Case Report

Bilateral Sudden Sensorineural Hearing Loss Following Unilateral Temporal Bone Fracture

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Temporal bone fractures usually cause unilateral sensorineural hearing loss (SNHL) by fracture that violated otic capsule of that side. Bilateral SNHL from unilateral temporal bone fracture were rarely seen. Labyrinthine concussion was considered to be the pathogenesis in these cases. This article reports an additional case of bilateral SNHL from unilateral temporal bone fracture but in a different pattern of SNHL which may result from an occlusion of the internal auditory artery.

Keywords: *Fractures, Bone, Hearing loss, Bilateral, Hearing loss, Sensorineural, Temporal bone*

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Temporal bone fractures have been reported about forty per cent of skull base injuries[1]. Otologic complications are well-recognized complications of temporal bone fractures, following which the reported incidence of hearing loss varies between 24 and 81%, CSF leak between 7 and 30%[1]. Sensorineural hearing loss (SNHL) is usually caused by fracture that violated otic capsule of that side. Bilateral SNHL from unilateral temporal bone fractures were rarely reported[2]. This paper reports an additional case but with a different audiologic result.

Case Report

A 45-year-old woman presented to the Department of Otolaryngology, HRH Princess Maha Chakri Sirindhorn Medical, Srinakharinwirot University, ten days after suffering blunt trauma to the head from motorcycle accident. Initially, she was brought to the emergency unit of the local hospital near the accidental site. She was treated for her right shoulder pain. X-ray finding revealed right scapular fracture, then arm sling was applied. She also had bleeding per right ear, hearing loss in both ear (right ear more than left ear) and tinnitus in the right ear. She had vertigo for 2 days and then resolved.

She was admitted for 10 days without any investigation about her ear symptoms. She was referred to this hospital on the next day. At the ENT outpatient department, she complained of having tinnitus in the right ear. She also reported loss of hearing in her right ear and decreased hearing in her left ear. Physical examinations revealed normal ear canal and tympanic membrane in both ears but with some clotted blood on the right tympanic membrane. Right lower motor neuron facial palsy was detected without patient awareness. Audimetry showed profound sensorineural hearing loss (SNHL) in the right ear and mild to severe SHNL in the left ear (Fig. 1). The speech reception threshold (SRT) was more than 100 dB and speech discrimination scores (SDS) was zero percent in right ear. The SRT was 40 dB and SDS was 88 percent in the left ear. The X-ray of the temporal bone suggested fracture of the right temporal bone (Fig. 2). The left temporal bone was normal. She was treated with oral prednisolone, mecobalamin and betahistine. One week later, she noted improvement in hearing acuity. Physical examinations showed right facial palsy with 80-100 % response by maximum stimulation tests (MST). Audiometry showed improvement in both ears. SRT was 80 dB and SDS was 48 percent in the right ear. SRT

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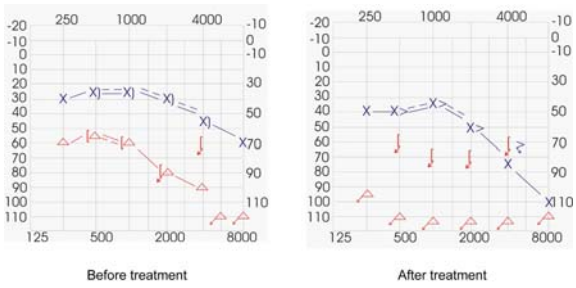

Fig. 1 Audiographic results show improvement in both ear

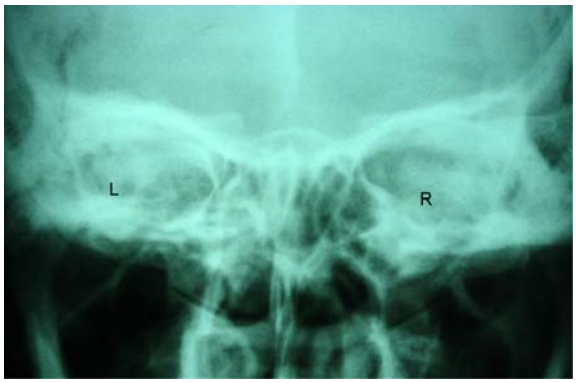

Fig. 2 X-ray film of the temporal bone

was 25 dB and SDS was 100 percent in the left ear. She was treated with the same drugs except prednisolone. Two weeks later, she still felt of tinnitus in her right ear but improved facial expression. Auditory brainstem evoked response showed latencies of wave I, III, V and interwave latencies of the left ear were within normal limit. These findings suggest cochlear lesion of SHNL in the left ear. One month later, she was reported decreasing in tinnitus and normal facial expression. The MST revealed 100% response of all branches of the right facial nerve. Audiometry showed mild SNHL in the left ear and moderately severe to profound SNHL in the right ear. SRT was 65 dB and SDS was 48 percent in the right ear. SRT was 25 dB, and SDS was 100 percent in the left ear (Fig. 1).

Discussion

Temporal bone fractures have been classified as longitudinal and transverse by the long axis of the temporal bone. About 80% of the fractures are longitudinal and 20% are transverse[3]. Longitudinal fractures typically run from the external auditory canal, through the middle ear and geniculate ganglion, and end in the foramen lacerum caused mostly by conductive hearing loss from any of these conditions such as hemotympanum, tympanic membrane perforation or ossicular chain disruption or combination of each.

Transverse fractures usually following direct temporofrontal or occipital trauma which required more force than longitudinal fractures and can be divided into the medial and lateral types. The lateral types run pass through the vestibule and the medial wall of the tympanic cavity and can involve the cochlea. The medial types runs medial to the otic capsule and pass through the fundus of the internal auditory canal. These types are usually more associated with SNHL, vertigo and fifty per cent cause facial palsy[4], CSF leak is also more common. The presentation can be hemotympanum without bleeding per ear. SHNL can result from damage to the cochlea, acoustic nerve or the intracranial pathways. Auditory evoked potentials are used to identify the site of damage.

Recently a new classification system has been proposed based on the involvement of the otic capsule[1]. If the otic capsule is violated the patient is seven times more possibility to develop SHNL, four times as chance to have CSF leak and twice as chance to develop facial nerve palsy.

SNHL resulted from temporal bone fractures is usually sudden and ipsilateral. However, bilateral SHNL from fractures and contralateral SHNL from other types of inner ear conditions can occur. Ulug and Ulubil[2] reported 3 cases of temporal bone fractures that had contralateral SHNL, 2 in the left ear and 1 in the right ear. All of them showed characteristic notch of hearing loss ranging between 50-60 dB at 4,000 Hz. Labyrinthine concussion was considered to be the pathogenesis of these findings. All patients were treated for facial palsy by surgery; middle cranial fossa or combined approach. SHNL was treated by expectant but none showed recovery.

Feneley and Murthy[5] reported a case of 57-year-old man who had acute bilateral deafness and vestibular dysfunction following occipital bone fracture. CT scan showed single fracture of the occipital bone. Hearing showed partial recovery over the following months.

Atkin et al[6] reported a case of bilateral sudden SHNL, vestibular dysfunction and a left hemiparesis without facial weakness following head injury. CT scan revealed bilateral transverse temporal bone fractures. His left hemiparesis resolved. He had undergone vestibular rehabilitation and cochlear

implant in the left ear. Bilateral SNHL caused by bilateral temporal bone fractures[7-9] were also reported. Also, SHNL following minor head injury[10] and mid-brain contusion without evidence of skull fracture[11] had been reported. Café et al[12] reported a case of SNHL in the right ear, 11 years previously he had left deafness following temporal bone fracture. Auto-antibodies to inner ear tissues were detected by immunochemistry and western blotting technique. Autoimmune-mediated sympathetic hearing loss was proposed to be the pathogenesis in this case. There are many reports of contra-lateral SHNL after vestibular schwannoma surgery. The proposed etiologies included loss of CSF[13], drill noise-induced acoustic trauma[14,15], ototoxicity[16], round membrane rupture due to nitrous oxide[16], sympathetic cochleo-labyrinthitis[17], and occlusion of internal auditory artery[18,19]. Loss of CSF usually caused temporary SHNL[13] but permanent deafness have been described[20,21]. Sympathetic cochleolabyrinthitis theorized by Harris[17] that immunocompetent cells become sensitized to previously unseen inner ear antigens after surgery and injury to the inner ear thus providing for autoimmune destruction against inner ear antigens in both the incident ear and the contra-lateral ear. This phenomenon usually takes several days, or even weeks or months, to occur. Occlusion of internal auditory artery can cause sudden SNHL and loss of vestibular function, by either thrombosis[19] or vasospasm[22,23].

The reported case in this article shows bilateral SHNL after a unilateral (right) temporal bone fracture confirmed by normal contra-lateral (left) otologic examinations and the X-ray of the temporal bone suggested fracture on the right side and normal left temporal bone. An auditory-evoked response showed bilateral cochlear lesions of SHNL. She also had incomplete right lower motor neurone facial palsy that completely resolved in 2 months. All these findings explain the clinical presentations of the fracture of the right temporal bone with otic capsule violation, but can't explain the sudden SNHL in the left ear. Hearing thresholds are dropping in all frequencies in the left ear which is different from that reported by Ulug and Ulubil[2] that showed notch of hearing loss at 4,000 Hz. The pathogenesis, labyrinthine concussion, may or may not be possible. Autoimmune-mediated sympathetic labyrinthitis is not thought to cause sudden SNHL in this patient because it usually takes time to show SHNL[17]. Loss of CSF, another possible cause, can produce temporary contra-lateral SHNL as seen in the patients having vestibular schwannoma surgery. Loss of CSF results in a decreased CSF pressure that is transmitted to the perilymph via the cochlear aqueduct. The perilymph depression generates a compensatory expansion of the endolymphatic component and a subsequent endolymphatic hydrops. The low frequencies were affected first after surgery, howere, after that, the higher frequencies also became involved[24]. Hearing loss, mostly normalized within 3 months, except for some cases where it did not[13,21]. In this reported case, there is no evidence of CSF otorrhea or leak, thus CSF lost-induced contra-lateral SHNL is unlikely to be the etiology.

In conclusion, the most probable cause in this case may be the occlusion of the internal auditory artery. Fortunately, there was complete recovery from her facial palsy and vertigo, and improved hearing occurred in both ears after treatment.

Conclusion

Contra-lateral SHNL after unilateral temporal bone fracture is extremely uncommon. Labyrinthine concussion is proposed for the etiology in the previously reported cases with SNHL notching at 4,000 Hz. This reported case show a different pattern of SNHL which may result from an occlusion of the internal auditory artery.

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โรคประสาทหูเสื่อมเฉียบพลันทั้ง 2 ข้าง ที่เกิดจากการแตกหักของกระดูก temporal ข้างเดียว: รายงานผู้ป่วย 1 ราย

นิรันดร์ หุ่นฉายศรี

การแตกหักของกระดูก temporal อาจทำให้เกิดโรคประสาทหูเสื่อมเฉียบพลันในด้านนั้น โดยมีผลกระทบต่อ otic capsule โดยตรง แต่ก็พบว่าทำให้เกิดประสาทหูเสื่อมเฉียบพลันทั้ง 2 ข้างพร้อมกันได้ ซึ่งเชื่อว่าเกิดจากภาวะ labyrinthine concussion รายงานฉบับนี้ได้นำเสนอผู้ป่วยประสาทหูเสื่อมเฉียบพลันทั้ง 2 ข้าง ที่เกิดจากการแตกหักของกระดูก temporal ข้างเดียว แต่กลไกในการเกิดโรคประสาทหูเสื่อมในผู้ป่วยรายนี้เชื่อว่าน่าจะเกิดจากการอุดตันของหลอดเลือดแดง internal auditory