

Herbal Strategies Against Drug-Induced Nephrotoxicity: A Review of Experimental Studies on Plant-Based Therapies

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Abstract

*Drug-induced nephrotoxicity is a major clinical challenge, often limiting the therapeutic use of essential medications such as antibiotics, chemotherapeutic agents, and NSAIDs. Conventional strategies for managing nephrotoxicity—such as dose adjustment or drug discontinuation—are largely supportive and fail to provide targeted renal protection. In response, increasing attention has turned toward herbal therapies as safer and potentially more effective alternatives. This review consolidates experimental findings on various medicinal plants with documented nephroprotective potential. Mechanistic insights reveal that phytochemicals—such as flavonoids, alkaloids, polyphenols, and terpenoids—exert renoprotective effects primarily through antioxidant, anti-inflammatory, and anti-apoptotic pathways. A wide array of plant species, including *Nigella sativa*, *Curcuma longa*, *Zingiber officinale*, and *Phyllanthus niruri*, have demonstrated efficacy in animal models of nephrotoxicity induced by drugs such as gentamicin, cisplatin, and cyclosporine. These findings underscore the therapeutic promise of plant-based interventions and support further translational research toward developing standardized phytopharmaceuticals for renal protection.*

Keywords: *Drug-induced nephrotoxicity; Herbal medicine; Nephroprotection; Medicinal plants; Oxidative stress; Phytochemicals; Renal injury; Antioxidants.*

1. Introduction

Drug-induced kidney injury, also known as nephrotoxicity, represents a significant clinical concern due to the kidneys' central role in eliminating waste products and maintaining metabolic balance. The kidneys perform a wide array of physiological functions essential to systemic homeostasis, including regulation of the extracellular environment, detoxification of

harmful compounds, and excretion of metabolic by-products and xenobiotics, including drugs. Given these roles, the kidneys are particularly susceptible to injury from exogenous toxicants, making them a frequent target of drug-induced damage [1].

Nephrotoxicity refers to kidney dysfunction that arises when the excretion of waste materials is compromised due to exposure to toxic substances, particularly pharmaceuticals [2,3]. While drug-induced nephrotoxicity accounts for approximately 20% of all cases, the prevalence significantly increases in elderly populations—rising to nearly 66%—largely due to polypharmacy and age-related physiological changes in renal function [4-6]. The nephrotoxic potential of various therapeutic agents, especially chemotherapeutic drugs, has become a major limitation in their clinical application, restricting their dosage and duration of use due to the risk of renal impairment.

Nephrotoxicity is mediated through several overlapping mechanisms including oxidative stress, inflammatory responses, mitochondrial dysfunction, and cell death via apoptosis or necrosis. As drugs and their metabolites are filtered and concentrated in renal tubular cells—particularly within the proximal tubules—these cells are frequently the first to sustain damage. Renal tubular injury can impair reabsorption, secretion, and filtration processes, ultimately leading to a decline in kidney function [7,8].

2. Mechanisms of Drug-Induced Nephrotoxicity

The pathophysiology of drug-induced nephrotoxicity involves multiple complex mechanisms, including oxidative stress, inflammation, mitochondrial dysfunction, and apoptotic or necrotic cell death. The kidneys, particularly the proximal tubules, are involved in the active transport and elimination of many drugs and their metabolites. As a result, these cells are often exposed to high drug concentrations, making them susceptible to damage. Several key factors contribute to nephrotoxicity, including:

2.1 Accumulation of toxic metabolites: Some drugs are metabolized into toxic intermediates that can accumulate in renal tissues, causing damage. For instance, certain chemotherapeutic agents like cisplatin generate highly reactive metabolites that induce cellular damage in renal tubules [9].

2.2 Disruption of cellular integrity: Drugs may impair cellular functions by altering ion homeostasis, disrupting cell membrane integrity, or inducing mitochondrial dysfunction, all of which contribute to kidney cell death [10].

2.3 Oxidative stress: Many nephrotoxic drugs increase the generation of reactive oxygen species (ROS), leading to oxidative damage to lipids, proteins, and DNA. This oxidative damage plays a pivotal role in the pathogenesis of kidney injury [11].

2.4 Inflammatory responses: Drugs may also initiate inflammatory responses in the kidneys, involving the activation of cytokines, chemokines, and immune cells that further exacerbate kidney damage [12].

3. Common Drugs Inducing Nephrotoxicity

Several substances are known to cause kidney damage due to their toxic effects on renal tissues. These include:

3.1 Heavy Metals: Toxic elements such as mercury, arsenic, lead, and bismuth.

3.2 Anticancer (Antineoplastic) Drugs:

Alkylating Compounds: include cisplatin and cyclophosphamide.

Nitrosourea Derivatives: Agents like streptozotocin, carmustine, lomustine, and semustine.

Antimetabolites: High doses of drugs such as methotrexate, cytarabine (cytosine arabinoside), 6-thioguanine, and 5-fluorouracil.

Antitumor Antibiotics: like mitomycin, mithramycin, and doxorubicin.

Biologic Therapies: Recombinant forms of leukocytes and interferons.

3.3 Anti-Infective Agents: Certain antibiotics and antivirals, including tetracycline, acyclovir, pentamidine, sulfadiazine, trimethoprim, rifampin, and amphotericin B.

3.4 Aminoglycoside Antibiotics: gentamicin, amikacin, kanamycin, and streptomycin.

3.5 Other Notable Agents:

Radiographic Contrast Media: Often used in diagnostic imaging, these can impair kidney function, especially in at-risk individuals.

Nonsteroidal Anti-inflammatory Drugs (NSAIDs): Medications like ibuprofen, indomethacin, and aspirin.

4. Need for Alternative Therapies in Drug-Induced Nephrotoxicity

Conventional drugs like antibiotics, NSAIDs, and chemotherapeutics are essential but often cause nephrotoxicity, particularly in vulnerable populations such as the elderly or those with renal impairment [13]. Agents like gentamicin and cisplatin, though clinically effective, have narrow safety margins due to dose-dependent renal toxicity [14]. Current strategies—such as hydration, dose adjustment, or drug withdrawal—are supportive and insufficient in fully

preventing or reversing kidney damage [15]. Additionally, no universally accepted nephroprotective drugs exist.

In this context, plant-based therapies have emerged as promising alternatives. Natural products rich in flavonoids, alkaloids, and polyphenols exhibit antioxidant and anti-inflammatory properties, potentially reducing renal injury by countering oxidative stress and stabilizing cell membranes [16]. Growing evidence supports their role in complementing conventional treatment with improved safety and patient tolerance [17]. Therefore, exploring phytochemicals through well-designed studies could fill a critical gap in managing drug-induced kidney injury.

4.1 Medicinal Plants with Renal Protective Properties

A growing body of preclinical research supports the nephroprotective potential of various medicinal plants. These plants exert renal protective effects primarily through their antioxidant, anti-inflammatory, and cytoprotective mechanisms. Several plant species have demonstrated promising results in experimental models of drug-induced nephrotoxicity.

Table 1. Medicinal plants with nephroprotective activity, their phytoconstituents, and experimental models used.

Plant Name	Family	Part used	Chemical constituents	Model used for screening
Nigella Sativa	Ranunculaceae	Seed oil (cold pressed)	Fixed oils, Essential oils, Thymoquinone, p-Cymene, Carvacrol, 4-Terpineol, trans-Anethole, Longifolene	gentamicin-induced
Curcuma longa	Zingiberaceae	Rhizome	Curcumin, Demethoxycurcumin, Bisdemethoxycurcumin, Volatile oils, turmerone, atlantone, zingiberene	Cisplatin-induced
Zingiber officinale	Zingiberaceae	Rhizome from which 6-Shogaol was extracted	Zingiberene (major sesquiterpene), β -Sesquiphellandrene, Bisabolene, Farnesene, Curcumene, Camphene, Cineole, Citral, Limonene 6-gingerol, paradols, zingerone, quercetin, kaempferol	Cisplatin-Induced

Annona squamosa	Annonaceae	Ethanollic extract of Leaves	annonaine, asimilobine, coreximine, quercetin, kaempferol, β -sitosterol, stigmasterol, annonacin, oleic acid, linoleic acid, and palmitic acid	paracetamol-induced
Phyllanthus niruri	Euphorbiaceae	Whole plant methanolic extract	Phyllanthin, Hypophyllanthin, Niranthin, Nirtetralin, Phyltetralin, Isolintetralin, Gallic acid, Ellagic acid, Corilagin, 4-Methoxy-securinine, Dihydrosecurinine, Norsecurinine, Lupeol, β -Sitosterol, Friedelin	Gentamicin induced
Aegle marmelos	Rutaceae	leaves	Rutin, Quercetin, Kaempferol, Isorhamnetin, Aegeline, Skimmianine, Marmeline, β -Sitosterol, Stigmasterol, Campesterol, Marmelosin, Limonene, Aegelinol	Gentamicin induced
Polyalthia longifolia	Annonaceae	Roots (ethanolic extract)	pendulamine A, 13-halimadien-15-oic acid, O-methyl bulbocapnin-N-oxide, N-methyl nandigerine-N-oxide, Oliveroline-N-oxide, 8-oxopolyalthiane, 8-Oxopolyalthiane, Ellagitannins, Gallotannins	vancomycin-induced
Costus afer	Zingiberaceae	Leaves (aqueous leaf extract)	Securinine, 4-Methoxy-securinine, Dihydrosecurinine, Norsecurinine, Phyllanthine, Phyllanthoside, β -Amyrin glycosides, Luteolin, Apigenin, Geraniin, Proanthocyanidins	Cyclosporine-A
Homonoia riparia	Euphorbiaceae	Whole plant	Gallic acid, Quercetin glycosides, Hydrolyzable tannins, β -sitosterol,	in vitro using cisplatin (8 μ g/mL) on

			Cycloartane-type triterpenoid saponins, Taxerone	Human Embryonic Kidney (HEK 293)
Sansevieria roxburghiana	Agavaceae	Leaves	Quercetin, Kaempferol, Luteolin, Gallic acid, Caffeic acid, Ferulic acid Roxburghianosides, quercetin-3-O-rutinoside, Chlorogenic acid, β -Sitosterol, Stigmasterol, Cyanidin-3-glucoside, Delphinidin derivatives	Gentamicin induced
Andrographis paniculata	Acanthaceae	Leaves	Andrographolide, neoandrographolide, Wogonin, apigenin, Caffeic acid, chlorogenic acid	Gentamicin-induced
Kalanchoe pinnata	Crassulaceae	Leaves	Quercetin, kaempferol, quercetin-3-L-rhamnosido-L-arabinofuranoside, α -Amyrin, β -amyrin, p-Coumaric, ferulic, caffeic acids, β -Sitosterol, n-Hentriacontane, n-triacontane	Gentamicin-induced
Moringa peregrina	Moringaceae	Leaves	chlorogenic acid, rutin, p-coumaric acid, caffeic acid, and 3,6,2',4'-tetrahydroxyflavone, gallic acid, quercetin	Gentamicin-induced
Vitis vinifera	Vitaceae	Aerial parts	Chlorogenic acid, p-coumaric acid, caffeic acid, Rutin, 3,6,2',4'-tetrahydroxyflavone	Carbon Tetrachloride-Induced
Dialium guineense	Fabaceae	Fruit pulp	Trigonelline, legumes, Gallic acid, ellagic acid, β -sitosterol, lupeol, Quercetin, kaempferol, or rutin	Cisplatin-Induced Kidney
Wedelia chinensis	Asteraceae	Leaves	Apigenin, Luteolin, Wedelolactone, β -amyrin, Oleanolic acid, Ursolic acid Chlorogenic acid, Caffeic acid	Gentamicin-induced
Hedychium spicatum	Zingiberaceae	Rhizome	1,8-Cineole, α -Terpinene, Limonene, α -Phellandrene, p-	Streptozotocin induce

			Cymene, Linalool, α -Terpineol, Sabinene, β -Pinene, Camphene, Lupeol, Oleanolic acid, Ursolic acid	diabetes-associated nephropathy
Carica papaya	Caricaceae	Seeds	Carpaine, Pseudocarpaine, Dehydrocarpaine I and II, Kaempferol, Quercetin, Caffeic acid, Chlorogenic acid, Gallic acid, Lupeol, β -amyrin, Oleanolic acid, Ursolic acid, Papain, Chymopapain, Lysozyme, Caricain	carbon tetrachloride (CCl ₄) in olive oil
Cichorium intybus	Asteraceae	Roots	Quercetin, Luteolin, Apigenin, Kaempferol, Rutin, Isoquercetin, β -Amyrin, Lupeol, Taraxasterol, Cichotyboside, Cichoriolide A & B, Chicoric acid glycosides, Cichoriin Chicoric acid, Ferulic acid, Protocatechuic acid, p-Coumaric acid	Gentamicin-induced
Zea mays	Poaceae	Husk	Anthocyanins, stigmasterol, β -sitosterol, p-hydroxycinnamic acid, octadecanoic acid, gallic acid, protocatechuic acid, chlorogenic acid	Gentamicin-induced

Nigella sativa

Yamana I. et al. (2009) examined the nephroprotective efficacy of *Nigella sativa* oil in gentamicin-induced nephrotoxicity in rats. Gentamicin administration led to elevated creatinine, urea, MDA, and NO levels, along with reduced SOD and GSH-Px activities and marked renal histopathology. Co-administration of *N. sativa* (0.2 and 0.4 mL/kg) significantly restored renal function and antioxidant status ($p < 0.05$). The findings highlight the potent antioxidant and free-radical scavenging effects of *N. sativa* in mitigating aminoglycoside-induced renal injury.

Curcuma longa

Tarladacalisir Y.T. et al. (2016) evaluated curcumin's nephroprotective effect against cisplatin-induced renal toxicity in rats. Pretreatment with curcumin (200 mg/kg) significantly reduced oxidative stress, inflammation (\downarrow MPO, \downarrow IL-6, \uparrow IL-10), and apoptosis (\downarrow caspase-3, \downarrow p53) markers, while preserving renal histoarchitecture ($p < 0.05$). The study underscores curcumin's ability to attenuate renal damage through its antioxidant, anti-inflammatory, and anti-apoptotic properties, supporting its potential as an adjuvant therapy in cisplatin-based chemotherapy.

Zingiber officinale

Gwon M. et al. (2021) investigated 6-shogaol's nephroprotective role against cisplatin-induced acute kidney injury. 6-shogaol markedly improved renal biomarkers and histology by reducing oxidative stress (\downarrow MDA, \uparrow GSH/GSSG), inhibiting apoptosis and necroptosis, and suppressing inflammatory mediators and immune cell infiltration. The study reveals the compound's broad cytoprotective mechanism, suggesting its utility as a multifaceted therapeutic agent against cisplatin-induced nephrotoxicity.

Annona squamosa

S. Neelima et al. (2020) assessed the nephroprotective potential of *Annona squamosa* leaf extract against paracetamol-induced renal damage in vitro and in vivo. The extract reversed oxidative stress and elevated biomarkers, improving GSH, SOD, and CAT levels and restoring kidney function ($p < 0.001$). Histological findings confirmed renal tissue recovery. These effects were attributed to its flavonoids, tannins, and polyphenols, suggesting *A. squamosa* as a promising phytotherapeutic candidate for renal protection.

Phyllanthus niruri

Reddy G.S. et al. (2015) explored the protective effect of methanolic *Phyllanthus niruri* extract against gentamicin-induced nephrotoxicity. Treatment with 200 and 400 mg/kg extract significantly normalized serum creatinine, BUN, and oxidative stress markers while improving GSH levels. Histological analysis showed reduced tubular necrosis. These findings support *P. niruri*'s nephroprotective role, likely mediated by its potent antioxidant constituents.

Aegle marmelos

Kalita B. et al. (2017) investigated the nephroprotective and nephrocurative effects of *Aegle marmelos* leaf extract in gentamicin-induced nephrotoxicity. Administration of 500 mg/kg aqueous extract significantly decreased BUN and creatinine levels and improved renal histology ($p < 0.001$). While protective effects were evident, curative effects were minimal compared to spontaneous recovery. The results affirm the prophylactic renal benefits of *A. marmelos*.

Polyalthia longifolia

Das K. et al. (2023) evaluated the nephroprotective efficacy of *Polyalthia longifolia* root extract against vancomycin-induced renal injury. High-dose PL (400 mg/kg), especially when combined with selenium (6 mg/kg), significantly improved renal function by reducing serum BUN, creatinine, and potassium levels ($p < 0.01$). The findings suggest synergistic antioxidant effects of PL and selenium, highlighting their therapeutic potential in combating drug-induced nephrotoxicity.

Costus afer

Ezeji for A.N. et al. (2016) assessed the nephroprotective and antioxidant potential of *Costus afer* aqueous extract in cyclosporin A-induced nephrotoxicity in rats. Doses of 375–1125 mg/kg markedly improved renal biomarkers and antioxidant enzyme levels (GSH, SOD, CAT, GST), while reducing MDA levels. Histopathological findings supported renal protection. The study concludes that *C. afer* confers significant protection against oxidative kidney damage, reinforcing its value in nephrotoxic drug management.

Homonoia riparia

Xavier S.K. et al. (2016) examined the nephroprotective and antioxidant activity of methanolic extract and fractions of *Homonoia riparia*. Using cisplatin-induced HEK-293 cell toxicity, butanol and aqueous fractions showed the highest cell viability. Antioxidant assays (DPPH, ABTS, NO) and HPTLC confirmed phenolic richness, especially gallic acid. The study supports the therapeutic relevance of *H. riparia* as a nephroprotective agent via antioxidant mechanisms.

Sansevieria roxburghiana

Aclan J.B. et al. (2020) investigated the nephroprotective effects of methanolic *Sansevieria roxburghiana* extract in gentamicin-induced nephrotoxicity. Co-administration (250–500 mg/kg) significantly improved serum creatinine and BUN levels and reduced histological damage ($p < 0.05$), comparable to ascorbic acid. The observed renal protection is likely due to antioxidant phytochemicals, supporting its therapeutic potential against aminoglycoside-induced renal injury.

Andrographis paniculata

Padmalochana K. et al. (2017) evaluated the nephroprotective potential of *Andrographis paniculata* leaf extracts (aqueous, ethanol, acetone) against gentamicin-induced renal toxicity in Wistar rats. Gentamicin (80 mg/kg, oral) elevated serum urea, uric acid, and creatinine ($*p < 0.05$), while reducing total protein. Ethanol extract (300 mg/kg) exhibited superior efficacy ($*p < 0.001$), normalizing biochemical markers and restoring renal histoarchitecture. Phytoconstituents like andrographolide and flavonoids likely mediated antioxidant and anti-

inflammatory effects. Findings validate *A. paniculata* as a promising nephroprotective agent, supporting its traditional use in renal disorders.

Kalanchoe pinnata

Harlalka G.V. *et al.* (2007) evaluated the nephroprotective potential of *Kalanchoe pinnata* aqueous leaf extract against gentamicin-induced renal damage in Wistar rats. Gentamicin (100 mg/kg, i.p.) administration caused elevated serum creatinine, BUN, and renal histopathological alterations. Co-treatment with *K. pinnata* (125 mg/kg, i.p.) significantly restored biochemical parameters and mitigated tissue injury ($p < 0.05$). In vitro assays revealed strong antioxidant activity (DPPH EC₅₀: 116.25 µg/mL; NO inhibition), likely attributed to flavonoids such as quercetin and kaempferol. These findings support *K. pinnata* as a promising agent for preventing drug-induced nephrotoxicity.

Moringa peregrine

Hasan A. *et al.* (2022) evaluated the nephroprotective effects of *Moringa peregrina* leaf aqueous extract against gentamicin-induced renal toxicity in mice. The extract exhibited high phenolic (555.57 mg/g) and flavonoid (40.08 mg/g) content with potent antioxidant capacity (DPPH IC₅₀: 3.10 µg/mL). HPLC-MS/MS identified ten bioactive constituents. Pre-treatment with the extract (500–1000 mg/kg) significantly reduced serum and urinary nephrotoxicity markers and improved renal histopathology. These results support *M. peregrina*'s therapeutic potential in renal protection through its antioxidant-rich phytochemical profile.

Vitis vinifera

Kumar A. *et al.* (2024) evaluated the hepatoprotective and nephroprotective potential of *Vitis vinifera* ethanolic extract against CCl₄-induced organ toxicity in rats. Pretreatment with the extract (100 and 200 mg/kg) significantly lowered elevated liver (AST, ALT, ALP, bilirubin) and kidney (creatinine, urea, uric acid) biomarkers, with the 200 mg/kg dose showing effects comparable to silymarin. Histological analysis revealed reduced hepatic necrosis and renal structural damage. The protective effects are attributed to the extract's polyphenolic antioxidants, supporting *V. vinifera*'s role in preventing toxin-induced liver and kidney injury.

Dialium guineense

Nyarko J.A. *et al.* (2024) investigated the nephroprotective effects of *Dialium guineense* fruit pulp aqueous extract against cisplatin-induced renal injury in rats. Phytochemical screening confirmed the presence of flavonoids, phenols, alkaloids, tannins, saponins, and terpenoids. Pretreatment with the extract (100–500 mg/kg) dose-dependently reduced serum creatinine, urea, AST, and ALT levels, with 100 mg/kg showing optimal efficacy. Histopathological analysis revealed reduced tubular necrosis and inflammation, with preserved renal structure. The protective effects are attributed to antioxidant phytochemicals, supporting *D. guineense* as a potential nephroprotective agent.

Wedelia chinensis

Gautam D.T et al. (2023) investigated the nephroprotective effects of *Wedelia chinensis* hydroalcoholic leaf extract (WCHAE) against gentamicin-induced renal injury in rats. Phytochemical analysis confirmed rich flavonoid and phenolic content. WCHAE exhibited strong antioxidant activity in vitro and molecular docking identified quercetin, rutin, and apigenin as key ligands targeting KIM-1 and NGAL. In vivo, WCHAE (500 mg/kg) significantly reduced serum creatinine, urea, and uric acid, and restored antioxidant enzymes. Histological analysis showed reduced tubular damage, supporting WCHAE's efficacy in gentamicin nephrotoxicity through antioxidant-mediated protection.

Hedychium spicatum

Mittal R. et al. (2020) investigated the antidiabetic and nephroprotective effects of *Hedychium spicatum* ethanolic rhizome extract in streptozotocin-induced diabetic rats. Phytochemical screening confirmed the presence of flavonoids, alkaloids, and terpenoids. Treatment (100–500 mg/kg) over 40 days significantly reduced blood glucose, BUN, creatinine, lipid peroxidation, and urinary albumin, while enhancing antioxidant enzymes (GSH, SOD, CAT). Histological analysis showed preserved glomerular structure and reduced tubular damage, especially at higher doses. The results suggest *H. spicatum* offers renal protection via hypoglycemic and antioxidant pathways, supporting its therapeutic potential in diabetic nephropathy.

Carica papaya

Sultana M.S. et al. (2022) the nephroprotective effects of aqueous *Carica papaya* seed extract (CPE) against CCl₄-induced renal toxicity in rats. Pretreatment with CPE (250–500 mg/kg/day) significantly reduced elevated serum creatinine and urea levels, particularly at 500 mg/kg ($p < 0.001$), compared to the CCl₄ group. Histological analysis revealed preserved renal structure in treated rats. The renoprotective effect is attributed to the extract's antioxidant phytochemicals, supporting the therapeutic potential of *C. papaya* seeds in managing chemically induced kidney damage.

Cichorium Intybus

Sharma P. et al. (2025) examined the nephroprotective effects of the ethyl acetate fraction of *Cichorium intybus* root extract (EAFCI) against gentamicin-induced renal injury in rats. Pretreatment with EAFCI (200–400 mg/kg) significantly reduced serum urea, creatinine, and BUN levels ($p < 0.01$), with greater efficacy at 400 mg/kg. Histological analysis showed preserved renal structure and reduced tubular damage. The protective effect is linked to flavonoid-rich antioxidant activity, supporting EAFCI's therapeutic potential.

Zea mays

Okokon J.E. et al. (2019) investigated the nephroprotective potential of *Zea mays* husk ethanol extract against gentamicin-induced renal toxicity in rats. Pretreatment with the extract (187–748 mg/kg) significantly reduced serum creatinine, urea, and electrolyte disturbances ($p < 0.05$ – 0.001), with histological analysis showing preserved renal structure and reduced tubular damage. The observed renoprotection is linked to antioxidant-rich compounds, including anthocyanins and phenolics, supporting the traditional use of *Z. mays* husk in renal protection.

5. Conclusion

Herbal medicines present a promising frontier in the prevention and management of drug-induced nephrotoxicity. The evidence compiled in this review highlights the potent nephroprotective properties of numerous plant extracts, often mediated by their antioxidative and anti-inflammatory bioactive compounds. While the preclinical data are compelling, clinical translation remains limited due to variability in extract standardization, dosage, and study design. Future research must prioritize well-designed clinical trials, phytochemical characterization, and safety profiling to validate these natural agents as adjuncts or alternatives to conventional nephroprotective strategies. Harnessing the therapeutic power of medicinal plants could bridge a critical gap in nephrology and improve patient outcomes in drug-induced kidney injury.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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