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Ossicular necrosis following head injury

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Abstract. *Ossicular necrosis following head injury. Problems/objectives*: The aim of this study is to report on ossicular necrosis in the middle ear occurring shortly after head injury.
Methodology: Our sample included 3 males and 2 females aged 9 to 37 years who complained of unilateral hearing loss after a head trauma that had occurred 3 to 6 months previously. The tympanic membranes were intact, and a CT-scan did not show fracture of the temporal bone. Audiometry showed a unilateral conductive hearing loss in all cases.
Results: On tympanotomy exploration, 4 of the 5 patients had a necrosis of the lenticular process of the incus and one patient had a necrosis of the posterior and anterior crura of the stapes. Additionally, the incus was displaced at the incudomalleolar joint towards the promontory in two patients. The long process of the incus was surgically aligned and adjusted to the head of the stapes through interposition of either temporal fascia or a bone chip. At the 2-year-postoperative follow-up, the pure tone audiometry showed that the preoperative air-bone gap was almost closed with a mean of 11.8 dB.
Conclusions: It is possible that the head injury resulted in ossicular displacement in the middle ear with disturbance of the local vascular supply. Due to the tenuous blood supply to the lenticular and long processes of the incus, this portion of the ossicular chain, including the stapes suprastructure, may become more vulnerable to an ischemic process and subsequent necrosis shortly after the head trauma.

Introduction

Head injury may result in fractures of the temporal bone and damage to the external auditory canal, middle ear, or cochlea. Temporal bone fractures are classically divided into longitudinal, transverse, and oblique fractures with the designation indicating the fracture line relative to the long axis of the petrous temporal bone. Trauma to the middle ear usually manifests as conductive hearing loss. Conductive hearing loss may be caused by middle ear hemorrhage or ossicular disruption. Middle ear bleeding and attendant hearing loss resolves in 60% to 80% of patients within 3 weeks.[1] In a few patients, ossicular injury is the cause of unresolved hearing loss and may require surgical intervention.

Although ossicular injuries usually manifest as a dislocation or fracture,[2] to our knowledge necrosis of the ossicular chain following head trauma has not been described previously.

Material and methods

The patient characteristics are shown in Table 1. There were 3 males and 2 females 9 to 37 years of age. According to their history, patients 1, 4, and 5 (Table 1) fell off their bicycle and hit their head against the curb; patient 2 slid in the bath, and patient 3 (a child) slid on the corridor next to a swimming pool (both hitting their head).

The patients presented in our clinic 3 to 6 months after the injury complaining of unilateral hearing loss. The patients reported that on the day of the injury they did not have loss of consciousness, bleeding from the ear, drowsiness, amnesia, or prolonged headaches. On admission, the tympanic membrane was intact in all patients without sign of recent perforation. Tuning fork testing revealed a negative Rinne with the Weber lateralizing to the affected ear. Pure tone audiometry confirmed a unilateral conductive hearing loss in all cases, ranging from 31 to 41 dB across all frequencies (median: 34.8 dB). The CT scan did not show temporal bone fracture or other intracranial abnormalities.

All patients underwent tympanotomy exploration a short time after their admission. Under general anaesthesia, a post-aural incision and tympanomeatal flap was used in order to expose the patients' middle ear.

Results

In patients 1 and 2, a necrosis of the lenticular process of the incus

Table 1

Patients' age, sex, and side of hearing loss due to head injury at the time of surgery (y = years)

Patient	Age, sex, side
1.	9 y, ♂, left ear
2.	27 y, ♀, right ear
3.	9 y, ♀, left ear
4.	15 y, ♂, right ear
5.	37 y, ♂, right ear

Figure 1

The necrosis of the lenticular process of the incus of a head injury patient (patient 2) is shown. S = head of the stapes, Inc = long process of the incus.

was noted (Figure 1). In patients 3 and 4, in addition to necrosis of the lenticular process of the incus, the ossicular chain was found to be discontinuous at the incudomalleolar joint and the remaining long process of the incus was displaced towards the promontory. However, a possible incudostapedial disarticulation was not excluded in 3 cases (cases 1, 2, and 3), but these were without evidence of associated dislocation at the level of the incudomalleolar articulation. In all patients (except case 5), the incus was slightly manipulated through its long process in order to be correctly aligned and adjusted to the head of the stapes. In patient 5, the posterior and the anterior crura of the stapes were completely missed. In patients 1 and 2, a bone chip was carved out from the cortical bone of the mastoid and positioned between the long process of the incus and the head of the stapes to bridge the small gap created after the ossicular necrosis. Similarly, in patients 3 and 4, a small piece of temporalis fascia was placed between the remaining long process of the incus and the head of the stapes to facilitate their anatomical contact. In patient 5, a piston was positioned between the long process of the incus and the footplate.

At the postoperative follow-up 2 years after surgery, the tuning fork tests demonstrated a bilaterally positive Rinne and a central Weber in all patients. Moreover, the pure tone audiometry showed that the preoperative air-bone gap was almost closed with a mean of 11.8 dB.

Discussion

Various forms of ossicular injuries have been described previously, such as incudomalleolar joint separation, dislocation of the malleoincudal complex, and stapediovestibular dislocation.[2] Isolated fractures of the long process of the incus,[3] malleus handle,[4] and arch of the stapes[5,6] have also been described which result in conductive hearing loss to a lesser degree. Incudostapedial joint and incus dislocation was found to be the most common ossicular abnormality in temporal bone fractures.[7,8] The pathophysiological mechanism of incudostapedial disarticulation remains controversial. Some cite the fragile enarthrosis of the incudostapedial disarticulation as a contributing factor,[9] while others refer to the fact that the incus is a heavier ossicle (27.5 mg) compared with the malleus and stapes. Also, having only poor ligamentous support, the incus is more vulnerable to dislocation, especially when incudomalleolar joint separation is associated with incudostapedial joint separation or a fracture of the stapes.[10] According to an alternative theory, the dislocation of the incus results from tetanic contraction of the stapedius and tensor tympanic muscles following the head trauma.[11] In this scenario, the incus is pulled medially while a similar violent contraction of the stapedius tendon would pull the stapes posteriorly.

In our study, the principal finding was the necrosis of the lenticular process of the incus (in 4 cases) and of the posterior and anterior crura of the stapes (one case). It is possible that the head injury resulted in ossicular displacement in the middle ear with disturbance of the local vascular supply. The blood supply to the lenticular and long processes of the incus is irregular and often tenuous (Figure 2), and may lead

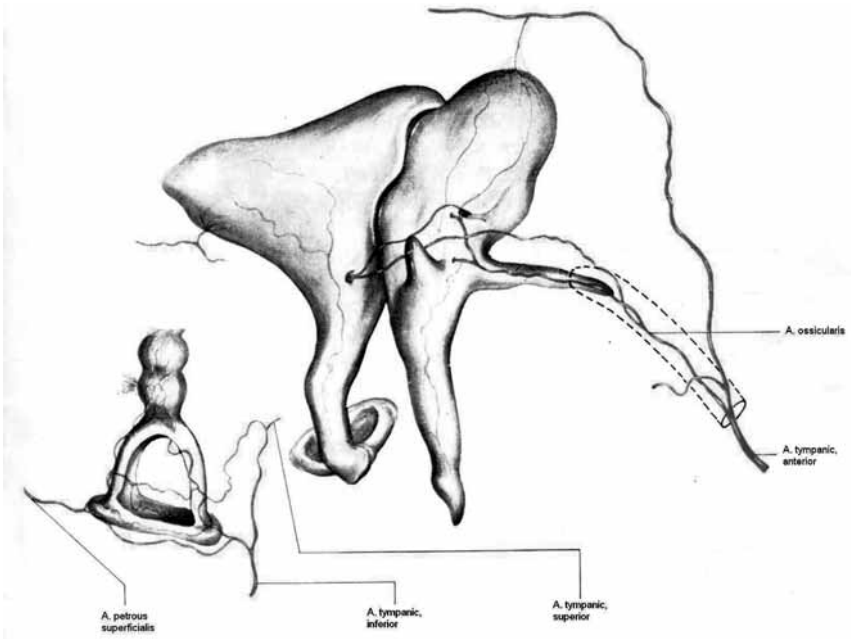

Figure 2
The irregular and tenuous blood supply to the lenticular and long processes of the incus is shown.[20]

to erosion of this portion of the ossicular chain.[12] Thus, shortly after the head trauma, it is possible that the impairment of the vascular supply in this segment of the ossicular chain could lead to necrosis, either of the lenticular process of the incus or the stapes suprastructure. However, Lannigan *et al*.[13] showed that the vascular anatomy of the lenticular and long processes of the incus is similar to that of the short process and body, and concluded that necrosis in the lenticular and long processes could not be related to vascular insufficiency. They also demonstrated that the lenticular and long processes possess a greater degree of osteoclastic pitting of the bone surfaces than is found elsewhere on the incus or in the rest of the ossicular chain. They also suggested that osteoclastic activity may account for the predilection of this portion of the ossicular chain to undergo resorption. Nevertheless, in our cases, although hypothetical, a local ischemic process seems more likely to have occurred shortly after the traumatic injury which resulted in ossicular necrosis. Apart from other infectious diseases of the middle ear, a similar clinical aspect of the missing lenticular process and suprastructure of the stapes has also been encountered in the case of a patient with diabetes mellitus[14] which was attributed to impaired vascular supply and subsequent increased osteoclast activity. In another study,[15] an absence of the inferior aspect of the long process of the incus (but with an intact lenticular process) was reported in one case, and it was hypothesized that since the long process of the incus undergoes remodeling through resorption and rebuilding throughout life, the above clinical finding was caused by either failed remodeling or impaired vascular supply to the incus. Moreover, a complete loss of the long arm of the incus was described in the context of a family expansile osteolysis with bony lesions similar to those found in Paget's disease.[16]

Regarding the surgical correction, either the ossicles are reshaped to reconstitute the ossicular chain, or a hydroxyapatite prosthesis and cartilage autografts are used to reconstruct the ossicular chain following ossicular injury.[17] In cases 1 and 2 presented here, a bone chip was positioned between the remaining long process of the incus and the head of the stapes, as described by Silverstein.[18] The temporalis fascia used in cases 3 and 4 had previously been advocated to be effective in the subluxation or separation of the incudostapedial joint with partial dislocation of the incus.[19] In this study,[19] the temporalis fascia was similarly interposed between the long process of the incus and the head of the stapes. This serves to minimize the frictional resistance to movement generated between these two dry, rough articular areas to ensure a firm, movable union of the repositioned incus with the stapes with full functional restoration (the follow-up ranged between 1 and 11 years). Finally, the necrosis of the stapes suprastructure in patient 5 was successfully treated by using a piston between the incus and footplate as described previously in fractures of the stapes arch.[3,5]

Conclusion

In conclusion, a displacement of the incus at the level of the incudomalleolar and incudostapedial articulation can occur following a head injury. Due to the

tenuous blood supply to the lenticular and long processes of the incus, this portion of the ossicular chain, including the stapes suprastructure, may become more vulnerable to an ischemic process and subsequent necrosis shortly after the head trauma.

References

1. Lee D, Honrado C, Har-El G, Goldsmith A. Pediatric temporal bone fractures. *Laryngoscope.* 1998;108: 816-821.
2. Meriot P, Veillon F, Garcia JF, *et al*. CT appearances of ossicular injuries. *Radiographics.* 1997;17:1445-1454.
3. Spector GJ, Pratt LL, Randall G. A clinical study of delayed reconstruction in ossicular fractures. *Laryngoscope.* 1973;83:837-851.
4. Applebaum EL, Goldin AD. Surgical management of isolated malleus handle fractures. *Laryngoscope.* 2000; 110:171-173.
5. Ballantyne JC. Review of traumatic conductive deafness. *Proc R Soc Med.* 1966;59:535-542.
6. Singh S, Salib RJ, Oates J. Traumatic fracture of the stapes suprastructure following minor head injury. *J Laryngol Otol.* 2002;116:457-459.
7. McGuirt WF Jr, Stoll SE. Temporal bone fractures in children: a review with emphasis on long-term sequelae. *Clin Pediatr (Phila).* 1992;31:12-18.
8. Tos M. Prognosis of hearing loss in temporal bone fractures. *J Laryngol Otol.* 1971;85:1147-1159.
9. Gulya AJ, Schuknecht HF, Ed. *Anatomy of the temporal bone with surgical implications.* 2nd ed. Parthenon Publishing, New York, London; 1995.
10. Hough JV, Stuart WD. Middle ear injury in skull trauma. *Laryngoscope.* 1968;78:899-937.
11. Swartz JD, Swartz NG, Korsvik H, *et al*. Computerized tomographic evaluation of the middle ear and mastoid for posttraumatic hearing loss. *Ann Otol Rhinol Laryngol.* 1985;94:263-266.
12. Smyth GD. The long process of the incus. *J Laryngol Otol.* 1964;78:400-407.
13. Lannigan FJ, O'Higgins P, McPhie P. The vascular supply of the lenticular and long processes of the incus. *Clin Otolaryngol Allied Sci.* 1993;18:387-389.
14. Tuz M, Dogru H, Yasan H, Doner F, Yariktas M. Incus and stapes necrosis associated with diabetes mellitus. *J Laryngol Otol.* 2006;120:E22.
15. Imauchi Y, Karino S, Yamasoba T. Acquired atrophy of the long process of the incus. *Otolaryngol Head Neck Surg.* 2005;132:156-158.
16. Esselman G, Goebel JA, Wippold FJ 2nd. Conductive hearing loss caused by hereditary incus necrosis: A study of familial expansile osteolysis. *Otolaryngol Head Neck Surg.* 1996;114:639-641.
17. McGuirt WF Jr. Injuries of the ear and temporal bone. In: Bluestone CD, Stool SE, Kenna MA, eds. *Pediatric Otolaryngology.* 3rd Ed. Saunders, Philadelphia PA; 1996:689-705.
18. Silverstein H. Trauma of the tympanic membrane and ossicles. In: Nadol JB, Schuknecht HF, eds. *Surgery of the ear and temporal bone.* Raven Press, New York; 1993:325-328.
19. Mundada PS, Dasgupta KS, Tapadiya GB. Temporalis fascia assisted ossicular arthroplasty for the dislocated incus: fascial arthroplastic ossiculoplasty. *Laryngoscope.* 2002;112:1699-1702.
20. Berendes J, Link R, Zöllner F, Eds. *Hals-Nasen-Ohren-Heilkunde in Praxis and Klinik*. Georg Thieme Verlag, Stuttgart; 1979.

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