INTRODUCTION
Penetrating injuries violating the tympanic membrane and resulting in middle ear trauma may cause minor temporary symptoms or significant debilitation. Knowledge of the anatomy of the middle and inner ear is vital to proper diagnosis and appropriate treatment of these injuries.

Initial work up of any facial nerve injury begins with a thorough history and physical exam with emphasis on the mechanisms (penetrating vs. blunt trauma), timing (progressive vs. sudden loss) and associated symptoms such as vertigo or hearing loss. In addition, otoscopic evaluation looking for canal lacerations, TM perforation or hemotympanum is key. Bedside Rinne and Weber tests can be used as the initial evaluation to estimate the type and magnitude of hearing loss. The facial muscles should be closely examined and their function classified by the House-Brackmann grading system.

Penetrating injuries are the second most common cause of perforations; these injuries are most often caused by objects such as Q-tips, bobby pins, keys and paper clips often used in attempts to clean the external ear canal.

Here we report a case of facial nerve paralysis following transtympanic penetrating middle ear trauma, a situation that has been rarely reported in the literature.

CASE REPORT
A 26-year-old woman without significant past medical history presented to our outpatient department with unilateral complete facial nerve paralysis and severe hearing loss.

One day prior to presentation the patient had experienced a transtympanic penetrating middle ear trauma when she had fallen and a long earring was pushed into the left external auditory canal. Approximately 6 hours after the trauma she noticed the onset of facial nerve weakness, subsequently progressing to complete paralysis.

Physical examination revealed a 40% superior tympanic membrane perforation, a posterior canal laceration and a House-Brackmann grade VI paralysis. Initial tuning fork examination was consistent with a conductive hearing loss.

An audiogram was performed confirming a maximal 60-dB left-sided conductive hearing loss (Figure 1). A high-resolution CT scan of the temporal bones showed significant ossicular displacement; there was no evidence of a fracture or penetrating bony spicule involving the tympanic segment of the facial nerve (Figure 2).

Figure 1. Pure-tone audiogram showing maximal conductive hearing loss; also noted was a SDS of 100% bilaterally.

Figure 2. Axial (A, B) and coronal (C, D) cuts from a high-resolution CT scan showing separation of the incudomalleal and likely incudostapedial joints, with possible stapes displacement. The incus appears to be displaced towards the tympanic segment of the facial nerve, which is grossly normal.

DISCUSSION
This case represents a rarely reported sequela of penetrating middle ear injury—facial paralysis. We postulate that either direct trauma from the earring itself or displacement of the incus into the tympanic segment of the facial nerve led to the development of edema and subsequent delayed-onset paralysis.

No specific management guidelines are available for this type of injury, so we followed a typical strategy for facial paralysis secondary to temporal bone fractures. Because the paralysis was delayed in onset and the CT showed no gross abnormalities of the nerve, we proceeded with conservative management, including high-dose steroid therapy.

Common causes of penetrating middle ear trauma include:
- Foreign bodies
- Thermal injuries (e.g. slag burns)
- Blast injuries
- Foreign bodies
- Thermal injuries (e.g. slag burns)
- Blast injuries

Typical findings are:
- Tympanic membrane perforation
- Hearing loss and tinnitus
- Vertigo
- Otorhea
- Ossicular injury
- Perilymph fistula
- Cholesteatoma (delayed)
- Facial nerve injury

Perforating trauma to the posterosuperior quadrant of the tympanic membrane specifically places a number of middle ear structures at risk because of their anatomical alignment. These include all three ossicles, the facial nerve and the oval window. Thus, dislocation at the malleoincudal articulation, incudostapedial joint, or both, is possible and commonly occurs.

The concomitant facial nerve injury described here is a rare event, but should be kept in mind as a possible finding and treated aggressively. We propose that management should be similar to that for facial paralysis secondary to temporal bone fractures from external forces.

REFERENCES

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