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CASE REPORT

Unusual complication in a child with lightning strike: cerebral salt wasting

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Abstract Several case reports have presented various neurological complications caused by lightning. However, there was no report related to cerebral salt wasting caused by lightning injury. We described a patient with lightning strike, who was subsequently diagnosed with cerebral salt wasting.

Keywords Lightning · Cerebral salt wasting · Hyponatremia · Rhabdomyolysis

Introduction

Lightning injury is one of the most frequent injuries by natural phenomenon. Although lightning may involve all organ systems, injuries to the cardiovascular system and central nervous system are the most common ones. Central nervous system injuries caused by a direct lightning strike to the head are rare but exceedingly harmful and often result in a high mortality. Electrical current passing through the brain can lead to coagulation of brain tissue, intracranial hemorrhage, and damage to the medullary respiratory center [1].

Several case reports have presented various neurological complications caused by lightning. On the other hand, there was no report related to cerebral salt wasting caused by lightning injury. We described a patient with lightning strike, who was subsequently diagnosed with cerebral salt wasting.

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Case report

The case is of a 9-year-old girl who was thrown to the ground by a lightning shock wave. The patient lost her consciousness for a couple of hours and was confused afterwards. She was brought to the hospital about 3 h after strike from different city, and Glasgow coma scale score was recorded as 12. On arrival, her size and vital signs were normal. Cardiac, pulmonary, ophthalmological, and otoscopic examinations were within normal limits. There were no burn injuries. She did not have a history of drug use. Medical and family history was also unremarkable.

The initial cranial CT scan showed no edema, hemorrhage, or skull fractures in emergency department. Abdominal CT, electrocardiography, and echocardiography were normal. Laboratory findings on admission were shown Table 1. Serum electrolytes, including sodium, potassium, and calcium, were normal. Urine analysis was unremarkable.

According to the patient's history and biochemical findings, a diagnosis of acute rhabdomyolysis due to a lightning strike was made. No signs of acute renal failure were seen. Isotonic saline solution and 5% glucose fluid daily for preserving urinary output (urine output >1.5 ml/kg/h) were administered to the patient. One day after admission, her consciousness was opened, and physical examination was normal. Although WBC and serum AST and ALT levels returned to within the normal ranges, serum CK levels were still high. Decline in the serum sodium level to 125 mEq/L was noted and urinary density was 1,028. The urinary sodium

Table 1 Laboratory findings on admission

	Patient's value	Normal range
White blood cell	32,900/mm^3	4–10,000/mm^3
ALT	91 lU/L	6–37 lU/L
AST	106 lU/L	6–40 lU/L
Creatine kinase (CK)	445 U/L	26–192 U/L
Creatinine-kinase muscle band	85 U/L	7–25 U/L
Myoglobin	326 μg/L	10–46 μg/L
Troponin I ultra	0.04 μg/L	<0.01 μg/L

level was 293 mmol/L, the urinary osmolality was 840 mOsm/kg H_2O. Pseudo-hyponatremia was excluded since he had normal serum glucose, protein and lipid levels, and low serum osmolality. Repeated MRI of brain was normal. Daily water intake was restricted to 60% of maintenance fluids. On the following day, circulating sodium level was still 121 mEq/L. Daily water intake was 950 ml, and urine output was 1,250 ml. Daily sodium excretion was 630 mmol/L. A diagnosis of hypovolemic hyponatremia was made and saline infusion started. Her serum Na level was still low (125 mEq/L). Serum uric acid level was found to be low (1.7 mg/dL) and plasma vasopressin level was less than 0.5 pmol/L. She had polyuria and body weight continuously decreased. These data prompted the diagnosis of cerebral salt wasting (CSW). Total fluid intake was higher than the combined renal and insensible losses. A careful evaluation of her Na intake was revealed. The rate of hypertonic saline infusion that is appropriate for raising the serum Na to a desired level was estimated from the following formula: the required amount of Na to be infused = the desired increase in serum Na×ECF volume (the body weight×0.6). One week after admission, a renal salt wasting was continuing although physical examination and biochemical tests were normal. Thyroid function tests and serum cortisol level were also normal. On the second week of admission, urine output, and urinary Na level gradually decreased. Because fludrocortisone is not present in Turkey, oral NaCl was started in dose 2 g/day and intravenous fluid treatment was stopped. Serum Na levels were within normal range with this oral NaCl dose. CSW resolved spontaneously over the ensuing 6 months. Repeat investigations after discontinuation of oral Na were normal. She was healthy.

Discussion

Effects of lightning injuries range from minor to life threatening. Only about 20% of people are killed, the remaining 80% having various degrees of disability. The major fatal causes are primary cardiac arrest and hypoxia-induced secondary cardiac arrest. But for survivors, the most complications are neurologic. Manifestations in survivor of lightning include changed consciousness, disorientation, impaired vision, ruptured ear drums, hearing loss, seizures, paralysis, burns on skin, internal burns to organs, and cardiac arrest [1–3].

If victims survive, they may be afflicted with various complications, such as myocardial dysfunction including arrhythmia, muscle necrosis resulting in acute renal failure, neurological pathologies including loss of consciousness, transient paraplegia and aphasia, and tympanic rupture [1, 4]. A lightning injury is included in the category of a electrical injury and high-voltage electric charge may pass through the body for a split second. Up to 10% of patients with lightning-related cardiac arrest have no signs of electrical burns [1, 5]. Generally, it is challenging to evaluate the significance and severity of damage to deep organs and tissue that occurs from a lightning strike. Biochemical data, including serum creatine kinase, are usually used as a clinical markers for deep organ and tissue damage, but they never reflect the actual site and extent of the damage [6]. The serum CK level is commonly used as a biochemical marker of muscle damage. The major complications of rhabdomyolysis are hyperkalemia and acute renal failure related to a release of massive amounts of potassium and myoglobin. Our case had rhabdomyolysis but acute renal failure did not occur. The incidence of acute renal failure in rhabdomyolysis ranges from 10% to 30% [6].

How does lightning damage tissues and organs? The exact mechanism of tissue damage is unknown. Mechanisms include thermal effects, electrical effects, induced electrical currents, blast effects, and injuries related to falls. Magnetic field changes associated with lightning may induce a loop current within the human torso. Lightning's electrical current is enormous, even though of brief duration. Electric fields can damage the structural integrity of membranes of nerve and muscle tissues. The defect to the lipid bilayer membrane caused by these electric fields is termed electroporation [1, 5].

Our patient had increased CK, CK-MB, troponin l, and cerebral salt wasting. Brain-type (B-type) natriuretic peptide (BNP) is a neurohormone with diuretic, vasodilatory, a drenin–angiotensin–aldosteron antagonist effects. BNP might be of cardiac and brain origin. It is secreted primarily by cells in the ventricular wall in response to increases in wall stress [7]. Several observations have suggested that elevated levels of BNP may be markers of the extent and severity of ischemia [8]. Traditional biomarkers to evaluate myocardial infarction, such as CK-MB and troponin, are released when there is irreversible injury to cardiac myocytes. In contrast, BNP is released by intact cells. Transient myocardial ischemia was found to be associated

with and immediate rise in circulating BNP levels in one study [8]. For this reason, we think our patient had transient myocardial ischemia related to lightning and ischemia may lead to altered regional myocardial stretch, causing active secretion of BNP. On the other hand, the cause of secretion of BNP might result from the ischemia itself or to left ventricular dysfunction caused by ischemia. Also, early fluid administration is essential and the volume administered is generally much greater than the urinary output in rhabdomyolysis. Our patient was treated for rhabdomyolysis. Volume load might increase in ventricular wall tension. We must be aware of electrolyte disturbance in patient with rhabdomyolysis.

To our knowledge, there was no article about CSW after a lightning strike. Kotagel et al. [2] reported one patient with inappropriate secretion of antidiuretic hormone (SIADH) 4 days after lightning. They suggested that it is relatively late onset complication, and close follow-up of the fluid and electrolyte status for up to a week is important [2]. In our case, CSW also occurred within first week. The critical difference between CSW and SIADH is that CSW involves marked renal salt and water loss, resulting in hyponatremia and decrease of extracellular fluid volume, whereas SIADH results from inappropriate secretion of the antidiuretic hormone, leading to the renal conservation of water and euvolemic or hypovolemic hyponatremia [9].

In summary, lightning injury may also result in the production of inappropriate secretion of BNP. Close follow-up of the fluid and electrolyte status are mandatory. This disturbance may onset late and complete recovery may take as long as 6 months.

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