Review Article

MENINGITIS CAUSING IVH IN A TERM NEONATE WITH SEIZURES MASQUERADING STROKE - A CASE REPORT

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Abstract

Intraventricular hemorrhage (IVH) occurs in preterm infants; however, the occurrence of this event is less frequent in term neonates. In this present case study our objective is to evaluate clinical, radiological features & outcome of a term neonate with IVH. Materials & Methods: This is a case report of full-term neonate admitted to the Neonatal Intensive Care Unit (NICU) of DR DY PATIL Hospital, PUNE March 2022. IVH was diagnosed using cranial ultrasonography & brain magnetic resonance imaging (MRI). Results: The age of onset of symptoms was 4 days. Seizure was the commonest clinical symptom. There is no history of poor feeding, lethargy, fever, hypothermia, vomiting, trauma or coma.The source of bleeding in the brain was parenchyma. Severity of bleeding include grade 4 IVH. Routine lab reports showed no coagulopathy. CSF routine showed bacterial meningitis & blood culture was positive for Streptococcus Parasanguinis. CSF culture had no growth. Treatment: Baby was kept on NBM & empirically treated with iv antibiotics. Conclusion: The main source of IVH in term neonate is choroid plexus; the most common clinical symptom include focal seizure, and IVH event is of grade 4. Novelty of this case is meningitis presenting as grade 4 IVH in a term neonate with hypoxic ischemic hypoglycemic injury with normal perinatal history. The neonate is discharged from NICU without any CNS complication.

Keywords: Intraventricular hemorrhage, Term neonate, Outcome.

INTRODUCTION

Neonatal Meningitis can present with refusal of feeds, temperature instability, respiratory distress, lethargy & convulsions. This case report highlights the rare finding of grade 4 IVH in a term neonate with hypoxic ischemic hypoglycemic injury. Incidence: In developed countries, culture-proven neonatal meningitis is estimated at 0.3 per 1000 live births. In developing countries, the incidence is higher, at 0.8 to 6.1 per 1000 live births, with a mortality rate of up to 58%.

CASE REPORT

A 4 day old full-term male baby born to a mother with a parity index of G5P1L1A3 with uneventful antenatal history was shifted to neonatal intensive care unit (NICU) in view of sudden onset of tonic posturing. The baby was born by normal vaginal delivery with birth weight of 2.3kg cried immediately after birth with an apgar score of 7/10 at 1 min & 9/10 at 5 min with no signs of perinatal depression. The liquor was meconium stained & labour was uneventful. There is no h/o consanguineous marriage. Baby was breast fed normally, passing normal colored urine and stools & presented to us with facial twitching & tonic clonic movements of all extremities lasting for 30secs. Examination showed heart rate 132/min, RR 6/min, SpO2 98% on room air, MBP 89/60(70)mmhg, urine adequately passing, BSL 77mg/dL, on breast feeds. CNS examination showed symmetrical tone in all extremities, cry & reflexes are appropriate for gestational age. There was no organomegaly. The baby was kept on NBM loaded with iv phenobarbitone, empirically stated on iv antibiotics piptaz & amikacin, maintenance intravenous fluid was commenced and euglycemia was maintained. Saturation maintained 95-97%. X-ray chest was normal.

Hemogram was normal with (Hb: 14.2 gm%, total leucocyte count: 11,100/cm3, P: 54, L: 41, E: 02, M: 03, PLT: 1.9 LACS), septic screen done showed C-reactive protein 10.9, blood culture positive for streptococcus parasanguinis.) ABG showed metabolic acidosis & CSF r/m was suggestive of bacterial meningitis. CSF culture sensitivity had no growth. Clinical examination showed wide open bulging anterior fontanel we did cranial ultrasonography which showed grade 4 IVH.MRI brain done suggestive of early subacute intraventricular hemorrhage in right ventricle with obstructive dilatation of left ventricle with restricted diffusion pattern in right peritrigonal periventricular white matter & splenium of corpus callosum likely to be result of hypoxic ischemic Encephalopathy with associated hypoglycemic brain injury. Angio-MR showed the main intracranial arterial trunks. There was no evidence of significant vascular defect.

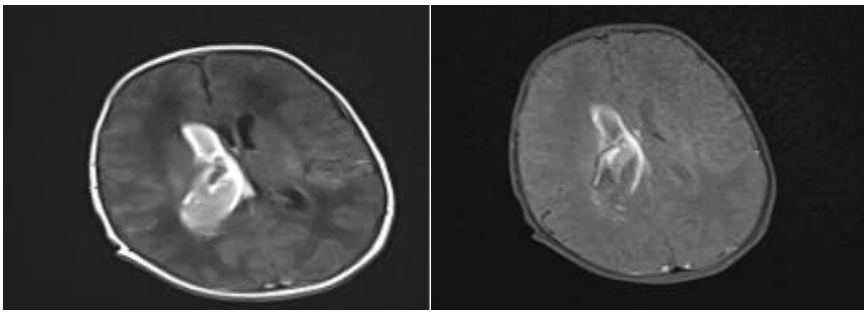

Figure 1 & 2 Showing Right Ventricle hemorrhage

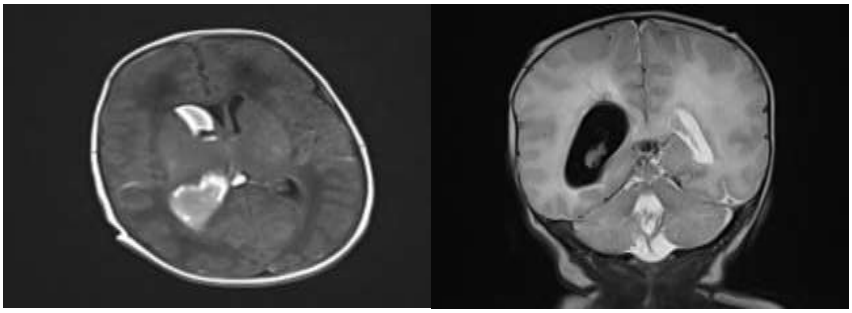

Figure 3 & 4 Showing Obstructive dilatation of left ventricle with restricted diffusion pattern in right peritrigonal periventricular white matter & splenium of corpus callosum

DISCUSSION

Intraventricular hemorrhage (IVH) is mostly documented in premature infants, and more younger the gestational age, more often IVH occurrence[1]. IVH is very rarely reported in full-term neonates and may occur with a variety of clinical conditions like perinatal trauma, asphyxia, and coagulation disorders. Very often, IVH in full-term neonates comes from choroidal plexus and is connected with venous thrombosis and ischemia of the thalamus. However, in some cases, it might be a result of damage to the residual periventricular germinal matrix. The high mortality rate in full-term newborns with IVH is explained by perinatal asphyxia [2].

IVH also contribute as an important source of neonatal morbidity and mortality. The incidence of IVH in term newborns is not known. The incidence of all types of symptomatic intracranial hemorrhages (epidural, subdural, subarachnoid, intraventricular, and intraparenchymal) is 0.27–0.49 per 1000 live births [1].

IVH in full-term infants usually occurs during labor due to mechanical factors; however, in the preterm infants, it occurs mostly as a result of the immature central nervous system and hemodynamic instability. Besides etiology, the location of hemorrhage, clinical presentation, and neurological outcome also differs in the term and preterm infants.

The term newborn with IVH typically presents with signs such as seizures, apnea, irritability or lethargy, and vomiting with dehydration. Flaccidity, loss of pupillary reaction, extraocular movements, coma, irritability, vomiting, shrill cry, central facial weakness, opisthotonic posturing, fever or hypothermia, hypo- or hyperglycemia, decreased lower extremity tone, neck flexor hypotonia, head lag, and brisk reflexes are less frequent in term newborns, however more often observed in preterms.[2,6]Our case presented with facial twitching & sudden onset GCTS but tone was normal in all extremities.

In the full-term infant, IVH usually results only when the infant has a developmental or structural anomaly, or when serious stress occurs. In our case structural lesions such as arteriovenous malformation, tumor, or aneurysm were not seen. No stressful events could be identified. Alterations in cerebral blood flow secondary to hypothermia, BP variation, hypoxia, hypercarbia, acidosis, and/or apnea, which may lead to IVH was not seen with our patient. Another contributing factor may include use of hypertonic solutions with resultant hyperosmolality and hypernatremia is not seen in our case.

In approximately 25% neonates IVH remains asymptomatic and can only be discovered on imaging procedures.cranial ultrasonography is a good choice for early identification of significant intracranial lesions in healthy full-term neonates [2,3].

In Meningitis the meningeal invasion by bacteria causes cellular damage, loss of cellular homeostasis and worsen cerebral edema. Damage to vessels lead to vasculitis and bleed or thrombosis leading to infarction or haemorrahage. In some cases exudate formation can obstruct CSF flow leading to hydrocephalus. Inflammatory mediators along with cytotoxic factors obstruct the blood vessels causing less blood supply & hypoxic insult leading to impaired metabolic adaptation. Bacteria further adjust to ongoing cellualr damage by using anaerobic glycolysis that leads to depletion of brain glucose stores & ATP, with increase in lactate production resulting in metabolic acidosis. In our case the MR angiography was normal. The IVH could be possibly explained by the inflammatory damage caused by the meningitis itself.

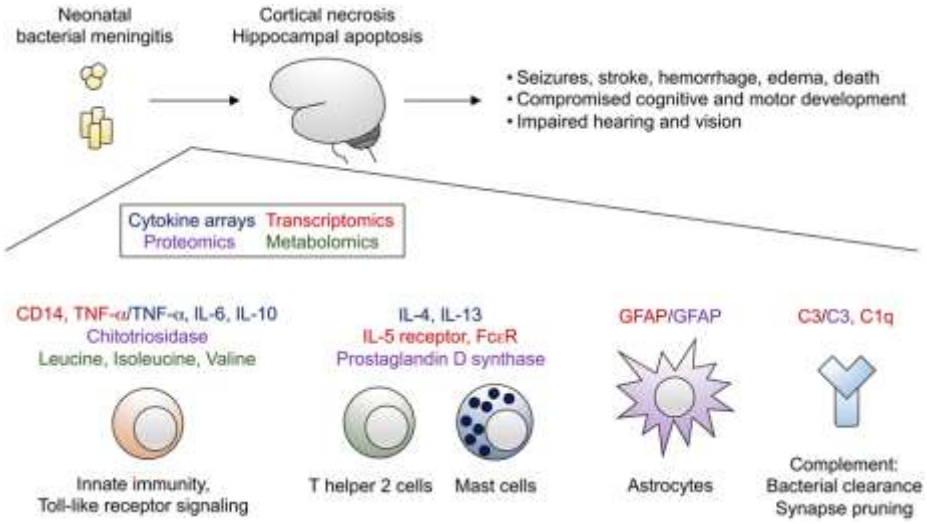

Figure: Showing Bacterial meningitis (Adapted from https://emcrit.org/ibcc/ich/) of the neonate leads to profound brain injury, characterized by cortical necrosis and hippocampal apoptosis. Selected transcripts (highlighted in red), cytokines (highlighted in blue), other proteins (highlighted in purple), and metabolites (highlighted in green) identified from omics analyses have been integrated into a proposed model of immune activation in the setting of bacterial meningitis that can lead to IVH.

Most IVHs of high grade arise from the posterior tufts in the choroid plexus, and less commonly arise from the small residual germinal matrix tissue near the thalamocaudate groove, the thalamus, and the watershed area of the foramen of Monro near the caudate nucleus. [4, 5].

Some arteriovenous malformations (AVM) may cause neonatal intracranial hemorrhage. Reports of AVM of choroid plexus have been limited to descriptions of individual cases [7] IVH may be followed by development of venous sinus, medullary, or cortical vein thrombosis [6]. In one third of cases, a hemorrhage mechanism cannot be determined from neuroimaging studies [5,6].

IVH bleeding in the full-term newborn may occur as a result of birth asphyxia and resuscitation at birth [7,8, 9].It can be a consequence to an interruption of placental blood flow and decreased blood flow to the brain and impairment of cerebral autoregulation. Therapeutic hypothermia can place newborns at greater risk of IVH by causing fluctuations of the cerebral blood flow, depressed cardiac function, hypotension, and changes in coagulation cascade [9]. It was demonstrated that severe stages of HIE may lead to development of severe IVH [10]. Fetal distress was identified as a significant risk factor for IVH and may compromise the vascular environment and may be the first sign of fetal compromise due to IVH in utero [5, 11]. However, since IVH itself may cause respiratory distress, it is difficult to demonstrate that asphyxia is a significant factor in IVH pathogenesis.

The vaginal birth process itself may be traumatic enough to cause IVH in term newborns. Moreover, instrumental deliveries have been shown as a risk factor for IVH in term newborns [12, 13], while other studies did not confirm this association [5].

Bleeding disorders are rare cause of IVH in term newborns. Study conducted by Chadd M, Gray P. Hypothermia and coagulation defects in the newbon. Arch Dis Child. 1972; (47):819-821 says thrombocytopenia (drug-induced, infectious, genetic, immune-related) is a condition that may lead to IVH [22]. Coagulation factors deficiencies (vitamin K-dependent coagulation factors deficiency, factor VIII and IX deficiency) have been implicated with intracranial hemorrhage [14, 15]. Coagulopathy significantly increases the risk for IVH, especially in patients treated with extracorporeal membrane oxygenation, therapeutic hypothermia, and in children with severe congenital heart diseases (secondary to altered hemodynamics or anticoagulant administration) [16]. our case the thrombotic profile was normal.

Neurodevelopmental impairment has been reported in nearly 50 % of patients with IVH. More than half of survivors required physical therapy, and rest seen with neurologic deficits. However, these deficits are mild in nature [17]

The treatment of newborns with IVH should be focused on adequate ventilation, feeding, prevention of metabolic acidosis, and normalization of coagulation disorders. Anticonvulsant therapy should be used to control seizure activity. Transfusion with fresh frozen plasma and platelets may be beneficial in newborns with IVH. Neurosurgical intervention in patients with IVH should be considered in posthemorrhagic hydrocephalus to relieve increasing intracranial pressure or to drain hemorrhagic ventricular cerebrospinal fluid [18].

Table showing Summary of case reports in term neonates with IVH

study	Gestational age	Birth weight	gender	Age at presentation	clinical	risks	Most common Source of bleeding	outcome
Bruno et al.,2014[5]	term			1 day	Seizure Apnoea	Congenital heart disease	Choroid plexus	
Afsharkes et al., 2015[6]	term			3.9 day	Seizure Poor oral intake	Coagulation disorder	Choroid plexus	
Ou Yang et al., 2010[17]	term			2 hours	Seizure Cyanosis		NA	
Dawid S et al., 2016[21]	term	3.6 kg	male	Day 3	Focal seizure	Nil	NA	discharge

					tachypnoea			
Dawid S et al., 2016[21]	term	3.84 kg	male	Day 3	Hypertonia in all limbs	Nil	Choroid plexus & tentorium cerebelli	Post hemorrhagic ydrocephalus
Ellen et al., 2018[19]	term	NA	NA	Day 15	Non focal seizure	Choroid plexus tumor	Choroid plexus	discharge
Maki & Shirai[20]	Term	3.2 kg	female	Day 4	Opisthotonus seizure	Nil	Choroid plexus	discharge
Palma et al;	Term			Day 1	Hyperpyrexia jitteriness	MAS	NA	NA

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