

Posttraumatic labyrinthitis ossificans with perilymphatic fistulization

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ABSTRACT

Labyrinthitis ossificans is fibrosis or ossification of the membranous labyrinth. Tympanogenic, meningogenic, and hematogenous etiologies are more common than trauma in the development of labyrinthitis ossificans. We present a case complaining of right-sided hearing loss and symptoms of otitis media and positional vertigo resulting from perilymphatic fistulization. Imaging revealed labyrinthitis ossificans secondary to temporal bone fracture crossing through the otic capsule.

Key words: • labyrinthitis • labyrinthitis ossificans • trauma • perilymphatic fistulization

Labyrinthitis ossificans (LO) (sclerosing labyrinthitis) is the result of the repair process following infective, inflammatory, or destructive insults to the otic capsule (1–3). The most frequent cause of LO is suppurative bacterial labyrinthitis. Trauma is very rarely a causative factor (2, 4). Temporal bone fractures crossing through the otic capsule may result in LO, sensorineural hearing loss, vestibular dysfunction, perilymphatic fistula, or cerebrospinal fluid leakage (4–7). Herein, we present a case of trauma-induced LO with sensorineural hearing loss and positional vertigo as a sign of perilymphatic fistulization.

Case report

A 33-year-old man complaining of vertigo for the last 20 days was referred for magnetic resonance imaging (MRI) for right-sided sensorineural hearing loss and otitis media. High-resolution 3D fast spin echo T2-weighted MR images were obtained for detailed visualization of the membranous labyrinth. There was decreased signal from the cochlea on the right side, and adhesive changes along the seventh and eighth nerves within the right internal auditory canal, suggestive of LO (Fig. 1).

High-resolution computed tomography (CT) of the temporal bone was performed with 1-mm contiguous axial images. Ossification within the cochlea confirmed LO and, additionally, a transverse fracture crossing the internal auditory canal and cochlea was seen (Fig. 2). The fracture appeared to be old, with sclerotic lines. There was minimal soft tissue surrounding the middle ear ossicles without any evidence of destruction, suggestive of otitis media. Further detailed history revealed that the patient had suffered a serious traumatic crush injury under a building 11 years earlier.

There was no ossification in the oval or round windows to suggest a tympanogenic etiology, and no history of bacteremia or meningitis to suggest a hematogenous route or meningogenic etiology for the development of LO. The patient had episodes of otitis media and difficulty in hearing on that side. He started having vertigo 20 days prior to presentation. The vertigo worsened with standing and Valsalva maneuver, suggesting perilymphatic fistulization. Antibiotics were given for otitis media. Conservative medical treatment was chosen for perilymphatic fistulization, although surgical closure is usually applied when there is antecedent trauma. Fortunately, the patient responded to strict bed rest, head elevation, and refraining from strenuous activity, with resolution of positional vertigo.

Discussion

LO is pathologic fibrosis or ossification of the membranous labyrinth. It is the final result of inflammatory, infectious, or destructive processes such as meningitis, middle ear infection, cholesteatoma, septic emboli,

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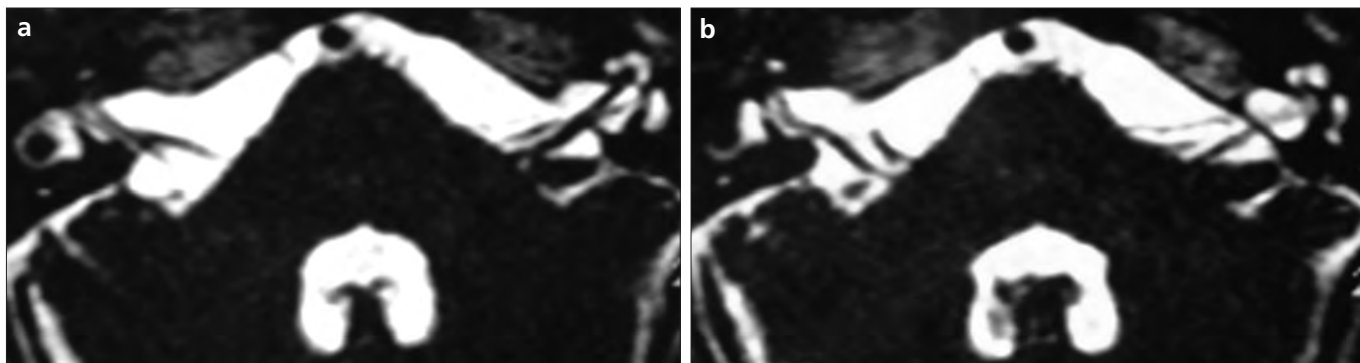


Figure 1. a, b. Axial T2-weighted MR images show T2 signal loss from the cochlea on the right. The vestibulocochlear nerve complex on the right is thinner than its counterpart, and is inseparable from its components, suggestive of atrophy and adhesion.

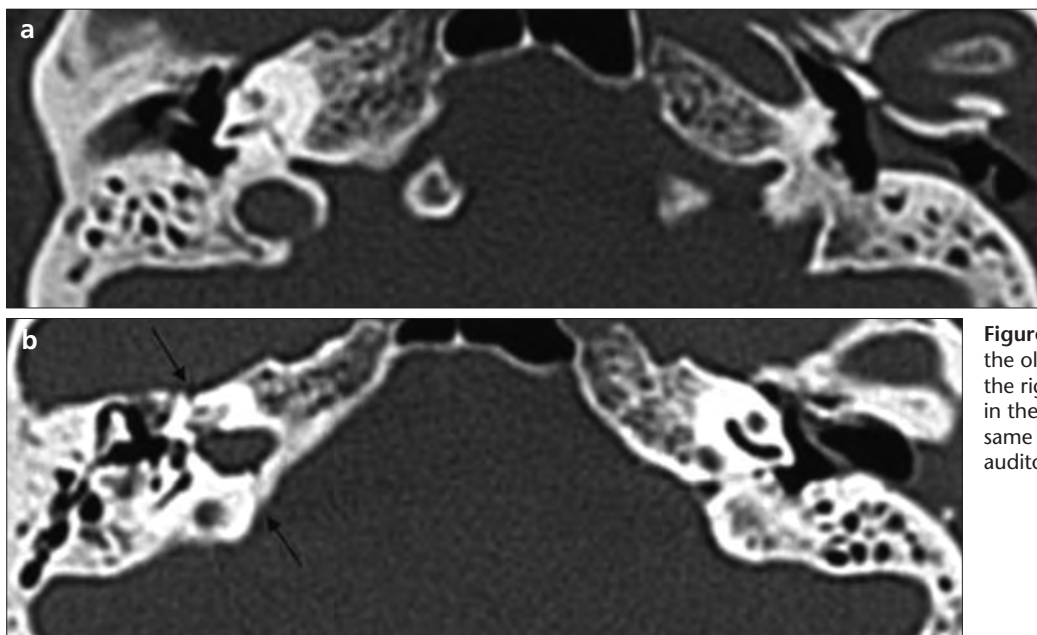


Figure 2. a, b. Axial CT images show the old fracture (*arrows, a*) crossing the right otic capsule, and ossification in the turns of the cochlea (*a, b*). The same fracture traverses the right internal auditory canal at a superior level (*b*).

viral or bacterial labyrinthitis, advanced otosclerosis, autoimmune inner ear disease, occlusion of the labyrinthine artery, previous labyrinthectomy, trauma, leukemia, or tumor of the temporal bone. The most common cause of LO is suppurative bacterial labyrinthitis following bacterial meningitis (1–3). The infection can reach the inner ear via a tympanogenic, meningogenic, or hematogenous route. Unilateral LO is usually of tympanogenic etiology, while bilateral is of either meningogenic etiology or from a hematogenous route (8). In bacterial meningitis, the infection spreads to the inner ear via the subarachnoid spaces (e.g., the cochlear aqueduct and the internal auditory canal). In chronic otitis media, it usually spreads via either the oval or the round window (3).

In the evolution of the LO, Paparella and Sugiura (9) identified three stag-

es: acute, fibrous, and ossifying. In the acute stage, purulent or serofibrous exudate fills the perilymphatic space within the membranous labyrinth, but spares the endolymphatic space between the membranous and bony labyrinths. The fibrous stage is marked by fibroblastic proliferation within the perilymphatic space, with angiogenesis. This stage begins approximately two weeks following the onset of infection. The third stage is the ossifying stage, characterized by bone formation, which can develop in the basal turn of the cochlea as early as two months after the onset of infection. The most common location for fibrosis/ossification is the basal turn of the cochlea, although the location may change according to the route of entry or spread of inflammation (3, 10). In the ossification of the basal turn of the cochlea, the spread

usually occurs through the cochlear aqueduct, while in the ossification of the first and second turns of the cochlea, spread is usually through the internal auditory canal (10). Severe LO may result in total ossification of the membranous labyrinth, which can be easily misdiagnosed as congenital otic dysplasia. Convexity of the otic capsule makes the differentiation easy, because it is preserved in LO, but reduced in congenital otic dysplasia, otherwise known as Michel deformity, or cochlear hypoplasia/aplasia (1, 8).

Temporal bone fracture is a very rare cause of LO. Complications of fractures of the temporal bone are sensorineural or conductive hearing loss, cerebrospinal fluid leakage, facial nerve weakness, vestibular dysfunction, and perilymphatic fistula. Fractures can be longitudinal, transverse, or mixed on the basis of their orientation relative to the

long axis of the petrous temporal bone. Longitudinal fractures cross the middle ear and are often associated with ossicular dislocation, conductive hearing loss, and facial nerve palsy. Transverse fractures traverse the fundus of the internal auditory canal or bony labyrinth, resulting in sensorineural hearing loss (1, 4, 6, 8). A new classification categorizes the fractures into two types: fractures violating the otic capsule, and fractures sparing the otic capsule. Fractures violating the otic capsule usually result in sensorineural hearing loss, vestibular dysfunction, perilymphatic fistula, or cerebrospinal fluid leakage (4, 5). Sometimes, even without fracture lines through the otic capsule, neural structures may be destroyed by concussion alone (7).

In our patient, LO is the result of an old temporal bone fracture crossing through the otic capsule and internal auditory canal. The patient had a history of bouts of otitis media following the traumatic event, and soft tissue surrounding the ossicles, but there was no ossification in the round or oval window that would support the theory that the chronic otitis media might have played a role in the development

of LO. He had also vestibular dysfunction. His vertigo could be due to LO itself, or to perilymphatic fistula secondary to fracture through the otic capsule. The positional vertigo sustained by the patient in the last 20 days was suggestive of perilymphatic fistula. There were no other signs of leakage, such as pneumolabyrinth, dependent fluid in the tympanomastoid cavity, enhancement of the membranous labyrinth, or meningitis (1, 11).

In conclusion, among the many causes of LO, trauma is very rare. In radiologic evaluation of LO, high-resolution CT and MRI of the temporal structures are complementary. CT enables the visualization of bony labyrinth, fractures through the temporal bone, ossification of bony labyrinth, and pneumolabyrinth. MRI, especially heavily T2-weighted images, enables the visualization of the membranous labyrinth and vestibulocochlear nerve complex. MRI is better than CT in the depiction of the acute and fibrotic stages of LO.

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