

How quickly can acute symptomatic hyponatremia be corrected?

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Abstract The systemic absorption of the flush liquid, including sorbitol, glycine or mannitol, can lead to complications, such as hyponatremia, volume overload and pulmonary or cerebral edema. Acute hyponatremia is defined as a reduction in the plasma sodium level in less than 48 h. Acute symptomatic hyponatremia should be corrected aggressively because it may cause irreversible neurological damage and death. Rapid correction of hyponatremia causes severe neurologic deficits, such as central pontine myelinolysis; thus, the optimal therapeutic approach has been debated. This article examined acute symptomatic hyponatremia in a patient undergoing transcervical myomectomy for a submucosal myoma. A thirty-seven-year-old patient was evaluated in obstetrics and gynecology clinic because of altered mental status and agitation. There was no history of chronic illness or drug use. It was discovered that during the operation, 12 L of the flush fluid, which contained 5 % mannitol, had been infused, but only 7 L of the flush fluid had been collected. On physical examination, the patient's general condition was moderate, her

cooperation was limited, she was agitated, and her blood pressure was 120/70 mmHg. The sodium level was 99 mEq/L. Furosemid and 3 % NaCl solution were given. Her serum sodium returned to normal by increasing 39 mEq/L within 14 h. Her recovery was uneventful, and she was discharged 24 h after her serum sodium returned to normal. In conclusion, if there is a difference between the infused and collected volumes of the mannitol irrigant, severe hyponatremia may develop due to the flush fluid used during transcervical hysteroscopy and myomectomy. In these patients, acute symptomatic hyponatremia may be corrected as rapidly as the sodium level dropped.

Introduction

Hyponatremia is generally defined as a serum sodium level less than 135 mEq/L, although the definition may vary slightly among laboratories [1]. This condition is observed in 15–30 % of hospitalized patients [2]. In severe hyponatremia (<115 mEq/L), the main symptoms include nausea, vomiting, headache, lethargy, agitation, fatigue, and disorientation [3]. In this situation, brain edema may develop due to the influx of water into the intracellular compartment. Neurological symptoms generally cannot be observed when the serum sodium level is more than 120 mEq/L [3].

Fluid flushes must be used to remove blood and cells during transurethral prostate resection or hysteroscopic transvaginal submucosal myomectomy. The

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systemic absorption of the flush liquid, including sorbitol, glycine or mannitol, can lead to complications, such as hyponatremia, volume overload and pulmonary or cerebral edema. Respiratory distress, cardiovascular collapse, coma and eventually death may subsequently occur. The management of acute and chronic hyponatremia is different and clinically very important because chronic hyponatremia, even for very low serum sodium levels, is well tolerated and rapid correction of this condition can lead to severe neurological sequelae. Acute hyponatremia is defined as a reduction in the plasma sodium level in less than 48 h [3]. Acute symptomatic hyponatremia should be corrected aggressively because it may cause irreversible neurological damage and death [4]. Rapid correction of hyponatremia causes severe neurologic deficits, such as central pontine myelinolysis; thus, the optimal therapeutic approach has been debated [5, 6].

This article examined acute symptomatic hyponatremia in a patient undergoing transcervical myomectomy for a submucosal myoma.

Case

A thirty-seven-year-old patient was evaluated in obstetrics and gynecology clinic because of agitation and altered mental status during the first postoperative hour after a vaginal myomectomy operation. The blood gas analysis showed the following: Na, 104 mEq/L; K, 3.8 mEq/L; pH, 7.32; PCO₂, 40.4; PO₂, 199; HCO₃, 21.3; and O₂ saturation, 96 %. There was no history of chronic illness or drug use. It was discovered that during the operation, 12 L of the flush fluid, which contained 5 % mannitol, had been infused, but only 7 L of the flush fluid had been collected. The difference between the infused and collected volumes of the mannitol irrigant was 5 L. On physical examination, the patient's general condition was moderate, her cooperation was limited, she was agitated, and her blood pressure was 120/70 mmHg. There were no pathological findings for heart or lung auscultation, and there was no pretibial edema. Biochemical results were not yet available, and iatrogenic acute hyponatremia was diagnosed based on the history, examination findings and the sodium level from the blood gas analysis. According to the results of the blood gas analysis, the target sodium level was determined to be 115 mEq/L, based on the

calculated sodium deficit. To increase the free water clearance, 10 mg of furosemide was given intravenously (IV), and 3 % NaCl solution at 30 mL per hour was given IV. The Na level was to be evaluated every 2 h. Initially, the following laboratory values were found: urea, 15 mg/dL; Cr, 0.5 mg/dL; sodium, 99 mEq/L; Cl, 71 mEq/L; K, 3.9 mEq/L; Ca, 7.2 mg/dL; and glucose, 142 mg/dL. The serum osmolarity was 280 mOsm, and the calculated serum osmolarity was 208 mOsm. Thus, the osmolal gap was 72 mOsm. The urine-specific gravity was determined to be 1015, but the spot urine sodium could not be detected. After 2 h, her sodium level was 112 mEq/L. The hypertonic saline solution was stopped because her sodium level had increased more rapidly than expected.

At this time, the patient was conscious, cooperative and not disoriented, but the patient had two episodes of projectile vomiting. A neurology consultation was obtained to evaluate for possible brain edema, but there were no localizing signs, and the cranial CT was normal. Her serum sodium was 123 mEq/L in the 4th hour, despite no further treatment for hyponatremia. Her serum sodium was 128 mEq/L, 133 mEq/L and 138 mEq/L at the subsequent 3-h intervals. Her serum sodium returned to normal by increasing 39 mEq/L within 14 h. Her recovery was uneventful, and she was discharged 24 h after her serum sodium returned to normal.

Discussion

Although hyponatremia is rare after transvaginal myomectomy, if there is a difference between the infused and collected volumes of the mannitol irrigant, the serum sodium level can significantly decrease in a short time (<110 mEq/L), and neurological symptoms may develop [7]. If sufficient consideration is not made for the treatment of hyponatremia, significant mortality and morbidity can occur in these patients.

The incidence of hyponatremia after hysteroscopy has been reported to be between 0.06 and 0.2 % [8]. The risk of developing hyponatremia increases as the gap between the amount of infused fluid and collected fluid increases [9]. Serum sodium levels generally decrease within 1–2 h after the operation [10]. In our case, symptoms of hyponatremia were observed 2 h after the operation. In these patients especially, close

monitoring of the fluid gap during the intraoperative period can help clinicians to more quickly detect and treat hyponatremia.

Based on our knowledge, the correction rate for acute symptomatic hyponatremia should be 0.5–1 mEq per hour and 12–15 mEq/L within 24 h [3]. However, the serum sodium can be raised by 8–10 mEq/L within 4–6 h in acute hyponatremia patients showing neurological symptoms [11]. In our case, acute symptomatic hyponatremia was diagnosed, and a target sodium level of 115 mEq/L was calculated based on the sodium deficit. She was treated with a 3 % NaCl infusion, and 10 mg of furosemide IV was administered to increase water clearance. In our patient, the serum sodium increased very rapidly, by 13 mEq/L within the first 2 h. Therefore, we stopped the hypertonic saline infusion and planned to spontaneously restore the electrolyte balance. As planned, her serum sodium level was 138 mEq/L after an increase of 39 mEq/L in 14 h (Fig. 1). The rapid correction of her hyponatremia did not cause any clinical problems.

Woo et al. [12] had presented a case where the patient's serum sodium level decreased to 87 mEq/L during the intraoperative period. Severe volume overload, non-cardiogenic pulmonary edema and cardiac instability developed in this patient. The patient was treated successfully with IV hypertonic saline solution and continuous venovenous hemofiltration without any sequelae. In our case, correction of the hyponatremia was considered to be a priority because there was no pulmonary edema or cardiac instability.

The prognosis is affected by either condition in patients with hyponatremia [13, 14]. First,

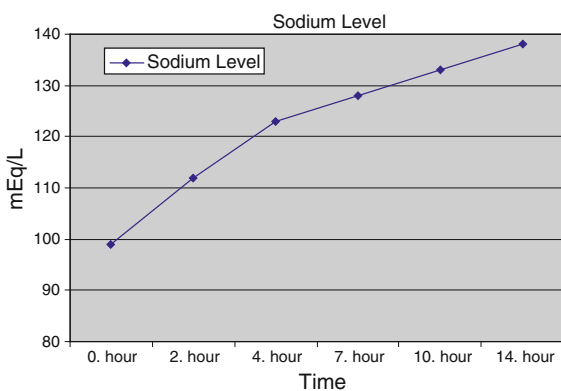


Fig. 1 Time course for the serum sodium level

hyponatremic encephalopathy is associated with brain edema and increased intracranial pressure. It is often observed in untreated or partially treated cases. Second, central pontine myelinolysis due to a rapid correction of hyponatremia is rare. It should be decided how to correct hyponatremia in a patient according to the presence or absence of neurological symptoms [14]. However, this approach may be risky due to the possibility of patients with unknown chronic severe hyponatremia [15]. It is thought that central pontine myelinolysis occurs with rapid correction of chronic hyponatremia that has developed over more than 48 h [13, 16]. Paradoxically, in situations of a rapid decline in serum osmolarity due to acute hyponatremia, the correction of hyponatremia may be useful to prevent cerebral edema and central pontine myelinolysis [12]. Hyponatremia may be an independent factor for demyelination damage [15].

Ayus et al. [17] have suggested that, to correct severe acute hyponatremia, hypertonic saline should be given rapidly with close monitoring until the serum sodium level is 121–134 mEq/L. However, they emphasized that the serum sodium level should not be increased to normal or hypernatremic levels or increased by more than 25 mEq/L within 48 h. However, it is difficult to control the correction rate, and it is often more rapid than expected. Indeed, in our case, the correction rate for hyponatremia was faster than expected, and the serum sodium spontaneously increased to 139 mEq/L within 14 h.

In conclusion, if there is a difference between the infused and collected volumes of the mannitol irrigant, severe hyponatremia may develop due to the flush fluid used during transcervical hysteroscopy and myomectomy. In these patients, acute symptomatic hyponatremia may be corrected as rapidly as the sodium level dropped.

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