



CASE REPORT

Extreme Hypernatraemia and Sepsis in a Patient with Huntington's Dementia: A Conundrum in Fluid Management

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Summary

We report a unique case of extreme hypernatraemia of 196 mmol/L and severe sepsis in a young 39-year-old adult with Huntington's dementia, which presented a challenge in fluid management. The hypernatraemia was thought to be caused by chronic severe dehydration from poor intake and the sepsis was thought to have started as an inadequately treated urinary tract infection. The patient was initially treated aggressively with hypotonic saline and intravenous antibiotics but was subsequently managed using a slower correction rate after identifying the chronic nature of the naatraemia. To our knowledge, this is the first reported case of extreme hypernatraemia and severe sepsis manifesting concomitantly in such a young patient. We highlight the difficulties of balancing the risks and benefits of rapid versus slow fluid resuscitation in such complex clinical situations. We advocate early identification of chronic hypernatraemia, accurate calculation of free water deficit and effective administration of intravenous fluids that is decided on a case-by-case basis.

to 60% [2-5]. According to one study, approximately 1 to 2% of patients attending the Emergency Department have hypernatraemia, often with a variety of aetiologies [6]. Hypernatraemia over 160 mmol/L is considered to be very severe [7] and as such needs to be treated with special caution. We report a unique case of extreme hypernatraemia and severe sepsis in a young adult with Huntington's dementia, which presented a conundrum in fluid management in our Emergency Department.

Case Presentation

In November 2014, a 39-year-old man with Huntington's disease presented to the Emergency Department with reduced consciousness and signs of dehydration. He lived in a nursing home receiving one-to-one care and was unable to communicate verbally due to Huntington's dementia. 3 days before his admission, he had been started on trimethoprim for a confirmed urinary tract infection by his General Practitioner. On admission he was tachycardic at 104 beats per minute, tachypneic at 26 breaths per minute and had an SpO₂ of 83%. Examination showed diaphoresis, signs of severe dehydration and basal crepitations on auscultation. Initial blood tests showed a sodium level of 196 mmol/L, leukocytosis (total white cell count of 26.3 × 10⁹/L, neutrophil count of 23.8 × 10⁹/L) and renal impairment (blood urea of 34.1 mmol/L and serum creatinine of 217 mmol/L). Chest radiograph revealed no active pulmonary patho-

Background

Hypernatraemia is a common electrolyte disturbance defined as a rise in serum sodium level exceeding 145 mmol/L, or more specifically as a loss of total body water relative to electrolyte content. It is a clinical condition which presents in various ways depending on the severity, ranging from headaches to seizures and coma [1]. It is more common in the elderly population and children [1], with adult mortality rates ranging from 20



Citation: Venkatesh H, Ramachandran S, Basu A, Nair H (2019) Extreme Hypernatraemia and Sepsis in a Patient with Huntington's Dementia: A Conundrum in Fluid Management. Int J Crit Care Emerg Med 5:064. doi.org/10.23937/2474-3674/1510064

Accepted: January 03, 2019; **Published:** January 05, 2019

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logy. A low urine output excluded diabetes insipidus. A diagnosis of extreme hypernatraemia and acute kidney injury secondary to presumed urosepsis was made and initial treatment in the Emergency Department included aggressive fluid therapy (1000 ml of 0.45% saline over 3 hours), Intravenous (IV) antibiotics and hydrocortisone.

However, after carefully reviewing the patient and his background history, we further deduced that his hypernatraemia was caused by chronic dehydration. After calculating his free water deficit, his IV fluid regime was slowed down with the aim of reducing his sodium levels by no more than 8 mmol/L per day. He was then transferred to the Acute Medical Unit under the care of the renal physicians.

Outcome and Follow-up

On Day 2 of his admission the patient's sodium levels had come down to 186 mmol/L, however he became more unresponsive and was generally deteriorating. His blood culture returned positive for *Staphylococcus aureus*, which was sensitive to the antibiotics he was already prescribed. After speaking to his family, it was agreed that he was forward-based care and not for resuscitation. It was decided that he be put on the End of Life Care Pathway, through which palliative care was initiated. By Day 3, sodium levels had lowered to 180 mmol/L, but the patient further deteriorated due to possible aspiration; his arterial blood gas was in keeping with a picture of severe hypoxia. He passed away on Day 4, after treatment was withdrawn the night before in order to keep him comfortable.

Discussion

We report the case of a patient with Huntington's dementia presenting with extreme hypernatraemia, complicated by severe sepsis. To the best of our knowledge, this is the first reported case of both clinical issues manifesting concomitantly, raising a challenge in fluid management. Extreme hypernatraemia is treated by calculating the free water deficit [8] and administering slow IV fluids generally aimed at lowering the sodium level cautiously in order to prevent cerebral oedema [9].

However, given the rarity of cases of extreme hypernatraemia, the evidence base supporting interventions in such situations is lacking. Few reports have discussed cases of extreme hypernatraemia in adults, which were managed using slow hypotonic saline [10] and haemodialysis [9]. Both methods were effective in treating extreme hypernatraemia, with no neurological sequelae. One study showed that there is a poorer prognosis for patients presenting to hospital with hypernatraemia and suggested that less aggressive fluid therapy may be beneficial for such patients [11]. In contrast, an important study retrospectively evaluating treatment and outcomes for 85 patients admitted to the Emergency Department with severe hypernatraemia (>

150 mmol/L) revealed that correcting hypernatraemia too slowly was also associated with increased mortality, regardless of initial natraemia [12]. One issue identified by this study pertaining to ineffective management of severe hypernatraemia was that only 3.5% of the patient cohort was weighed - an essential component for calculating free water deficit. Furthermore, the study highlighted that patients who were institutionalised and suffered from dementia were particularly at risk of developing severe hypernatraemia, reflecting the characteristics of our patient. Although consensus has not been reached regarding optimal correction, experts agree that correction speed should not exceed 1 mmol/L/hour, whilst simultaneously aiming to shorten period of hypernatraemia [1,13-15]. In our case, however, the aforementioned considerations need to be weighed up against the need for more aggressive fluid therapy to maintain end organ perfusion in the context of sepsis.

In summary, we highlight the difficulty of balancing the risks and benefits of rapid fluid resuscitation, necessitated by severe sepsis and acute kidney injury, against the complexities of fluid resuscitation in correcting such an extreme hypernatraemia.

Learning Points

- Fluid therapy regimes in complex clinical scenarios should be decided on a case-by-case basis, taking into account all clinical factors and the underlying physiology of the patient.
- The presence and aetiology of chronic hypernatraemia should be identified as early on in the course of the patient's care as possible. This is especially important in high risk patients such as those institutionalised and suffering from dementia.
- In cases of severe and extreme hypernatraemia in particular, accurate calculation of free water deficit is essential to manage the patient effectively. In line with this, we must ensure that such patients are always weighed on admission.

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