

**Research Paper**

## Evaluation of Clinical Predictors and Outcome in Newborn with Hypoxic Ischemic Encephalopathy

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**Abstract**

**Introduction:** In the present days, Neonatal encephalopathy is a heterogenous syndrome characterized by signs of central nervous system dysfunction in newborn infants often lead to long lasting detrimental neurological defect.

**Material and Method:** Prospective type of study was conducted on 150 infants in Obstetrics and Gynecological department of Rajindra Hospital, Patiala and admitted to Neonatology section of Department of Pediatrics with birth asphyxia and were divided into study group (delivered with perinatal asphyxia) and control group (normally delivered without any history of perinatal asphyxia). The data was recorded and subjected to statistical analysis.

**Results:** It was observed that out of 150 cases of group-I (study group), 87(58%) progressed into Sarnat and Sarnat stage-I had good outcome. 9 (6%) cases progressed into stage-II had poor outcome and 54(36%) in stage-III had poor outcome.

**Conclusion:** Major cause of morbidity and mortality is birth asphyxia which indicates the need for early detection of maternal risk factors and better obstetric management.

**Keywords:** neonate, encephalopathy, perinatal, hypoxic.

**Introduction**

In Neonatal encephalopathy [NE], Apgar scores at 1 min and 5 min reflect the neonate's general condition immediately after birth and are predictive of neurological outcome respectively<sup>[1]</sup>. The adverse events in the developing brain often lead to long lasting detrimental neurological defects later on in life such as mental retardation,

epilepsy, cerebral palsy, learning disabilities, gross development delay, motor disabilities and other neurological handicaps.<sup>[2]</sup> The initial most striking feature to neonatal asphyxia is delayed onset of breathing at birth, followed by difficulty in making all of the physiologic transitions from intrauterine to extra uterine life.<sup>[3]</sup> Hypoxic ischemic encephalopathy is the primary cause of 15% to

28% of cerebral palsy among children.<sup>[4]</sup>  
 Longitudinal monitoring of educational and behavioural development is therefore recommended in both children with mild and moderate NE.<sup>[5]</sup>

**Aims and Objective**

The aim of the study was to evaluate the clinical predictors and outcome in newborn with hypoxic ischaemic encephalopathy by doing regular follow-up for 18 months.

**Material and Methods**

This study was conducted on 150 children to evaluate the clinical predictors of outcome in newborn hypoxic ischemic encephalopathy. Follow up up to the age of 3, 6, 9, 12, 15, 18 months was done. The group-1 (study group) will consist of 150 babies, delivered with perinatal asphyxia and group-II (control group) will consists of 50 babies, normally delivered without any history of perinatal asphyxia.

**Inclusion Criteria**

- 1) Full term babies with low Apgar score (i.e. a 5 min score of <= 7) or post- asphyxia symptoms admitted within 24h of delivery

**Exclusion Criteria**

- 1) Infants with gestational age <37 weeks (as the neurological complications of prematurity may interfere with the results),
- 2) Presence of perinatal infection.
- 3) Those who did not complete the course of the follow up.
- 4) All infants with obvious congenital malformations, congenital mental disorders.
- 5) Maternal drug addiction.

All cases of birth asphyxia who landed into hypoxic ischaemic encephalopathy [HIE] were evaluated in detail clinically for signs and symptoms of birth asphyxia and were graded into three groups i.e. HIE-I, HIE-II, HIE-III as per Sarnat and Sarnat's Staging System. All the babies were followed and complete examination for vitals and anthropometry, general physical and systemic examination and development were recorded and data was analyzed statistically.

**Results**

In our study 150 full terms neonate were enrolled who were delivered at Obstetrics and Gynecological department of Rajindra Hospital, Patiala as per inclusion and exclusion criteria and following observations were made in the study.

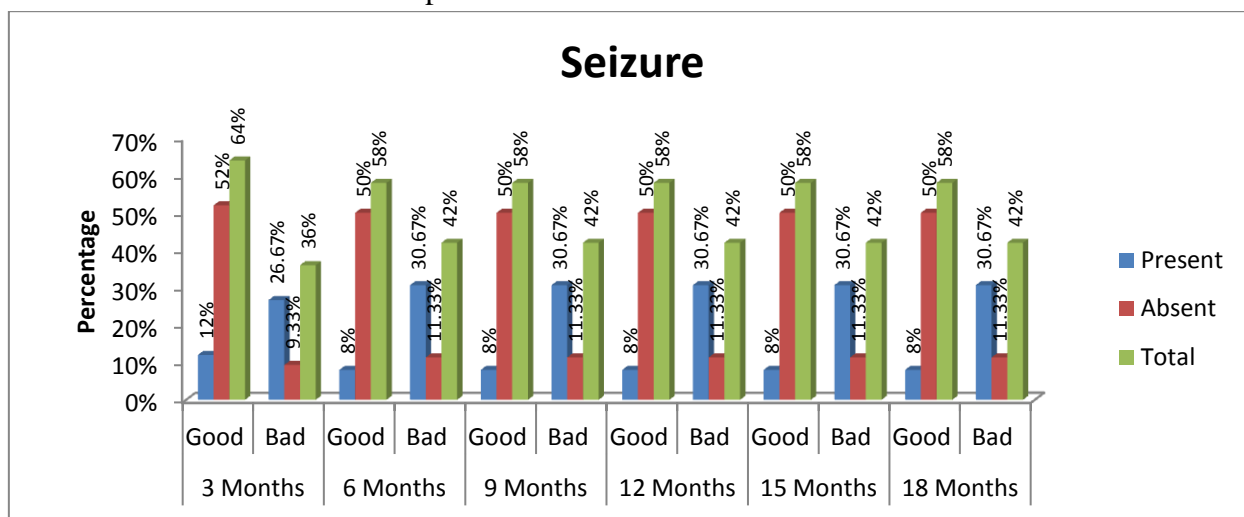
**Table 1: Clinical Predictors**

Clinical Predictors	Group I (Study Group) (N=150)				Group II (Control Group) (N=50)				X <sup>2</sup>	P value
	Good Outcome		Poor Outcome		Good Outcome		Poor Outcome			
	Present n (%)	Absent n (%)	Present n (%)	Absent n (%)	Present n (%)	Absent n (%)	Present n (%)	Absent n (%)		
Maternal Factors	20 (13.33%)	67 (44.67%)	18 (12%)	45 (30%)	15 (30%)	35 (70%)	0 (0%)	0 (0%)	0.12	0.728 (NS)
Fetal Factors	37 (24.67%)	50 (33.33%)	29 (19.33%)	34 (22.67%)	22 (44%)	28 (56%)	0 (0%)	0 (0%)	0.71	0.401 (NS)
Placental Factors	34 (22.67%)	54 (36%)	24 (16%)	39 (26%)	19 (38%)	31 (62%)	0 (0%)	0 (0%)	0.37	0.542 (NS)
Mode of Delivery	87 (58%)	0 (0%)	63 (42%)	0 (0%)	50 (100%)	0 (0%)	0 (0%)	0 (0%)	1.27	0.259 (NS)
Cessarian delivery	25 (16.67%)	0 (0%)	17 (11.33%)	0 (0%)	17 (34%)	0 (0%)	0 (0%)	0 (0%)	0.03	0.864 (NS)
Vaginal delivery	62 (41.33%)	0 (0%)	46 (30.67%)	0 (0%)	33 (66%)	0 (0%)	0 (0%)	0 (0%)	1.82	0.177 (NS)

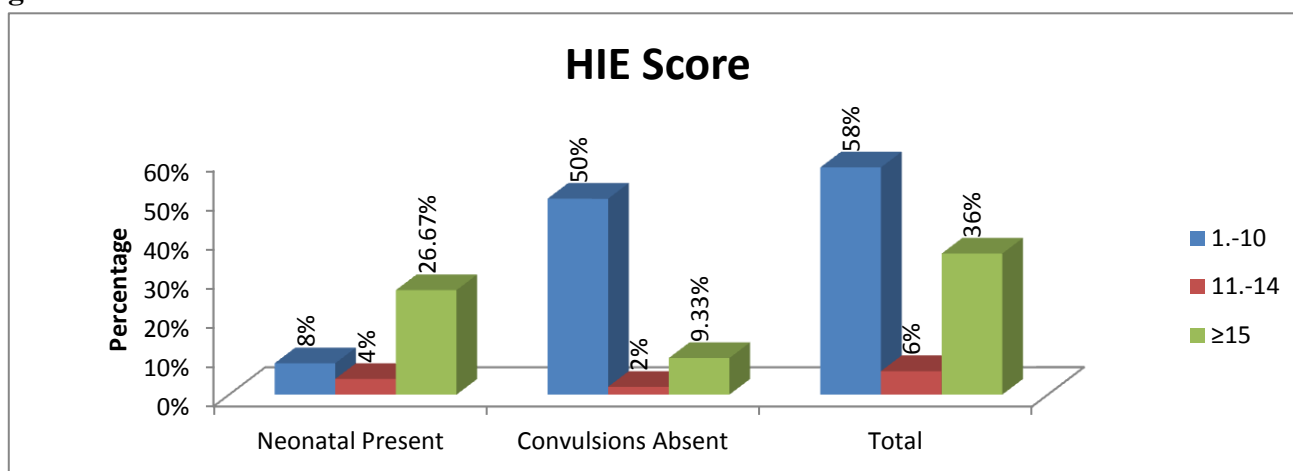
It was observed that out of 150 cases of group-I (study group), 87(58%) progressed into Sarnat and Sarnat stage-I had good outcome. 9 (6%) cases

progressed into stage-II had poor outcome and 54(36%) in stage-III had poor outcome as per table1.

**Diagram 1:** Distribution of Babies as per occurrence of Seizure



**Diagram 3:** HIE score in neonates



It was observed that out of 150 cases in group-I (study group), 87 (58%) has THOMPSON’S HIE score 1-10, out of which 12 cases had convulsions and 75 (50%) had no convulsions. 9 (6%) cases had HIE score of 11-14. Out of which 6 (4%) had convulsions and 3 (2%) had no convulsions. 54 (36%) cases had HIE score 15-22, out of which

40 (26.67%) had convulsions and 14 (9.33%) had no convulsions as per diagram 2 and 3.

**Discussion**

The present study was comparable with other study on the basis of Sarnat and Sarnat HIE staging.

**Table 2:** Comparison of outcome of neonates as per Sarnat and Sarnat staging

Studies	Sarnat and Sarnat HIE stage				Outcome	
	NO HIE	Stage-I	Stage-II	Stage-III	good	Poor
Sekela mwakyusa et al 2008 <sup>[6]</sup>	0	98	26	16	111	29
Khaled Abdulqawi et al 2011 <sup>[7]</sup>	0	16	19	20	22	33
Helen Trotman et al 2011 <sup>[8]</sup>	0	29	41	25	61	34
Prachipaliwal et al 2014 <sup>[9]</sup>	18	20	41	21	68	32
Serdar et al 2014 <sup>[10]</sup>	0	29	36	29	57	37
Ashaq h thoker et al 2017 <sup>[11]</sup>	0	45	40	8	66	27
Present study	0	87	9	54	87	63

Results of present study were comparable to Serdar et al. Results of studies by Khaled Abdulqawi et al, Sekela Mwakyusa et al, Prachipaliwal et al, Helen Trotman et al, Ashaq Thoker et al were in

agreement with present study that all newborns in stage-III had poor neurodevelopmental outcome and were comparable as in table 2.

**Table 3:** Comparison of various studies with neonatal seizures and their outcome with HIE

Studies	Seizures		Outcome	
	Present	Absent	Good	Poor
Matthew ellis et al 2007 <sup>[12]</sup>	57	74	40	91
Sekela D Mwakusa et al 2008 <sup>[6]</sup>	58	82	111	29
Helen Trotman et al 2011 <sup>[8]</sup>	54	31	53	32
Ashaq H Thoker et al 2017 <sup>[11]</sup>	28	65	66	27
Present study	58	92	87	63

Present study results were in agreement with Ashaq H Thoker et al that occurrence of seizures had poor neurodevelopmental outcome with statistically significant p-value ( $p < 0.0001$ ) as in present study ( $p < 0.029$ ). Also study by Helen Trotman et al is in consistent with present study that 97% (31/32) of neonates with poor outcome had clinically apparent seizures compared to 43% (23/53) of neonates who had good outcome ( $p < 0.001$ ).

### Conclusion

Birth asphyxia still remains a major cause of morbidity and mortality during neonatal period in India. Overall mortality was 22.4 % which clearly indicates the need for early detection of maternal risk factors, better obstetric management and prompt resuscitation measures. Both antepartum and intrapartum factors are important in the causation of neonatal encephalopathy in developing countries.

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