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Evaluation of neonates suffered from cerebral edema in birth asphyxia by using transcranial color Doppler

Dr. Anup Kumar[1], Dr. Manoj Kumar Ram[2*], Dr. Anil Kumar Jaiswal[3]
[1, 3] Senior Resident, Upgraded Department of Paediatrics, Patna Medical College & Hospital, Patna, Bihar, India
[2] Professor and HOD, Upgraded Department of Paediatrics, Patna Medical College & Hospital Patna, Bihar, India
[*] Corresponding Author: Dr. Manoj Kumar Ram

Abstract
Hypoxic-ischemic encephalopathy, or HIE, is the brain injury caused by oxygen deprivation to the brain, also commonly known as intrapartum asphyxia. The new-born's body can compensate for brief periods of depleted oxygen, but if the asphyxia lasts too long, brain tissue is destroyed. Hypoxic-ischemic encephalopathy due to fetal or neonatal asphyxia is a leading cause of death or severe impairment among infants. From the above findings the present study was planned to evaluate the ultrasound presentation of the brain and cerebral haemodynamic in neonates with hypoxic-ischemic encephalopathy (HIE).
The study was planned in Upgraded Department of Paediatrics, Patna Medical College & Hospital, Patna from Jan 2018 to Aug 2018 by enrolling 25 neonates. The 25 neonates having gestational age of more than 37 to less than 42 completed weeks with perinatal asphyxia delivered PMCH were enrolled. Measurement of cerebral blood flow velocity was done by the use of colour Doppler ultrasound in Department of Radiology.
The study had proved that colour Doppler USG would be of practical importance in evaluating the cerebral blood flow velocity in neonate with HIE. Although MRI is a gold standard practice of initial screening of high risk neonates by means of transcranial ultrasound in early life is recommended because it provides useful information regarding extent and severity of hypoxic ischemic encephalopathy (HIE). Transcranial ultrasound proved to be very useful bedside screening tool for detection of hypoxic-ischemic encephalopathy (HIE).

Keywords: colour doppler, perinatal asphyxia, HIE

Introduction
Neonatal encephalopathy (NE), formerly known as Hypoxic - ischaemic encephalopathy (HIE) is a major challenge in newborn care worldwide. Literatures suggest that it is a major cause of perinatal morbidity and mortality as well as a cause of post-neonatal neurologic deficits. There are many confounding issues surrounding the definition, aetiologies and scope of NE which have often made it a matter for litigations in the developed world. While the developed world is pre-occupied with defining the precise basis of NE, the developing world still gropes with the occurrence of perinatal asphyxia which is widely believed to be a major cause of NE. Perinatal asphyxia remains a topical issue in health circles in the developing world. No doubt, there may be other yet undefined mechanisms behind NE.
Although, hypoxaemia and circulatory changes remain central to the pathogenesis of NE, the precise mechanism by which these hypoxic and ischaemic situations result in NE is still largely unknown. However, there are new ideas of the pathophysiology of NE and these have yielded new management options which are aimed at preventing further brain damage after NE. This paper aims to highlight the clinical applicability of some of these new developments about the pathophysiology and management of NE.
This is a state of acute neurologic dysfunctions resulting from the effects of perinatal asphyxia on the brain tissue. The constellation of cerebral hypoxic and ischaemic changes which follows perinatal asphyxia results in increased cerebral blood flow, cerebral oedema and massive cellular necrosis involving the cortex, basal ganglia and brain stem. Intraventricular and intracerebral hemorrhage may also occur. Depending on the extent of cerebral involvement, NE presents in different shades of severity described by Sarnat and Sarnat.6 However, the major clinical presentations include severe central nervous system depression and abnormalities of respiration, muscle tone and deep tendon reflexes.
Neuroimaging features of NE include cerebral oedema and haemorrhages within the first one week of asphyxial insult while cerebral necrosis and atrophy occur subsequently. The resuscitation of the asphyxiated infant is followed by a therapeutic window where the infant may appear grossly normal despite an on-going brain damage. Therefore, steps need to be taken to prevent secondary brain damage that occurs in the therapeutic window. The handling of this therapeutic window determines the outcome of NE. The outcome includes mortality in about 20% and neurologic sequelae in another 45%. These neurologic sequelae include handicapping motor and sensorial dysfunctions like cerebral palsy, seizure disorders, mental retardation, deafness and speech defect [1].

Hypoxic-ischemic encephalopathy, or HIE, is the brain injury caused by oxygen deprivation to the brain, also commonly known as intrapartum asphyxia. The new-born's body can compensate for brief periods of depleted oxygen, but if the asphyxia lasts too long, brain tissue is destroyed. Hypoxic-ischemic encephalopathy due to fetal or neonatal asphyxia is a leading cause of death or severe impairment among infants. Such impairment can include epilepsy, developmental delay, motor impairment, neurodevelopmental delay, and cognitive impairment. Usually, the severity of impairment cannot be determined until a child is three to four years old. Asphyxia was long thought to be the cause of Cerebral Palsy, but two studies have shown that only 9% of cases are a direct result of asphyxia. In the remaining 91% of cases, factors such as premature birth, complications of birth or problems immediately following birth cause Cerebral Palsy. In some cases, cause cannot be definitively determined.

Hypoxic-ischemic encephalopathy is most common in full-term infants, although it does occur in premature infants, as well. The timing and severity of asphyxia can affect the area of the brain that sustains the injury. If injury occurs before week 35 in fetal development, hypoxic-ischemic encephalopathy is likely to produce periventricular leukomalacia, or PVL. At 40 weeks, the degree of hypoxia correlates to the area of the brain that is injured; mild hypoxia will affect the parasagittal white matter while severe hypoxia affects the putamen, thalamus, and paracentral white matter. The area of the brain that is affected will have a significant bearing on symptoms the child experiences [2].

HIE is managed using a treatment called therapeutic hypothermia, where the baby's brain or body is cooled down below normal temperatures to slow the cascade effect that causes widespread damage. This allows the baby's brain to recover and reduces the level of disability they may have as they grow. According to current guidelines, the treatment must be given within six hours of birth, although there is some evidence to suggest it may be beneficial when given up to 24 hours.

Therapeutic hypothermia lasts for around 72 hours, allowing the baby's metabolic rate to slow. This prevents further damage known as reperfusion injury, which occurs when normal oxygenation and blood flow are restored too quickly to the brain's cells. While it may seem counter-intuitive that restoring flow quickly could cause further injury, the brain's cells react differently to rapid oxygenation after being oxygen deprived. After oxygen deprivation injury, rapid oxygenation can cause more inflammation and the release of certain harmful compounds. Hypothermia treatment works to stabilize the brain's cells and prevent or limit damaging inflammation [3].

From the above findings the present study was planned to evaluate the ultrasound presentation of the brain and cerebral haemodynamic in neonates with hypoxic-ischemic encephalopathy (HIE).

Methodology

The study was planned in Upgraded Department of Paediatrics, Patna Medical College & Hospital, Patna from Jan 2018 to Aug 2018 by enrolling 25 neonates. The 25 neonates having gestational age of more than 37 to less than 42 completed weeks with perinatal asphyxia delivered PMCH were enrolled. Measurement of cerebral blood flow velocity was done by the use of colour doppler ultrasound in Department of Radiology. Cranial Ultrasound was done in neonates with birth asphyxia in first 24 hours of life and repeat scan on 3rd postnatal day.

Inclusion Criteria

- Neonates with birth asphyxia
- Preterm neonates
- Neonates with convulsion of unknown etiology

Exclusion Criteria

- Babies having jaundice
- Congenital anomalies

Results & Discussion

The data from the enrolled patients were collected and presented as below.

The table 1 indicates the distribution of the patients as per the grades of the HIE.

Table 1: Grades of the patients

Grade	Males	Female
HIE-1	3	1
HIE-2	12	5
HIE-3	2	2
Total	17	8

Table 2: Mean RI value in Anterior Cerebral artery according to grading of birth asphyxia

	Number of cases	1st day	3rd day
HIE-1	4	0.67 – 0.71	0.45 – 0.61
HIE-2	17	0.52 – 0.58	0.51 – 0. 63
HIE-3	4	0.20 – 0.24	0.25 – 0.29

This table show that means RI value in Middle cerebral artery decrease with severity of birth asphyxia, it is lowest in HIE-3, and in grade -2.

Birth asphyxia in neonates and preterm mainly affects deep gray matter like putamen, ventrolateral thalami, hippocampi, brainstem, and lateral geniculate nuclei. These areas of the brain contain the highest concentrations of NMDA receptors and have high metabolic demands (because of ongoing myelination) therefore, the most susceptible to neonatal hypoxic-ischemic encephalopathy (HIE) [4]. Donna Ferriero *et al.,* suggested that mortality and morbidity from neonatal brain injury is significantly high and associated with long term effects in form of stroke, status epilepticus and cerebral palsy [5]. Transcranial ultrasound in early life first 7 days have proven less sensitive for detection of changes of hypoxic-ischemic encephalopathy (HIE). After first week changes are more evident. Early transcranial ultrasound findings include increase cortical echogenicity, obliteration of CSF spaces because of edema, increase thalamic echogenicity, persistent periventricular echogenicity. Thalamic involvement associated

with poor outcome and more severe injury. Late findings include cystic changes involving cortex and subcortical area, encephalomalacia, ex vacuo ventricular dilatation, prominent extra-axial CSF spaces, rarely porencephalic cyst formation [6].

Brain edema is a pathological change that is characteristically observed after asphyxia [7], and the main ultrasound manifestation of brain edema is diffuse parenchymal echo enhancement. More intense patterns of parenchymal echo indicate more severe neuronal damage. With brain edema, the intracranial structures appear fuzzy with shallow sulci and narrowed or undetected ventricles [8]. The extent and time required for the cerebral edema to subside are closely related to the neonatal prognosis.

Hemodynamic changes are involved in the major pathophysiological mechanisms that underlie post asphyxia brain damage [9]. Many studies from China and other countries have used color Doppler ultrasound to monitor the hemodynamic changes that occur in the brain after asphyxia, but the results have been inconsistent. Ilves *et al*. [10] have found that infants with severe HIE exhibit increased blood flow velocities of cerebral artery blood and decreased RI values within 24 h of asphyxia, thus indicating high velocity and low resistance. Those authors suggested that hyperperfusion was the most important manifestation of brain injury in the early stages after asphyxia. Liu *et al*. [11] have suggested that the cerebral blood flow of HIE patients is disordered and that the RI can significantly increase or decrease within the first 24 h of asphyxia. Patients with severe HIE have RIs that are less than 0.50 or greater than 0.90, and brain death occurs when the RI is greater than 1.0.

When asphyxia is followed by hypoxic ischemic injury to brain, a syndrome has been described known as 'Hypoxic ischemic encephalopathy'. Early assessment of the degree of resulting hypoxic– ischaemic encephalopathy (HIE) can provide prognostic information for both clinical management and the potential use of cerebroprotective strategies. However, clinical assessment is often difficult because the neurological state of the infant may be altered by pharmacological interventions such as sedation, muscle relaxation, or anticonvulsant treatment. Moreover, clinical signs of HIE may not develop until at least 12 hours after birth.

The haemodynamics in term infants with acute encephalopathy are deranged during the first 24 hours after presumed perinatal asphyxia. These consist of an increase in cerebral blood flow velocity (CBV) and a significant reduction in cerebral blood flow velocity resistance (CBVR). CBVR tended to return to normal values after the first 24 hours of age. CBV and CBF were frequently increased in the first 24 hours after birth were associated with a greater severity of acute encephalopathy signs and adverse outcome [12].

Attempts have been made to diagnose ischemic encephalopathy by use of CT, MRI and ultrasonography. Various authors in different study have emphasized that Doppler sonography is a practical, non-invasive and accurate method of diagnosing a wide spectrum of intra-cranial conditions due to HIE in neonate. Sonography is now established as a primary method for recognizing neonatal cerebral ischemia.

Having found normal RI values with Doppler method, the clinician can confidently reassure parents that their baby has a little risk of death. Prognostication of long term outcomes is one of the main objectives in Doppler sonography of the brain of full term neonate who experienced perinatal asphyxia. In this study maximum number of cases occurred in male.

Our study analyzed the relationship of cerebral blood circulation parameters (PSV, EDV & RI) registered in the anterior & middle cerebral arteries evaluated at 12-24 hr of life and its relation to long term neurological outcome.

Doppler studies done in intracranial vessels in asphyxiated infants found an increase in diastolic flow and lowering of the RI to be the usual initial response detectable in initial 4 days of birth asphyxia [13].

Conclusion

The study had proved that colour Doppler USG would be of practical importance in evaluating the cerebral blood flow velocity in neonate with HIE. Although MRI is a gold standard practice of initial screening of high risk neonates by means of transcranial ultrasound in early life is recommended because it provides useful information regarding extent and severity of hypoxicischemic encephalopathy (HIE). Transcranial ultrasound proved to be very useful bedside screening tool for detection of hypoxic-ischemic encephalopathy (HIE).

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