Severe Symptomatic Diuretic Induced Hyponatremia

Dear Editor,

A 66 year old man, a known case of primary hypertension and primary hypothyroidism was admitted with complaints of fatigue, weakness, lethargy and unsteadiness of gait of about 24 hours duration. He complained of dull headache for the last one week. There were no visual symptoms, loss of consciousness, seizures, sensory or motor symptoms, fever or vomiting. His comprehension was preserved. His treatment included Telmesartan 40 mg; Amlodipine 5 mg; Metoprolol XL 50 mg; Hydrochlorothiazide 25 mg and Thyroxine 50 mcg daily. Two days prior to admission his thiazide was increased from 12.5 mg to 25 mg and he had curtailed his salt intake.

Clinical examination revealed a conscious but confused patient. He was disoriented to time, drowsy but easily arousable. His pulse was 72/min and had a blood pressure of 114/78 mmHg. He had no focal neurological deficits or any meningeal signs. His systemic examination was essentially normal.

Investigations revealed haemoglobin 12.9 gm/dl, total leucocyte count 7800 cells/cmm, blood urea 13.0 mg/dl and serum creatinine 0.8 mg/dl. The liver function tests showed serum bilirubin of 1.0 mg/dl, total proteins 7.0 g/dl, albumin 4.0 g/dl, SGOT 42 IU/L, SGPT 36 IU/L, alkaline phosphatase 79 IU/L. The serum sodium was markedly low 98 mEq/L, serum potassium was 3.1 mEq/L. A urinary spot sodium revealed 148 mEq/L. TSH was 6.0 μ IU/ml (normal 0.3-6.5) and basal cortisol was 19.6 μ g/dl. His serial serum sodium values are depicted in the graph (Fig 1).

He was diagnosed as a case of chronic severe hyponatremia possibly due to use of diuretic and restriction of dietary salt intake (high urinary spot sodium in presence of hyponatremia). He tolerated severe hyponatremia well without any severe neurological manifestation probably indicating underlying chronic hyponatremia. Hence a gradual correction of sodium with hypertonic saline was planned (10 mEq/day). He was started with 3% normal saline at the rate of 30 ml/min (0.5ml/kg) [1]. On second day at 1800 hours his infusion was stopped in view of overcorrection. His serum sodium level again fell to 105 mEq/L in the morning next day which prompted restarting the 3% normal saline infusion at the rate of 20 ml/min. His serum sodium was gradually corrected to 126 mEq/L within 48 hours and his clinical condition improved. However his urinary sodium loss also increased. He was shifted to oral sodium replacement, 300 mEq with oral fluid replacement (1 Litre). His sodium level gradually increased by 8 mEq/day and he was discharged.

The principal mechanism of thiazide induced hyponatremia is thought to be through the blockage of sodium chloride co transporter in the proximal part of distal tubule leading to excess sodium excretion [2]. Thiazides also induce excessive anti diuretic hormone activity with renal resistance attributable to associated hypokalemia [3]. Excessive water drinking during summer months also contributes to thiazide induced hyponatremia. Another proposed mechanism

Fig. 1 : Serial serum sodium values during the hospital course

is the upregulation of aquaporin – 2 receptors in collecting duct by thiazides resulting in increased water absorption [4]. Severe hyponatremia is associated with high mortality, when treated aggressively (>20mEq/day) or when treatment is delayed. Our patient had the right setting for thiazide induced hyponatremia – his thiazide dose was recently increased, he had curtailed his salt intake and drank free water in excess due to prevailing extreme hot and humid weather. He was successfully treated with hypertonic saline, fluid restriction followed by oral salt. Recovery occurred in eight days. It has been reported that it usually takes 2-12 days for recovery of diuretic induced hyponatremia [3]. It must be remembered that overzealous and rapid correction of chronic symptomatic hyponatremia can have disastrous consequences, with irreversible neurological sequelae.

References

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