

CASE REPORT

Facial Palsy in an Extremely Low Birth Weight Neonate due to Acute Suppurative Otitis Media

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Abstract

We report an extremely low-birth-weight neonate with new onset unilateral facial palsy on ninth day of life. The child had neonatal sepsis, meningitis was ruled out and ear discharge was noted from the ipsilateral ear. Otoscopy suggested acute suppurative otitis media, while magnetic resonance imaging was suggestive of mastoiditis. The facial weakness improved with physiotherapy over next two weeks.

Key Words

Lower motor neuron, Mastoiditis, Facial canal, House-Brackmann stage, Physiotherapy

Introduction

Facial palsy is an uncommon manifestation in neonates. Typically, the onset of palsy is appreciated at birth. While birth trauma is by far the commonest cause, some cases result from developmental defects like Mobius syndrome and nerve hypoplasia. Postnatal onset can be due to trauma from respiratory interfaces, or other acquired lesions of inflammatory, infectious or neoplastic origin. We report acute suppurative otitis media as an unusual cause of postnatal onset facial nerve palsy in an extremely low birth weight neonate.

Case report

A 28-week female child, first of dichorionic diamniotic twins was born by preterm vaginal delivery because of pre-labor rupture of membranes. The child cried after birth; birth weight, length and head circumference were 980 grams (-0.57 z-score in Fenton 2013 charts), 38 cm and 25.5 cm, respectively. The mother was 31-year-old, with two previous first trimester abortions, hypothyroidism and gestational diabetes mellitus. She conceived

spontaneously, received single course of antenatal steroids and magnesium sulfate. There were no features of chorioamnionitis.

The child had respiratory distress syndrome needing surfactant administration. Nasal continuous positive airway pressure (nCPAP) was continued for seven days. On third day of life, baby developed lethargy, hemodynamic instability and the work up revealed blood stream infection due to *Pseudomonas aeruginosa*. Although baby improved, left facial nerve palsy was noted on ninth day of life (*Fig 1a*). The severity was graded as House-Brackmann stage IV. The possibilities considered were meningitis, brain abscess, pressure injury due to non-invasive respiratory support and idiopathic Bell's palsy. Cerebrospinal fluid examination and cranial ultrasound were normal. Physiotherapy was initiated in the subsequent days and serial monitoring of weakness was done. Eye care with banding and hydroxy methyl

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Fig 1a. Image highlighting left facial nerve palsy at diagnosis; **1b.** Pus discharge noted from left ear on day 18; **1c.** Magnetic resonance imaging (axial section of skull) showing obliteration of middle ear cavity on left and bulky mastoids; **1d.** Facial palsy as on day 35

cellulose drops was provided to prevent exposure keratitis.

On day 18 of life, pus discharge was noted from the left ear (*Fig 1b*). In the subsequent days, we have noted improvement in the facial palsy, with partial closure of eyes, some frowning on forehead and lesser deviation of mouth to right (House-Brackmann stage 3). Tragus sign was negative, making otitis externa less likely. Otoscopy revealed perforated tympanic membrane. Pus was sent for culture, which again grew *Pseudomonas aeruginosa*. An MRI brain was done to look for intracranial complications, which showed obliteration of middle ear cavity and mastoid suggestive of acute suppurative otitis media (ASOM) with mastoiditis (*Fig 1c*). As a result, antibiotics were continued for a duration of 14 days. The physiotherapy was continued and weakness gradually improved. By day 35 of life, the facial weakness was graded House-Brackmann stage 2 (*Fig 1d*).

Discussion

Facial nerve palsy is an uncommon finding in neonates. While approaching a neonate with facial palsy, the age at onset and the type of lesion are important diagnostic clues. Most cases presenting at birth are related to birth trauma and underlying congenital disorders like Mobius syndrome and nerve hypoplasia.^[1] Onset after birth can be seen in

infectious, inflammatory, traumatic, compressive and neoplastic causes.^[2,3] It is important to differentiate between upper (UMN) and lower motor neuron (LMN) lesions; the former have preserved furrowing of brow, eye closure and blinking.

The index case had postnatal onset of unilateral LMN facial palsy, suggesting an acquired lesion in the facial nerve motor neuron or in the facial nerve. The possibility of trauma to facial nerve related to nCPAP interface was considered. However, the onset was noted after cessation of nCPAP. As pus discharge was noted in the ipsilateral ear, the possibilities of otitis externa and ASOM with or without mastoiditis were considered. A negative tragus sign followed by Otoscopy clinched the diagnosis.

ASOM is not an uncommon occurrence in neonates, however, it's rarely diagnosed because of technical difficulties. The incidence ranged from 2.3 to 25% in various studies.^[4] Preterm neonates are predisposed to ASOM because of the anatomical orientation of eustachian tube (horizontal and patulous), immature immune system, persistent supine posturing and inability to clear the eustachian tube resulting in negative pressure in middle ear. The additional risk factors include male sex, nasotracheal intubation, mechanical ventilation,

neuromuscular diseases and craniofacial anomalies. ^[4] None of the later factors were present in the index case.

Facial nerve palsy as a result of ASOM in neonate is very rarely reported, although it was reported in older children. ^[5] In the facial canal, facial nerve passes very close to middle ear and mastoids and hence prone to injury. Moreover, the ossification is incomplete and there are several dehiscences in the bony covering of facial canal, bridged by soft tissue. Also, the bony structure separating the middle ear cavity and mastoid from the facial canal is thinner and the mastoid antral cells are underdeveloped. ^[6] In the index case, the injury to facial nerve could have occurred in either the tympanic segment of facial canal (secondary to ASOM) or the mastoid segment (secondary to mastoiditis). But the differentiation was not possible in a preterm neonate, because it is challenging to assess the function of chorda tympani and nerve to stapedius.

The index case highlights an uncommon, but important cause of facial nerve palsy in neonatal age.

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Conflicts of Interest

There are no conflicts of interest.

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