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Incidence, Patterns, Risk Factors and Clinical Outcomes of Intravenous Acyclovir Induced Nephrotoxicity

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INTRODUCTION

Nephrotoxicity is toxicity of the kidney. This is the toxic effect of both toxic chemicals and drugs on kidney function. It comes in many forms, and some drugs can affect kidney function in more than one way. Nephrotoxins are substances that exhibit nephrotoxicity. Nephrotoxicity should not be confused with some drugs that are eliminated primarily by the kidneys and whose doses must be adjusted as kidney function declines. Drugs are normally metabolized in the liver, intestine, and kidneys. There are two routes of drug excretion. Renal excretion of drugs can basically be divided into three processes: Glomerular filtration, tubular reabsorption, and tubular secretion. Tubular secretion enables the transport of drugs from the blood to the urine *via* tubular cells. Apical contact with luminal secreted compounds exposes the proximal tubules. In addition, uptake by tubular epithelial cells or their apical efflux (movement of substances from the region of the plasma membrane located at the apex of the epithelial cells) can lead from the basolateral region of the tubular cells into the tubular lumen. These nephrotoxic compounds are cleared from the body *via* glomerular filtration and tubular secretory transport from the proximal tube to the Henle loop.

DESCRIPTION

The drug then travels to the distal tubule where it precipitates, crystallizes, or forms casts. This obstructs the renal tubules and causes tubular injury leading to acute tubular necrosis. Tubular obstruction by crystals or casts can also occur, eventually leading to interstitial nephritis. The kidney plays an important role in mediating the toxicity of many drugs, environmental pollutants, and natural products. Drugs known to be nephrotoxic include some cancer drugs, drugs of abuse, antibiotics, and x-ray contrast agents. Environmental contaminants known to target the kidneys include cadmium, mercury, arsenic, lead, trichlorethylene, bromate, brominated flame retardants, diglycolic acid, and ethylene glycol. Natural nephrotoxins include aristolochic acid and mycotoxins such as ochratoxin, fumonisin B1, and citrinin. There are some common features between nephrotoxin-induced renal failure mechanisms and exogenous causes. This commonality is primarily due to similarities in the molecular mechanisms that mediate renal cell death. This review summarizes the current state of affairs in the field of nephrotoxicity. It highlights the synthesis of our understanding of pathologically induced renal failure and nephrotoxicity. Such approaches are necessary to address important questions in this field, such as the diagnosis, prognosis, and treatment of both acute and chronic renal failure, and the progression of acute kidney injury to chronic kidney disease.

CONCLUSION

These substances include molds and fungi, cancer drugs such as cisplatin, antibiotics such as aminoglycosides, metals such as mercury, arsenic, and lead, and drugs of abuse such as cocaine. Evidence of nephrotoxicity is alterations in renal function assessed by Glomerular Filtration Rate (GFR), Blood Urea Nitrogen (BUN), Serum Creatinine (sCr), or urine output. However, nephrotoxic drugs can cause renal damage without altering established clinical markers of renal function. For example, studies have shown that in male He Sprague-Dawley rats exposed to gentamicin, proximal tubular necrosis can reach 75% before BUN or sCr increases. However, nephrotoxicity is difficult to predict during preclinical drug development and nephrotoxicity is usually recognized later. The nephrotoxic potential of newly approved drugs is also often underestimated. No regulatory approved or validated

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in vitro models for predicting nephrotoxicity are currently available. Here, we present current approaches to the development of such models. This includes discussions of three-dimensional and microfluidic models and recently developed stem cell-based approaches.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.