Generalized tonic–clonic seizure in recent polytrauma: a case of severe hyponatraemia

Introduction

Desmopressin is a vasopressin analogue used in the treatment of central diabetes insipidus, haemostatic disorders such as von Willebrand disease and for enuresis. It is generally well tolerated but hyponatraemia is a known complication. There are numerous reports of hyponatraemia as a result of long-term desmopressin treatment, but only a few reports describe severe hyponatraemia caused by short-term desmopressin treatment.

This article presents a 47-year-old man with von Willebrand disease who developed generalized seizure and severe hyponatraemia (112 mmol/litre). Two days earlier, the patient had suffered a traffic accident which resulted in a fracture of the nasal bones and haematoma of the left arm, and desmopressin had been started for the prophylaxis of bleeding. The patient was admitted, desmopressin was discontinued, the patient was placed on fluid restriction and hyponatraemia was corrected, following which he made a full recovery.

Discussion

Desmopressin is used to treat haemostatic disorders, central diabetes insipidus and nocturnal enuresis. This drug works in haemostasis by stimulating the release of endogenous von Willebrand factor and factor VIII into the plasma and in diabetes insipidus by increasing absorption of free water in the nephron (Juul et al, 2014). Desmopressin has a relatively benign side-effect profile (Hjälmås et al, 1998) – the primary associated adverse reaction is hyponatraemia (Vande Walle et al, 2007). Severe hyponatraemia causes life-threatening bradycardia, hypertension, altered temperature regulation, obtundation, generalized seizure, respiratory arrest, coma and death (Fraser and Arieff, 1997).

There are multiple reports of mild to moderate cases of desmopressin-induced hyponatraemia as a result of long-term treatment, but few reports of desmopressin-induced severe hyponatraemia as a result of short-term therapy.

In the current report there are several possible causes for the severe hyponatraemia. Desmopressin was probably prescribed at excessive dosage and the patient did not restrict his fluid intake as is typically indicated when taking desmopressin as he had not been advised to do so.

This case shows the importance of advising both the patient and the patient's family about the need to restrict the patient's fluid intake during desmopressin therapy to prevent desmopressin-induced hyponatraemia.

When severe hyponatraemia has developed, more aggressive treatment is indicated to prevent further morbidity. In the first instance desmopressin should be stopped, the patient must restrict his/her fluid intake and then the hyponatraemia

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CASE REPORT

A 47-year-old man presented to the emergency department with general malaise, nausea and a confused mental state. In the first minutes of clinical observation he had a generalized tonic–clonic seizure. The past medical history was positive for von Willebrand disease. Two days earlier, the patient had suffered a traffic accident which resulted in a fracture of the nasal bones and haematoma of the left arm, and he had been given desmopressin for the prophylaxis of bleeding (intravenous desmopressin 26 µg/day for 2 days). His family reported that the patient typically drank 3 litres of water daily. The patient was not advised to restrict his water intake when the desmopressin was started.

Initial vital signs were temperature 37.3°C, blood pressure 170/90 mmHg, heart rate 97 beats/min, respiratory rate 20 breaths/min and oxygen saturation 96% on room air. On neurological examination he had a post-ictal state characterized by drowsiness, confusion, nausea and disorienting symptoms. Positive findings also included minimal epigastric tenderness. Electrocardiogram revealed sinus rhythm without ST segment alterations.

Admission laboratory values were significant for severe hyponatraemia (112 mmol/litre, normal range 135–145 mmol/litre), serum osmolarity was 238 mmol/litre (normal range 275–300 mmol/litre), the complete blood count was normal as were liver function tests, renal function, remaining electrolytes and urine toxicology.

A non-contrast computed tomography of the head demonstrated no acute intracranial processes.

Hyponatraemia as a result of desmopressin was suspected, so desmopressin was discontinued and intravenous infusion of 0.9% saline was started at 100 ml/h. After 8 hours the patient was admitted to the internal medicine department for careful monitoring of fluid balance.

When he entered the department his neurological state worsened and he became unresponsive. His serum sodium level was 113 mmol/litre. An electroencephalogram showed slowing diffuse rhythms, signs of a post-ictal state.

In the internal medicine department a 3% hypertonic saline infusion was administered at 45 ml/h. After 1 day of treatment the serum sodium level was 119 mmol/litre and his mental status and symptoms had improved.

The patient was also placed on an 800 ml/day fluid restriction and the saline infusion was combined with intravenous furosemide (40 mg/day). After another 3 days of therapy the patient's neurological state had completely recovered.

On discharge the serum sodium level was 13 mmol/litre and serum osmolarity was 282 mmol/litre. The patient was instructed to discontinue the desmopressin.

At follow up his sodium levels remained stable at between 137 and 142 mmol/litre.

corrected with intravenous saline infusion (Reddy and Mooradian, 2009). The plasma sodium level can be raised rapidly by 1–2 mEq/litre/h (no more than 8–10 mEq/litre/24 h) until the neurological symptoms stop (Palmer et al, 2003).

Infusion of 3% hypertonic saline is the best way to raise the sodium concentration to treat acute and symptomatic hyponatraemia. In patients prone to develop volume overload, hypertonic saline infusion is often combined with furosemide to prevent rapid expansion of extracellular fluid (Adrogué and Madias, 2000).

In this case correction was started with intravenous normal saline infusion and then, following poor clinical and laboratory response, with intravenous hypertonic saline (3%). With this approach the patient's neurological state completely recovered. **BJHM**

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LEARNING POINTS

- Severe desmopressin-induced hyponatraemia may develop after a few days of treatment.
- During desmopressin therapy it is essential to monitor the patient's daily fluid intake.
- In cases of severe hyponatraemia aggressive therapy is indicated to prevent neurological disability.

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A huge cavity in the right upper abdomen

A 71-year-old woman with uncontrolled diabetes presented with a 1-week history of fever and abdominal pain. She also reported poor appetite, nausea and dysuria. Physical examination revealed knocking tenderness over her right flank. Laboratory analysis revealed anaemia, leukocytosis and elevated C-reactive protein level. Abdominal radiograph revealed a huge cavity in the right upper abdomen (*Figure 1a*), with computed tomography showing complete duplication of the right collecting system and emphysematous pyelonephritis (*Figure 1b*). Her clinical condition recovered after percutaneous nephrostomy tube drainage and antibiotic treatment. The pus culture finally yielded *Propionibacterium acnes*.

Emphysematous pyelonephritis is a severe necrotizing infection caused by gas-forming organisms within the renal parenchyma. Complete ureteral duplication may result in one ureter normally opening into the bladder and the other being ectopic. Ectopic insertion usually causes reflux, obstruction and infection. Treatments include adequate drainage and antibiotic treatment. Minimal invasive surgery, such as laparoscopic heminephrectomy, is recommended for recurrent infection. **BJHM**

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Figure 1. a. Abdominal radiograph revealed a huge cavity over the right upper abdomen. **b.** Abdominal computed tomography revealed complete ureteral duplication (arrows) and emphysematous pyelonephritis.