THE KOREAN SOCIETY OF NEPHROLOGY
APCN & KSN 2024
June 13(Thu) - 16(Sun), 2024 Coex, Seoul, Korea
APSN
Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

Abstract Submission No.: A-0243

Acute tubular necrosis with unusually prolonged and marked polyuria: a pediatric case report

JI HYUN KIM[1], Hee Gyung Kang[2], Yo Han Ahn[2]
[1]Department of Pediatrics-Nephrology, Seoul National University Bundang Hospital, Korea, Republic of
[2]Department of Pediatrics-Nephrology, Seoul National University Hospital, Korea, Republic of
[3]Department of Pediatrics-Nephrology, Seoul National University College of Medicine, Korea, Republic of

Case Study : Acute tubular necrosis (ATN) is the leading cause of acute kidney injury (AKI), primarily triggered by ischemia, sepsis, or nephrotoxins. Typically, both the oliguric (urine output (UOP) < 500 ml/day) and diuretic phases of ATN last about 12 days, with UOP seldom exceeding 7 to 8 L/day. We report the first pediatric ATN case, exhibiting an unusually prolonged and marked polyuria. A previously healthy 15-year-old girl presented with abdominal pain, chest discomfort, and decreased UOP. She was diagnosed with acute fulminant myocarditis, leading to multiorgan failure, affecting her kidneys and liver. Despite improvements in myocarditis and liver failure, her oliguric AKI persisted and required kidney replacement therapy for 14 days. Ultrasound revealed diffuse, slightly increased echogenicity in both kidneys. Laboratory findings necessitated the exclusion of thrombotic microangiopathy or glomerulonephritis, leading to a kidney biopsy, which confirmed ATN only. From the 15th hospital day, oliguric AKI steadily improved and stabilized over 10 days, with UOP of 1.5 to 2.5L/day. During that period, a fever lasted for 6 days, attributed to a parainfluenza infection. Remarkably, she then experienced prolonged diuresis for 30 days, with UOP peaking at over 23 L/day and averaging 10 L/day, persisting for 14 days even after kidney function normalization. Urinalysis showed mixed polyuria, and no other causes for this condition were found. Continuous fluid and oral hydration were maintained to prevent further prerenal injury. On the 24th day of diuresis, desmopressin was initiated, leading to an increase in urine concentration and a dramatic improvement in polyuria to less than 1L/day. This case highlights an unusual presentation of extended polyuria (31 days, peaking at 23 L/day) following ATN recovery, emphasizing the need for vigilant UOP monitoring and fluid replacement to prevent secondary prerenal insult. While desmopressin markedly improved polyuria, its role and effectiveness in this case warrant further investigation.