



OBSEVATIONAL STUDY ON ROLE OF NEUROSONOGRAM IN INTRACRANIAL PATHOLOGIES IN NEONATES INA TETIARY CARE CENTER

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ABSTRACT

Background: Neonates face a lot of challenges especially based on their gestational age, maternal factors, birth weight at the time of delivery, immediate cry following birth. Hypoxic ischemic encephalopathy and Germinal matrix haemorrhage constitutes major portion of those challenges. To identify varied conditions, we can take help of imaging in form of neurosonogram. **Methods :** Neurosonogram was carried out on all n tool including follow neonates both as screening and diagnostic up scans. A total of 100 preterm babies with positive findings were included in the study. An analysis was performed over different pathologies including grading of specific condition in required areas. **Results:** Our study comprising of 100 neonates with brain pathologies showed following results. In our study of 100 neonates, most of the disease burden is by vascular causes (56%), then followed by infective (19%), congenital (14%), trauma (8%), metabolic (2%) and miscellaneous (2%). 52% of PVL showed associated risk factors. **Conclusion:** In our study 100 preterm babies with suspected brain injuries were studied with ultrasound through anterior fontanelle and mastoid sutures. Neurosonogram can detect various intracranial pathologies on par with CT scan. Transcranial doppler can reflect the cerebral blood volume and perfusion status through resistive index and abnormal values according to literature are >0.8 and <0.4 and can be noticed in severe hypoxic ischemic encephalopathy and conditions which increases intracranial pressure. Any measure that decreases the vasospasm which decreases RI can eventually prevent the neonate having progression of ischemia and further new infarct.

Keywords: Anterior cerebral artery system, Cerebroventricular hemorrhage; Germinal matrix hemorrhage

INTRODUCTION

Second Many radiologists are not quite familiar with spectrum of radiological findings with application of neurosonography due to inadequate exposure. this observational study includes various spectrum of findings using neurosonography, its various techniques and indication [1] Indications of neurosonography includes intraventricular haemorrhage, periventricular leukomalacia and porencephaly, hydrocephalus, chiari malformations dandy-walker complex, agenesis of corpus callosum, holoprosencephaly, vascular malformations, lenticulostriate vasculopathy and colour doppler ultrasound [2]. The incidence of intraventricular haemorrhage (IVH) and cystic periventricular leukomalacia (PVL) has diminished considerably with help of early cranial ultrasound imaging. Current studies aim more at detecting subtle white matter disease, assessing brain growth and maturation, and predicting neurodevelopment outcome from the cranial ultrasound [3]. CT is not typically used in the premature infant because of the instability of the infant and the lack of good grey / white matter differentiation from the high water content in the new-born brain. The advantages of sonography over computed tomography (CT)/ magnetic resonance imaging (MRI) include portability, lower cost, speed, no ionizing radiation, and no sedation. Screening of prem-

ature infants for intracranial haemorrhage has proven highly sensitive and specific. Ultrasound is essential to the neonatal evaluation and follow up of hydrocephalus and periventricular leukomalacia (PVL) [4]. When performed according to protocol, neurosonography is a reliable technique for most newborn illnesses. With the help of the ultrasound, which is portable and can be done at the bedside, a neurosonogram is widely used as an initial screening tool of the neonatal brain and can be repeated as many times as possible. By increasing the fair and efficient use of neurosonogram in the neonates to screen the cranial abnormalities, various conditions causing severe morbidity or mortality can be detected early in their course and thereby be efficiently managed for better prognosis [5].

MATERIAL AND METHOD

A prospective observational study of 100 neonates with Neurosonogram was performed from January 2021 to June 2021 in the department of radiodiagnosis Osmania General hospital, Hyderabad.

Source of data: Niloufer child and maternity hospital affiliated to Osmania general hospital.

Selection of patients:

Inclusion criteria:

- Babies under one month period including term and preterm
- Babies under NICU admission
- Asymptomatic children included for screening NSG
- Very low birth babies $<1500g$



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Exclusion criteria:

- Babies exhibiting poor window for NSG like thick hair and premature closure of fontanelles
- Babies for whom unable to do NSG due to excessive cry Preparation of the patient:
- Neonates were transported to ultrasound room by wrapping them in warm clothing to maintain normal body temperature.
- Baby was fed adequately before examination.
- No sedation was used
- Baby was laid in supine.
- Hand washing and cleansing of the transducer was done.

Equipment: All the preterm babies in this study underwent neurosonogram using curvilinear transducer and linear array high frequency transducer of ESAOTE ultrasound equipment with both curvilinear probe (2-5hz) and linear probe (6-10hz) are used .in necessary cases, Plain/Contrast CT was advised using Canon 16 Slice CT.

Performa: NEUROSONOGRAM REPORT

1. Falx cerebri: seen- central /displaced not seen
2. Cerebral echotexture : normal / increased / decreased sulci and fissures : normal/ increased echo texture
3. focal parenchymal echodense lesion : absent / present (location)
4. periventricular echogenecity: present/absent
5. Size of the ventricle : normal/not- visualised /slit like/ enlarged
6. Caudothalamic grooves /intraventricular hemorrhage : yes / no if yes : grade i/ii/iii/iv
7. Choroid plexus: bleed/normal
8. Cerebellum :normal/ hyperechoic/ hypoechoic
9. Impression :normal/) gmh (grade)/pvl (grade/ ventriculomegaly)

Case report:

1. Baby of (Mother's Name)
2. Sex
3. Date and time of birth
4. Hospital number
5. Presenting complaints
6. Maternal history
7. Gestational age (in weeks)
8. Mode of delivery: a) Vaginal delivery b) assisted breech c) forceps d) c section
9. Vitals: a) pulse b) respiratory rate c) temperature
10. Birth weight of body (in grams)
11. General physical examination (positive if any)
12. RS, CVS, P/A (positive if any)
13. CNS EXAMINATION : Anterior fontanelle : a. Normal b) Bulged c) Depressed
14. Level of consciousness: Hyperactive b) Lethargic c) Stuporous d) Coma
15. Tone: a) Normal b) hypotonic c) hypertonic
16. Investigations (positive if any)

RESULT

Our study comprising of 100 neonates with brain pathologies showed following results

Table 1: Gender wise distribution of cases

SEX	NUMBER
MALE	60
FEMALE	40

Table 1 shows gender wise distribution of cases where 60 neonates were male and 40neonates were female.

Table 2: Gestational age wise distribution of cases: Pre-term

GESTATIONAL-AGE (WEEKS)	MALES	FEMALES	%
<28	1	1	2%
28-31.6	4	5	9%
32 – 36.6	24	13	37%
TOTAL	30	18	42

Table 2 shows maximum cases among pre-terms. In our study, out of 100 neonates,45 neonates are preterm among them 1% constitutes for gestational age <28 weeks, 6% constitutes for gestational age 28-31.5 weeks and 38% constitutes for gestational age between 32-36.5.

Table 3: Gestational age-wise distribution of cases: term

Age (weeks)	Males	Females	%
37-38.6	19	13	32
39-40.6	9	9	18
41-41.6	2	0	2
Total	30	22	52

In our study, out of 100 people, 52 neonates are term gestation. Among them, 32 neonates fall under 32 % of gestational age between 37- 38.6 weeks; 18 % fall between 39-40.6 weeks and 2 % fall between 41-41.6 gestational age.

Table 4: Percentage of incidence of cases using cranial neurosonography

Pathology	Percentage
congenital	14
infective	19
PVL	24
GMH	22
trauma	8
Metabolic	2
miscellaneous	2

In our study of 100 neonates, most of the disease burden is by PVL & GMH accounting to 56 % combined, then

followed by infective (19%), congenital (14%), trauma (8%), metabolic (2%) and miscellaneous (2%)

Table 5: weight distribution among 100 neonates

Birth weight (kgs)	No. of cases	Percentage
<1.5	3	3
1.5 – 1.9	18	18
2.0–2.4	25	25
2.5-2.9	21	21
3.0 -3.5	26	26
3.5-4.0	7	7
TOTAL	100	100

Table 6: Distribution of gmh among 22 neonates in 100 study group

GRADE	PERCENTAGE
GMH GRADE 1	7
GMH GRADE 2	4
GMH GRADE3	9
GMH GRADE4	2
TOTAL	22

In our study , GMH cases were 22 . GMH Grade1 was seen in 7 neonates. GMH Grade 2 was seen in 4 neonates . GMH Grade 3 was seen in 9 neonates . GMH grade4 seen in 2 neonates which is accounting for 7%, 4%, 9%,2% of population respectively

Table7: Incidence of GMH based on birth weight

WEIGHT Kg	GMH Grade			
	1	2	3	4
<1.5	-	-	1	1
1.5 kg to 2.5	6	4	7	1
2.5 kg to 3.5	1	0	1	0

In our study, <1.5 kg birth weight, GMH grade 3 and grade 4 cases were seen with contributing to 2 % of entire population. GMH grade 4 cases not noted in 2.5 kg to 3.5kg birth weight cases. Grade 1 and grade 3 cases were observed more in birth weight group 1.5 to 2.5 kg.

Table 8 : Incidence of GMH among gestational week at delivery

	<28 WEEKS	28-31.6W	32-36.6W
GMH 1	0	2	7
GMH 2	0	0	3
GMH 3	0	5	3
GMH 4	2	0	0

In our study , more cases of GMH grade 1 seen in 32 - 36.6 weeks gestational birth age accounting to 7 %. more cases of GMH grade 2 seen in 32 -36.6 weeks gestational birth age accounting to 3 %. more cases of GMH grade 3 seen in 28-31.6 weeks gestational birth age accounting to 5%. cases of GMH Grade4 seen in <28 weeks gestational birth age accounting to 3 %.

Table 9: Incidence of PVL

	PVL GRADE			
	1	2	3	4
NUMBER	5	5	6	8

In our study, PVL Grade 1 Is contributing to 20.8% of 24 neonates. PVL Grade 2 is contributing to 20.8 % of 24 neonates sharing same percentage a PVL GRADE 1.PVL grade 3 is contributing to 25 % of cases and PVL grade 4 contributing highest as 33.3% cases.

Table 10: PVL and Clinical picture

	PVL NEONATE
APGAR SCORE <7	8
MATERNAL PREECLAMPSIA	4
RDS	8
SEIZURES	14
PPROM	2
PROLONGED VENTILATION	9

The table and chart demonstrating the risk factors associated with PVL . 58% of cases showed associated with risk factors whereas 42% of cases there is no associated risk factors or not found

Table 11: Number of cases due to infection

Infection	NUMBER
Ventriculitis	8
Meningoencephalitis	7
TORCH	4

Table 11 showing different infection cases found during our study period . Total number of cases were 19 cases . 21.05 % of cases were TORCH infections .36.8 % cases were related to meningoencephalitis . 42.1% of cases were related to hydrocephalus contributing to 8 % of total population.

Table 12: Number of cases attributing to various trauma

TRAUMA	NO OF CASES
SCALP HEMATOMA	1
SDH	2
CONTUSION	2
EDH	2
CEREBRAL HEMATOMA	1

Table 12 demonstrating different traumatic cases that were diagnosed through NSG total cases were 8 in number. In them, 1 was case of scalp hematoma case, 4 were cases of extra axial hematomas, 2 were cases of cerebral contusion and 1 was cerebral hematoma.

Table 13: Demonstrating doppler changes in cases of PVL and Gross hydrocephalus

	HIGH RI	LOW RI	Reversal/Absent of Diastolic flow
PVL 1	0	0	0
PVL 2	0	1	0
PVL 3	1	2	0
PVL 4	3	2	1
Hydrocephalus	4	0	1

Out of 24 cases of PVL, 9 cases were noted to have abnormal doppler findings. 5 cases of 8 IN HYDROCEPHALUS showing abnormal doppler findings. Neonates with abnormal Doppler findings were been seen to be at poor prognosis. The neonates demonstrating reversal of diastolic flow died in subsequent 2 days.

Table 14: Intracranial doppler findings and corresponding clinical picture

	Lethargic/Decreased tone	Active
HIGH/LOW RI	12	3
NORMAL RI	17	18

Out of 29 cases, abnormal doppler changes seen in 15 neonates. Out of them, 12 neonates are in lethargic state which accounts for 80% which is showing p value of 0.02 which is statistically significant.

Table 15: Follow-up cases of PVL and clinical picture

	PVL 1	PVL 2	PVL 3	PVL 4
Follow-up	5	6	2	4
Lost follow-up	2	0	2	1
Normal	5	4	0	0
Developmental delay	0	2	1	1
Cerebral palsy	0	0	1	3

Table 15 showing follow-up cases of PVL. PVL grade 1 showed no neurological developmental delay. 2 cases of PVL grade 2 showed developmental delay. Majority of cases i.e. 6 were related to PVL grade 3 and 4 accounting to 75% of cases.

Table 16: No. of Scan cases

	CT scan done	CT scan not done
CASES	38	62

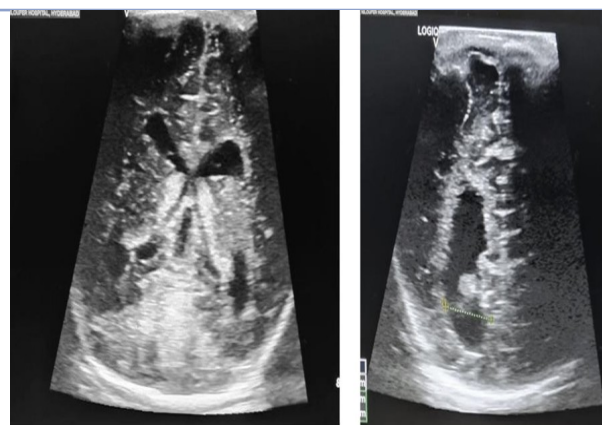


Fig1: showing colpocephaly with widely placed frontal horns and mild prominent third ventricle with poor visualization of corpus callosum – partial corpus callosal agenesis.

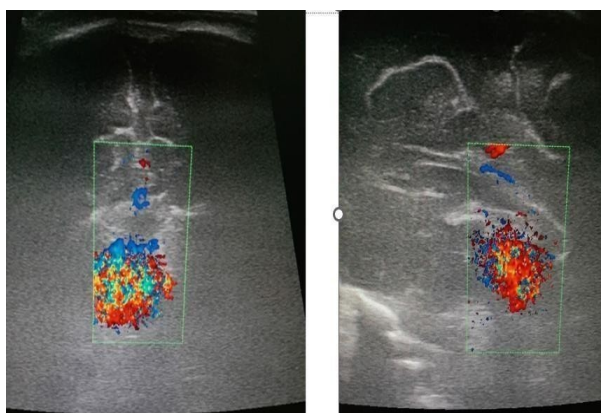


Fig 2: Showing colour aliasing at vein of galen (internal cerebral vein) region with hyperdense focus in that region on plain CT suggesting thrombosis

DISCUSSION

Neurosonography has now been routinely performed in premature infants. This has produced wealth of information about the central nervous system like GMH, PVL and ventriculomegaly. This information has included timing and evolution of these lesions and their eventual correlation with suspected brain injuries.[6] In our study of 100 neonates, most of the disease burden is by vascular causes (56%), then followed by infective (19%), congenital (14%), trauma (8%), metabolic (2%) and miscellaneous (2%). Study group comprise of babies who are neonates. Most of the neonates' Study group comprise of babies who are neonates. Most of the neonates' birth weight falls within the range of 1.5 to 2.4 kg. In our study, out of 100 neonates, 45 neonates are preterm [7]. Initial sonogram was performed within 7 days of birth followed by a repeat scan after 1,2,3,4 weeks. Initial neurosonogram showing periventricular echogenicity later turn out to be normal or with periventricular leukomalacia.

Table 17: Periventricular leukomalacia incidence

	Rajendra K. Diwakar	Present study
PVL	2	24
INFECTIVE (19%)	13	19
CONGENITAL (14%)	1	14
GMH	6	22
CEREBRAL EDEMA	6	1

When compared to our study with Rajendra K. Diwakar et al.,(63) our study has periventricular leukomalacia as highest incidence, where as in their study the most common incident cases are of hydrocephalus.

Table 18: Comparison with other study

Grade	Ramenghi La (2011) (%)	Nilgun Kosal (2002) (%)	Kadri (2006) (%)	Present study (%)
I	57.9	50.0	52.4	20.8%
II	19.3	17	30.95	20.8%
III	24.15	11	11.9	25%
IV	4.8	22	4.76	33.3%

In Ramenghi La et al study on grades of PVL, grade 1 PVL is most common accounting to 57.9% and grade 4 accounting for least In a study conducted by Nigun Kosal et al, 50 % cases are of Grade 1 PVL and 11 cases are of Grade 3 PVL.

In a study conducted by Kadri et al., most cases are of Grade1 PVL accounting to 52.4 % and PVL grade 4 is 4.7 % In our study , most case are of PVL Grade 4 accounting for 33.3% and least cases are of Grade 1 and 2 account in to 20.8 %of cases each Hypoxic ischemic encephalopathy (HIE) is a serious birth complication affecting full term infants. The majority of the underlying pathologic events of HIE are a result of impaired cerebral blood flow and oxygen delivery to the brain with resulting primary and secondary energy failure. By the age of 2 years, up to 60% of infants with HIE will die or have severe disabilities including mental retardation, epilepsy, and cerebral palsy (CP). In our study , 33.3 % of neonates developed either developmental delay /cerebral palsy. In an article by Seetha shankaran et al., events for HIE include placental abruption, uterine rupture, amniotic fluid embolism, tight nuchal cord, cord prolapse/avulsion, maternal haemorrhage, trauma or cardio respiratory arrest, severe and sustained fetal bradycardia, and prolonged labor. The majority of infants with encephalopathy do not have an

obvious cause for the encephalopathy. This is in correspondence of our study where 42 percent of neonates with HIE didn't show any risk factors .In our study , we followed De Vries classification of PVL[9].

According to De vries et al., transient echogenicity persist for at least a week and cystic changes takes 2 to 4 weeks to develop which was observed in our study . Therefore serial screening of neonates was done in our study. In our study , PVL GRADE 1 and Grade 2 are associated with less adversity than PVL Grade 3 & 4. Transcranial doppler can reflect the cerebral blood volume and perfusion status through resistive index and abnormal values according to literature are >0.8 and <0.5 and can be noticed in severe HIE and conditions which increases intracranial pressure. Any measure that decreases the vasospasm which decreases RI can eventually prevent the neonate having progression of ischemia and further new infarct According to study by Salas J. et al., Abnormalities in RI have been correlated with prognosis. An abnormal RI (equal to or less than 0.55), in the first 72 h after birth, has been found to be highly predictive of a poor prognosis with either death or severe disability which is in accordance of our study where low RI is found to be associated with a poor outcome. In our study , out of 15 people , 12 were showing abnormal RI whose clinical condition is not well . out of 12 , low ri is seen in 5 neonates who were when followed up , death occurred in 4 neonates so 80% of neonates with low RI were associated with poor prognosis . this is important because these neonates can be taken extra care of and protective hypothermia can be done in those neonates . In a study by Jing lui et al., The cerebral blood flow velocity decreased or increased markedly as resistive index (RI) decreased or increased markedly, which usually suggested the diagnosis of HIE, $RI < 0.50$ or $RI > 0.90$ usually occurred in severe patients, while $RI > 1.0$ would be associated with later brain death. This is in accordance with our study where increased RI associated with poor clinical outcome. in their study($12/16 = 75\%$) were accompanied with $RI < 0.50$ (four patients) or $RI > 0.90$ (eight patients) were

Grade	
I	transient periventricular densities, persisting for >7 days
II	transient periventricular densities, evolving into small localized fronto-parietal cysts
III	periventricular densities evolving into extensive periventricular cystic lesions
IV	densities extending into the deep white matter evolving into extensive cystic lesions

severely ill. In our study (15/12 =80%) were severely ill showing good agreement [10].

Alessandro parody et al., conducted a study over germinal matrix haemorrhage in preterm population [11-13]. In this study, they mentioned The overall incidence ranges between 20 and 25% among very low birth weight (VLBW) infants. In our study we have 9% of incidence of GMH in VLBW (<1.5KG). And we have 90% of incidence in low birth weight babies compared to normal weight babies (p value =0.02) which is statistically significant. In their study, the incidence of severe lesions (GMH Grade 3 & 4) ranges between 10 - 25 % in <28 weeks. In our study, 2 cases of GMH were observed under 28 weeks and they were GMH Grade 4. Severe GMH -IVH (grade 3 and 4) observed of 5% percentage incidence in their study beyond 28 weeks. In our study we observed severe GMH grade in 22 % of population beyond 28 weeks, after 32 weeks the study mentioned the cases are rare. This is in correspondence of our study, 3 out of 22 neonates are having severe grade of GMH [14].

CONCLUSION

In our study 100 pre-term babies with suspected brain injuries were studied with ultrasound through anterior fontanel and mastoid sutures. In case of PVL, most common clinical presentation is dull activity followed by RDS. The abnormalities found on neurosonogram in our study were germinal matrix haemorrhage, periventricular leukomalacia, extra axial hematoma, congenital venous malformations, meningitis, ventriculitis, metabolic diseases and other miscellaneous. The most common abnormality was periventricular leukomalacia. Even when PVL changes not seen in NSG in first week, serial followup NSG revealed PVL changes and doppler initially can show ongoing insult and doppler changes are related to hypocarbia which is seen in various scenarios. PVL grade 3 and grade 4 are associated with low intelligent quotient, developmental delay and cerebral palsy. In 40% of cases of PVL, appropriate insult could not be found out. High grade of GMH was detected in babies born less than 32 weeks of gestation, weighing less than 1500 gm. Most of the cases showing abnormalities on CT are detected on NSG initially.

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