CASE REPORT

Perinatal infratentorial haemorrhage: a rare but possibly life-threatening condition

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SUMMARY

Background Perinatal infratentorial haemorrhage (PIH) is a rare birth complication associated with abnormal labour.

Case presentation A baby boy was born by vacuum extraction at 41 weeks' gestational age. The pregnancy was uneventful and Apgar scores were 3/6/9. Following initial resuscitation, insufficient and irregular breathing, non-reactive pupils and absence of spontaneous movements were noted. A diagnosis of perinatal asphyxia with hypoxic–ischaemic encephalopathy (HIE) was considered. Therapeutic hypothermia (TH) for 72 hours was initiated. Cerebral ultrasound showed only a mildly hyperechogenic periventricular substance. A brain MRI on the fourth day of life revealed a subdural haemorrhage in the posterior fossa with compression of the fourth ventricle.

Conclusion PIH is an important differential diagnosis to HIE that can be missed with ultrasound. PIH is a treatable condition but may be aggravated by TH. Therefore, in neonates at risk for PIH, a more detailed ultrasound protocol or brain MRI should be considered early.

BACKGROUND

Asymptomatic perinatal intracranial haemorrhage (PIH) is reported in up to 25% of full-term neonates after vaginal delivery. Symptomatic intracranial haemorrhage in full-term neonates is less common and its incidence ranges from 2.7 to 4.9 per 10 000 live births.¹ Complicated labour requiring instrumentation or caesarean section is considered a risk factor for PIH, which is demonstrated by a higher incidence in this patient population (8 to 21 per 10 000 live births).^{2–4} Symptomatic PIH is a rare and potentially life-threatening condition. It is likely to occur during labour when vertical moulding of the skull leads to distortion and disruption of blood vessels. Compression of vital structures (ie, brainstem) and evolution of hydrocephalus are life-threatening complications of PIH.

A diagnosis of PIH is challenging as the signs are often non-specific and the neurological examination showing an encephalopathic neonate can be mistaken for an underlying hypoxic–ischaemic encephalopathy (HIE). Furthermore, cerebral ultrasound—a fast and frequently used bedside tool in neonatal intensive care units—might miss the ongoing process in the posterior fossa. A high index of suspicion in combination with appropriate neuroimaging techniques is needed in order to make a timely diagnosis of PIH.⁵

The aim of this case report is to increase the awareness of PIH in neonates with a history of complicated labour and unusual presentation.

CASE PRESENTATION

A 33-year-old previously healthy woman was admitted to a primary care hospital for delivery at 41 weeks' gestational age. No previous stillbirths or haematological problems were noted in the medical and extended family history. The pregnancy was uneventful and the woman attended regular follow-up visits with her obstetrician during her pregnancy, which were in keeping with the national recommendations. Clear amniotic fluid was noted 1 hour prior to delivery. Fetal bradycardias were detected on cardiotocogram during labour. The baby boy was delivered by kiwi vacuum extraction and required immediate resuscitation with positive pressure ventilation. Shortly after delivery, discharge of meconium-stained amniotic fluid was observed.

Apgar tests were performed 1, 5 and 10 min after birth and revealed scores of 3, 6 and 9. Blood gas analysis showed a pH at 7.26, Pco₂ of 34 mm Hg, bicarbonates of 16.4 mmol/L and base excess of −11.4 mmol/L. Insufficient spontaneous breathing was noted after initial resuscitation with blood oxygenation levels around 80%, requiring on and off mask ventilation. The neonate was intubated 1 hour after birth and transferred to the intensive care unit of the University Children's Hospital in Berne.

Due to the absence of both spontaneous breathing and movements and non-reactive wide pupils, the diagnosis of neonatal asphyxia with HIE was postulated. Scoring for asphyxia was performed, revealing a Thompson score of 18/22 and Sarnat III. Therapeutic hypothermia was induced according to the guidelines.⁶ A temperature of 33.5°C (±0.5°C) was reached within 1 hour after hypothermia start and within the 6-hour window after birth. Cooling was achieved by passive cooling and application of external cooling devices. Cerebral function monitoring (CFM) showed seizure activity, and as such, an anticonvulsant treatment, Phenobarbital (5 mg/kg/day), was initiated. Other investigations that were performed included a cerebral ultrasound on admission (through the anterior fontanel) showing mildly hyperechogenic periventricular substance; testing serum levels of creatinine (67 µmol/L), aspartate transaminase (62 U/L) and alanine transaminase

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Figure 1 As demonstrated in the first and second row, large acute to early subacute subdural haemorrhage can be found in the posterior fossa (see arrows) with hypointense signal in T2-weighted images (A–C) and hyperintense signal in T1-weighted images (D–F). The haemorrhage had a compressive effect on the aqueduct.

(67 U/L) indicating no renal or hepatic abnormalities; and an echocardiogram with normal results.

The therapeutic hypothermia lasted for 72 hours. Subsequently, low normothermia ($36.0 \pm 0.5^\circ\text{C}$) was achieved by a stepwise increase of 0.5°C every 2 hours and strictly monitored for 48 hours. According to national guidelines, electroencephalography (EEG) and MRI were performed at physiological body temperature.⁶ The EEG showed normal baseline activity, no focal findings and no seizure activity. In contrast, unexpectedly, cerebral MRI revealed an acute to early subacute subdural infratentorial haemorrhage with compression of the fourth ventricle (figure 1). There were no areas of restricted diffusion. X-ray images of the cervical bone structures were normal with no signs for occipital osteodysplasia. Haemostatic analysis showed normal platelet count ($>150 \text{ G/L}$) and elevated international normalized ratio (maximum 1.82; normal range, 0.85–1.27).

The patient was alert and spontaneously breathing by the fourth day of life; consequently, extubation was possible. Apnoeas and intermittent bradycardias were observed during the first 2 days after extubation. Daily cerebral ultrasound monitored for development of hydrocephalus. Lethargy and progressive size of the lateral ventricles on ultrasound led to a follow-up MRI at the seventh and ninth days of life (figure 2). Neurosurgical intervention was not necessary due to spontaneous remission of intermittent apnoeas and bradycardias.

Figure 2 As a complication of perinatal infratentorial haemorrhage, follow-up MRI demonstrates a progressive enlargement of the first to third ventricles (see arrows) due to the compressive effect on the aqueduct; G = 7th day of life, H = 9th day of life (FLAIR, fluid attenuated inversion recovery, images).

The patient was transferred to the regular ward on the 13th day of life and discharged from hospital at the age of 20 days.

OUTCOME AND FOLLOW-UP

At 3 months of age, the cerebral ultrasound and EEG were normal and no further signs of hydrocephalus were noted. Phenobarbital was successfully tapered off over 3 weeks without any further signs of clinical seizure activity. On clinical examination at the age of 3, 6 and 12 months, no focal neurological deficits were identified. In addition, normal psychomotor development was noted (Griffiths Mental Developmental Scale global quotient 0.96 at 12 months).

DISCUSSION

Here, we have presented an unusual case of PIH with coexisting perinatal asphyxia in a full-term neonate. It is known that full-term newborns delivered during complicated labour and with instrumentation are at risk for occult head injuries.⁵ The use of vacuum extraction applies additional traction on vessels, which may lead to rupture and bleeding.⁷ In this case, the Apgar scores and early laboratory investigations were not indicative of a diagnosis of HIE. However, the need for continuous resuscitation during the first hour of life in the context of a severely neurologically altered neonate with findings of hyperechogenic white matter on the ultrasound and seizure activity on the CFM leads to the idea that HIE may have evolved secondary to the PIH. Therefore, we postulate that PIH was responsible for the main neurological/encephalopathic symptoms and the development of HIE.

The clinical signs and symptoms of PIH are non-specific. They can mimic or coexist with other neonatal conditions such as HIE, which often results from similar risk factors. This similar presentation can easily cause a therapeutic dilemma. In the case of perinatal asphyxia, timely initiation of therapeutic hypothermia is crucial to improve neurological outcome.⁸ However, therapeutic hypothermia can hide symptoms of PIH, for example, signs of increased intracranial pressure. Furthermore, therapeutic hypothermia alters the coagulation cascade and may provoke or aggravate haemorrhages.⁹ In our case, one would expect that a haemorrhage of that size would be sonographically detected. The ultrasound protocol applied for patients with HIE does not include mastoid views on a regular basis. In the case, there were no signs of hydrocephalus detected in the early ultrasounds; however, hydrocephalus could be seen during follow-up. Therefore, we suspect that the haemorrhage was evolving under hypothermia.

Literature is sparse concerning this unusual condition. PIH can be life threatening because of brainstem compression or development of hydrocephalus requiring surgical intervention.¹⁰ In contrast, long-term neurological outcome is good in the majority of patients with PIH; therefore, maximal therapy should be provided.^{10 11} In a series of 16 patients, surgery was performed in 13 cases because of signs of brainstem compression or progressive hydrocephalus.¹⁰ Conservative management has been described but needs close monitoring and frequent evaluations of the patient.¹² Early interdisciplinary management is crucial to provide optimal, individually adjusted management of these patients. To improve the guidelines, we suggest to include a more comprehensive ultrasound protocol including mastoid views for the monitoring of patients with HIE, especially in those with a history of instrumented labour. Furthermore, cerebral MRI appears to be crucial to exclude PIH as a potentially treatable condition and should be considered in severely affected

children with presumed HIE, where the clinical situation might implicate treatment limitation.

In conclusion, symptomatic PIH is an unusual complication of instrumented birth and can be easily missed with initial cerebral ultrasound. A more detailed ultrasound protocol, early cerebral MRI, close monitoring and interdisciplinary management are required to detect potentially life-threatening compression of the brainstem and development of hydrocephalus.

Learning points

- ▶ Perinatal infratentorial haemorrhage (PIH) is an important differential diagnosis to hypoxic–ischaemic encephalopathy.
- ▶ PIH can be missed with an ultrasound.
- ▶ PIH may be aggravated by therapeutic hypothermia.
- ▶ Physicians should consider early cerebral MRI in neonates at risk for PIH.

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Competing interests None declared.

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