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Acute Kidney Injury in Children

Çocuklarda Akut Böbrek Hasarı

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ABSTRACT

Acute kidney injury (AKI) is a clinical condition characterized by sudden deterioration in kidney functions, increase in blood urea nitrogen (BUN) and serum creatinine levels, hyperkalemia, metabolic acidosis and hypertension. When defining AKI, current guidelines that consist of criterias determined by serum creatinine level and urine output are used. There are three main causes of AKI; prerenal, renal and postrenal. Prerenal AKI is most common etiology in children. Clinical symptoms of AKI vary depending on etiology. When evaluating a child with AKI, it should be noted that an increase in creatinine typically occurs 48 hours after renal injury and is the result of events 2-3 days earlier. The prognosis of AKI varies depending on the etiology.

ABSTRACT

Keywords: child, acute kidney injury, serum creatinine, urine output

ÖZ

Akut böbrek hasarı (ABH), böbrek fonksiyonlarında ani bozulma, kan üre nitrojeni (BUN) ve serum kreatinin düzeyinde artış, hiperkalemi, metabolik asidoz ve hipertansiyon ile karakterize klinik bir durumdur. ABH tanımlanırken, serum kreatinin düzeyi ve idrar miktarına göre belirlenen kriterlerden oluşan güncel kılavuzlar kullanılmaktadır. ABH'nın üç ana nedeni vardır; prerenal, renal ve postrenal. Prerenal ABH, çocuklarda en sık görülen etyolojidir. ABH'nın klinik semptomları etiyolojiye göre farklılık gösterir. ABH'li bir çocuğu değerlendirirken, kreatinin düzeyindeki artışın tipik olarak böbrek hasarından 48 saat sonra meydana geldiği ve 2-3 gün önceki olayların sonucu olduğu unutulmamalıdır. ABH'nın prognozu etyolojiye göre değişiklik göstermektedir.

Anahtar kelimeler: çocuk, akut böbrek hasarı, serum kreatinin, idrar miktarı

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Acute kidney injury (AKI) is a clinical condition characterized by sudden deterioration in kidney functions, increase in blood urea nitrogen (BUN) and serum creatinine, hyperkalemia, metabolic acidosis and hypertension [1]. In the past, acute renal failure (ARF) was used and it has been called AKI since 2004 [2]. The development of AKI has been reported in 2-3% of hospitalized children and 8% of those in the neonatal intensive care unit [3].

When defining AKI, current guidelines such as RIFLE, pediatric RIFLE, Acute Kidney Injury Network (AKIN) and Kidney Disease Improving Global Outcomes (KDIGO) are used [4-7, Table 1]. Early diagnosis and determining the clinical severity of AKI are important issues for prognosis. RIFLE criteria is a definition consisting of the initials of English words such as risk (R), injury (I), failure (F), loss (L) and end stage renal disease (E) and began to be used in 2004. Subsequently, the pediatric RIFLE criteria, which recommended the use of the same abbreviation in children, came into use. Pediatric RIFLE includes criteria determined based on serum creatinine and urine output. AKIN criterias and KDIGO criterias were published in 2007 and 2012 respectively. Both criterias evaluated AKI at three stages.

The etiology of AKI is evaluated under 3 main causes: prerenal, renal and postrenal causes. Prerenal causes are most commonly seen in

children. Dehydration and gastroenteritis are the most common causes of AKI [1]. Conditions such as extracellular fluid loss, systemic vasodilation, decreased renal blood flow, and increased resistance to flow are the causes of prerenal AKI. Conditions that cause tubular damage and glomerulonephritis are causes of renal AKI. Congenital or acquired conditions that impair urine flow are also considered as postrenal AKI [8]. Clinical findings differ according to etiology. For example; oliguria along with clinical signs of dehydration may be observed in a child with AKI secondary to gastroenteritis, one of the causes of prerenal AKI. Hypervolemia findings due to oliguria may be observed in a child with AKI secondary to glomerulonephritis due to renal causes, and anuria may be observed in a patient with AKI secondary to renal stone due to postrenal causes [1]. Although an infant presenting with vomiting and diarrhea for three days is more likely to have prerenal AKI with signs of dehydration, the diagnosis of hemolytic uremic syndrome (HUS) that is renal etiology of AKI should also be considered. If there is history of pharyngitis, periorbital edema, hypertension and macroscopic hematuria, renal AKI secondary to acute post-infectious glomerulonephritis should be considered as differential diagnoses. Acute tubular necrosis should be considered in patients with resistant hypotension or a history of nephrotoxic drug use. A male newborn with bilateral hydronephrosis on prenatal ultrasonography and a palpable bladder on physical examination should be evaluated for the posterior urethral valve. A detailed physical examination should be performed and the patient's volume status should be carefully evaluated. If AKI is accompanied by rash and arthritis, SLE (systemic lupus erythematosus) or IgA vasculitis should be considered. If there are palpable kidneys on physical examination, renal vein thrombosis, tumor, cystic diseases or urinary system obstruction should be considered in the differential diagnosis [3].

When evaluating a children with AKI, it should be noted that an increase in creatinine typically occurs 48 hours after renal injury and is the result of events 2-3 days before. For this reason, hypotension, hypoxia, sepsis, surgical intervention, contrast material and drug exposure should be questioned 48-72 hours before AKI is identified [8]. Serum creatinine is considered a late biomarker of AKI as that increases after AKI develops. Biomarkers such as Neutrophil gelatinase associated lipocalin (NGAL), Kidney injury molecule-1, and IL-18, which help to define AKI earlier, have been identified, but have not yet come into routine use [9]. Development of sensitive biomarkers to define AKI earlier is important to initiate treatment at the appropriate time [10]. In the diagnostic evaluation of AKI; urine examination, basic serum electrolyte levels, kidney function tests and urinary system imaging, especially ultrasonography, are the most important diagnostic tools [8].

Table1: Criterias Used in Defining Acute Kidney Injury In Children (ESRD: end stage renal disease, e GFR: estimated glomerular filtration rate, h: hour)

Criteria	Stage	Serum creatinine	Urine output
pRIFLE	Risk	eGFR decrease by 25%	< 0.5ml/kg/h for 8 h
	Injury	eGFR decrease by 50%	< 0.5ml/kg/h for 16 h
	Failure	eGFR decrease by 75%	< 0.3ml/kg/h for 24 h or anuria for 12 h
	Loss	Persistent failure > 4 weeks	
	ESRD	Persistent failure > 3 months	
AKIN	1	Increase in serum creatinine x 1,5-2 or increase in serum creatinine > 0.3mg/dl	<0.5ml/kg/h for 6h
	2	Increase in serum creatinine x 2-3	<0.5ml/kg/h for 12 h
	3	Increase in serum creatinine x 3 or serum creatinine >4 mg/dl (acute increase >0,5 mg/ dl) or Renal Replacement Treatment	<0.3ml/kg/h for 24h or anuria for 12 h
KDIGO	1	Increase in serum creatinine x 1,5-1,9 or increase in serum creatinine >0,3mg/dl within 48 h	<0.5 ml/kg/h for 6 h
	2	Increase in serum creatinine x 2-2,9	<0.5ml/kg/h for 12 h
	3	Increase in serum creatinine x 3 or serum creatinine ≥ 4mg/dl or renal replacement therapy	<0.3ml/kg/h for 24 h or anuria for 12h

The prognosis of AKI depends on the etiology of AKI. Children who develop AKI as a component of multisystem failure have a higher mortality rate than AKI that develops secondary to primary renal diseases such as HUS, rapidly progressive glomerulonephritis (RPGN) and acute interstitial nephritis (AIN). Recovery from AKI secondary to

primary renal disease varies depending on the underlying etiology. Nephrotoxic AKI and hypoxic/ischemic AKI usually result in recovery of normal renal function [10].

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