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ROLE OF CRANIAL ULTRASONOGRAPHY IN PRETERMNEONATES

Dr. Aditi Parekh *, Dr. Alpa Parekh **, Dr. Gargi Pathak***

* Assistant Professor , SVP hospital , Ahmedabad

**Associate Professor , Civil hospital , Bhavnagar

*** Associate Professor , civil hospital, Ahmedabad

Corresponding Author : Dr. Aditi Parekh

ABSTRACT

Prematurity is associated with variable degree of brain injuries and adverse neurodevelopmental outcomes. Therefore cranial ultrasound is used as a screening tool to assess further neurodevelopmental abnormalities. A prospective study was conducted over a period of 4 months .50 preterm infants admitted to neonatal intensive care unit were subjected to ultrasonography on selected days. Total 50 preterms were included of which 30(60%) male, 20(40%) female. Correlation between gestational age and cranial ultrasonography findings was statistically significant. 10% had evidence of intracranial bleed, 2 %- periventricular echogenicity , 2% - ventriculomegaly and 2%- periventricular echogenicity.

INTRODUCTION

Cranial ultrasonography has become an essential diagnostic tool in modern neonatology for depicting normal anatomy and pathological changes in neonatal brain. In the neonate the fontanalles are open and these can be used as acoustic window to look into the brain.

It is a reliable tool for detecting most of the haemorrhagic, ischaemic and cystic lesions as well as calcifications, cerebral infections and major structutral abnormalities

MATERIALS AND METHODS

A prospective study was conducted over a period of 4 months from in neonatal intensive care unit of civil hospital Ahmedabad. 50 preterm neonates admitted to neonatal intensive care unit were selected as per inclusion criteria and subjected to ultrasonography on selected days.

If cranial ultrasonography revealed any significant findings than repeat ultrasonography was done to follow up sequale if any.

Informed consent of relative was taken

INCLUSION CRITERIA

- Preterms with abnormal neurological manifestations like seizures, lethargy, apnoea, sudden onset pallor, increase in muscle tone, bulging anterior fontanalle.
- All preterms born prior to 32 weeks

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- All preterms weighing less than 1500 gms

EXCLUSION CRITERIA

- Any major congenital lethal malformation
- Term and post term babies

Assesment of risk factors was done taking a detailed maternal history . All perinatal details were received and detailed clinical examination of newborn was done

Gestational age assessed as per modified ballards scoring method.

Basic routine investigations like septic screening, random blood sugar, ionised calcium, chest xray for respiratory symptoms was done. Cranial ultrasonography was done for all neonates included in the study

Intraventricular haemorrhage grading was done according to volpe staging and clinical correlation with ultrasonography findings was done

OBSERVATION AND RESULTS

1. **Gestational age wise distribution of cases**

Out of 50 neonates enrolled in the study only 8 showed abnormal findings

GESTATIONAL AGE	NUMBER OF NEONATES
28-30 WEEKS	2
30-32 WEEKS	3
32-34 WEEKS	2
34-36 WEEKS	1

2. **Incidence of various cranial ultrasound abnormalities in neonates**

CRANIAL ULTRASOUND	NUMBER OF NEONATES	PERCENTAGE
NORMAL	42	84%
ABNORMAL	8	16%
GMH	5	10%
PERIVENTRICULAR ECHOGENICITY	1	2%
LEUCOMALACIA	1	2%
VENTRICULOMEGALY	1	2%

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3. Cranial ultrasound findings in relation to days of life

USG cranium	Number of neonates	Days of life		
		<24 hours	24-48 hours	>72 hours
ABNORMAL	8	0	6	1
GMH	5	0	5	0
Periventricular echogenicity	1	0	1	0
Leukomalacia	1	0	0	1
ventriculomegaly	1	0	1	0

4. Outcome of the neonates enrolled in the study

OUTCOME	NUMBER OF NEONATES
Relieved	1
Cured	4
Death	1
DAMA	2

5. Distribution of various clinical presentation

Presentation	Number of neonates	Percentage
Seizure	3	37.5%
Lethargy	1	12.5%
Pallor	1	37.5%
Bulging fontanalle	3	12.5%

Total 50 preterm neonates were included in the study of which 30 were male and 20 were female. No correlation was found between sex of the patient and ultrasonography finding.
Co relation between gestational age and cranial ultrasonography was significant.
10% of the neonates has evidence of intracranial bleed, 2 % had periventricular echogenicity, 2% had ventriculomegaly and 2 % had periventricular echogenicity.

Discussion

Intracranial hemorrhage in preterm infants develops usually spontaneously. Less frequently, it may be also caused by trauma or asphyxia, rarely, it occurs from a primary hemorrhagic disturbance or congenital cerebrovascular anomaly. Intracranial hemorrhage often involves the ventricles (intraventricular hemorrhage, IVH) of premature infants delivered spontaneously without any apparent trauma. The IVH in premature infants is usually not present at birth but may develop during 1st week of life. Primary hemorrhagic disturbances and vascular malformations are usually rare and give rise to subarachnoid or intracerebral hemorrhage. In utero hemorrhage associated with maternal idiopathic or, often, fetal alloimmune thrombocytopenia may appear as severe cerebral hemorrhage or as a porencephalic cyst after resolution of a

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fetal cortical hemorrhage. Intracranial bleeding may be associated with disseminated intravascular coagulation, isoimmune thrombocytopenia, and neonatal vitamin K deficiency, especially in infants born to mothers receiving phenobarbital or phenytoin.

EPIDEMIOLOGY

The overall incidence of IVH has decreased over the past decades as a result of improved perinatal care, increased use of antenatal corticosteroids, surfactant to treat respiratory distress syndrome (RDS), and possibly prophylactic indomethacin

PATHOGENESIS

The major neuropathologic lesions associated with very-low-birthweight (VLBW) infants are IVH and PVL. IVH in premature infants occurs in the gelatinous subependymal germinal matrix. This periventricular area is the site of origin for embryonal neurons and fetal glial cells, which migrate outwardly to the cortex. Immature blood vessels in this highly vascular region of the developing brain combined with poor tissue vascular support predispose premature infants to hemorrhage. The germinal matrix involutes as the infant approaches full-term gestation, and the tissue's vascular integrity improves; therefore IVH is much less common in the term infant. The cerebellum also contains a germinal matrix and is susceptible to hemorrhagic injury.

The Predisposing factors for IVH include prematurity, RDS, hypoxiaischemia, exaggerated fluctuations in cerebral blood flow (hypotensive injury, hypervolemia, hypertension), reperfusion injury of damaged vessels, reduced vascular integrity, increased venous pressure (pneumothorax, venous thrombus), or thrombocytopenia. PVL is characterized by focal necrotic lesions in the periventricular white matter and/or more diffuse white matter damage. Instead, diffuse injury leading to abnormal maturation of neurons and glia is more frequently seen. The risk for PVL increases in infants with severe IVH or ventriculomegaly. Infants with PVL are at higher risk of cerebral palsy because of injury to the corticospinal tracts that descend through the periventricular white matter

CLINICAL MANIFESTATIONS

Many infants with IVH, including some with moderate to severe hemorrhages, have no initial clinical signs (silent IVH). Some premature infants in whom severe IVH develops have acute deterioration on the 2nd or 3rd day of life (catastrophic presentation). Hypotension, apnea, pallor, stupor or coma, seizures, hypotonia, metabolic acidosis, shock, and decreased hematocrit (or failure of hematocrit to rise after transfusion) may be the first clinical indications. A saltatory progression may evolve over several hours to days and manifest as intermittent or progressive alterations of levels of consciousness, abnormalities of tone and movement, respiratory signs, and eventually other features of the acute catastrophic IVH. PVL is clinically asymptomatic until the neurologic sequelae of white matter damage become apparent in later infancy as spasticity and/or motor deficits. PVL may be present at birth but usually occurs later, when the echodense phase is seen on ultrasound (3-10 days of life), followed by the typical echolucent/cystic phase (14-20 days). The

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severity of hemorrhage is defined by the location and degree of bleeding and ventricular dilation on cranial imaging.
In a grade I hemorrhage, bleeding is isolated to the subependymal area.
In grade II hemorrhage, there is bleeding within the ventricle without evidence of ventricular dilation.
Grade III hemorrhage is IVH with ventricular dilation.
In grade IV hemorrhage, there is intraventricular and parenchymal hemorrhage.
Another grading system describes 3 levels of increasing severity of IVH detected on ultrasound:
In grade I, bleeding is confined to the germinal matrix–subependymal region or to 10% of the ventricle is involved, with dilated ventricles.
Grade II is defined as intraventricular bleeding with 10–50% filling of the ventricle (40% of IVH cases);
And in grade III, >50% of the ventricle is involved, with dilated ventricles .
Ventriculomegaly is defined as mild (0.5-1 cm dilation), moderate (1.0-1.5 cm dilation), or severe (>1.5 cm dilation). Ventriculomegaly is defined as mild (0.5-1 cm dilation), moderate (1.0-1.5 cm dilation), or severe (>1.5 cm dilation).

CONCLUSION

Neurosonogram remains the accurate rapid imaging technique of choice for detecting brain injuries in preterm neonates. This technique is both sensitive and specific for detecting various brain anomalies. Cranial ultrasonography is best initial method of investigation on preterm babies with suspected neurological injuries

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