ISBN NO978-81-990189-9-0

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ACUTE KIDNEY INJURY SECONDARY TO NEPHRTOXIC DRUGS: A CLINICAL OVERVIEW

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Abstract

Acute Kidney Injury (AKI) is a sudden decline in renal function characterized by an increase in serum creatinine and/or reduction in urine output. Drug-induced AKI is a significant and preventable cause of morbidity and mortality, especially among hospitalized patients. A wide range of medications—including nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics such as aminoglycosides, radiographic contrast agents, diuretics, and certain chemotherapeutic drugs—are known to impair renal function through various mechanisms.The pathophysiology of drug-induced AKI primarily involves hemodynamic alterations, direct tubular toxicity, interstitial nephritis, and crystal-induced obstruction. Risk factors include advanced age, pre-existing renal impairment, dehydration, polypharmacy, and comorbid conditions such as diabetes and hypertension. Clinical presentation may range from asymptomatic biochemical changes to severe complications like oliguria, electrolyte imbalance, and uremia.Early identification through monitoring of renal parameters, along with prompt discontinuation or dose adjustment of the offending drug, is crucial in preventing progression. Management strategies focus on supportive care, optimization of fluid balance, and in severe cases, renal replacement therapy. Preventive measures such as appropriate drug selection, dose modification based on renal function, and therapeutic drug monitoring play a vital role.In , drug-induced AKI remains a critical clinical concern, emphasizing the importance of rational drug use and vigilant monitoring to minimize renal complications and improve patient outcomes.

Key words: Acute Kidney Injury, Oliguria, uremia, Aminoglycosides