A 5-DAY-OLD boy was seen for poor feeding, nonbilious vomiting, lethargy, and decreased urine output. The history was notable for an unremarkable pregnancy with normal findings from 16-week fetal ultrasound. Findings from initial physical examination revealed a large, nontender right flank mass. Results of subsequent laboratory analysis revealed acute renal failure, metabolic acidosis, hyperkalemia, azotemia, and hematuria. Anemia and thrombocytopenia were also present. Renal ultrasonography was performed, and results of a subsequent computed tomographic scan of the abdomen are shown in Figure 1 and Figure 2.

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Denouement and Discussion

Inferior Vena Cava and Renal Vein Thrombosis in a Neonate

All abdominal masses detected in the neonatal period require further investigation with ultrasonography. Most of these masses originate in the genitourinary tract, and ultrasonography should be performed to localize and differentiate between cystic and solid structures. The differential diagnosis of cystic abdominal masses includes hydronephrosis; multicystic-dysplastic kidney disease; intestinal duplication; hydrometrocolpos; and choledochal, ovarian, renal, and pancreatic cysts. The differential diagnosis of solid abdominal masses includes neuroblastoma, teratoma, and Wilms tumor. A solid flank mass of a neonate in the presence of hematuria, hypertension, renal failure, and thrombocytopenia suggests a diagnosis of renal vein thrombosis.

Two types of renal vein thrombosis are recognized. In the first, the thrombus begins intrarenally at the level of the arcuate and interlobar veins, successively extending into the larger renal veins and to the inferior vena cava. It is thought that this type is more frequent during infancy. However, Demirci et al presented a case of bilateral renal vein thrombosis in which the inferior vena cava was completely filled with thrombus, suggesting that the thrombus arose initially in the inferior vena cava with subsequent retrograde extension into the renal veins.

Clinically, renal vein thrombosis is characterized by the presence of a palpable flank mass associated with acute renal failure. Hematuria, proteinuria, and oliguria are usually present. Predisposing factors are dehydration, asphyxia, polycythemia, sepsis, shock, coagulopathies, and maternal diabetes. Ultrasound examination will show perivascular streaking with increased echogenicity of the renal parenchyma. Computed tomography will demonstrate persistent parenchymal opacification and enlargement of the affected kidney, dilation of the renal vein, and renal vein and/or inferior vena cava thrombus.

Surgical management with removal of the affected kidney has historically been the treatment of choice, but it has been recognized more recently that with supportive therapy alone, the short-term prognosis of renal vein thrombosis occurring during infancy is good. However, the involved kidney progressively becomes atrophic resulting in a small, scarred kidney. Treatment with systemic anticoagulation agents may result in a better long-term prognosis. In our patient, treatment with low-molecular-weight heparin resulted in a return of normal renal function by age 4 months.

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REFERENCES