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Drug-induced Kidney Injury: Prevention and Detection

Oliver Hastings*

Department of Medicine Renal Division, Kingsford Biomedical University, Northbridge, UK

Introduction

Drug-induced nephrotoxicity represents a substantial and complex clinical issue, stemming from the use of a wide array of pharmacological agents. The underlying mechanisms contributing to this adverse effect are diverse and often involve direct damage to the renal tubules, the induction of interstitial inflammation within the kidneys, or injury to the renal vasculature. Proactive prevention strategies are critical and hinge on a multifaceted approach that includes judicious drug selection, meticulous dose monitoring, accurate patient risk stratification based on individual factors, and the prompt and accurate recognition of early indicators of declining kidney function.

Antimicrobial agents are consistently identified as a primary category of drugs responsible for inducing kidney injury, with specific classes such as aminoglycosides and vancomycin posing particularly elevated risks. A thorough understanding of both dose-dependent toxic effects and idiosyncratic reactions is paramount for effective management. Consequently, implemented strategies often involve rigorous therapeutic drug monitoring and, where clinically feasible, the judicious selection of alternative antimicrobial agents with a lower nephrotoxic profile.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are well-recognized for their capacity to impair renal function, primarily through the inhibition of prostaglandin synthesis. This mechanism can lead to a significant reduction in renal blood flow, potentially culminating in acute kidney injury. Several risk factors are known to exacerbate this effect, including states of dehydration, the presence of pre-existing kidney disease, and the concurrent administration of other nephrotoxic medications.

Contrast-induced nephropathy (CIN) continues to be a significant concern and a recognized complication following the administration of radiocontrast agents. Established prevention strategies encompass ensuring adequate patient hydration, minimizing the total volume of contrast agent administered, and prioritizing the utilization of renal-sparing contrast agents whenever clinically appropriate. The specific role and efficacy of N-acetylcysteine in preventing CIN remain a subject of ongoing debate and investigation.

Chemotherapeutic agents, with particular emphasis on platinum-based drugs and certain tyrosine kinase inhibitors, are associated with a spectrum of nephrotoxicity mechanisms. These can include direct tubular damage and the development of crystal nephropathy within the renal tubules. Therefore, careful patient selection prior to initiating therapy and appropriate dose adjustments throughout the treatment course are essential for mitigating renal risks.

Immunosuppressants, notably calcineurin inhibitors such as cyclosporine and tacrolimus, are indispensable in post-transplantation management. However, their use carries a substantial risk of inducing chronic kidney disease. Strategies to mitigate this risk involve close monitoring of drug trough levels and, when necessary, the consideration of newer immunosuppressive agents or the implementation of combination therapies.

The consumption of herbal and alternative medicines presents another potential source of nephrotoxicity. The mechanisms underlying these effects are frequently not well-understood, largely owing to a lack of standardization and regulatory oversight for these products. Consequently, the proactive reporting of adverse events by patients and heightened awareness among healthcare providers are crucial for the timely identification of such cases.

Acute interstitial nephritis (AIN) stands out as a common manifestation of drug-induced kidney injury, often triggered by hypersensitivity reactions to various medications. The cornerstone of management for AIN involves the prompt identification and discontinuation of the offending agent. In select cases, the administration of corticosteroids may be considered to ameliorate the inflammatory response within the renal interstitium.

Biomarkers that facilitate the early detection of drug-induced kidney injury are currently an active area of research and development. The ultimate goal is to identify renal damage at its earliest stages, ideally before significant functional decline becomes apparent. Promising candidates for such biomarkers include neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1).

Pharmacogenomics holds significant promise as a future approach for the personalized prevention of drug-induced nephrotoxicity. By identifying individuals who possess a genetic predisposition for a higher risk of kidney damage, this personalized medicine strategy allows for the tailoring of drug selection and precise dose adjustments, thereby enhancing patient safety.

Description

Drug-induced nephrotoxicity is a significant clinical challenge, arising from diverse pharmacological agents. The mechanisms often involve direct tubular damage, interstitial inflammation, or vascular injury. Prevention hinges on careful drug selection, dose monitoring, patient risk stratification, and prompt recognition of early signs of kidney dysfunction.

Antimicrobials are a leading cause of drug-induced kidney injury, with aminoglycosides and vancomycin posing particular risks. Understanding dose-dependent and idiosyncratic toxicity is key. Strategies include therapeutic drug monitoring and alternative agent selection when possible.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can impair renal function through prostaglandin inhibition, leading to reduced renal blood flow and potentially acute kidney injury. Risk factors include dehydration, pre-existing kidney disease, and concomitant use of other nephrotoxic agents.

Contrast-induced nephropathy (CIN) remains a significant concern after radiocontrast administration. Prevention strategies include adequate hydration, minimizing contrast volume, and utilizing renal-sparing contrast agents when feasible. N-acetylcysteine's role is debated.

Chemotherapeutic agents, particularly platinum-based drugs and tyrosine kinase inhibitors, are associated with varied mechanisms of nephrotoxicity, including tubular damage and crystal nephropathy. Careful patient selection and dose adjustments are crucial.

Immunosuppressants, such as calcineurin inhibitors (cyclosporine, tacrolimus), are vital in transplantation but pose a significant risk of chronic kidney disease. Monitoring trough levels and considering newer agents or combination therapies can mitigate risk.

The use of herbal and alternative medicines can also lead to nephrotoxicity through various mechanisms, often poorly understood due to lack of standardization and regulation. Patient reporting and physician awareness are crucial for identification.

Acute interstitial nephritis (AIN) is a common pattern of drug-induced kidney injury, often mediated by hypersensitivity reactions. Identifying and discontinuing the offending agent is paramount, with corticosteroids sometimes used to reduce inflammation.

Biomarkers for early detection of drug-induced kidney injury are under active investigation, aiming to identify renal damage before significant functional decline. Neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1) show promise.

Pharmacogenomics offers a future avenue for personalized prevention of drug-induced nephrotoxicity by identifying individuals at higher risk based on their genetic makeup. This approach allows for tailored drug selection and dosing.

Conclusion

Drug-induced nephrotoxicity is a prevalent clinical concern arising from numerous medications, including antimicrobials, NSAIDs, chemotherapeutics, immunosuppressants, and contrast agents. Mechanisms vary from direct tubular damage and inflammation to vascular injury and prostaglandin inhibition. Prevention strategies involve careful drug selection, dose monitoring, risk assessment, and prompt recognition of renal dysfunction. Acute interstitial nephritis is a common pattern often caused by hypersensitivity. Herbal and alternative medicines also pose a risk. Emerging biomarkers like NGAL and KIM-1 aid in early detection, while pharmacogenomics offers personalized prevention. Management often requires discontinuing the offending agent and, in some cases, using corticosteroids.

Acknowledgement

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Conflict of Interest

None.

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Address for Correspondence: Oliver, Hastings, Department of Medicine Renal Division, Kingsford Biomedical University, Northbridge, UK, E-mail: o.hastings@kingsfordbio.uk

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