Acute Kidney Injury in Children

Introduction

Acute Kidney Injury (AKI) (formerly known as acute renal failure) is defined by a reversible increase in blood creatinine and nitrogenous waste products, as well as the kidney’s inability to maintain proper fluid and electrolyte homeostasis. AKI in children appears to be on the rise, and the etiology of AKI has switched from primary renal illness to multifactorial reasons in recent decades, particularly in hospitalised children. Some children may be predisposed to AKI due to genetic causes. Pre-renal failure, intrinsic renal illness, including vascular insults, and obstructive uropathies are the three types of renal injury. The pathophysiology of hypoxia/ischemia-induced AKI is not fully known, although over the last several years, tremendous progress has been made in clarifying the cellular, biochemical, and molecular mechanisms. The likely cause(s) of AKI can be determined through the history, physical examination, and laboratory testing, including urinalysis and radiographic studies. Many treatments, including “renal-dose dopamine” and diuretic therapy, have been demonstrated to have little effect on the progression of AKI. The underlying etiology of AKI has a significant impact on the prognosis of AKI. Children who have had AKI for whatever reason are at risk for developing kidney disease later in life, even years after the initial insult. Because of the complicated nature of AKI’s etiology, the fact that serum creatinine concentration is an insensitive indicator of kidney function, and co-morbid variables in patients, therapeutic efforts in AKI have been mainly disappointing. For the development of viable therapeutic options for the treatment of AKI, a better understanding of the pathophysiology of AKI, early biomarkers of AKI, and better classification of AKI are required. Some AKI causes, such as Rapidly Progressive Glomerulonephritis (RPGN), can produce AKI but quickly proceed 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 improvement of renal function to normal or near-normal levels, but the child’s renal function may gradually deteriorate, leading to CKD months to years later.

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) are more common in children with AKI caused by hypoxic/ischemic insults, HUS, acute glomerulonephritis, and other causes. AKI with normal urine output is more common in children with acute interstitial nephritis, nephrotoxic renal insults such as aminoglycoside nephrotoxicity, and contrast nephropathy. Non-oliguric AKI has a lower morbidity and mortality rate than oliguric renal failure.