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Case Study

Hyponatraemic seizures following prostate brachytherapy

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Abstract

Aim: To demonstrate the importance of fluid management in the perioperative period by presenting a case of hyponatraemic seizures following prostate brachytherapy.

Case: A 61-year-old gentleman, who had prostate cancer but was otherwise well, developed confusion and word-finding difficulties the day after prostate brachytherapy. This was followed by tonic–clonic seizures that necessitated treatment, intubation and ventilation, and admission to the intensive care unit. Investigations revealed serum sodium of 116 mmol/L. Fluid balance was inadequately recorded, but the patient had drank more than 3 L of water before he developed hyponatraemia.

Discussion: Postoperative severe hyponatraemia and hyponatraemic encephalopathy develop because of anti-diuretic hormone release and hypotonic fluid administration. These are medical emergencies and should be managed in an intensive care unit. Symptoms range from headache, nausea and confusion to seizures, respiratory arrest and death, and are related to cerebral oedema. Treatment is done using hypertonic sodium chloride to increase the serum sodium to safe levels. Care should be taken to avoid overly rapid correction of serum sodium. Complete documentation of fluid balance is essential to allow proper assessment of fluid status. Patients should be advised on appropriate oral fluids in the postoperative period.

Key words: Hyponatraemia; prostate brachytherapy; seizures

CASE

A 61-year-old gentleman with locally advanced prostate adenocarcinoma was treated with high-dose rate (HDR) brachytherapy under general anaesthesia. He was on hormone manipulation with Bicalutamide for prostate cancer, but was otherwise well and took no other regular medication. As part of the general anaesthesia and pre-operative medications during HDR brachytherapy, he was administered Propofol 160 mg, Fentanyl 100 μg, Ondansetron 4 mg, Gentamicin 160 mg and Rocuronium 30 mg. About 2 L of compound sodium lactate were administered intravenously.

Overnight, after the procedure, the patient vomited around 1,000 mL in total and was noted to have low blood pressure (96/62 mmHg). Urine output from the urinary catheter decreased. Bladder scan demonstrated no residual volume. He was encouraged to increase his oral intake of fluid and was retrospectively noted in nursing documents to have drunk more than 3 L of water over

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the course of the morning. Fluid balance charts were incomplete, however.

In the early afternoon, the patient became confused, disorientated and developed word-finding difficulties. Observations of haemodynamic status, temperature and respiratory function were stable compared with before.

Shortly after, the patient had four tonic–clonic seizures, which were terminated with intravenous Diazepam and Phenytoin. Arterial blood gases revealed a serum sodium of 116 mmol/L. Before the procedure, he had normal serum sodium levels. He was intubated and ventilated to protect his airway and was transferred to intensive care unit. An urgent computed tomography scan was performed, which did not demonstrate any intracranial abnormality. Intravenous 0·9% sodium chloride was cautiously administered to avoid rapid correction of hyponatraemia.

Paired serum and urine electrolytes and osmolalities demonstrated a low serum osmolality 271 mmol/L, low serum sodium 119 mmol/L, high urinary osmolality 464 mmol/L and high urinary sodium 116 mmol/L.

Over the next 48 hours, the sodium level corrected to 134 mmol/L. Urine output was noted to improve.

The patient recovered successfully with no neurological sequelae resulting from this episode. Subsequent blood tests revealed normal serum sodium.

DISCUSSION

Hyponatraemia is the most common electrolyte abnormality[1] and is defined as a serum sodium level of < 135 mmol/L.[2] It usually reflects a state of excess body water relative to sodium either because of dilution of sodium levels by excess water or by losses of sodium exceeding that of water.[3]

Severe hyponatraemia is defined as a serum sodium <125 mmol/L and has been observed in around 1% of patients.[4] Where is it occurs over 48 hours or less, a potentially fatal hyponatraemic encephalopathy may occur as a complication.[3,5,6] Features range from headache, nausea and vomiting, agitation and confusion to seizures, non-cardiogenic pulmonary oedema, cerebral herniation and respiratory arrest. These effects are related to cellular oedema caused by movement of water along an osmotic gradient from the extracellular to the intracellular space.[3,7]

Postoperative hyponatraemia has been shown to occur in around 4% of patients.[8] In the postoperative period, most patients are well hydrated not only from intravenous fluid administration during the procedure but also because of release of anti-diuretic hormone (ADH). A study has shown that all patients have elevated ADH levels after the surgery.[8] ADH acts at the kidney to retain water leading to dilution of the blood. Under normal circumstances, ADH is released in response to hypovolaemia to prevent dehydration. However, other stimulants for the release of ADH include stress, pain, trauma, nausea, vomiting and sub-clinical volume depletion,[9] all of which are common after surgery and general anaesthesia. The resulting excess of water to sodium in the blood results in hyponatraemia.

Hyponatraemic encephalopathy occurs postoperatively when hypotonic fluid is administered. Clinically, the effects of ADH secretion manifest as a reduced urine output, as water is being conserved at the kidney; however, most patients with postoperative hyponatraemia are euvolaemic and are not dehydrated. If a careful assessment of clinical fluid status and correlation with biochemical evidence of dehydration is not undertaken, and fluid replacement initiated merely because of low urine output, the patient may develop overhydration, compounding the effects from ADH release. If the fluid replacement used is hypotonic, for example, intravenous 5% dextrose or oral tap water, then intracellular oedema may result, as in the presence of ADH the kidneys cannot effectively excrete the excess fluid.[7–9]

Acute severe hyponatraemia is a medical emergency. Patients with neurological symptoms that may herald impending cerebral oedema should be cared for in an intensive care unit. There is a high risk of life-threatening complications including seizures, cerebral herniation and respiratory arrest.

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Prompt treatment with hypertonic sodium chloride 3% should be started, with the increase in serum sodium by 1–2 mmol/L/hour. Boluses of 3% sodium chloride should be initially administered to increase the sodium by 4–6 mmol/L. The overall increase per 24, 48 and 72 hours should be 6–8, 12–14 and 14–16 mmol/L. Serum sodium should be monitored every 2 hours, and close monitoring of urine output is essential as patients may begin to self-diurese and risk rapid increases in sodium levels. Once symptoms have resolved treatment with hypertonic sodium chloride should be stopped.

The main risk with overcorrection or too rapid correction of sodium levels is the development of osmotic demyelination syndromes. The risk with acute hyponatraemia is lower than with chronic hyponatraemia, as brain cells have not yet adjusted to the lower sodium levels; however, correction of sodium in line with the above ranges is recommended.[3,7,9–11]

We report the first case in the literature of acute severe hyponatraemia after prostate brachytherapy. Symptoms that might have initially suggested hypovolaemia and dehydration, including vomiting, low blood pressure and reduced urine output, were in retrospect more likely caused by a combination of factors including general anaesthesia and operative trauma from placement of the brachytherapy seeds, with subsequent ADH release. Careful clinical assessment of hydration status and biochemical correlation is important in managing appropriately the postoperative symptoms. Assessment was hindered by incomplete fluid balance charts. Complete and accurate documentation in relation to recording of fluid input and output is crucial so that fluid status might be accurately determined. Although intravenous fluids are prescribed, the volume of oral fluids taken by patients often receives less attention. Patients should be better informed about the appropriate volume and type of fluids to drink.

CONCLUSION

Postoperative patients are at risk for overhydration and acute severe hyponatraemia when hypotonic fluid replacement by intravenous or oral routes is used. Meticulous fluid input and output should be recorded and patients should be advised not to overhydrate. Low urine output in the absence of clinical or biochemical dehydration may reflect a number of factors including ADH release postoperatively. If intravenous fluid replacement is required, isotonic fluid should be used. Symptoms that may herald acute severe hyponatraemia require prompt assessment and measurement of serum electrolytes. Acute severe hyponatraemia requires monitoring in an intensive care unit and treatment with hypertonic 3% sodium chloride to prevent life-threatening complications.

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Conflicts of Interest

None.

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