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Case Report

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Suboptimal care in a case of severe hyponatremia in an elderly male patient in the emergency department. A three-question guide for basic assessment and treatment of hyponatremia

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Abstract

Hyponatremia is a common medical disorder with high mortality and morbidity. Extensive guidelines are developed by internal medicine and critical care specialists. However, every physician should be familiar with initial assessment and treatment of hyponatremia. A case of a severe hyponatremia presenting to an emergency room is described where care was suboptimal. This case illustrates that diagnosis and treatment are not always straight forward. Three questions are proposed to guide physicians in the immediate care of hyponatremic patients.

Introduction

Hyponatremia (HN) is a common metabolic disorder in up to 30% of hospitalized patients and might be underreported [1,2]. The most frequent cause of HN is the syndrome of inappropriate antidiuretic hormone (SIAD). HN is associated with high in-hospital and long-term mortality and high morbidity [1-3]. Even mild HN may lead to falls and attention deficits in the elderly [4]. Guidelines are traditionally developed by internal medicine- and critical care societies, based on sparse scientific evidence [5-7]. HN can be categorized according severity (severe or profound <120 mmol/l, moderate 120-130 mmol/l, mild 130-135 mmol/l), tonicity (hypotonic, isotonic, hypertonic), volume status (hypovolemic, euvolemic, hypervolemic), duration (acute <48 hours of chronic > 48 hours) or symptomology. The various guidelines present different diagnostic and therapeutic algorithms, and contain extensive expert diagnostic options like co-peptin, fractional excretion of urea of uric acid and treatment options like demeclocycline, urea, vaptans [5,7-10]. Despite these guidelines HN remains challenging to diagnose and treat, demonstrated by the reported suboptimal care [11]. Complicating factors include the presence of multiple contributing factors (e.g. malignancies, drugs, low sodium intake) [12]. Also, classifying HN based on the recommended physiological assessment of volume status may lead to a wrong diagnosis [13].

The responsibility for treating HN does not lie solely with internal medicine or critical care specialists. Every physician, especially in the field of emergency medicine, should be familiar with the urgent diagnostic and treatments steps in HN. A case is described of severe HN in an elderly male patient, in which initial care was suboptimal. Three questions are formulated, and related to recent literature, which may guide every physician in assessing an hyponatremic patient.

Case report

A male octogenarian was referred to the emergency department because of general deterioration (day 0). His medical history mentioned an ischemic cerebrovascular accident and hypertension. His prescribed home medications were acetylsalicylic acid, atorvastatin, perindopril/indapamide; which were last ingested on the day before admission. He did not drink alcohol but had smoked more than fifty pack years. The last months he had no appetite and lost 20 kilograms in weight. In the last week there was almost no food intake; his fluid intake seemed adequate. He became increasingly confused and had fallen two times in the last week. Also, the patient became incontinent for urine. Blood testing showed: sodium 103 mmol/l, potassium 4,6 mmol/l, magnesium 0,6 mmol/l, corrected calcium 2,2 mmol/l, phosphate 0,98 mmol/l, glucose 6,1 mmol/l, creatinine 102 umol/l, hemoglobin 8,2 mmol/l, CRP 14 mg/l with a mild leukocytosis. At physical examination the patient was restless and disorientated in time, place and person. His blood pressure was normal; there were no evident signs of hypovolemia or fluid overload. A CT-scan of the brain showed no posttraumatic or other lesions. Catherization showed a urinary retention of 1000 milliliter.

For a symptomatic HN 100 ml of sodium chloride 3% (30 mg/ml) was administered. Also, 500 milliliter of sodium chloride 0,9% was infused. Because of the severity of the sodium disturbance the Intensive Care Unit (ICU) was consulted for correcting the sodium level and to monitor for adverse events. Next adjuvant testing was performed, which showed serum osmolality of 221 mosmol/kg, urine osmolality 378 mosmol/kg and urine sodium level of 38 mmol/l. In the ICU the patient was put on fluid restriction of 1000 milliliter per day. The ACE-inhibitor and diuretic were stopped. Because of the severity of HN and because the mild cognitive symptoms possibly related to the

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low sodium level, a slow continuous infusion of NaCl 3% was started, aiming for a 5-8 mmol/l increase in the first 24 hours (Figure 1). After 24 hours serum sodium level was 109 mmol/l. On the second day of admission after another 14 hours the sodium level was 115 mmol/l. The sodium chloride 3% infusion had been decreased and stopped in two days. Patient was transferred to the general ward on day 2, his cognitive deficits were minimal. A fluid restriction was maintained of 1500 milliliter per day, the sodium level slowly increased to 130 mmol/l. On day 9 he was discharged from the hospital. During admission in the ICU no somatic cause was found for the HN: X-ray of the thorax was normal PSA was normal, as were kidney-adrenal-thyroid-liver functions. Follow up will take place on an outpatient basis.

Discussion

Every physician should be familiar with the initial urgent diagnostic and treatment steps for HN. Three important questions, which are described below, can guide the physician.

Is the hyponatremia symptomatic?

The most important complication of HN is cerebral edema due of movement of water following an osmotic gradient into the cell. After 48-hour active osmotic particles are transported out of the cell as a compensating mechanism [6]. Therefore, an acute decrease in sodium level is more likely to cause edema. To cause cell swelling hyponatremia has to be hypotonic, in isotonic or hypertonic hyponatremia other osmoles are present (e.g. glucose, glycine) which attract water and dilute the serum sodium. Low serum osmolality is often used to exclude isotonic or hypertonic hyponatremia; however, serum osmolality also measures urea which is not an osmole. When serum osmolality is not available the formula for effective osmolality can be used: Effective osmolality (mmol/kg H2O) = 2 × (serum sodium (mmol/l) + serum K (mmol/l)) + serum glycaemia (mmol/l) [6]. Also, pseudohyponatremia might be excluded, using serum for example triglycerides or protein levels.

Hypotonic hyponatremia can cause a spectrum of signs and symptoms ranging from life threatening to subtle cognitive disorders or can even be asymptomatic [8,14]. A severe complication of overcorrecting serum sodium level is osmotic demyelination syndrome. Whether immediate treatment is indicated, depends on the severity of symptoms. Risk of edema is deemed more severe than the risk of osmotic demyelination in case of severe symptoms. Guidelines and treatment algorithms offer various and different categorizations of symptoms based on severity [5-7]. Well known examples of severe symptoms are coma and convulsions. But less clear are the patients with nausea, somnolence, confusion, cognitive deficits or headache. Vomiting, cardiorespiratory distress, seizures and coma are consequently rated as severe. Muscle rigidity is sometimes also rated as severe. Nausea without vomiting and headache are rated as moderately severe. Somnolence is rated as severe or moderate symptom depending on source. Confusion is categorized as moderate in some guidelines.

In case of severe symptoms guidelines and treatment algorithms recommend immediate administration of a bolus of sodium chloride 3%, the recommended dosage varies from 100 to 150 ml or 2 ml/kg (5-7). With persistent severe symptoms a bolus can be repeated twice; with a target increase of 4-6 mmol to reverse serious complications. Treatment recommendations differ in case of moderate severe symptoms, a single bolus of hypertonic saline or slow continuous infusion of hypertonic saline is advocated.

Is the hyponatremia acute?

As stated above acute HN is more likely to be (come) symptomatic due to cerebral edema. Because of the incomplete adaptation mechanism, the cut-off between acute and chronic HN is 48 hours. If the duration of hyponatremia is not documented, it is regarded as a chronic hyponatremia. With acute hyponatremia, adaptation of the brain is not completed and the risk for pontine demyelination might be. According to the European guideline a single infusion of 150 ml sodium chloride 3% can be given [6]. In chronic HN the goals and

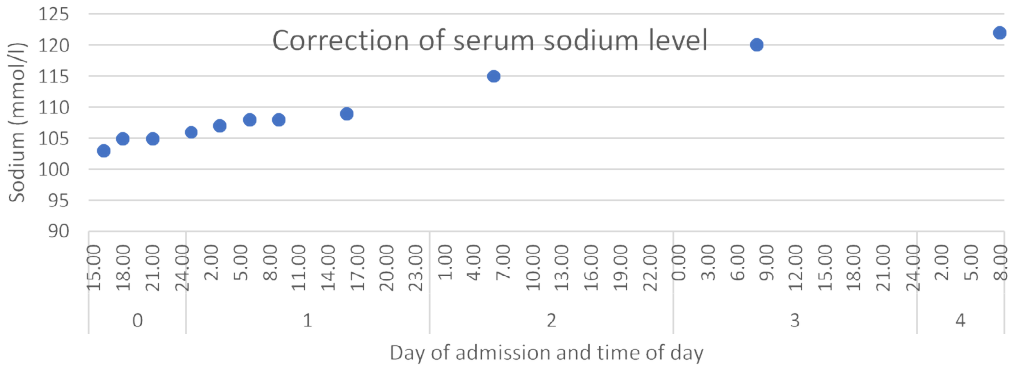

Figure 1. Rate of increase of sodium lever during the first five days of admission

Table 1. Three questions guiding initial assessment with basic diagnostic and treatment considerations

Questions	Diagnostic considerations	Treatment considerations
Is the hyponatremia symptomatic?	Pseudohyponatremia, Alternative diagnosis, Serum osmolality, Plasma glucose, Effective Osmolality	Yes: 100ml – 2ml/kg bolus NaCl 3%, up to three times in severe symptoms No: slow correction, cause specific
Is the hyponatremia acute?	Documented serum sodium levels	Yes: Faster correction, bolus NaCl 3% or continuous infusion No: slow correction, cause specific
What is the cause of the hyponatremia?	Expert consultation, Serum and Urine electrolytes, Urine osmolality, Liver- kidney- adrenal- thyroid insufficiency, Drugs, Sodium intake SIAD: infection, malignancy	Cause-specific treatment: e.g. stop precipitating factors, fluid restriction

limits of sodium level correction became more conservative over time, the limits are around 8-10 mmol/day.

What are the precipitating factors for hyponatremia?

Hyponatremia is a heterogenous disorder with many possible causes. After the first questions are answered, the treatment of HN should be cause-specific. Syndrome of inappropriate antidiuretic hormone (SIAD) is the most frequent cause of hyponatremia, however, to diagnose SIAD other possible causes should be excluded. Also, SIAD can be caused by various underlying diseases [8,15,16]. Guidelines present various diagnostic algorithms using different clinical or laboratory parameters [5,7-9]. As stated earlier, the use of recommended clinical parameters might lead to wrong diagnoses. Also, patients may have multiple factors contributing to the, and the etiology might not become clear (immediately). After finding HN and after the immediate treatment steps, the diagnostic steps and correction of sodium levels are often under the responsibility of internal medicine of critical care specialists. However, basic investigations should be requested by every physician. Prescribed medication should be screened and drugs which may contribute to HN should be stopped. Table 1 contains diagnostic and treatment considerations for each question (Table 1).

Hereafter, the questions are applied to the described case about an older male patient with a severe hyponatremia.

Is the hyponatremia symptomatic?

Important basic tests were not performed before administering a bolus of hypertonic saline. Afterwards a low serum osmolality was found, and the effective osmolality was 227,6 mmol/kg H2O. The patient was confused and disorientated, and these symptoms are generally not categorized as moderate. Moreover, the symptoms can also be explained as a delirium caused by the urinary retention. So, it can be argued whether the hypertonic sodium bolus should have been given, with potential pontine demyelination as severe complication. Another option is to stop precipitating factors and observe sodium levels, or to administer a slow continuous infusion.

Is the hyponatremia acute?

Since the duration of the hyponatremia is unknown, the disorders should be treated as a chronic. Regarding the duration of HN, a bolus of hypertonic saline does not seem to be indicated.

What are the precipitating factors for hyponatremia?

Low blood and high urine osmolality and urine sodium levels are consistent SIAD, however low sodium intake and use of diuretic exclude a formal diagnosis of SIAD. The patient is treated with fluid restriction as first line treatment. Elderly should be screened for a malignancy as a precipitating cause. In this a case an X-ray did not show signs of pulmonary disease. Also, prostatic carcinoma seems unlikely due to low serum PSA-levels. A rare cause of SIAD described in literature is acute urinary retention [17]. Other factors might have contributed to the low sodium levels in this case are: low dietary sodium intake and the use of angiotensin converting enzyme inhibitor and thiazide-related diuretic. Kidney, adrenal, liver and thyroid testing were normal. This case points out that especially in elderly multiple factors may contribute to the development of HN.

In this case important basic testing like serum osmolality, urine testing and liver-adrenal-thyroid function was not performed before administering hypertonic saline. Also, this case shows that it is not always clear if subtle or moderate symptoms are related to the HN and whether they are severe enough to administer hypertonic saline. Often multiple causes of HN can be found.

Conclusion

Every physician should be familiar with the initial diagnostic steps and urgent treatment of hyponatremia. Three questions can be used as guidance. Is the hyponatremia symptomatic? Is the hyponatremia acute? What are the precipitating factors for the hyponatremia?

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Conflict of Interest

The author has no conflict of interest.

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