A Case Of Severe Hyponatremia After Donor Nephrectomy

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BACKGROUND

- Living kidney donation reduces the growing gap between the demand for and supply of renal allografts, and offer recipients the best opportunity for dialysis-free survival(1).
- Hyponatremia is the most common electrolyte disorder in clinical practice, associated with increased morbidity, mortality, and length of hospital stay(2).
- Here, we report a woman in her 60s who was admitted to our hospital with severe hyponatremia following donor nephrectomy.

CASE REPORT

A 60-year-old woman with no significant past medical history, who underwent left donor nephrectomy. One week after nephrectomy, she presented to the emergency department with 5 days history of nausea, vomiting, diarrhea and abdominal pain. The patient denied use of alcohol or illicit drugs. **Physical examination** revealed a blood pressure of 115/70 mmHg with mild postural drop and a regular pulse of 78 beats/min. She looked mildly dehydrated and had no fever. Results of further physical examination were unremarkable and revealed no goiter, pigmentation, or vitiligo. Her **laboratory** results showed severe hyponatremia and a high urine sodium and osmolality together with metabolic acidosis (? Renal tubular acidosis, RTA). Additional diagnostic tests included chest x-ray, abdominal ultrasound, and brain computed tomography, none of which revealed relevant abnormalities (Table 1).

Table 1. Laboratory results

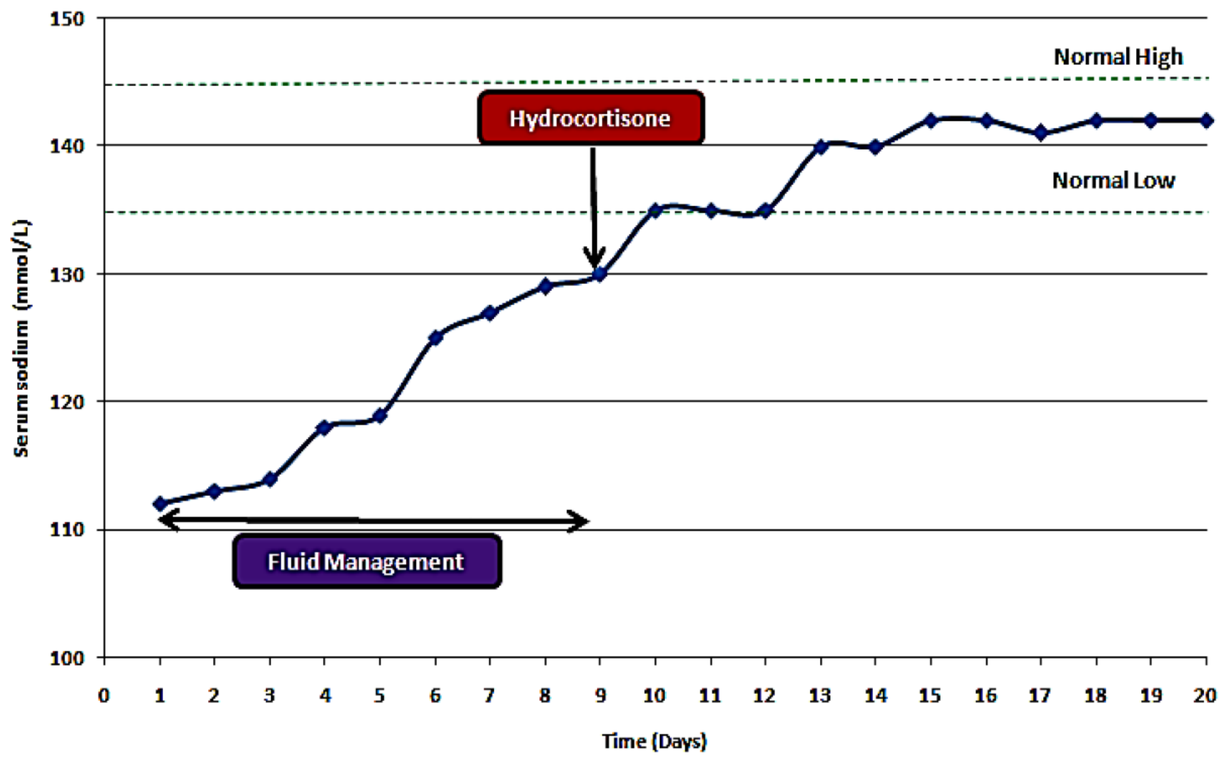
	Parameter	Patient	Reference
Serum	Sodium, mmol/L	113	135 - 147
	Potassium, mmol/L	4.2	3.5 - 5
	Osmolality, mOsm/Kg	234	275 - 300
	Glucose, mmol/L	3.1	4.0 – 7.6
	Calcium, mmol/L	2.23	2.10 – 2.55
	$HCO3^-$, mmol/L	17	22 - 29
	Creatinine, µmol/L	74	46 - 96
	Urea, mmol/L	3.8	2.5 – 7.5
	Uric acid, µmol/L	119	140 - 390
	Hemoglobin, g/L	97	110 - 160
	Platelets, 10^9 /L	592	155 - 435
	Albumin, g/L	42	32 - 48
Urine	Sodium, mmol/L	91	---[a]
	Osmolality, mOsm/Kg	655	50 – 1200

[a]There are no reference interval values for urine sodium, because measured values depend on the diet and the clinial circumstances. During hyponatremia, high or low urine sodium concentrations (typically with 20 mmol/L as cutoff) can be used for differential diagnosis.

Because of apparent hypovolemia, intravenous NaCl 0.9 % then 2 % were initiated with no appreciable improvement in serum sodium. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) was suspected(3). However, fluid therapy (NaCl 2% with fluid restriction) showed incomplete response. Because of the lack of response to therapy for SIADH, the diagnosis was reconsidered and hypothyroidism and/or adrenal insufficiency were suspected. Serum TSH was 6.3 mU/L (reference interval 0.27–4.2 mU/L) with free thyroxine of 7.6 pmol/L (reference interval 12–22 pmol/L) denoting primary hypothyroidism. L-thyroxine (75 µg daily) was initiated but again with no much improvement. Random cortisol was 124 nmol/L (reference interval 150–700 nmol/L), and a stimulation test with ACTH showed a baseline cortisol of 140 nmol/L (reference interval 171–536 nmol/L), which increased to 463 nmol/L (normal response 500 – 550 nmol/L) denoting suboptimal response.

CASE REPORT (cont'd)

These results confirmed the presence of primary adrenal insufficiency. Oral hydrocortisone was started (10-5 mg daily), which corrected serum sodium to within the reference interval (Fig. 1). Hydrocortisone replacement quickly resolved her symptoms and greatly ameliorated the hyponatremia and RTA.



DISCUSSION

- This case has 2 salient features. First, it illustrates the diagnostic challenges of severe hyponatremia with high urine sodium and osmolality. Second, it illustrates quite strikingly how atypical the presentation of adrenal insufficiency can be.
- The differential diagnosis of hyponatremia with a high urine sodium and osmolality consists of diuretic use, primary or secondary adrenal insufficiency, hypothyroidism, cerebral salt wasting, salt-wasting nephropathy, and SIADH (1).
- Many physicians tend to diagnose SIADH before excluding the other causes. However, according to the criteria, SIADH is a diagnosis of exclusion(3).
- Adrenal insufficiency must always be ruled out in cases of severe hyponatremia, because the classic clinical and biochemical features may not be present(2). The mechanism of adrenal insufficiency in this case could be due to interruption of the venous drainage of the left adrenal gland during left-sided donor nephrectomy.

CONCLUSION

- In this case with complex hyponatremia following donor nephrectomy, a mechanism of severe hyponatremia caused by primary adrenal insufficiency, could be due to interruption of the venous drainage of the left adrenal gland during left-sided donor nephrectomy; for which the administration of hydrocortisone exerted additional therapeutic efficacy.
- Observational prospective studies of adrenal function following donor nephrectomy are needed to verify this proposed mechanism.

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