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Case Report

Acute otitis media in pregnancy with unilateral lower motor neuron facial nerve palsy: a case report

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ABSTRACT

We report the case of a pregnant female of 32-years at 37-weeks of gestational period with four days history of left sided lower motor neuron (LMN) facial palsy with a recent history of bilateral ear pain. She is in her last trimester of pregnancy and expected for a normal vaginal delivery. She had no other ailments and had no history of similar facial palsy in past. The case was managed conservatively with good outcome. Following our experience and review of literature on the case, antibiotic therapy and corticosteroid therapy, with or without myringotomy were found to be the first-line of management. To our knowledge, this is a rare report of LMN facial palsy in association with acute otitis media (AOM) and pregnancy. Some factors involved for LMN palsy in this case could be: compression of horizontal part of facial nerve due to otitis media, increases incidence of Bell's palsy in pregnancy.

Keywords: Acute otitis media, Corticosteroid, Lower motor neuron, Facial nerve palsy

INTRODUCTION

Facial nerve palsy has become an uncommon complication of AOM in the recent era, with an estimated incidence of about 0.005%.[1] It was a very common complication in the pre-antibiotic era, with an estimated incidence of around 0.5-0.7%.[1] Facial nerve paralysis secondary to AOM is thought to be mediated by intrafallopian inflammatory edema and consequent ischemia with neuropraxia. The possible factors causing the facial nerve paralysis in AOM are likely to be alterations in the middle ear microenvironments, such as elevated pressure, osteitis, or acute inflammation, retrograde infection or due to reactivation of viruses within bony facial canal wherein facial nerve physiology may be directly affected.[2] Facial nerve palsy secondary to AOM requires proper care with appropriate antibiotics, so that the requirement of any surgical intervention can be minimised.

CASE REPORT

A 32-year-old lady presented to us in outpatient basis with history of bilateral mild ear pain 10 days back; more on left side and mild left ear discharge. Oral analgesics were not required for the pain. Later, she also complained of drooling of saliva from left side of the mouth while drinking water and eating food, along with an asymmetry of face and inability to close her left eye for last 4 days.

On facial examination, she was found to have left sided facial palsy of LMN type, grade V House Brackmann scale as seen in Figure 1.

Local examination of left ear was done to rule out Herpes zoster. No vesicular rash was noted in concha, external ear canal (EAC). Oral cavity was also thoroughly examined to rule out such lesions. No parotid masses were observed. Furthermore, no postauricular swelling or

erythema was detected. No auricular displacement or mastoid region tenderness was documented.

Minimal whitish flakes with mild discharges were noted in ear canal on the left side, which was cleaned by suctioning. Tympanic membrane was found to be erythematous and bulging with purulent fluid visible in middle ear. Similar findings were noted in right ear as well but less pronounced as compared to left side; oto-endoscopy as shown in Figure 2. Hearing tests revealed mild conductive hearing loss in both ears.

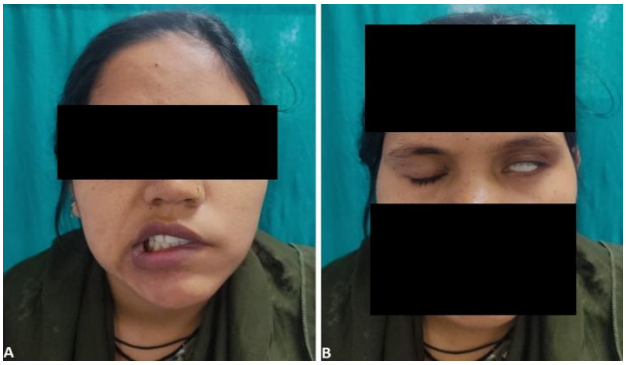

Figure 1 (A and B): At presentation showing grade 5 house Brackmann scale facial palsy.

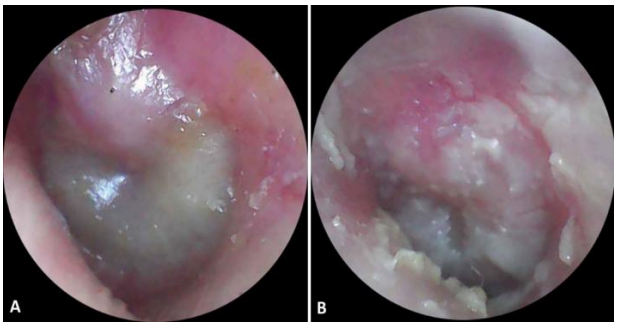

Figure 2 (A and B): A-Right tympanic membrane, B-Left tympanic membrane; both showing erythema and bulging with purulent fluid visible in middle ear, more on left side.

Investigations

Complete blood counts were done which showed slightly raised WBCs and erythrocyte sedimentation rate, about 20 mm/h.

Considering the pregnancy status, CT scan was not done to prevent radiation exposure and discomfort to the patient considering her last trimester of pregnancy. Also, electroneuronography was not done. The approach was to be as conservative as possible due to her pregnancy.

Treatment

With the suspected diagnosis of AOM with left facial nerve palsy of LMN type, patient was planned for conservative management. Since, facial nerve compression (suspected dehiscent horizontal part) was one of our hypotheses, myringotomy and pus drainage from middle ear was one of our considerations, but it was deferred as an initial management since the case was of a pregnant lady. Our plan was to initiate conservative management for couple of days and if no improvements were noted, then the plan was for myringotomy and release pus from middle ear. Patient was treated with oral antibiotics (cefpodoxime 200 mg twice a day), oral corticosteroids (prednisolone 60 mg once a day) and rehabilitative measures for facial palsy in the form of eye lubricants and physiotherapy. The facial palsy was seen to be improving after two days of medication and ear findings also improved. Thus, conservative management was continued and myringotomy was deferred. Antibiotic course was discontinued after 10 days and oral steroid tapered after a week of initial dose. Facial palsy recovered to grade II after 2-weeks.

DISCUSSION

The incidence of facial nerve paralysis is drastically declined from the pre-antibiotic era to present days, and meanwhile, the incidence of facial nerve paralysis dropped from 2 in 100 cases to 1 in 2000.[3]

Paralysis of the facial nerve will result in weakness of the facial muscles, impacting on verbal communication, social interaction in respect to facial expression, oral competence, corneal protection, and vision.

In the post-antibiotic era, the incidence of facial nerve paralysis due to AOM is 0.2% and so conservative treatment is encouraged towards timely eradication of infecting agent with antibiotic therapy.[4] The rate of peripheral facial nerve paralysis secondary to otitis media varies from 0 to 30%.[5]

Though, Bell's palsy is more common in pregnancy, three times that of an average person, the cause of LMN palsy in this case was attributed to AOM as the identifiable ear cause was present.[6]

The etiology of facial nerve palsy in patients with AOM is unclear, although many hypotheses have been postulated. Firstly, retrograde transmission of infection within the facial nerve canal through dehiscent canal or via corda tympani nerve into the facial nerve.[7] Secondly, the presence of inflammatory bacterial toxins may induce peripheral demyelination of the facial nerve. Finally, the spread of the inflammation into the mastoid region may provoke inflammation or compression of the facial nerve. Ischemia due to pressure through dehiscence of the facial canal may cause facial nerve paralysis in AOM. This suggests that drainage of the middle ear could be an important treatment of the facial nerve palsy. But drainage was kept as second option in our case as she was a pregnant lady and no aggressive mode of management was thought of as primary treatment, to be on safer side.

Acute inflammation of middle ear is one of the most common infections in children and early adulthood. The infection usually involves middle ear and mastoid cavity. Occasionally, if the infection and inflammation is severe, it spreads to the surrounding structures in middle ear cleft. Fallopian canal can be involved, especially in cases of anatomical dehiscence of the canal, resulting in peripheral facial paralysis as seen in our patient. Nager et al reviewing anatomical variations of the facial nerve, found 55% fallopian canal dehiscence.[8] Another theory suggests that the infection and inflammation cause compression to the vessels that nourish the facial nerve and this could cause local ischemia and nerve infarction, and consequent paralysis.

In cases of reduced immunity, reactivation of latent viral infection caused by middle ear suppuration has been postulated for facial nerve paralysis.[9] Rarely, AOM is known to cause acute neuritis with venous thrombosis leading to inflammatory edema of the nerve.[10]

When facial palsy appears late in the course of disease, it seems to be caused by direct extension of middle ear inflammation to the fallopian canal and poor vascular perfusion caused by inflammation.[11] During this stage it depends on silent or masked mastoiditis, a complication of acute middle ear infection which is due to inadequate antibiotic therapy and aggressiveness of infectious agents.

Also, it is worth discussing regarding the eustachian tube dysfunction (ETD) during pregnancy. ETD may be due to patulous eustachian tube during pregnancy when there is inadequate weight gain. This is mostly noted during 3rd trimester. This patulous tube may permit reverse infection of viruses or bacteria and may result in increased incidence of middle ear infections. Likewise, oedema of the respiratory mucosa as seen in pregnant women may lead to hypoventilation of the middle ear cavity and otitis media.

Similarly, “pregnancy rhinitis” is a condition that causes nasal congestion or a stuffy nose during pregnancy, which results from an increased level of estrogen and blood in the body. Occasionally, this temporally rhinitis may trigger secondary viral rhinitis, which may in turn progress into otitis media.

CONCLUSION

The treatment for facial nerve palsy in AOM should be managed on emergency basis with appropriate antibiotics and with corticosteroids for complete recovery.

Myringotomy and grommet insertion should be considered when there is a bulging tympanic membrane, collection of fluid in the middle ear or if the spontaneous perforation of the tympanic membrane does not occur. Surgery is indicated when there is history of recurrent ear infection and there is suspected silent or masked mastoiditis due to inadequate antibiotic therapy.

In our patient, the recovery was optimized due to early diagnosis, and appropriate intervention with antibiotic and steroids. It is always safer to be less aggressive and judicious in managing such cases in a pregnant lady as the body is already in hyperdynamic and hypercoagulative state. It was wiser on our side not to intervene with myringotomy as first line; eventually patient responded with conservative management.

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