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Correlation between cranial ultrasonography findings in neonates with perinatal asphyxia and severity of hypoxic ischemic encephalopathy

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Abstract

Introduction: Hypoxic Ischemic Encephalopathy (HIE) is a dreaded neurological complication associated with perinatal asphyxia of the newborn. The injury pattern in brain imaging has crucial implications in therapies and predicted neurodevelopmental outcomes. Concurrent use of cranial ultrasound and clinical staging systems are evolving to predict the prognosis.

Objective: To study the cranial ultrasonographic finding in neonates with perinatal asphyxia and its clinical correlation with severity of hypoxic ischemic encephalopathy.

Materials and Methods: It is an observational cross sectional study of 60 term neonates with perinatal asphyxia admitted in the Neonatal ICU of Krishna Hospital, Karad. Neurosonography (NSG) was done within3-5 days of life.

Results: It is observed that, higher the grade of HIE, more was the complications seen in NSG findings in the neonates with perinatal asphyxia. Mortality rate was also high in those neonates that had a higher grade of HIE and NSG findings.

Conclusion: Good correlation between neurosonography findings in neonates with perinatal asphyxia and severity of hypoxic ischemic encephalopathy was found. Routine NSG in neonates with moderate to severe perinatal asphyxia done between 3-5 days of life will help in early diagnosis, assessment of severity of hypoxic ischemic encephalopathy and management of the affected neonates in a better way.

Combined use of Sarnat staging and neurosonography findings has got good predictive value for severity of disease and outcome in terms of treatment and mortality.

Keywords: HIE, Perinatal Asphyxia, Neurosonography.

Introduction

Hypoxia-ischemia-induced brain injury is a prevalent cause of short-term mortality and long-term neurological morbidity in infants and children.[1] Statistics indicate that approximately 20 out of every 1000 live births in term newborns are affected by systemic asphyxia. Among those newborns experiencing asphyxia and subsequent brain damage due to hypoxia-ischemia, 5 to 20 percent succumb to death early in life. At birth, neonates with hypoxic-ischemic encephalopathy (HIE) often present with a low APGAR score characterized by bradycardia, poor respiratory effort, hypotonia, decreased alertness, a weak or absent cry, and abnormal skin coloration, along with metabolic acidosis in the umbilical cord blood.[2]

Imaging is crucial for diagnosing and managing hypoxic-ischemic encephalopathy (HIE). In acute scenarios, it guides appropriate treatment strategies and offers valuable insights into the long-term prognosis of affected infants.[3] Both brain sonography and MRI are commonly employed to monitor intracranial changes in newborns with hypoxic-ischemic injuries.[4] Of these, MRI is particularly effective in identifying hypoxic-ischemic lesions in the brain and has consequently been increasingly utilized for evaluating such conditions.[5]

Hypoxic-Ischemic Encephalopathy (HIE) in a full-term infant is a clinically defined syndrome characterized by disturbed neurological function in the first days of life. It manifests through difficulty in initiating and maintaining respiration, decreased muscle tone and reflexes, reduced consciousness, and often seizures. The severity of asphyxial insult in infants over 36 weeks of gestation is typically assessed using the Sarnat clinical staging system. Infants who survive severe HIE often face significant long-term neurological disabilities such as cerebral palsy, intellectual disabilities, and epilepsy.As a result, the sequelae of hypoxic-ischemic brain injury demand considerable healthcare resources. Accurate assessment of the severity of HIE is crucial for providing appropriate parental counseling and initiating early stimulation therapies to promote better developmental outcomes for the affected infant.[6]

It is already known that MRI is the preferred imaging modality for the selective evaluation of newborns with hypoxic-ischemic encephalopathy.However, due to practical limitations, such as the inability to transport critically ill neonates for MRI, cranial ultrasonography remains the initial diagnostic tool for imaging and screening in hypoxic conditions. This is particularly crucial for newborns severely affected by encephalopathy, where immediate and accessible imaging is necessary to obtain vital information regarding the progression of the condition and the location of the lesion. [7]Although certain lesions may be exclusively detectable by MRI, cranial USG still plays a complementary role in the comprehensive evaluation of newborns with encephalopathy.[8]

The most common brain sonography findings in newborns with hypoxic-ischemic damage include brain edema, increased echogenicity of the subcortical white matter and brain tissue, and sometimes intra ventricular hemorrhage, although the latter is less common. Brain sonography shows compatible findings in neonates with HIE and is essential for early prognosis and guiding treatment decisions. Periodic evaluation with imaging and EEG is crucial for timely intervention and predicting outcomes, with sonography playing a key role in early

HIE prognosis and decision-making for neuroprotective therapy.[9]

By elucidating the link between cranial USG imaging and the clinical presentation of HIE, this study intends to contribute to improved diagnostic strategies, enhance the prognostication of neonatal outcomes, and ultimately support better clinical care for newborns experiencing perinatal asphyxia.

Material and Methods

A prospective cross sectional study involving 60neonates was conducted over a 18-month period from June 2022 to November 2023 at Department of Pediatrics, Krishna Vishwa Vidyapeeth, Karad. Ethical clearance from the institute's Ethical Committee was obtained before commencing the study.

Inclusion Criteria: All term neonates with perinatal asphyxia admitted in NICU was included in this study.

- Failure to initiate and sustain breathing at birth (WHO).
- Moderate asphyxia as slow gasping breathing or an APGAR score of 4-6 at 1 minute of life and severe asphyxia as no breathing or a score of 0-3 at one minute of life (NNPD).
- History of difficult labour with poor or no cry immediately after birth (in cases of babies referred to out born NICU where APGAR score was not available)

Exclusion Criteria

- Injury to brain caused by metabolic derangement, cerebral dysgenesis, infections, hyperbilirubinemia.
- Neonates with life threatening congenital malformations.
- Birth trauma to the head due to assisted delivery or precipitated labor or any other condition causing injury to the head.
- Neonates with Vit K deficiency, thrombocytopenia or thrombotic events (example : sinovenous thrombosis, etc).

Informed consent was obtained from parents after explaining the procedure to them.All data including gender, birth weight, mode of delivery, maternal risk factors, presence of MSL,APGAR score at 1 minute and 5 minutes, neurological signs (if any),days of hospitalization, status of the baby at discharge was recorded.

The neonates were graded into HIE stages using Sarnat and Sarnat staging. NSG was done between days 3-5 of life. Scans were performed through the anterior fontanelle in both sagittal and coronal planes.

NSG Grading was done according to Papile's classification:

Grade 1 : Restricted to subependymal region

Grade 2 : IVH with no ventricular dilation

Grade 3 : IVH with ventricular dilation

Grade 4 : IVH with parenchymal extension

IVH= Intra Ventricular Hemorrhage

Statistical Analysis

Statistical analysis was done with Statistical Package for Social Sciences (IBM SPSS Statistic for window, version 21.0. Armonk, NY: IBM Corp.) at 95% CI and 80% power to the study. Kolmogorov-Smirnov and Shapiro Wilk test was done to check for normal distribution of the data. Descriptive statistics was performed in terms of Mean, Std Deviation. Chi Square test was applied to check the statistical significance of the observations. Statistical significance was calculated at $p<0.05$ and $p<0.001$ was considered highly significant.

Results

Table 1: Average gestational age of the study subjects (n=60)

	N	Minimum	Maximum	Mean	Std. Deviation
Age	60	37.00	41.10	38.6850	.99963

Mean gestational age of the study subjects was 38.68±0.99 weeks.

Table 2: APGAR score at 1 and 5 mins of life of the study subjects (n=60)

APGAR score (N=60)	Minimum	Maximum	Mean	Std. Deviation
At 1 minute	1	3	2.56	0.738
At 5 minutes	3	5	4.66	0.723

Mean APGAR score at 1 minute is 2.56±0.738 and at 5 minutes is 4.66±0.723 respectively.

Table 3: Gender wise distribution of study subjects (n=60)

Gender	No.	%
Female	25	41.7
Male	35	58.3

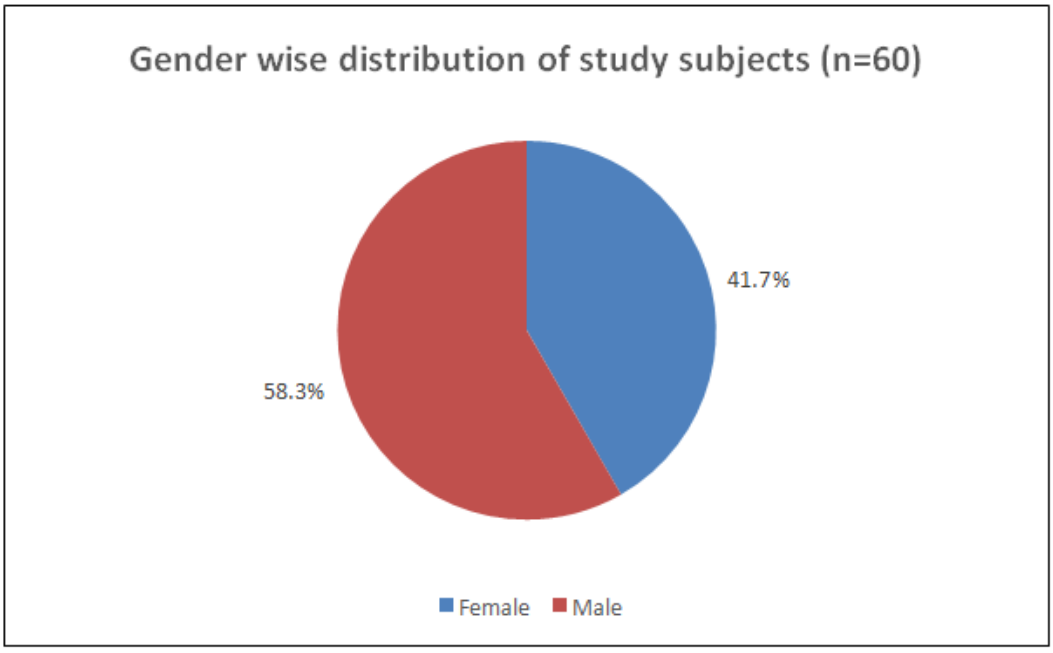

Figure 1 : Gender wise distribution of study subjects(n=60)

Table 4: Birth weight of study subjects (n=60)

Birth weight	No.	%
<2.5 kgs	5	8.3
≥2.5 kgs	55	91.7

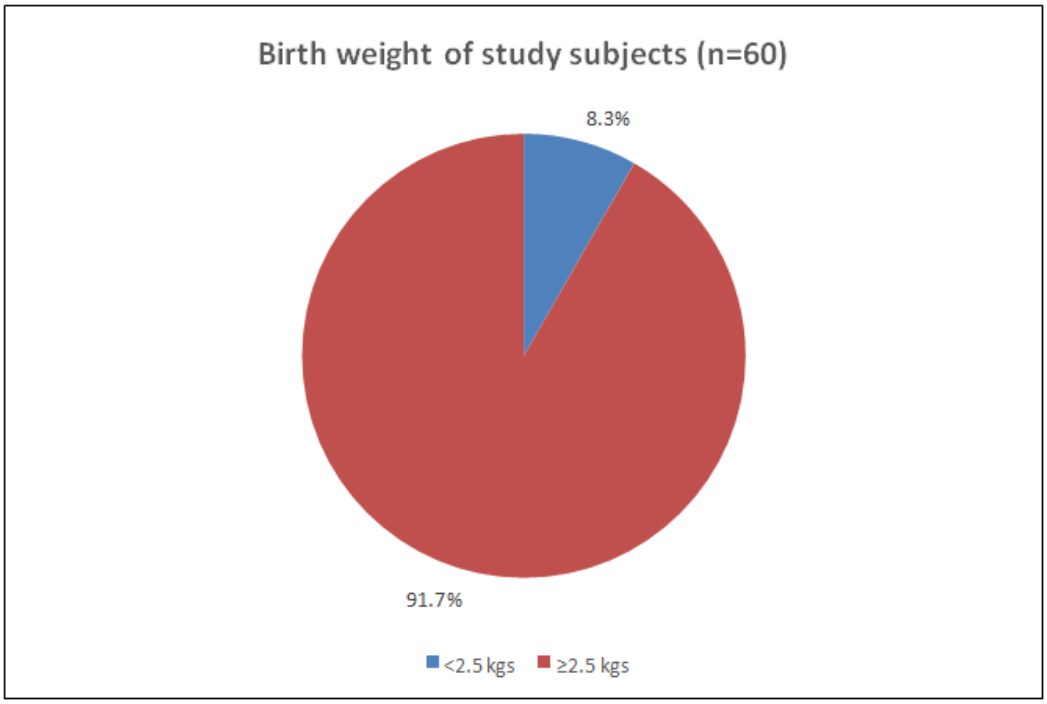

Figure 2: Birth weight of study subjects (n=60)

Mean birth weight of the neonates was 2.86+0.38kgs.

Table 5: Mode of delivery in study subjects (n=60)

Mode of delivery	No.	%
LSCS (Lower Segment Cesarean Section)	21	35.0
NVD (Normal Vaginal Delivery)	39	65.0

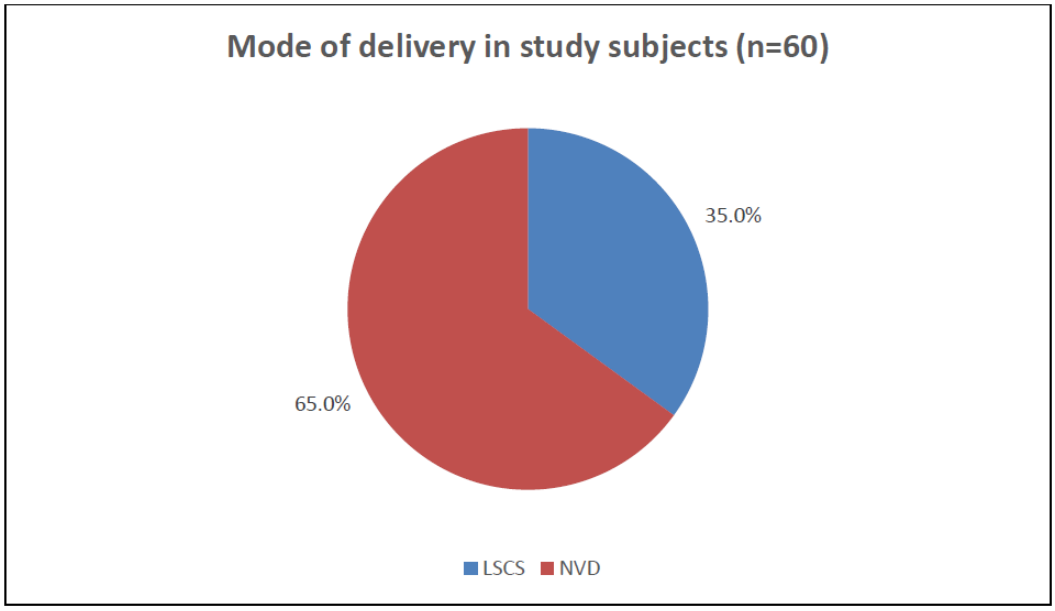

Figure 3: Mode of delivery in study subjects (n=60)

Table 6: Maternal risk factors in study subjects (n=60)

Maternal risk factors	No.	%
None	43	71.6
Eclampsia	2	3.3
Severe Pre Eclampsia	3	5.0
Maternal anaemia	3	5.0
GDM	4	6.6
Maternal hypotension	1	1.6
Polyhydrominos	1	1.6
Abruptio Placenta	3	5.0

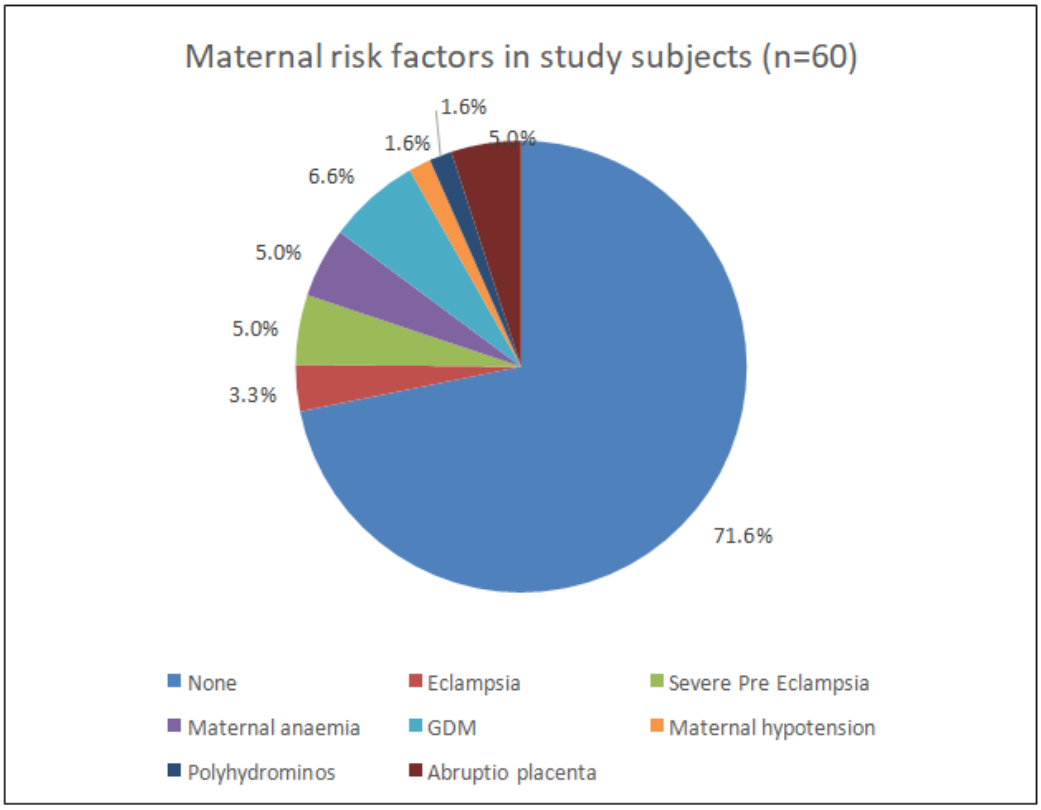

Figure 4: Maternal risk factors in study subjects (n=60)

Table 7: Meconium Stained Liquor (MSL) in study subjects (n=60)

MSL	No.	%
No	29	48.3
Yes	31	51.6

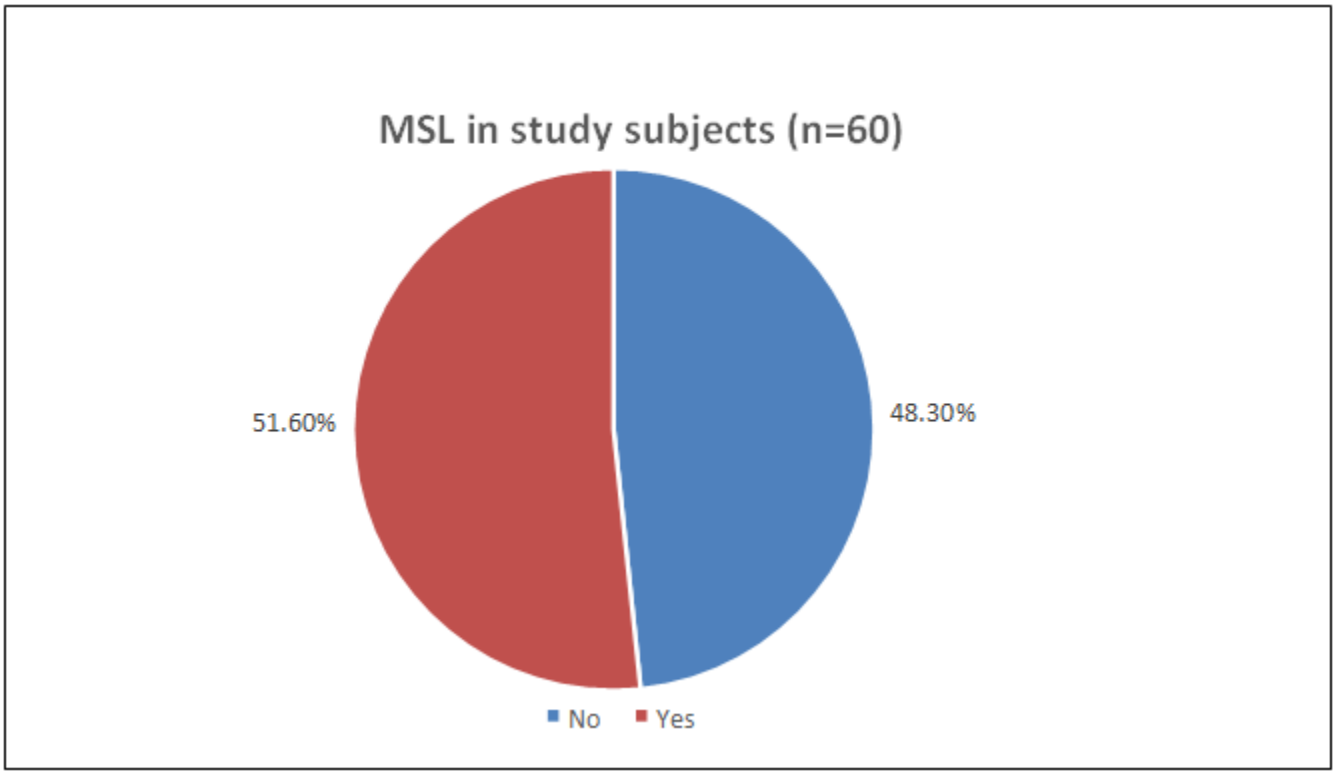

Figure 5: Meconium Stained Liquor (MSL) in study subjects (n=60)

Table 8: HIE grade in study subjects (n=60)

HIE grade	No.	%
1	28	46.7
2	17	28.3
3	15	25.0

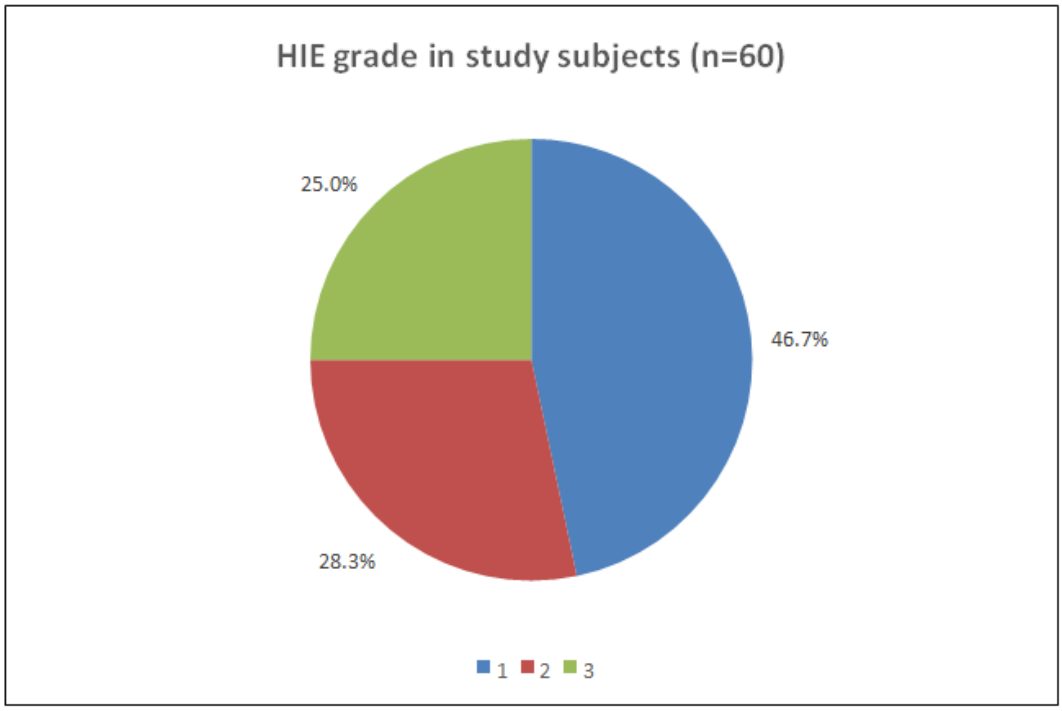

Figure 6: HIE grade in study subjects (n=60)

Table 9: NSG finding in study subjects (n=60)

NSG finding	No.	%
Normal	32	53.3
Cerebral oedema	17	28.3
Grade 2 IVH	4	6.7
Grade 3 IVH	6	10.0
Grade 4 IVH	1	1.7

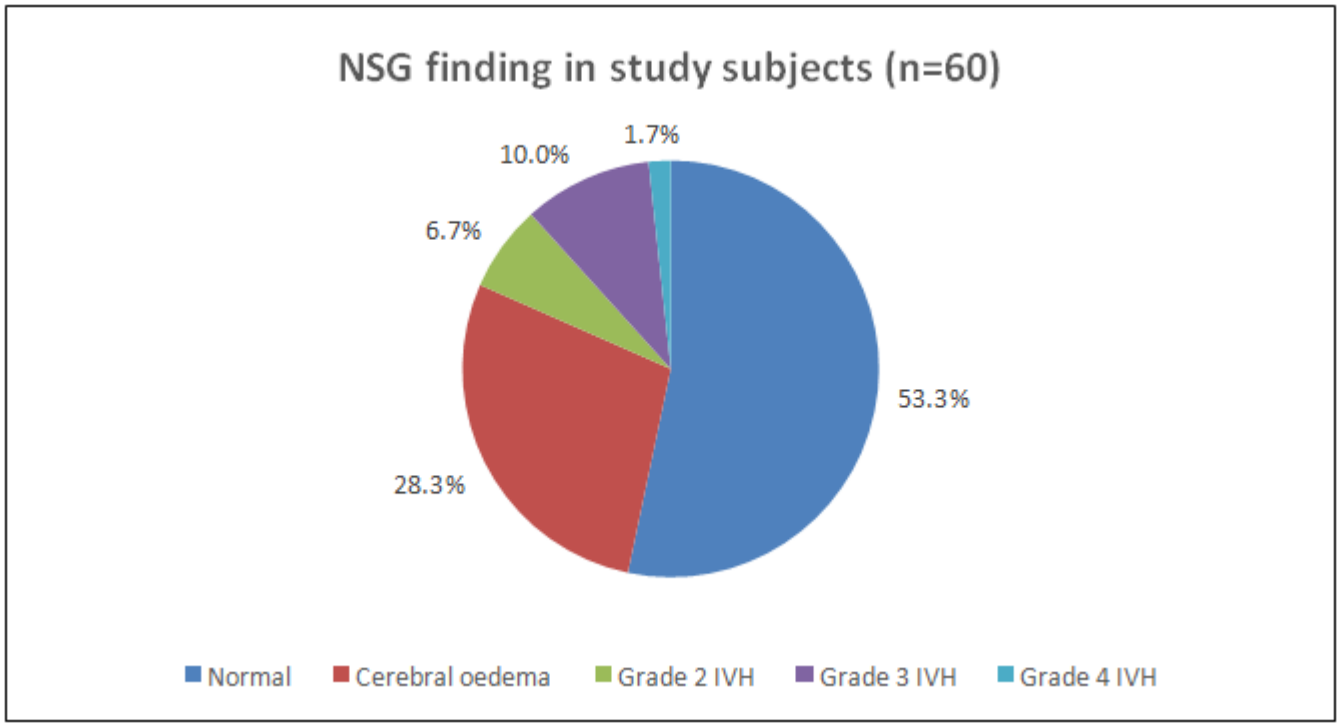

Figure 7: NSG finding in study subjects (n=60)

Table 10 a): Association of APGAR score at 1 minute with NSG findings (n=60)

NSG findings	AP	GAR score at 1 min	
	1	2	3
Normal	0	3(5%)	29(48.3%)
Cerebral oedema	3(5%)	5(8.3%)	9(15%)
Grade 2 IVH	2(3.3%)	0	2(3.3%)
Grade 3 IVH	3(5%)	0	3(5%)
Grade 4 IVH	1(1.6%)	0	0

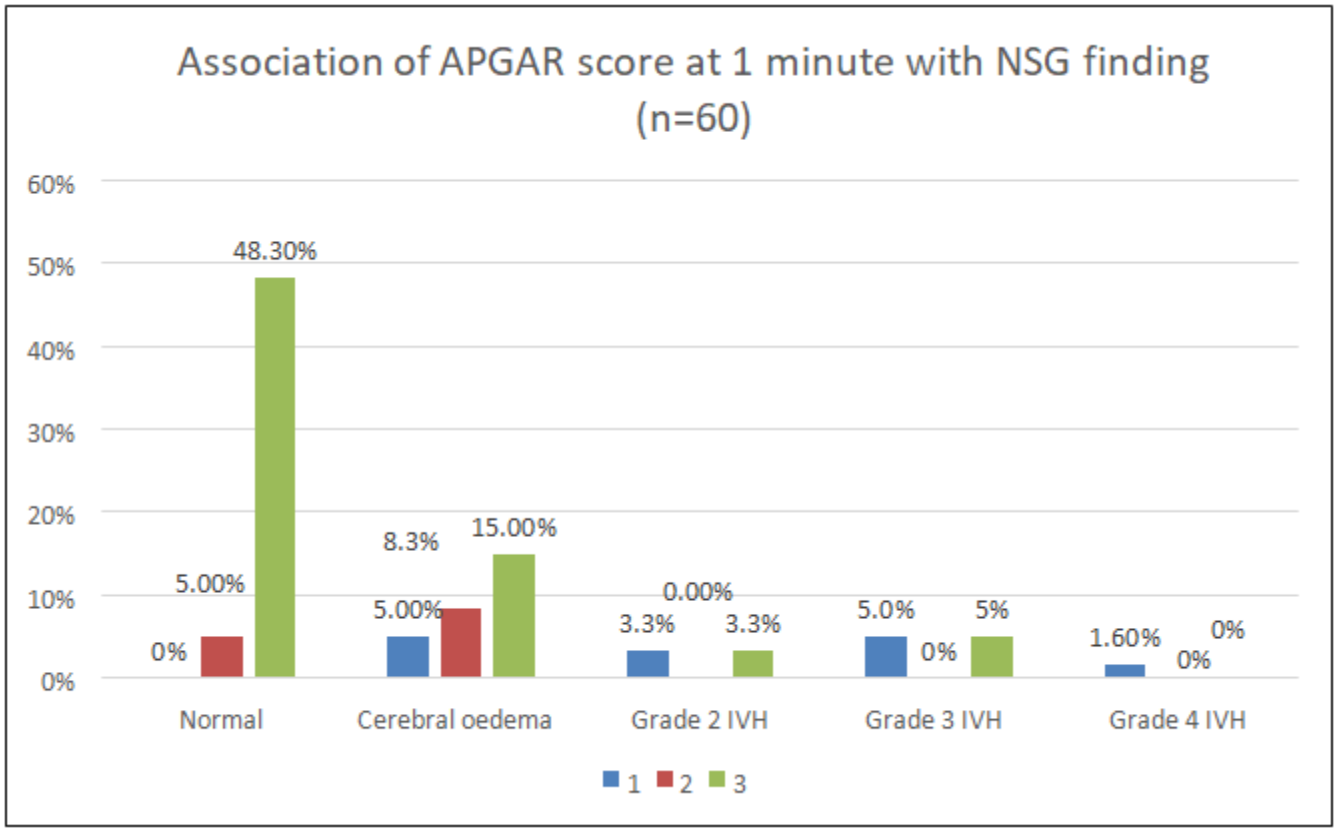

Figure 8a): Association of APGAR score at 1 minute with NSG findings (n=60)

Table 10b): Association of APGAR score at 5 minutes with NSG findings (n=60)

NSG findings	APGAR score at 5 min		
	3	4	5
Normal	0	0	32(53.3%)
Cerebral oedema	3(5%)	2(3.3%)	12(20%)
Grade 2 IVH	2(3.3%)	0	2(3.3%)
Grade 3 IVH	3(5%)	0	3(5%)
Grade 4 IVH	1(1.6%)	0	0

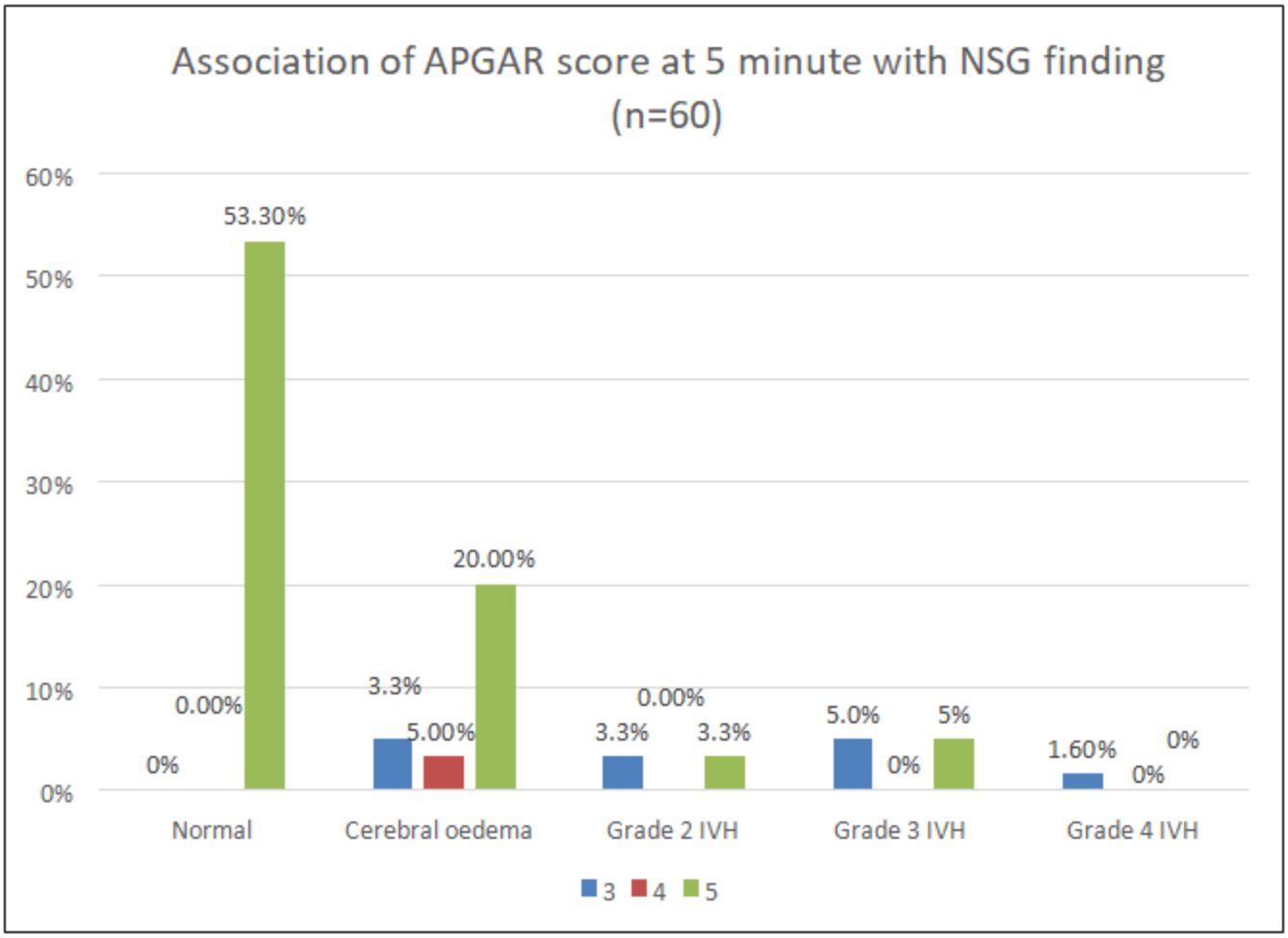

Figure 8b): Association of APGAR score at 5 minutes with NSG findings (n=60)

Table 11: Age in days at which NSG was done in study subjects (n=60)

Age in days at which NSG was done	No.	%
3	30	50.0
4	25	41.7
5	5	8.3

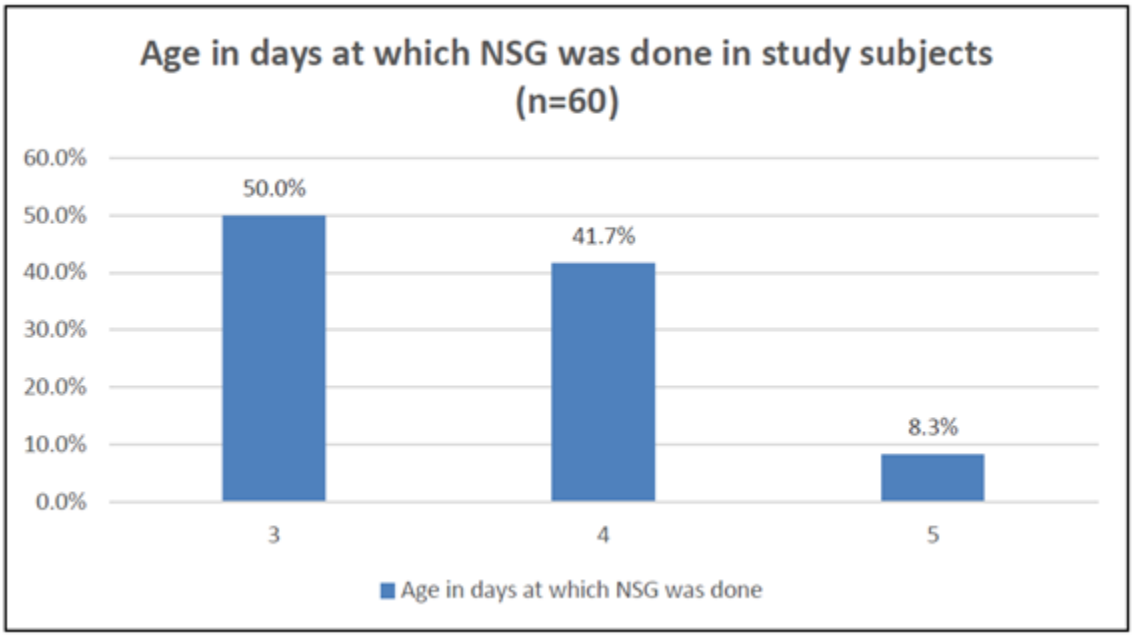

Figure 9: Age in days at which NSG was done in study subjects (n=60)

Table 12: Neurological signs associated with grades of HIE(n=60)

CNS SYMPTOMS	
1)Tone	Hypertonia : 1 Hypotonia : 8
2)Difficulty feeding	6
3)Lethargy	7
4)Irritability	2
5)Poor reflexes(suck,swallow,moro)	7
6)Abnormal breathing pattern	6
7)Seizures	10
8)Coma	6

Neurological signs	Stage 1 HIE	Stage 2 HIE	Stage 3 HIE
Present	1(1.6%)	10(16.6%)	11(18.3%)
Absent	27(45%)	7(11.6%)	4(6.6%)

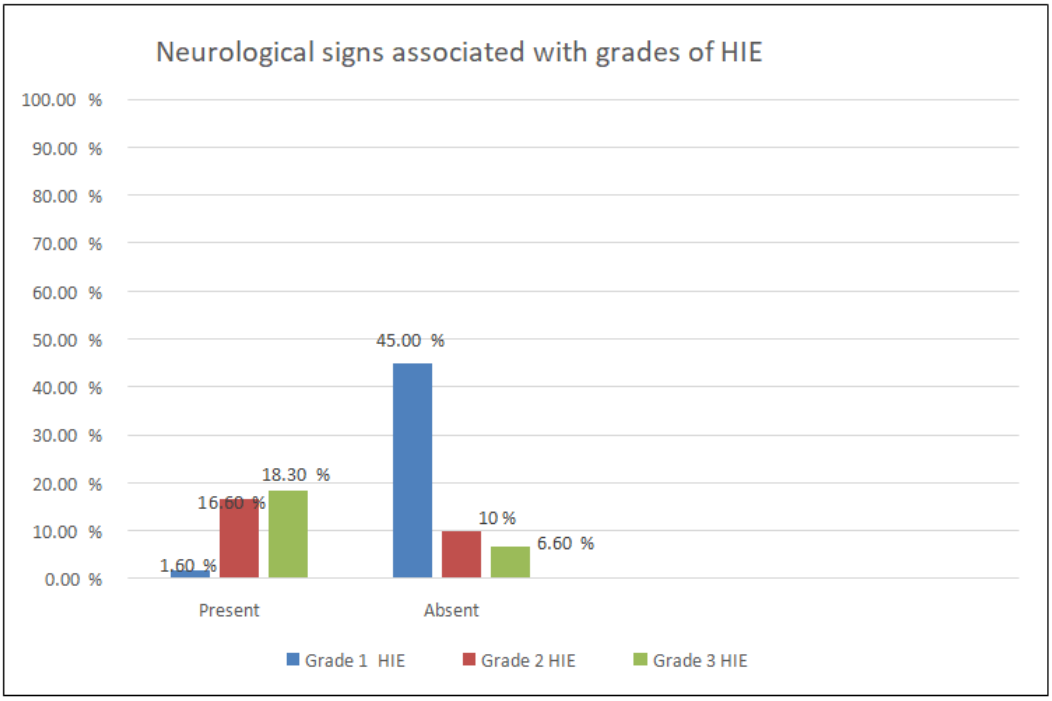

Figure 10: Neurological signs associated with grades of HIE (n=60)

Table 13: Status of baby at discharge in study subjects (n=60)

Status of baby at discharge	No.	%
Death	14	23.3
Survived	46	76.7

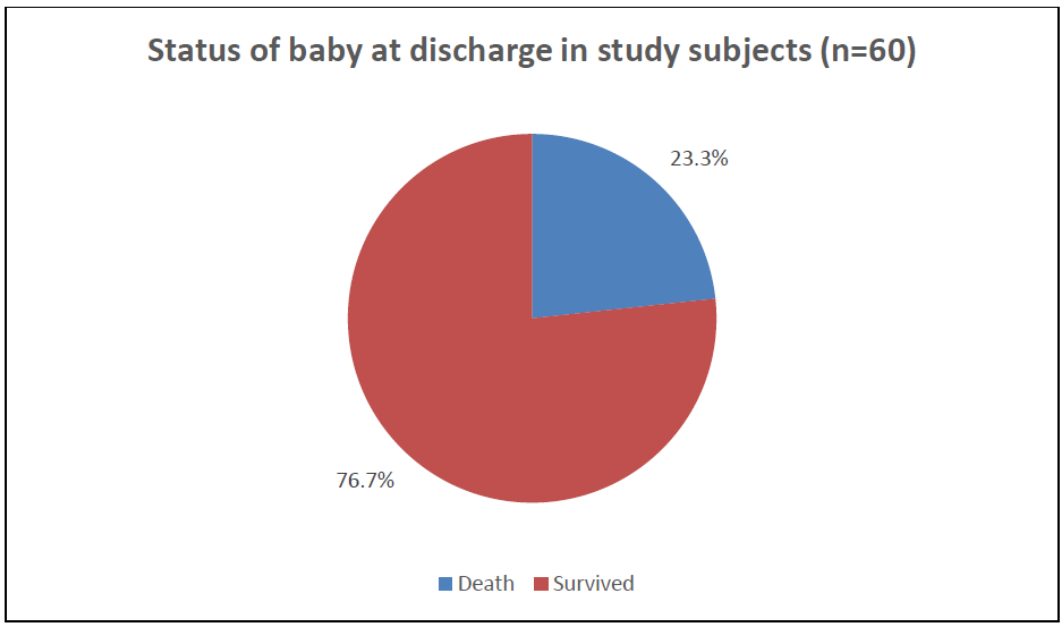

Figure 11: Status of baby at discharge in study subjects (n=60)

Table 14: Association of HIE Stage with NSG findings

	Normal	Cerebral oedema	Grade 2 IVH	Grade 3 IVH	Grade 4 IVH	
Stage 1	26 (92.1%)	2 (7.9%)	0	0	0	P value <0.001
Stage 2	6 (35.3%)	10 (58.8%)	0	1 (5.9%)	0	
Stage 3	0	5 (33.3%)	4 (26.7%)	5 (33.3%)	1 (6.7%)	

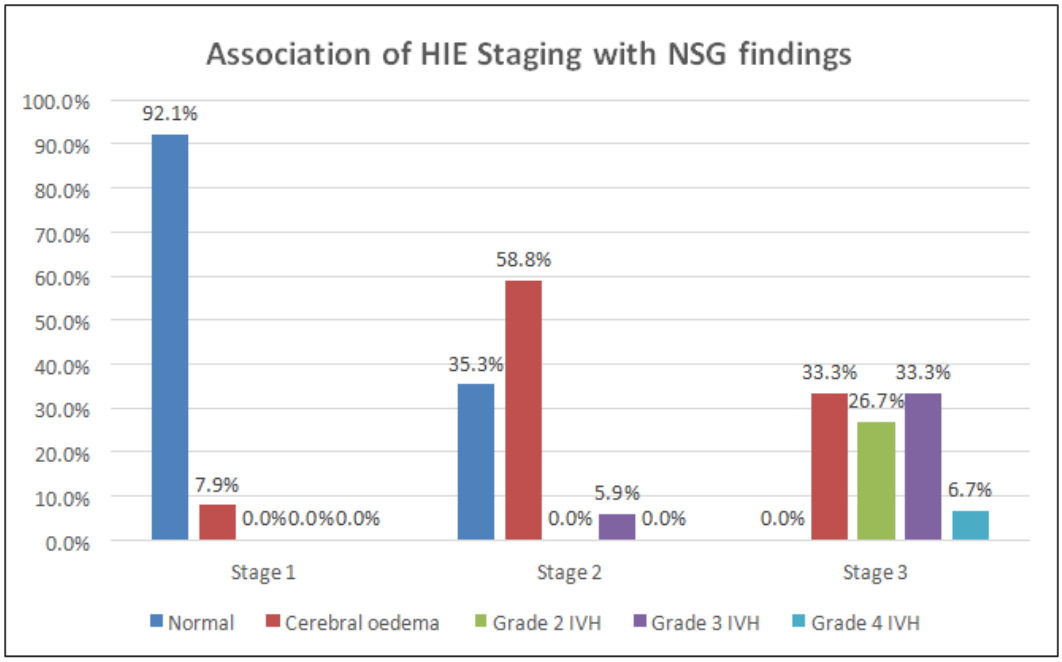

Figure 12: Association of HIE staging with NSG findings

Table 15: Association of HIE staging with mortality in study subjects (n=60)

HIE grading	Death	Survived	
1	0	28 (100%)	P value <0.001
2	2 (11.8%)	15 (88.2%)	
3	12 (80%)	3(20%)	

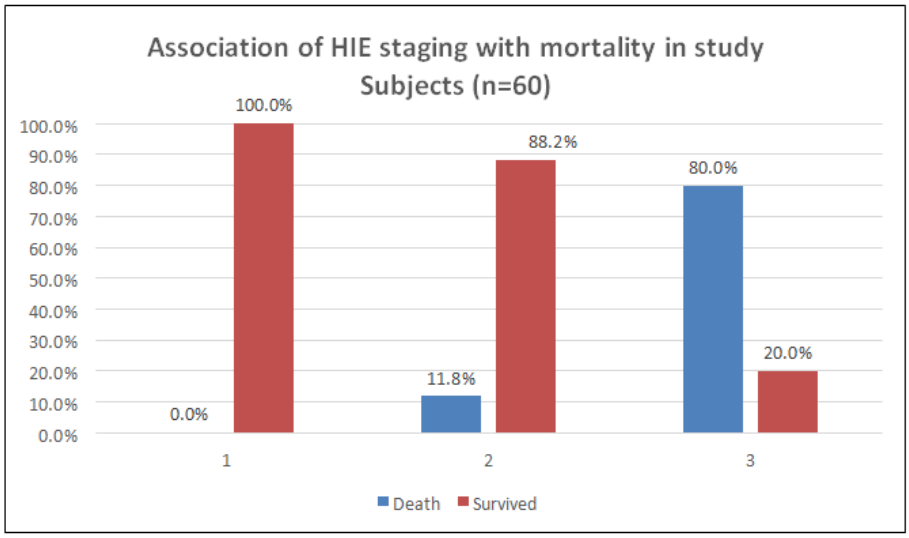

Figure 13: Association of HIE staging with mortality in study subjects (n=60)

Table 16: Association of NSG finding with mortality in study subjects (n=60)

NSG finding	Death	Survived	
Normal	0	32 (100%)	P value <0.001
Cerebral oedema	4 (23.5%)	13 (76.5%)	
Grade 2 IVH	4 (100%)	0	
Grade 3 IVH	5 (83.3%)	1 (16.7%)	
Grade 4 IVH	1 (100%)	0	

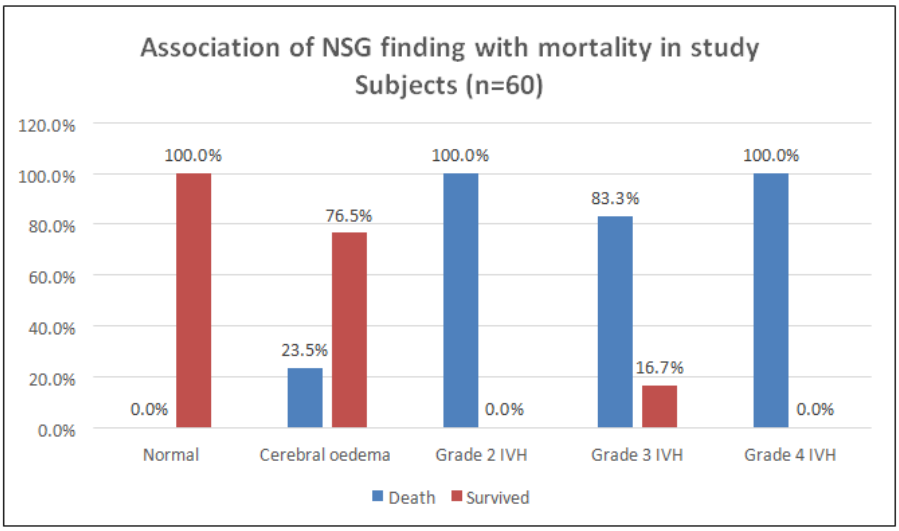

Figure 18: Association of NSG finding with mortality in study subjects (n=60)

Discussion

In this study, mean gestational age of the study subjects was 38.68±0.99 weeks. In the study conducted by Ganesan et al[10] ,mean gestational age was 39.24 ±0.94 weeks and the study conducted by Mangaraj et al [11], mean gestational age was 36.44±0.58 weeks respectively. The present study correlates well with Ganesan et al.[10]

Present study showed male dominance having 35(58.3%) males and 25(41.7%) females. In the study conducted by Ganesan et al [10],males were 39(52%) and females were 36(48%) which were similar to present study findings. In the study conducted by Cally J Tann et al[12] in sub-Saharan Africa, males were 136(64.8%) and females were 74(35.2%).

Mean birth weight in study population in present study was ≥ 2.5kgs in 55(91.7%) neonates whereas rest 5(8.3%) neonates had birth weight below 2.5kg. In Cally J Tann et al[12] study,198 (94.2%) neonates and 12(5.7%) neonates had birth weights ≥ 2.5kgs and <2.5 kgs respectively.

The Mode of delivery was LSCS (Lower Segment Cesarean Section) and NVD (Normal Vaginal Delivery),out of which, 21 (35%) neonates were delivered by LSCS and delivery by NVD was in 39(65%) neonates. In the study conducted by Ganesan et al [10]neonates delivered by LSCS were 29(38.7%) and by NVD were 46(61.3%) respectively.

Maternal risk factors were evaluated and showed, eclampsia was observed in only 2 (3.3%) mothers and severe preeclampsia was observed in 3(5%) mothers.3(5%),4(6.6%),1(1.6%),1(1.6%)3(5%) mothers had maternal anaemia, GDM, maternal hypotension, polyhydrominos and abruptio placenta respectively. There were 43(91.7%) mothers in which there was no risk factors. Meconium stained liquor was present in most of the neonates ,that is 31(51.6%)neonates. No MSL was seen in the remaining 29 (48.30%) neonates.

The present study showed stage 1 HIE was in 28(46.7%) neonates , stage 2 in 17(28.3%) neonates and stage 3 in 15(25%) neonates respectively. In the study conducted by Nath B et al [13] Stage 1 HIE was seen in 39(39%) neonates, stage 2 HIE in 49(49%) neonates and stage 3 HIE in 12(12%) neonates respectively. In the study conducted by Mangaraj et al [11] Stage 1 HIE was seen in 7(14%) neonates, stage 2 HIE in 26(52%) neonates and stage 3 in 17 (34%) neonates respectively. It is observed that present study had similar findings to study of Nath et al [75] and that majority of neonates had stage 1 HIE and stage 2 HIE.

In this study normal NSG findings were seen in 53.3% neonates, cerebral oedema was seen in 28.3% neonates and Grade 2,grade 3 and grade 4 IVH was seen in 6.7%,10% and 1.7% neonates respectively. According to Nath B et al [13], NSG showed normal study in 32% and cerebral oedema in 14% neonates and varying degrees of intra-cranial hemorrhage in the rest 46 % neonates. Zhu L et al [14] had reported 10.4 % of cerebral oedema and 18.5% of intra-cranial hemorrhage. Prithviraj et al[15] had found 46% normal study and 38 % of cerebral oedema in NSG of neonates with HIE. A study by Anand N K et al [16] in New Delhi revealed 86% normal scans and 14% cerebral oedema.

Mean values of APGAR score at 1 and 5 minutes was 2.56±0.738 and 4.66±0.723 respectively whereas in the study conducted by Cally J Tann et al [12] in sub-Saharan Africa at 1 and 5 minutes was 3±0.688 and 7±0.123 respectively.

In the study, for an APGAR score (at 1 minute) of 1, there was no normal NSG findings. 3(5%), 2(3.3%),

3(5%) and 1(1.6%) neonates had cerebral oedema, grade 2 IVH, grade 3 IVH and grade 4 IVH respectively. For an APGAR score of 2,3 (5%) neonates had normal NSG findings,5(8.3%) neonates had cerebral oedema. No IVH was documented in these neonates. For an APGAR score of 3,24 (48.3%) neonates had normal NSG findings,9(15%),2(3.3%),3(5%) and 6(10%) neonates had cerebral oedema, grade 2 IVH and grade 3 IVH respectively. Here, it is observed that neonates with normal NSG findings predominantly had higher APGAR scores at 1 minute of life. Neonates with cerebral oedema were more evenly distributed across the APGAR scores. Higher grades of IVH (grade 2 to 4) were associated with lower scores with very few having scores of 3.The severity of IVH appears to correlate with lower initial APGAR scores.

For an APGAR score (at 5 minutes) of 3,there was no normal NSG findings.3(5%),2(3.3%),3(5%) and 1(1.6%) neonate had cerebral oedema, grade 2 IVH, grade 3 IVH and grade 4 IVH respectively. For an APGAR score of 4, no neonates had normal NSG findings,2(3.3%) neonates had cerebral oedema. No IVH was documented in these patients. For an APGAR score of 5,32 (53.3%) neonates had normal NSG findings,12(20%) ,2(3.3%) and 3(5%) neonates had cerebral oedema, grade 2 IVH, grade 3 IVH and no grade IVH. Here, it is observed that neonates with normal NSG findings predominantly had the highest APGAR score of 5 at 5 minutes. Neonates with cerebral oedema were more evenly distributed across the APGAR scores, with a few achieving a score of 5.The severity of IVH correlates with lower APGAR score at 5 minutes. In conclusion it was seen that those neonates that had a lower APGAR score at 1 and 5 minutes had a higher grade of intracranial bleed.

In the present study, it was observed that in 30(50%) neonates, NSG was done at the age of 3 days**.** In 25(41.7%) neonates, NSG was done at the age of 4 days and in 5(8.3%) neonates, it was done at the age of 5 days. Hence in majority of neonates, NSG was done before 5 days. At discharge it was observed that 46 (76.7%) of the neonates survived which constituted the majority of the study group and 14 (23.3%) of neonates died.

In this study, the neurological signs observed in the neonates correlated with HIE grades. There was no neurological signs seen in 27(45%) neonates with stage 1 HIE, no neurological signs seen in 7(11.6%) neonates with stage 2 HIE and no neurological signs seen in 4(6.6%) neonates with stage 3 HIE. Neurological signs (hyper/hypotonia, difficulty feeding, lethargy, irritability, poor reflexes, abnormal breathing pattern, seizures and coma) was seen in 1(1.6%) neonate with stage 1 HIE, 10(16.6%) neonates with stage 2 HIE and 11(18.3%) neonates with stage 3 HIE. Thus, it can be observed that neurological signs are more common in the moderate (stage 2 HIE) and severe (stage 3 HIE) grades of HIE.

In this study, neonates with Stage 1 HIE, 92.1% were normal,7.9% neonates with Stage 1 HIE had cerebral oedema. In neonates with Stage 2 HIE, 35.3% neonates were normal ,cerebral oedema was present in 58.8% neonates , and 5.9% neonate had grade 3 IVH. In neonates with Stage 3 HIE,33.3% neonates had cerebral oedema, Grade 2 IVH was seen in 26.7% neonates , Grade 3 IVH was seen in 33.3% neonates and 6.7% neonates had Grade 4 IVH ,all of this being statistically significant(p value <0.001). Ganesan D et al [10] reported

cranial ultrasound findings of HIE stage I, 89.7% had normal cranial ultrasound findings, and 10.3% had abnormal findings, among which 2.6% had Cerebral oedema, 5.1% had hydrocephalus and 2.6% had intra ventricular hemorrhage which was similar to the study conducted by Tarana Yasmin et al[17] which showed 82% of babies with stage 1 HIE had normal NSG findings. A study conducted by Bijay et al[18] in Odisha concluded that 45% of stage 2 HIE and 30.8% of stage 3 HIE neonates had abnormal cranial USG findings. A study by Nagraj et al [19] from India showed 24.6% neonates who had abnormal APGAR scores, 50% with moderate asphyxia, and 80% with severe asphyxia had abnormal findings on cranial ultrasound. Among babies with stage 2 HIE,15.4% had cerebral oedema, 15.4% had hyper intensities and 11.5% had intra cerebral hemorrhage. Cranial ultrasound findings in stage 3 HIE , showed 40% neonates had cerebral oedema and 30% had hyperintensities in deep white matter.

In the current study, all neonates with stage 1 HIE survived. It was observed that in neonates with stage 2 HIE, 2(11.8%) neonates died and 15(88.2%) neonates survived. In neonates that had stage 3 HIE,12 (80%) died and 3(20%) neonates survived.This was statistically significant($p<0.001$) Thus in terms of mortality

Stage 1 HIE : associated with no mortality indicating that mild HIE has a good prognosis

Stage 2 HIE : showed some mortality(11.8%) but most neonates (88.2%) survived indicating that moderate HIE has a lower survival rate as compared to stage 1

Stage 3 HIE : had a high mortality rate(80%) and only a small fraction (20%) survived indicating the critical nature of severe HIE

Thus, in this study, lower HIE stages (stage 1) were associated with better outcomes (100% survival rate, 0% mortality).As the HIE stage increases, the mortality rate increases significantly (80% in stage 3), and the survival rate decreases. This data highlights the critical importance of early detection and intervention to improve outcomes in subjects with higher HIE stage.

All neonates with normal NSG findings survived. It was observed that in neonates with cerebral oedema, 4(23.5%) neonates died and 13(76.5%) neonates survived. All neonates with Grade 2 IVH died. Out of total neonates with Grade 3 IVH , 5(83.3%) died and 1(16.7%) survived. Neonate with Grade 4 IVH died. This was found to be statistically highly significant.($p<0.001$). In a similar study conducted by Khan RH et al [20] in 2014 in Bangladesh ,out of 178 neonates, 28 % had developed intra-ventricular hemorrhage and all 7 cases of Grade 4 intra-ventricular hemorrhage had died.

In present study, higher grades of IVH(grade 2,3,4) were associated with very high mortality rates and poor outcomes, highlighting the severity of these conditions. Cerebral oedema also showed a notable impact on mortality but had a better prognosis compared to higher grades of IVH. These findings emphasize the importance of NSG findings in predicting outcomes and guiding clinical decisions in study subjects.

Benefits of USG are that it can be performed serially, at the bedside, and in the sickest of neonates.[21] It allows the early detection of congenital or other causes of NE(Neonatal Encephalopathy) and informs the timing of hypoxic injury; as a consequence, it continues to be recommended as an important adjunct to MRI scanning for encephalopathic infants with NE.[22] Previous studies have found abnormal ultrasonography findings to be

predictive of outcome , with good correlation between MRI and ultrasonography findings among infants with HIE in some, but not all studies. USG used in this study successfully identified encephalopathic neonates with major abnormalities on ultrasound, and differentiated between abnormalities suggestive of significant brain injury and other changes frequently seen among well term infants.[23]Mejaski et al [24] reported in a follow up of 10 infants with grade-3 HIE that 100% developed severe cerebral atrophy on follow up by ultrasound.

Defining the nature and timing of newborn brain injury in encephalopathy is key to understanding the most appropriate prevention and intervention strategies for encephalopathy. Since changes seen on ultrasound are recognized to take some time to develop after the original brain insult , this may imply that the injury pathway begins several hours before delivery, or a very severe acute exposure to hypoxic injury. It is unclear whether severe injury may appear on ultrasound more rapidly than moderate injury; experimental studies show that secondary energy failure develops more rapidly in severe hypoxic injury than moderate hypoxic injury . The accelerated cell death cascade may be reflected in earlier evidence of injury on ultrasonography. As in South India, a high prevalence of white matter injury was seen among cases on early imaging. This may be associated with perinatal infection and inflammation or may reflect other exposures, including a less acute and more chronic hypoxic ischemic insult.

Conclusion

In this study, good correlation between neurosonography findings in neonates with perinatal asphyxia and severity of hypoxic ischemic encephalopathy was found. Routine NSG in neonates with moderate to severe perinatal asphyxia done between 3-5 days of life will help in early diagnosis, assessment of severity of hypoxic ischemic encephalopathy and management of the affected neonates in a better way.

Combined use of Sarnat staging and neurosonography findings has got good predictive value for severity of disease and outcome in terms of treatment and mortality.

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