Severe Hyponatremia Complicating Urinary Tract Malformation with Pyelonephritis

A 7-month-old developmentally normal girl presented with high fever and crying during micturition for 3 days. There was no history of convulsions, diarrhea, vomiting, lethargy or refusal to feed. She weighed 7.2 kg, blood pressure was normal, and she was hemodynamically stable. Anterior fontanelle was level, and there was no hepatosplenomegaly or dehydration. Urinalysis showed 100 neutrophils/HPF. Intravenous ceftriaxone was administered in view of a presumptive diagnosis of urinary tract infection; and continued for 14 days. Investigations revealed hemoglobin 11.2 g/dL, total leukocyte count 15×10^9/L (70% neutrophils), and platelet count 2.1×10^9/L. Blood urea was 12 mg/dL, and serum creatinine was 0.4 mg/dL. There was severe hyponatremia (serum sodium 110 mEq/L) while serum potassium was 5 mEq/L. Mild metabolic acidosis was observed on venous blood gas sample (pH 7.36, bicarbonate 16.2 mEq/L). Urine culture revealed significant growth of *Escherichia coli*. Since the genitalia were female, without any virilization, a diagnosis of type 1 pseudohypoaldosteronism (renal variety) secondary to the renal malformation and urinary tract infection, was considered. Serum aldosterone levels were 1696 pg/mL (reference range 20-1100 pg/mL), confirming pseudohypoaldosteronism. Sodium levels were restored to normal with 3% hypertonic saline supplementation.

Subsequently, a renal ultrasound revealed left sided enlarged duplex kidney (renal size 7.5 cm) with hydronephrosis of the lower moiety (anteroposterior diameter of renal pelvis 12 mm). The right kidney was of normal size (5.9 cm). Micturating cysturethrogram was normal. EC-Diuretic Renogram showed pelviureteral junction obstruction in the left kidney with differential function 40%. DMSA radionuclide scan (done after 2 months) did not show renal scars. Sodium levels continue to be normal. The infant was started on cephalexin chemoprophylaxis, and is being planned for operative intervention.

Severe hyponatremia in infants may be part of salt losing crisis such as congenital adrenal hyperplasia, adrenal hypoplasia, or aldosterone resistance (pseudohypo-aldosteronism). Pseudohypoaldosteronism is characterized by renal tubular unresponsiveness to aldosterone and may complicate pediatric renal disorders such as obstructive uropathy, duplex kidneys, pyelonephritis, vesicoureteral reflux, tubulointerstitial nephritis etc [1-3]. The entity may manifest with hyponatremia, hyperkalemia and mild metabolic acidosis. Pyelonephritis and urinary tract malformations increase the intrarenal synthesis of cytokines such as Transforming Growth Factor β1 which can induce inhibition of action of aldosterone [4]. Impairment of the aldosterone receptor by circulating factors as well as bacterial toxins has also been proposed [5]. Hyperkalemia may not be noticed beyond the neonatal period in such cases [1]. Our patient had urinary malformation (duplex kidney) with pyelonephritis leading to transient Type 1 pseudohypoaldosteronism.

Many cases of pseudohypoaldosteronism secondary to renal causes can be erroneously diagnosed and treated as congenital adrenal hyperplasia [1,2]. We emphasize that in any infant who presents with severe hyponatremia outside the neonatal period, a renal cause should always be considered, especially when there are signs/symptoms of pyelonephritis and/or urinary tract malformations. Prompt realization of this entity is essential to ensure favorable outcomes and avoid unnecessary interventions.

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***Sriram Krishamurthy and VS Venkateswaran**
Department of Pediatrics,
Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry, India.
**drsriramk@yahoo.com*

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