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Case Report

A rare case of bilateral acute otitis media leading to bilateral facial paresis in an adult

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ABSTRACT

Facial nerve paresis is a known complication of middle ear disease. However it is more commonly seen as a complication of chronic otitis media as compared to acute otitis media (AOM). There are very few reported cases of AOM leading to facial palsy and even fewer ones of bilateral acute otitis media leading to bilateral facial palsy. Since this is a very rare presentation its management and treatment are not very well outlined as per standard guidelines. Here we have presented a very rare case of bilateral AOM leading to bilateral facial paresis, how the case progressed and how it was managed. We have tried to bring forth the salient features of the presentation, the progression and the resolution of the disease due to the successful management.

Keywords: Acute otitis media, Facial paresis, Bilateral, Myringotomy

INTRODUCTION

Facial nerve paralysis complicating acute otitis media is thought to be due to intrafallopian inflammation and consequent ischemia with neuropraxia. Mostly this occurs either due to (i) preexistent bony dehiscences, (ii) physiologic canaliculi between the middle ear and fallopian canal, or (iii) the vascular connections between the fallopian canal and the mastoid air cells. Facial nerve paralysis due to otitis media was not so rare in the pre-antibiotic era, occurring in 0.5–0.7% cases of middle ear inflammation.[1] Nowadays, this complication has become an exceptional phenomenon and its incidence is reduced by a factor of 100 to 0.005%.[2] Reported cases of bilateral acute otitis media (AOM) leading to bilateral LMN facial palsy are very few and mostly seen in the paediatric population. Here we report an extremely rare case of bilateral AOM leading to bilateral FNP in a young adult.

CASE REPORT

A 24 year old male presented with complaints of otalgia and hearing loss in both ears for the past 1 week. He also had on and off low grade fever for a week. The patient gave history of facial weakness on the (R) side since one day. On examination he was found to have bilateral hyperemic and bulging tympanic membranes which were more prominent in the region of the pars flaccida above the lateral process of malleus. He had a moderate conductive hearing loss on the (R) and a mild conductive hearing loss on the (L). The same was shown by the pure tone audiogram as well. Tympanometry showed a bilateral 'B' type curve suggestive of fluid in the middle ear. He also had a Grade III House Brackmann facial paresis on the (R) side. A nasal endoscopy was done to rule out any central cause for the bilateral pathology. However the nasopharynx and bilateral middle meatus

were found to be normal. On detailed oral cavity and oropharyngeal examination no feature suggestive of any central pathology that could give rise to a bilateral acute otitis media was found. The patient underwent an urgent CT scan of the temporal bone to rule out any other intracranial or extra cranial complications of AOM and any anatomical abnormality like fallopian canal dehiscence leading to the facial palsy.

The CT scan showed features suggestive of bilateral mastoiditis and bilateral acute otitis media. However the facial nerve and the bony facial canal appeared normal bilaterally. The patient was started in intravenous antibiotics Cefotaxime and Amikacin. Adjunctive treatment such as anti histaminics, Xylometazoline nasal drops and steam inhalation was also given. On 2nd day of admission the facial paresis on the (R) worsened to Grade III and the patient underwent myringotomy of the (R) TM in OPD and thick glue was aspirated. On day 3 of admission the patient the developed Grade II facial palsy on the (L) side as well. He again underwent myringotomy on the (L) side in OPD and thick glue was drained out from the (L) ear. The antibiotics were stepped up and the patient was started on a 6 day short course of oral sterioids and tablet Chymoral Forte. By 4th day of admission the patient had symptomatic improvement in hearing in both the ears and the hyperemia of the tympanic membrane on both sides had started reducing. On day 6 of admission the facial paresis on both sides started improving and was grade II bilaterally by day 7 when he was discharged. No surgical exploration was attempted or deemed necessary due to the evident improvement in facial function with the conservative measures. The patient was discharged with a course of oral antibiotics, anti histaminics and nasal steroid sprays. During follow up 5 days after discharge, a repeat pure tone audiogram showed normal hearing sensitivity in both ears. Facial muscle function returned to normal bilaterally on follow up after 25 days. Informed consent was taken from the patient for publication of the above information for educational and research purposes.

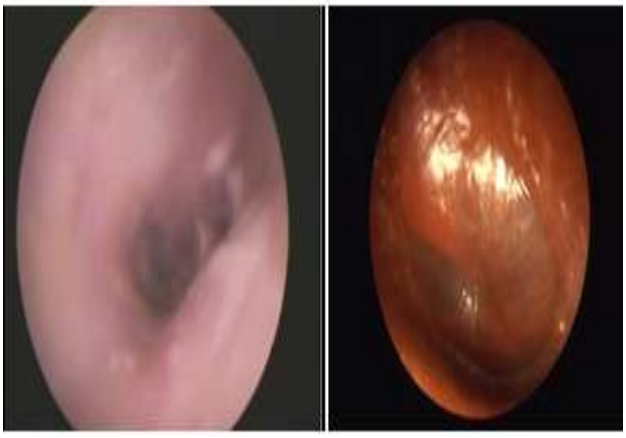

Figure 1: Showing bulging tympanic membrane b/l especially in the pars flaccid region.

Figure 2: Inability to close eyes bilaterallly.

Figure 3: Deviation of angle of mouth to (L).

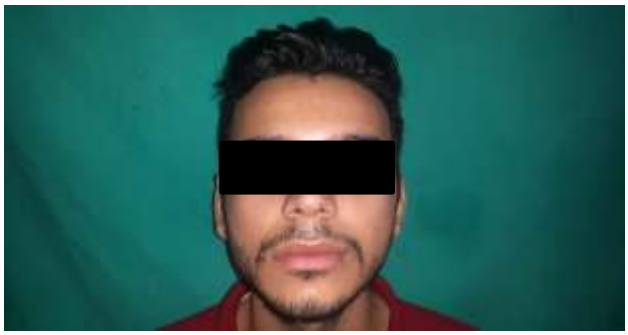

Figure 4: Absence of forehead wrinkling.

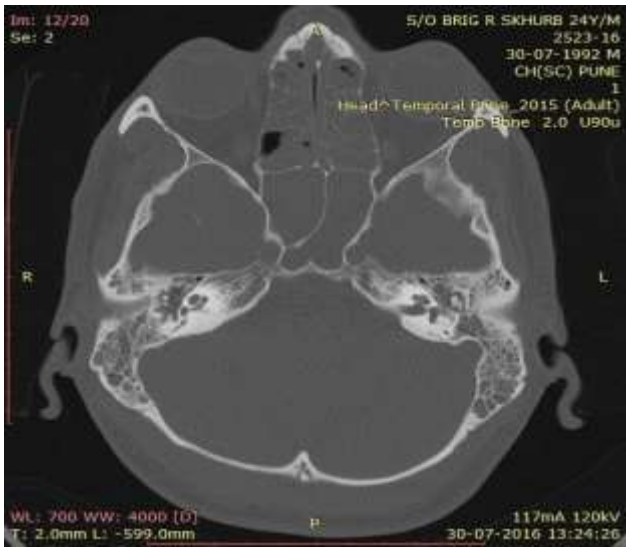

Figure 5: CT scan showing soft tissue attenuation in middle ear and mastoid.

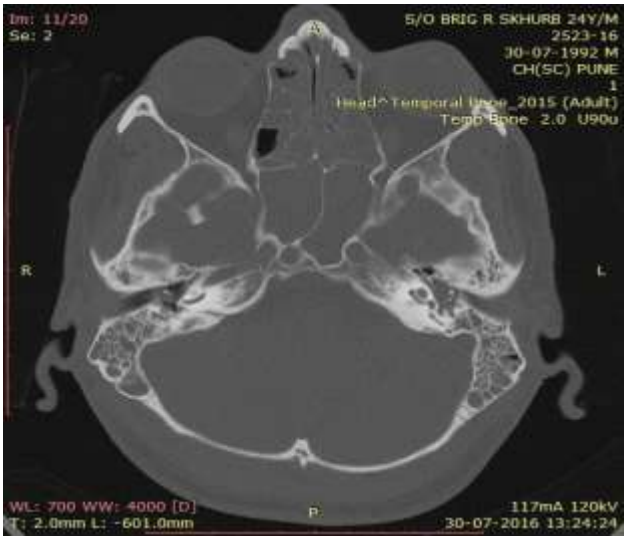

Figure 6: CT scan showing preserved anatomy of facial nerve bilaterally.

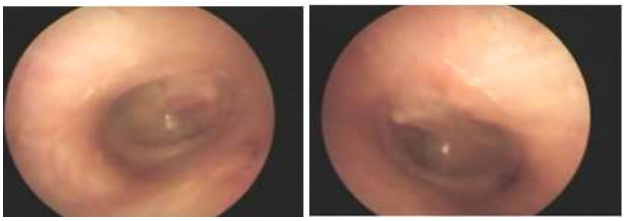

Figure 7: Appearance of bilateral tympanic membranes 25 days post treatment.

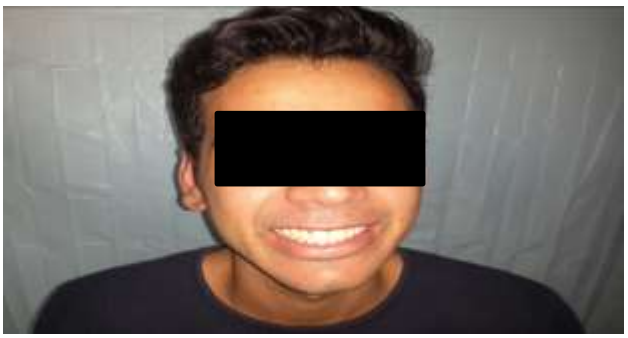

Figure 8: Return of facial muscle function.

Figure 9: Complete closure of eyes.

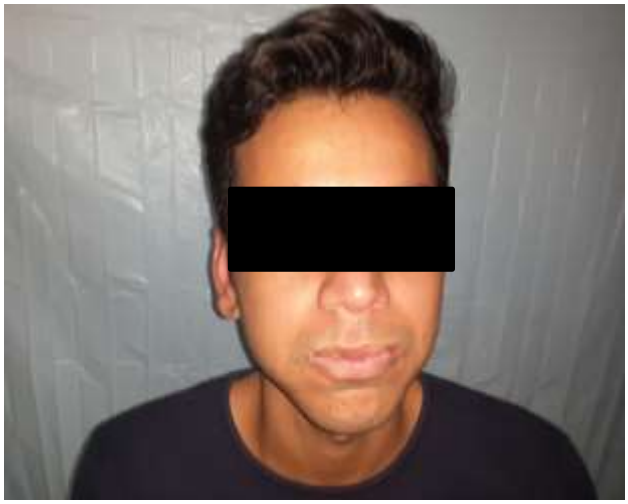

Figure 10: Patient able to puff cheeks.

DISCUSSION

Cause of FNP in AOM

The exact pathophysiology by which facial nerve paralysis occurs during AOM is still uncertain. Proposed mechanisms have been based on the anatomic intimacy between the facial nerve and the temporal bone. Middle ear microenvironments, such as elevated pressure, osteitis, or acute inflammation, may directly affect the facial nerve physiology and may be responsible for its dysfunction.

In the early stages of acute otitis media, facial paralysis may be explained by:

- Retrograde infection within the facial nerve bony canal or retrograde infection within the tympanic cavity ascending the chorda tympani to the facial nerve.[3]
- Reactivation of the latent virus infection caused by middle ear suppuration.[4]
- Demyelination of the facial nerve due to the presence of bacterial toxins.[5]
- Acute neuritis with venous thrombosis leading to inflammatory oedema of the nerve.[6]

In chronic middle ear disease, facial nerve paresis occurs by a slowly growing cholesteatoma or by an osteitis affecting of the fallopian canal. As middle ear infection persists, bone infection and resorption begin, leading to coalescent mastoiditis, then osteitis, bone erosion, and nerve destruction.[7] However, osteitis would likely take 2 to 3 weeks to develop after the onset of infection, making it an unlikely cause of Facial nerve paresis in AOM.

The pathophysiology by which acute otitis media leads to early facial nerve paresis remains one of speculation. Bacteriologic research has yet to support the idea that bacteria-liberated toxins are responsible because no

toxins have been associated definitely with the pathogens causing AOM. However, the temporal relationship of FNP in AOM suggests that unknown mediators acting either directly as neurotoxins or indirectly by reactivation of a latent virus are most probably responsible.

Ischemia secondary to pressure through a dehisced facial canal may cause facial palsy associated with acute otitis media. This would mean that draining the middle ear could be a key treatment statergy for facial palsy with otitis media even when there are no signs of infection on the tympanic membrane.[8]

Management

When AOM is complicated by facial nerve paresis, management should involve aggressive antimicrobial therapy for the AOM. Although myringotomy has an unknown effect on the reversal of FNP, it continues to be the standard of care and should remain so until scientific study can refute its role. When used, myringotomy should probably be accompanied by ventilation tube insertion to prevent early closure and to allow ongoing assessment of the middle ear. In our case we did not insert a ventilation tube as the symptoms started resolving on the next day the myringotomy was performed and the need to insert a ventilation tube was not felt.

We also strongly advocate the use of a short course of oral steroids as we did in our case and as shown in other studies.[9] Because corticosteroid agents remain the best treatment for inflammatory, virally induced, immune-mediated disease, we advocate their use unless contraindicated by another comorbid condition.

Adour and Ruboyianes showed prednisone plus Acyclovir to be more effective than prednisone plus placebo in patients with Bell's palsy.[10] Because of the lack of clinical trials, it is uncertain whether Acyclovir should be added to a management regimen for FNP in AOM, and further investigation is warranted.

In case of unilateral paralysis if the paralysis does not improve within 7 days, a CT scan of the temporal bones is indicated to exclude the presence of an occult tumor or mastoid coalescence. If coalescent mastoiditis is seen, mastoidectomy is indicated. With surgical intervention, decompression of the bony fallopian canal is discouraged because the risk of damage to the inflamed and friable facial nerve is thought to be high.

However as our case had bilateral AOM at the onset and unilateral facial nerve paresis, it was deemed prudent to get a CT scan to rule out any other causes of facial nerve palsy or any central cause leading to the bilateral AOM.

CONCLUSION

Bilateral AOM leading to bilateral FNP is a very rare presentation and of which there are very few recorded cases most of which are in the paediatric population. In this paper we want to highlight the management with aggressive antimicrobial therapy, myringotomy to help drain the pus from middle ear cavity and release pressure from the middle ear and the use of oral corticosteroids to decrease inflammatory process. All of the above were together responsible in successful management of the case.

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