

## CASE REPORT

## FACIAL PALSY IN A NEWBORN: A CASE REPORT

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**Received:** 12-Sep-2022; **Revised:** 10-Dec-2022;**Accepted:** 15-Dec-2022; **Published:** 20-Dec-2022**Copyright:** © 2022. Snezana Jancevska, Sanja Ristovska, Igor Isjanovski, Nikolina Zdraveska. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.**Competing Interests:** The author have declared that no competing interests

Unilateral congenital facial palsy in the newborn occurs more often as isolated than in the context of syndromes or developmental defects. Recognition of the etiological factor, the severity of the clinical presentation, the dynamics of recovery are a guide for a multidisciplinary approach, a range of investigations and treatment. In a non-therapeutic approach, unsupported by sufficiently reliable data from the literature, there is a dilemma whether and how long one has to wait and observe the improvement of clinical results, before determining the range of examinations in the newborn. In facial paresis of prenatal or perinatal origin, identical to non-congenital Bell's palsy, infectious and anatomical-structural causes should initially be excluded. This paper presents a case of a symmetrical hypotrophic premature infant with a manifestation of acute Bell's palsy at birth, with negative biomarkers for infectious etiology and a normal brain ultrasound. The newborn underwent a special neonatal care with targeted local treatment. Towards the end of the first postnatal week, there was an evident withdrawal of clinical symptomatology.

## ПРИКАЗ НА СЛУЧАЈ

## ФАЦИЈАЛНА ПАРЕЗА КАЈ НОВОРОДЕНЧЕ: ПРИКАЗ НА СЛУЧАЈ

Снежана Јанчевска<sup>1</sup>, Сања Ристовска<sup>1</sup>, Игор Исјановски<sup>2</sup>, Николина Здравеска<sup>3</sup><sup>1</sup> Универзитетска клиника за гинекологија и акушерство; Универзитет Св. Кирил и Методиј во Скопје, Медицински факултет, Република Северна Македонија<sup>2</sup> Универзитетска клиника за очни болести; Универзитет Св. Кирил и Методиј во Скопје, Медицински факултет, Република Северна Македонија<sup>3</sup> Универзитетска клиника за детски болести; Универзитет Св. Кирил и Методиј во Скопје, Медицински факултет, Република Северна Македонија**Извадок****Цитирање:** Јанчевска С, Ристовска С, Исјановски И, Здравеска Н. Фацијална пареза кај новороденче: Приказ на случај. Арх Ј Здравје 2022;14(2)

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**Клучни зборови:** новороденче, Бел-ова парализа, пренатална траума**\*Кореспонденција:** Снежана Јанчевска, Универзитетска клиника за гинекологија и акушерство, Универзитет Св. Кирил и Методиј во Скопје, Медицински факултет, Република Северна Македонија.

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**Примено:** 12-сеп-2022; **Ревидирано:** 10-дек-2022;**Прифатено:** 15-дек-2022; **Објавено:** 20-дек-2022**Печатарски права:** ©2022 Снежана Јанчевска, Сања Ристовска, Игор Исјановски, Николина Здравеска. Оваа статија е со отворен пристап дистрибуирана под условите на некаленизирана лиценца, која овозможува неограничена употреба, дистрибуција и репродукција на било кој медиум, доколку се цитираат оригиналните автор(и) и изворот.**Конкурентски интереси:** Авторот изјавува дека нема конкурентски интереси.

Еднострана вродена парализа на лицето кај новороденчето почесто се јавува изолирано, одошто во склоп на синдроми или од дефект во развојот. Препознавањето на етиолошкиот фактор, тежината на клиничката презентација, динамиката на опоравувањето ќе бидат смерница за мултидисциплинарен пристап, палета на иследувања и третман. При вон-терапевтски приод, непоткрепен со доволно сигурни податоци од литературата, постои дилема дали и колку да се чека и обсервира подобрувањето на клиничките резултати, пред да се одреди палетата на иследувања кај новороденото дете. Кај фацијалната пареза од пренатално или перинатално потекло, идентично како кај неконгениталната парализа на Бел, првично треба да се исклучат инфективни и анатомско-структурни причини. Во овој труд се презентира случај на симетрично хипотрофично прематурно новороденче со манифестација на акутна парализа на Бел на раѓање, со негативни биомаркери за инфективна етиологија и уреден ултрасонографски наод на мозочните структури. Новороденото дете беше на специјална неонатална нега со целно спроведуван локален третман. Кон крајот на првата постнатална седмица покажа евидентно повлекување на клиничката симптоматологија.

## Introduction

The presentation of congenital facial palsy in neonatal period is represented by asymmetric facial movements on the affected side, incomplete eye closure, and possible feeding problems. In the future development of the child, the untreated facial palsy may affect the speech, the facial emotional expression or chewing and may also lead to cosmetic discomfort and social isolation. The congenital facial palsy can be divided into traumatic or non-traumatic (developmental), unilateral or bilateral, total (palsy) or incomplete (paresis); it can occur as an isolated change or as part of other syndromes. The incidence of facial palsy in neonatal period is 0.8-2.1 per 1,000 live births. Bell's palsy is an idiopathic mononeuritis. It is a unilateral facial palsy and occurs both in adults and in children<sup>1</sup>. There is insufficient data in the literature on the incidence and frequency of this condition in premature newborns.

The seventh cranial nerve (nervus facialis) is a mixed nerve that contains motor, parasympathetic and sensory fibers. This nerve provides innervations to the facial muscles and is responsible for the perception of taste in the first two thirds of the tongue. The movements of the lateral side of the mouth and of the nasolabial fold are enabled by a group of four muscles, the most important of which is the musculus depressor angularis oris. All of these muscles are innervated by a branch of the seventh cranial nerve, and, in case of injury, there is a dysfunction of these muscles, which results in asymmetric facial expression.

From etiological point of view, the congenital facial palsy can be considered a traumatic or developmental disorder of brain structures or of facial nerves<sup>2</sup>. The traumatic cause for the occurrence of facial palsy is much more common, and the risk factors include: termination of vaginal birth by using typical forceps with extended second delivery time; a primipara mother, giving birth to a macrosomic fetus (> 3,500 g), cranio-pelvic disproportion<sup>3</sup>. Facial nerve palsy as a secondary trauma when using forceps at birth was first studied by Landouzy as a research subject of his doctoral thesis in 1839<sup>4</sup>. Intrauterine trauma occurs due to increased pressure on the face of the fetus with the sacral bone at birth<sup>5</sup>. Recent studies show that traumatic facial palsy may be secondary to the use of continuous positive air pressure (CPAP) nasal application, due to the continuous and severe pressure on the newborn's face<sup>6</sup>. It is considered that the underlying patho-physiological process is ischemic tissue damage, mediated by free radicals<sup>7</sup>. Given that this is about a very young age group and that there is nonlinear correlation with the type and duration of injury, the full knowledge of the patho-physiological mechanism remains unknown<sup>8</sup>.

From the point of view of developmental disorders of brain structures and of facial nerves, the congenital facial palsy can be presented as part of a series of other syndromes, such as Möbius syndrome, hemifacial microsomia, velocardiofacial syndrome (DiGeorge Syndrome), CHARGE syndrome, facio-scapulo-humeral muscular dystrophy, Goldenhar syndrome. The intrauterine

hypoplasia of the nucleus of the seventh cranial nerve is often associated with congenital malformations and permanent facial palsy<sup>9</sup>.

Teratogenic risk factors during pregnancy, such as thalidomide and misoprostol, are responsible for the occurrence of facial palsy in newborns.

### A Case Report

The presented case is a third child from a third planned, desired and controlled pregnancy of a 33-year-old mother. In this pregnancy, the mother was exposed to multiple risk factors: pre-existent arterial hypertension, treated with methyl-dopa, as well as with gestosis of hypertension. In the prenatal period, the fetus was diagnosed with intra-uterine growth retardation (IUGR), decreased amniotic fluid (Oligohydramnion). Due to the condition

of fetal distress, the birth was terminated via abdominal surgical route, with emergency caesarean section; the fetus was extracted in capital presentation; a male newborn was born with birth weight of 2,100 g, Apgar score 7/8, with gestational maturity suitable for 36 gestational weeks or a late preterm newborn. The newborn was in good general condition. According to the birth weight and gestational age illustrated on the percentile curve, the newborn was hypotrophic. The head was properly configured, with craniofacial symmetry. Decreased movements on the left side of the face were noticeable. When crying, the nasolabial fold was lost; the newborn was unable to move the lower muscle of his lip, he had the so-called „limp“ lip and pulled the lip to the side of the mouth that was not affected. The eye on the left side was partially closed (Figure 1 and Figure 2).



**Figure 1 and Figure 2** Newborn with left-sided facial palsy, presented when crying and when not crying

In the first hours after birth, peripheral venous blood was collected for examination of biomarkers to rule out congenital infection (complete blood count with leukocyte formula, C-reactive protein, procalcitonin, blood culture). The results of the examination were negative. Ultrasonographic examination of the central nervous system showed an orderly finding of brain structures.

The newborn infant was treated with special care, which included gentle minimal manipulation, general measures for protection from cold and drafts and local treatment of the eyes with a solution for prevention of eye irritation, bathing the face with warm water.

## Discussion

There is a logical link between intrauterine risk factors and the clinical expression of facial palsy, as well as the absence of facial asymmetry in the newborn. The reduced volume of amniotic fluid prevents free movement

of the fetus and leads to anchoring and complete immobility, followed by pressure on the supine side in the pelvic bone structures (sacral part). All these unfavorable conditions for the fetus indicate a possible etiology of facial palsy in the newborn.

The compromised fetal mobility and dull pressure affect blood circulation, causing mild to moderate tissue ischemia. Timely completion of the delivery prevents reflection on soft and bony structures in terms of facial developmental asymmetry of the supine side relative to the free side.

Differentially diagnosed, facial palsy should be distinguished from congenital hypoplasia of musculus depressor angularis oris, which is a benign change and causes facial asymmetry when the newborn is crying<sup>9</sup>.

There are grading systems and scales for assessing facial palsy. The most commonly used are the House-Brackmann Scale<sup>10</sup> (Table 1) and the Terzis-Noah Scale<sup>11</sup>.

**Table 1.** House-Brackmann Scale

Grade	Characteristics
I. Normal	Normal facial function in all areas
II. Mild dysfunction	<ul style="list-style-type: none"> <li>♦ Slight weakness noticeable on close inspection;</li> <li>♦ May have very slight synkinesis;</li> <li>♦ At rest: normal symmetry and tone.</li> <li>♦ Motion: Forehead - moderate to good function; Eye - complete closure with minimum effort; Mouth - slight asymmetry.</li> </ul>
III. Moderate dysfunction	<ul style="list-style-type: none"> <li>♦ Obvious weakness and asymmetry;</li> <li>♦ At rest: normal symmetry and tone.</li> <li>♦ Motion: Forehead - none; Eye - incomplete closure;</li> <li>♦ Mouth - asymmetric with maximum effort.</li> </ul>
IV. Severe dysfunction	<ul style="list-style-type: none"> <li>♦ Only barely perceptible motion.</li> <li>♦ At rest: asymmetry.</li> <li>♦ Motion: Forehead - none; Eye - incomplete closure; Mouth - slight movement.</li> </ul>
V. Totalpalsy	♦ No movement.

The case presented belongs to the third grade according to the House-Brackmann Scale, although there is no strict mathematical limit in ranking patients from one grade to another.

The management and treatment approach of neonatal facial palsy is careful. The artificial tears are the first protection against dry eye. The use of corticosteroids in neonatal facial palsy is controversial. The use of corticosteroids in adult facial palsy population gives satisfactory results. However, there are still not enough randomized and controlled trials for this condition, which would justify the use of corticosteroids in the neonatal period<sup>12, 13</sup>.

The surgical treatment of traumatic facial palsy depends on the clinical and electro-physiological tests. Total facial palsy (Grade V. on the House-Brackmann scale), temporal bone injury proven by computed tomography of the brain, and lack of improvement after 5 weeks of birth are possible indications for surgical treatment. The surgical treatment carries a risk of iatrogenic injuries.

Physiotherapy is a possible supportive therapy in newborns with facial palsy, through stimulating exercises, opening/closing of the eyelid and provoking a smile.

There is a complete withdrawal of facial palsy in most newborns. This process may take 3-6 months and does not require surgery<sup>14, 15</sup>.

Negative prognostic factors for traumatic facial palsy are unilateral total palsy present at birth, temporal bone dislocation fracture, absence of spontaneous and evoked

motor unit responses in all muscles innervated by the facial nerve for 3 to 5 days of life, and impaired function of the facial nerve by the fifth week after delivery<sup>16</sup>. The available literature on the prognosis of isolated congenital unilateral facial palsy is scarce and inconsistent, probably due to the more frequent encounter of patients with traumatic etiology.

Developmental monitoring and regular check-ups by a neonatologist and pediatric neurologist are important in newborns with congenital unilateral facial palsy to detect additional deficits such as abnormal ocular mobility, hypoglossal or trigeminal nerve involvement, which are not visible in the neonatal period<sup>17</sup>.

## Conclusion

Recovery time and degree significantly differ in patients with facial nerve paresis compared to patients with total palsy. Since the term "palsy" includes both entities, the term "palsy" should be used only to describe the total loss of nerve function. Patients, including newborns with incomplete acute Bell's palsy (paresis), should show early improvement in facial function (1-2 weeks after onset), which was the case with this newborn. Full recovery is expected within 3 months. Treatment with antiviral drugs and steroids is debatable, whereas examining patients with varying grades of facial palsy could lead to controversial results.

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