

# Cerebellopontine Angle Cholesterol Granuloma: A Case Report

M. Hariz Shahrudin<sup>1</sup>, Nur Hamizah<sup>2</sup>

<sup>1,2</sup>Doctor, Department of Otorhinolaryngology, Hospital Shah Alam

## Abstract

Cholesterol granulomas are benign growths that occur due to an inflammatory response to cholesterol and hemosiderin. These lesion typically originate in the temporal bone and middle ear, although cerebellopontine angle cholesterol granuloma is exceptionally rare. This report describes a rare occurrence of a cholesterol granuloma developing in the cerebellopontine angle.

The patient exhibited unilateral hearing impairment in the left ear accompanied by tinnitus. High resonance computed tomography revealed a lobulated lesion situated at the right cerebellopontine angle, which is also causing erosion of the right mastoid bone, right posterior wall semicircular canal, and right jugular foramen. Magnetic resonance imaging demonstrated hyperintensity with peripheral hypointense hemosiderin rim on both T1-weighted and T2-weighted images.

According to the authors' understanding, this is the third documented instance of cholesterol granuloma manifesting as a tumour in the cerebellopontine region. The authors provide a comprehensive description of the uncommon lesion, including its presentation, imaging findings, histological characteristics, pathophysiology, and surgical therapy.

**Keywords:** Cholesterol granuloma, Cerebellopontine angle

## Introduction

A cholesterol granuloma is a benign, inflammatory growth that usually develops in the middle ear. The condition is distinguished by the buildup of cholesterol crystals inside a fibrous capsule. The precise aetiology of cholesterol granulomas remains incompletely elucidated, however it is hypothesised to be associated with persistent inflammation and the occlusion of the middle ear drainage routes. This can arise due to persistent ear infections, dysfunction of the Eustachian tube, or the existence of a cholesteatoma.

## Case Report

The patient is a 27-year-old gentleman who has been experiencing right ear hearing loss and ringing sounds for the past 2 weeks. He has a history of chronic otitis media and underwent a myringoplasty operation in 2015. Otoscopic examination revealed an intact tympanic membrane, a clear external auditory canal, and no discharge. Pure-tone audiometry testing showed profound sensorineural hearing loss with no response at frequencies ranging from 500 Hz to 8000 Hz. The acoustic evaluation of middle ear functions was normal, as the tympanometry test showed type A.

The high-resonance computed tomography (HRCT) of the temporal bone revealed a lobulated lesion located at the right cerebellopontine angle, which also involved the mastoid bone. The dimensions of the

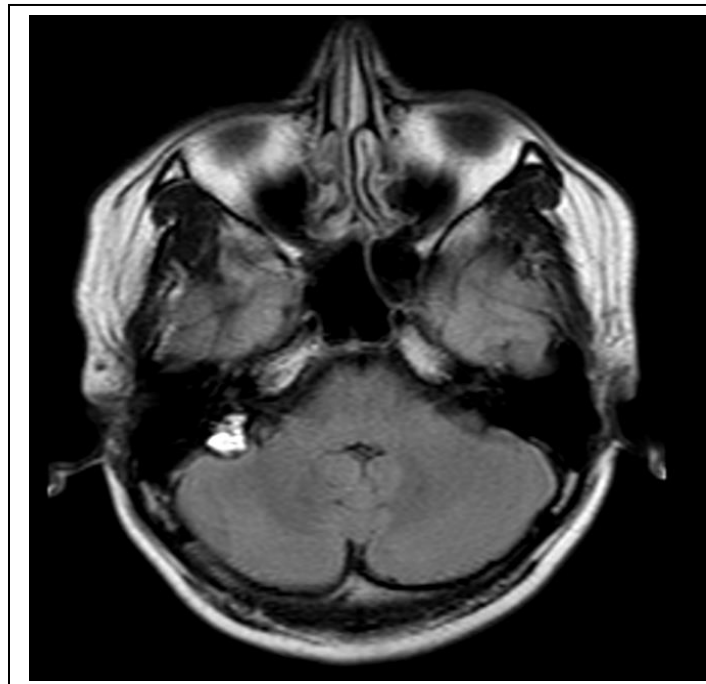
lesion were approximately 1.1 x 1.3 x 1.6cm (APxWxCC). Antero-superiorly, there is associated erosion of the right mastoid bone and right posterior semicircular canal noted on HRCT. Posteriorly, it is mildly compressing onto the adjacent right cerebellar hemisphere; however, there is no focal oedema within the right cerebellum. Inferiorly, the lesion is abutting the right jugular bulb. Erosion of the right jugular foramen wall was noted. Laterally, there is no extension into the right middle ear cavity.

On magnetic resonance imaging (MRI), the lesion showed hyperintensity with a peripheral hypointense hemosiderin rim on T1-weighted and T2-weighted images (Fig1). A high signal is seen on fluid-attenuated inversion recovery (FLAIR) (Fig 2). It shows a blooming artefact on gradient echo sequences (GRE) due to the hemosiderin rim. Loss of normal T2W signal noted in the right inner ear structures, corresponding to HRCT findings of abnormal density within the basal and middle turns of the right cochlea, as well as the right semicircular canals. These features suggest right labyrinthitis ossificans, which is likely the cause of hearing loss. On HRCT of the right temporal, the right middle ear cavity is clear. The right facial canal is preserved. Internal auditory meati (IAM) are normal in calibre and symmetrical bilaterally; there is no focal IAM enlargement or erosion. Cranial nerves VII and VIII appear normal bilaterally. The diagnosis of cholesterol granuloma was made in light of these findings.



**Figure 1 MRI of a cholesterol granuloma**

(a)coronal T1W (b) axial T2 demonstrate hyperintensity with a peripheral hypointense hemosiderin rim



**Figure 2 MRI of a cholesterol granuloma**

A high signal is seen on fluid-attenuated inversion recovery (FLAIR)

### **Discussions**

In 1894, Manassé recorded the existence of cholesterol crystals within the granuloma, suggesting a potential correlation between cholesterol metabolism and the formation of this pathological condition<sup>1</sup>. The aetiology of cholesterol granuloma development continues to be a subject of continuing discussion and discordance<sup>2</sup>.

There are two ideas that elucidate the formation of petrous apex cholesterol granulomas. Originally, it was believed that these were caused by the obstruction of air cells, resulting in a decrease in pressure and the seepage of fluid into the mucosa. Extended inflammation leads to the deterioration of the mucosa, leading to haemorrhaging into the air cells. The breakdown of blood products initiates an inflammatory response, resulting in the development of granulomas. This ongoing process leads to bone degradation<sup>3</sup>.

Jackler RK et al. propose a hypothesis called the "expose marrow" hypothesis. As per the notion, the marrow-filled spaces in the petrous apex erode as the air cells form, causing the occurrence of subacute bleeding. This bleeding induces an inflammatory response and the formation of cysts, which subsequently enlarges and leads to more haemorrhaging from the marrow cavities<sup>4</sup>.

Cholesterol granuloma very rarely occurs in the cerebellopontine angle. So yet, only two cases have been documented. Lunardi et al. documented a case featuring cerebellar syndrome and neurological symptoms<sup>5</sup>. House and Brackmann witnessed the destruction of the petrous apex, indicating a possibly severe and progressed state of cerebellopontine angle cholesterol granuloma<sup>6</sup>.

CT scans reveal cholesterol granulomas as distinct, non-enhancing, expanding masses accompanied by bone weakening and erosion<sup>7,8,9</sup>. On magnetic resonance imaging (MRI), the presence of haemoglobin breakdown byproducts results in increased signal intensity on both T1- and T2-weighted MRI sequences. Additionally, there is increased signal intensity on fluid-attenuated inversion recovery (FLAIR) MRI, whereas diffusion-weighted MRI shows lower signal intensity<sup>7,8,9</sup>. There is no sign of a central

enhancement after the administration of gadolinium. The T2-weighted pictures display a peripheral ring that appears hypointense, indicating the presence of hemosiderin deposits<sup>7,8,9</sup>.

The identification of these distinctive features can aid in differentiating cholesterol granulomas from other prevalent abnormalities, including cholesteatomas, petrous apex effusions, mucoceles, cysts, apical petrositis, and tumours<sup>8,9</sup>.

Cholesterol granulomas are pathologically characterised by an inflammatory response to the by-products of haemoglobin breakdown found extravascularly<sup>10</sup>. The microscopic study of the cholesterol granuloma showed the presence of several cholesterol clefts, macrophages, chronic inflammation, and hemosiderin pigment<sup>11</sup>.

Cholesterol granulomas are treated surgically, and these lesions can be removed using a number of surgical techniques. For cerebellopontine angle cholesterol granuloma in particular, House and Brackman performed translabyrinthine exploration to remove the mass, which also resulted in the preservation of neurological functions<sup>6</sup>. Lunardi et al. reported surgical removal of granuloma by using posterior fossa approach, however recurrence occur after a year<sup>5</sup>.

## Conclusion

Cerebellopontine angle cholesterol granulomas are very rare. Radiological examinations encompass computed tomography (CT) and magnetic resonance imaging (MRI). Surgical treatment is necessary.

## References

1. Manasse P. Uber granulations geschwülste mit fremdkNrperriesenzellen. Virchows Arch 1894;136:245.
2. Miura M, Sando I, Orita Y, Hirsch BE. Histopathologic study of the temporal bones and Eustachian tubes of children with cholesterol granuloma. Ann Otol Rhinol Laryngol 2002;111:609/15.
3. DiNardo LJ, Pippin GW, Sismanis A. Image-guided endoscopic transsphenoidal drainage of select petrous apex cholesterol granulomas. Otol Neurotol 2003; 24:939–941.
4. Jackler RK, Cho M. A new theory to explain the genesis of petrous apex cholesterol granuloma. Otol Neurotol 2003;24:96—106.
5. Lunardi, P., Missori, P., Salvati, M., & Licastro, G. (1992). *Cholesterol granuloma of the cerebello-pontine angle. Clinical Neurology and Neurosurgery*, 94(3), 265–268. doi:10.1016/0303-8467(92)90102-9
6. House, J. L., & Brackmann, D. E. (1982). *Cholesterol Granuloma of the Cerebellopontine Angle. Archives of Otolaryngology - Head and Neck Surgery*, 108(8), 504–506. doi:10.1001/archotol.1982.0079056
7. Hoa, M., House, J. W., Linthicum, F. H., & Go, J. L. (2013). *Petrous apex cholesterol granuloma: pictorial review of radiological considerations in diagnosis and surgical histopathology. The Journal of Laryngology & Otolaryngology*, 127(04), 339–348. doi:10.1017/s0022215113000091
8. Royer, M. C., & Pensak, M. L. (2007). *Cholesterol granulomas. Current Opinion in Otolaryngology & Head and Neck Surgery*, 15(5), 319–322. doi:10.1097/moo.0b013e3282a08f74
9. Chapman, P. R., Shah, R., Curé, J. K., & Bag, A. K. (2011). *Petrous Apex Lesions: Pictorial Review. American Journal of Roentgenology*, 196(3\_supplement), WS26–WS37. doi:10.2214/ajr.10.7229

10. Rinaldo A, Ferlito A, Cureoglu S, et al. Cholesterol granuloma of the temporal bone: a pathologic designation or a clinical diagnosis? *Acta Oto-Laryngologica* 2005; 125:86–90.
11. Grossi, P. M., Ellis, M. J., Cummings, T. J., Gray, L. L., Fukushima, T., & Sampson, J. H. (2008). Cholesterol granuloma of the lateral ventricle. *Journal of Neurosurgery*, 108(2), 357–360. doi:10.3171/jns/2008/108/2/0357



Licensed under [Creative Commons Attribution-ShareAlike 4.0 International License](https://creativecommons.org/licenses/by-sa/4.0/)