

Brain herniation into the middle ear cavity: MR imaging

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Summary. A CT and MR study of a case of brain hernia within the middle ear cavity is reported. MR allows differentiation between brain tissue and other abnormalities of the ear with a similar appearance on CT.

Key words: CT-MR imaging - Brain hernia - Cholesteatoma - Cholesterol granuloma

Brain hernia within the temporal bone, also referred to as brain fungus, brain prolapse, endaural encephalocele or fungus cerebri was not unusual in the pre-antibiotic era as a complication of mastoid surgery associated with brain abscess. Meningeal complications are now rare and little reference is made in the recent and almost exclusively otologist literature [1-6]. This case report describes CT and MR characteristics of a post-surgical lesion and their correlation with surgical findings.

Case report

A 25-year-old man had a complex history of bilateral chronic otitis media with previous otologic surgery that included in childhood a left open radical mastoidectomy for cholesteatoma and a right functional mastoidectomy. He also had since childhood a long history of epilepsy (petit-mal seizures followed 9 years ago by grand-mal seizures) for which he is now treated by Carbamazepine. He later presented recurrent meningitis: the first meningococcal meningitis in 1985, and a second pneumococcal one in February 1988.

Otological examination revealed a normal right eardrum with subnormal hearing.

CT scans (Fig. 1) showed a defect of the tegmen tympani with a non contrast-enhanced soft-tissue mass filling the attic cavity. The ossicular chain, especially the heads of the malleus and the incus was surrounded by the mass without displacement or erosion.

On MR, the lesion appeared as a continuous mass from the brain through the tegmen; on sagittal (Fig. 2) and coronal T1-WIs, the mass depicted an isointense signal relative to the brain, with a T2-WIs, a small external underlying hyperintense area (Fig. 3).

Surgery with a combined transmastoid-middle crania fossa extradural approach disclosed a bony defect in the tegmen tympani with a 1 cm-long dural defect through which appeared a temporal lobe herniation. The brain hernia surrounded by an arach-

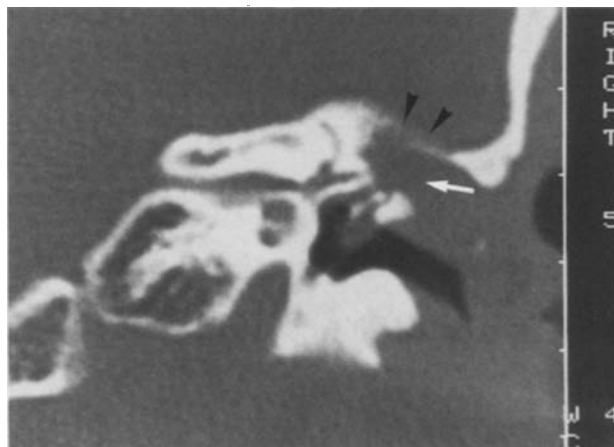


Fig. 1. Post contrast CT scan: erosion of the tegmen tympani (black arrows). A non contrast-enhanced soft-tissue mass (white arrow) fills the attic cavity without ossicular chain erosion

noid covering, filled the attico-antral cavity. Peripheral inflammatory tissue located in the attic at the anterior part of the hernia was removed. No CSF leakage was noted. After reducing the hernia, the defects were grafted with dura and temporalis fascia. A small resected area of brain presented no microscopic abnormality. Postoperative hearing was not modified.

Discussion

Brain herniation into the middle ear or mastoid cavities may be spontaneous, related to congenital bone defects, or posttraumatic; however, infection and

surgery for chronic ear infection continue to be the major cause of brain hernia [1-9].

The pathophysiology of brain herniation remains poorly understood. It is frequently associated with surgical trauma, but it seems that isolated chronic low grade infections involving the mastoid and middle ear can lead to destruction of the tegmen with extension to the dura, followed by involvement of adjacent brain with localized increased intracranial pressure and hernia. So, combinations of trauma and infection could explain the majority of brain extrusions [1, 6]. On the other hand, Ahren has shown a high incidence (21%) of dehiscences in the tegmen area in 94 cadavers, providing an easy pathway for the spread of infection [6].

Through the defect of the tegmen of which the size may vary from a few millimeters to several centimeters, brain hernia may appear pedunculated as in our case, or extensive throughout the mastoid, mixed with granulations [1, 2]. Absence of dural covering is the rule, the hernia thus not appearing as a true encephalocele; however arachnoid can follow the hernia for varying distances [1, 6]. The extruded tissue, contrary to our case, is most often composed of chemically infected, necrotic brain, and is usually considered as non functional [1, 2, 9].

Of the cases previously described in the literature, the presenting clinical picture was primarily that of chronic otitis media and chronic mastoiditis. Progressive conductive hearing loss is the most common symptom. CSF otorrhea or rhinorrhea can occur, however they are most frequently observed with congenital defect, and with occasionally recurrent meningitis [1-4, 6-9]. Seizure activity, most often as temporal lobe attacks, has also been reported [3, 6, 7, 9]. Otoscopic examination can reveal a soft, whitish, pulsatile mass [2, 5, 6, 9]. However brain hernia can also simulate cholesterol granulomas with a blue eardrum [3, 10].

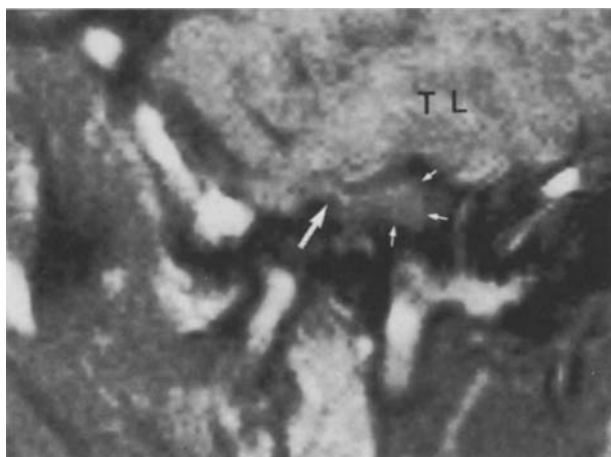
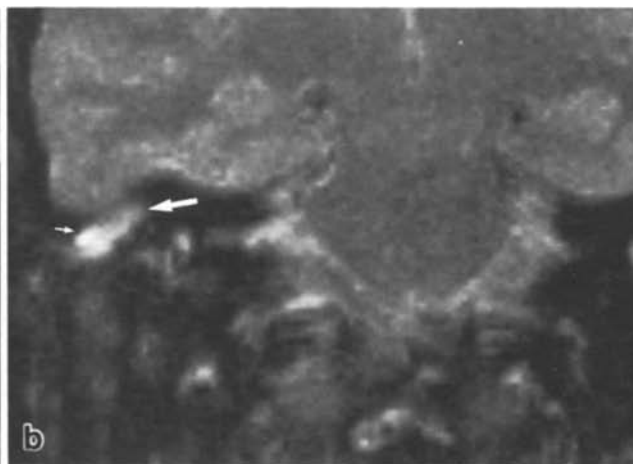
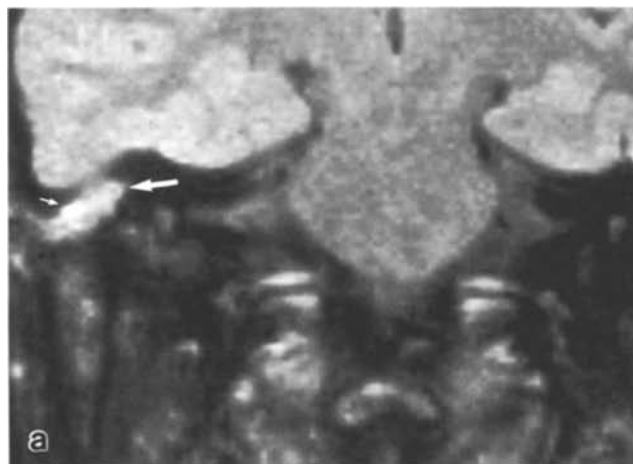


Fig. 2. Sagittal T1-weighted, (SE 420/30) 4 mm thick, MR image (short arrows). The mass initiates a signal similar to grey matter, and is continuous with the adjacent temporal lobe (TL) (arrow)

Fig. 3a, b. Coronal T2-weighted, (SE a 1700/60; b 1700/120) 4 mm thick, MR image: the mass exhibits a signal similar to that of the adjacent brain (arrow), underlined by a small hyperintense area (short arrow)



The surgical literature [4, 6-9] has emphasized the necessity of a combined transmastoid-middle cranial fossa extradural approach to extrude the brain prolapse and repair the conjoined usually dural and bony tegmen defects. So, an accurate characterization is crucial in planning surgical approaches, particularly with secondary cholesteatomas and cholesterol granulomas which can result as well from chronic middle ear infections with a similar clinical appearance, and can be variously associated [1, 6].

In the few previous reports, CT was found to detect the bony tegmen abnormality, with soft-tissue mass filling the middle ear or the mastoid, but not to differentiate these features from non contrast-enhanced cholesteatoma or granulation tissue [2, 7, 9]. CT together with surgical findings can show erosion of the ossicular chain and of the fallopian canal; the heads of the ossicles can be enveloped by the hernia, sometimes with a dislocation, but as in our case, the integrity of the ossicular chain was not helpful since occurring also with the other soft-tissue masses such as cholesterol granuloma and even cholesteatoma [6, 7].

To our knowledge, the MR appearance has not been previously reported; in our case, as suggested by Bowes [9], MR can specifically demonstrate brain hernia as a continuous mass from the brain, and of the same signal. In contradistinction, the recently reported MR appearance of cholesteatomas consists of an iso- or hypointense signal on T1-WIs with a high signal intensity on T2 images, while cholesterol granulomas have been shown as hyperintense areas on both T1- and T2-WIs [9, 11-13]. The hyperintense area underlying the hernia (Fig.3) is compatible with the surgically proven localized granulation tissue often found in association with the hernias.

MRI consequently appears very useful in differentiating brain hernia from other pathologic processes with similar clinical and/or CT features. However, subtle bone abnormalities of the ossicular chain and the tegmen are better detected by CT.

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Received: 10 January 1989

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