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Mini Review

Risk Factors, Clinical Features and Diagnosis of Cerebellar Hemorrhage in Neonates

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Abstract

Cerebellar hemorrhage is the most common acquired lesion in the posterior cranial fossa, especially in neonates born before 32th weeks of gestation. The incidence of cerebellar hemorrhage is inversely proportional to gestational age and birth weight. The main risk factors to cerebellar hemorrhage are perinatal asphyxia, resuscitation at birth, traumatic delivery, assisted vaginal delivery, emergency cesarean section, use of mechanical ventilation, systemic hypotension, patent ductus arteriosus, etc. The association of cerebellar hemorrhage and high grade of intraventricular hemorrhage in preterm neonates is common. It is recommended that cranial ultrasound through the mastoid fontanelle be part of the routine examination of neonates born prematurely during hospitalization. Also, cranial ultrasound through the mastoid fontanelle is required in all full-term neonates with neonatal infections, perinatal asphyxia and hypoxic-ischemic encephalopathy, as well as unexplained neurological manifestations.

Keywords: Brain Injury, Cerebellar Hemorrhage, Newborn, Prematurity, Cranial Ultrasound

Introduction

Cerebellar hemorrhage (CH) is the most common cause of cerebellar injury in the neonatal period, especially in neonates born before 32th weeks of gestation and less than 1500g birth weight (BW). The overall incidence of CH is estimated at about 3%, and increases to about 9% in neonates with a BW of less than 750g [1,2]. Cerebellar hemorrhage is a rare condition in late preterm and full-term neonates, but affects critically ill neonates, which require treatment in the neonatal intensive care unit [3]. Although it occurs rarely, and mostly in neonates born prematurely, CH represents a significant perinatal event, given that it can lead to death, and in surviving neonates to long-term neurodevelopmental disabilities, including cognitive and behavioral impairments, learning and language difficulties [2,4,5].

The increase in the incidence of neonatal CH during the last decade is noted thanks to the progress of neuroimaging diagnostic techniques, such as cranial ultrasound (CUS) through the mastoid fontanel and brain magnetic resonance imaging (MRI) [2,6,7].

Risk factors

The vulnerability of the cerebellum and sensitivity to numerous perinatal risk factors is particularly pronounced between the 23rd and 27th weeks of gestation, so CH is most often seen in extremely preterm neonates. The pathogenesis of CH is multifactorial and differs in preterm and full-term neonates. Immaturity of the cerebellar structure as well as the autoregulatory mechanisms of cerebral blood flow increase the risk of CH in neonates born prematurely. Factors that cause hemodynamic disturbances play an important role in the development of preterm CH. The association of CH and high grade (III and IV) of intraventricular hemorrhage (IVH) in preterm neonates is common. The spread of IVH or subarachnoid hemorrhage (SAH) into the cerebellum is the most common form of CH in preterm neonates [2,5,6,8-10]. Risk factors for CH in preterm neonates are shown in (Table 1).

Table 1: Risk factors for preterm cerebellar hemorrhage.

low gestational age
low birth weight
perinatal asphyxia
respiratory failure requiring endotracheal intubation and mechanical ventilation
systemic hypotension requiring the use of volume expanders and inotropes
patent ductus arteriosus
pulmonary hemorrhage

In contrast, birth trauma due to complicated delivery is the most common cause of CH in neonates born at term. Occipital osteodiastasis can also be a cause, leading to direct traumatic laceration of the cerebellar pontine veins or occipital sinuses. Other risk factors for CH in full-term neonates include assisted vaginal delivery, emergency cesarean section, resuscitation at birth, perinatal asphyxia and hypoxic-ischemic encephalopathy [2,3,6]. Also, neonatal sepsis is also identified as a risk factor for cerebellar injury in full-term neonates [11]. The one cross-sectional study identified 35% of mothers positive for group B β-hemolytic Streptococcus (GBS) at the time of delivery, whose neonates had cerebellar injury. However, there was no neonates with CH with GBS positive blood culture [3]. Other, rare causes of CH in full-term neonates have been described in the literature, such as fetal and neonatal alloimmune thrombocytopenia, vitamin K deficiency, and organic acidopathies [2,12-14].

Clinical Presentation

The clinical presentation of CH is directly related to the gestational age and the size of the hemorrhage. In large hemorrhagic lesions in the cerebellum that compress the brainstem, with frequent association with supratentorial hemorrhage, there is a severe degree of disturbance of consciousness, respiratory failure accompanied by apneic crises and irregular breathing, bradycardia, decerebrate position and neonatal seizures. There may be clinical signs of intracranial hypertension, a bulging anterior fontanelle, separated cranial sutures, and an increase in head circumference. In these neonates, death most often occurs in the first 36 hours after the onset of CH. However, clinical manifestations may be completely absent or nonspecific in the neonates with small hemorrhagic lesions in the cerebellum. These lesions diagnosed exclusively by neuroimaging [2,11,15].

Diagnosis

Neuroimaging diagnostic techniques are used to diagnose neonatal CH. Due to its numerous advantages over other neuroimaging techniques, including bedside examination, minimal disturbance to the neonates, and the absence of ionizing radiation, CUS through the mastoid (posterolateral) fontanelle is the first choice for examining the posterior cranial fossa. All neonates with signs of brainstem compression, and/or increased intracranial pressure should undergo an CUS through the mastoid fontanelle [2,15,16]. Small, punctiform lesions (< 4 mm), as the most common type of CH in preterm neonates, are often not detected by CUS. In order to more precisely define the extent of CH, as well as the distribution of lesions, a more sensitive type of neuroimaging is brain MRI. Classification of CH based on CUS or brain MRI was addapted from Steggerda et al. [6] and Parodi et al. [17], and presented in (Table 2).

Table 2: Classification of grading of the severity of cerebellar hemorrhage in neonates.

Grade 0	normal echogenicity of the cerebellar vermis and hemispheres withnormal anatomic features and with no signs of destruction or atrophy
Grade I	small (≤4 mm) (focal) punctate lesion(s) in the cerebellar parenchyma
Grade II	limited CH, lesion (>4 mm) but involving at the most 1/3 of the cerebellar hemisphere
Grade III	extensive CH, involving more than 1/3 of the cerebellar hemisphere

Conclusion

Given the long-term neurodevelopmental disabilities, the importance of early diagnosis, is emphasized. It is recommended that CUS through the mastoid fontanelle be part of the routine examination of all preterm neonates and critically-ill full-term neonates in neonatal intensive care unit. All neonates who have had CH require additional neuroimaging (brain MRI) and follow-up by pediatric neurologist during childhood.

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Conflict of Interest

The author declare no competing interests.

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