

Case Report

Acute necrotizing otitis media in infant: a rare and challenging case

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ABSTRACT

Acute necrotizing otitis media (ANOM) is a highly virulent middle ear infection that results in necrosis of the tympanic cavity. Here a rare case report is presented of a 4 month infant who presented with bilateral ANOM and right facial nerve palsy which was aggressively managed with debridement and intravenous antibiotics obviating the need of surgery. The child was followed up for a period of 6 months where both the ears remained disease free accompanied with hearing gain and grade of facial palsy improvement from grade V to grade II. ANOM is still a possibility in the modern era of higher antibiotics and a high index of suspicion is required. Timely and aggressive management has to be done in order to prevent the dreaded complications and sequelae like facial nerve palsy and hearing loss which are likely to be permanent.

Keywords: Acute necrotizing otitis media, Facial palsy, Otitis media

INTRODUCTION

Acute necrotizing otitis media (ANOM) is a highly virulent middle ear infection that results in necrosis of the tympanic cavity. It was far more common in the pre-antibiotic era than it is today. Classically, ANOM occurs in infants and young children who are critically ill with scarlet fever, measles, pneumonia, or other systemic disease that suppresses immunologic defences. The bacterial origin is nearly always beta-haemolytic *Streptococcus*.¹ It has been reported in literature that during the first year of life 63-85% and by 2 years of age 66-99% of children suffer from an episode of acute otitis media.² There are notable common complications like hearing loss, mastoiditis and facial palsy but one of the most dreaded complications is ANOM. Here a rare case report is presented of a 4 month infant who presented with ANOM and right facial nerve palsy which was aggressively managed with debridement and intravenous antibiotics obviating the need of radical surgery.

CASE REPORT

An otherwise asymptomatic, healthy and adequately immunized 4-month-old male child presented with a 3 days history of high-grade fever, excessive inconsolable cry and bilateral brownish ear discharge.

The child was started on intravenous amoxicillin-clavulunate and anti-pyretics. On day 3 the patient developed bilateral blood tinged ear discharge with deviation of angle of mouth to the left side, drooling of saliva from the right angle of mouth and inability to close the right eye completely. On examination profuse mucopurulent discharge was present in bilateral external auditory canal. Right facial nerve lower motor neuron type palsy was seen (House Brackmann grade V) (Figure 1). Patient was switched to intravenous piperacillin-tazobactam and syrup prednisone along with adequate eye care for facial palsy. Suction clearance was done and copious mucopurulent discharge was suctioned out. Tympanic membrane was not visualized on otoscopy as

there was significant external auditory canal (EAC) edema. Culture swab was taken from both the ear canals.



Figure 1: Day-3 right facial grade V and 2-month follow-up with facial palsy grade II.

On day 6 culture swab was suggestive of *pseudomonas aeruginosa*. Culture directed antibiotics were started (intravenous *Meropenem*).

Otoendoscopic suction clearance was done due to no improvement in the ear discharge, which revealed necrotic tissue in both the external auditory canals and patient was immediately subjected to microscopic ear examination with removal of slough under general anesthesia (Figure 2).

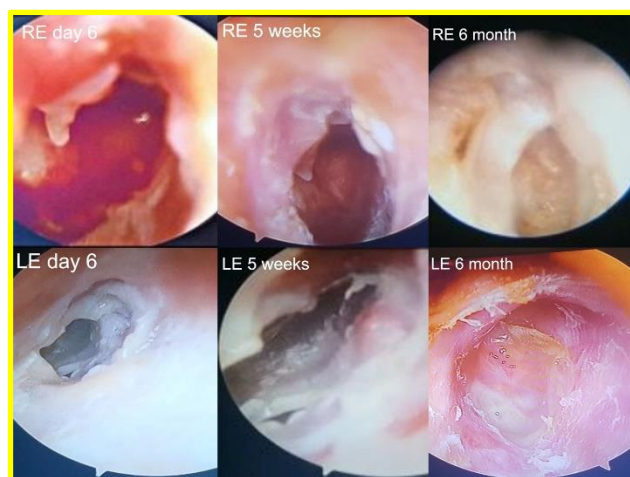


Figure 2: Progression of disease and healing as seen on otoendoscopy, day 6 both ears show slough and necrosis, at 6 weeks epithelization in right ear and granulations in left EAC and by 6 months both healed epithelized ears.

In the right external auditory canal entire canal wall skin was sloughed off with underlying bone exposed. Posterior bony canal wall showed a defect of about 4mm with exposed mastoid antrum and the tympanic membrane was absent. The middle ear mucosa was hemorrhagic and edematous.

In the left ear the tympanic membrane was intact with a defect in posterior bony canal wall of about 4 mm leading

into mastoid antrum and anterior canal wall defect with exposed temporomandibular joint capsule was visualized. Debrided tissue was sent for histopathology and was reported as necrotic tissue. The sample was negative for fungal elements.

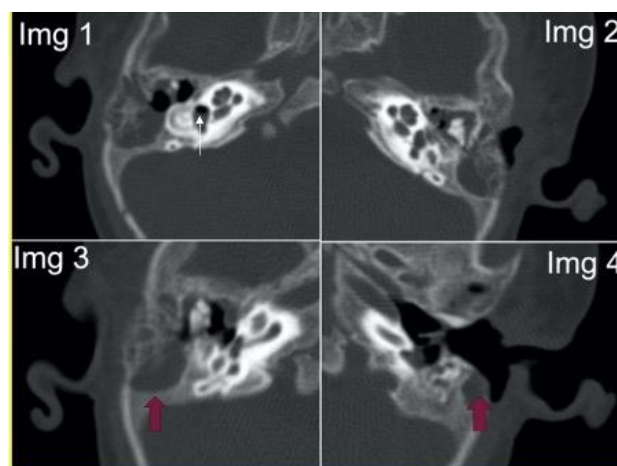


Figure 3: (Img 1) Air fluid level in labyrinth (white arrow), (Img 2) otomastoiditis, (Img 3, 4) significant EAC wall erosion (blue arrow).

In view of no perceptible hearing and extent of disease High resolution computed tomography (HRCT) scan of temporal bone was done. HRCT temporal bone with 0.625 mm cuts was taken which showed bilateral otomastoiditis with intact ossicular chain. Right labyrinth showed air fluid level, suggestive of labyrinthitis. Erosion of anterior and posterior walls of external auditory canals on both sides was noted. However, bilateral fallopian canals were intact (Figure 3).

Over the next 1 week the general condition of the patient improved and the ear discharge reduced. At 3 weeks microscopic examination of the ear was done which revealed minimal necrotic slough and discharge for which adequate suction clearance was done. Regular otoscopic evaluation was done with aural toileting.

On week 4 there was no evidence of discharge and no necrotic debris. Intravenous antibiotics were changed to oral *Clarithromycin* and oral steroids were discontinued after 2 weeks of usage.

On day 31 Both the middle ear showed no evidence of disease and patient was discharged on physiotherapy for facial weakness. On discharge facial function and hearing was not improved.

On 5-week follow-up, the ears were dry with no evidence of infection however, facial nerve examination revealed improvement in facial palsy grade from V to III and eye closure with maximum effort was present. In both ears satisfactory epithelization was present (Figure 2).

On 6 weeks follow-up right ear healing was satisfactory. Patient started minimal response to loud sound. Facial nerve was grade III. Left external auditory canal showed presence of granulations with narrowing. In view of canal stenosis, a snugly fitting FR 11 silicon catheter was used. (Figure 4)



Figure 4: Left external auditory canal stenosis and a 3 mm silicon stent fitted in situ.

On 8 weeks follow up the right ear shows marked healing (Figure 2) and left external auditory canal stent was in situ. Child started responding to sound stimulus very well. The facial palsy improved to grade II (Figure 1).

The child is under regular follow-up with both the ears completely disease free and well epithelized. On 6 months follow-up left EAC stent was removed and adequate diameter of canal was achieved (Figure 2).

DISCUSSION

Acute Necrotizing otitis media (ANOM) is an extremely virulent infection of the middle ear cleft associated with necrosis and sloughing off of the contents of tympanic cavity. The incidence of this condition has greatly declined in the present time due to advent of modern antibiotics. It is the disease of elderly immune-compromised patients having diabetes mellitus or human immunodeficiency virus.¹ However, ANOM has also been reported in infants and children who have immunocompromised status with childhood infections like measles, scarlet fever, pneumonia or other systemic illnesses.³

ANOM usually follows an episode of necrotizing otitis externa which progressively erodes the external auditory canal, tympanic cavity, skull base and eventually spreads to the cranial nerves and intracranial cavity.⁴ The most common organism documented in literature is beta hemolytic streptococcus however in our case it was pseudomonas aeruginosa.⁵ Similar finding was documented in a case report by Shen et al.³

The treatment is directed towards regular debridement of external auditory canal and middle ear, use of topical antibiotic drops, long term intravenous antibiotics and

nutritional support to the infant. Surgery is indicated only in refractory cases where radical debridement of temporal bone is the modality of choice. In our case the disease was managed with the above measures and the need for a radical surgery was negated.⁶

The association of facial nerve paralysis in children is well documented with acute otitis media, chronic otitis media with or without cholesteatoma, Lyme's disease, herpes zoster oticus and necrotizing otitis media.⁷ Though the chances of improvement of facial nerve function in children is higher as compared to adults, the prognosis is always guarded in cases of infectious etiology. The proposed mechanisms of involvement of facial nerve is by either the effect of cholesteatoma or diffusion of infection to the dehiscence fallopian canal, hence making the damage likely to be permanent.

Keeping this pathogenesis in mind the use of systemic corticosteroids is advocated in order to relieve the edema within the fallopian canal. In contrast to Bell's palsy or traumatic facial nerve palsy where the oral steroid therapy is initiated within 2 weeks of the onset of palsy, no such guideline has been mentioned in cases of infective etiology for facial nerve palsy in pediatric age group. However, in a study done by Adour in 1982 a 10 day course of corticosteroids was suggested.⁸ We have tried a course of oral dexamethasone for 14 days with no immediate clinical improvement in the facial palsy. Similar inference was made in the studies done by Wang et al, Duval et al and Shih et al in 2009-2010.⁹⁻¹¹

It has also been shown that contractures, muscle spasm and synkinesis develop on the ipsilateral side of palsy in about 5% of cases.¹² The therapeutic approach in infants requires a multidisciplinary team of otolaryngologists, paediatricians, ophthalmologists and physiotherapists. Facial nerve decompression is not recommended in children due to the risk of development of sensory neural hearing loss possibly due to the intricate relationship of the nerve with the vital structures like the labyrinth. Also, there are not many robust studies claiming the benefits outweighing the risks involved with surgery.^{13,14}

The mainstay of treatment remains aggressive debridement of ear and culture directed intravenous antibiotics along with adequate facial physiotherapy.

CONCLUSION

In the present era of higher antibiotics, ANOM is still a possibility even in otherwise healthy paediatric population with adequate immunization. The diagnosis is based on history and physical examination. Culture directed antibiotics should be started as soon as possible. Surgery remains the last resort to achieve disease clearance.

A high index of suspicion is required and timely and aggressive management has to be done in order to

prevent the dreaded complications and sequelae like facial nerve palsy and hearing loss which are likely to be permanent. Reassurance and motivation to the parents is extremely paramount and timely referral to otolaryngologist is highly stressed upon.

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