

Neonatal Cerebellar Hemorrhage and Facial Nerve Palsy: An Unusual Association

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Abstract

Cerebellar hemorrhage is rare in term newborns and is most often seen after traumatic birth. Lifelong sequelae include motor and cognitive impairment. We report the uncommon case of a late preterm infant born by spontaneous delivery who showed right peripheral facial palsy at 24 hours of life. Cranial ultrasound showed lateral ventricles dilatation and a diffuse hyperechoic round lesion in the right cerebellar hemisphere. The computed tomography scan confirmed a hemorrhagic lesion in the right cerebellar hemisphere and in the vermis with midline shift and intraventricular bleeding. Ommaya reservoir was inserted and used for a few days. The facial palsy gradually recovered to a complete remission after 6 weeks. Follow-up examinations at 12 and 18 months evidenced infant's delayed motor function, hyperreflexia, tremors, and speech delay.

Keywords

- ▶ cerebellar hemorrhage
- ▶ facial palsy
- ▶ infant

Cerebellar hemorrhage (CH) is a frequent complication in preterm infant, and its recognition has increased in high-risk newborn requiring intensive care due to improved neuroimaging techniques.^{1,2} Conversely, CH is a rare pathology in late and term infants and its incidence at birth or shortly thereafter, has been reported to be approximately 2/1,000 live births.³ However, the exact incidence is unknown, and probably underestimated because only a fraction of newborns show neurological symptoms in the first postnatal days.^{4,5} However, Limperopoulos et al reported a case series of 17 term infants with cerebellar hemorrhagic injury, examined by magnetic resonance imaging (MRI), over a 5-year period⁶; more recently, in a retrospective study, through MRI, Hong and Lee identified CH in 2 out of 42 term infants with symptomatic intracranial hemorrhage over an 11-year period.⁷

The pathogenesis of CH is poorly understood, but its etiology has been ascribed to the distortion and rupture of

veins in the subdural space, laceration of the falx cerebri or tentorium, direct cerebellar contusion, or, less commonly, to bleeding disorders or congenital vascular malformations.^{1,2} Moreover, prolonged labor, precipitous delivery, instrumental delivery, primiparity, and extreme multiparity are recognized risk factors.^{8–10}

Symptoms typically occur within the first 24 to 36 hours of life, and are generally represented by recurrent crisis of apnea and/or bradycardia, seizures, and signs of obstruction to cerebrospinal fluid flow with cerebral ventricle dilation.¹¹

CH may severely impair infants' neurodevelopment affecting particularly motor functions and expressive language, and inducing behavioral disturbances.⁶ On the other hand, early diagnosis of CH is critical to allow for prompt medical treatment, such as in the case of coagulation disorders, or a neurosurgical approach, in case of intracranial hypertension, as well as to plan a proper neurodevelopmental follow-up.⁶

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To underscore these considerations, we report the unusual case of a late preterm infant with a peripheral facial palsy as presenting sign of CH.

Case Report

A Caucasian male was born at 36^{0/7} weeks of gestation by vaginal delivery after a regular pregnancy with an Apgar score of 9 and 10 at 1 and 5 minutes, respectively. His birth weight was 2,575 g (30th percentile). The newborn had a regular postnatal adaptation and normal physical examination. At 24 hours of life, he showed inconsolable crying episodes but physical and neurologic examinations, blood gas analysis, complete blood count, glycemia, sepsis workup, toxicologic data, chest, and abdominal X-ray were all normal. However, 4 hours later, he developed 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. The clinical picture was completed by the subsequent development of a progressive axial hypotonia and abnormal Moro's reflex. We performed a cerebral ultrasound through the anterior and mastoid fontanelles, which revealed the dilatation of both lateral ventricles and a diffuse hyperechoic round lesion in the right cerebellar hemisphere (► Fig. 1). Coagulation tests were normal. A computed tomography (CT) scan confirmed a hemorrhage in the right cerebellar hemisphere with midline shift and intraventricular bleeding (► Fig. 2). An Ommaya's reservoir was inserted and 15 mL per day of cerebrospinal fluid were withdrawn for 2 weeks. Postintervention MRI performed at 10 days of life showed a slightly reduction of lateral ventricle size (► Fig. 3). Our patient was discharged at 25 days of life and the facial palsy gradually recovered with complete remission at 6 weeks of life. MRI scans at 6 months showed loss of substance in the cerebellar right hemisphere and vermis (► Fig. 4). At 9 months, the Ommaya reservoir was removed, and the baby underwent an endoscopic third ventriculocisternostomy for progressive ventriculomegaly.

Nearly 12 and 18 months of follow-up examinations (with the Bayley-III scales and Gross Motor Function Classification

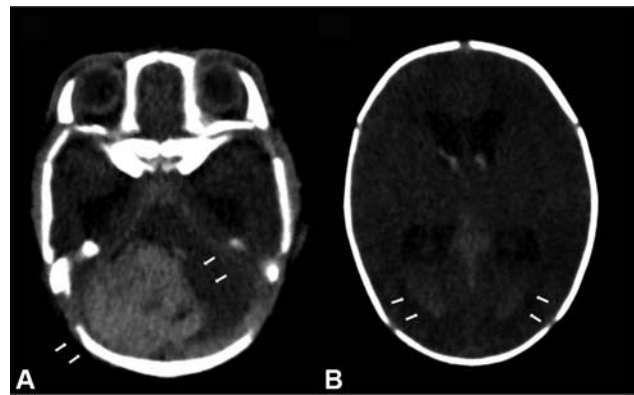


Fig. 2 Axial views of CT scan showing hyperdense round lesion on the right cerebellar hemisphere (A), ventricular dilatation, and intraventricular bleeding (B) (arrows). CT, computed tomography.

System) demonstrated infant's motor function and speech delay associated with hyperreflexia and tremors.

Discussion

We report the unusual case of CH with inconsolable cry and right facial nerve palsy as presenting sign. Unilateral facial palsy as the first symptom of CH is an extremely rare finding,¹² and can therefore lead to a lack of or delayed recognition.

The early onset of symptoms may suggest that in our case cerebellar injury occurred during the delivery inducing progressive bleeding. The imaging and the complete remission of palsy in few weeks' time support the hypothesis that it was caused by a transient compression of right facial nerve at the cerebellopontine angle due to the hemorrhage. In addition, our patient exhibited another relevant, but non-specific symptom: an inconsolable cry probably due to the development of posthemorrhagic ventricular dilatation, as previously described.^{5,12,13} Interestingly, the most common symptoms of CH, such as seizures, apnea, bulging fontanelle, and abnormal level of consciousness,^{5,7,13} did not occur in this case.

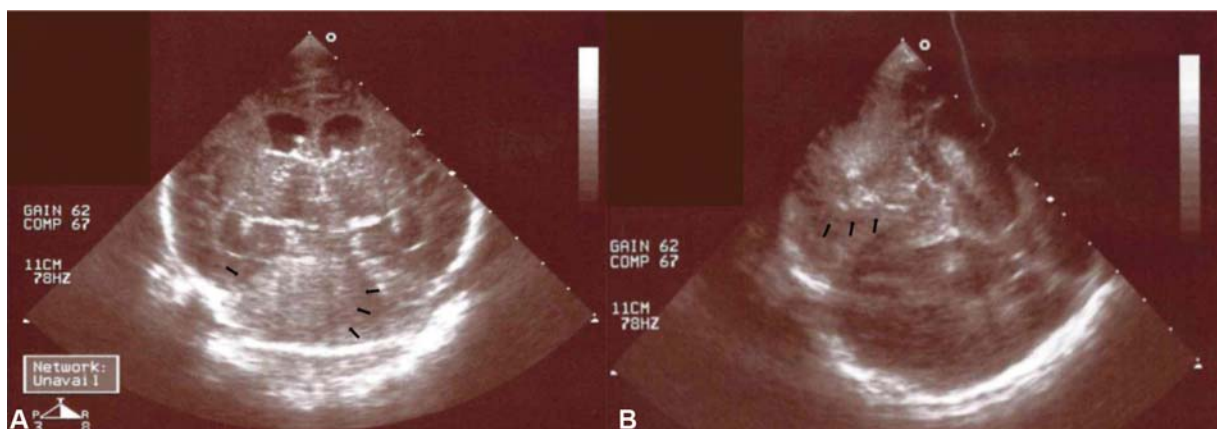


Fig. 1 Cranial ultrasound (Sonos 7500, Philips, Amsterdam, The Netherlands) through the anterior (A) or mastoid fontanelle (B) showing dilatation of both lateral ventricles and a diffuse hyperechoic round lesion on the right cerebellar hemisphere (arrow).

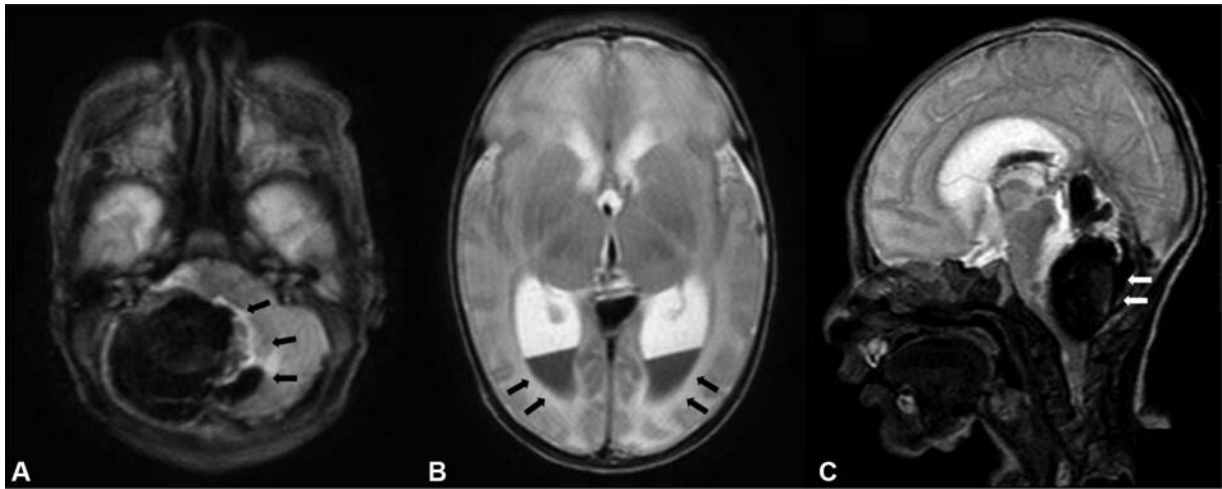


Fig. 3 Postintervention MRI at 10 days of life. T2-weighted axial (A, B) and midsagittal view (C), showing huge round hypointense bleeding causing shift of the midline with involvement of the vermis and ventricular bleeding (arrows). MRI, magnetic resonance imaging.

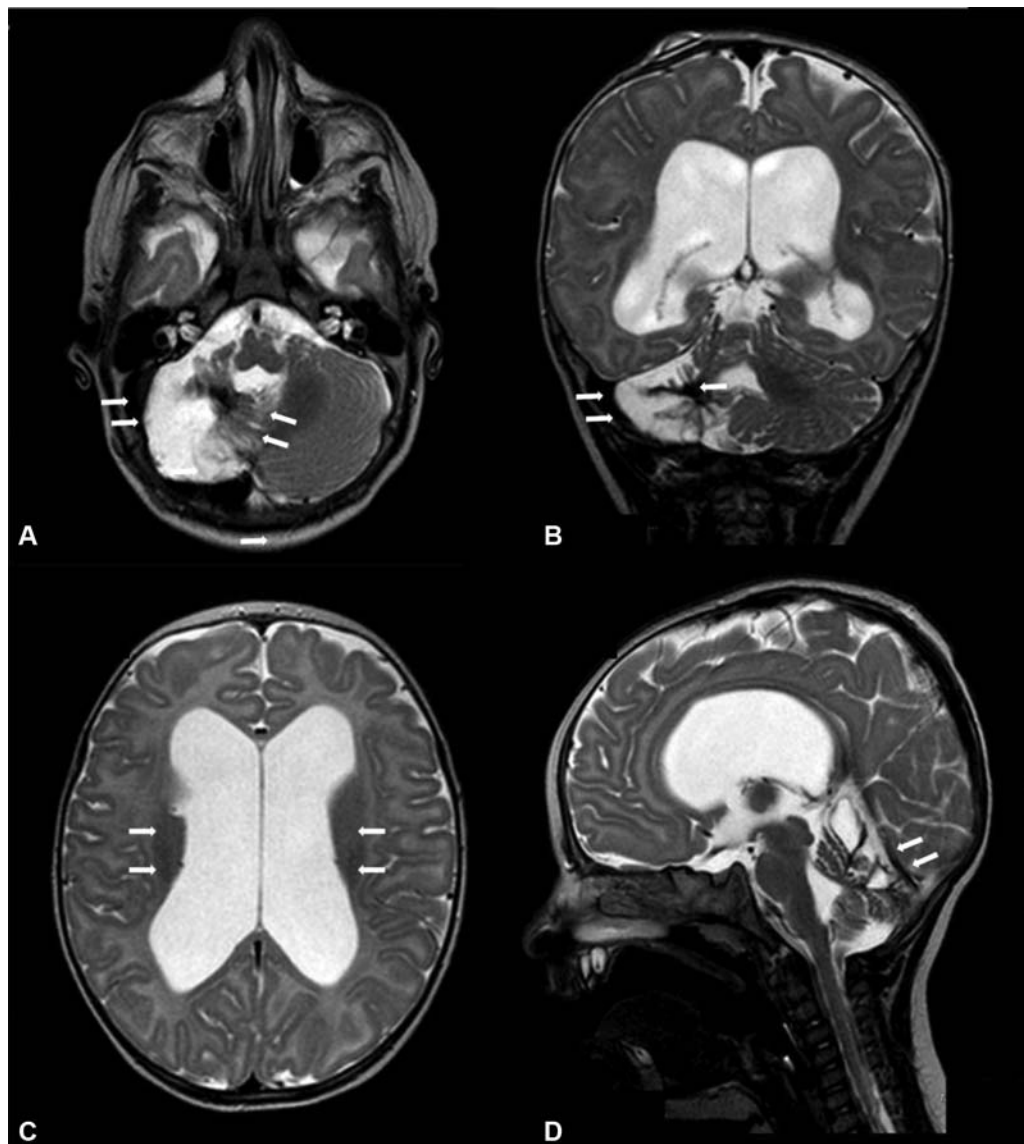


Fig. 4 MRI T2-weighted axial (A–C) and midsagittal view (D) scans at 6 months showing an isolated substance defect of the right cerebellar hemisphere and of the vermis, and ventricular dilatation (arrows). MRI, magnetic resonance imaging.

Recently, Mühlbacher and coworkers described a late preterm newborn who showed peripheral facial palsy at birth due to cerebellum infarction. Unlike our case, the baby developed focal tonic seizures contralaterally to the stroke with secondary generalization.¹⁴

Our patient did not present two of the most frequently reported risk factors for the development of intracranial hemorrhage, and in particular for CH, that is, bleeding disorders⁵ or thrombocytopenia.¹⁵ Only primiparity could be identified as a classical risk factor. However, the increased risk of CH in nulliparous women is due to a greater occurrence of instrumental delivery (i.e., vacuum extraction)^{9,12}, which did not occur with our patient.

The differential diagnosis of neonatal facial nerve palsy includes several pathologies, such as perinatal trauma, intrauterine malposition, intrapartum compression, and congenital aplasia of cranial nerve nucleus.³ However, perinatal injuries are the most frequent causes of facial nerve palsy and are commonly associated with birth trauma due to the relatively superficial course of the extracranial facial nerve, meaning it is susceptible to damage during labor and delivery. Congenital facial nerve palsies include Moebius, cardiofacial, Poland's, and Goldenhar's syndromes.¹⁶ As expected, perinatal posttraumatic facial nerve palsy is a self-resolving condition, while congenital facial nerve palsy is not reversible.^{3,16}

Our case was complicated by the development of posthemorrhagic ventricular dilation, which was initially treated by serially withdrawing cerebrospinal fluid through an Ommaya reservoir, and later with a third ventriculo-cisternostomy. This complication is frequent and neurosurgical intervention has been reported in almost 25% of cases of CH.^{2,7}

Follow-up examinations revealed a delay in infant's motor function and speech. CH is associated in childhood with a wide spectrum of neurodevelopmental disabilities, such as motor, cognitive, language, and behavioral deficit—particularly in infants with large lesions (>1 cm).⁷ Thus, early diagnosis is needed to allow for adequate neurodevelopmental follow-up and intervention to limit possible deficits.⁶

In conclusion, our case suggests that the postnatal finding of an unusual symptom, such as facial nerve palsy, may be rarely associated with the diagnosis of CH. The rarity of this association might make its recognition difficult but, in any case, it should be suspected and quickly diagnosed by cerebral ultrasound to allow for proper in-hospital management and postdischarge neurodevelopmental follow-up.

Funding

None.

Conflict of Interest

None declared.

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