



“To Study the Association of Severity of Birth Asphyxia Done by Sarnat Staging with the Neuro-Sonographical Changes and Prognosis at the Time of Discharge in Term Neonates”

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ABSTRACT

INTRODUCTION

Perinatal asphyxia describes the interruption of blood flow or gas exchange to and from the fetus in the perinatal period. Hypoxic-ischemic injury to the brain and vital organs may result if the perinatal asphyxia is of a sufficient degree or prolonged beyond the ability of the fetus to compensate. Approximately 20-30% of infants with HIE die in the neonatal period, and 33-50% of survivors are left with permanent neurodevelopmental abnormalities (cerebral palsy, mental retardation). The greatest risk of adverse outcome is seen in infants with severe fetal acidosis (72% mortality). Cranial USG is Safe, Bedside-comptible, Inexpensive, Enables early imaging and serial imaging such as Brain maturation, growth and evaluation of lesions that makes it suitable for screening

Material and methods:-

This is hospital based prospective analytical study conducted in year of 2022 in the department of pediatrics at Pt JNMCH, Raipur. The sample size calculated for the study was 65 observations. Scientific and ethical committee approval obtained. Patients admitted in the NICU with history of birth asphyxia are enrolled. USG cranium done between 24 to 48 hours of life and at the time of discharge. Consent taken and Proforma was filled. Data was analysed and conclusion derived.

Results-

Neuro-sonographical changes in study subjects between 24 to 48 hours shows that majority 36% had normal findings, 29.33% had cerebral edema, 22.67% had hyperleucency present with normal echo texture. Neuro-sonographical changes in study subjects. Neuro-sonographical changes in study subject's at time of discharge shows that majority 85.33% had no significant abnormality, (6) 8% had Leucen cysts, 2 (2.67%) had Hyperleuceny present with normal echo texture. At 24 to 48 hrs, Among 33 HIE-1 cases, majority 25 (75.8%) had no significant abnormality, 4 (12.1%) had cerebral edema and 3 (9.1%) had

Periventricular hemorrhage. Whereas among 42 HIE-2 cases, majority 17 (40.5%) had cerebral edema, 14 (33.3%) had Periventricular hemorrhage. Among 33 HIE-1 cases, majority 32 (97%) had no significant abnormality and 1 case had hyperleucency with normal echotexture.

Where as among 42 HIE-2 cases, majority 32 (76.2%) also had no significant abnormality, 6 (14.3%) had leucen cysts found.

Conclusion

HIE 2 cases are more compared to HIE 1 & 3. Majority of abnormal findings are found in babies with NVD and low APGAR scores at 1min and 5min of life. The most common consequence is cerebral edema followed by periventricular hemorrhage. We observed significant statistical association between neuroimaging abnormality and severity of HIE. Thus, neuroimaging may help to predict neurodevelopmental outcome in term infants with HIE.

I. INTRODUCTION

Perinatal asphyxia describes the interruption of blood flow or gas exchange to and from the fetus in the perinatal period [1]. This may be prolonged partial asphyxia, sudden sub-total asphyxia due to a sentinel event or a combination of both [2]. Hypoxic-ischaemic injury to the brain and vital organs may result if the perinatal asphyxia is of a sufficient degree or prolonged beyond the ability of the fetus to compensate [3-5]. Approximately 20 per 1000 deliveries will require significant resuscitation, with biochemical and clinical evidence of perinatal asphyxia [6]. Of these only 1.6 per 1000 will go on to develop signs of evolving encephalopathy consistent with hypoxic-ischaemic encephalopathy (HIE) [7]. HIE must be differentiated from other causes of neonatal encephalopathy (NE), such as sepsis, meningitis or a metabolic disorder [1,8,9]. There may be a high suspicion of hypoxic-ischaemic injury following a known perinatal insult such as placental abruption or cord accident or if typical clinical signs,



biochemical evidence of metabolic acidosis or depressed Apgar scores are present. However it can be very difficult to make this differentiation quickly after birth[10]. Approximately 50%-80% of NE can be attributed to hypoxia-ischaemia and given the potential benefit of early treatment, the need to identify infants with hypoxic-ischaemic induced encephalopathy is becoming increasingly important. A detailed classification of HIE in term neonates was proposed by Sarnat and Sarnat.

Aim and Objective

Aim: To study the association of severity of birth asphyxia in term neonates done by sarnat staging with the neuro-sonographical changes and prognosis at the time of discharge.

Objective:

To study association between neuro-sonography changes and HIE staging in neonates with Birth Asphyxia after 24hrs to 48 hours of age.

To find out the association through sonography investigations in term babies with perinatal asphyxia at 24 to 48 hours of age and to find prognosis at the time of discharge .

II. MATERIALS AND METHODS:-

This was a hospital based prospective cross-sectional analytical study conducted on 2022. The study was conducted in the department of pediatrics at Pt JNMCH and Dr. BRAM Hospital, Raipur, Chhattisgarh. All term babies with birth asphyxia admitted in the NICU, DR. BRAM Hospital. This calculator uses the following formula for the sample size $n = (Z\alpha/2 + Z\beta)^2 * 2 * \sigma^2 / d^2$, Sample size was calculated for comparing two sample means by using formula $n = (Z\alpha/2 + Z\beta)^2 * 2 * \sigma^2 / d^2$, and taking confidence interval at 95%, power 80%, at hypothesised difference of 12. The sample size for was 65. All patients will undergo clinical and neurological evaluation, grading at the time of admission (time of admission to study will be arbitrarily taken as the time of first neurologic assessment) and divided into stage I, II, III of HIE according to Sarnat staging. cranial USG done between 24 to 48 hours of life and at the time of discharge. The history, general examination, the neurological profile at the time of admission, important positive investigation findings, the diagnosis and outcome of all cases will be presented in tabular form (master chart) and analyzed. Statistical significance was taken P value <0.05. Written informed consent was taken from the parents were explained about the study.

III. RESULTS:-

Neuro-sonographical changes in study subject's b/w 24 to 48 hours

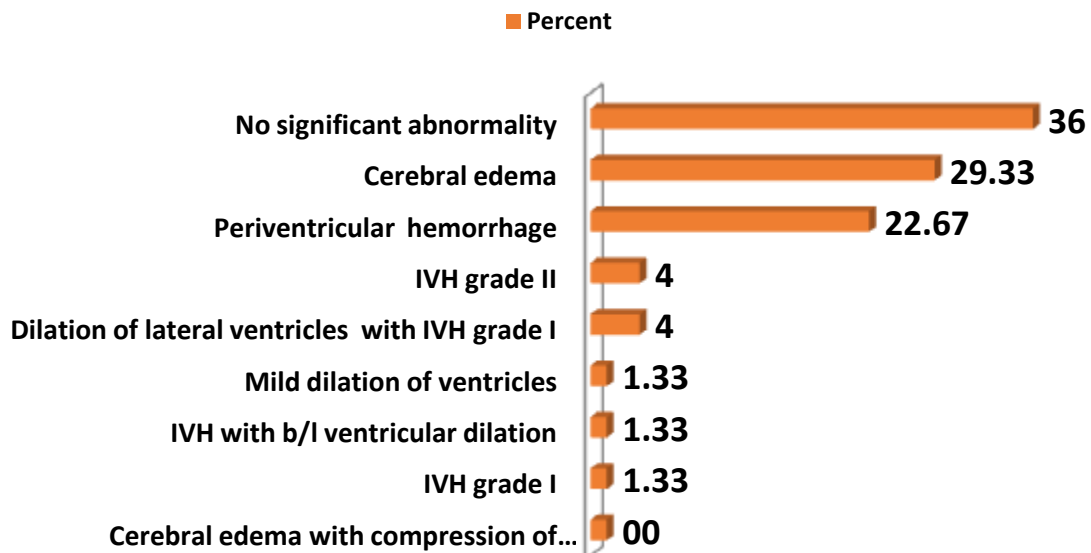
Neuro-sonographical changes B/W 24 to 48 hours	Freq.	Percent
No significant abnormality	27	36%
Cerebral edema	22	29.33%
Periventricular hemorrhage	17	22.67%
IVH grade I	1	1.33%
IVH with b/l ventricular dilation	1	1.33%
Mild dilation of ventricles	1	1.33%
Dilation of lateral ventricles with IVH grade I	3	4%
IVH grade II	3	4%
Total	75	100%

Among study subjects Neuro-sonographical changes in study subject's b/w 24 to 48 hours shows that majority of the findings are

normal and 29.33% had Cerebral edema, 22.67% had Periventricular hemorrhage and 36% had no significant abnormality.



Figure 7: Neuro-sonographical changes in study subject's b/w 24 to 48 hours(N-75)



Neuro-sonographical changes in study subject's at discharge

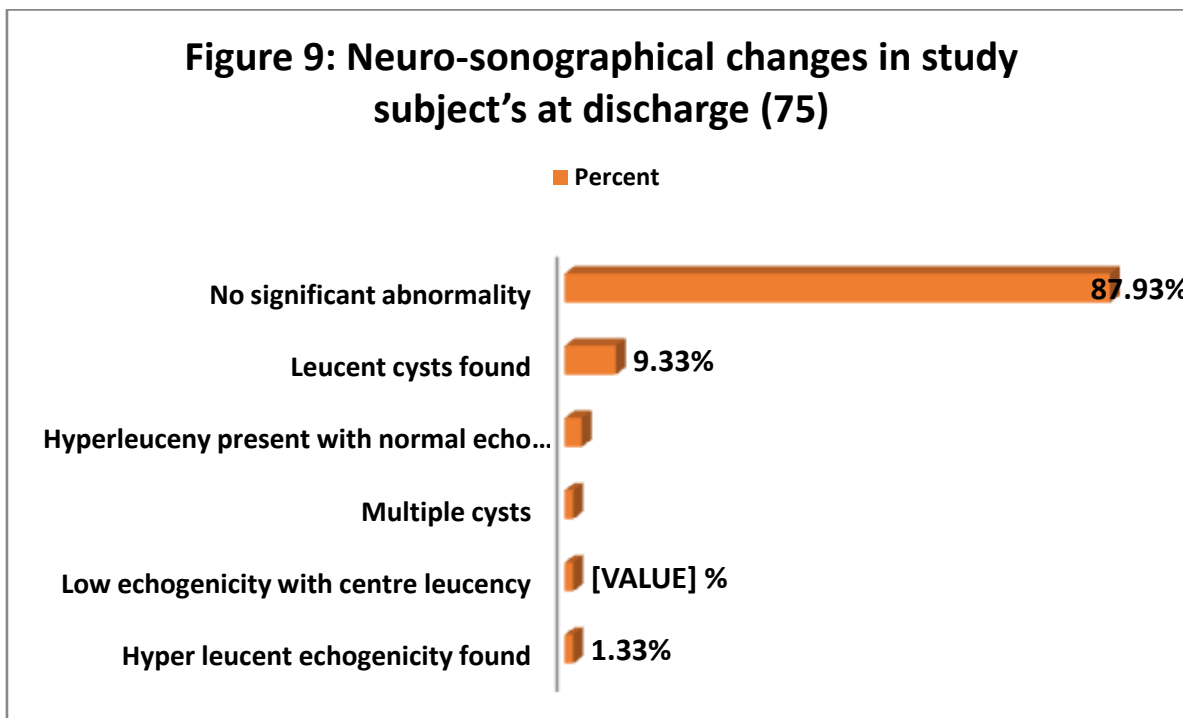
Findings on the day of discharge	Freq.	Percent
Hyper leucent echogenicity found	1	1.33
Low echogenicity with centreleucency	1	1.33
Leucent cysts found	7	9.33
No significant abnormality	66	87.93
Total	75	100

Among study subjects Neuro-sonographical changes in study subject's at time of

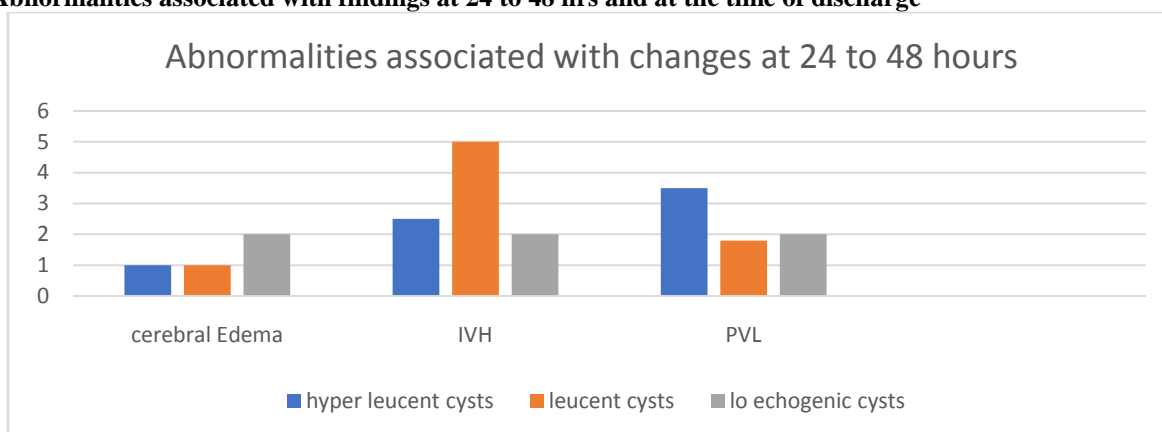
discharge shows that majority 87.93% had no significant abnormality ,9.33% had Leucent cysts.



Figure 9: Neuro-sonographical changes in study subject's at discharge (75)



Abnormalities associated with findings at 24 to 48 hrs and at the time of discharge



It is found that patients with IVH changes had found majority of abnormal findings such as

leucogenic cysts at the time of discharge followed by Periventricular hemorrhage and cerebral edema.

Assoiation b/w Neuro-sonographic findings b/w 24 to 48 hours and SARNAT staging

Neuro-sonographic findings b/w 24 to 48 hours	SARNAT STAGE			Total	P value
	HIE1	HIE2	HIE3		
Cerebral edema	4 12.1%	16 40.5%	01 2.4%	21 28.0%	p<0.01
Cerebral edema with compression of lateral ventricles	0 0.0%	1 2.4%	00 0.0%	1 1.3%	
Dilation of lateral ventricles with IVH grade I	0 0.0%	2 4.8%	01 2.4%	3 4.0%	



IVH grade I	0	2	00	2
	0.0%	4.8%	00	2.6%
IVH grade II	0	3	00	3
	0.0%	7.1%	00	4.0%
Mild dilation of ventricles	1	0	00	1
	3.0%	0.0%	00	1.3%
No significant abnormality	25	2	00	27
	75.8%	4.8%	00	36.0%
Periventricular hemorrhage	3	14	00	17
	9.1%	33.3%	00	22.7%
Total	33	42	00	75
	100.0%	100.0%	100	100.0%

Among 33 HIE-1 cases, majority 25 (75.8%) had no significant abnormality, 4 (12.1%) had cerebral edema and 3 (9.1%) had Periventricular hemorrhage. Whereas among 41 HIE-2 cases, majority 16 (40.5%) had cerebral

edema, 14 (33.3%) had Periventricular hemorrhage and a HIE 3 case with IVH with dilation of b/l ventricles seen. Association was tested using chi square test and it was statistically significant ($p < 0.01$).

Table 12: Association b/w Neuro-sonographic findings at time of discharge and SARNAT staging

Neuro-sonographic findings at day of discharge	SARNAT STAGE			Total	P value
	HIE1	HIE2	HIE3		
Hyper leucent echogenicity found	0	1	0	1	0.154
	0.0%	2.4%	0	1.3%	
Hyperleuceny present with normal echo texture	1	1	0	2	
	3.0%	2.4%	0	2.7%	
Leucent cysts found	0	6	0	6	
	0.0%	14.3%	0	8.0%	
Low echogenicity with centroleucency	0	1	0	1	
	0.0%	2.4%	0	1.3%	
Multiple cysts	0	1	0	1	
	0.0%	0	2.4%	1.3%	
No significant abnormality	32	32	0	64	
	97.0%	76.2%	0	85.3%	
Total	33	42	0	75	
	100.0%	100.0%	100%	100.0%	

Among 33 HIE-1 cases, majority 32 (97%) had no significant abnormality and 1 case had Hyperleuceny present with normal echo texture. Whereas among 41 HIE-2 cases, majority 32 (76.2%) also had no significant abnormality, 6

(14.3%) had Leucent cysts found and HIE3 case with multiple cysts found. Association was tested using chi square test and it was statistically not significant ($p = 0.154$).



Table 14: Association b/w neuro-sonographic changes b/w 24 to 48 hrs and findings on the day of discharge

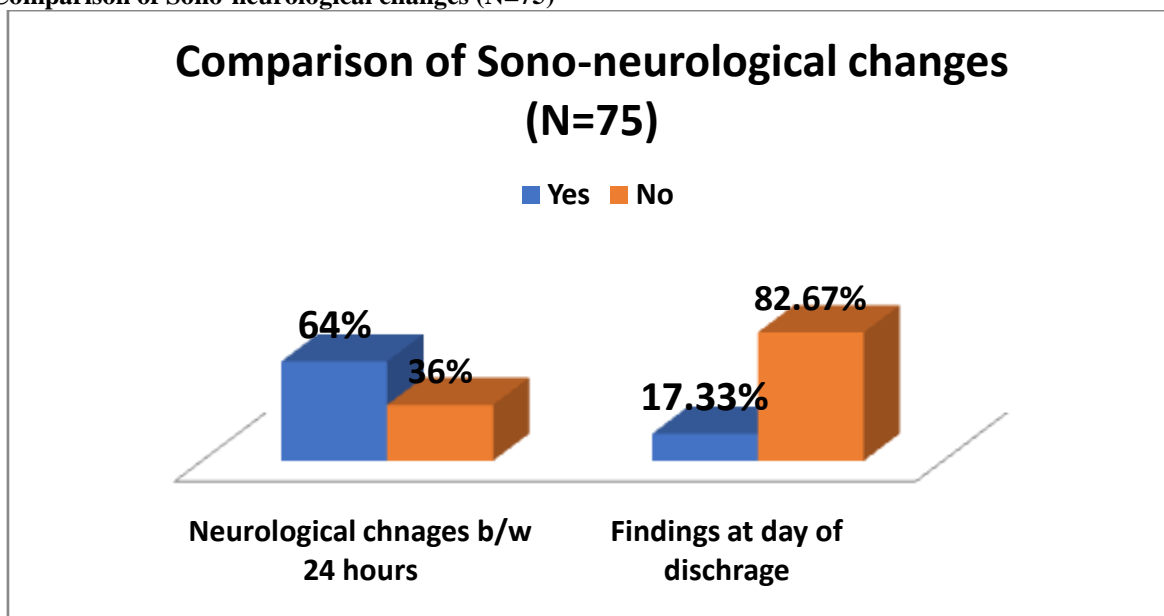
Findings b/w 24 to 48 hours	Findings on the day of discharge						Total
	Hyperleucic echogenicity found	Hyperleuceny present with normal echo texture	Leucic cysts found	Low echogenicity with centrelucency	Multiple cysts	No significant abnormality	
Cerebral edema	0	0	0	0	0	21	21
	0.0%	0.0%	0.0%	0.0%	0.0%	32.8%	28.0%
Cerebral edema with compression of lateral ventricles	0	0	0	0	0	1	1
	0.0%	0.0%	0.0%	0.0%	0.0%	1.6%	1.3%
Dilation of lateral ventricles with IVH grade I	0	0	2	0	0	1	3
	0.0%	0.0%	33.3%	0.0%	0.0%	1.6%	4.0%
IVH grade I	0	0	0	0	0	1	1
	0.0%	0.0%	0.0%	0.0%	0.0%	1.6%	1.3%
IVH grade II	0	1	1	0	1	0	3
	0.0%	50.0%	16.7%	0.0%	100.0%	0.0%	4.0%
IVH with b/l ventricular dilation	0	0	0	0	0	1	1
	0.0%	0.0%	0.0%	0.0%	0.0%	1.6%	1.3%
Mild dilation of ventricles	0	0	0	0	0	1	1
	0.0%	0.0%	0.0%	0.0%	0.0%	1.6%	1.3%
No significant abnormality	0	0	0	0	0	27	27
	0.0%	0.0%	0.0%	0.0%	0.0%	42.2%	36.0%
Periventricular hemorrhage	1	1	3	1	0	11	17
	100.0%	50.0%	50.0%	100.0%	0.0%	17.2%	22.7%
Total	1	2	6	1	1	64	75
	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Association b/w neuro-sonographic changes b/w 24 to 48 hrs and findings on the day of

discharge was tested using chi-square test and it was found statistically significant (p=0.002).



Comparison of Sono-neurological changes (N=75)



At the time of discharge 82.67% of HIE babies found to have normal findings and were 36% were normal at the 24 to 48 hours of life. 17.33% of the patients have left with minimal changes.

IV. DISCUSSION

The present study was conducted in department of pediatrics at Pt. JNMCH Raipur with the objective to study the association of severity of birth asphyxia in term neonates with the neuro-sonographical changes and prognosis at the time of discharge. This study duration was 1 year and total 75 term babies included, among them 60% were Male and 40% were Female with 21.33% LSCS and 78.67% were NVD and some of the taken cases were under exclusion criteria.

In present study the mean gestational age among study subjects was 38.53 ± 1.18 weeks. Majority 54.67% were born b/w 38-39 weeks of gestational age followed by 22.67% each were born b/w 36-37 weeks and 40-41 weeks of gestational age. Thakkar P A et al. (2017) study the clinical profile, outcome and clinical indicators for poor prognosis in full term babies born with severe birth asphyxia. The present study was carried out at Intramural Neonatal Nursery of Department of Pediatrics at Medical College SSG Hospital, Vadodara, Gujarat, India. The mean gestational age of study subjects was 38.62 ± 1.1 weeks. [95] Debnath B et al (2021) study the severity and immediate neurodevelopmental outcome in term neonates with hypoxic-ischemic encephalopathy admitted in NICU at a tertiary hospital in Bangladesh. The mean gestational age of study

subjects was 38.73 ± 1.03 weeks. [104] which is similar to our study.

In present study majority 60% were male and 40% were female. The male female ration was 1.5:1. Thakkar P A et al. (2017) study the clinical profile, outcome and clinical indicators for poor prognosis in full term babies born with severe birth asphyxia. They reported that in their study male were 55% and male female ratio was 1.25:1. [95] Debnath B et al (2021) study the severity and immediate neurodevelopmental outcome in term neonates with hypoxic-ischemic encephalopathy admitted in NICU at a tertiary hospital in Bangladesh. In this study, there was male predominance (61.7%) with male to female ratio 1.6:1. [104] which is similar to our study.

In present study 56% of the study subjects were inborn and 44% were out born. Majority 78.67% were born through normal vaginal delivery and 21.33% were born through LSCS. Patil B et al (2015) study the clinicoradiological correlation in birth asphyxia. In their study 68% were born through normal vaginal delivery, 24% were born through LSCS and 8% through assisted vaginal delivery. [91] Yasmin T et al (2016) study the assessment of Cranial Sonographic Findings of Hypoxic Ischemic Brain Injury in Perinatal Asphyxia. Mode of delivery was normal vaginal delivery for 46% neonates and 54% via LUCS for various reasons. [93] Thakkar P A et al. (2017) study the clinical profile, outcome and clinical indicators for poor prognosis in full term babies born with severe birth asphyxia. They



reported that most of the neonates were inborn (58.3%).[95] which is similar to our study.

Among study subjects the mean birth weight was 2.70 ± 0.36 Kg. Majority 68% were having normal birth weight and 32% were low birth weight. Boo NY et al (2000) study the early cranial ultrasound changes as predictors of outcome during first year of life in term infants with perinatal asphyxia. They reported that in term neonates the mean birth weight was 3.081kg. [87]Yasmin T et al (2016) study the assessment of Cranial Sonographic Findings of Hypoxic Ischemic Brain Injury in Perinatal Asphyxia. In term babies, birth weight between 2.5-3.5 kg was in 40 (71%) cases and between $>3.5-4$ kg was in 16 (29%) cases.[92]which is similar to our study.

In present study the mean APGAR score of term neonates at 1 minute was 3.02 ± 0.66 and mean APGAR score at 5 minute was 5.71 ± 0.46 .

Boo NY et al (2000) study the early cranial ultrasound changes as predictors of outcome during first year of life in term infants with perinatal asphyxia. They reported that the mean APGAR score at 1 minute was 2.9 ± 1.4 and mean APGAR score at 5 minute was 5.1 ± 0.1 . [87]Thakkar P A et al. (2017) study the clinical profile, outcome and clinical indicators for poor prognosis in full term babies born with severe birth asphyxia. They reported that in their study 13/45 (29%) had APGAR score of < 5 at 5 minute, and 4/45 (9%) had an APGAR < 5 at 10 minutes of age. 46% of babies required < 1 min duration of IPPR. [95]Although APGAR is not specific indicator of Birth Asphyxia we have taken it according to available resources.

In present study among study subjects only 5.41% were on inotropes and 54.67% were on antiepileptic drugs. Raj Prakash (2016) did a prospective observational study study the clinical profile and neurobehaviour at discharge of term neonates with perinatal asphyxia. Of 120 asphyxiated infants 9% were on inotropes and 37% were on antiepileptic drugs. [94]which is not similar to our study as there are more HIE 2 cases and all cases have treated with anti epileptics.

In present study among study subjects 54.7% were on HIE-2 as per SARNAT staging and 44% were on HIE-1 and 1.33% were HIE3 as per SARNAT staging. Boo NY et al (2000) study the early cranial ultrasound changes as predictors of outcome during first year of life in term infants with perinatal asphyxia. They reported that among study subjects 48.1% were in HIE grade-1, 15.4% in HIE grade-2 and 21.2% in HIE grade-3. [87]Patil B et al (2015) study the clinicoradiological correlation in birth asphyxia.

They reported that 57% neonates were in HIE-2 and 43% in HIE-3.[91] similar to our study.

BIBLIOGRAPGY

- [1]. Volpe JJ. Neurology of the Newborn. Philadelphia: Saunders; 2001. [Google Scholar]
- [2]. Fatemi A, Wilson MA, Johnston MV. Hypoxic-ischemic encephalopathy in the term infant. Clin Perinatol. 2009;36:835–858, vii.
- [3]. Ugwumadu A. Understanding cardiotocographic patterns associated with intrapartum fetal hypoxia and neurologic injury. Best Pract Res Clin Obstet Gynaecol. 2013;27:509–536.
- [4]. Low JA. Determining the contribution of asphyxia to brain damage in the neonate. J Obstet Gynaecol Res. 2004;30:276–286.
- [5]. Parer JT. Effects of fetal asphyxia on brain cell structure and function: limits of tolerance. Comp Biochem Physiol A Mol Integr Physiol. 1998;119:711–716.
- [6]. Vannucci RC. Hypoxic-ischemic encephalopathy. Am J Perinatol. 2000;17:113–120.
- [7]. Lee AC, Kozuki N, Blencowe H, Vos T, Bahalim A, Darmstadt GL, Niermeyer S, Ellis M, Robertson NJ, Cousens S, et al. Intrapartum-related neonatal encephalopathy incidence and impairment at regional and global levels for 2010 with trends from 1990. Pediatr Res. 2013;74 Suppl 1:50–72.
- [8]. Edwards AD, Nelson KB. Neonatal encephalopathies. Time to reconsider the cause of encephalopathies. BMJ. 1998;317:1537–1538.
- [9]. Nelson KB, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia? Am J Dis Child. 1991;145:1325–1331.
- [10]. Volpe JJ. Neonatal encephalopathy: an inadequate term for hypoxic-ischemic encephalopathy. Ann Neurol. 2012;72:156–166.
- [11]. Dammann O, Ferriero D, Gressens P. Neonatal encephalopathy or hypoxic-ischemic encephalopathy? Appropriate terminology matters. Pediatr Res. 2011;70:1–2.
- [12]. Cowan F, Rutherford M, Groenendaal F, Eken P, Mercuri E, Bydder GM, Meiners LC, Dubowitz LM, de Vries LS. Origin and timing of brain lesions in term infants



- with neonatal encephalopathy. *Lancet*. 2003;361:736–742.
- [13]. Shah DK, Lavery S, Doyle LW, Wong C, McDougall P, Inder TE. Use of 2-channel bedside electroencephalogram monitoring in term-born encephalopathic infants related to cerebral injury defined by magnetic resonance imaging. *Pediatrics*. 2006;118:47–55
- [14]. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute systemic organ injury in term infants after asphyxia. *Am J Dis Child* 1989;143:617-20.
- [15]. Dr. Mohamed A. El-Gamasy¹, Dr Mohammed Abd Ellatif Nassar, NICUs of Tanta Universality hospital, *International Journal of Research Studies in Medical and Health Sciences* V2 I12 2017
- [16]. Sarnat HB, Sarnat MS: Neonatal encephalopathy following fetal distress: A clinical and electroencephalographic study. *Arch Neurol* 33: 695-706, 1976.
- [17]. Epelman M, Daneman A, Kellenberger CJ, Aziz A, Konen O, Moineddin R, et al. Neonatal encephalopathy: A prospective comparison of head US and MRI. *Pediatr Radiol* 2010;40:1640-50
- [18]. Lowe LH, Bailey Z. State-of-the-art cranial sonography: Part 1, modern techniques and image interpretation. *AJR Am J Roentgenol* 2011;196:1028-33
- [19]. Ilves P, Lintrop M, Talvik I, Muug K, Maipuu L, Metsvaht T. Low cerebral blood flow velocity and head circumference in infants with severe hypoxic ischemic encephalopathy and poor outcome. *Acta Paediatr* 2009;98:459-65
- [20]. Ghei SK, Zan E, Nathan JE, Choudhri A, Tekes A, Huisman TA, et al. MR imaging of hypoxic-ischemic injury in term neonates: Pearls and pitfalls. *Radiographics* 2014;34:1047-61

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