ORIGINAL RESEARCH PAPER

VOLUME-6 | ISSUE-6 | JUNE-2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

Study of Ultrasonographic Evaluation of Patterns of Neonatal Brain Injury in Cases of Perinatal Asphyxia

Radiology

Dr. Mukesh Bihari Assistant Professor, Department of Radiology J.L.N. Medical College & Hospital, Bhagalpur

Dr. A. K. Murarka H.O.D. . Department of Radiology, J.L.N. Medical College & Hospital, Bhagalpur

Dr. Ankur Priyadarshi Assistant Professor, Department of Paediatrics, J.L.N. Medical College & Hospital, Bhagalpur

ABSTRACT

Background: Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Hypoxic ischemic encephalopathy (HIE) refers to the CNS dysfunction associated with perinatal asphyxia. HIE is foremost concern in an asphyxiated neonate because contrary to other system derangements this has the potential to cause serious long term neuromotor sequelae among survivors.

Objectives: To study the different varieties of gross anatomical defects of the neonatal brain in cases of HIE as seen by ultrasonography.

Materials and Methods: Ultrasonography (USG) of the neonatal brain was done in 100 cases of hypoxic ischemic encephalopathy (HIE) during the first and second weeks of life, through anterior, posterior and mastoid fontanelles The results were analysed to display the spectrum of abnormalities in the affected babies.

Results: A large proportion of the neonates had a normal study (41%), followed by intracranial haemorrhage (30%), cerebral oedema and infarction (16%), dilatation of ventricles (6%), PVL (4%)and thalamic lesion in (3%).

Conclusion: Intracranial haemorrhage is the commonest consequence of hypoxic ischemic encephalopathy. The large number of normal studies shows the need for combining ultrasonography with other imaging modalities in selected cases

KEYWORDS:

INTRODUCTION

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. It ranks as the second most important cause of neonatal death after infections, accounting for around 30 % mortality worldwide. In India, between 250,000 to 350,000 infants die each year due to birth asphyxia, mostly within the first three days of life. In addition, ante-partum and intra-partum asphyxia contributes to as many as 300,000 to 400,000 stillbirths. Perinatal asphyxia may result in adverse effects on all major body systems. Many of these complications are potentially fatal. In a term infant with perinatal asphyxia renal, neurologic, cardiac and lung dysfunction occurs in 50%, 28%, 25% and 23% cases respectively. The manifestations of hypoxic and ischemic injury to the brain in a developing foetus in utero or during the birth process consist of a pattern of abnormal neurological signs occurring in sequence over a period of the first few days of life. This is known as hypoxic ischemic encephalopathy (HIE) [1]. HIE occurs in about 25% infants following severe birth asphyxia [2]. HIE is foremost concern in an asphyxiated neonate because contrary to other system derangements this has the potential to cause serious long term neuromotor sequelae among survivors. A clinical staging system has been described in the Sarnat staging system. A clinical diagnosis of hypoxic ischemic encephalopathy automatically initiates a search for the anatomical location and extent of the brain lesions and a follow up of their progress over the next few weeks. This helps the clinician to formulate a line of management for the affected neonate and form some idea of the short and long term prognosis for the affected child.

MATERIALAND METHODS

The present study is a descriptive study which was conducted in the Department of Radiology and Department of paediatrics, at *J.L.N. Medical College & Hospital, Bhagalpur* The study was conducted over a period of one year from January 2016 to December 2017. The patients were selected from the NICU, Department of Paediatric , *J.L.N. Medical College & Hospital, Bhagalpur.* Proper informed consent was taken from the Care givers or guardians of the babies during the study and the Institutional Ethics Committee prior to the study. A total of 100 cases of birth asphyxiated babies showing clinical features of Hypoxic Ischemic Encephalopathy (HIE) were selected for the present study. Of these, 70 babies were born at term and 30 were pre-term.

Cases included in our study were those neonates who were suspected to be affected by hypoxic ischemic encephalopathy (HIE) according to the clinical features displayed in the Sarnat staging system. Neonates who showed features of altered consciousness, diminished muscle tone and reflexes as a result of correctable electrolyte disturbances, neonatal infections and developmental anomalies and malformations were excluded from the study. While all neonates suspected of having HIE were thoroughly investigated and treated, only the first 100 cases referred to the Department of Radiology for neonatal USG after being diagnosed with suspected hypoxic ischemic encephalopathy were included in our study. In all cases, USG of the brain was done in the 1st and 2nd weeks of post natal life. Ultrasonographic scans were done through the anterior, posterior and mastoid fontanelles.

RESULTS

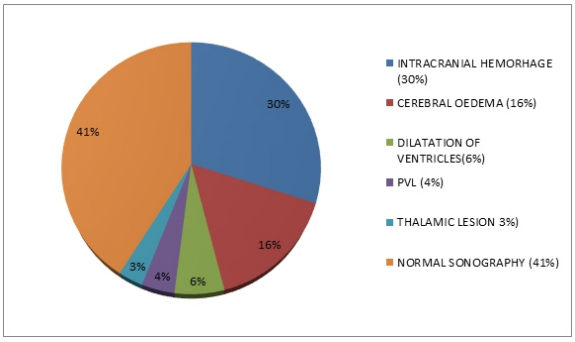
In the current study, 100 babies were asphyxiated at birth including term and preterm births. Among these 100 babies, intracranial haemorrhage (ICH) was found in 30 babies (30%), cerebral oedema in 16 babies (16%), various degrees of dilatation of ventricles in 6 babies (6%), other abnormalities in 7 babies and normal study was seen in 41 cases (41%). These findings are displayed in [Table/Fig-1]. Intracranial haemorrhage (30%) included cases of subependymal haemorrhage, intraventricular haemorrhage and intra parenchymal haemorrhage. The cases of intra parenchymal haemorrhage included cases of periventricular and subcortical haemorrhage. Intraventricular and periventricular haemorrhages may be considered together as Germinal Matrix Haemorrhages (GMH). In the present study out of 100 neonates, 30 were preterm and 70 were mature (term). 41 cases of clinically diagnosed hypoxic ischaemic encephalopathy (HIE) showed a normal sonographic scan..

Table 1 Distribution of Sonographic lesions in Hypoxic Ischaemic Encephalopathy

Intracranial Lesion		Term Babies	Pre-term Babies	Total Percentage
Intracranial haemorrhage	Sub ependymal haemorrhage (SEH)	0	7	30%
	Intra ventricular haemorrhage (IVH)	5	10	
	Intra parenchymal haemorrhage (IPH)	3	2	
	Subarachnoid haemorrhage (SAH)	3	0	

Cerebral oedema and infarction	Diffuse oedema	13	1	16%
	Infarction	2	0	
Dilatation of Ventricles		4	2	6%
PVL		0	4	4%
Thalamic lesion		3	0	3%
Normal Sonography		35	6	41%

Fig. 1Distribution of Sonographic lesions in Hypoxic Ischaemic Encephalopathy



DISCUSSION

Cranial ultrasound is used as the first imaging modality for newborns [6]. Selection of neonates for ultrasonographic screening of the brain to determine the pattern of hypoxic injury requires a high index of suspicion. Patterns of hypoxic ischemic brain injury detected by sonography have been shown to correlate very well with autopsy findings [7].

Features of Hypoxic Ischemic Encephalopathy

Preterm neonates exhibit very few clinical features suggestive of hypoxic-ischemic brain injury. Detection needs meticulous observation of subtle signs and symptoms. In all neonates the problem may be suspected by the following features –

Impairment or of consciousness or irritability, impairment of muscle tone, tendon reflexes, sucking, Moro response, grasping and oculo cephalic reflex; abnormal pupils, respiration, heart rate; seizures and EEG changes.

Summary of Human Brain Development

Brain development starts from the epiblast cells of the germ disc of the embryo. The epiblast further differentiates into surface ectoderm and neuro ectoderm. The neuro ectoderm in turn, undergoes a folding to form a neural tube. The cephalic expanded part of the neural tube forms the forebrain, midbrain and hindbrain vesicles.

The cells of the neural tube differentiate in three layers – from inside outwards ependymal, mantle and marginal layers. Cells of the ependymal layer migrate outwards and differentiate into neuroblasts and spongioblasts forming nerve cells and glial cells respectively [8].

Maturational Changes in the Human Brain in the 3rd Trimester

It is generally agreed that the dividing line between the immature and the mature brain is around 34 weeks. The germinal matrix is a fine network of blood vessels and primitive neural tissue that lines the ventricular system in the subependymal layer during fetal life. As the fetus matures, the germinal matrix regresses toward the foramen of Monro so that by full term only a small amount of germinal matrix is present in the caudothalamic groove. These delicate vessels are highly susceptible to pressure and metabolic changes, which can lead to rupture of the vessels. Premature infants are therefore at risk of spontaneous hemorrhage in and around the ventricles [9]. Full-term infants rarely experience this type of hemorrhage [10]. There is a high degree of vascular supply to the basal ganglia and the germinal matrix on one side and numerous small penetrating branches from the leptomeningeal vessels to a thin cortex on the other side. A watershed area, therefore, exists between these two vascular territories in the periventricular white matter. HIE related pathological lesions in the immature brain are germinal matrix hemorrhage and periventricular leukomalacia (PVL) which are highly specific and are observed in this watershed area. After 34 weeks of gestation, the cerebral vascular architecture matures with less chance of haemorrhage. In a nutshell, it may be said that in full term babies, parasagittal cerebral injury occurs as a result of generalised reduction in the cerebral blood flow. In preterm babies, the area of infarction involves the deeper periventricular white matter . In the present study 30% of the neonates showed features of intracranial hemorrhage. This is detected on USG by increased echogenic masses occupying ventricles, separating and thickening the sulci. Among the hypoxic preterm babies, the greatest incidence was of intracranial hemorrhage while among the full term hypoxic babies the maximum number of cases had a normal scan. This in general highlights the lability of intracranial blood vessels in the developing foetus as well as the discrepancy between structure and function, between gross anatomical impressions and pathological conditions in full term hypoxic babies.

Limitations Of USG study

Over half of the total number of cases showed a normal USG scan despite having features of Hypoxic Ischemic Encephalopathy (51%). This may be due to several reasons –

- USG may not be the optimal investigation for all cases of HIE. Although USG is invaluable as a screening tool, the level of differentiation and clarity may be insufficient for minor alterations in cerebral structure.
- Some cases of small subependymal haemorrhages resolve quickly and are not visible by one week.
- Some cases of periventricular leukomalacia and hydrocephalus manifest beyond two weeks
- Many cases of HIE may have functional abnormalities, but may not have gross structural anomalies
- Alterations in cerebral blood flow and effect of metabolic toxins may not be detectable by routine USG.

A more detailed analysis of the neonatal brain may be obtained by combining USG with cerebral Doppler studies, CT scan and functional imaging methods. However, the portability, availability, ease of application, low cost and lack of side effects and radiation exposure make USG the most desirable initial investigation for neonatal hypoxia.

CONCLUSION

The term birth asphyxia denotes a constellation of signs and symptoms resulting from inadequate perfusion and oxygenation of the foetus during the process of childbirth. This is known as hypoxic ischemic encephalopathy (HIE). It results in several forms of pathology of the brain characterized by hemorrhage and softening of brain tissue around a periventricular watershed zone. Although, USG is easy to perform and cost effective, functional hypoxic damage to the cerebral neurons may not always be evident on ultrasonography. Therefore, USG studies may be supplemented with MRI and functional imaging of the brain for better analysis of hypoxic ischemic encephalopathy in neonates.

REFERENCES

[1] Namasivayam A, Carlo WA. Nelson textbook of pediatrics 19th ed. Philadelphia: Saunders Elsevier; 2011. p. 569 – 73.
[2] Singh M. Care of the newborn. 5th ed. New Delhi: Sagar Publications, 2002. p.114.
[3] Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. Arch Neurol .1976; 33(10): 696- 705.
[4] Pigadas A, Thompson JR, Grube GL. Normal infant brain anatomy: correlated real-time sonograms and brain specimens. AJR. 1981; 137(4): 815 – 20.
[5] Yousefzadeh DK, Naidich TP. Ultrasound anatomy of the posterior fossa in children : Correlation with brain sections. Radiology .1985; 156 (2) : 353 – 61.
[6] Gupta SN, Amer MK, Uday SK. Intracranial hemorrhage in term newborns: Management and outcomes. Pediatric Neurology. 2009; 40(1): 1-12.
[7] Eken P, Jansen GH, Groenendaal F, Rademaker KJ, de Vries LS. Intracranial lesions in the fullterm infant with hypoxic ischaemic encephalopathy: ultrasound and autopsy correlation. Neuropediatrics. 1994; 25(6): 301-07.
[8] Sadler TW. Langman's medical embryology. 10th ed. Philadelphia: Lippincott Williams & Wilkins; 2006; p. 285 – 316.
[9] Soni JP, Gupta BD, Soni M, Gupta M, Dabi DR, Nemal KR. Ultrasonic diagnosis of intracranial hemorrhage in high risk neonates. Indian Pediatrics. 1995; 32(4): 453-60.
[10] Bowerman RA, Donn SM, Silver TM, Jaffe MH. Natural history of neonatal periventricular / intraventricular haemorrhage and its complication : sonographic observation. AJR Am J Roentgenol. 1984;143(5):1041-52.