LETTER TO THE EDITOR

More is not always better: a case postrenal transplant large volume diuresis, hyponatremia, and postoperative seizure

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Polyuria is common in the period immediately following the renal transplantation [1]. Such postoperative polyuria, which is associated with renal tubular dysfunction, can be exacerbated by aggressive i.v. fluid resuscitation [2]. With prolonged high volume polyuria, proper medical management may be difficult as fluid and electrolyte balance may be disturbed. Furthermore, some of the electrolyte disturbances associated with polyuria may lead to complications such as seizure, arrhythmia, and death [3]. We present a case of a renal transplant recipient who experienced polyuria and subsequently developed severe hyponatremia and generalized tonic-clonic seizures. Permission was obtained from our institutional human subjects committee and informed consent was obtained from the patient.

A 39-year-old woman, with a history of end-stage renal disease secondary to membranoproliferative glomerulonephritis type I (MPGN) underwent elective living unrelated renal transplant from her husband. Of note, she was 155 cm tall and weighed 44.5 kg while her husband (donor) was 195.6 cm tall and weighed 111.4 kg. The surgery was completed at 7 PM during which she received 3700 cc of Ringer's Lactate [(Na) = 130 MEq/l], 12.5 g of mannitol, and 80 mg of furosemide. At 9 PM, her serum [Na] was 141 MEq/l. Overnight, the patient had substantial diuresis with urine output of 1–2 l/h (25–50 cc/kg/h). Per our postoperative kidney transplant protocol, the patient's urine was replaced in equal volume by half normal saline [(Na) = 77 MEq/l]. During this period, the patient had stable blood pressure and heart rate. She received noninduction immunosuppression including a steroids, cyclosporine, and mycophenylate. Nine hours later, our service was informed that she was having a tonic-clonic seizure. At that time, a stat laboratory panel was drawn, which revealed a sodium of 113 MEq/l and a creatinine of 0.8 mg/dl (5.3 at 9 PM the prior evening). The seizure resolved spontaneously, but she remained unresponsive. The patient was intubated for her poor mental status and proceeded to have several more generalized seizures despite loading with anticonvulsant medications. The seizure was determined to be metabolic and gradual return of sodium levels was begun. We calculated her sodium deficit [total body water (22.3 l) × (140 MEq/l − 113 MEq/l)] to be 602 MEq of Sodium. Our goal was to correct her Sodium to 120 MEq/l in the first 12 h and not to exceed correction by >10 MEq/l per 24 h. Too rapid a correction risks central pontine myelinolysis. We initially administered 3.0% NS (513 mEq of Na^+/l) at a rate of 25 ml/h for 12 h. The seizures stopped shortly after initialing hypertonic saline therapy. After 12 h, her sodium was 122 and the fluids were changed to 0.9% NaCl. With the above therapy, the patient's neurologic status returned to normal, and she was discharged 6 days postoperatively. Six months postoperatively, she has excellent graft function and is back to work without apparent sequalae. No follow-up CT scan was carried out.

Following renal transplant, a brisk diuresis is generally welcomed, but in this case it was excessive. This patient received replacement fluids that were significantly hypotonic relative to her urine output. Currently, no consensus exists postrenal transplant fluid management [4,5], but a decrease in urine replacement fluid resuscitation with closer monitoring of electrolytes would have been appropriate in this case. Intra-operative administration of mannitol is routine at our center. There is no clear data that this improves graft function and it may have contributed to this case of hyponatremia. Additional study on mannitol in renal transplantation is needed. We suggest careful monitoring of patients with a diuresis >25 cc/kg/h and adjustment of their urine replacements to 1/2 cc per cc of urine.

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