Abstract. Cholesterol granuloma (CG) may erode into the middle ear, the mastoid bone and the petrous apex. However, aggressive erosion into the cranial cavity is extremely rare. Here we report a case of huge CG extending to the middle cranial fossa. Temporal bone computerized tomography showed a soft tissue mass which destroyed the bony plate of the posterior and middle cranial fossa. On magnetic resonance imaging, the mass revealed a high signal on both T1 and T2-weighted images. The mass compressed the middle cranial fossa without invasion into the brain. The CG was removed by extended cortical mastoidectomy. The postoperative course was uneventful and there were no neurological complications.

Cholesterol granuloma (CG) of the middle ear typically presents with a conductive hearing loss and a blue eardrum; those at the petrous apex either manifest with side-effects from bony erosion (with sensorineural hearing loss, tinnitus, vertigo or cranial nerve impairment), or are identified as incidental findings (1). CG can be a perfectly localized and isolated mass in any pneumatized area in the temporal bone, the middle ear cavity (2, 3), mastoid antrum (4), external auditory canal (5) and the petrous apex (1). CG has been found in as many as 12-20% of temporal bones with chronic otitis media (2-4). According to a histopathological study, CG has been identified in 12% of patients with chronic otitis media with intact tympanic membranes and in 21% of a group of patients with a perforated tympanic membrane (6). Cholesterol granuloma may erode into the middle ear, the mastoid bone and the petrous apex. However, aggressive erosion into the cranial cavity is extremely rare (7, 8). Here a case of huge cholesterol granuloma extending to middle cranial fossa is presented.

Case Report

A 53-year-old woman had right otitis media with hearing difficulty since childhood. Her hearing gradually became worse. On otomicroscopic examination, the right tympanic membrane showed attic retraction. An audiogram revealed a conductive hearing loss of 31.5 dB in the right ear. Temporal bone computed tomography (CT) showed a soft tissue mass which had destroyed the bony plate of the posterior and middle cranial fossa (Figure 1). Pneumatization of both temporal bones was poor. On magnetic resonance imaging (MRI), the mass revealed a high signal on both T1 and T2-weighted images. The mass compressed the middle cranial fossa without invasion into the brain (Figures 2 and 3). Extended cortical mastoidectomy showed the huge cholesterol cyst to have a dark chocolate-colored effusion. The cyst wall of the cholesterol granuloma was removed from the dura by gentle dissection using cottonoid. Histologically, the lesion showed typical features of a cholesterol granuloma with numerous cholesterol clefts surrounded by foreign-body giant cells embedded in granulation tissue (Figure 5). The postoperative course was uneventful and there were no neurological complications. There has been no recurrence of the cholesteatoma for over 20 months after surgery.

Discussion

CGs, first reported in the mastoid and middle ear in 1894, may occur anywhere in the air cell system of temporal bone when eustachian tube obstruction, mucosal edema, temporal bone fracture, cholesteatoma, chronic otitis media or any another process blocks the air cell tracts. They are thought to be caused by a foreign-body reaction to cholesterol crystals released by the breakdown of blood and local tissue. CG of the middle ear typically presents with a conductive hearing loss and a blue eardrum; those of the petrous apex either manifest themselves as a consequence of bony erosion (with sensorineural hearing loss, tinnitus, vertigo or cranial nerve compression) or as incidental findings (1).
CG may erode into the middle ear, the mastoid bone or the petrous apex. However, aggressive erosion into the cranial cavity is extremely rare (7, 8). The vast majority of middle ear and mastoid CG conforms to the anatomic compartment in which they have arisen and do not erode adjacent bone. Maerta et al. reviewed their 11 series of CGs in young patients and observed that there was no evidence of any extension of disease beyond the middle ear and mastoid cavity or of any aggressive bony involvement. The reasons for the aggressive extension of CG are unknown. Martin et al. (8) suggested that distinction between benign and aggressive CG is often macroscopically self-evident: the benign variant consists of an agglomeration of cholesterol-rich fluid that fills available potential space and is amenable to simple evacuation, whereas the aggressive type is organized into a cyst with a thick wall of inflammatory tissue.
and may more properly be labeled a ‘granuloma’. Recently, Pfister et al. speculated that the difference in aggressiveness between CGs is the result of variability in the vigor of their vascular source. Actually, CG of the petrous apex appears to have a more aggressive nature.

Initial radiological investigation is performed by temporal bone CT scan, however CT appearance may be indistinguishable from those of cholesteatoma. The MRI characteristic of CG is helpful for its differentiation from cholesteatoma or other diseases. CG characteristically gives a high signal intensity on both T1- and T2-weighted MRI images, owing to the paramagnetic effect of hemoglobin breakdown products derived from microhemorrhages around the crystals.

With regards to treatment of CG, a variety of approaches ranging from drainage to surgical extirpation are plausible. In the present case, an extirpation and obliteration were performed. The postoperative course was uneventful and there were no neurological complications.

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References


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