



Gabriela de Andrade Lopes¹, Laiane Cristina de Camargos Silva¹, Livia Lemos Oliveira¹, Thais Carvalho Marinelli¹, Rogério Saint-Clair Pimentel Mafra^{2*} and Ivana Mussel²

¹Medical Students, FAMINAS BH, Brazil

²Professor, FAMINAS BH Medical School, Brazil

Dates: Received: 19 June, 2017; **Accepted:** 12 July, 2017; **Published:** 13 July, 2017

***Corresponding author:** Rogério Saint-Clair Pimentel Mafra, Department of renal care, Belo Horizonte - MG, Brazil, Tel: 31 32732100; Email: rogerioscm@yahoo.com.br

Keywords: Hyponatremia; Hydroelectrolytic disturbance; Sodium; Sodium metabolism; Vasopressin

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Case Report

Elderly Severe Hyponatremia: Case Report and Literature Review

Abstract

This article describes hydroelectrolytic disorder in a 77-years-old patient accepted at an emergency room with atypical hyponatremia that has been analyzed through laboratory tests. The aim of this study is to understand cause and proper care in cases of abnormal variations of sodium levels in the body and review the literature about severe hyponatremia.

associated with each patient and for sodium saline solution parenteral replacement indication.

Methodology

Bibliographic review was performed at PubMed, LiLACS and SciELO databases with following terms: hyponatremia, hydroelectrolytic disorder, sodium. Were selected and reviewed articles of greatest relevance corresponding to the period of years between 2003 and 2017. Clinical case description was carried out, and all research rules and regulations were followed according to current legislation.

Case Report

A 77-year old female patient, 70 kg, was admitted at emergency room due of syncope followed by fall of her own height. Patient had systemic arterial hypertension (SAH), esophageal achalasia and depression, and continuous use of the following medications: indapamide 25 mg, metoprolol 25 mg, losartan 50 mg, zolpidem 10 mg, duloxetine hydrochloride 30 mg and domperidone 10 mg. Previous history of thymoma and subsequent thymectomy in 2006, and valvuloplasty due achalasia correction in 2015, with improvement of 60% of symptoms.

Patient developed prostration, lethargy, frequent falls, drowsiness and memory failures five days before admission; and urinary incontinence two days before admission. She was attended with Glasgow 15/15, without deficits and evaluated by general surgery team and neurosurgery. Computed tomography scan of cervical spine and skull was performed, demonstrating no changes.

Laboratory tests with hyponatremia, showed sodium = 99meq/L, and after repeat sodium test = 102meq/L. Due to arterial hypertension venous nitroglycerin was started.

Introduction

Most authors define hyponatremia as a serum sodium concentration below the lower limit of normality, lower than 135 meq/L, but there is still a discussion on best values of reference [1].

Characterized as a hydroelectrolytic disorder, hyponatremia is very common in elderly, hospitalized individuals and may be commonly associated with neoplasms, liver cirrhosis, heart failure and renal failure. Physiologically, it is inferred that the increase of water triggers hyponatremia and not a direct sodium low, even in situations of hypovolemic hyponatremia [1].

Thus, excess water leads to an increase in serum sodium dilution, and as a negative feedback under normal conditions, the level of antidiuretic hormone (ADH) decreases, so that the kidney collecting tubules become impermeable to water and it can be excreted in urine. However, in pathological situations, there may be a decompensation in this functioning, with several etiologies related to inadequate ADH secretion, such as antidepressant medications use [2].

Patient's symptoms vary according to hyponatremia severity, but in general, clinical manifestations are neurological, since with hypotonic extracellular environment there is a deviation of water into cells that evolves in cellular edema and intracranial hypertension, since skull prevents natural expansion of the organ [1,2].

Treatment for hyponatremia depends on clinical condition

The patient was referred to ward and intravenous administration of 0.9% saline solution was started. She was transferred to an intensive care unit on the same day, where progressed uneventfully with nitroglycerin suspension and with mild and progressive improvement of hyponatremia. Discharge from intensive care unit occurred after one day, with sodium values = 117meq/L.

After five days, patient was discharged with sodium = 130 mEq/L for outpatient control, with medication modification, indapamide suspension, and was instructed to seek medical assistant. She was evaluated by a nephrologist after 15 days, presenting normal renal function and sodium = 136 mEq/L.

Discussion

The most prevalent cation in extracellular fluid is sodium [3] Serum levels of this cation under normal conditions are maintained between 136 and 145 mEq/L. Regardless of total body sodium, a reabsorption of more than 99% (from sodium filtered by kidney) occurs through proximal tubule and Henle's loop. However it is the lowest proportion of sodium, reabsorbed in distal tubule and collecting ducts, which makes greater influence on total sodium balance [4].

Hyponatremia consists at most often electrolyte disturbance found in clinical practice, and occurs when the body's sodium concentration is less than 136 mEq/L. Although such dysfunction is associated with several causes, main one is almost always water retention. The disease may be asymptomatic (up to 125 mEq/L) or symptomatic [5].

Symptoms most commonly seen in hyponatremia are: lethargy, apathy, disorientation, paresthesia, anorexia, nausea, vomiting, agitation and seizures. Neurological symptoms are those that first present, because plasma osmolarity decrease in brain, creates a gradient that forces water into neurons, leading to edema and subsequent injury. Patients' clinical signs most commonly found are altered sensitivity, deep reflexes decrease, Cheyne-Stokes respiration, hypothermia, pathological reflexes, pseudobulbar palsy and seizures [1].

Disease can be classified as: pseudo hyponatremia, which is the cause of lipids high concentration and paraproteinemias that moves a portion of extracellular water, reducing sodium plasma portion; hypertonic hyponatremia, which occurs due to presence of body osmotically active solutes; and hypotonic hyponatremia which occurs, since kidneys ability to excrete water is less than intake [3].

Classification can also be made according to extracellular volume: Expansion of extracellular volume, which results in kidneys' water excretion decrease, with consequent increase in body's water volume. Normal extracellular volume, when hyponatremia is associated with hypovolemia, being caused by hypothyroidism, corticosteroid deficiency, emotional stress, drugs, pain, and solutes intake decrease. Extracellular volume contraction, in which there may be sodium loss through kidneys (with urine concentration increase), skin and gastrointestinal tract; and water intake excess due mainly to pathological thirst stimulus [3].

The management with a suspected patient presenting hyponatremia, consists in clinical history evaluation, volume status and physical examination with special attention to neurological exam [6].

It is known that a patient with symptomatic acute hyponatremia, which lasts less than 48 hours, may present convulsions or hyponatremic encephalopathy resulting from cerebral edema. Serum sodium usually presents in concentrations lower than 130 mEq/L and its correction should be made with replacement of 1 to 2 mEq/L/h until symptoms improvement, or until serum sodium levels reach 120 mEq/L. In patients with neurological symptoms, no more than 12 mEq/L should be restored in 24 hours and an additional 18 mEq/L in 48 hours due to osmotic demyelination risk, which causes central pontine myelinolysis, an acute disease that results in symmetrical demyelination of pontis base central part. The most commonly used treatment is 3% physiological solution administration at 1-2 mL/kg/h rate and a loop diuretic [2].

In patients who present symptomatic chronic hyponatremia, which lasts longer than 48 hours, great care is needed because of brain's osmotic adaptation to prolonged hyponatremia, and it is necessary to monitor them frequently. Correction should be limited to no more than 10-12 mEq/L at treatment's first day and 6 mEq/L on following days until serum values correction, and this correction is usually done with 0.5 mEq/L/h rate [2].

Treatment with vasopressin antagonists is already known and although they are being released, little is known about its future repercussions. These drugs are based on water's reabsorption reduction at renal collecting tubules, increasing free water excretion and decreasing sodium excretion [7].

The best therapeutic choice still consists in volume replacement with sodium and other electrolytes replacement, always prioritizing to treat hyponatremia main cause and other pathological conditions associated. In addition, some authors advocate water restriction as a simpler way of treating hyponatremia [8].

Antidepressants are associated with hyponatremia [9]. The association is strongest with citalopram and lowest with duloxetine, venlafaxine and mirtazapine [10]. In this case report, the patient was a duloxetine long-time user.

Selective serotonin reuptake inhibitors (SSRIs) are widely prescribed among the general population, despite their benign side effect profile, these drugs can cause significant adverse effects in elderly patients, including severe hyponatremia [11] Although SSRIs are the first treatment option for elderly depressed patients, they should be prescribed cautiously in this population because of the risk of potentially severe adverse effects such as hyponatremia [12]. Physicians should be aware of the significance of these medication classes.

There are several guidelines on hyponatremia, this is because the prevalence of hyponatremia in a wide variety of conditions and the fact that hyponatremia is managed by clinicians with a broad variety of backgrounds have fostered diverse institution and specialty based approaches to diagnosis and treatment [13].

Conclusion

There is no consensus on treatment, however, it is known that in order to improve it, individualized procedures should be adopted that take into account patient's comorbidities, type and hyponatremia cause.

According to presented case, it is concluded that drug association is a determining factor for hyponatremia, especially in elderly. Thus, early diagnosis is essential to avoid other complications that may arise associated with the patient's symptoms.

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