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Decompression of Facial Nerve in Case of Cholesteatoma (Case Report)

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Abstract **Case Report**

Middle ear cholesteatoma is a destructive, keratinizing lesion capable of eroding ossicles, mastoid air cells, and in severe cases, the fallopian canal leading to facial nerve palsy. Although facial nerve paralysis secondary to cholesteatoma is rare, its presence demands urgent surgical management to prevent permanent neurological damage. We present the case of a 45-year-old male named Abdur Rahman, who suffered for decades with progressive facial weakness, hearing loss, chronic otorrhea, and eventually facial nerve dysfunction caused by an extensive cholesteatoma involving the mastoid cavity and middle ear. He underwent tympanomastoid surgery followed by decompression of the facial nerve. The operation was complex due to adhesions, severe canal erosion, and anatomical distortion. Post-operative recovery demonstrated significant functional improvement. This case emphasizes the importance of early diagnosis, high clinical suspicion, advanced imaging, and timely surgical intervention in preventing irreversible facial paralysis and life-threatening intracranial complications.

Keyword: Middle ear cholesteatoma, facial nerve palsy, tympanomastoid surgery, mastoid erosion.

INTRODUCTION

Cholesteatoma is a chronic, destructive disease of the middle ear characterized by the proliferation of keratinizing squamous epithelium within the temporal bone. Despite being histologically benign, its potential for local tissue destruction makes it clinically significant. The progressive accumulation of keratin debris exerts mass effect and enzymatic degradation over ossicular structures, mastoid bone, and occasionally the facial nerve canal. Facial nerve paralysis is reported in 1–2% of cholesteatoma cases, and when present, it represents advanced disease requiring urgent surgical management (Gulotta *et al*., 2023; Selesnick & Lynn-Macrae, 2001).

The facial nerve is vulnerable when the fallopian canal is dehiscent either congenitally or secondarily due to chronic inflammation and osteolysis. Studies reveal facial canal dehiscence in 30–35% of patients with chronic

otitis media and cholesteatoma (Miyanaji *et al*., 2020; Rivas & Arrigunaga, 2024). Once cholesteatoma reaches this exposed nerve segment, compression, inflammation, and direct erosion may lead to neuropraxia or axonotmesis. Surgical decompression is therefore considered the gold standard to reverse neuropathic deficits and restore function.

In the present report, we document a rare and severely advanced case of cholesteatoma in a Bangladeshi patient, Mr. Abdur Rahman, who presented with chronic hearing loss, persistent ear discharge, imbalance, and ultimately facial weakness. Imaging revealed extensive mastoid and middle ear involvement with compression of the labyrinthine and tympanic segments of the facial nerve. A staged tympan mastoidectomy with facial nerve decompression was performed. The complexity of the case required meticulous dissection, anatomical reconstruction, and intensive perioperative care. The successful outcome highlights the importance of multidisciplinary teamwork, precise surgical technique, and early rehabilitation.

This report aims to present the clinical features, diagnostic challenges, operative strategy, and postoperative progress of this unique patient, while correlating findings with available literature. The case not only demonstrates surgical success but also emphasizes how early intervention can change the natural course of advanced cholesteatoma.

Case Study

Patient Description

The patient was a 45-year-old Bangladeshi male named Mr. Abdur Rahman, a resident of the Cumilla district. He was a father of seven children and had spent most of his life as the sole earning member of the family. Despite advancing age, he remained physically active and continued to work to support his household. For almost two decades, he suffered from chronic right-sided ear discharge and progressive hearing impairment. Due to financial limitations and lack of access to specialized ENT services in rural areas, he repeatedly ignored his symptoms or treated them conservatively. Over time, his condition deteriorated significantly. In the last one year, the disease progressed to the point where it began affecting his facial function, speech, and daily life activities, causing social embarrassment and emotional distress.

Case History

The patient had a long history of recurrent otorrhea, mild to moderate otalgia, hearing difficulty, foul-smelling discharge, and episodes of imbalance. Initially, the symptoms were intermittent, but over the years they became continuous. In the last twelve months, his family noticed progressive right-sided facial weakness. He developed inability to close his right eye completely, deviation of mouth toward the opposite side, slurred speech, and difficulty in chewing. Eating became messy as food frequently dribbled from the affected side. He also complained of frequent headaches, occasional vertigo, and discomfort while lying down, especially at night.

He sought treatment at multiple private hospitals within Bangladesh and even visited facilities abroad. However, due to the extensive nature of disease and the high risk of nerve injury and postoperative complications, surgical intervention was repeatedly postponed. As facial weakness continued to worsen and hearing declined further, he was finally referred to an advanced otology surgical team for definitive management. At this stage, concern for possible intracranial extension or permanent facial paralysis made urgent intervention necessary.

Physical Examination

General condition was stable and the patient was oriented. Local ear examination revealed persistent mucopurulent discharge from the right ear, an attic retraction pocket, keratinous debris, and a large central perforation of the tympanic membrane. There was marked mastoid tenderness on palpation. Pure-tone audiometry confirmed significant

conductive hearing loss on the right side, with an average air-bone gap of 40–50 dB.

Facial nerve assessment showed Grade IV weakness on the House-Brackmann scale. The patient had lagophthalmos, drooping of the oral commissure, loss of nasolabial fold depth, and reduced tone of facial muscles. Despite these findings, there were no signs of intracranial involvement no papilledema, neck stiffness, altered consciousness, or involvement of other cranial nerves.

Investigations

Pure-tone audiometry demonstrated a severe conductive hearing loss in the affected ear. High-resolution CT scan of the temporal bone revealed extensive cholesteatoma involving the epitympanum, mesotympanum, aditus, and mastoid antrum, with almost complete opacification of the mastoid cavity. The ossicular chain was eroded, the scutum was destroyed, and there was clear dehiscence of the tympanic segment of the facial nerve canal. In addition, part of the labyrinthine segment was exposed, and the mastoid air cells were markedly reduced due to chronic mastoiditis. These findings were consistent with advanced cholesteatoma known to erode bone and expose neural structures.

Facial nerve conduction study indicated reduced amplitude and delayed conduction velocity, suggesting neuropraxia secondary to nerve compression rather than complete degeneration. Baseline blood tests, ECG, electrolytes, coagulation profile, and chest X-ray were within normal limits, making him suitable for general anesthesia.

Treatment Plan

After correlating clinical and radiological findings, the surgical team planned a canal-wall-down modified radical mastoidectomy with complete cholesteatoma removal. Due to facial canal dehiscence and encasement of disease around the nerve, facial nerve decompression was mandatory. The plan included meticulous removal of cholesteatoma matrix, ossicular chain assessment, cartilage grafting for reconstruction, and tympanoplasty with temporalis fascia. Intraoperative nerve monitoring was arranged to avoid iatrogenic injury.

The patient and his family were counseled extensively. They were informed about risks of facial paralysis, hearing loss, CSF leak, vertigo, recurrence, and anesthesia-related complications. Considering his progressive nerve dysfunction, surgery was performed urgently to prevent irreversible axonal damage.

Operative Details

Modified Radical Mastoidectomy (MRM) followed by Type-3 Tympanoplasty (Right ear) + Facial Nerve Decompression Date: Sept. 2023. Under general anesthesia, a post-auricular incision was made to access the mastoid. Upon entering the mastoid cavity, a large cholesteatoma sac with dense granulation tissue was visualized, spreading throughout the aditus, antrum, and middle ear space. The incus was fully eroded and the malleus head partially destroyed. The disease extended into the facial recess and encased the tympanic segment of the facial nerve. The bony covering over the nerve was dehiscent and extremely thinned.

With high-magnification microscopy, the cholesteatoma matrix was carefully peeled away from the nerve without causing trauma. Granulation tissue was removed, and the facial nerve was decompressed from the labyrinthine to the mastoid segment. A palisade cartilage graft was placed to reinforce middle ear structure, and tympanic membrane reconstruction was achieved using temporalis fascia. Blood loss was minimal and intraoperative neuromonitoring confirmed nerve activity throughout the procedure.

Expected Outcome

Because nerve decompression was done before permanent degeneration, gradual improvement in facial movement was expected within weeks to months. Hearing restoration depended on graft uptake, ossicular reconstruction, and absence of further

infection. The canal-wall-down procedure also reduced the chance of cholesteatoma recurrence.

Actual Outcome

The patient recovered smoothly from anesthesia with no immediate postoperative neurological deficits. Within 48 hours, partial improvement of facial symmetry was observed; eyelid closure improved and oral movement became easier. Ear discharge stopped and pain significantly reduced, allowing him to sleep comfortably for the first time in years. At three-month follow-up, the patient showed remarkable progress. Facial nerve function improved to House-Brackmann Grade II, indicating near-normal facial symmetry and muscle strength. The tympanic membrane graft was well-positioned, there was no discharge, and his hearing improved sufficiently for everyday conversation. The patient regained confidence, social interaction, and quality of life. His family, especially his children who had seen him disabled for decades, were emotionally overwhelmed by his transformation and recovery.

Figure 1: Patients outcome before and after surgery

DISCUSSION

Cholesteatoma, although histologically benign, behaves in an aggressive and destructive manner within the temporal bone. Its pathogenicity arises from chronic infection, enzymatic bone resorption, and continuous expansion of keratinizing squamous epithelium, ultimately leading to erosion of surrounding anatomical structures. Among its severe complications, facial nerve paralysis is one of the most alarming. Chronic inflammation, osteolysis, and mechanical compression can progressively damage the facial canal, resulting in neuropraxia or permanent axonal injury if left untreated. Reported incidence of facial canal dehiscence in cholesteatoma is high, ranging between 30% and 70% in surgical series, underscoring the vulnerability of the nerve as disease advances (Gülüstan *et al*., 2014; Di Martino *et al*., 2000). Therefore, timely diagnosis and surgical intervention are vital to prevent irreversible neurological sequelae.

Why This Case Is Noteworthy

This case is particularly remarkable for several reasons. First, facial palsy associated with cholesteatoma remains a rare clinical finding compared to more common presentations such as otorrhea and hearing loss.

The majority of cholesteatoma cases are diagnosed and managed before neurological involvement occurs, especially in centers with widespread access to otologic care. Second, the disease in this patient persisted for decades, demonstrating how socioeconomic barriers and healthcare limitations can lead to extraordinary delays in treatment. Third, the cholesteatoma involved multiple segments of the facial nerve canal, causing progressive compression and dehiscence of the tympanic portion, with partial exposure of the labyrinthine segment an unusual and high-risk pattern of disease extension. Fourth, the surgical challenge was significant. The nerve had to be decompressed and diseased tissue meticulously removed without causing iatrogenic damage. Despite extensive anatomical distortion, the surgical team preserved the nerve, achieved complete disease clearance, and restored middle ear integrity. Finally, the rapid postoperative improvement highlights that even longstanding paralysis can recover when neural continuity remains intact. The reversal of facial deformity and restoration of expression had profound emotional and psychological benefits for the patient and family, adding meaningful human impact to the clinical success.

Correlation with Literature

Findings in this case strongly correlate with existing scientific literature. Arias-Marzán *et al.,* (2019) demonstrated that although CT imaging plays a vital role in preoperative planning, discrepancies may exist between radiologic interpretation and actual intraoperative nerve exposure. In this case, however, CT accurately predicted the extent of canal erosion and the risk to the facial nerve, guiding the surgical approach. Miyanaji *et al.,* (2020) emphasized that early decompression leads to better neurological recovery, particularly when paralysis is incomplete or recent. Our patient exhibited neuropraxia rather than axonal degeneration, which explains the rapid postoperative improvement within days and near-normal functional restoration at three months.

Bhagat *et al.,* (2024) reported significant improvement in facial nerve function following decompression in cholesteatoma-associated palsy, supporting the strategy used in this patient. Gulotta *et al.,* (2023) further noted that cholesteatoma-induced facial palsy tends to have a more favorable prognosis than traumatic palsy, as the primary mechanism is inflammatory compression rather than structural transection. Once pressure is relieved and infection controlled, nerve recovery is often rapid consistent with the present outcome. Regarding auditory rehabilitation, Kim (2024) described favorable hearing restoration using incus reposition and ossicular reconstruction techniques. However, in this case, ossicular remnants were insufficient for repositioning, necessitating alternative reconstruction using cartilage grafts to stabilize the middle ear architecture.

Message for Clinical Practice

This case carries several important clinical implications. Persistent, foul-smelling ear discharge should never be considered a minor or harmless symptom; it may represent underlying destructive pathology. Facial nerve weakness in the setting of chronic otitis media is a surgical emergency and requires immediate referral to an otologic specialist. High-resolution CT scanning remains an essential diagnostic tool for preoperative planning; however, definitive assessment of nerve exposure or erosion can only be confirmed intraoperatively. Most importantly, early surgical decompression prevents permanent neural injury and maximizes postoperative recovery. Finally, this case reinforces the importance of patient education and public health awareness in low-resource regions, where delayed presentation continues to contribute to avoidable disability. Improving access to specialized care and promoting awareness can prevent complications that carry lifelong physical, social, and emotional consequences.

CONCLUSION

This case demonstrates the successful surgical management of an advanced cholesteatoma complicated by long-standing facial nerve compression. Despite decades of untreated middle ear disease, severe anatomical distortion, ossicular destruction, and dehiscence of the facial canal, timely surgical intervention resulted in remarkable recovery. Canal-wall-down tympanomastoidectomy with meticulous cholesteatoma clearance and facial nerve decompression restored facial symmetry, improved hearing to a functional level, prevented further neurological or intracranial complications, and dramatically enhanced the patient's quality of life. The outcome highlights that even chronic cases can recover when neural continuity is preserved and appropriate operative strategies are used. Clinically, this case reinforces several important lessons. Facial nerve palsy in the setting of chronic otitis media with cholesteatoma should always be considered a surgical emergency, as ongoing inflammation and compression rapidly increase the risk of permanent nerve damage. Timely decompression offers excellent prognosis and allows for functional restoration when performed before irreversible axonal degeneration occurs. Furthermore, optimal outcomes require a multidisciplinary approach, including careful radiological evaluation, expert microscopic surgical technique, facial nerve monitoring, and structured postoperative rehabilitation.

Finally, this case underscores the importance of early detection and public health awareness. Improved patient education and greater access to specialized ENT care particularly in low-resource communities could prevent such advanced presentations in the future. Early diagnosis of chronic otitis media, prompt referral to otologic surgeons, and community awareness campaigns have the potential to reduce disability, prevent severe complications, and protect auditory and neural function in vulnerable populations.

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