Trigeminal Neuralgia Following Lightning Injury

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Lightning and other electrical incidents are responsible for more than 300 injuries and 100 deaths per year in the United States alone. Lightning strikes can cause a wide spectrum of neurologic manifestations affecting any part of the neuraxis through direct strikes, side flashes, touch voltage, connecting leaders, or acoustic shock waves. This article describes the first case of trigeminal neuralgia induced by lightning injury to the trigeminal nerve, thereby adding a new syndrome to the list of possible lightning-mediated neurologic injuries. *J Oral Facial Pain Headache 2017;31:e7–e9. doi: 10.11607/ofph.1871*

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Lightning strikes can cause a wide spectrum of neurologic syndromes, including visual dazzling, deafness, vertigo, loss of consciousness, amnesia, paralysis, sensory disturbances, cerebral edema, or death.[1–3] The type and extent of lightning injury are determined by the strength of the discharge that reaches the patient and the path that the electrical current takes through the body.[1] Lightning can strike directly—which is often lethal—or flashes from bolts that strike nearby objects and travel through conducting materials may reach the patient indirectly. Lightning discharges can affect any part of the neuraxis, including peripheral nerves.[1,2,4] This article describes the first case of trigeminal neuralgia induced by lightning injury to the trigeminal nerve.

Case Description

A 40-year-old woman with a past medical history notable only for migraine was answering a telephone call at work during a thunderstorm when she heard a sudden crackling sound in the landline receiver that was positioned against her right cheek and ear. The sound was quickly followed by a painful sensation that shot into the right side of her face so intensely that the telephone receiver flew from her hand. Afterward, the telephone no longer worked, nor did the computers and other electrical appliances that drew power from the same circuit; their wires had been damaged by a lightning bolt that had struck the building. She sustained brief amnesia and a minor electrical burn presenting as a red line across her right cheek.

For 5 years following the incident, she experienced paroxysmal stabbing pains that were sharp and sudden with the subjective quality of electricity. The pains lasted a few seconds and were superimposed on intermittent burning sensations involving the V2 distribution of the right trigeminal nerve. She rated the pain intensity as 15 on a scale of 10 and described its severity as "suicidal." The pain occurred spontaneously or was triggered by slightly touching any part of the right side of the face—especially the cheek—or by applying facial cosmetics, speaking, opening her mouth, or brushing her teeth.

The patient initially went through a series of dental procedures, including two dental extractions, even though no evidence of dental pathology was found. These did not alleviate her pain. Carbamazepine

initially abolished the pain—supporting the diagnosis of trigeminal neuralgia—but eventually became ineffective at the highest tolerated doses (at or exceeding 600 mg daily) until continued use was poorly tolerated because of dizziness and ataxia. Thereafter, many other medications were utilized in an effort to alleviate her pain, but the pain proved to be refractory to numerous standard and off-label drugs. These included the prophylactic agents gabapentin (up to 2,700 mg daily), oxycodone (up to 60 mg daily), pregabalin, topiramate, phenytoin, lamotrigine, divalproex sodium, amitriptyline, paroxetine, fluoxetine, and milnacipran, as well as acute symptomatic drugs, including meperidine, morphine, sumatriptan, rizatriptan, ketorolac, and prednisone. These drugs had been prescribed by her previous physician, and although she did not recall the specific doses for all of them, she indicated that each had been increased to the highest tolerated dose. She also tried topical capsaicin, topical lidocaine, acupuncture, and biofeedback, but with no improvement.

Neurologic examination was notable for tactile allodynia and hyperalgesia localized to the right trigeminal maxillary division and subtle flushing and relative warmth of the right cheek. Infrared measurements of facial cutaneous temperature during a pain-free interval were 35.7°C on the right cheek and 34.9°C on the left. Corneal reflexes were intact. No Horner syndrome was present. Masseter muscle bulk and strength and the remainder of her neurologic examination were normal.

Magnetic resonance imaging (MRI) of the brain with gadolinium was unremarkable and showed no evidence of central demyelination or a compressive lesion along the course of the trigeminal nerves.

Because of the severe and pharmacologically refractory nature of the pain, the patient underwent a suboccipital microvascular decompression procedure. Intraoperative inspection discovered the trigeminal nerve root to be compressed by the superior cerebellar artery, which was mobilized free and padded away from the nerve. Her pain subsided for 5 months postoperatively, but then returned as before. The pain then resolved following a balloon compression procedure. At the last follow-up 1 month after the procedure, she had successfully discontinued all her neuropathic pain medications without the pain returning. Four years later, she still has not reported any return of facial pain.

Discussion

This patient's facial pain was consistent with the diagnosis of trigeminal neuralgia as defined by the criteria of the International Headache Society, which specifies it as "a disorder characterized by recurrent unilateral brief electric shock–like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli."[5] Ironically, her facial pain was electrical both in quality and etiology.

Many patients describe the pain of trigeminal neuralgia, whether from neurovascular compression or other causes, as an electric shock–like sensation.[6] This may be because the temporal profile of classic trigeminal neuralgia persuades patients to describe their pain by the analogy of electricity, as each painful paroxysm typically begins and ends abruptly—like a switch turned on and off—and because the paroxysms are sometimes described as pulsating rapidly, like the feeling one experiences when touching a live wire carrying alternating current. This is not to suggest that the patient's trigeminal nerve still carried residual electrical impulses from the original lightning strike; rather, it is concluded that the injury that she sustained to the trigeminal nerve resulted in a chronic state of painful neural discharges or action potentials.

It is plausible that the lightning side flash was responsible for the development of her pain, since the symptoms began precisely following the lightning strike, she had no prior history of facial pain, and the location of the chronic paroxysmal pain coincided with the same branch of the trigeminal nerve that innervated the part of her face where she sustained a minor electrical burn. Beyond these temporal and neuroanatomical correlations, the underlying mechanism is uncertain, but at least two potential mechanisms seem possible.

First, the electrical current of the lightning strike may have caused a direct electrothermal injury to the trigeminal nerve, and potential irreversible nerve ischemia leading to necrosis might have been the cause of peripheral nervous system injury.[3] In other reported cases of electrical injury, multiple pathophysiologic mechanisms for acute and delayed peripheral nervous system damage have been proposed, including direct electrical insult damaging the vasculature, cytotoxicity secondary to increased free radicals, necrosis, and demyelination.[7] For each of these mechanisms, focal axonal or myelin injury has been hypothesized to generate aberrant neural discharge or ephaptic neural transmission. Cases of peripheral nervous system involvement following lightning injury have rarely been reported in the literature. Hawkes and Thorpe[4] described a patient who, following a lightning strike, suffered quadriplegia secondary to acute polyneuropathy, as demonstrated by low amplitude or absent sensory nerve action potentials on nerve conduction testing. Cases of median mononeuropathy,[8] acoustic and vestibular nerve injury,[9] and unilateral diaphragmatic paralysis[10] have also been reported.

Second, the electric current may have indirectly injured the trigeminal nerve via vascular damage, compromising the nerve's blood supply. Arteries readily conduct electricity, and arterial thrombosis or coagulation are known sequelae of lightning injuries.[1,4]

It is possible, moreover, that neurovascular compression, which was identified intraoperatively and was asymptomatic prior to the lightning strike, might have predisposed the patient to developing pain from the lightning injury as a mechanical phenomenon.

Conclusions

This case history report presented a unique case of trigeminal neuralgia that developed immediately after a lightning bolt injury to the patient's face. The clinical characteristics (ie, short duration of paroxysms, sharp or electrical quality of pain, triggering of pain by remote facial stimuli, excellent initial response to carbamazepine, and eventual progression to medical refractoriness) were identical to those of idiopathic trigeminal neuralgia. This case adds a new presentation to the list of possible lightning-mediated neurologic injuries.

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