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Kidney Damage in Children

Editorial

Acute Kidney Injury (AKI) is defined as a range of rapidly compromised renal functions that result in a fluid, electrolyte, and waste product balance that is disrupted. It is becoming more widely recognised as a leading cause of illness and mortality in children. This review covers the most important recent publications in the paediatric clinical and research areas, concentrating on the definition, epidemiology, impact and management, and early diagnostic tools of AKI. It has been challenging to construct consistent measurements of incidence and prevalence trends across time due to past discrepancies in AKI criteria in the paediatric population. Furthermore, most previous studies had been limited to tiny single-centre retrospective studies. The implementation of consistent definitions based on the KDIGO AKI staging criteria in numerous centres throughout the world will make assessing paediatric AKI epidemiology easier.

Olivia Taylor*

Editorial Office, Pediatrics Infectious Diseases: Open Access, London

*Corresponding author: Olivia Taylor

Taylor.O@gmail.com

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However, there will continue to be limitations in capturing AKI cases, such as the fact that most data is still focused on inpatients rather than outpatients. As a result, more research on specific populations at higher risk, such as children with chronic renal disease, nephrotic syndrome, and so on, is needed.

Although considerable progress has been made in understanding the cellular, metabolic, and molecular pathways of hypoxia/ischemia-induced AKI during the last several years, the pathophysiology of hypoxia/ischemia-induced AKI is still unknown. The history, physical examination, and laboratory tests, including urinalysis and radiographic examinations, can all be used to determine the likely cause(s) of AKI. Many treatments have been shown to have minimal effect on the course of AKI, including "renal dose dopamine" and diuretic medication. The underlying cause of AKI has a substantial impact on its prognosis. Children who have experienced AKI for any cause are at risk for renal disease later in life, even years after the initial insult. The fact that serum creatinine concentration is an insensitive indicator of AKI's etiology is due to the intricate nature of AKI's etiology.

Acute kidney injury (AKI) in children is becoming more common around the world, as are the accompanying morbidities and mortality. This review focuses on epidemiology and summarizes the most recent literature. The RIFLE (Risk, Injury, Failure, Loss of kidney function, End-stage renal disease) criteria, the later pediatric RIFLE (pRIFLE) score, and the Acute Kidney Injury Network (AKIN) criteria have all been utilized. Each of these classifications characterized and staged kidney injury differently, making comparison studies and uniform therapeutic recommendations more challenging.

Children who have experienced AKI for any cause are at risk for renal disease later in life, even years after the initial insult. Because of the intricate nature of AKI's pathogenesis, the fact that serum creatinine concentration is an insensitive predictor of kidney function, and the presence of co-morbid factors in patients, therapeutic efforts in AKI have largely failed. A greater understanding of the pathogenesis of AKI, early biomarkers of AKI, and improved classification of AKI are required for the development of viable therapeutic alternatives for the treatment of AKI. AKI can be caused by a variety of conditions, including Rapidly Progressive Glomerulonephritis (RPGN), which can trigger AKI but soon progress to Chronic Kidney Disease (CKD). Several renal diseases, such as Hemolytic-Uremic Syndrome (HUS), Henoch-Schonlein purpura, and obstructive uropathy with associated renal dysplasia, can present as AKI with normal or near-normal renal function, but the child's renal function may gradually deteriorate, leading to CKD months to years later. Children with AKI caused by hypoxic/ischemic insults, HUS, acute glomerulonephritis, and other causes are more likely to have oliguria or anuria (urine output less than 500 mL/24 h in older children or less than 1 mL/kg per hour in younger children and babies). Children with acute interstitial nephritis, nephrotoxic renal insults such as aminoglycoside nephrotoxicity, and contrast nephropathy are more likely to have AKI with normal urine production. In comparison to oliguric renal failure, no oliguric AKI has a lower risk of morbidity and mortality.

2021 Vol.6 No.11:36

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