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Case Report

Behavioural Induced Severe Hypernatremia without Neurological Manifestations

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ABSTRACT. Hypernatremia is a relatively common entity and is more prevalent among the elderly and critically ill. A number of medical conditions are commonly associated with hypernatremia, and these differ substantially among children and adults. Severe hypernatremia is usually associated with central nervous system manifestations and carries a high mortality rate. We report a case of a female patient who presented to the emergency department of the King Khalid University Hospital, Riyadh, Saudi Arabia with severe hypernatremia and without any associated co-morbid conditions or neurological manifestations. We did not find any etiological background despite extensive evaluation other than under hydration due to decreased fluid intake, which was secondary to behavioural causes.

Introduction

The serum sodium concentration and thus, serum osmolality, are closely controlled by water homeostasis, which is mediated by thirst, arginine vasopressin, and the kidneys.[1] A disruption in the water balance is manifested as an abnormality in the serum sodium concentration in the form of hypernatremia or hyponatremia.[2,3] Because sodium is functionally an impermeable solute, hypernatremia contributes to hypertonic hyperosmolality leading to movement of water across the cell membrane causing cellular dehydration, at least transiently. The resultant morbidity may be inconsequential, serious, or even life-threatening, associated with mortality rates ranging from 42 to 60%.[4-7] Elderly patients are believed to be at high-risk of severe hypernatremia, because with advancing age their renal response to dehydration alters and they have a poorer thirst response.[8,9] Also, renal concentrating ability declines with age.[10] The principle serious clinical feature associated with hypernatremia is neurological involvement, which probably contributes to the high mortality rate.[11-14]

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Case Report

A 45-year-old female patient, not known to have any prior medical illness, presented to the

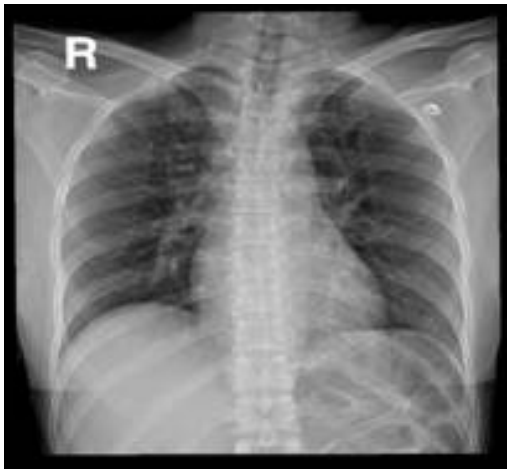

Figure 1. X-ray of the chest of the study patient

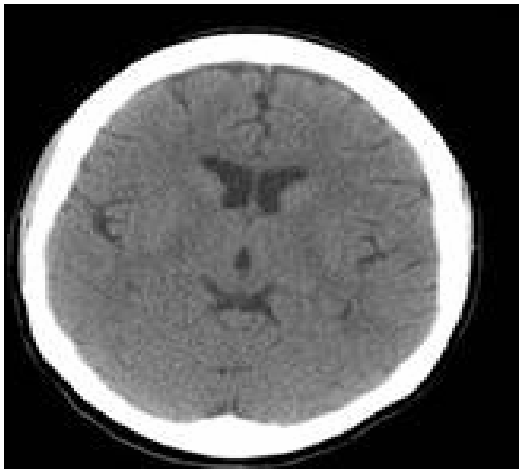

Figure 2. CT scan of the brain showing no abnormalities

emergency room (ER) with two weeks history of decreased appetite, decreased fluid intake followed by lethargy, and four days history of constipation, decrease in urine frequency and volume. She denied history of diarrhea, nausea, vomiting, fever, and excessive sweating. She also denied history of visual symptoms, recent head trauma, or history suggesting tuberculosis (TB), sarcoidosis or any possible underlying cause. There was no history of taking any drug, which can cause hypernatremia. Initially, the patient was reluctant to talk with the attending physician but later she was convinced to talk. Detailed history did not reveal any clue to justify her recent decrease in appetite, reduced fluid intake and behavioural change, but her attendant (son) was mainly concerned about fatigue, lethargy and recent behavioural change. On further questioning in the absence of her son, she gave some hints about her recent mental stress due to family matters, which were insignificant.

On physical examination, she looked dehydrated. Her body temperature was 36.8°C; heart rate, 90 beats/min; respiratory rate 18/min, blood pressure 113/78 mmHg with mild postural drop (10-15 mmHg difference) and O_2 saturation was 96% at room air. Head and neck examination was normal, and the jugular veins were not distended. Examination of the heart disclosed normal sounds without gallop or murmurs. Her peripheral pulses were full and regular. Examination of the chest revealed bilateral equal vesicular breath sounds. Abdominal examination was negative for organomegaly, masses or tenderness. There was no clubbing, cyanosis, edema, arthritis, lymphadenopathy, or rash. Neurological examination showed normal orientation to time, place and person, normal cranial nerve examination, normal power and tone of all muscle groups without any focal deficit; reflexes were brisk and plantars were flexor. Fundal examination was unremarkable.

Initial lab work-up revealed blood urea of 42.2 mmol/L, serum creatinine of 179 µmol/L, sodium (Na^+) of 191 mmol/L, potassium (K^+) of 4.0 mmol/L, chloride of 135 mmol/L, random blood sugar of 7.8 mmol/L and osmalility of 421 mosm/L. The white blood cell count (WBC) was 11,000/mm^3, with normal differentials, red blood cell count was 5.6×10^5, hemoglobin (Hgb) was 12.6 gm/dL with low MCV and MCH, mid stream urine showed Ketone 1^+, Blood 1^+, Hgb 1^+, WBC: 11, RBC: 45 and nitrites were negative. Arterial blood gas analysis showed pH of 7.39, PaO_2 of 88.6 mmHg, $PaCO_2$ of 38.3 mmHg, bicarbonate (HCO_3) of 23.8 mmol/L and oxygen saturation of 94% at room air.

She underwent radiological exam including a chest x-ray (Figure 1) followed by computed tomography of brain (Figure 2) to rule out any space occupying lesion or edema; both were normal.

On the basis of the above findings, a preliminary

Table 1. Serial Lab investigations in the study patient

S.#	Sodium (mmol/L)			Potassium (mmol/L)			Urea (mmol/L)			Creatinine (µmol/L)		
Day 1	191	182	162	4.0	3.7	3.4	42.2	30.1	20.8	179	118	107
Day 2	173	176	177	4.1	3.6	3.5	14.4	13.0	12.2	85	77	79
Day 3	157	153	149	3.3	3.2	3.6	3.6	3.1	2.9	73	68	70
Day 4	147	144	na	3.5	3.5	na	2.5	na	na	71	na	na
Day 5	142	140	na	3.4	na	na	2.8	na	na	68	na	na
Day 6	140	140	na	3.2	3.0	na	2.3	na	na	64	na	na
Day 7	140	na	na	3.0	na	na	2.7	na	na	60	na	na

diagnosis of pre-renal azotemia with severe hypernatremia possibly due to decreased fluid intake supposedly secondary to behavioural changes, was made. Since the patient's weight was 51 kg at presentation, the total body water deficit calculated with standard protocol was approximately 8500 mL. Therefore, treatment was started with 5% dextrose normal saline infusions administered at 200 mL/hour for six hours followed by 150 mL/hour; the urea and electrolytes were monitored six-hourly. The following day, the patient was treated with slow replacement of fluid under the supervision of the nephrologists. Subsequent serial lab results for urea and electrolytes showed satisfactory improvement (Table 1). The patient was then encouraged to take orally; although she was reluctant initially, the patient eventually agreed to take oral fluids.

The patient remained in the ER as a long stay patient; when she was declared to be free of any medical or nephrological problems, she was referred to the psychiatrist for evaluation. Thorough review was inconclusive for major psychiatric disorder except mild depression. After two sessions with the psychotherapist, she started to take fluids orally and was quite stable from mood point of view. Finally, on day seven in the ER, all the lab parameters of the patient reached normal limits with serum Na of 140 mmol/L, K of 3.0 mmol/L, urea of 2.7 mmol/L and creatinine of 60.0 µmol/L.

She was then discharged with the advice to attend follow-up after one week with the psychiatrist.

Discussion

Hypernatremia can be caused by derangement of the thirst response or the behavioural response thereto (primarily in infants, psychiatric patients, and elderly patients who are institutionalized) and by pathological process involving the renal concentrating mechanism such as diabetes insipidus (DI) either due to kidney pathology (nephrogenic DI) or due to difficulty with the neurohormonal control of this concentrating mechanism (central DI).[6,11-18] It can also occur due to loss of free water from other sources including gastro intestinal losses,[19] administration of diuretics such as mannitol,[20,21] impaired thirst mechanism due to hypothalamic involvement in granulamatous diseases like sarcoidosis, crohn's disease, Cushing's disease, tumours or vascular pathology.[22,23] The main feature associated with hypernatremia is neurological involvement that begins with lethargy, weakness, irritability, twitching of muscles, and can progress to disorientation, delirium, seizures and if untreated, to coma. In severe cases, it has been reported that initial low Glasgow Coma Scale and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores are associated with high mortality and poor outcome.[24-26] In the literature, some rare neurological manifestations of hypernatremia described include hydrocephalus,[27] bilateral choroid plexus haematomas and extensive destruction of choroid plexus due to diffuse intravascular coagulation.[28]

The signs and symptoms of hypernatremia are variable and evidence suggests that higher the level of sodium worse the outcome, with a mortality of up to 40%. This is explained by the complex patho-physiological phenomenon initiated by hypernatremia itself and the other coexisting conditions like metabolic acidosis, hyperglycemias, etc. which eventually lead to cellular necrosis, demyelinating lesions and hypoxic

ischemia of brain tissue.[26]

The most common cause of hypernatremia in elderly or institutionalized patients is lack of free water intake adequate to meet losses. As explained earlier, thirst is the body's main defence against increased serum tonicity. Most patients with an intact thirst mechanism and access to water can prevent the development of hypernatremia. Even patients with a defective renal concentrating mechanism such as those with DI generally can keep up with water losses (even up to 20 L/day) if they are allowed free access to water.

Some patients, however, cannot respond to their thirst drive. Infants and elderly patients who are debilitated depend on their caregivers to provide fluids. Similarly, institutionalized patients may have limited access to water secondary to either external or internal constraints. Intrinsic water losses cannot be avoided, and some urine must be produced, even if it is maximally concentrated. Without access to water, these patients develop a free water deficit, and their serum sodium level increases. It is defined as “adipsic hypernatremia” which could be of behavioural origin as seen in our patient.

In conclusion, since our patient recovered fully and no pathologic cause was identified, we speculate that the possible underlying cause of hypernatremia in our patient was behavioural neglect of fluid intake for more than two weeks.

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