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CASE REPORT

BELLS PALSY IN A CASE OF SQUAMOUS CHRONIC SUPPURATIVE OTITIS MEDIA – A CASE REPORT WITH REVIEW OF LITERATURE

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ABSTRACT

We present the case of an 11-year old boy diagnosed with chronic suppurative otitis media, who was admitted to the ENT Department with bilateral ear discharge with decreased hearing which wasgradual in onset and progressive in nature, associated with right-sided facial nerve paralysis. The patient's facial nerve paralysis was scored as IV, according to the House-Brackmann scale. A cranio-facial computer tomography examination revealed mastoid cavity opacification, mucosal hypertrophy, and signs of chronic osteitis, with soft tissue accumulation over middle ear cavity. There was focal dehiscence of lateral wall of facial canal. The patient underwent Modified Radical Mastoidectomy with Ossicular reconstruction and tympanoplastyto verify the presence of a cholesteatoma, and to remove the offending lesions. Post-operatively, patient's recovery was favorable, and prognosis remained encouraging. The patient's evolution was followed by check-ups every three months to assess progress and benefits of the treatment.

Keywords: facial nerve paralysis; chronic suppurative otitis; chronic Mastoiditis; house-Brackmann scale

INTRODUCTION

Facial nerve paralysis is a rare but serious complication associated with chronic otitis media (COM). While the use of antibiotics has reduced its occurrence, preventing this complication remains a challenge. Studies have shown that the prevalence of facial nerve paralysis in cases of COM ranges between 0.16% and 5.1%(1,2)

Chronic Otitis Media (COM) is a long-standing inflammatory condition of the middle ear that leads to permanent alterations in the tympanic membrane. These changes may include tympanosclerosis, perforation, atelectasis, dimer formation, retraction pocket development, or cholesteatoma. The extent of ossicular chain involvement varies. Dysfunction of the Eustachian tube (ETD) contributes to COM by causing poor ventilation

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in the middle ear, leading to recurrent episodes of acute otitis media, persistent infections, or chronic inflammatory triggers. Cholesteatoma, when in contact with bone, leads to degradation of the normal mucosal lining, prompting an inflammatory response from macrophages, monocytes, and osteoclasts, which results in bone destruction. Increased mast cell presence is observed in the granulation tissue and on eroded ossicular surfaces, while multinucleated osteoclasts are the primary cells responsible for bone resorption in cholesteatoma.(3)

The pathological process in chronic suppurative otitis media leads to alterations in the middle ear mucosa, characterized by edema, sub mucosal fibrosis, and infiltration by chronic inflammatory cells. As these inflammatory changes progress, they can result in osteitis, triggering bone invasion and destruction in areas such as the inner ear, dura mater, or facial canal, which may ultimately lead to facial nerve palsy.

The exact mechanism by which COM leads to facial nerve paralysis is not entirely clear. However, treatment strategies typically involve a combination of antibiotics and surgical interventions, such as Myringotomy, mastoidectomy, and nerve decompression, to restore normal function to the facial nerve.(4,5) Facial nerve paralysis (FNP) caused by chronic suppurative otitis media (CSOM) can have either a sudden or gradual onset. Sudden onset typically occurs due to an acute infectious flare-up superimposed on the existing CSOM, whereas gradual onset FNP is usually caused by compression from a cholesteatoma.(6)

CASE REPORT

A 11-year-old male came in the ear nose and throat (ENT) OPD clinic following a referral by the local general practitioner. He presented with abrupt three-day history of right sided facial numbness, paresis and Otalgia. He had history of bilateral ear discharge and was diagnosed of bilateral Chronic Suppurative Otitis Media (CSOM) since 3 years. The discharge was scanty, mucopurulent, and intermittent andfoul smelling in nature. Discharge was not blood tinted. There was a history of unilateral right sided tinnitus with dizziness which was on and off since 1 year. The disease progressed over a period since last 6 months. He was on a course of oral amoxycillin-clavulanic acid. The discharge was not relived by medication. One week previously he was suffering from low grade fever which wascontinous in nature followed by lower respiratory tract infection. He also had complained of hard of hearing since one and half years which was gradually progressive in nature. On presentation at the ENT clinic he had a House-Brackmann (HB) of grade IV facial palsy. He complained that his eye is irritated, as it does not close even in sleep and tears rolls over. Food catches in the cheek,fluid when taken squirts from the angle of the mouth. There is inability to blow out the cheeks, and speech was mildly deficient. There was no change or absence of taste sensation. No complaint of dryness of mouth.No abnormal acuity of hearing and special sensitivity to deep notes. He had no respiratory disorders like allergy, asthma and did not smoke or had a history of any substance abuse. No history of trauma to head and neck. No history of any previous head and neck surgery.

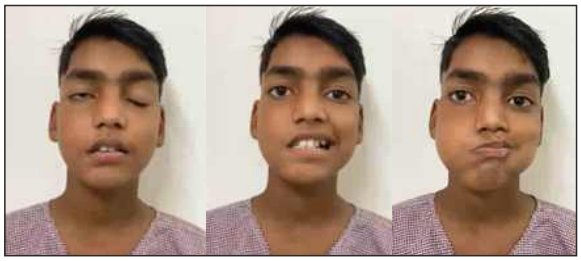

Fig. 1—Preoperative photographs showing facial paralysis

On Physical examination the patient was well oriented to time place and person, with healthy built.On facial examination flaccidity of right side of the face present. No swelling over neck or face. On ear examination no abnormal deformity seen over pinna, pre and post auricular regions. No erythema or eruptions over pinna. Otoscopy and microscopy revealed bilateral grade 4 retraction of tympanic membrane. On right ear retraction pockets were present on the postero superior part at the attic. EAC was mildly congested with discharge of pus in the canal. There is no presence of vesicles within the external auditory canal. No hyperesthesia of posterior wall of external auditory canal. On examination of nasal cavity slight devation of nasal septum towards right present. No tonsillar or adenoid hypertrophy in oral cavity examination. Tongue normal, no fissures present. No evidence of corneal abrasions and corneal reflex showed normal as checked by ophthalmologist.Other cranial nerve examination was unremarkable.Systemic examination wasnormal, no history of high blood pressure or diabetes.

The patient's facial nerve paralysis showed facial asymmetry at rest, on right side there was inability to close eye, mild brow ptosis with loss of forehead and brow movements, lower eyelid laxity or drooping,Nasolabial fold effacement with drooping of lower lip, deviation of the upper lip philtrum, inferior malpositioned oral commissure. On dynamic physical examinationof facial nerve , forceful closing the eyes showed failure to do so on right side, angle of mouth deviation from right lip absent, patient not able to raise his eyebrows of right side, inability to close right eye, no tongue deviation, No change in taste sensation on anterior two third of the tongue.

The severity of facial paralysis is assessed using the House-Brackmann scale, with a Grade IV classification indicating significant weakness. This is determined by evaluating the baseline level of asymmetry and the amount of effort needed to close the affected eye. (7)

On pure tone audiometry he had bilateral conductive hearing loss with 40 and 31 decibels loss in both right and left ear respectively. CT scan revealed mastoid cavity opacification, mucosal hypertrophy, and signs of chronic osteitis, with soft tissue accumulation over middle ear cavity. There was focal dehiscence of lateral wall of facial canal but without any intracranial complication. Culture sensitivity report of right ear came to show growth of Staphylococcus aureus. Comprehensive blood testing was done showing values within the normal range.

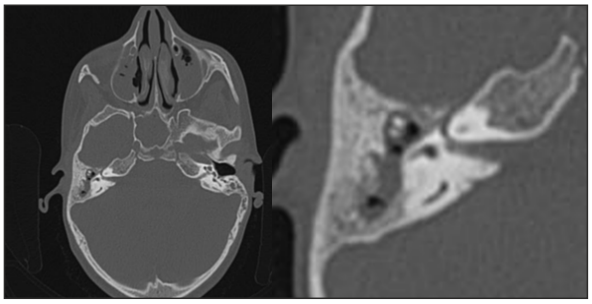

Fig.2: Transvese CT-section showing modifications of Chronic Mastoiditis, with

cavity opacification and mucosal hypertrophy. The Facial canal appears to be dehiscent.

Based upon the findings, the diagnosis of exacerbated chronic suppurative otitis media with grade IV facial nerve paralysis as a complication of CSOM was reached, along with moderate right ear hearing loss.

On admission the patient was given intravenous antibiotics along with steroids in divided doses. His Otalgia and discharge was significantly improved after administration of the medication. Meticulous eye care was followed. Additionally, the patient is advised to have a canal wall down mastoidectomy with facial nerve decompression with tympanoplasty. The surgery was carried out under general anesthesia. The attic portion of the middle ear and mastoid cavity presented with minimal mucous accumulation and numerous granulation tissue deposits which were surgically removed. Middle ear inspection confirmed both osteitis lesions, as well as the presence of small cholesteatoma flakes. The facial nerve was not dehiscent in the tympanic portion. Intraoperative findings showed necrosis of malleus, incus, stapes and stapes supra structure eroded. Stapes foot plate intact. Anterior and posterior buttress, facial bridge removed. Facial ridge lowered and all the disease removed. Mastoid and attic obliterated with periosteal and mucosal flap. Ossicular reconstruction done with Teflon prosthesis that was rested over the footplate and covered with cartilage. Tympanic membrane repaired with temporalis fascia followed by mastoid dressing. Postoperatively patient was instructed to prevent water from entering their ears while bathing and to avoid water-related activities, such as swimming.

In 3 days his signs and symptoms of Facial nerve palsy improved gradually. After seven days of pre-operative preparation, the patient's facial nerve paralysis and eye closure drastically improved, and facial expression was also regained. On evaluation after 3 days House-Brackmann score of III was seen.

The patient was seen 2 weeks later in an outpatient setting. His Facial nerve weakness improved with only a very mild weakness remaining; House Brackman grade II was seen. The patient reported that his hearing was also improved. Eventually in due course normal facial functions were restored; symmetry of the face was established as shown in the photographs (Fig-3).

Fig. 3: Postoperative photographs showing improved facial function (Post op- 3weeks)

DISCUSSION

Bell's palsy in a Case of Squamous Chronic Suppurative Otitis Media (CSOM) Bell's palsy is a condition characterized by sudden, unilateral facial paralysis due to dysfunction of the facial nerve. It is usually idiopathic in origin. Chronic suppurative otitis media is a long-standing infection of the middle ear characterized by ear discharge (otorrhea) and a perforated tympanic membrane, which can lead to various complications, including facial nerve palsy. This discussion will explore the occurrence of Belly's palsy in a case of squamous CSOM, which otherwise is mostly because of a complication

of CSOM. Bell's palsy occurs due to inflammation and swelling of the facial nerve, leading to temporary paralysis. In cases of squamous CSOM, facial nerve palsy may arise due to the direct involvement of the nerve through infection, inflammation, or compression by cholesteatoma, which is a destructive and expanding growth of keratinizing squamous epithelium. The cholesteatoma may erode the bone that separates the facial nerve from the middle ear, causing nerve damage or compression. Studies have shown that patients with squamous CSOM are at higher risk of developing complications such as facial nerve paralysis compared to those with mucosal CSOM due to the more aggressive nature of cholesteatoma.
The incidence of facial nerve paralysis in CSOM varies across studies, with some reporting it as a rare but serious complication. A study by Bakshi et al.(8) (2018) reported that Bells palsy occurs in approximately 1-2% of patients with CSOM, while other studies, such as those by Ramalingam et al. (2016)(9) suggest a slightly higher incidence when cholesteatoma is present.

Browning et al.(10)(2012) emphasized that facial nerve paralysis in CSOM is most commonly associated with cholesteatoma, highlighting the importance of early detection and treatment of cholesteatoma to prevent this severe complication. They found that aggressive surgical management, including mastoidectomy, was necessary to relieve the pressure on the nerve and allow recovery.
There is also a difference of Clinical presentation of Bell's palsy and Facial palsy in CSOM. (Monstad et al.(11) - 2019) Patients presenting with Facial palsy secondary to squamous CSOM typically report a history of ear discharge, hearing loss, and in some cases, pain. The onset of facial nerve paralysis is usually gradual, correlating with the slow progression of cholesteatoma or chronic infection, unlike the sudden onset typically seen in idiopathic Bell's palsy. This distinction is critical for diagnosis, as delayed management may lead to permanent nerve damage.

Verma et al.(12)(2020) echoed these findings, noting that the gradual progression of facial paralysis in squamous CSOM can often lead to delayed diagnosis, which worsens the prognosis. Their study emphasized that patients with squamous CSOM often report ear symptoms for months before the facial nerve is affected, distinguishing it from idiopathic Bell's palsy, which usually resolves spontaneously without long-term sequelae.

Several studies have compared the outcomes of patients with Bell's palsy due to idiopathic causes versus secondary to CSOM. For instance, Monstad et al.(11)(2019) showed that patients with Bell's palsy secondary to squamous CSOM had a lower rate of spontaneous recovery and often required surgical intervention, such as canal wall down mastoidectomy or tympanoplasty, compared to those with idiopathic Bell's palsy. Their recovery depended significantly on the extent of nerve involvement and the timely administration of antibiotics and steroids.

In a comparative study, Kazikdas et al.(13)(2015) found that the prognosis for facial nerve recovery in patients with squamous CSOM was dependent on early surgical intervention. They reported that patients who underwent surgery within a few days of the onset of facial nerve palsy had better outcomes, with 80% achieving

full recovery. Conversely, those with delayed intervention had poorer prognoses, with only 40% achieving significant nerve function improvement. This suggests that early recognition and prompt surgical management are essential for preventing permanent nerve damage.

CONCLUSION

Bell's palsy in the context of squamous CSOM presents a unique clinical challenge due to the risk of permanent facial nerve damage if not treated promptly. Various studies highlight the differences in the clinical presentation, importance of radiological investigations and importance of early diagnosis and early intervention which is different in the two categories. Comparisons across studies show consistent findings that surgical management yields better outcomes than conservative treatments, particularly when intervention occurs early in cases of facial palsy with CSOM whereas better results are achieved by medical management in Idiopathic Bells palsy. Given the progressive nature of squamous CSOM and its potential to cause serious complications like facial nerve palsy, early identification and treatment of patients at risk are critical. Additionally, more research is needed to refine treatment protocols and improve prognostic outcomes in these patients.

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