Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Otorhinolaryngology

Facial Nerve Paralysis Revealing a Petrous Apex Cholesteatoma: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i02.021 | **Received:** 06.10.2025 | **Accepted:** 29.11.2025 | **Published:** 19.02.2026

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Abstract **Case Report**

Petrous apex cholesteatoma is an uncommon entity whose clinical presentation may be subtle or misleading due to the complex anatomy of this region. We report the case of a 64-year-old man initially referred to ophthalmology for a corneal abscess secondary to lagophthalmos, in whom clinical examination revealed a right peripheral facial paralysis (House–Brackmann grade IV) with a normal otoscopic evaluation. Lack of improvement after medical therapy and rehabilitation prompted imaging. MRI demonstrated a right petrous apex lesion, hypointense on T1, intermediate on T2, with marked diffusion restriction, extending to the geniculate ganglion and the labyrinthine segment of the facial nerve. High-resolution CT confirmed erosion of the superior semicircular canal. Overall correlation of clinical and radiologic findings was highly suggestive of petrous apex cholesteatoma. This case highlights the importance of considering petrous apex lesions in unexplained facial paralysis, even when otoscopic findings are normal.

Keywords: Cholesteatoma; Petrous apex; Facial nerve paralysis; Diffusion-weighted MRI; Geniculate ganglion.

INTRODUCTION

Cholesteatomas of the petrous apex are rare lesions that pose significant diagnostic challenges. Their clinical expression depends largely on lesion topography within the petrous bone, and symptoms may be otologic, neurologic, or entirely non-specific. Facial nerve paralysis is an unusual but important presentation, especially when it occurs in the absence of otorrhea or middle ear pathology. Diffusion-weighted MRI plays a central role in differentiating petrous apex cholesteatoma from other cystic or expansile lesions of the region.

We present a case of isolated House–Brackmann grade IV facial paralysis revealing a petrous apex cholesteatoma, illustrating the diagnostic pitfalls and therapeutic considerations associated with this rare pathology.

CASE PRESENTATION

A 64-year-old man with no significant medical or surgical history, employed as a baker, was referred to ophthalmology for a right corneal abscess secondary to lagophthalmos. History revealed the onset of a progressively worsening right peripheral facial paralysis over several days, without otalgia, otorrhea, fever, or subjective hearing loss. Otoscopic, cervical, and parotid examinations were unremarkable. Facial paralysis was graded House–Brackmann IV.

The patient received oral corticosteroids with tapering, vitamin therapy, ocular protection, and facial physiotherapy, with no clinical recovery after one month.

MRI of the temporal bone and internal auditory canal demonstrated an 18-mm expansile lesion of the right petrous apex, hypointense on T1, intermediate on T2, with marked diffusion restriction, encroaching on the geniculate ganglion and the labyrinthine segment of the facial nerve, and encasing the acoustic–facial bundle. (Figure 1-2)

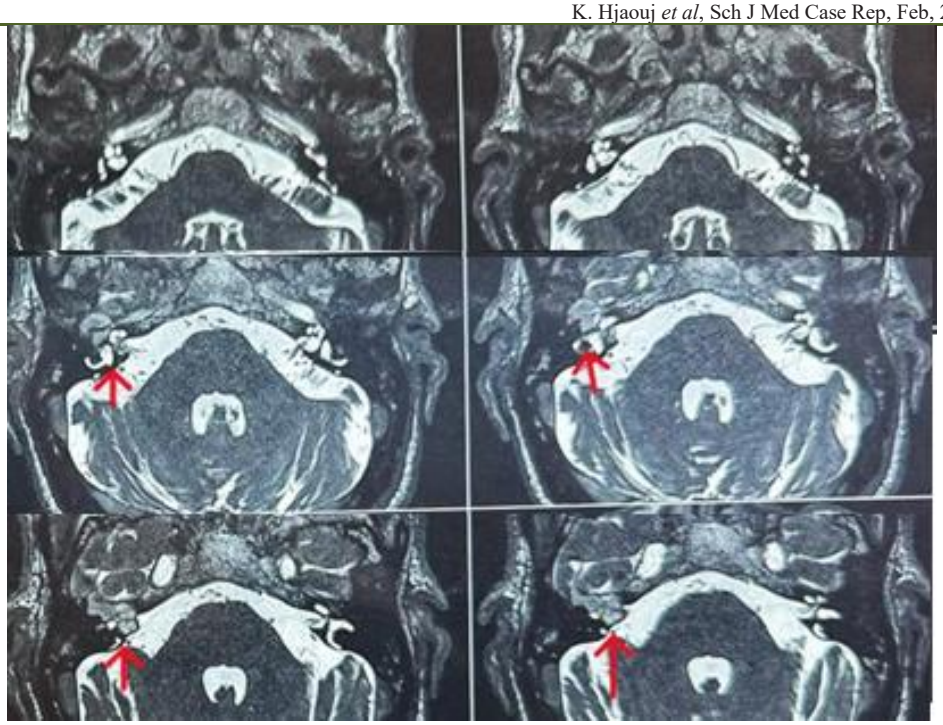

Figure 1: Temporal bone MRI – Axial T2-weighted sequence: Intermediate T2 signal lesion (arrow) involving the right petrous apex, with extension to the geniculate ganglion and the labyrinthine segment of the facial nerve

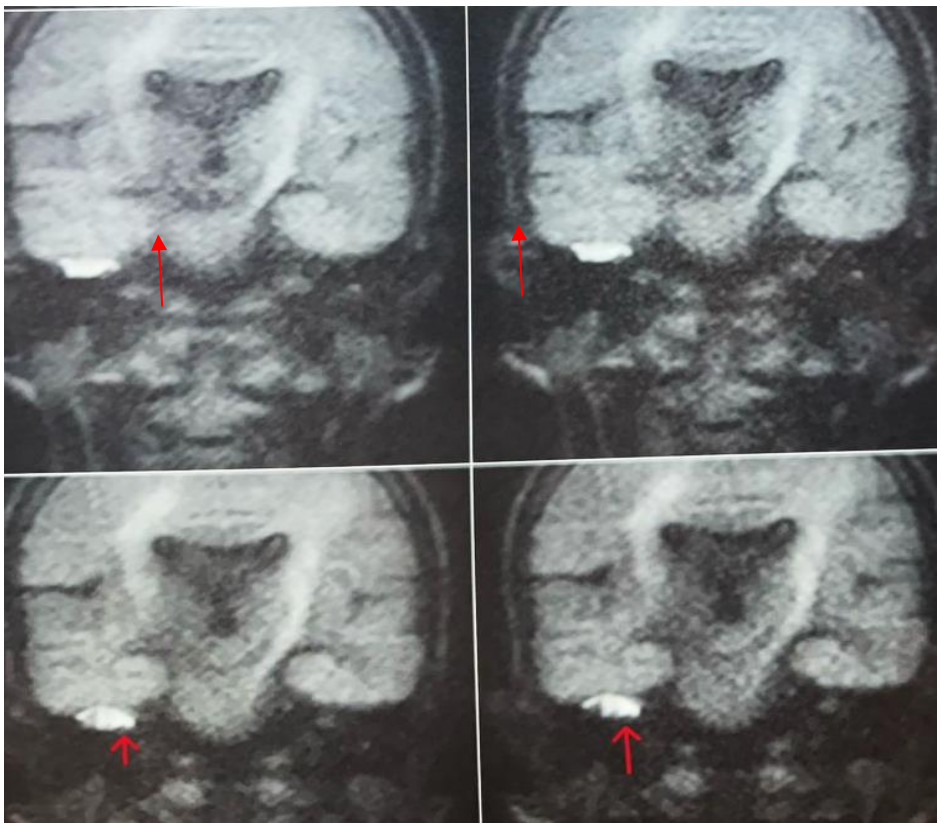

Figure 2: MRI – Diffusion-weighted sequence (DWI): Marked diffusion hyperintensity (arrow) with corresponding low ADC values, a key finding strongly suggestive of a cholesteatoma

High-resolution CT confirmed erosion of the superior semicircular canal and extension into the epitympanum with lysis of the short process of the incus and the malleus. Despite the anatomical involvement, the patient reported no vertigo, suggesting effective vestibular compensation described in chronic or slowly progressive lesions. (Figure 3)

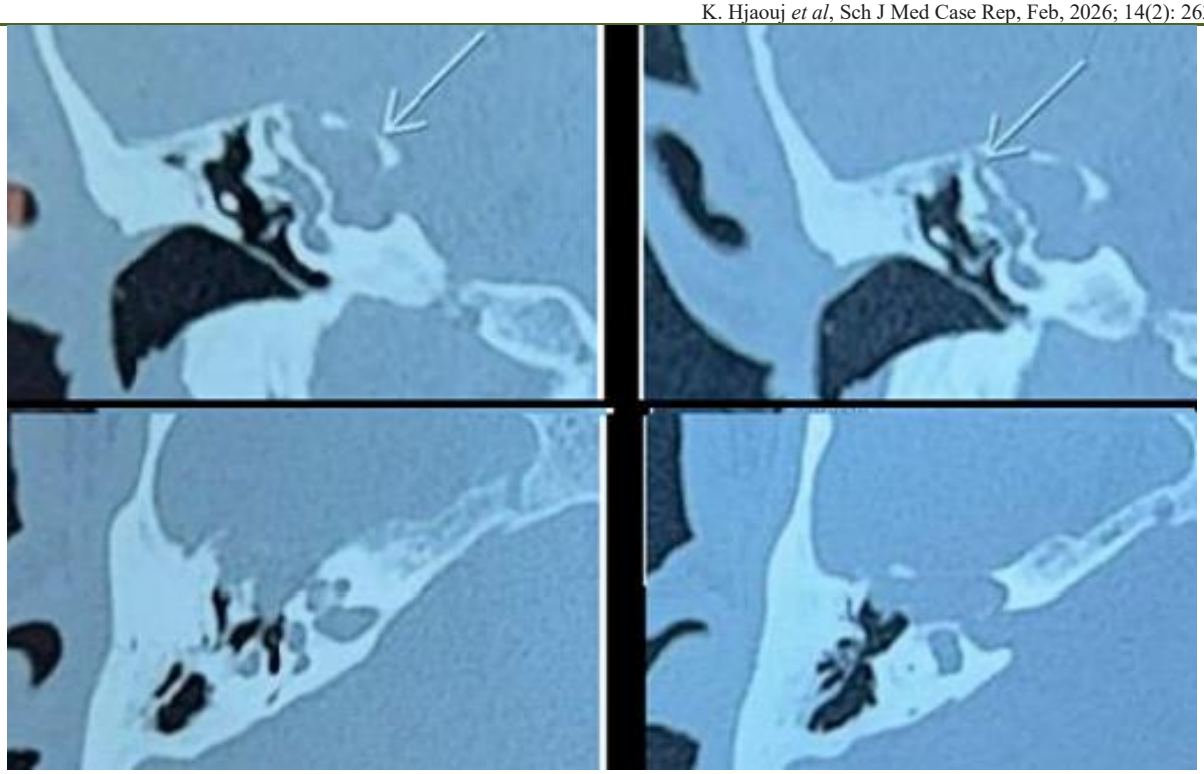

Figure 3: Temporal bone CT – Bone window: (a) Coronal view, (b) Axial view. Lytic soft-tissue process of the right petrous apex extending into the epitympanum, with erosion of the short process of the incus and the malleus. Note the lysis of the superior semicircular canal (arrow)

Electroneuromyography showed severe axonotmesis. Audiometry revealed mild right conductive hearing loss with normal tympanometry. The clinico-radiologic findings were consistent with a petrous apex cholesteatoma.

Given the involvement of the geniculate ganglion and the labyrinthine segment of the facial nerve and preserved hearing, an otologic–neurosurgical approach via the middle fossa (suprapetrous) was recommended. This route offers direct exposure of the affected facial nerve segments while sparing functional labyrinthine structures.

Surgical objectives included:

1. Complete excision of the cholesteatoma,
2. Targeted facial nerve decompression, and
3. Hearing preservation.

After detailed counseling, the patient declined surgery. Regular clinical and radiological follow-up every six months was therefore instituted.

DISCUSSION

1. Rarity and Clinical Presentation

Petrous apex cholesteatomas are rare lesions, with limited cases reported in specialized series [1]. Facial nerve paralysis is an unusual presentation and occurs less frequently than hearing loss or otorrhea. Isolated facial paralysis with normal otoscopy, as in this case, may delay diagnosis. MRI is essential to detect perineural involvement, particularly at the geniculate ganglion or labyrinthine segment [2,3].

2. Imaging Features and Differential Diagnosis

A combined CT–MRI approach is the cornerstone of diagnosis.

Cholesteatoma features:

- T1 hypointensity
- T2 intermediate/high signal
- Marked diffusion restriction (DWI positive)
- Irregular bone erosion on CT

Our patient fulfilled all the radiologic criteria: intense DWI restriction, petrous apex erosion, and extension toward the labyrinthine segment [4,5].

Differential diagnosis includes:

- **Cholesterol granuloma:** T1/T2 hyperintense, no DWI restriction [6]
- **Arachnoid cyst:** CSF-like signals, no erosion
- **Mucocele:** expansile, no DWI restriction
- **Meningioma/schwannoma:** enhancement after contrast, smooth remodeling or canal enlargement [7]

DWI is a highly reliable discriminator, with a sensitivity ≥ 90–100% for cholesteatomas [4,8].

Table 1: Differential diagnosis of petrous apex lesions

Entity	Cholesteatoma	Cholesterol Granuloma	Arachnoid Cyst	Mucocele	Petrous Meningioma	Schwannoma (VII/VIII)
Nature	Keratinizing lesion	Hemorrhagic cavity	CSF-like cavity	Trapped mucosal cavity	Meningeal tumor	Neurofibromatous tumor
T1 signal	Hypointense	Hyperintense	CSF-like	CSF-like	Iso- to hyperintense	Iso- to hypointense
T2 signal	Intermediate to high	Very hyperintense	Very hyperintense	Hyperintense	Variable	Hyperintense
Diffusion (DWI)	***Marked restriction***	***No restriction***	DWI negative	DWI negative	Variable	Variable
CT bone findings	Irregular bone erosion	Smooth expansion	No erosion	Slow erosion	Frequent hyperostosis	Enlargement of the internal auditory canal
Facial nerve (VII) involvement	Frequent (geniculate ganglion / labyrinthine segment)	Occasional	Rare	Rare	Possible compression	Frequent (VII/VIII)
Treatment	Surgical excision + facial nerve decompression	Drainage / fenestration	Observation	Surgery	Resection / radiosurgery	Microsurgery / radiosurgery

3. Therapeutic Strategy: Rationale for the Suprapetrous Approach

Complete surgical excision is the treatment of choice, frequently combined with facial nerve decompression when perineural involvement is present [1,3]. The middle fossa approach is ideal for supralabyrinthine lesions with preserved hearing, as it provides direct access to the geniculate ganglion and labyrinthine segment while preserving the functional labyrinth [1,5].

Translabyrinthine approaches, although effective, sacrifice hearing and are not indicated in patients with serviceable hearing [6]. Endoscopic techniques are promising but may offer limited exposure to the petrous apex, risking residual disease [7].

4. Facial Nerve Functional Outcomes

Recovery depends on:

- Delay between paralysis onset and decompression,
- Site of involvement (geniculate and labyrinthine segments carry the worst prognosis),
- ENMG severity,
- And type of neural management (decompression, grafting, neurorrhaphy) [9,10].

Within this clinical presentation, severe axonotmesis suggests a guarded prognosis even with optimal surgery. Declining operative management further reduces the likelihood of full recovery.

5. Case Application and Follow-Up

The recommended management (complete excision + facial nerve decompression via a suprapetrous route) was fully justified.

Due to patient refusal, strict follow-up was implemented:

- Serial House–Brackmann assessments,
- Ocular protection measures,
- Periodic audiometry,
- MRI-DWI and temporal bone CT every 6–12 months.

Untreated petrous apex cholesteatoma may progress, causing further cranial nerve deficits, labyrinthine destruction, or intracranial extension.

CONCLUSION

Petrous apex cholesteatoma is a rare and potentially deceptive condition. Isolated facial paralysis with normal otoscopy should raise suspicion of petrous bone pathology and prompt dedicated imaging. High-resolution CT combined with diffusion-weighted MRI is critical for diagnosis and for differentiating cholesteatoma from other petrous apex lesions. When feasible, surgical management aims to achieve complete excision and targeted facial nerve decompression while preserving hearing. In cases where surgery is declined, close clinical and radiologic surveillance is essential to prevent irreversible complications.

Learning Points

- Petrous apex cholesteatoma may present with isolated facial paralysis despite a normal otoscopic examination.
- Diffusion-weighted MRI is a key diagnostic tool, often eliminating the need for histology.
- The suprapetrous (middle fossa) approach offers optimal access to the geniculate ganglion and labyrinthine facial nerve with maximal hearing preservation.

Patient Perspective

The patient expressed concerns about the potential risk of postoperative hearing loss, which strongly influenced his decision to decline surgery. He reported that maintaining his remaining auditory function was a major priority, and he preferred a close clinical and radiologic follow-up despite the possibility of disease progression.

Ethical Approval and Consent

Written informed consent for publication of this case report and accompanying images was obtained from the patient.

Conflict of Interest: The authors declare no conflict of interest.

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