
CASE REPORT**Unusual multifocal cholesterol granuloma following canal wall-up mastoidectomy***Mohammad Naksh Kamar¹, Apurva Anil Jarandikar^{1*}**¹ Department of ENT, Dr. D. Y. Patil Medical College, Hospital and Research Centre,
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Abstract

This case report outlines an unusual multifocal presentation of Cholesterol Granuloma (CG), a lesion typically found in the petrous apex, middle ear, or mastoid process. A 52-year-old male, with a history of right canal wall-up mastoidectomy 4 years earlier, presented with right ear pain and was initially suspected to have recurrent cholesteatoma based on otoendoscopy and high-resolution computed tomography findings. Surgical exploration led to the identification of a cyst simultaneously involving the external auditory canal, attic, middle ear, and mastoid antrum, which histopathology confirmed as cholesterol granuloma. Complete excision with creation of a single, well-aerated mastoid cavity resulted in an uneventful recovery. This case underscores that cholesterol granuloma must be considered as a differential diagnosis in a postoperative ear presenting with a mass, even if there is simultaneous involvement of multiple sites or the presence of a disease free petrous apex.

Keywords: Cholesterol granuloma, Mastoidectomy, Multifocal, Postoperative, Differential diagnosis

Introduction

Cholesterol Granulomas (CG) have been classically defined microscopically as giant cell reactions surrounding cholesterol clefts which are encapsulated by a fibrous capsule. They are expansile lesions of the temporal bone, occurring typically in the petrous apex, mastoid air cells, antrum, or the middle ear cavity. Although rare, CG has been reported in isolated sites in literature [1]. The simultaneous multifocal involvement of the External Auditory Canal (EAC), attic, middle ear, and mastoid antrum is highly unusual, especially in postoperative cases.

Case Report

A 52-year-old male came to our department with complaints of right ear pain that had started 1 month back. He had a history of right ear canal wall up mastoidectomy done 4 years back. He had no history of postoperative complications. As seen on otoendoscopy (Figure 1), the right ear EAC showed sagging and bulging of the posterior wall

and roof throughout the EAC, causing partial obliteration of the EAC. The mass was soft to touch. The tympanic membrane was partially visible and intact.

Pure tone audiometry reported moderate degree conductive hearing loss for the right ear. High-resolution computed tomography temporal bone reported “soft tissue density completely obliterating the inner third of the EAC, attic, and remaining mastoid antrum-likely a recurrence of cholesteatoma (Figure 2). The right middle ear showed minimal mucosal thickening, and the ossicular chain was not visualised.”

The patient underwent right ear revision type 3 cartilage tympanoplasty with mastoid exploration under general anaesthesia. The bulge in the EAC was aspirated partially by a 21G needle and a yellowish-brown glistening sticky fluid was aspirated, which was sent for Gram stain and culture sensitivity. The fluid showed no microbial growth.

This was consistent with a non-infectious process, in contrast to reports showing that the large majority of ear discharge samples in chronic middle ear disease yield positive bacterial cultures [2]. Intra-operatively, we found a cyst lined by a smooth membrane lying in close adherence with the posterior wall and roof of the EAC. It extended into the mastoid antrum, attic, and middle ear. The cyst was exposed, excised completely, and sent for histopathology. A single large mastoid cavity was made with adequate lowering of the canal wall and exenteration of the remaining air cells.

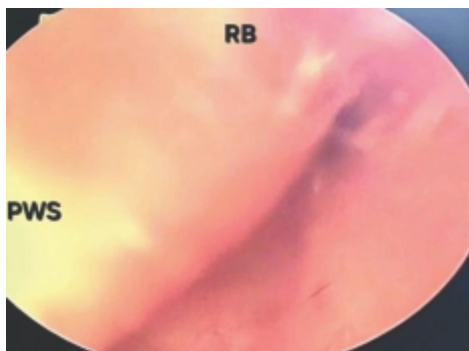


Figure 1: Otoendoscopy showing PWS- posterior canal wall sagging & RB- bulge in the roof of EAC

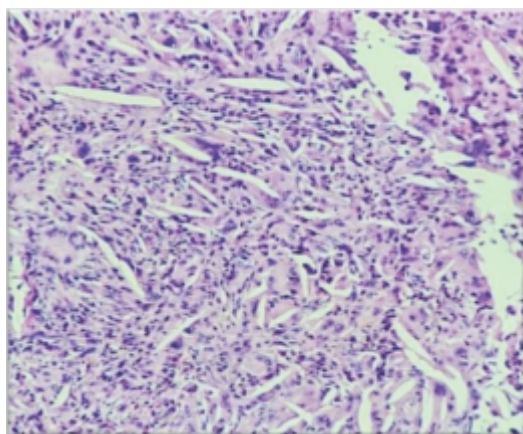


Figure 3: HPE report of cholesterol granuloma

Histopathology (Figure 3) showed “fibro collagenous tissue densely infiltrated by lymphocytes and histiocytes containing haemosiderin pigment. Many foreign giant cells and cholesterol clefts were seen.” Histopathology confirmed cholesterol granuloma.

Postoperative recovery was uneventful, and otoendoscopy showed a healthy neotympanum after 3 months (Figure 4).

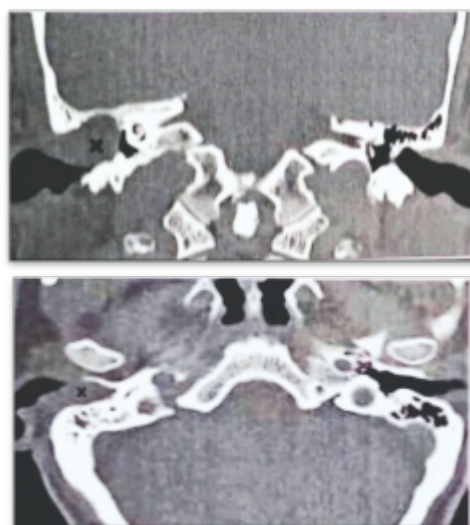


Figure 2: HRCT temporal bone showing soft tissue (marked with x)

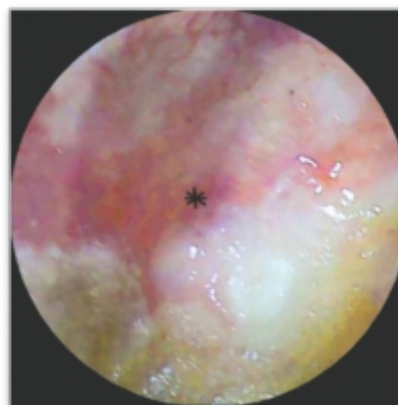


Figure 4: Successful uptake of graft seen postoperatively (Neotympanum marked with *)

Discussion

Classically, CG formation is explained by a mucosal obstruction in the temporal bone leading to air getting trapped within without any aeration. Departing from the traditional obstruction-vacuum hypothesis, Jackler & Cho (2003) argued that attributing the condition to hypoventilation would imply that nearly all patients with Eustachian tube obstruction should develop CG [3]. They introduced the exposed marrow theory as another possible mechanism. The bone-wax foreign body reaction explained by Wong *et al.* (2020) [4] and the post-surgical air cell isolation mechanisms proposed by Bizakis *et al.* (2000) and Khairi & Ramza (2012) are examples of more recent theories [5-6].

Shambaugh (1929) proposed *idiopathic hemotympanum* as a precursor to the development of cholesterol granuloma in the middle ear [7]. Matt *et al.* (1990) defined the classic CG as a blue mass medial to the tympanic membrane [8]. In our case we found the rare presentation of the CG simultaneously involving the EAC, attic, middle ear, and mastoid antrum in a postoperative patient. The usual differential diagnosis of CG includes osteoma, meningocele, encephalocele, glomus, high jugular bulb, hemotympanum. In previously operated ears, atypical postoperative temporal bone pathology may further broaden the differential [9]. As the petrous apex, the usual site of CG, was disease-free, our preoperative diagnosis became even more challenging. While the imaging findings were consistent with cholesteatoma, the cystic lesion with a soft consistency and the absence of bony destruction

raised concern for CG. MRI may differentiate between these entities based on signal characteristics, where the CG would show high T1/T2 signal, while cholesteatoma may show variable T1 signal. However, in our case, due to financial constraints, this was not possible.

Recently, complete cyst excision surgery with removal of all potential isolated spaces and providing good permanent aeration with drainage has proved to be most effective [10]. In our case, the approach was customized according to the hearing status and altered anatomy, with meticulous clearance of the lesion and remnant air cells to create a single mastoid cavity.

Conclusion

Our knowledge of CG has changed over the last thirty years, moving from discrete case reports to more extensive surgical series and developing etiological theories. Although novel ideas like the exposed marrow hypothesis and bone-wax reaction offer insightful information, the persistent finding of postoperative CG, frequently occurring 6–9 years after surgery, points to a shared underlying mechanism. Permanent drainage and aeration are necessary for long-term success, and surgical intervention remains the mainstay of treatment. CG must be considered a differential in postoperative cases even if the usual sites are uninvolved. Validating more recent pathophysiological models and improving recurrence prevention techniques should be the goals of future research.

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