

## End Stage Renal Disease Presenting with Acute Renal Failure

A 3-year-old boy presented with sudden onset of edema, oliguria and vomiting of 15 days duration with no significant contributory preceding history. His height was above the 50th centile for age and blood pressure was 150/110 mmHg. Apart from generalized edema, the physical examination was unremarkable. Urinalysis showed a spot urine protein to creatinine ratio of 2, without significant erythrocyturia. The hemoglobin level was 8.3 g/dL, platelet counts were normal and peripheral blood smear was normal. The blood level of urea was 227 mg/dL, creatinine 7.1 mg/dL, sodium 125 mEq/L, potassium 5.3 mEq/L, bicarbonate 6 mEq/L, cholesterol 385 mg/dL, total protein 5.2 g/dL (albumin 2.6 g/dL), calcium 8.0 mg/dL, phosphate 4.8 mg/dL and SAP 282 IU/dL. Blood levels of CPK, LDH and complement C3 were normal; ASO titre, CRP, ANA and ANCA were negative. Viral markers for hepatitis B and C and HIV antibodies were negative. Ultrasonogram abdomen showed normalized kidneys with mild increase in renal echogenicity; doppler study was normal.

A diagnosis of nephrotic syndrome (NS) with acute renal failure was made and the patient managed with intermittent peritoneal dialysis and IV methylprednisolone (30 mg/kg/day) for 5 days followed by oral prednisolone (60 mg/m<sup>2</sup>/day), nifedipine and hydralazine. After one week, the urine output improved and blood level of creatinine decreased to 1.6 mg/dL. Peritoneal dialysis was discontinued and a renal biopsy was performed. Two days later he developed severe hyponatremia, hyperkalemia with creatinine level of 6.3 mg/dL. The renal biopsy showed 20 glomeruli with extensive glomerulosclerosis with moderate tubular atrophy and interstitial inflammation. Immunofluorescence findings showed mesangial granular deposits of IgM and C3. The child was managed with continuous ambulatory peritoneal dialysis and supportive measures.

The etiology of acute renal failure in patients with nephrotic syndrome is multifactorial, and include hypovolemia, renal venous thrombosis, rapid progression of the original glomerular disease, crescentic glomerulonephritis, intratubular obstruction by proteinaceous casts and allergic interstitial nephritis (due to antibiotics, diuretics and NSAIDs)(1). This child presented as nephrotic syndrome for the first time and on evaluation was found to have a severe renal failure needing peritoneal dialysis. No etiological or predisposing cause was available. The renal biopsy showed extensive glomerular sclerosis and it was surprising that he was asymptomatic and presented as nephrotic syndrome with histological features of end stage renal disease. The “collapsing” variant of focal segmental glomerulo sclerosis especially, has been associated with rapidly progressive renal failure(2). Infants with congenital NS can have diffuse mesangial sclerosis, and present with irreversible renal failure. Nakahata, *et al.*(3) have reported a 4-year-old previously healthy Japanese girl who developed irreversible renal failure with the first episode of NS. She was hypertensive at presentation with no other significant clinical features. Extensive investigations did not reveal any cause for the renal failure and the renal biopsy showed a similar histology to our case, with global sclerosis of most glomeruli. She was continued on maintenance dialysis without any recovery over 15 months.

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### REFERENCES

1. Agarwal N, Phadke KD, Garg I, Alexander P. Acute renal failure in children with idiopathic nephrotic syndrome. *Pediatr Nephrol* 2003; 18: 1289-1292.
2. Weiss MA, Daquiaoag E, Margolin EG, Pollak VE. Nephrotic syndrome, progressive irreversible renal failure, and glomerular “collapse”: a new clinicopathologic entity? *Am J Kidney Dis* 1986; 7: 20-28.
3. Nakahata T, Tanaka H, Tsuruga K, Shimada J, Tsugawa K, Suzuki K, *et al.* End-stage kidney at the onset of nephrotic syndrome in a 4-year-old girl. *Tohoku J Exp Med* 2003; 200: 151-154.