

Case reports

Bilateral sudden sensorineural hearing loss following indirect lightning exposure successfully treated with hyperbaric oxygen therapy: a case report

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Abstract

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Introduction: Lightning injuries may result in a wide spectrum of neurological and otological complications. Sudden sensorineural hearing loss (SSNHL) following indirect lightning exposure is rare, and evidence regarding the role of hyperbaric oxygen therapy (HBOT) remains limited. We report a case of severe bilateral SSNHL following indirect lightning exposure with substantial recovery after HBOT.

Case report: A 26-year-old man developed sudden bilateral hearing loss after a lightning strike hit a lightning rod approximately 20 m from his location. He experienced transient syncope and limb weakness consistent with keraunoparalysis. Audiometry demonstrated severe bilateral sensorineural hearing loss, with pure-tone averages (PTA) of 70 dB HL in the right ear and 75 dB HL in the left ear. Because of persistent hearing loss despite systemic corticosteroid treatment, HBOT was initiated approximately 105 days after injury. A total of 30 HBOT sessions were administered together with intratympanic steroid injections. After 20 sessions, PTAs improved to 39 dB HL and 42 dB HL. Following completion of treatment, PTAs improved to 15 dB HL and 12 dB HL, with speech discrimination scores of 96% bilaterally.

Conclusions: Severe bilateral SSNHL following indirect lightning exposure is rarely reported. Proposed mechanisms include cochlear injury, microvascular compromise, and oxidative damage to inner-ear structures. This case demonstrates that indirect lightning exposure may result in both keraunoparalysis and severe bilateral SSNHL. Substantial hearing recovery was observed despite initiation of HBOT more than three months after injury, suggesting a potential role for HBOT as part of a multidisciplinary treatment approach in selected cases.

Introduction

Injuries due to lightning strikes are uncommon but potentially devastating, associated with substantial morbidity and mortality. Although cardiac and respiratory arrest remain the principal immediate causes of lightning-related death, survivors may experience a wide range of neurological, cardiovascular, dermatological, and otological sequelae.^{1,2} The auditory system is particularly vulnerable because lightning can cause direct electrical injury, blast effects, thermal damage, and microvascular compromise affecting the structures of the middle and inner ear.^{2,3}

Otologic manifestations of lightning injuries include tympanic membrane perforation, tinnitus, vertigo,

conductive hearing loss, and sensorineural hearing loss. Tympanic membrane rupture is considered the most common otologic injury due to the intense shock wave generated by lightning strikes.^{3,4} However, sudden sensorineural hearing loss (SSNHL) following lightning exposure is rarely reported, and its underlying pathophysiological mechanisms remain incompletely understood. Proposed mechanisms include cochlear ischaemia, disruption of hair-cell function, microvascular injury, and direct damage to auditory pathways.^{2,3,5} Another neurological manifestation of lightning injury is keraunoparalysis, a transient syndrome characterised by motor weakness, sensory disturbances, and peripheral vascular symptoms that typically resolves spontaneously within hours after the event.^{6,7} Most lightning injuries result from indirect mechanisms such as ground

current, side flash, or blast effects, and auditory injury may occur even in the absence of direct contact with the lightning discharge.^{8,9}

Hyperbaric oxygen therapy (HBOT) is an established adjunctive treatment for idiopathic SSNHL and is believed to enhance oxygen delivery to the ischaemic cochlea, improve inner-ear oxygen tension, and facilitate recovery of injured auditory structures.¹⁰⁻¹² Although HBOT has been investigated in idiopathic SSNHL, data regarding its use in lightning-associated hearing loss remain limited. We report a case of severe bilateral SSNHL that developed after indirect lightning injury following a nearby lightning strike to a lightning rod and demonstrated near-complete audiometric recovery after a substantially delayed course of HBOT.

Case report

Written informed consent was obtained from the patient for publication of this case report and the accompanying audiometric figures.

A 26-year-old previously healthy, non-smoking man was exposed to a lightning-related incident when a lightning strike hit a lightning rod located approximately 20 metres from his position. Immediately following the event, he experienced syncope and was transported to the emergency department for evaluation. After regaining consciousness, he

reported bilateral hearing loss. He had no previous history of hearing impairment, chronic medical illness, or tobacco use.

In addition to hearing loss, the patient developed transient weakness involving both the upper and lower extremities immediately after the incident. The weakness gradually improved approximately three hours after the event and resolved completely within four hours. No residual motor deficits were identified on subsequent neurological examinations. This transient neurological presentation was considered consistent with keraunoparalysis, a well-recognised and typically reversible neurological manifestation of lightning injury.

Radiological investigations showed no intracranial or otological abnormalities. Otoloscopic examination demonstrated intact tympanic membranes bilaterally without perforation.

The patient was subsequently referred to the otolaryngology department because of persistent bilateral hearing loss. Oral corticosteroid therapy (Deltacortril) was initiated eight days after the incident for bilateral SSNHL. Despite treatment, severe bilateral hearing loss persisted. Pure-tone audiometry performed prior to HBOT demonstrated pure-tone averages (PTA) of 70 dB HL in the right ear and 75 dB HL in the left ear (Figure 1). The patient was referred to our hyperbaric medicine unit approximately 105 days after the lightning-related incident.

Figure 1

Initial pure-tone audiometry demonstrating severe bilateral sensorineural hearing loss after lightning exposure. Pure-tone averages were 70 dB in the right ear and 75 dB in the left ear

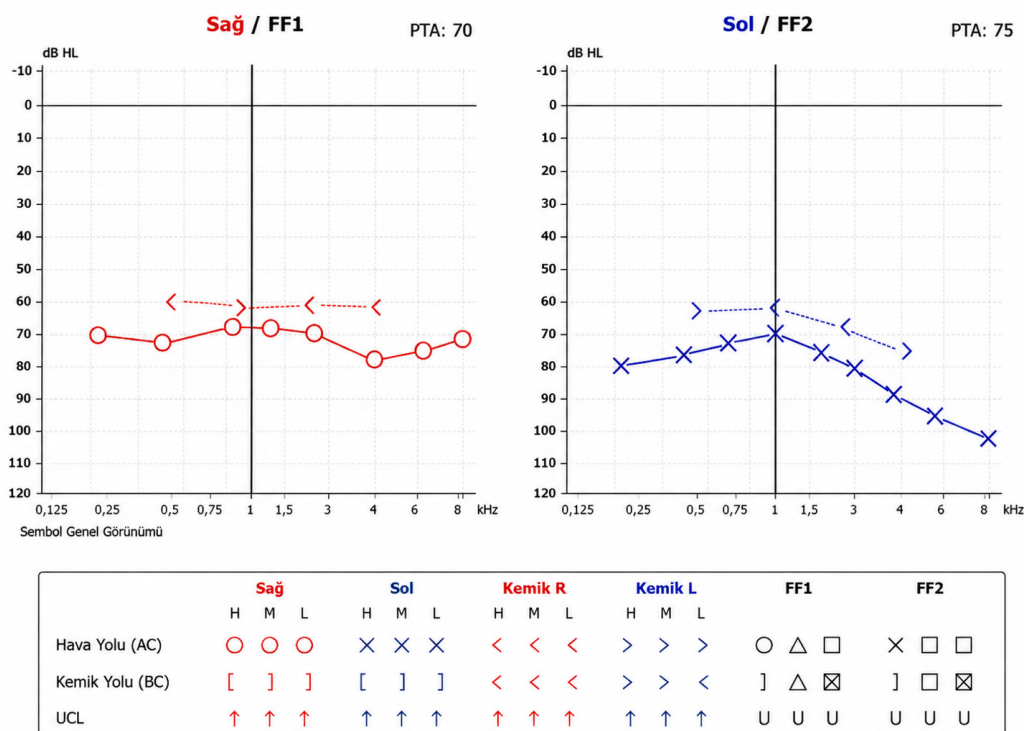
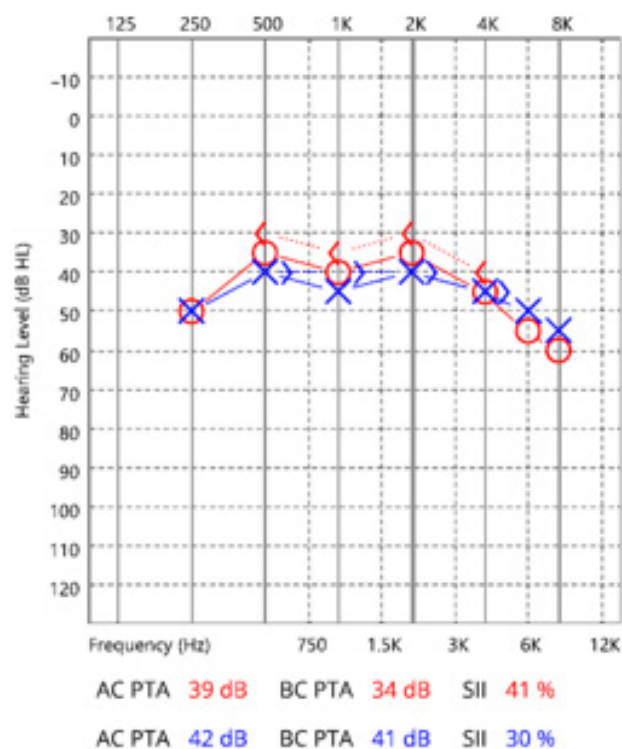
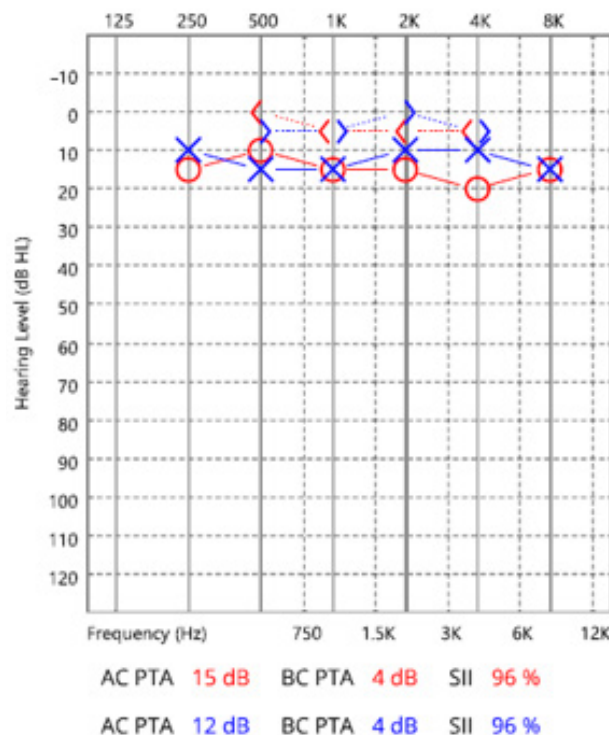


Figure 2

Pure-tone audiometry obtained after 20 sessions of hyperbaric oxygen therapy demonstrating partial recovery of hearing thresholds. Pure-tone averages improved to 39 dB in the right ear and 42 dB in the left ear, although speech discrimination scores remained limited (41% and 30%, respectively)

**Figure 3**

Final pure-tone audiometry obtained after completion of 30 hyperbaric oxygen therapy sessions and five bilateral intratympanic steroid injections demonstrating near-complete recovery. Pure-tone averages improved to 15 dB in the right ear and 12 dB in the left ear, with speech discrimination scores of 96% bilaterally



HBOT was initiated and administered in a multiplace hyperbaric chamber according to the institutional protocol for SSNHL. A total of 30 HBOT sessions were completed. In addition, bilateral intratympanic steroid injections were administered concurrently with HBOT at two-day intervals for a total of five injections in each ear.

Repeat audiometric evaluation after 20 HBOT sessions demonstrated substantial improvement in hearing thresholds. Pure-tone averages improved to 39 dB HL in the right ear and 42 dB HL in the left ear. Although hearing thresholds improved significantly, speech discrimination remained limited (41% in the right ear and 30% in the left ear) at this stage of treatment (Figure 2).

Following completion of 30 HBOT sessions and five bilateral intratympanic steroid injections, further audiometric recovery was observed. Final pure-tone audiometry demonstrated near-complete recovery, with pure-tone averages of 15 dB HL in the right ear and 12 dB HL in the left ear. Speech discrimination scores improved to 96% bilaterally (Figure 3). The patient reported marked subjective improvement in hearing and successfully returned to his normal daily activities.

The diagnosis of lightning-associated bilateral sudden sensorineural hearing loss was supported by the temporal relationship between the lightning incident and symptom onset, the presence of transient keraunoparalysis immediately after exposure, the absence of previous otological disease, the presence of severe bilateral sensorineural hearing loss on audiometric evaluation, and the exclusion of alternative neurological and otological causes. Although the patient had previously received systemic corticosteroid therapy without measurable benefit, substantial hearing recovery was observed following treatment with HBOT combined with intratympanic steroid administration.

Discussion

Lightning injuries are associated with a broad spectrum of neurological and otological complications. Otological manifestations most commonly include tympanic membrane perforation, tinnitus, vertigo, and hearing loss. Among these, tympanic membrane rupture is considered the most frequent otologic finding because of the intense blast wave generated during a lightning strike.^{3,4} In contrast, SSNHL following lightning exposure is uncommon, particularly in the absence of direct lightning strike or structural ear injury.²⁻⁴ Most lightning injuries result from indirect mechanisms such

as ground current, side flash, or blast effects rather than direct strikes.^{8,9} The occurrence of severe bilateral SSNHL highlights the potential for significant auditory injury even after indirect exposure.

Isolated sensorineural hearing loss following lightning injury has been reported only rarely in the literature.^{4,13} In the present case, severe bilateral SSNHL developed after indirect exposure to a nearby lightning rod strike, without tympanic membrane rupture, temporal bone injury, or radiologically detectable abnormalities. These findings suggest that inner-ear injury may occur even in the absence of overt structural damage.

The patient developed transient weakness involving both upper and lower extremities immediately after the event. The neurological deficits resolved completely within four hours and were consistent with keraunoparalysis, a characteristic but typically reversible neurological manifestation of lightning injury.^{6,7} Keraunoparalysis is believed to result from transient autonomic dysfunction and intense vasospasm induced by the electrical discharge.^{6,7} The presence of this phenomenon further supports a causal relationship between the lightning exposure and the subsequent auditory injury observed in our patient.

The precise mechanism responsible for hearing loss in lightning injuries remains uncertain. Several pathophysiological pathways have been proposed, including cochlear ischaemia, microvascular compromise, disruption of hair-cell function, oxidative stress, and direct neural injury.^{2,3,5} In the present case, the absence of tympanic membrane perforation, temporal bone injury, or intracranial abnormalities suggests that cochlear injury and microvascular compromise may have contributed to the development of hearing loss.

HBOT has become an accepted adjunctive treatment for idiopathic SSNHL and is recommended by contemporary clinical practice guidelines.¹⁰ HBOT increases dissolved oxygen content in plasma, improves oxygen delivery to the cochlea, and may enhance recovery of ischaemic inner-ear structures.^{11,12} Although evidence supporting HBOT in idiopathic SSNHL has grown, data regarding its use in lightning-associated hearing loss remain extremely limited.

Hyperbaric oxygen therapy has also been associated with hearing improvement in conditions other than idiopathic sudden sensorineural hearing loss, including acute acoustic trauma. Studies of both acute acoustic trauma and idiopathic sudden sensorineural hearing loss have generally reported better outcomes when treatment is initiated early.^{10–14} In contrast, HBOT in our patient was initiated approximately 105 days after the lightning-related injury, well beyond the conventionally accepted therapeutic window. Despite this prolonged delay, substantial audiometric recovery was observed, making the favorable outcome in the present case particularly noteworthy.

Substantial audiometric recovery was observed following treatment, with near-complete normalisation of hearing thresholds and restoration of speech discrimination scores. Oral corticosteroid therapy failed to produce measurable clinical improvement and HBOT was initiated approximately 105 days after the lightning-related injury, well beyond the conventionally accepted therapeutic window for SSNHL. Although causality cannot be established from a single case, the progressive improvement documented on serial audiometric examinations and temporal relationship between HBOT and recovery after a long treatment latency suggests a potential therapeutic benefit of HBOT-based treatment strategies in selected patients with lightning-associated SSNHL.

This case has several limitations. The combined use of HBOT and intratympanic steroid therapy precludes assessment of the independent effect of either intervention. Nevertheless, the temporal relationship between lightning exposure, bilateral SSNHL, keraunoparalysis, and subsequent audiometric recovery provides valuable clinical observations. To our knowledge, reports describing successful treatment of lightning-associated bilateral SSNHL with HBOT remain exceedingly limited.

Conclusions

Severe bilateral SSNHL may occur following indirect lightning exposure even in the absence of direct lightning strike or radiologically detectable injury. Clinicians should be aware of this uncommon presentation and consider early audiological assessment in symptomatic patients. HBOT, particularly when incorporated into a multidisciplinary treatment approach, may represent a useful therapeutic option in selected cases of lightning-associated SSNHL.

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