Case Report

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Transient Oliguria during Anesthesia in Cerebral Salt Wasting Syndrome

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Cerebral salt wasting syndrome is a hyponatremic and hypovolemic condition caused by intracranial disorders, such as head injury, subarachnoid hemorrhage, brain tumor, and brain operations. We report a case of a 5-year-old girl that had cerebral salt wasting syndrome with marked polyuria who showed transient oliguria during general anesthesia. The patient had undergone an operation for traumatic intracranial hemorrhage three months prior and has had marked polyuria and hyponatremia since then. After induction of anesthesia for cranioplasty, the patient had oliguria during surgery and then resumed polyuria in the post-operative period.

Key Words: Anesthesia, Cerebral salt wasting, Oliguria, Polyuria

INTRODUCTION

Cerebral salt wasting syndrome (CSW) commonly occurs in patients with traumatic brain injury or brain tumors. It is characterized by low blood sodium concentrations and hypovolemia. Despite normal kidney function, hyponatremia occurs due to excessive sodium excretion. Hyponatremia is present in both syndromes with inappropriate secretion of antidiuretic hormone (SIADH) and CSW. While SIADH is a euvolemic state that requires strict volume restriction, CSW is a hypovolemic state that requires adequate fluid replacement and sodium chloride substitution. Therefore, it is crucial for clinical treatment to differentiate between SIADH and CSW. Cerebral salt wasting syndrome occurs about a week after brain injury and then spontaneously resolves within 2-4 weeks [1]. It is rare for patients with CSW to undergo an operation during this period. We report a case of a child with CSW who experienced marked hyponatremia and excessive urine output for three months. After undergoing general anesthesia, she showed severe intra-operative oliguria and then became polyuric during the post-operative period.

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CASE REPORT

A five-year-old female patient (111 cm, 17 kg) was scheduled for cranioplasty. Her medical history included surgical repair of a ventricular septal defect, attention deficit hyperactivity disorder, and language disorder. The patient was initially hospitalized in the emergency department after being referred from a local clinic after being diagnosed with traumatic intracranial hemorrhaging. Cardiopulmonary resuscitation was performed during the transfer. At the time of arrival, her vital signs were as follows: blood

pressure 120/74 mmHg, heart rate 112 beats/min, body temperature 39.8°C, and she was in a semicoma. She was immediately placed on ventilator support with a norepinephrine infusion as her blood pressure fell. Brain computed tomography showed traumatic intracranial hemorrhage of the basal ganglia, which prompted the decision to perform intracranial hematoma evacuation and decompressive hemicraniectomy. She was anesthetized with sevoflurane, cisatracurium and remifentanil. The total anesthesia time was three hours and thirty minutes. A total of 1,100 mL of crystalloid was infused during the operation, and total urinary output was 580 mL. After surgery, the patient was placed under coma therapy. Three days later, her mental state was a deep stupor. Norepinephrine was completely tapered a week later. The patient was weaned from the ventilator and extubated ten days later.

On day 8, laboratory testing revealed an increase in brain natriuretic peptide (BNP 1,411.19 pg/mL, <160). Electrocardiogram revealed normal sinus rhythm and echocardiography showed no evidence of cardiac dysfunction. The patient's blood pressure was within a normal range (110/50-160/85 mmHg). On day 10, there was a marked increase in urine output (3,000 mL/day) accompanied by hyponatremia. Her minimum sodium level was 116 mmol/L (138-145). Serum osmolaltiy was 286 mmol/kg (275-295), urine osmolality was 386 mmol/kg (50-1200), and urine sodium was 183 mmol/L. To maintain a serum sodium level above 130 mmol/L, 3% NaCl and 0.9% normal saline was infused at a rate of 20-50 mL/h, and fluid was replaced to compensate for volume loss. Her central venous pressure (CVP) was 4 cm H_2O.

Until the day of surgery, the patient's urine output was between 1,700-6,000 mL per day. An electrocardiogram and pulse oximetry monitoring were performed when the patient arrived in the operating room. Her blood pressure was 140/104 mmHg and her heart rate was 121 beats/min. Skin turgor was slightly decreased and her eyes were not sunken.

General anesthesia was induced with thiopental sodium and cisatracurium. Maintenance was achieved with sevoflurane (1.5-2.5%) and remifentanil infusion (0.1-1 μg/kg/min). After induction of general anesthesia, normal saline was infused at a rate of 300 mL/h. Blood pressure was maintained between 100/60-130/95 mmHg, and heart rate was sustained at 120-140 beats/min.

During the first hour of general anesthesia, the patient's urinary output was only 15 mL. Even after rapidly infusing 600 mL of normal saline an hour later, her urinary output was 10 mL. During this period, the patient's heart rate increased to 140-150 beats/min and her blood pressure dropped to 70/40 mmHg. Norepinephrine infusion was administered immediately. Three hours after general anesthesia, there were still only 15 mL of urine output, which increased to 30 mL after another hour. Body temperature was preserved between 36.6 and 36.8°C. Total anesthesia time was four hours and fifteen minutes. Total urinary output was 75 mL and total blood loss was 80 mL. A total of 1,500 mL normal saline and 100 mL packed red cells were infused intravenously. After the surgery, the patient was sent to the intensive care unit (ICU) without extubation.

After arriving in the ICU, normal saline was given at a rate of 300 mL/h. During the first hour the patient produced 90 mL of urine, which increased to 480 mL two hours later. The patient's heart rate was 115-145 beats/min and her blood pressure was 110/60-145/90 mmHg. The norepinephrine infusion was stopped three hours later and the patient was extubated the next day. The patients' urine output during the first 25 days after surgery was 2,000-4,000 mL/day, which later decreased to 1,000 mL/day.

DISCUSSION

CSW is a clinical condition that can involve weight loss, increased urinary volume, and excessive loss of urinary and serum sodium after intracranial hemorrhage or injury [2]. CSW usually spontaneously resolves after two to four weeks. Jimenez et al. [3] reported 14 cases of children with acute central nervous system injury that were accompanied by CSW. Among them, 11 patients experienced CSW 48 hours after admission, while 3 patients showed symptoms of CSW 11 days later. The mean duration was 6.3 ± 5.4 days and the range was between 1-19 days. It is uncommon for symptoms to persist for more than three months, as discussed in this case. CSW has several clinical features: (1) hypovolemia with net negative fluid balance; (2) hyponatremia and hypo-osmolality; (3) elevated urine osmolality (>100 mOsm/kg); and (4) elevated urinary sodium (>40

mEq/L) 4). However, there are no definitive diagnostic tests for CSW, and it is difficult to distinguish CSW from SIADH based on serum and urine lab findings [4].

The pathophysiology of CSW remains unclear. Since CSW is accompanied by primary natriuresis that causes hypovolemia and sodium depletion, it is believed that antidiuretic hormone (ADH) release, decreased aldosterone secretion, and BNP secretion play an important role [1,5]. Lu et al. [6] reported increased BNP levels in a patient with CSW and concluded that the level of BNP (a peptide with natriuretic, vasorelaxant, and aldosterone inhibiting properties) may be related to CSW. They also suggested that BNP plays a causative role in traumatic brain injury-induced CSW. Natriuresis and increased urine output were demonstrated when BNP was given intravenously. We suggest that elevated BNP level and hyponatremia were connected in this case where a 5-year-old child experienced CSW, and that the plasma concentration of BNP affects urinary sodium excretion.

Hypovolemic hyponatremia occurs when there is renal volume loss and hypotonic fluid replacement. When total body sodium and total body water decrease, the pituitary increases ADH secretion to maintain intravascular volume, causing free water retention from hypotonic fluid replacement. Hypo-osmolalitiy is used to maintain intravascular volume [1]. The extracellular volume state is the primary factor that distinguishes CSW from SIADH. It is difficult to precisely assess extracellular fluid volume and since CSW and SIADH can both be present in the same patient, it can be difficult to distinguish these conditions. Central diabetes insipidus (DI) also includes polyuria and must be differentiated from CSW. In central DI, the serum sodium level is greater than 145 mEq/L with polyuria. In many cases, CSW occurs after the onset of central DI [7].

Although there is no gold standard for assessing extracellular volume states, CVP or isotope-labeled albumin may be used as a diagnostic tool. Patients with a CVP below 5 cm H_2O are considered to have CSW because such a condition is uncommon in SIADH [8]. Depending on the patient's condition, CVP may not accurately reflect cardiac filling pressure. Along with laboratory findings, clinical signs such as decreased skin turgor, low blood pressure, elevated heart rate, and weight loss may help to assess the volume state of a patient with CSW [9]. In this case, CVP was not intra-operatively measured because the patient's central line was placed in the right femoral vein, which may cause an inaccurate value. In addition, the blood pressure and heart rate may not correlate with blood volume state. The patient's blood pressure and heart rate were stable before induction of anesthesia and we did not consider the patient to be in a severely hypovolemic state. However, since her body weight decreased 2 kg during admission, the possibility of a hypovolemic state should not be ruled out. Additionally, appropriate fluid replacement should have taken place before surgery. However, in this case, the patient's fluid levels were maintained based on urinary loss and changes in blood pressure. Thus, sufficient compensation to control hypovolemia might not have taken place.

We believe that the use of an inhaled agent during general anesthesia might have caused systemic vasodilation, further worsening the patient's hypovolemic state. This might have led to decreased prerenal blood flow and glomerular filtration rate, causing oliguria. Therefore, even if initial hemodynamic parameters were normal, the possibility of altered volume status must be considered. Thus, the volume state must be accurately assessed during the peri-operative period. In conclusion, hyponatremia frequently occurs in patients with brain injury. CSW is similar to SIADH in clinical symptoms and laboratory tests, so it is difficult to differentiate these two conditions. CSW is a hypovolemic state with opposing treatment protocols to SIADH. Therefore, before anesthetizing a patient with CSW, it is important to assess blood volume state and actively replace fluids to maintain stable conditions throughout surgery to reduce potential complications.

REFERENCES

1. Cho KC. Electrolyte & acid-base disorders. In: Papadakis MA, McPhee SJ, Rabow MW, editors. Current Medical Diagnosis & Treatment. (55th ed). McGraw-Hill Education. 2016. pp869-97.
2. Peters JP, Welt LG, Sims EA, Orloff J, Needham J. A salt-wasting syndrome associated with cerebral disease. *Trans Assoc Am Physicians* 1950;63:57-64.
3. Jimenez R, Casado-Flores J, Nieto M, Garcia-Teresa MA. Cerebral salt wasting syndrome in children with acute central nervous system injury. *Pediatr Neurol* 2006;35:

261-3.
4. Yee AH, Burns JD, Wijdicks EF. Cerebral salt wasting: pathophysiology, diagnosis, and treatment. *Neurosurg Clin N Am* 2010;21:339-52.
5. Berendes E, Walter M, Cullen P, Prien T, Van Aken H, Horsthemke J, Schulte M, von Wild K, Scherer R. Secretion of brain natriuretic peptide in patients with aneurysmal subarachnoid haemorrhage. *Lancet* 1997; 349:245-9.
6. Lu DC, Binder DK, Chien B, Maisel A, Manley GT. Cerebral salt wasting and elevated brain natriuretic peptide levels after traumatic brain injury: 2 case reports. *Surg Neurol* 2008;69:226-9.
7. Yang YH, Lin JJ, Hsia SH, Wu CT, Wang HS, Hung PC, Chou ML, Hsieh MY, Lin KL. Central diabetes insipidus in children with acute brain insult. *Pediatr Neurol* 2011;45:377-80.
8. Damaraju SC, Rajshekhar V, Chandy MJ. Validation study of a central venous pressure-based protocol for the management of neurosurgical patients with hyponatremia and natriuresis. *Neurosurgery* 1997;40:312-6; discussion 6-7.
9. Kumar A, Anel R, Bunnell E, Habet K, Zanotti S, Marshall S, Neumann A, Ali A, Cheang M, Kavinsky C, Parrillo JE. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med* 2004;32: 691-9.