

Recognizing acute hyponatremia

The nurse grows suspicious when a patient with acute abdominal pain becomes lethargic.

By Ira Gene Reynolds, MSNEd, RN, PCCN-CMC



SAM JOHANSEN, age 42, is admitted to the medical-surgical unit after 3 days of acute nausea, vomiting, and watery diarrhea associated with acute abdominal pain. His nurse, Megan, obtains initial vital signs: blood pressure (BP) 122/71 mm Hg, heart rate 85 beats/minute (bpm) and regular, respiratory rate (RR) 18 breaths/minute, and temperature 36.5° C (97.7° F). She notes his history of Crohn's disease and childhood seizures.

An abdominal X-ray shows a possible small-bowel obstruction. Significant laboratory results on admission include serum sodium level 132 mEq/L, serum potassium 3.2 mEq/L, blood urea nitrogen (BUN) 28 mg/dL, serum creatinine 1.6 mg/dL, hemoglobin 19.3 g/dL, and hematocrit 47.2%.

A nasogastric (NG) tube is placed to help resolve the bowel obstruction. Mr. Johansen is put on nothing-by-mouth status. As ordered, Megan starts an I.V. infusion of 0.9% normal saline with potassium 20 mEq at a rate of 200 mL/hour for 1 liter, and then decreases the rate to 125 mL/hour.

History and assessment hints

Seven hours later, Megan documents NG tube output at 1,500 mL of greenish/brown liquid. She notes Mr. Johansen has had three episodes of watery diarrhea.

When he complains of a headache, she administers acetaminophen 650 mg, as ordered. Two hours later, he reports his headache is worse. He seems irritable and has a hard time remembering where he is and why he's in the hospital. Soon he becomes lethargic. Megan notes his heart rate is 112 bpm and irregular; his other vital signs are normal. But she's concerned about his neurologic findings and decides to call the rapid response team (RRT).

On the scene

The latest laboratory results come back: sodium 122 mEq/L, potassium 2.8 mEq/L, BUN 18 mg/dL, creatinine 1.1 mg/dL, hemoglobin 12.3 g/dL, and hematocrit 41.6%. These findings indicate Mr. Johansen has lost a lot of sodium and potassium through NG-tube and urine output, as well as some dilution from I.V. fluids; fortunately, his kidney function is intact. The physician diagnoses him with hyponatremia and hypokalemia,

and Mr. Johansen is transferred to the intensive care unit (ICU) for electrolyte replacement and ECG monitoring.

Outcome

In the ICU, Mr. Johansen receives I.V. hypertonic saline boluses followed by normal saline solution, vasopressin antagonists, and I.V. potassium. Clinicians monitor his fluid intake and output closely. Within a few days, Mr. Johansen's electrolyte levels return to normal and his neurologic status improves, eventually returning to normal. His small-bowel obstruction, abdominal pain, and nausea resolve.

Education and follow-up

In acute hyponatremia, the serum sodium level drops abruptly over less than 24 hours. The most common causes are nausea, vomiting, and diarrhea. Also, the patient loses potassium and fluid volume. Many hyponatremic patients present with dehydration; I.V. fluids given to correct dehydration must be monitored closely to avoid serum sodium and potassium dilution. With Mr. Johansen, the small-bowel obstruction complicated the situation by necessitating an NG tube; suctioning gastric contents via an NG tube removes additional sodium and potassium.

Early signs and symptoms of hyponatremia include nausea and malaise, which may arise when sodium concentration falls below 130 mEq/L. As the level continues to drop, the patient may experience headache, irritability, lethargy, confusion, and obtundation. When the sodium level falls below 120 mEq/L, seizures, coma, and respiratory arrest may occur. A sodium-water mismatch in the brain leads to hyponatremic encephalopathy. Acute encephalopathy can be reversed, but it must be done slowly over several days to avoid permanent damage.

Thanks to Megan's suspicion about Mr. Johansen's neurologic findings, he averted catastrophic consequences of acute hyponatremia. ★

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