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Acute otitis media with facial nerve palsy: our experiences at a tertiary care teaching hospital of eastern India

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ABSTRACT

Objective: To assess acute otitis media with facial nerve paralysis, its prognosis, and treatment.

Methods: It is a retrospective study. Thirteen patients of acute otitis media associated with facial nerve paralysis attended the outpatient department of otorhinolaryngology between May 2017 and April 2019. All patients were clinically assessed with appropriate investigations, prognosis, and treatment.

Results: Out of 13 patients, 2 were identified with complete facial paralysis and the remaining 11 patients presented with incomplete paralysis. Medical treatment including antibiotics and corticosteroids failed, while myringotomy and facial nerve decompression were done with a favorable outcome. Eleven patients recovered to grade-I (House-Brackmann) and 2 cases to grade-II (House-Brackmann).

Conclusion: Peripheral facial nerve paralysis in acute otitis media is rare. Antibiotics and steroids yield good outcome as conservative management. In case of failure by conservative treatment, facial nerve decompression yields a favorable outcome.

1. Introduction

The facial nerve has fascinated and interested almost all physicians, otolaryngologists and neurologists for several decades and extensive studies have been carried out on disease. As the course of the facial nerve is intimately related to the temporal bone and middle ear, any lesions or infections in the middle ear can cause facial nerve paralysis. Acute otitis media (AOM) is defined clinicopathologically as inflammation of the middle ear cleft with rapid onset and infective in origin and associated with collections of clinical manifestations. It is sometimes called as acute suppurative otitis media. AOM is a common illness in pediatric age, but it is rare in adults. Facial nerve

paralysis is a rare complication of AOM in the clinical practice and its incidence is about 0.005% AOM[1]. The possible cause for facial nerve paralysis in AOM are likely to be changes of the middle ear environment such as raised pressure, osteitis or acute inflammation where the physiology of facial nerve may be affected[2]. It was a common clinical complication of AOM in the pre-antibiotic era with an incidence of around 0.5%-0.7%[3]. In AOM, facial nerve paralysis is thought to be caused by intra-fallopian inflammatory edema and followed by ischemia with neuropraxia. Raised intratympanic pressure is due to accumulation of inflammatory fluid or

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osteitis or acute inflammation, retrograde infection or by reactivation of facial nerve of viruses inside the fallopian canal where the physiology of facial nerve may be affected directly. Facial nerve paralysis due to AOM requires proper care with antibiotics, so the need of any surgical intervention can be minimized. Our study aims to describe the experience of AOM with facial nerve paralysis with its management and outcome at a tertiary care teaching hospital.

2. Materials and methods

This study was conducted between May 2017 and April 2019 and was approved by the Institutional Ethics Committee with reference number CRL/IMS/SOAU/IEC/23/2017. The patients with previously facial palsy due to traumatic origin, iatrogenic origin and unusual infection like Ramsay Hunt syndrome were excluded. Clinical parameters included age, gender, duration, radiographic findings, audiological findings, surgical treatment, and outcome. All patients were clinically assessed for confirmation of facial nerve paralysis in AOM. The facial nerve paralysis was classified according to the House-Brackmann scale (Table 1). The facial nerve lesions were assessed topographically through a study of tearing, stapedial reflex test and imaging. The prognosis of the facial nerve was assessed by electroneurography. Broad-spectrum antibiotics were used to treat the majority of the bacteria causing AOM and steroids. We also prescribed eye drops and eye protection ointments. Surgical treatment included myringotomy and/or mastoidectomy and facial nerve decompression without opening the facial nerve sheath.

3. Results

A total of 13 patients were included in this study. Out of 13 patients, 8 were male (61.54%) and 5 were female (38.46%) with age varying from 1 year to 65 years (mean age: 14.32 years). A total of 2 (15.38%) were identified with complete facial paralysis and 11 (84.62%) presented with incomplete paralysis. Nine (69.23%) patients presented with right side facial palsy whereas 4 (30.77%) patients presented in left sides. Topographic tests showed 12 cases (92.31%) of infra-geniculate and 1 case (7.69%) of supra-geniculate facial nerve paralysis. The mean duration of infection before facial paralysis was 3.4 d with mean duration of facial paralysis as 17.53 d. Ten patients underwent CT scan of the temporal bone and revealed opacification of the middle ear and coalescence of mastoid air cells without any evidence of bony erosion. Nine patients underwent an audiological evaluation and showed a conductive hearing loss. Fluid was aspirated from nine cases and seven showed negative culture for bacteria, which may be due to previous antibiotic course. Two cases showed streptococcus pneumoniae in culture. All patients received high dose broad-spectrum antibiotics (Amoxyclav) following myringotomy with grommet insertion. Two cases underwent mastoidectomy and facial nerve decompression, which showed

Table 1. House-Brackmann grading for evaluation of facial nerve function.

Grade	Functional impairment
1	Normal
2	Mild dysfunction (Slight weakness, complete eye closure and symmetric at rest)
3	Moderate dysfunction (Obvious weakness but not totally disfiguring, complete eye closure with effort and symmetric at rest)
4	Moderately severe dysfunction (Disfiguring weakness, incomplete eye closure and asymmetric at rest)
5	Severe dysfunction (extreme weakness with minimal executive function, no motion in forehead and incomplete eye closure)
6	Total paralysis <i>i.e.</i> no movement

improvement of the facial nerve paralysis after surgery. An intact canal wall mastoidectomy was done and granulations tissue was seen in the middle ear particularly over horizontal portion of the facial nerve where dehiscence of the canal was identified. The granulation tissues from dehiscence area of the facial canal were cleaned. The facial nerve paralysis recovery occurred in 11 cases to grade I (House-Brackmann) and 2 cases to grade II (House-Brackmann). One patient had purulent otorrhea at the time of examination and not required nerve decompression. The first patient recovered on 8th day whereas the last patient recovered on 45th day (Table 2).

4. Discussion

Facial nerve paralysis is a rare but a serious complication of AOM with functionally, emotionally and esthetically devastating consequences. 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[4]. AOM is a commonly encountered disease among children and early adulthood. In this disease, the inflammation is found in the middle ear cavity and mastoid whereas, in severe disease, the inflammation will spread to adjacent structures in the middle ear cleft. The fallopian canal is a bony canal covering the facial nerve which is involved in case of persistent inflammation of the middle ear cleft, leading to facial nerve paralysis. Paralysis of the facial nerve in a child or an adult will result in weakness of the facial muscles, impacting on verbal communication, social interaction in respect to facial expression, oral competence, taste, corneal protection, and vision. The facial nerve paralysis is more devastating when it is seen in pediatric patients where parents present with an unreasonable concern for the well being of their children[5]. The exact etiology of facial nerve paralysis in AOM is not well understood. The possible mechanisms of facial nerve paralysis include direct neurotoxic effects of middle ear effusion, inflammation, and edema of the nerve and ischemia[6]. Infections in the middle ear usually affect the facial nerve in the presence of the dehiscence of the fallopian canal, usually tympanic segment. In AOM, early facial nerve paralysis is thought to be due to toxic neuritis by direct involvement of bacterial toxins and it will lead to hyperemia or edema of the loose fibrous tissue of the nerve. This

Table 2. Detail clinical profiles of the patients with facial nerve paralysis with acute otitis media.

Patient number	Age (years)	Sex	Degree of paralysis	Pure tone audiometry	CT scan findings	Treatment	Recovery in days
1	1.0	Male	Incomplete	Not done	Not done	Myringotomy +Grommet	15 days
2	3.5	Male	Incomplete	Not done	OC	Myringotomy +Grommet	11 days
3	5.0	Female	Complete	Not done	OC	Myringotomy +Grommet	21 days
4	5.5	Female	Incomplete	Not done	Not done	Myringotomy +Grommet	15 days
5	7.0	Male	Incomplete	30 dB loss	Not done	Myringotomy +Grommet	14 days
6	8.5	Male	Incomplete	34 dB loss	OC	MMF	1 month
7	9.0	Male	Complete	30 dB loss	OC	MMF	45 days
8	11.0	Female	Incomplete	32 dB loss	OC	Myringotomy +Grommet	10 days
9	19.0	Male	Incomplete	40 dB loss	OC	Myringotomy +Grommet	12 days
10	32.0	Female	Incomplete	42 dB loss	OC	Myringotomy +Grommet	18days
11	36.0	Female	Incomplete	30 dB loss	OC	Myringotomy +Grommet	16 days
12	37.0	Male	Incomplete	32 dB loss	OC	Myringotomy +Grommet	8 days
13	65.0	Male	Incomplete	34 dB loss	OC	Myringotomy +Grommet	13 days

OC: opacification & coalescence; MMF: myringotomy+mastoidectomy+facial nerve decompression.

edema may be due to vasomotor paresis of the epineural vessels. The bacterial toxins in AOM may reach the nerve via dehiscence in the fallopian canal[7]. In the post-antibiotic era, the incidence of facial nerve paralysis due to AOM is 0.2% and so traditional treatment is encouraged towards timely eradication of infecting agent with antibiotic therapy[8]. 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. In one study, out of 2 758 patients, 40 were presented with facial palsy AOM[9]. Most of the patients suffering from facial paralysis with AOM have facial nerve lesion beneath the geniculate ganglion as this region usually have direct contact with the infections[10]. Diagnosis of facial nerve paralysis in AOM is based on physical examination and imaging. Patients present with otalgia, fever and headache. Otoloscopic examination shows congested tympanic membrane. The patient often shows radiographic evidence of facial canal dehiscence but in a few cases, it may not show the dehiscence as micro-dehiscence is not appreciated on thin slice CT. Even a very small communication to the facial nerve can provide infection and compression to the nerve and cause facial nerve paralysis. Fluctuations of the middle ear pressure may compress directly the facial nerve or compress the vascular plexus present over the facial nerve sheath and can cause temporary ischemic neuropraxia[11]. When AOM is complicated with facial nerve paralysis, treatment should include aggressive antimicrobial therapy for otitis media. Although myringotomy has an unknown outcome on curing the facial nerve paralysis, it still continues as the standard treatment care and remain part of the treatment until scientific study refute its role in AOM with facial palsy. We had done myringotomy along with ventilation tube insertion for preventing early closure and help to assess the middle ear. Facial nerve paralysis in AOM is an otolaryngologic emergency. Urgent myringotomy with or without placement of grommet and culture of the middle ear fluid with aggressive IV antibiotic therapy are required for the treatment. The role of IV corticosteroids in this clinical condition is controversial but often routinely used in the management of facial nerve paralysis in AOM[12]. If facial nerve paralysis does not improve

within one week of conservative treatment, a temporal bone CT scan is advised[13]. Electroneurography is an important tool to identify the prognosis and it can show the functional outcome of the facial nerve paralysis. It is usually done at 3 to 4 days after onset of complete facial nerve paralysis to find out the extent of the nerve injury. If the outcome of electroneurography is positive, a surgical decompression of the facial nerve is required. In majority of cases of facial nerve paralysis in AOM, the paralysis lasts for longer than 3 weeks[13]. In this study, the longest duration of recovery of the facial nerve was 45 d and the shortest was 8 d.

As the prevalence of facial nerve paralysis due to AOM is low, the treatment of choice is often difficult. Management of facial nerve paralysis includes multidisciplinary team efforts. The first step in the management is medical treatment targeted for underlying etiology like antibiotics or corticosteroids. Management with aggressive antimicrobial treatment and myringotomy will help to drain the pus from the middle ear cavity and release the pressure over the facial nerve. Oral corticosteroids help to reduce the inflammatory process in the middle ear. If pharmacologic interventions are failed, surgical interventions may be indicated to restore the function. Many physicians recommend aggressive treatment with antibiotic and myringotomy with or without grommet insertion[14]. In addition to this, steroid therapy is also advised for facial paralysis[15]. For two patients of our study, mastoidectomy and facial nerve decompression were done where intra-operative bony dehiscence and edema of the tympanic segment of the facial nerve were seen and this is similar to Tschiasny's theory which described by Zinis *et al.*[16]. This theory says infectious involvement of the facial nerve occurs via bony dehiscence and neurovascular communication between the facial nerve and middle ear. The study showed anatomical variations of the facial nerve, documented 55% fallopian canal dehiscence, which helps us to think about other pathological mechanisms, as facial nerve paralysis as a complication of AOM is uncommon[17]. One theory suggests that the infection causes compression of the vessels that feed the facial nerve and causes local ischemia and nerve infarction and leads to paralysis finally[18]. One study concluded the mechanism of the facial nerve paralysis in AOM

by direct involvement of the facial nerve to the bacterial or viral toxins[19]. The patients underwent operation showed improvement of the facial nerve regardless of the grading of the paralysis. The decompression of the facial nerve is usually done by removal of the matrix which drapes over the dehiscent nerve as it can be easily peeled off by using suction and sickle knife or gently peeling it off with help of a moist cotton ball followed by irrigation with saline. In this study, two cases underwent intact canal wall cortical mastoidectomy and granulations identified the tympanic segment of the facial nerve where bony dehiscence was identified. The granulation tissues from dehiscence area over the tympanic segment of the facial nerve were cleared. Eye care is also an important part of management if incomplete eye closure is seen in AOM with facial nerve paralysis. Ophthalmologic consultation is required if the patient complains any eye pain, redness or vision changes. In AOM with facial nerve paralysis, more than 95% of patients will have complete recovery whereas patients with complete facial paralysis have worse outcomes with complete recovery in 58%-70% only[20]. Aggressive and appropriate treatment in the earliest period help patients to recover from facial nerve paralysis.

Facial nerve paralysis has a tremendous impact on the patients and their family, particularly when the patient is a child. AOM with facial nerve paralysis is rare. We presented a small series demonstrating facial nerve paralysis with AOM. The treatment of this clinical entity included antibiotics and steroids. However, myringotomy with grommet insertion for drainage of fluid from the middle ear is helpful for immediate facial nerve decompression.

This study has a relatively small sample sizes due to the rarity of this clinical entity. However, the detail clinical profile of the patients with AOM and its management will definitely help to provide a management strategy and its awareness among patients suffering from AOM and facial nerve paralysis.

Conflict of interest statement

The authors report no conflict of interest.

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