Rhabdomyolysis and Disseminated Intravascular Coagulation Associated with Severe Exercise-induced Hypernatremic Dehydration

Sik Lee, M.D., Won Kim, M.D., Sung Kwang Park, M.D
Eun Seok Kang, M.D.*and Sung-Kyew Kang, M.D

Department of Internal Medicine, Chonbuk National University Medical School, Jeonju,
Yonsei University College of Medicine*, Seoul, Korea

The most common causes of clinically significant hypernatremia occur as a consequence of three pathologic mechanisms: impaired thirst, solute or osmotic diuresis, and excessive losses of water (either through the kidneys or extrarenally), or in combination of these derangements. Any condition that increases insensible fluid loss such as severe exercise, exposure to high temperatures, high fever, respiratory infections, or burns predisposes toward the development of severe hypernatremia. Severe hypernatremic dehydration may be associated with coagulopathy and rhabdomyolysis. We report that a case of severe exercise-induced hypernatremic dehydration, disseminated intravascular coagulation, and rhabdomyolysis in a 15-year-old boy associated with weight loss.

Key Words : Hypernatremia, Dehydration, Severe exercise

Introduction

Hypernatremia is defined as an increase in Na^+ concentration in plasma water. Although the upper limit for normal serum Na^+ is 150 mEq/L, most subjects with a serum Na^+ in the high normal range experience intense thirst, which is an important physiological response in defending against the development of hypernatremia[1]. Hypernatremia rarely develops with an increase in water loss alone; there must be a mechanism that interferes with the water intake. Insensible fluid losses from the skin and respiratory tract are hypoosmotic in respect to plasma and average from 800 to 1,000 mL/day in adults. Any condition that increases these losses such as severe exercise, exposure to high temperatures, high fever, respiratory infections, or burns predisposes toward the development of hypernatremia. The normal defense against the development of hypernatremia is the stimulation of both the antidiuretic hormone release and thirst by activation of hypothalamic osmoreceptors. The combination of decreased water excretion and increased water intake results in water retention and restoration of the plasma sodium concentration into normal. There have been several reports that severe hypernatremic dehydration is associated with coagulopathy and rhabdomyolysis[2-5].

A case of severe exercise-induced hypernatremic dehydration, disseminated intravascular coagulation, and rhabdomyolysis in a 15-year-old boy associated with weight loss is presented.

Case report

A 15-year-old boy was admitted to the ER with high fever and generalized convulsion.

His mentality was a comatose state. He was a

Correspondence : Sung Kyew Kang, M.D.
Tel : 82-63-250-1677 Fax : 82-63-274-0781
E-mail : hope@chonbuk.ac.kr

wrestler. During several days before the admission, he had been exercising hard to take off weight and his weight had decreased from 55 kg to 46 kg. Initially, he had visited the local hospital for falling down during running. He was managed as a patient with a heat stroke. At that time, he was drowsy with complaining of high fever and myalgia. He developed a deterioration of the level of consciousness. Therefore, he was referred to our hospital twelve hours after the visit to the local hospital. On admission, he was unresponsive to verbal and deep painful stimuli. The corneal reflex and oculocephalic motion were normal. In addition, Babinski's reflexs were absent bilaterally. His vital signs were:blood pressure 90/60 mmHg, temperature 40℃, pulse 110 bpm, and respiratory rate 22/min. Laboratory findings on admission were as follows: hemoglobin 13.1 g/dL, hematocrit 41%, white blood cell 10.9×10^9/L, platelet 148×10^9/L, BUN 42 mg/dL, creatinine 3.5 mg/dL, Na^+ 161 mmol/l, K^+ 4.6 mmol/L, ionized calcium 0.76 mmol/L, aspartate transaminase (AST) 543 U/L, alanine transaminase (ALT), 218 U/L, amylase 1,074 U/L, lipase 327 U/L, serum myoglobin over 500 ng/mL (normal 12-76 ng/mL), CK over 1252 U/L (normal 32-187 U/L), PT 40.6 sec (normal 11.7-13.7 sec), aPTT over 120 sec (normal 29.8-41.8 sec).

The urinalysis showed pH 5.0, specific gravity 1.030, protein +3, with 10-15 RBC/HPF, 1-4 WBC/HPF. ABGA : pH 7.29, PCO_2 30.2 mmHg, PO_2 67 mmHg, bicarbonate 14.3 mmol/L, O2 saturation 96%. The patient was moved to the intensive care unit for cold compression because of high fever. The estimated water deficit was about 3.6 liters. One point eight liters of fluid replacement was begun. It was supplied during the first 12 hrs through both parenteral and oral route as recommended by Adrogne and Madias[6].

After five hours, the patient had several episodes of hematemesis and massive hematochezia. Laboratory examinations showed the following values, the day after the blood loss:hemoglobin 3.8 g/dL, white blood cells 2.4×10^9/L, platelet 27×10^9/L, BUN 51 mg/dL, creatinine 4 mg/dL, Na^+ 149 mmol/L, K^+ 5.4 mmol/L, ionized calcium 0.66 mmol/L, AST 789 U/L, ALT 442 U/L, serum myoglobin over 500 ng/mL (normal 12-76 ng/mL), CK over 3,420 U/L (normal 32-187 U/L), ABGA under artificial ventilator (FiO_2, 0.6):pH 7.41, PCO_2 16.7 mmHg, PO_2 130 mmHg, bicarbonate 9.1 mmol/L, O2 saturation 98.7%. PT 40 sec (normal 11.7-13.7 sec), aPTT over 120 sec (normal 29.8-41.8 sec), fibrinogen 263 mg/dL (normal 200-400 mg/dL), FDP 10 μg/mL (normal below 5 μg/mL). The patient's general condition deteriorated rapidly. Therefore, blood transfusion and conservative treatments were intensively attempted but without effect.

Twenty-four hours after the admission, the patient's state deteriorated, and the systolic blood pressure dropped down below 60 mmHg, in spite of an infusion of inotropic agent.

He was managed out from circulatory collapse.

Discussion

The most common causes of clinically significant hypernatremia occur as a consequence of three pathologic mechanisms:impaired thirst, solute or osmotic diuresis, and excessive losses of water (either through the kidneys or extrarenally), or in combination of these derangements[7]. Insensible water losses are approximately 1 L/day in the average adult. These losses are not subject to the osmotic regulation but may be increased by a wide variety of factors, including fever, exercise, increased ambient temperature, and hyperventilation. Patients with increased insensible losses manifest oliguria with maximally concentrated urine. In contrast, patients with diabete insipidus have polyuria less than maximally concentrated urine. In our case, urine specific gravity was 1.030.

Severe hypernatremic dehydration may be complicated by acute renal failure, disseminated intravascular coagulation, circulatory collapse, rhadomyolysis,

and a wide array of signs and symptoms of central nervous system dysfunction, including delirium, confusion, and seizures. Abramovici et al. suggests that hypernatremia is a direct cause and affects rhabdomyolysis[3]. Mongalgi et al. also report that acute hypernatremic dehydration is associated with consumption coagulopathy and peripheral gangrene[2].

In our case, abnormal FDP, prolonged PT/aPTT, and thrombocytopenia revealed DIC.

Rhabdomyolysis was diagnosed by symptoms (such as muscle pain and weakness), elevated muscle enzymes, acute renal failure, electolyte abnormalities (such as hyperkalemia, hypocalcemia, hyperuricemia and metabolic acidosis). Severe hypernatremia has the mortality rate approaching 60% in adults and almost certainly contributes to the morbidity and mortality in respect of the underlying disease[8]. We thought the rate of correction of hypernatremia was adequate in the present case.

However, the bad results of the patient were due to the bleeding tendency and acute renal failure by DIC and rhabdomyolysis.

In summary, we present here a case of acute renal failure, rhabdomyolysis, and disseminated intravascular coagulation associated with severe exercise-induced hypernatremic dehydration for reducing body weight. This case shows a possibility that hypernatremic dehydration may be associated with coagulopathy and rhabdomyolysis.

References

1) Kang SK, Kim W, Oh MS : Pathogenesis and treatment of hypernatremia. *Nephron* **92**(Suppl 1):14-17, 2002
2) Bouguerra L, Bibi S, Mongalgi M : Peripheral gangrene associated with consumption coagulopathy in acute hypernatremic dehydration. *Arch Fr Pediatr* **44**:65, 1987
3) Abramovici MI, Singhal PC, Trachtman H : Hypernatremia and rhabdomyolysis. *J Med* **23**:17-28, 1992
4) Mongalgi M, Cheour M, Debbabi A:Periostal effusion in hypernatremic dehydration with disseminated intravascular coagulation. *Arch Pediatr* **1**(7):655-658, 1994
5) Comay SC, Karabus CD:Peripheral gangrene in hypernatremic dehydration of infancy. *Arch Dis Child* **50**:616-619, 1975
6) Adrogne HJ, Madias NE:Hypernatremia. *N Engl J Med* **342**:1493-1499, 2000
7) Kokko JP:Fluids and electrolytes. In:Goldman L and Bennett JC, Editors, Cecil textbook of medicine (21st ed), WB Saunders, Philadelphia (2000), p540-558
8) Arieff AI : Central nervous system manifestations of disordered sodium metabolism. *Clin Endocrinol Metab* **13**:269-294, 1984