

Reversible congenital facial nerve palsy: An uncommon cause of asymmetric crying facies in the newborn

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Citation

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Abstract

Neonatal asymmetric crying facies is commonly due to the absence or hypoplasia of the depressor anguli oris muscle on one side of the mouth and is sometimes associated with major congenital anomalies. Infrequently it may be due to unilateral partial peripheral facial nerve paralysis. Here we report a case of congenital peripheral facial palsy in a newborn probably caused by trauma to the facial nerve during labor which improved spontaneously.

INTRODUCTION

Neonatal asymmetric crying facies, described 75 years ago [1], is commonly due to the absence or hypoplasia of the depressor anguli oris muscle on one side of the mouth, and is associated at times with major congenital anomalies, most commonly in the cardiovascular system. Infrequently it may be due to unilateral partial peripheral facial nerve paralysis. Asymmetric crying facies caused by absence or hypoplasia of the depressor anguli oris muscle or developmental anomaly of facial nerve or its nucleus is unlikely to recover and may require surgical correction for cosmetic reasons. Pooled data from 5 prospective studies estimate the incidence of neonatal asymmetric crying facies to be approximately 1 per 160 live births [2]. The reported incidence of congenital facial palsy (CFP) varied from 0.2-6.9% of all live births [3]. A recent estimate from the United States found the incidence of CFP to be 2.1 per 1000 live births [4]. There are no reports available from India regarding the incidence of congenital facial palsy though the incidence of congenital asymmetrical crying facies has been reported to be 6.3 per 1,000 infants [5]. Here we report a case of congenital peripheral facial palsy in a newborn which improved spontaneously.

CASE REPORT

A female baby was born to a sixth gravida woman at full term by emergency caesarian section for obstructed labor. She was brought to the hospital for obstructed labor and had been earlier managed outside by an untrained Dai (Traditional Birth Attendant who assists the mother during childbirth. She may have acquired skills by delivering babies

herself, or through apprenticeship of other Traditional Birth Attendants, without any formal training). There were five previous female living issues, all were delivered at home and are apparently alive and well. The maternal pelvis was asymmetric in dimensions due to polio leading to cephalopelvic disproportion. On per vaginal examination, the right pelvic wall was divergent while left pelvic wall was convergent with prominent left ischial spine. The baby was born through thick meconium stained liquor and had meconium aspiration syndrome which subsided at 72 hours of life. APGAR scores at 1, 5 and 10 minutes were 4, 7 and 8.

The baby was noticed to have complete left hemi facial palsy at birth (figure-1). The weakness was lower motor neuron type as there was absence of forehead wrinkles. There was no other cranial nerve palsy or associated major malformations and rest of the neurological examination was normal. Cranial ultrasound done at 72 hours and at seventh day was normal. The facial palsy improved spontaneously within a week and at discharge on day 7 of life, there was only minimal facial asymmetry on crying.

Figure 1

Figure 1: Left facial nerve paralysis with deviation of angle of mouth, loss of nasolabial fold, wide left palpebral fissure and absence of forehead wrinkles.



DISCUSSION

Congenital facial palsy (CFP) is clinically defined as facial palsy of the 7th cranial nerve which is present at birth or shortly thereafter [1]. This is an infrequent condition; the most recent estimate of the incidence of CFP reported is 2.1 per 1000 live births [4]. The suggested causes of facial palsy include perinatal trauma, intrauterine posture, intrapartum compression, and familial and congenital aplasia of the nucleus; the last being most frequently reported for bilateral cases [6]. Harris et al divided neonatal facial paralysis in two types; conditions acquired during or soon after birth, and conditions that result from developmental disorders [7].

There are a number of syndromes which encompass congenital facial palsy as part of their symptoms, including the cardiofacial, Moebius, Poland's, and Goldenhaar's syndrome [6, 8]. The cardiofacial syndrome comprises facial asymmetry when crying, but not at rest. This is attributed to an isolated weakness of the depressor anguli oris and quadratus labii inferiores muscles; the syndrome has been linked to congenital heart defects. Moebius syndrome is a

bilateral facial and abducens nerve palsy. Necropsy studies in patients of Moebius syndrome have shown defects ranging from hypoplasia to agenesis of the respective cranial nuclei. Poland's and Goldenhaar's syndromes are only occasionally associated with facial palsy. Some cases of CFP have been attributed to agenesis of the petrous portion of the temporal bone, with resulting agenesis of the facial and auditory nerves, the external ear, and the mastoid region [9]. Underlying CNS abnormalities were found using MRI scans, in four of 15 (27%) patients who presented with CFP as their sole symptom suggesting that a central cause is not infrequent [6].

Though the causes of CFP are known to be varied, but the focus so far has been on perinatal factors. The majority of neonatal facial palsies are caused by trauma to the facial nerve during labor. There are ample opportunities for the extracranial facial nerve to be damaged during birth owing to its relatively superficial course; several mechanisms have been suggested, apart from instrumentation in assisted delivery. These include: intrauterine posture, where pressure from the shoulder produces a notable displacement of the jaw and the periaural portions of the head, resulting in sufficient pressure on the peripheral portion of the facial nerve to cause facial weakness; and intrapartum compression, where the foetal head is compressed against a maternal bony prominence such as the pubic rami, the ischial spines, or the sacral prominence, again damaging the peripheral facial nerve [9, 10, 11, 12]. The most probable cause of facial palsy in the index case was intrauterine facial nerve compression by the left ischial spine in maternal pelvis due to asymmetric pelvic wall. Falco and his coworker found forceps delivery, birth weight of 3500 gm or more, and primiparity to be significant risk factors for acquired facial palsy [2]. Traumatic facial palsy in neonates is associated with good prognosis as seen in the index case. In contrast nontraumatic facial palsies carry a poor functional outcome [13, 14].

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