EXCESSIVE WATER INGESTION AND REPEATED SEIZURES

The domino effect

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Despite variations in sodium and water concentrations, our body relies on several mechanisms to maintain the plasmatic osmolality within the normal range of 275–290 mOsm/L[1]. Hyponatremia is defined as a reduction in plasma sodium concentration below 136 mmol/L[2]. It occurs in 12% of the hospitalized patients and in 30% of the patients in the intensive care unit (ICU)[3]. Hyponatremia installed progressively usually causes no symptoms. If installed acutely, severe symptoms such as vomiting, progressive somnolence and confusion, coma and seizures often occur[2,4]. Hyponatremia related to the ingestion of free water is uncommon in healthy subjects that are neither athlete nor have psychiatric disorders[2-5]. Thus, after approval of the Hospital Ethics Committee and signed informed consent obtained from a patient representative.

We report a case of hyponatremia associated with fast ingestion of large amounts of potable water causing severe neurological impairment.

CASE

A 34-year-old male, previously healthy, was admitted in ICU of the Hospital da Cidade (Salvador BA, Brazil). The patient was transferred from the emergency department with a history of ingestion of 40 glasses of potable water, equivalent to approximately 8 liters, during a period of a few hours. The patient was playing domino and the players bet that one who loses a game should drink a full glass of water. The unlucky patient lost one game after the other and started to become sleepy, culminating with a generalized tonic-clonic seizure. Taken to the hospital, he developed new episode of seizure, lingered, requiring intubation and mechanical ventilation. The admission tests revealed plasma sodium of 123 mmol/L and mild cerebral edema at CT scan. The diagnosis of hypovolemic hyposmolar hyponatremia with low urinary osmolality ("the marathon runner's hyponatremia") was made based on plasmatic osmolality (262 mmol/L), urinary osmolality, urinary sodium (undetectable) and the volemic status was estimated by pre-load pressures. Since the installation of hyponatremia was rapid with severe symptoms, 3% saline solution was started with infusion rate of 0.5 mL/kg/h. Plasma sodium was measured hourly, aiming for an elevation of sodium level by 0.5 mEq/L/hour. After discontinuing sedation he gradually recovered, being disconnected from mechanical ventilation in the third day of hospitalization. He was discharged from ICU after five days without any neurological deficit.

DISCUSSION

The initial approach to hyponatremia requires determination of plasmatic osmolality , in order to classify the hyponatremia in hypertonic, isotonic or hypotonic. The determination of osmolar gap – difference between measured and calculated osmolality, normal <10 mOsm/kg – helps with the interpretation and suggests which solutions should be utilized in the treatment[6]. Hypertonic hyponatremia is typically observed during hyperglycemia or after mannitol use and is also known as translocational hyponatremia. Isotonic hyponatremia is diagnosed in settings of severe hypertriglyceridemia or paraproteinemias. High levels of plasma proteins and lipids increase the non-aqueous, non-Na^+ fraction of plasma while overall H_20 stays stable. That interferes in the laboratorial evaluation characterizing a pseudohyponatremia[1,2,4,6,7].

The hypotonic hyponatremia deserves special attention and should be stratified according to volemic status as hypervolemic, hypovolemic or euvolemic. The hypervolemic hypotonic hyponatremia associated with water retention is often observed in congestive heart failure, renal failure, nephrotic syndrome, hepatic cirrhosis or pregnancy. The normovolemic hypotonic hyponatremia occurs characteristically in the syndrome of inappropriate

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antidiuretic hormone secretion (SIADH), hypotireoidism, adrenal failure, beer potomania and after use of thiazide diuretics. In the hypotonic hypovolemic hyponatremia, we should measure urinary sodium to determine the etiology appropriately. A low urinary sodium (lower than 20 to 30 mmol/L) suggests an extra-renal loss of sodium, such as vomiting, diarrhea, extensive burns, excessive blood loss, retention of liquids in the third space (pancreatitis, peritonitis, extensive muscular lesion) and fluid overload as observed with fast ingestion of water by marathon runners and psychiatric patients. High urinary sodium (above 20 to 30 mmol/L) characterizes a renal loss of sodium and is an adverse effect of diuretics, agents used for osmotic diuresis (glucose, urea or manitol), salt-losing tubulopathies, ketonuria, bicarbonaturia and also in adrenal failure[1,2,4,6,7]. In our patient, determining the plasma and urine calculated osmolality and urinary sodium allowed classifying his hyponatremia as hypotonic hypovolemic.

Kugler et al. still suggest an evaluation of the urinary osmolality, because in the hypotonic hyponatremias associated with the ingestion of water, a urinary osmolality below 100 mOsm/kg is usually found[4]. In our patient, a direct measurement of osmolality was not performed, but no medications were being used causing osmotic diuresis.

Serious hyponatremia can cause important cerebral edema, neurological sequelae and progression to death[1,2]. There are several approaches to hyponatremia[2,9-12] but the key for its treatment involves the identification of the type, the rate of installation, presence of symptoms and determination of etiology. A fast installation suggests the need for fast reversion. If symptoms are mild and hyponatremia is not severe (>125 mEq/L), a conservative monitoring approach is advised[8].

If the hyponatremia is moderate (<125 mmol/L), but there are no clinical symptoms, an urgent intervention is unnecessary. If symptoms like seizures, progressive somnolence or mental confusion appears and the hyponatremia has less than 48 hours of evolution or the onset is ignored, 3% saline solution should be infused at 1–2 mL/kg/h until resolution of symptoms; this should be followed by maintaining sodium replacement with 0.9% saline by 0.5 mmol/every hour until plasma sodium rises above 125 mmol/L. After the initial approach, treatment should be driven based on patient's volemic status.

A slow correction of sodium levels is important in order to avoid osmotic demyelination syndrome of the central nervous system. There are reports in the literature on the occurrence of this syndrome with an increase of plasma sodium as low as 8 to 12 mEq/24 h[12].

In conclusion, hyponatremia was the cause of symptomatic seizures in our patient. A stratified approach to detecting the etiology of hyponatremia in patients presenting with seizures and other neurological symptoms allows prompt correction of the disturbance in an ICU setting while avoiding the complications of the treatment itself.

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