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Neonatal Stroke: A Review on Epidemiology, Pathogenesis, Diagnostics and Therapy

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Abstract

Neonatal stroke, including perinatal arterial ischaemic stroke and cerebral sinovenous thrombosis, remains a serious problem in the neonate. This article reviews the current evidence on epidemiology, pathogenesis, diagnostics and therapeutic options.

Introduction

Neonatal stroke can be defined as a cerebrovascular injury that occurs around birth. It can be focal or multifocal and may include both ischaemic and haemorrhagic injury. Neonatal stroke is most frequently referred to as perinatal cerebral injury of ischaemic origin [1]. In an international workshop on this subject, held in 2006, ischaemic perinatal stroke was narrowed down to 'a group of heterogeneous conditions in which there is focal disruption of cerebral blood flow secondary to arterial or cerebral venous thrombosis or embolisation, between 20 weeks of foetal life through to the 28th postnatal day, confirmed by neuroimaging or neuropathological studies. Two common subtypes are perinatal arterial ischaemic stroke (PAIS) and cerebral sinovenous thrombosis (CSVT). However, there is still an ongoing discussion about which other patterns of injury should be regarded as perinatal stroke. Periventricular haemorrhagic infarction (PVHI), which is commonly seen in preterm infants, is frequently regarded as being part of the stroke spectrum. Another one is haemorrhagic stroke. Finally, presumed perinatal stroke, encompassing both arterial ischaemic stroke and PVHI, is also considered part of the neonatal stroke spectrum, though by definition they are diagnosed beyond the 28th postnatal day. In this article, we review both PAIS and CSVT in terms of their aetiology, clinical presentation and the diagnostic and therapeutic options.

Perinatal Arterial Ischaemic Stroke

Perinatal arterial ischaemic stroke has an incidence of between 1/1600 and 1/5000. The wide range ofreported incidences may be explained by differences in the definition and diagnosis of PAIS. Some only include PAIS, while others also include presumed perinatal stroke and haemorrhagic stroke [2]. All studies are of retrospective design and therefore depend on the accuracy of the databases used. In view of the fact that magnetic resonance imaging (MRI) scans are not routinely obtained for all infants presenting to neonatal intensive care units with neonatal seizures, the true incidence is likely to be even higher. Perinatal arterial ischaemic stroke may affect both term and preterm born infants. It has been suggested that the incidence of PAIS is higher in preterm born infants, but this might also reflect the routine use of cranial ultrasound in preterm infants . Several studies have reported a male predominance of approximately 60%, and PAIS is known to involve the left hemisphere more often .

Pathogenesis and risk factors

Although several risk factors have been associated with PAIS, little is known about the exact pathophysiological mechanisms responsible for most cases [3]. It is generally thought that the patent foramen ovale allows passage of thrombi derived from the placenta or venous circulation, resulting in occlusion of an artery. Most potential risk factors have been identified in small retrospective case series and do not necessarily reflect a causal relation. Maternal risk factors include infertility, primiparity, maternal fever, meconium-stained amniotic fluid, chorioamnionitis, pre-eclampsia and intrauterine growth retardation. Complicated deliveries, both instrumental and emergency caesarean section, low Apgar scores and hypoglycaemia are more frequently observed in infants with PAIS . Studies have reported prothrombotic factors in more than half of the population studied. In a large case–control study, prothrombotic factors were more often observed in the cases (68%) being studied than in age and sex-matched controls (24%). The presence of prothrombotic factors has also been associated with poor neurological outcome, including development of unilateral spastic cerebral palsy (USCP). It is of interest to note that, despite the presence of these prothrombotic factors, recurrence of arterial ischaemic stroke is rare, suggesting a multifactorial cause of PAIS. Congenital heart disease is an important risk factor for PAIS. Due to the disturbed anatomy, cardiac thrombi are easily formed, which may result in a spectrum of cerebral injury, including PAIS [4]. Other factors associated with the development of PAIS are infectious disorders such as meningitis and sepsis, dehydration, arterial dissection, catheterisation and extracorporeal membrane oxygenation, which all increase the risk of thrombus formation. The role of the placenta remains uncertain, as data on placental abnormalities in infants suffering PAIS are scarce, probably because the placenta is often discarded before the onset of clinical symptoms.

Clinical presentation

The first manifestation of PAIS occurs most frequently during the first week. Seizures are the most common first clinical sign of PAIS, occurring in 70–90% of all infants with PAIS (13). When compared to seizures observed following hypoxic ischaemic encephalopathy (HIE), seizures following PAIS tend to arise later and are more often lateralised . Seizures may be subtle and may remain unnoticed, causing

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a delay in diagnosis. Additional signs may include apnoea, sometimes as clinical sign of a seizure, lethargy, feeding difficulties and hypotonia [5]. Some infants may show asymmetry of tone during the first days to weeks, with hypotonia rather than hypertonia on the affected side [6]. Infants may, however, remain asymptomatic. Imaging studies performed in children presenting with USCP and born at term suggest that approximately half of the infants with PAIS are diagnosed during the neonatalperiod . Infants who are not diagnosed during the neonatal period, but develop neurological deficits attributable to focal infarction later in infancy, will be diagnosed as presumed perinatal stroke, based on later neuroimaging findings [7]. Clinical presentation of these children will depend on the age at which they are recognised, and the extent of injury, and include early handedness, decreased hand use and rigidity of the upper limb or fisting. Other presentations include seizures, developmental delay and cognitive deficits [8].

Therapy

Cerebral sinovenous thrombosis therapy should primarily be aimed at treating any underlying cause that may have led to the development of the thrombus [9]. This includes treatment of dehydration, sepsis, meningitis and significant congenital heart disease. Studies in infants without CSVT show that in the supine position, compression of the occipital bone occurs, which may result in a reduced cerebral venous flow. Adjusting the infant's pillow decompresses the occipital bone and has been shown to increase flow in the sigmoid and superior sagittal sinus and may offer a noninvasive therapy for infants with CSVT [10]. However, this has not yet been studied in this population, and its efficacy still needs to be proven. There is ongoing discussion about whether anticoagulants should be used to prevent propagation of the initial thrombus and to enhance recanalisation. In the absence of randomised control trials [11], evidence for the use of anticoagulants is limited to case series. Withholding treatment does, however, seem to be associated with an increased risk of thrombus propagation of about 25%. Even in the presence of a thalamic haemorrhage, anticoagulation therapy appears to be safe [12]. That is why a recent guideline issued by the American College of Chest Physicians recommends administering unfractionated heparin or low molecular weight heparin to infants without an intracranial haemorrhage, followed by low molecular weight heparin for 6–12 weeks. In the presence of a significant haemorrhage, anticoagulation therapy may be initiated, but a conservative approach with supportive care and radiological monitoring of the thrombosis at 5–7 days may also be chosen [13]. If propagation of the thrombus is noted at that time, anticoagulation therapy may still be initiated.

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