ESETISMERTETÉS

CASE REPORT

Lightning strike-induced cauda equina syndrome: a case report

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Peripheral nerve injuries after being struck by lightning have been documented. Here, we report a case of cauda equina syndrome induced by lightning. A 27-year-old man presented with numbness, a burning sensation in the saddle region, and increased urinary urgency after being struck by lightning. He had absent Achilles reflexes and paresthesia in the saddle region upon neurological examination, and magnetic resonance imaging of the spine was normal. Electrophysiological studies indicated involvement of bilateral L5, S1, and S2 myotomes and revealed cauda equina lesions.

Peripheral nerve injury induced by lightning is rare, and the evaluation of people with neurological complaints using electromyography will help determine the true incidence.

Keywords: cauda equina, lightning strike, nerve conduction study, peripheral nerve injury, somatosensory evoked potential

Villámcsapás okozta cauda equina szindróma: esetismertetés

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A szakirodalomban található beszámoló villámcsapás után kialakuló perifériás idegsérülésről. Esetismertetésünkben villámcsapás által kiváltott cauda equina szindrómáról számolunk be. Egy 27 éves férfi „lovaglónadrág" eloszlású zsibbadással, égő érzéssel és fokozott vizelési ingerrel jelentkezett, miután villámcsapás érte. A neurológiai vizsgálat során hiányzó Achilles-reflexet és „lovaglónadrág" eloszlású paraesthesiát találtunk, míg a gerinc mágneses rezonanciás képalkotása normális volt. Az elektrofiziológiai vizsgálatok a kétoldali L5, S1 és S2 myotomák érintettségét jelezték, és cauda equina laesiókat mutattak ki.

A villámcsapás okozta perifériás idegsérülés ritka; a neurológiai panaszokkal küzdő személyek elektromiográfiás kiértékelése segít a valódi előfordulási gyakoriság meghatározásában.

Kulcsszavak: cauda equina, villámcsapás, idegvezetési vizsgálat, perifériás idegsérülés, szomatoszenzoros kiváltott potenciál

Lightning is estimated to occur with a frequency of 50 times per second worldwide, and lightning-induced fatalities are thought to be around 24,000 per year[1]. Cardiac arrest and arrhythmias are the main causes of death from lightning strikes. Survivors can experience various complications, such as burns and neurological manifestations[2]. Neurological complications can involve the central and/or peripheral nervous system[3]. Peripheral nervous system complications such as generalized polyneuropathy, brachial plexopathy, and autonomic nervous system involvement have been documented[4–6]. Here, we report a case with cauda equina syndrome after a lightning strike.

Case report

A 27-year-old man presented with a history of lightning strike 6 weeks prior. While on a field mission in heavy rain in November 2022, the patient was struck by lightning and thrown to the ground. Subsequently, he suffered

from numbness and a burning sensation in the saddle region and right leg, and the sensation of increased urinary urgency. There was no history of paralysis and no direct trauma to his body parts. Upon physical examination, burn scars on the skin of the patient's left upper arm, left side of the trunk, and right lower leg indicated that the current entered his body through the left upper arm and exited from the right lower leg **(Figure 1)**, showing that the current crossed his body. The neurological examination was normal except for the absence of the Achilles reflexes and paresthesia in the saddle region. Magnetic resonance imaging (MRI) of the cervical, thoracic, and lumbar spine were normal. Although nerve conduction studies of the bilateral peroneal, median, ulnar motor and median, ulnar, sural sensory nerves were normal, the amplitudes of the compound muscle action potentials of bilateral tibial nerves were low. Needle electromyography testing of the biceps, triceps, brachioradialis, first dorsal interosseous, and bilateral vastus medialis muscles were normal. Nevertheless, abnormal spontaneous activity in the form of fibrillations and positive sharp waves were observed in the bilateral tibialis anterior, gastrocnemius, extensor hallucis longus, tensor fasciae latae, and lumbosacral paraspinal muscles. The motor unit potentials of these muscles were morphologically polyphasic and had a reduced recruitment pattern, showing involvement of bilateral L5, S1, and S2 myotomes. Somatosensory evoked potentials (SEPs) by stimulation of the bilateral tibial nerves could not be obtained from thoracic recording. However, scalp-recorded SEP elicited by stimulation of bilateral tibial nerve showed prolonged peak latencies, demonstrating a conduction delay in the lumbar region **(Figure 2)**. All the clinical and electromyographic findings indicated a cauda equina lesion. The patient was referred to physiotherapy, but no progress was noted in the 2-month follow-up.

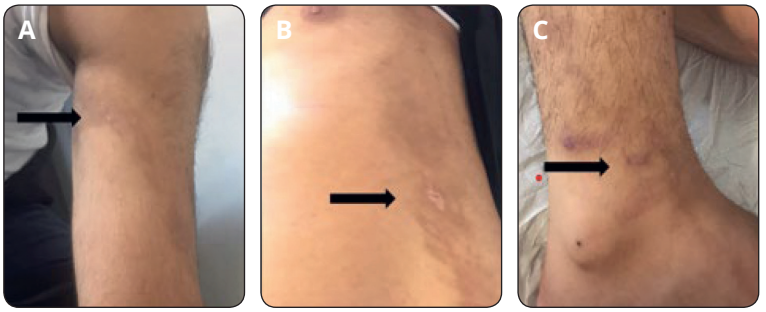

Figure 1. *Burn scars on the skin of the left upper arm (***A***, entry point of the current), the left side of the trunk* **(B)**, *and the right lower leg (***C***, exit point of the current)*

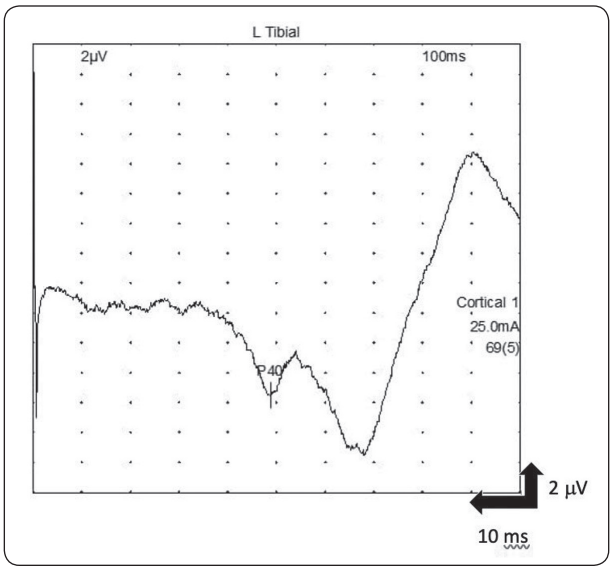

Figure 2. *Prolonged latency of scalp-recorded somatosensory evoked potential by stimulation of the left tibial nerve (P40 latency is 46.8 ms, N<40 ms)*

Discussion

The most catastrophic and most often reported neurologic complications of lightning injuries involve intracranial hemorrhages, cerebral infarction, and post hypoxic–ischemic encephalopathy secondary to cardiopulmonary arrest[3, 7]. However, some lightning strike victims suffer from peripheral nerve damage that affects their quality of life. Neurological complications after lightning strikes can be classified according to time of onset, duration of symptoms, and severity of clinical symptoms[8]. The first kind of complications consist of transient and immediate symptoms, such as loss of consciousness, amnesia, confusion, headache, paresthesia, and weakness. These symptoms are thought to be caused by the temporary excretion of catecholamines. The second kind of complications include immediate and prolonged or permanent symptoms. The majority of patients have structural lesions involving the central nervous system. Peripheral nerves can also be involved. In one case, a 67-year-old male experienced unilateral diaphragmatic paralysis that was attributed to a lightning strike 55 years ago; other possible causes of paralysis were excluded, but it could not be explained why the symptoms appeared so late[9]. *Hawkes* et al. reported a case of acute polyneuropathy due to lightning injury in a young man with many medical complications; the patient presented with flaccid quadriplegia but made a near complete clinical recovery 14 months after the accident[4]. The patient showed lower motor neuron symptoms and small or absent sensory

nerve action potentials suggesting direct damage to the peripheral nerves. However, due to acute renal failure, rhabdomyolysis, respiratory distress syndrome, and autonomic dysfunction, critical illness neuromyopathy might be an alternative diagnosis for this patient. Autonomic nervous system complications after lightning injury are also well-documented[6]. Another documented peripheral nerve injury induced by lightning is brachial plexopathy; the diagnosis of brachial plexopathy was made 6 weeks after the incident and it was not mentioned whether nerve injury was transient or permanent[4]. The third group comprises possible delayed neurologic syndromes attributed to lightning. Progressive spinal cord damage resembling motor neuron disease had been described[10]. The fourth group involves lightning-related secondary trauma from falls or explosions and evaporation of water[8].

Conclusion

Normal MRI findings ruled out structural, inflammatory, neoplastic, and infectious causes. However, if available, analysis of cerebrospinal fluid by lumbar puncture, would have strengthened our diagnosis. To the best of our knowledge, this is the first reported cauda equina syndrome after a lightning strike. The symptoms of our patient are consistent with the second group of neurological symptoms of lightning, immediate and prolonged signs for now. However, clinical follow up of the patient is necessary to determine whether the syndrome is a prolonged or permanent disease. Evaluation of lightning survivors by electromyography will give the chance to determine the true incidence of lightning-induced peripheral neuronal damage.

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