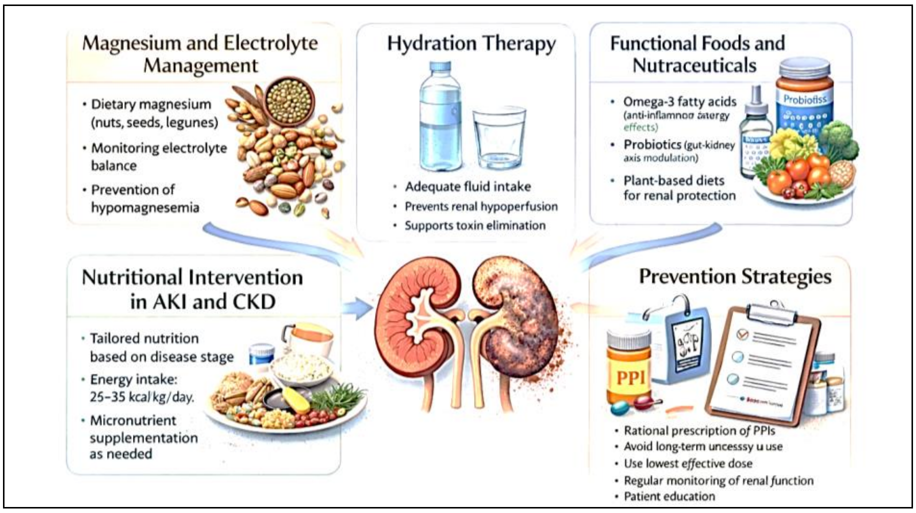

Fig 3: Role of Therapeutic Nutrition in the Management of Drug-Induced Kidney Injury

Prevention Strategies

Prevention of drug-induced kidney injury associated with acid-suppressive therapy primarily relies on judicious use of medications, particularly proton pump inhibitors (PPIs). Rational prescription practices are essential, ensuring that PPIs are used only when clinically indicated and for the appropriate duration. Avoiding long-term and unnecessary use is critical, as prolonged exposure significantly increases the risk of renal complications. Clinicians should aim to prescribe the lowest effective dose to achieve therapeutic benefits while minimizing adverse effects.

Regular monitoring of renal function through parameters such as serum creatinine and eGFR is also important for early detection of potential kidney impairment. Furthermore, patient education plays a key role in prevention, as informed individuals are more likely to adhere to prescribed regimens, avoid self-medication, and report early symptoms of renal dysfunction, thereby facilitating timely intervention and reducing the risk of irreversible kidney damage.

Table 1: Summary of Drug-Induced Kidney Injury from Acid-Suppressive Therapy

Aspect	Key Findings	Mechanism/Details	Clinical Implications
Drug Class	Proton Pump Inhibitors (PPIs)	Omeprazole, Pantoprazole commonly implicated	Higher nephrotoxicity risk
	H2 Receptor Antagonists (H2RAs)	Ranitidine, Famotidine	Lower renal risk compared to PPIs
Primary Mechanism	Acute Interstitial Nephritis (AIN)	Immune-mediated inflammation of renal interstitium	Major cause of drug-induced AKI
Immunological Response	Hypersensitivity reaction	T-cell activation, cytokine release	Unpredictable onset
Oxidative Stress	Increased ROS production	Mitochondrial damage, apoptosis	Contributes to AKI progression
Electrolyte Imbalance	Hypomagnesemia	Reduced intestinal Mg absorption	Tubular dysfunction, renal injury
Tubular Damage	Fibrosis and nephron loss	Chronic inflammation → fibrosis	Leads to CKD
Clinical Outcome	Acute Kidney Injury (AKI)	Sudden ↑ creatinine, oliguria	Often reversible
	Chronic Kidney Disease (CKD)	Progressive renal decline	May occur without AKI
	End-Stage Renal Disease (ESRD)	Irreversible kidney failure	Requires dialysis/transplant
Risk Factors	Long-term PPI use	Dose & duration dependent	Increased renal burden
	Elderly population	Reduced renal reserve	Higher susceptibility
	Polypharmacy	NSAIDs, antibiotics	Synergistic nephrotoxicity
Diagnosis	Serum creatinine, eGFR	Renal function markers	Early detection critical
	Urinalysis	Proteinuria, pyuria	Indicates inflammation
	Renal biopsy	Gold standard for AIN	Confirms diagnosis
Therapeutic Nutrition	Renal diet	Low protein, low sodium	Reduces kidney workload
	Antioxidants	Vitamins C, E, polyphenols	Reduces oxidative stress
	Magnesium intake	Nuts, seeds, legumes	Prevents hypomagnesemia
	Hydration	Adequate fluid intake	Maintains perfusion
	Functional foods	Omega-3, probiotics	Anti-inflammatory, gut-kidney axis
Prevention	Rational PPI use	Avoid unnecessary long-term use	Minimizes risk
	Monitoring	Regular renal assessment	Early intervention
	Patient education	Awareness of drug risks	Improves compliance

Conclusion

Drug-induced kidney injury from acid-suppressive therapy, particularly PPIs, represents a growing clinical concern. The primary mechanisms involve immune-mediated interstitial nephritis, oxidative stress, and electrolyte disturbances, which may lead to AKI and progression to CKD or ESRD. Early diagnosis and discontinuation of the offending agent are critical for recovery. Therapeutic nutrition plays a vital supportive role by mitigating oxidative stress, correcting electrolyte imbalances, and preserving renal function. A multidisciplinary approach integrating pharmacological vigilance and nutritional therapy is essential to reduce the burden of PPI-associated nephrotoxicity.

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