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Case Series

Neonatal facial palsy without forceps use: report of two cases and review

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ABSTRACT

Neonatal facial palsy is a very rare diagnosis with only a few previously published cases of newborns, it can have varying etiologies apart from traumatic causes. The congenital, infectious, inflammatory, neoplastic causes must be eliminated before making the definitive diagnosis of traumatic facial paralysis. A multidisciplinary approach is recommended to differentiate various causes and initiate treatment in a timely manner. Corticosteroids have been used to minimize nerve damage and hasten recovery in the most of studies. We report in this work two cases at 2 and 3 days old of congenital unilateral facial palsy in newborns without history of obstetrical trauma with a good prognosis after corticosteroids with complete resolution.

Keywords: Newborn, Facial palsy, Corticosteroid

INTRODUCTION

Facial palsy is not a common phenomenon in newborns. It can be congenital or developmental. Congenital palsy occurs due to perinatal trauma. Developmental palsy occurs due to error in development, for example, aplasia or hypoplasia of the cranial nerve nuclei, nuclear agenesis, and aplasia or hypoplasia of the facial nerve. This can be associated also with syndromes like Mobius syndrome.[1]

Most traumatic facial nerve palsies resolve spontaneously within days, and surgical intervention is rarely needed.[2] The role of steroids in treatment of the condition is an ongoing debate. Prognosis is good, though residual dysfunction may occasionally be seen. Here, we present two cases of traumatic unilateral facial palsy without forceps use with full recovery in a few days.

CASE SERIES

Case 1

A 1-day old male infant was born at 39 weeks of gestation by spontaneous vaginal delivery following an uneventful pregnancy (the mother was 32 years old, no gestational diabetes or other complications). The labor delivery monitoring was without worthy alteration. He required no resuscitation at birth and the Apgar score was respectively 10 and 10 at first and fifth minute. His birth weight was 3000 g (appropriate for gestational age), he suffered no respiratory distress.

The physical examination showed a Charles bell sign made of an asymmetry of the face with mouth left side deviation and incomplete right eye closure during crying which suggested the diagnosis of right facial nerve palsy (Figure 1). Ear, nose and throat (ENT) exploration revealed an inferior marginal perforation of the right eardrum without associated otorrhea, the patient was active and alert. He had a normal pulse rate and blood pressure readings compatible with his age. His head circumference was 34 cm with a normal head shape and flat anterior fontanel.

Initial laboratory evaluation was unremarkable (hemoglobin: 18,5 g/dl; platelet count: 245000 /mm^3; leucocytes: 10400/mm^3; C-reactive protein (CRP): 2.5 mg/l; calcemia: 84 mg/l). Brain magnetic resonance imaging (MRI) showed congestion of the right middle ear reaching the right facial nerve (Figure 2).

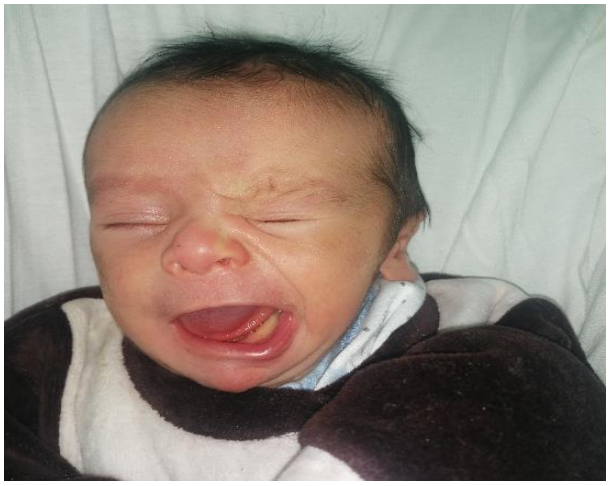

Figure 1: Right-sided lower motor neuron facial palsy (case 1).

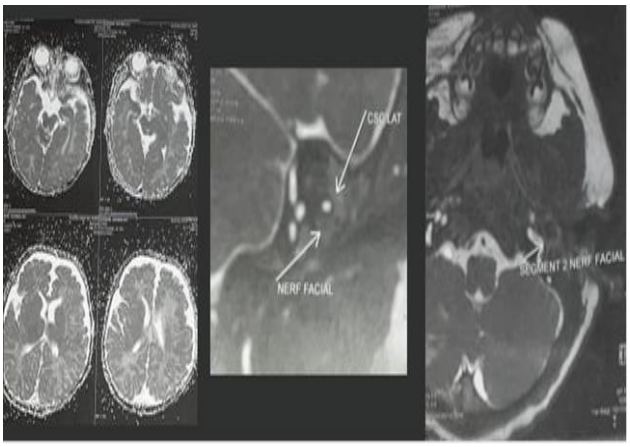

Figure 2: MRI axial views scans at 6 days of life showing congestion of the right middle ear reaching the right facial nerve.

In the light of these data, we concluded to an obstetric trauma secondary to a physical pressure during the passage of the birth canal.

The initial course of action was to start a corticosteroid therapy, artificial tears and eye padding to prevent dryness of the affected eye. The evolution was marked by the disappearance of the paralysis within 10 days (Figure 3).

Case 2

A 3-day old male infant was born at 38 weeks of gestation by spontaneous vaginal delivery following an uneventful pregnancy (the mother was 27 years old, no gestational diabetes or other complications). With meconium-stained amniotic fluid. He required no resuscitation at birth and the Apgar score was respectively 10 and 10 at the first and fifth minutes. His birth weight was 3450 g (appropriate for gestational age), he suffered no respiratory distress. He was admitted to our department for a fever (39ºC).

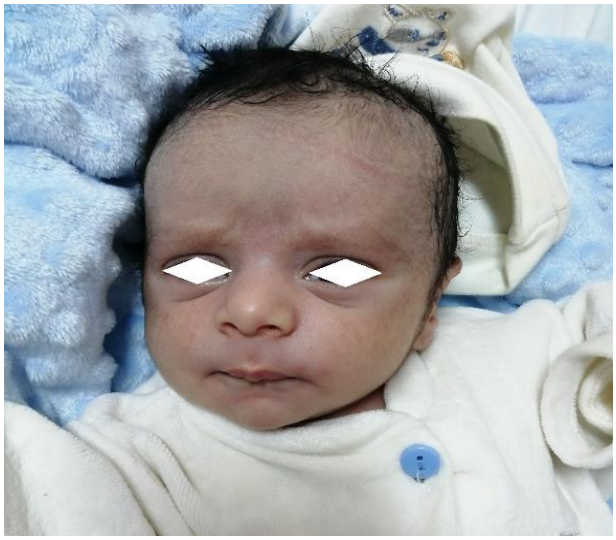

Figure 3: Complete recovery on follow-up.

The clinical examination did not show any abnormality. Initial laboratory evaluation shows (hemoglobin: 17.3 g/dl; platelet count: 315000/mm^3; leucocytes: 17350/mm^3 CRP: 42 mg/l; calcemia: 80 mg/l). the result of the lumbar puncture was normal.

The initial course of action was to start an antibiotic therapy based on third generation cephalosporin and aminoglycoside. Four days later the newborn has developed facial asymmetry with a Charles bell sign (Figure 4).

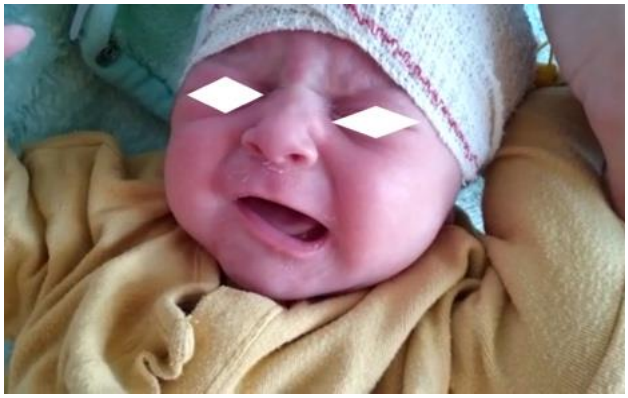

Figure 4: Right-sided lower motor neuron facial (case 2).

The decision was to start corticosteroids, we did not do any radiological imaging given the favorable evolution in a few days. The facial paralysis was gone in 7 days.

DISCUSSION

Neonatal nerve palsies can be either developmental or acquired. The incidence in the literature has been quoted as 2 per 1000 births most commonly caused by birth trauma. Rarer associations include Möbius's syndrome, hemifacial microsomia, Goldenhar's syndrome, DiGeorge

syndrome and Poland syndrome. Neonatal idiopathic facial palsy is even rarer. Some of the risk factors for birth trauma are a particular position of fetus where the face is compressed against the mother's sacral promontory or fetus's shoulder, application of forceps, primigravida mother and baby's birth weight more than 3500 g. However, Laing et al in a retrospective case–control study found no association between permanent congenital facial palsy and presence of risk factors for trauma during delivery. They emphasized that it being a serious medicolegal issue for obstetricians, care should be taken before announcing that facial palsy is due to birth trauma.[3]

Syndromic and genetic causes represent a small yet diverse group of etiologies associated with congenital facial palsy. It can be the result of central brain malformation syndromes, such as Arnold Chiari disorder, often along with other cranial nerves dysfunction.[4] Facial palsy is one of the diagnostic criteria for Möbius syndrome, but it is usually bilateral.[5] It has also been described in patients with branchial arch anomalies like Goldenhar, Poland, and branchiootorenal syndromes.[6,7] Rarely, hereditary neuromuscular disorders can present with facial palsy that is noticeable at birth. Examples of such disorders include congenital myotonic dystrophy and congenital myasthenic syndromes.[8] There is a current debate about new emerging genetic loci possibly responsible for a special disorder of hereditary congenital facial paralysis, which has been reported in a few families.[9]

There are also disorders related to absence of the facial muscles that can mimic Bell's palsy, which are also congenital. Syndromes such as hemifacial microsomia, and Cayler cardiac syndrome can also present with congenital Bell's palsy. Facial nerve palsies that are related to birth trauma are likely to resolve by 3 to 6 months of age without intervention.[10]

Although forceps delivery is a significant risk factor for facial nerve palsy, However, Altawil et al in a retrospective case–control study found that it could occur spontaneously in some infants due to the compression on the nerve prior to or during the process of delivery, which has been suggested by several previous studies. Direct pressure on the nerve on the mastoid region by bony structures such as the sacral promontory or ischial spines could cause nerve injury.[2] In a recent study the use of CPAP has also been implicated in the causes of neonatal facial palsy.[1]

Diagnosis of lower motor facial palsy as in this newborn is by physical examination and careful analysis of delivery history like history of prolonged labor, forceps delivery, periauricular or facial ecchymosis, or hemotympanum.[11] Mostly, traumatic lower motor facial nerve palsy in a newborn is self a resolving condition. Dryness of the affected eye is prevented by artificial tears and eye padding. If there is no improvement by 3 months of age, then electromyography (EMG) should be done. In infants who have a clinically complete facial nerve palsy, needle EMG may show some potentials on stimulation of nerve which tells that the nerve is still in continuity and have the potential for regrowth. Motor nerve conduction study (NCS) (motor NCS or electroneurography or evoked EMG) is done for prognostication.[12] This test involves stimulation of the main branch of the facial nerve near the stylomastoid foramen and recording the compound muscle action potential in millivolts. In the case of permanent facial nerve palsy, definitive treatment involves muscle and nerve transplantation.[13]

Pharmacological treatment options in neonatal facial palsy are controversial. Since inflammation and edema have been implicated in its pathogenesis, corticosteroids have been used to minimize nerve damage and hasten recovery.[14] However, other studies have shown no benefit of corticosteroids.[15]

Our newborn has evolved well under oral corticosteroid therapy only and the paralysis disappeared in 10 days without sequelae. We concluded through our experience that neonatal facial palsy due to birth trauma is not always associated with the use of forceps, then the importance of careful clinical examination. We also concluded to the good prognosis of this pathological condition specially under oral corticosteroid.

CONCLUSION

Congenital facial nerve paralysis should be differentiated from traumatic facial nerve paralysis as this determines the course of the disease process and treatment plan.

Careful physical examination and detailed birth history help in finding out the cause of traumatic facial paralysis.

Resolution of the symptoms on neonatal facial palsy is important to prevent long-term side effects that include social isolation, future surgeries, and short-term difficulties with eating.

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