**A rare case of acquired infantile Bell’s palsy**

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**INTRODUCTION**

Bell’s palsy is an acute onset, acquired, isolated peripheral facial palsy which usually follows a viral illness.1 Named after Sir Charles Bell who first described a weakness in facial nerve, it has an incidence of 6.1 cases per year per 100000 in those aged between 1 and 15 years.2 Bell’s palsy is unusual in infancy and we report one such 3-month-old infant with idiopathic facial nerve palsy below.

**CASE REPORT**

A 3-month old male infant presented to our emergency department with new onset feeding complaints and deviation of angle of mouth to left side while crying for 1 day. Birth history was significant with late preterm vaginal delivery, respiratory distress at birth requiring intratracheal surfactant and perinatal asphyxia stage with stage 2 hypoxic ischemic encephalopathy. He was discharged after neonatal intensive care for 14 days. He was followed up at high risk new-born clinic and was doing well prior to these complaints.

The day prior to presentation mother noticed right side of face did not move when he cried and he could not fully close his right eyelid. Baby was otherwise active and playful. Child did not have any associated fever, rash, swelling of the parotid glands or ear discharge. He had received pentavalent vaccine 15 days prior with no adverse reactions. Neurological examination was notable for lack of forehead wrinkling on right while crying, absence of movement of right cheek, and corner of mouth on right side while crying and inability to completely close the right eyelid. (Figure 1) No motor or other cranial nerves affection were noted. All development milestones were attained for the age with normal neonatal reflexes.

As there was concern for sepsis, sepsis screen, blood and CSF cultures were undertaken and were u revealing. Ultrasound of neck revealed a small, ovoid, well defined, heterogeneous, hypoechocic mass lesion in the superficial lobe of right parotid gland measuring 15×10×10 mm. There was no associated cervical lymphadenopathy.

Oral prednisolone was started at 1 mg/kg/day, given totally for 10 days as we initially suspected inflammatory aetiology for the palsy. He demonstrated dramatic improvement in symptoms from 5th day of therapy. Post treatment head and neck MRI showed T1W, FLAIR hypointensities involving both cerebral hemispheres and...
subcortical white matter regions attributed to the hypoxic insult in the neonatal period, but no mass lesions in parotid with normal bilateral seventh/eighth cranial nerve complexes and cerebellopontine angles. On follow-up at one month post discharge, he was thriving well with no deficits. (Figure 2)

![Figure 1: Infant showing right Bell’s palsy.](image1)

**Figure 1: Infant showing right Bell’s palsy.**

![Figure 2: No residual deficits post treatment with steroids.](image2)

**Figure 2: No residual deficits post treatment with steroids.**

**DISCUSSION**

Pediatric facial nerve palsy can be congenital (traumatic, syndromic, genetic) or acquired. Bell’s palsy is the idiopathic and the most common form constituting nearly 50% of the cases. It usually has a favourable prognosis with spontaneous resolution without sequelae within 3 months and the degree of recovery is dictated by the severity of paralysis at onset.¹

Clinically absence of nasolabial groove motility, asymmetry of the face with buccal deviation when crying, rises the suspicion of unilateral facial palsy in infants. The child cannot close the eye due to a complete absence of movement on the affected side hampering breastfeeding in severe cases.²,³

EBV, Hemophilus influenza, TB, CMV, HIV, Adenovirus, Rubella, Mumps, Mycoplasma pneumoniae are known acquired infectious causes. Tumors (schwannomas, hemangiomas, bone tumors, rhabdomyosarcoma, histiocytosis), vasculitis (Henoch-Schönlein purpura, Kawasaki disease), temporal bone fracture or surgery of middle ear, mastoid or parotid glands can also cause facial palsy.⁵

A comprehensive history is necessary to investigate regarding the onset and the time course of the paralysis and its eventual progression. Blood pressure measurements and blood counts should be done in all cases. Specific laboratory and imaging tests are recommended for patients with recurrent paralysis or when there has been no improvement after 3 weeks of therapy. Imaging studies are necessary when the child has associated neurological manifestations or in suspected middle ear pathology. Electrophysiological studies, though useful to identify the cause of the paralysis, define the prognosis and follow-up of functional recovery, are not considered necessary in all pediatric cases.⁶

The treatment of facial palsy is aetiology and severity related, aimed to resolve the specific underlying cause. Cortico-steroids, preferably oral prednisone 1-2 mg/kg per day for 10 days with gradual dose tapering, is recommended preferably within 3 days from onset of symptoms. The majority of children have a spontaneous recovery. In children with persistent, severe paralysis, direct neurorrhaphy or dynamic facial reanimation maybe required. Regenerative medicine with stem cells to restore neuronal integrity could offer future options for the condition.⁷

Our infant had right sided LMN type facial palsy, cause for which couldn’t be ascertained and hence considered Bell’s palsy or the idiopathic variety. Ultrasound neck had revealed an ovoid mass in parotid gland of possible inflammatory aetiology which disappeared on treatment with steroids with no residual palsy post treatment, highlighting the benign course of the illness.

**CONCLUSION**

Resolution of symptoms of Bell’s palsy is important to prevent long term side effects. Though rare in early infancy, low dose corticosteroids have shown to be effective and can be used once other causes are excluded.

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