Lightning strike-induced brachial plexopathy

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ABSTRACT

We describe a patient who presented with a history of lightning strike injury. Following the injury, he sustained acute right upper limb weakness with pain. Clinically, the lesion was located to the upper and middle trunk of the right brachial plexus, and the same confirmed with electrophysiological studies. Nerve damage due to lightning injuries is considered very rare, and a plexus damage has been described infrequently, if ever. Thus, the proposed hypothesis that lightning rarely causes neuropathy, as against high-voltage electric current, due to its shorter duration of exposure not causing severe burns which lead to nerve damage, needs to be reconsidered.

Key words: Axonal plexopathy, brachial plexopathy, lightning strike

Introduction

Lightning injuries are known to cause neurological diseases, as also death due either to arrhythmias or other associated injuries.[1] The presentation varies from catastrophic and irreversible neurological deficits to minor transient symptoms.[2] Greater number of cases may be noted in certain geographical areas with greater average rainfall and thunderstorms, though no place is immune. The actual number of fatalities and injuries associated with lightning strikes is not known due to gross underreporting, especially so in developing countries.

Case Report

A 53-year-old man, a field worker in a government establishment, presented to us with a history of lightning strike while working outdoors during a thunderstorm. The incident occurred during mid August, 2013 at around 20:00 hours. He was the only person injured as his colleagues were working at a distance. Immediately following the strike, the patient dropped to the ground unconscious, but did not have any direct blow to his arms or other parts of his body. There was no history of any blast effects or of anything falling on the patient’s body during the incident. There was no history of any injuries due to his body rolling on the ground or falling into trenches. He was immediately rushed to the local hospital by coworkers. There, the patient became conscious within 3 hours and noticed severe pain, weakness, and swelling in his right upper limb, with severe burns on the upper back. The patient was treated symptomatically and his pain and swelling resolved, but the weakness persisted, for which he was referred to our hospital after 6 weeks of the incident.

On examination, his vital parameters were normal. Examining his back revealed a large healing scar of the burns sustained due to the lightning strike, as also right suprascapular hollowing. Power in rhomboids, spinatii, and serratus anterior was preserved. Further testing revealed weakness of his right supraspinatus, infraspinatus, biceps brachii, deltoid, teres major, and pectoralis major (power 2/5 in all muscles). Biceps and wrist flexors were weaker compared to triceps and wrist extensors, while small muscles of the hand were spared. Right biceps, supinator, and triceps jerks were absent. Sensory loss was noted in right C5,6,7 dermatomes. There was no vision loss, dysarthria, or gait disturbance. Ophthalmological examination as also rest of the examination was unremarkable. Routine blood investigations and Magnetic Resonance Imaging [MRI] of cervical spine and brain were normal. Nerve conduction study showed reduced amplitude and normal latency and conduction velocity of the sensory
component of right median nerve and lateral antebrachial cutaneous nerve. Motor nerve study showed decreased amplitude and normal latency and conduction velocity in right median nerve, musculocutaneous nerve, radial nerve, and axillary nerve. Ulnar nerve study was normal. EMG showed profuse fibrillations in right deltoid, biceps brachii and supraspinatus, and some fibrillations in left abductor pollicis brevis and triceps. Rest of the study was normal. A diagnosis of a brachial plexopathy was made, and localized to the upper and middle trunk (C5-6-7).

Discussion

Lightning injuries tend to occur in men in their mid-thirties in the months of July-August while doing outdoor recreational activities during thunderstorms. Lightning causes damage either by thermal effects, electrical effects, magnetic field changes, blast effects, and injuries due to falls. Direct damage to the cell membrane lipid bilayer leads to electroporation followed by cell damage. Lightning-induced neurological disorders may be either immediate and transient, or immediate and permanent. The third category is of delayed syndromes, which includes motor neuron diseases and movement disorders, which has been well described by Jafari et al. Our patient’s symptoms fell in the second category, that is, immediate and permanent. Intracerebral hemorrhage, hypoxic encephalopathy, cerebral infarction, and cerebellar syndromes are more common. Critchley assumed that blood vessels constitute the main routes for the passage of currents through the brain as nerve tissue is a poor conductor of electric current, which causes damage in lightning and electric injuries. For the cervical area, this might imply that the sympathetic plexus around the carotid and vertebral-basilar arteries sustains electrical damage leading to autonomic dysfunction. Spinal cord syndromes are also well documented. However, lightning injuries rarely cause neuropathy, myopathy, or neuromuscular junction disorders, as against electric injuries. Unlike lightning strikes, the longer exposure time in high-voltage electric current causes severe third and fourth degree burns, which leads to neuropathy. However, conclusive evidence for the same is lacking.

As noted above, involvement of the peripheral nervous system due to lightning strikes itself is very rare, a plexopathy even more. This is against the current hypothesis that the short duration of the lightning strike precludes damage to the nerves. It could be likely that the symptoms of neuropathy may have been missed secondary to the more serious complications like cardiac arrhythmia, hypoxic ischemic encephalopathy, and stroke. Also, some of the previously reported cases of generalized polyneuropathy have been suspected. Thus, we would suggest performing routine electrophysiological testing for all patients suffering from lightning strikes in order to find out the true incidence of lightning-induced neuropathy, and not simply rule out the possibility of a lightning strike-induced neuropathy.

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References


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