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Unexpected Fatal Hypernatremia after Successful Cardiopulmonary Resuscitation with Therapeutic Hypothermia: A Case Report

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Central diabetes insipidus (DI), characterized by unexpected fatal hypernatremia, is a rare complication after successful cardiopulmonary resuscitation with therapeutic hypothermia, but may be potentially fatal if recognition is delayed. We describe here a patient who experienced cardiac arrest due to a pulmonary embolism, followed by successful resuscitation after induction of therapeutic hypothermia. The patient, however, suddenly developed unexpected hypernatremia with increased urine output and was diagnosed with central DI as a complication of cerebral edema, and eventually died. Our findings suggest that central DI should be considered as a possible complication following unexpected hypernatremia with increased urine output during therapeutic hypothermia and that desmopressin acetate should be used to treat central DI.

Key Words: Hypernatremia; Diabetes Insipidus; Hypoxia, Brain; Hypothermia, Induced

INTRODUCTION

The 2010 report of the American Heart Association highlighted post-cardiac arrest care as the fifth chain of survival in advanced cardiopulmonary life support (1). Several studies have shown that therapeutic hypothermia can benefit patients after cardiac arrest, and this therapy is now considered a level one recommendation for survivors of ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest (1). Less is known, however, about the complications of post-cardiac arrest care and therapeutic hypothermia and about methods to monitor and treat these conditions. Central diabetes insipidus (DI) arises due to a deficiency in antidiuretic hormone and is characterized by hypernatremia and polyuria with hyperosmolar dehydration (2). The most common causes of central DI are tumors and traumas in the neurohypophyseal area, although it may also occur after severe hypoxic/ischemic brain damage or at the terminal stage of a critical illness (3-5). Central DI during therapeutic hypothermia following successful cardiopulmonary resuscitation, however, is less known and uncommon. We describe here a patient who experienced central DI as a complication of therapeutic hypothermia following cardiac arrest.

CASE DESCRIPTION

On May 12th 2011, A 43-yr-old woman with no medical history except for an ovarian cystectomy 4 months earlier felt a sudden shortness of breath and, 20 min later, was found at home in cardiac arrest by her husband. Immediate life support was started by her husband. Paramedics arrived at the scene 13 min later and observed pulseless electrical activity (PEA). The patient was transferred to the emergency room of a secondary hospital within 10 min. Return of spontaneous circulation (ROSC) was noted 6 min later. To find the cause of arrest, echocardiography and chest CT were performed, and she was diagnosed with a pulmonary embolism. She was treated with 100 mg of tissue plasminogen activator and transferred to a tertiary medical center for post-cardiac arrest care.

On arrival at our emergency room, she was comatose (GCS = 1-1-T) with pinpoint pupils unresponsive to light, but her corneal reflex was intact. Her vital signs were blood pressure 122/77 mmHg, heart rate 117 beats/min, and temperature 36.2°C, and she was intubated with controlled mechanical ventilation. Initial laboratory results showed that her hemoglobin was 12.0 g/dL, her D-dimer was 170.9 μg/mL, and her electrolytes were sodium 139 mM/L, potassium 3.6 mM/L, and chloride 113 mM/L. She was sedated, and therapeutic hypothermia was initiated with 2 L cold saline infusion (4°C) in combination with an external cooling mattress. She reached the target temperature (33°C) 3 hr 30 min after ROSC. Therapeutic hypothermia was maintained for 24 hr, during which time active upper gastrointestinal bleeding occurred. She underwent an emergency gastroduo-

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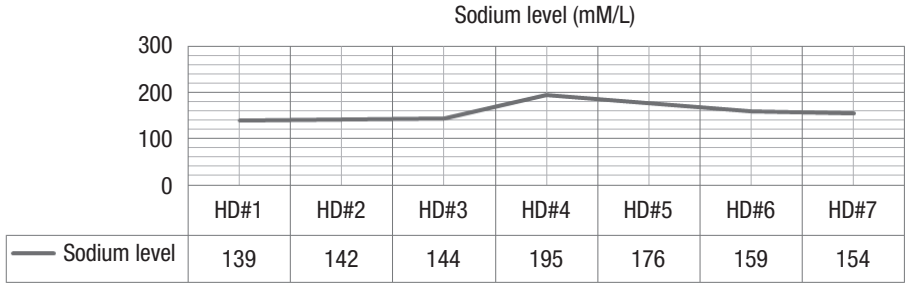

Fig. 1. Trends of sodium level (mM/L) after admission.

denoscopy, which showed hemorrhagic gastritis. She was treated with epinephrine spray and there were no additional bleedings. After 24 hr of therapeutic hypothermia, she was slowly rewarmed at a controlled rate of 0.3°C/h. On the third day, the patient developed polyuria (> 300 mL/h), with a total daily urine output of 8 L (input < 5 L). On the next day, her serum sodium concentration was 195 mM/L, 45 mM/L higher than on the previous day and her urine osmolarity was 142 mM/L. She was diagnosed with central DI. A brain CT scan with enhancement was performed to detect the cause of central DI, but there were no space-occupying lesions without severe cerebral edema, which was too severe to show her cerebral artery. The patient was treated with desmopressin acetate. During the 48 hr that followed, her urine output gradually decreased and her serum osmolarity, urine osmolarity, and serum sodium concentration returned to normal levels (Fig. 1). However, an electroencephalogram (EEG) showed severe cerebral dysfunction and a neurological examination showed clinical brain death. On the seventh day, brain death was confirmed by a committee consisting of a neurologist, a cardiologist, and a neurosurgeon and she died after multi-organ donation.

DISCUSSION

We have described a patient who experienced cardiac arrest due to a massive pulmonary embolism and underwent successful cardiopulmonary resuscitation and thrombolytic therapy, followed by successful induction of therapeutic hypothermia. Despite this appropriate post-cardiac arrest care, she developed sudden and unexpected hypernatremia with increased urine output and was diagnosed with central DI. She died after multi-organ donation.

During the last several years, therapeutic hypothermia has become an essential therapeutic modality, and may have significant neurological benefits for survivors of cardiac arrest. Nevertheless, therapeutic hypothermia has potentially serious side effects, including coagulopathy, increased risk of infection, reduction in insulin secretion, shivering, and electrolyte disturbance (6-10). Diuresis during hypothermia may result from decreased anti-diuretic hormone (ADH) and a reduction of renal medullary hypotonicity augmenting water loss, but this dysfunction is usually reversible (11). Therefore, hypernatremia has been considered a benign complication during therapeutic hypothermia. However, our experience with this patient shows that hypernatremia may be serious, and even fatal, in some patients. Despite general supportive treatment, her serum sodium concentration increased suddenly and unexpectedly, to 195 mM/L within 12 hr. Following a diagnosis of central DI, she was treated with desmopressin acetate.

The development of central DI during therapeutic hypothermia following successful cardiopulmonary resuscitation may be due to severe hypoxic brain damage. Despite decreases in cerebral circulation and hypoxia, supraoptic and paraventricular nuclei are not easily destroyed, because of the many neurosecretory cells and collateral circulation in the neurohypophyseal system (12). Thus, central DI can occur when up to 90% of the hypothalamic neurons are destroyed. In an experiment of dogs and monkeys, central DI occurred when almost 90% of the neurosecretory cells had been lost (13). Moreover, central DI has been reported to occur in 11%-87% of patients with irreversible brain damage during donor management in the intensive care unit (5, 7, 10, 11, 14). and a recent retrospective study on complications of cardiac arrest showed that central DI developed in up to 78.4% of patients scheduled for organ donation in a state of hypoxemic encephalopathy (15). To our knowledge, however, there have been no reports of patients with central DI as a complication of appropriate therapeutic hypothermia. Our findings suggest that unexpected hypernatremia is not merely a benign electrolyte disturbance but may be fatal, and highlight the importance of monitoring for central DI, one of the major complications of post-cardiac arrest care. Early diagnosis of central DI and treatment with desmopressin acetate may reduce secondary brain injury caused by hyperosmolar hypernatremia and resultant mortality and may result in better neurologic outcomes.

In conclusion, therapeutic hypothermia is an essential modality in post-cardiac arrest care, with hypernatremia considered a benign complication of therapeutic hypothermia. We describe a patient who presented with unexpected hypernatremia with increased urine output during therapeutic hypother-

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mia, was diagnosed with central DI and eventually died. Due to the increased use of therapeutic hypothermia, patients who develop unexpected hypernatremia with increased urine output should be considered central DI as a possible complication.

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