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Case Report

Case study on patient with CSOM with intracranial and intratemporal complications managed with craniotomy and facial nerve decompression

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ABSTRACT

Facial nerve palsy, together with the intracranial abscess and labyrinthitis is a representative complication of advanced middle ear cholesteatoma. It is rare now a day due to ready access to medical care and antibiotics. Facial palsy is not life threatening but impairs facial movement and markedly affects patient's social life and causes serious psychological damage. Appropriate management of this complication is necessary to ensure healing without sequelae. We present a case of CSOM with intracranial and intratemporal complications. The patient had undergone craniotomy for intracranial abscess drainage and underwent facial nerve decompression by transmastoid approach.

Keywords: Facial nerve palsy, Facial nerve decompression surgery, Mastoid, Geniculate ganglion, Fistula, Cholesteatoma, House Brackmann scale, Semi circular canal

INTRODUCTION

Facial palsy can be a consequence of untreated chronic suppurative otitis media but has become rare owing to ready access to medical care and antibiotics.[1] Whenever it is present along with intracranial infection, it represents complication of advanced middle ear cholesteatoma. Cholesteatoma was first described by Friedmann and later redefined by Schucknecht.[2,3] It is a retraction pocket, lined by the stratified squamous epithelium containing proliferative keratin within the temporal bone. It causes enzymatic destruction of the bony ossicles, pressure necrosis, osteolysis.[4,5] The granulation tissue, cholesteatoma can cause irritation, edema and dysfunction of the nerve.

The infection can last for many years manifesting as partial deafness and ear effusion resulting in palsy. The patient cannot frown his forehead, nor close his eyes on the involved side, while his cheek and lips just fall.

Facial nerve canal dehiscence varies from 0.5–74% and it occurs in the tympanic portion of facial nerve near the oval window.[6]

In a recent study, it was found that 80% of the patients who had developed facial nerve dysfunction in the setting of cholesteatoma, had incomplete recovery after treatment of the underlying disease.[7] If greater than 90% degeneration occurs within 2 weeks, facial nerve should be decompressed to avoid compression and hypoxia.[8] However, the efficacy of surgical treatment is controversial.[9]

Depending on the site of lesion and preoperative hearing, facial nerve may be exposed via MCF, translabyrinthine,

and/or transmastoid approaches but no surgical approach is established as perfect.[10-13] Most commonly performed approach is transmastoid especially when the tympanic and mastoid segments of facial nerve are involved. It obviates the need for craniotomy in 60% cases of labyrinthine segment involvement.

We present a case of B/L chronic suppurative otitis media with cholesteatoma with right sided unilateral facial nerve palsy with oval window fistula in which modified radical mastoidectomy along with transmastoid facial nerve decompression of mastoid and tympanic part was done. Patient had undergone craniotomy for the intracranial abscess drainage.

CASE REPORT

A 15 years old female child patient visited the ENT department of GGSMC& Hospital, Faridkot complaining of intermittent discharge from both ears since childhood which turned foul smelling from right ear for the last 2 years. She was having history of progressive hearing loss from both ears, more in the right ear since childhood. She was having history of episodic vertigo for 2 years which turned to continuous for the last 6 months. Patient used to take symptomatic medication from local practitioner but was not relieved of her ailment. Patient started having headache and seizures 2 weeks back. She was diagnosed to be having intracranial abscess and underwent emergency craniotomy for drainage of the same and management of hydrocephalus. She was on anti-epileptic treatment afterwards. There was also history of right sided facial muscle weakness for the last 10 days.

She was also a known case of hypothyroidism since childhood and was on medication for that. There was no past history of DM, hypertension, TB, jaundice.

Patient belonged to the family with low socioeconomic status. She was a student of 12th standard. No immunization record regarding communicable diseases especially *H. influenza* virus B was produced by the parents. No relevant family history was recorded.

On general physical examination, patient was oriented to time place and person. She was averagely built and poorly nourished. Vital parameters were normal.

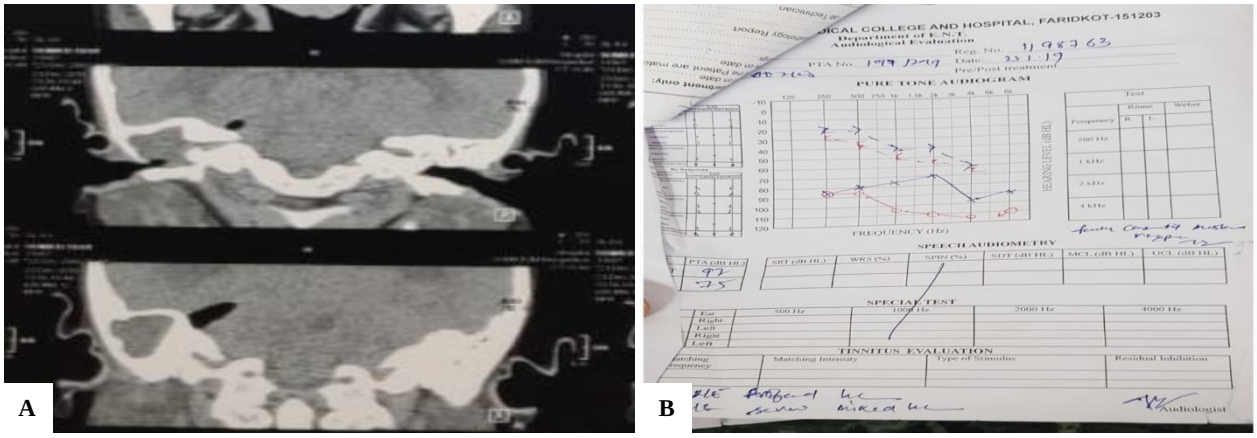

Figure 1: Preoperative investigations. (A) HRCT temporal bone showing middle ear cholesteatoma and inner ear fistula, (B) PTA.

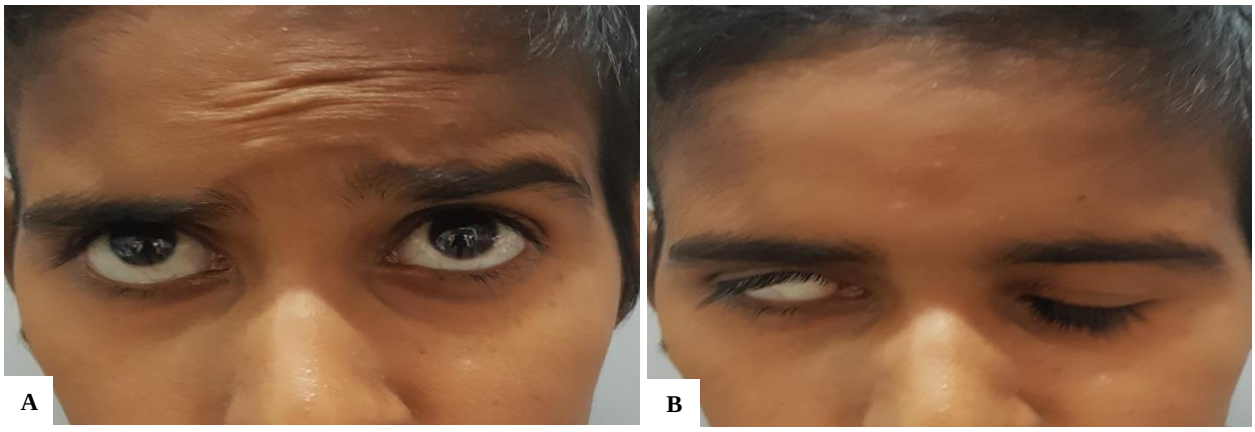

Figure 2: Preoperative facial expressions right side: (grade 6 on House Brackmann scale). (A) Forehead frowning, (B) Eye closure.

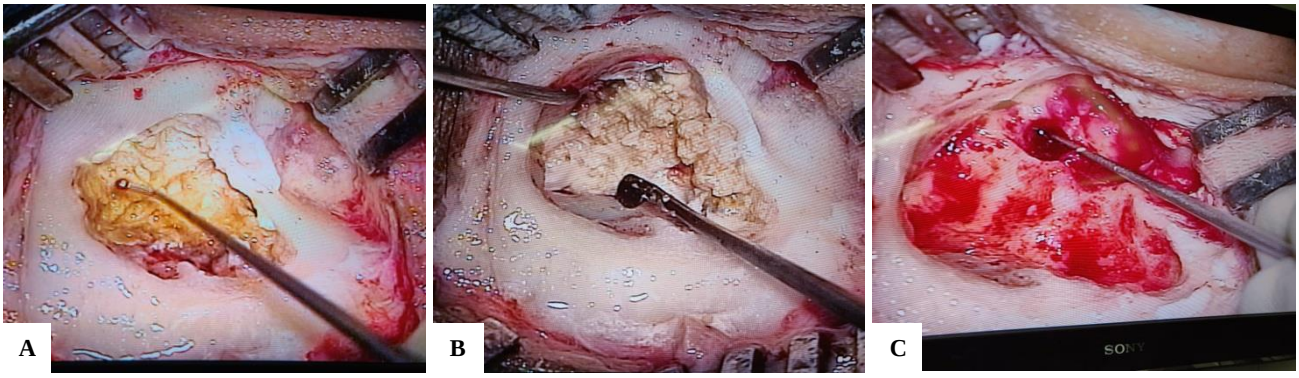

Figure 3: Intra operative figures/pictures. (A) Cholesteatomatous sac, (B) Vertical portion of facial nerve, (C) Oval window fistula.

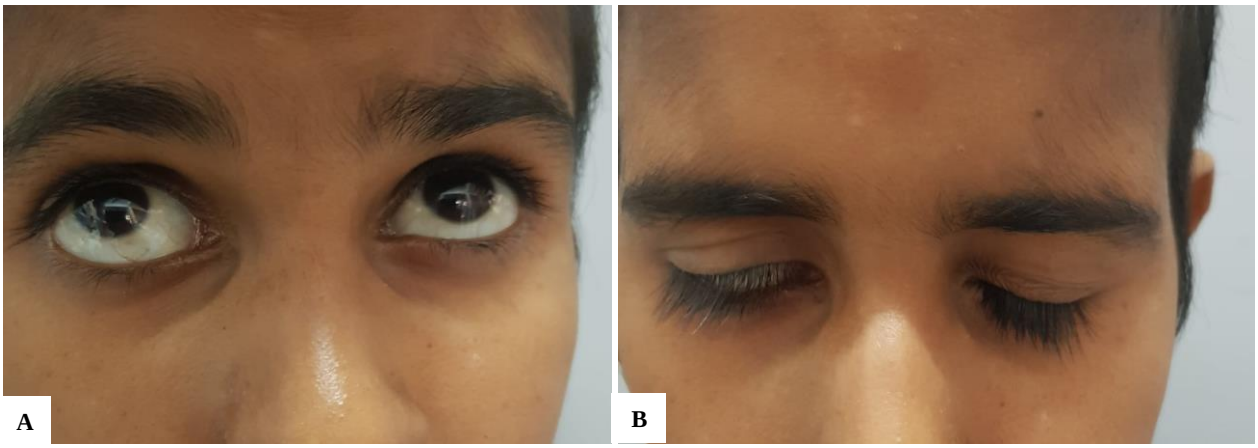

Figure 4: Postoperative facial expressions right side: (score 4 on House Brackmann scale). (A) Forehead frowning, (B) Eye closure.

On otoscopic examination of the right ear, patient had grade 4 retraction of TM in the posterior superior quadrant, attic region (as per Sade classification of pars tensa and tos classification of pars flaccida) with foul smelling discharge. Granulation tissue was visualized in the middle ear. On left ear otoscopy, there was total perforation of TM. Patient had right sided infranuclear facial nerve palsy of grade-6 on House Brackmann scale. Patient had healed stitch lining of craniotomy which she underwent 2 weeks back.

On PTA, patient was having B/L hearing loss with profound hearing loss on right side and severe mixed hearing loss on left side (Figure 1B).

On HRCT temporal bone (Figure 1A), there was soft tissue mucosal thickening involving right middle ear and mastoid air cells, causing destruction of the ossicles and mastoid air cells, with sclerosis and erosion of scutum and formation of fistula with inner ear.

Surgery

Under GA, a routine post auricular incision was made in the hairline on the right ear and 2-3 cm wide palvaflap was created. A self-retaining retractor holds the ear forward. A large cutting burr and continuous suction-irrigation was used. A complete modified canal wall down mastoidectomy was performed under microscope, with exposure of middle and posterior fossa dural plates, sinodural angle, sigmoid sinus, digastric ridge, incuidal fossa, lateral semicircular canal. Extensive cholesteatoma lying in the middle ear cleft involving epitympanum, mesotympanum, hypotympanum was removed for visualization of facial nerve (Figure 3A). The landmarks for the vertical segment are fossa incudis, chorda tympani nerve (Figure 3B). The middle ear ossicles were absent on account of destruction by the cholesteatoma. Diamond burr was used to delineate the course of facial nerve leaving only an eggshell layer of bone which was removed gently for decompression. Second genu was identified. The procedure was supplemented with trans EAC endoscopic approach for scutum dissection and better visualization of geniculate ganglion. 0 degree endoscope with HD camera was used for the purpose. Choleasteatoma was involving the labyrinthine segment and the same was cleared of. The oval window fistula (Figure 3C) repair was done with temporalis fascia graft along with surgicel. Wound was sutured in layers and mastoid dressing done. The greyish white soft tissue

collected from middle ear cleft was confirmed afterwards on HPE as cholesteatoma.

On first postoperative day, the patient stopped having the complaint of vertigo. After 1 month, facial nerve function evaluation of the operated side upgraded to grade 4, which was graded as 6 preoperatively in House-Brackmann scale (Figure 2A and B- preoperative, Figure 4A and B postoperative). There was no significant improvement in hearing post operatively.

DISCUSSION

Facial nerve palsy has several implications, particularly when occurring in childhood. It represents a serious clinical problem as it causes significant concern in doctors because of its etiology, treatment options, its functional and aesthetic outcomes. CSOM causing facial palsy is most frequently due to cholesteatoma.[12] This has been observed in study conducted by Altuntas, Yetiser et al.[14,15] The onset of palsy can be gradual or acute, gradual onset found to be more frequent.[13,14,16] Any surgical intervention for the facial palsy must carefully take into account the patient's age, medical history, residual hearing, segment of nerve injured, patient's expectation and risk tolerance.

In general, bony dehiscence of tympanic segment is observed in about 33% of cases during middle ear surgery with cholesteatoma and is mostly at the tympanic portion of facial nerve in the studies conducted.[17] This, accompanied by the high incidence of labyrinthine fistula, shows that the removal of cholesteatoma from around the tympanic portion of facial nerve should be done carefully. Rarely, In case of extensive inflammation and cholesteatoma, transaction of facial nerve may develop.[18]

In our case, surgical exploration revealed that cholesteatoma was present extensively in the middle ear cleft which damaged the tympanic, mastoid segment of facial nerve and formed a labyrinthine fistula. In the transmastoid approach, it is simple and easy to expose the vertical and partial tympanic segment of facial nerve and the same was adopted.

Classically, the exposure of geniclate ganglion requires middle cranial fossa approach. In case of facial nerve involvement in the labyrinthine segment and the part within internal acoustic meatus, mastoid-middle cranial fossa approach should be adopted. In present case, we adopted mastoid combined with transcanal endoscopic approach which exposed the geniculate ganglion and distal labyrinthine segment.

We opened the facial canal for the decompression. However, Cawthorne postulated in his study that nerve sheath incision should be performed for cases of complete paralysis.[16]

Savic and Djeric reported complete recovery in 70%, partial in 24% and failure in 6% of cases.[19] following decompression surgery. In our case, surgery was performed after 10 days of onset. This resulted in incomplete recovery as recorded on House-Brackmann scale.

CONCLUSION

Facial nerve involvement in CSOM is rare on account of ready access to antibiotics and medical care.

The transmastoid combined with transcanal endoscopic approach combines the advantages of access to geniculate ganglion without craniotomy.

In our case, surgery was performed after 2 months of onset of facial palsy. This resulted in incomplete recovery as recorded on House-Brackmann scale. Hence, it is concluded that the decompression should be carried out as soon as possible after palsy following injury due to cholesteatoma.

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