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Case Report

Acute transient hemiparesis induced by lightning strike

Abstract

According to data from the National Oceanic and Atmospheric Administration, in the years from 1959 to 1994, lightning was responsible for more than 3000 deaths and nearly 10,000 casualties. The most important characteristic features of lightning injuries are multisystem involvement and widely variable severity. Lightning strikes are primarily a neurologic injury that affects all 3 components of the nervous system: central, autonomic, and peripheral. Neurologic complications of lightning strikes vary from transient benign symptoms to permanent disability. Many patients experience a temporary paralysis called keraunoparalysis. Here we reported a 22-year-old mountaineer man with complaining of left sided hemiparesis after being hit by a lightning strike in the mountain 3 hours ago. There was no loss of consciousness at hitting time. On arrival the patient was alert, awake and hemodynamically stable. In neurologic examination cranial nerves were intact, left sided upper and lower extremity muscle force was I/V with a combination of complete sensory loss, and right-sided muscle force and sensory examination were normal. There is not any evidence of significant vascular impairment in the affected extremities. Brain MRI and CT scan and cervical MRI were normal. During 2 days of admission, with intravenous hydration, heparin 5000 unit SC q12hr and physical therapy of the affected limbs, motor and sensory function improved and was normal except mild paresthesia. He was discharged 1 day later for outpatient follow up while vitamin B_1 100 mg orally was prescribed. Paresthesia improved after 3 days without further sequels.

Until the last few years, lightning injuries have been the second most common cause of storm-related death in the United States [1]. According to data from the National Oceanic and Atmospheric Administration (NOAA), in the years from 1959 to 1994, lightning was responsible for more than 3000 deaths and nearly 10,000 casualties [2]. Although most injuries occur outdoors, a few people are injured indoors every year, including individuals who incur landline telephone-mediated strikes [3, 4]. The most important characteristic features of lightning injuries are multisystem involvement and widely variable severity [5].

Less than one third of affected persons have signs of burns. When burns do occur, they are usually superficial. Internal burns are rare. Myoglobinuria is rarely encountered in lightning injuries, whereas cardiac and respiratory arrest, vascular spasm, neurologic damage, and autonomic instability play a greater role. Blunt force injuries from falling, being thrown by muscle contractions, or barotrauma from the explosive force of a nearby lightning strike may occur [6]. Major complications are rare in mild and moderate lightning injuries, although musculoskeletal discomfort and subjective sensations of parenthesis, irritability, and other nonspecific neurologic sequel may be present, depending on the location and intensity of the strike. In severe lightning injury with cardiopulmonary resuscitation (CPR) required in the field, permanent neurologic deficit and hypoxic injury are common [7–9].

Most people struck by lightning survive. Approximately 10% of lightning strike cases are fatal. The fatalities are largely the result of cardiac arrest. Neurologic complications of lightning strikes vary from transient benign symptoms to permanent disability. These complications can be classified as falling into 1 of the following 4 categories:

1. Immediate and transient. These symptoms are often dramatic but the most benign because they are fleeting. The symptoms include brief loss of consciousness, amnesia, confusion, headache, paresthesia, and weakness. Nearly 75% of patients have a brief loss of consciousness and 80% have brief limb weakness and paresthesia. Many patients experience a temporary paralysis called keraunoparalysis. The paralysis is accompanied by sensory loss, pallor, vasoconstriction, and hypertension. It affects the lower limbs more than the upper limbs and is specific to lightning victims.
2. Immediate and prolonged or permanent.
3. Delayed and progressive complications.
4. Lightning-linked secondary trauma from falls or blast [10].

Immediate 'lightning paralysis' is characterized by complete tetraplegia and loss of sensory awareness of the trunk and all four limbs may be considered virtually pathognomonic [11]. Sometimes only the lower limbs, or an arm and a leg are affected, depending on where the lightning had struck [12]. Very often the sensory and motor paralysis is accompanied by marked autonomic and vasomotor disturbances [13–15]. As described, the affected limbs become pale and cold or cyanotic. The arterial pulses may transiently disappear. The affected limb can rarely become edematous. In typical uncomplicated cases of 'lightning paralysis', sensation and motor function usually return after a few minutes and at the latest up to a few days, accompanied by paresthesia [12].

Case presentation

A 22-year-old previously physically healthy mountaineer man was admitted to the emergency department (bodyweight, 75 kg) complaining of left sided hemiparesis after being hit by a lightning strike in mountain 3 hours ago. There was no loss of consciousness at hitting time. On arrival, he was hemodynamically stable. Oral temperature was 37.5 °C (99.5 °F), pulse rate was 84 beats per minute with normal rhythm, blood pressure was 132/82 mm Hg, respiratory rate was 15 breaths per minute without abnormal breath sounds and initial oxygen saturation was 95% (in room air). In physical examination, the abdomen was soft and flat with normal bowel sounds and there was no evidence of skin burning. He was alert, was awake with no apparent distress and speaks normally. He remembered events exactly. He did not mention any trauma and in physical examination, there is not any evidence of it. In neurologic examination, cranial nerves were intact, left

sided upper and lower extremity muscle force was I/V with combination of complete sensory loss, and right-sided muscle force and sensory examination were normal. Muscle tone had been decreased in the affected side. Deep tendon reflexes in the left and right sides were +1 and +2. The plantar reflex was flexor in both sides. He did not complain of any pain and there was not any evidence of significant vascular impairment in the affected extremities. A 12-lead electrocardiogram revealed normal sinus rhythm at a rate of 85 bpm; lab data in admission time were as follows: a complete blood count in patient was normal except for a mildly elevated white blood cell count of $8.5 \times 103/\mu L$, with 65.8% neutrophils, 24.4% lymphocytes, 9.8% mixed cell count, hemoglobin 13.9 g/dl, and Hct 41.7%. His serum blood urea nitrogen, creatinine, creatinine kinase, and liver function tests were normal (sodium 142 mEq/L, potassium 3.8 mEq/L, calcium (total) 9.3 mg/dL, magnesium 1.9 mg/dL). Coagulation studies, including a prothrombin time and activated partial thromboplastin time, were normal. Serum glucose was 120 mg/dL. Urine analysis was normal. Arterial blood gases were as follows: pH = 7.46, $PCO_2 = 34$, and $HCO_3 = 23.7$. Brain MRI and CT scan and cervical MRI were normal. We admitted him to the neurology ward. During 2 days of admission, with intravenous hydration (2 liters normal saline 0.9% per day), heparin 5000 unit SC q12hr and physical therapy of affected limbs, motor and sensory function improved and was normal except mild paresthesia. He was discharged 1 day later for outpatient follow-up while vitamin B_1 100 mg orally was prescribed. Paresthesia improved after 3 days without further sequels.

Discussion

The type of damage that lightning inflicts, falls into two categories: trauma due to the high temperature of the strike and injury caused by the electromechanical forces elicited by lightning [16]. Unlike electricity, which is an alternating current (AC), lightning is a direct current (DC) [17]. Resistance of the body is critical to the tissue insult experienced by a strike victim. Lightning, like electricity, follows the path of least resistance [18]. The most resistant tissues in the human body are (in descending order) bone, fat, tendon, skin, muscle, blood vessels, and nerves, with the last being the most susceptible to injury and trauma [17–19].

Autonomic nervous system (ANS) dysfunction is a serious complication of lightning and electrical trauma. When ANS is compromised after lightning and electric trauma, patients are vulnerable to serious medical problems. Three conditions of ANS dysfunction are complex regional pain syndrome (CRPS), cardiovascular abnormalities, and keraunoparalysis (KP). The patient with CRPS presents with pain, hyperpathia, sweating, and edema hours to days after trauma. Cardiovascular abnormalities associated with lightning and electrical trauma can be life threatening. Keraunoparalysis is a frightening and distressing complication of lightning strikes. The syndrome consists of limb paralysis, sensory symptoms, pallor, coolness and pulselessness. Release of excessive catecholamines is said to be responsible for these findings. Fortunately, the condition is transient [20].

Keraunoparalysis is a transient paralysis after lightning strike that is postulated to result from an overstimulation of the autonomic nervous system leading to vascular spasm. Typically, lower limbs are affected more than upper limbs. Signs and symptoms include pulselessness, pallor or cyanosis and motor and sensory loss in the affected extremities. Keraunoparalysis typically resolves within several hours. As keraunoparalysis may mimic a pulseless victim, responders must be vigilant about checking for a central pulse before starting cardiopulmonary resuscitation. We recommend hospital observation for keraunoparalysis. This phenomenon typically resolves spontaneously but may indicate more serious underlying trauma. Keraunoparalysis can mimic a spinal injury, thus spinal precautions should be maintained and diagnostic imaging should be performed to rule out spinal cord pathology if neurologic deficits persist despite resolution of pallor or pulselessness. Lightning victims frequently present with keraunoparalysis in which the extremities, usually the legs, become transiently cold, cyanotic, pulseless, and mottled. Keraunoparalysis is the result of vascular spasm and autonomic nervous system instability. It usually resolves after a few hours, though some patients have long-term neurologic sequelae, including paralysis and paresis [21].

Keraunoparalysis is a temporary paralysis of one or more extremities following a lightning strike. The clinical symptoms of keraunoparalysis include flaccid paralysis of the extremities involved, mostly the legs, in combination with complete or partial sensory loss. In many cases there are signs of disturbed peripheral circulation: loss of pulse, extreme coolness and pale or livid blue discoloration of the skin. As a rule paralysis, sensory loss and circulation disturbance disappear completely within 12–24 h and in the majority of cases even within1 h. Many authors describe this phenomenon by ascribing to a motor, sensory and vasomotor peripheral nerve reaction to the high current flow through the affected limb. Some authors believed the flaccid paralysis and sensory disturbance to be secondary to impaired peripheral circulation, because return of limb function seemed to be directly related to restoration of the peripheral limb circulation. However it is also possible that the activating mechanism works in just the opposite manner because in some cases paralysis has been observed without the complete loss of arterial circulation which is needed to provoke the actual neurological disturbance [22].

Cherington et al. [23] in 1995 reported 13 patients with neurologic complications following lightning injuries over the past ten years. Patients represent a wide spectrum of neurologic problems. Two new cases were reported and the remaining 11 have been reported previously. New case 1 was a 57-year-old man who was struck by lightning. He was diagnosed as having severe hypoxic brain injury due to cardiac arrest. A CT scan of the brain showed findings consistent with hypoxic encephalopathy. The patient died three days after admission. Case 2 was a 30-year-old man with isolated right facial nerve palsy. At follow-up clinical examination three weeks later, the condition of the patient was improved with only mild residual facial weakness. The patients' neurologic complications included the following: patients with hypoxic encephalopathy, 4 cases; spinal cord lesion, 3 cases; cerebellar lesion, 3 cases; concussion, 2 cases; and cranial nerve VII injury, 1 case. The surviving ten patients were divided into three groups based on the level of their neurologic lesions (lesions above the foramen magnum). All of these patients suffered from an initial major insult. All of them eventually recovered, however.

Ashish Kumar et al. [24] in 2012 reported a 50-year-old man with history of lightning strike when he was standing under a tree in the course of a lightning storm. He was unconscious for 15 min, and after he regained consciousness, he was unable to move both his lower limbs with loss of sensations and urinary retention. On examination, tone in both lower limbs was normal. He had grade 0 powers in both lower limbs, with a sensory level up to D12. Reflexes were depressed and plantars were equivocal. On the 2nd day, his power improved to grade III bilaterally which improved to grade IV the very next day so that he was ambulant by now. Brain MRI was normal, while spine imaging showed the evidence of ruptured posterior longitudinal ligament with evidence of hemorrhage. This could also have been a result of trauma. The patient had a group I injury where the imageology showed nothing substantial correlating to the pattern of injury, and hence diagnosis of keraunoparalysis was made. In this patient, the presence of hemorrhage substantiates their belief, but reversal of the neurological deficits in due course of time proved otherwise. They explained that as secondary trauma cannot be ruled out completely, it is always a diagnosis of exclusion. However, if we, as a community, are aware of this entity, we can save our energy and may try expectant management in cases where clear and definitive history of lightning strike is there. If patients recover within hours and no bony fractures are seen on X-rays, MRI of the spine may not be required in the emergent setting. They conclude that any lightning injury victim presenting with paraparesis can

have etiology of varied types. After a primary survey is over, close observation may be instituted till the patient shows some improvement in the paraparesis and if no other obvious bony injuries are suspected.

In contrast to other case reports and texts this patient had no evidence of vascular disturbance on affected limbs or concomitant acidosis; neurological impairment was unilateral and did not resolve in first hours. There is some controversy about keraunoparalysis treatment; administration of heparin and intravenous hydration may be rational in the case of concomitant vascular impairment but not in all cases.

Conclusion

With regard to this case and some other case reports with no complete loss of arterial circulation we do not believe impaired peripheral circulation as a sole cause of keraunoparalysis.

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