SPECIAL FEATURE

SECTION EDITOR: BEVERLY P. WOOD, MD

Radiological Case of the Month

LT Derek S. Wheeler, MC, USNR

A FULL-TERM, male infant presented at 4 days of life with decreased urine output, fever, and poor feeding. Delivery was complicated by a shoulder dystocia, and the infant had required a brief period of continuous positive airway pressure ventilation for apnea and central cyanosis. The infant was discharged from the hospital at 48 hours of life, following an uneventful postnatal course.

On initial presentation, blood, urine, and cerebrospinal fluid cultures were obtained. A urinalysis revealed microscopic hematuria and proteinuria. Further laboratory evaluation was notable for the following levels: serum sodium,155 mmol/L; serum urea nitrogen, 21.8 mmol/L (7.8 mg/dL); serum creatinine, 424.3 µmol/L (4.8 mg/dL); fractional excretion of sodium, 4.6%; and serum uric acid, 1118.2 mmol/L. Results of a complete blood cell count were normal.

A renal sonogram was obtained (**Figure**). The infant's kidney function normalized (on the third day of hospitalization) with fluid restriction and diuretic therapy. Laboratory studies revealed a serum creatinine level of 44.2 µmol/L (0.5 mg/dL) and a serum uric acid level of 285.5 mmol/L. Results of a renal sonogram at 2 months of age were normal.

From the Department of Pediatrics, US Naval Hospital, Guam.

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Denouement and Discussion

Transient Acute Renal Failure of the Newborn

Sonogram of one kidney shows hyperechoic renal medullary pyramids with acoustic shadowing. There is no evidence of hydroureteronephrosis.

Renal ultrasonography plays a significant role in the diagnostic evaluation of pediatric renal disease. The normal sonographic appearance of the neonatal kidney differs greatly from that of older children. The renal medullary pyramids are hypoechoic when compared with the renal cortex, where echogenicity is comparable to the liver and spleen.[1] Increased echogenicity of the renal pyramids during the neonatal period has been associated with serious renal disease, including renal vein thrombosis, nephrocalcinosis, congenital nephrotic syndrome, and cystic kidney disease.[2,3] There are several reports of a distinct variety of neonatal renal disease characterized by transient oliguric renal failure and associated with hyperechoic renal medullary pyramids. These cases have been associated with an excellent prognosis in which sonographic findings and renal function return to normal.[4,5]

The cause of transient acute renal failure of the newborn is poorly understood. Several authors have noted significant proteinuria during the recovery phase. The protein has been identified as the Tamm-Horsfall mucoprotein, formation of which is thought to be related to tubular maturation. It is postulated that some infants are unable to excrete the increased load of mucoprotein that coincides with the transition from intrauterine to extrauterine life, and the protein is deposited in the renal tubules with subsequent obstruction of urinary flow and renal dysfunction.[6,7]

Other investigators postulate that transient renal failure in association with hyperechoic renal pyramids is caused by transient hyperuricemia secondary to perinatal asphyxia.[8,9] Newborns generally have a transient elevation of serum uric acid concentration. The association between increased uric acid production and perinatal asphyxia is well-recognized.[10] It is possible that the increased uric acid production associated with perinatal asphyxia leads to deposition of uric acid in the renal tubules, producing tubular obstruction and subsequent renal dysfunction. As the renal uric acid load is excreted, normal kidney function is restored. Therefore, uric acid would account for the increased echogenicity of the renal medullary pyramids and the transient reduced kidney function. Further support for this hypothesis is provided by the fact that serial renal ultrasonographic examinations of patients with Lesch-Nyhan syndrome, a syndrome characterized by hyperuricemia, have revealed hyperechoic renal medullary pyramids.[11]

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