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Primary hyperoxaluria as a cause of renal failure in an infant

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Clinical approach to acute kidney injury in infants may be challenging. When pre-renal and post-renal azotemia is ruled out and no obvious cause is found, inherited disease such as primary hyperoxaluria should be considered and renal biopsy may be diagnostic.

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Renal failure in infants and children has many different causes from pre-renal azotemia due to gastroenteritis (which is common in daily practice) to glomerular, tubular and vascular injuries (1,2). When there is no positive history, clinical sign and laboratory finding in favor of dehydration, a clinician should consider direct renal damage or obstructive uropathy. Obstruction may be easily ruled out by urinary tract imaging. The absence of significant hematuria, proteinuria and vascular damage leads to probable tubulointerstitial involvement in the setting of infection, nephrotoxins, ischemia, pigment nephropathy or crystal nephropathy.

Primary hyperoxaluria type 1 (PH1) is one of the causes of crystal induced renal failure. This autosomal recessive condition with the prevalence of 3 in 10⁶ population is responsible for more than 1% of end-stage renal disease (ESRD) in children (3) and should be considered in patients with consanguineous parents and significant history of renal stone in family (4). Primary hyperoxaluria type 1 results from mutations of gene of hepatic enzyme; alanine glyoxylate aminotransferase (AGXT). Patients may present in infancy with renal failure and ESRD or in children and adults with renal stone. Diagnosis is highly suggested by an elevated level of oxalate excretion in urine and is made definite by renal

biopsy, genetic assessment and enzyme measurement (5). Oxalosis occurs when glomerular filtration rate (GFR) declines to 30 mL/min/1.73m² and calcium oxalate deposits in virtually all vital organs causing significant morbidity and mortality (6).

The patient was 5-month-old-boy presented with irritability, poor feeding and decreased urine output. His parents were first cousins. Sonography only showed a significant increase in echogenicity of both kidneys. Laboratory results were: BUN=120 mg/dL, creatinine=15 mg/dL, K=6 mEq/L, Na=126 mEq/L, pH=6.91, Hco₃=3.6 mEq/L, Hb=6.9 g/dL. Urinalysis consisted of +3 protein, +1 blood, +1 glucose, WBC=3-4/hpf, many RBC, granular and RBC casts/hpf and finally a random urine Na was 80 mEq/lit. Anuria of the patient persisted and peritoneal dialysis was started for him. According to unexplained acute kidney injury, a renal biopsy was performed for the patient. In renal biopsy, immunofluorescence microscopy showed no significant deposits while light microscopy showed diffuse and significant deposits of birefringent crystals indicative of hyperoxaluria (Figure 1A-B).

Authors' contribution

HEM wrote the manuscript and AD reviewed renal biopsy pathologic samples.

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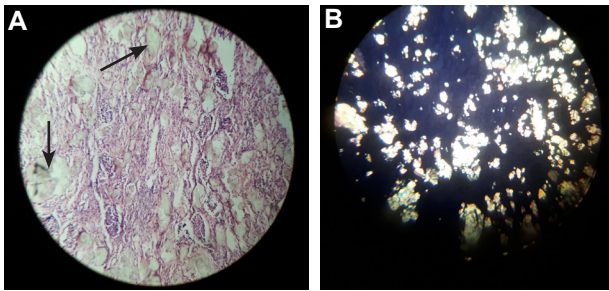


Figure 1. (A) Crystals in H&E stain ($\times 200$); (B) Birefringent crystals indicative of hyperoxaluria ($\times 200$).

Conflicts of interest

The authors declare no conflict of interest.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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