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CLINICAL PROFILE, SHORT-TERM NEUROLOGICAL OUTCOME AND PREDICTORS OF ADVERSE OUTCOME AMONG FULL-TERM NEONATES WITH SEVERE BIRTH ASPHYXIA: A PROSPECTIVE COHORT STUDY

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ABSTRACT

Background: Perinatal asphyxia remains an important cause of neonatal morbidity and mortality, particularly in resource-limited settings. Hypoxic-ischaemic injury may result in hypoxic-ischaemic encephalopathy (HIE), seizures, multiorgan dysfunction and long-term neurodevelopmental impairment. Early recognition of clinical predictors of poor outcome may facilitate risk stratification and appropriate neonatal care. The objective is to study the clinical profile and short-term outcome of full-term neonates with severe birth asphyxia and to identify clinical factors associated with adverse neurological outcome and mortality.

Materials and Methods: A prospective cohort study was conducted in the Intramural Neonatal Nursery of the Department of Pediatrics and Department of Obstetrics, M. P. Shah Medical College and G.G. Hospital, Jamnagar, Gujarat. Full-term neonates with severe birth asphyxia, defined according to the study protocol as an Apgar score ≤3 at 1 minute, were enrolled. Neonates born outside the study hospital, preterm infants, babies with major congenital malformations, birth weight <1,800 g and other predefined exclusion criteria were excluded. Clinical characteristics, resuscitation requirements, Apgar scores, HIE stage according to Sarnat and Sarnat classification, seizures, organ dysfunction, duration of NICU admission and mortality were recorded. Survivors were followed at 3 months using clinical neurological assessment and the Amiel-Tison scale. Statistical analysis included descriptive statistics, chi-square testing and multivariable analysis. A p value <0.05 was considered statistically significant.

Results: Fifty full-term neonates with severe birth asphyxia were enrolled. The mean gestational age was 38.6 ± 1.1 weeks and mean birth weight was 2561.2 ± 363.02 g. Males constituted 56% of the cohort. Hypoxic-ischaemic encephalopathy developed in 39/50 (78%) neonates, including HIE grade I in 10 (20%), grade II in 13 (26%) and grade III in 16 (32%). Common morbidities included acute kidney injury (44%), mechanical ventilation (42%), shock (34%), sepsis (56%), seizures (54%) and multiorgan dysfunction (32%). Longer duration of intermittent positive-pressure ventilation was significantly associated with increasing severity of HIE (p=0.0012). Overall mortality was 42%. Among survivors assessed at 3 months, 29 had normal and 12 had abnormal neurological findings. Multivariable analysis identified abnormal neurological findings at day 7, Apgar score <6 at 10 minutes and HIE grade II or higher as significant predictors of adverse neurological outcome. Difficult-

to-control seizures, multiorgan dysfunction and Apgar score <4 at 10 minutes were associated with mortality.
Conclusion: Severe birth asphyxia in full-term neonates was associated with substantial HIE, multisystem morbidity and mortality. Severity of HIE, persistent neurological abnormalities and low Apgar scores at 10 minutes were important predictors of adverse outcome. Early identification of these clinical markers may assist in prognostication and planning of intensive neonatal care and follow-up.
Keywords: Severe Birth Asphyxia, Hypoxic-Ischaemic Encephalopathy, Neonate, Apgar Score, Neonatal Seizures, Neurological Outcome, Mortality.

INTRODUCTION

Perinatal asphyxia is an important cause of neonatal morbidity and mortality and remains a major contributor to adverse neurological outcomes in newborns. Hypoxia and ischaemia occurring during the perinatal period may result in systemic organ injury as well as cerebral injury. The clinical consequences range from transient neurological abnormalities to hypoxic-ischaemic encephalopathy (HIE), seizures, cerebral palsy, developmental impairment and death.[1–4]

The burden is particularly relevant in developing countries where timely antenatal surveillance, intrapartum monitoring, neonatal resuscitation and advanced neonatal intensive care may not be uniformly available.[3,5–7]

The uploaded study material identifies birth asphyxia as an important contributor to neonatal mortality and emphasizes the relationship between severity of encephalopathy and subsequent neurological outcome.

HIE is one of the most clinically important manifestations of perinatal hypoxic-ischaemic injury. The Sarnat and Sarnat clinical staging system provides a practical method for classifying the severity of encephalopathy.[8]

Mild encephalopathy generally has a favourable prognosis, whereas moderate and severe encephalopathy are associated with increasing risks of death and neurodevelopmental disability.[8–10]

The source material similarly emphasizes that increasing HIE severity is associated with poorer prognosis, with severe HIE carrying a particularly high risk of mortality and neurological disability.

The outcome following perinatal asphyxia is influenced by several antenatal, intrapartum and neonatal factors. Apgar scores, duration of resuscitation, requirement for assisted ventilation, seizures, metabolic abnormalities, multiorgan dysfunction and persistence of neurological abnormalities have all been considered potential prognostic markers.[11–16]

The uploaded material specifically identifies low Apgar score, difficult-to-control seizures, repeated seizures, multiorgan dysfunction, prolonged ventilation and higher grades of HIE among important adverse prognostic factors.

Although biochemical markers, electroencephalography (EEG) and magnetic resonance imaging (MRI) can provide additional prognostic information, these investigations may not be consistently available in resource-constrained neonatal units.[17–20]

Consequently, simple clinical variables that can be assessed at the bedside remain important for early risk stratification. Previous studies have demonstrated that clinical neurological examination, neonatal seizures and severity of HIE can provide useful prognostic information.[9,21]

The present study therefore focused primarily on clinical parameters and short-term neurological outcomes among full-term neonates with severe birth asphyxia.

The study aimed to describe the clinical profile and outcome of full-term neonates with severe birth asphyxia, determine the frequency and severity of HIE, and identify clinical factors associated with adverse neurological outcome and mortality. This objective is consistent with the original study protocol, which specifically evaluated 3-month neurological outcome and risk factors associated with adverse outcome.

MATERIALS AND METHODS

Study design and setting: This was a prospective cohort study conducted in the Intramural Neonatal Nursery of the Department of Pediatrics and Department of Obstetrics at M. P. Shah Medical College and G.G. Hospital, Jamnagar, Gujarat. The study was conducted during the study period specified in the original research protocol. The uploaded methodology describes the study as a prospective, time-bound study conducted in the neonatal nursery.

Study population: The study population consisted of consecutive full-term neonates born at the study institution who fulfilled the study definition of severe birth asphyxia.

Severe birth asphyxia was defined using the National Neonatology Forum-based criterion adopted in the original study protocol, with an Apgar score ≤3 at 1 minute.

Inclusion criteria

The following neonates were eligible:

- Full-term neonates (≥37 weeks of gestation).
- Neonates born at the study institution.
- Neonates fulfilling the study definition of severe birth asphyxia.

- Neonates with birth weight ≥1,800 g.

Exclusion criteria

Neonates were excluded if they had:

- Delivery outside the study hospital followed by referral.
- Prematurity (<37 weeks).
- Birth weight <1,800 g.
- Major congenital malformations, particularly central nervous system malformations.
- Pyogenic meningitis.
- Severe hyperbilirubinaemia requiring exchange transfusion.
- Refusal of consent.

These exclusion criteria were specified in the original study methodology.

Data collection: After enrolment, demographic and clinical information was recorded prospectively using a structured study proforma. Variables included maternal and neonatal characteristics, gestational age, birth weight, sex, antenatal care, mode of delivery, Apgar scores at 1, 5 and 10 minutes, details and duration of resuscitation, need for mechanical ventilation, seizures, HIE stage, requirement for inotropes, multiorgan dysfunction, duration of NICU stay and mortality.

Gestational age was determined from first-trimester ultrasonography whenever available. When this was unavailable, the last menstrual period was considered; if neither was available, gestational age was assessed using the New Ballard scoring system. Complete antenatal care was defined as at least three antenatal visits.

Clinical management: All neonates received standard supportive neonatal care according to NICU protocols. Management included maintenance of normothermia, adequate oxygenation and ventilation, maintenance of tissue perfusion, correction of glucose and calcium abnormalities, nutritional support and treatment of seizures.

Neonatal seizures were initially treated with phenobarbitone. Additional anticonvulsant therapy with phenytoin and subsequently midazolam infusion was used when seizures remained uncontrolled.

Assessment of HIE: Neurological status was assessed clinically and HIE was graded according to the Sarnat and Sarnat classification. The highest grade reached during the neonatal period was recorded. Because EEG was not available for all infants, the study relied primarily on clinical criteria for staging.

Assessment of adverse prognostic factors

The following clinical variables were evaluated as potential predictors of poor outcome:

1. Repeated seizures.
2. Difficult-to-control seizures.
3. Multiorgan dysfunction.
4. Requirement for inotropic support.
5. Persistent abnormal neurological findings beyond 7 days.
6. Apgar score <4 at 10 minutes.
7. Assisted ventilation for >24 hours.
8. Hypoglycaemia.
9. Polycythaemia.
10. HIE grade II or III.
11. Persistent feeding difficulty.

These factors were prospectively recorded during hospitalization.

Follow-up and neurological assessment: At discharge, neonates were assessed for neurological abnormalities, including abnormalities of tone, primitive reflexes and feeding difficulty. Growth parameters were recorded.

Surviving infants were followed through a high-risk neonatal clinic. Neurological and developmental assessment was performed at 3 months of age using the Amiel-Tison neurological assessment together with assessment of growth parameters. Tone, primitive reflexes, posture and neurosensory function were assessed.

The final neurological outcome was classified as favourable when the neurological assessment was normal and adverse when abnormal neurological findings were present.

Statistical analysis: Data were entered into Microsoft Excel and analysed using descriptive and analytical statistical methods. Categorical variables were expressed as frequencies and percentages, while continuous variables were summarized using mean ± standard deviation or median as appropriate. Associations between categorical variables were assessed using the chi-square test. Multivariable analysis was used to assess factors associated with adverse neurological outcome and mortality. A p value <0.05 was considered statistically significant.

Ethical considerations: The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from the parents/guardians of participating neonates.

RESULTS

Study population

During the study period, 72 neonates fulfilling the initial screening criteria were identified. Twenty-two were excluded, including 18 premature neonates, one neonate with pyogenic meningitis and three neonates with major congenital malformations. A total of 50 full-term neonates with severe birth asphyxia were included in the final analysis.

Among 2,105 live births during the relevant period, 72 neonates were identified with severe birth asphyxia. The reported incidence of severe birth asphyxia was 34 per 1,000 live births. Among full-term births, the reported incidence was approximately 27 per 1,000 full-term live births.

Baseline characteristics

The mean gestational age was 38.6 ± 1.1 weeks and the mean birth weight was 2561.2 ± 363.02 g. Males accounted for 28 (56%) neonates and females for 22 (44%). The majority of neonates were appropriate for gestational age. Seventy percent of mothers had

not received adequate antenatal care according to the study definition.

Table 1: Baseline characteristics of neonates with severe birth asphyxia

Characteristic	n (%) / Mean ± SD
Total neonates	50 (100)
Gestational age, weeks	38.6 ± 1.1
Birth weight, g	2561.2 ± 363.02
Male sex	28 (56.0)
Female sex	22 (44.0)
AFD	43 (86.0)
SFD	7 (14.0)
Adequate ANC	15 (30.0)
Inadequate/no ANC	35 (70.0)

Apgar scores and resuscitation

All enrolled neonates had severe depression at birth according to the study inclusion criterion. At 5 minutes, 14/50 (28%) had an Apgar score <5, while 4/50 (8%) had an Apgar score <5 at 10 minutes.

Regarding intermittent positive-pressure respiration (IPPR), 12 (24%) required <30 seconds, 10 (20%) required 30–60 seconds, 14 (28%) required 60–120 seconds and 14 (28%) required >120 seconds. Overall, 44% required less than one minute of IPPR.

Clinical morbidities

The common neonatal complications observed during hospitalization were acute kidney injury (44%), sepsis (56%), seizures (54%), mechanical ventilation (42%), shock (34%), respiratory distress syndrome (32%), multiorgan dysfunction (32%), persistent feeding difficulty (48%), disseminated intravascular coagulation (16%), polycythaemia (12%) and hypocalcaemia (6%).

Table 2: Clinical morbidities among neonates with severe birth asphyxia

Morbidity	n (%)
Sepsis	28 (56)
Seizures	27 (54)
Persistent feeding difficulty	24 (48)
Acute kidney injury	22 (44)
Mechanical ventilation	21 (42)
Shock	17 (34)
RDS	16 (32)
Multiorgan dysfunction	16 (32)
DIC	8 (16)
Polycythaemia	6 (12)
Hypocalcaemia	3 (6)
Apnoea	2 (4)
NEC	1 (2)

Incidence and severity of HIE

HIE developed in 39/50 (78%) neonates, while 11/50 (22%) had no clinical evidence of HIE. Among the entire cohort, HIE grade I was present in 10 (20%), grade II in 13 (26%) and grade III in 16 (32%).

Table 3: Severity of hypoxic-ischaemic encephalopathy

HIE stage	n (%)
No HIE	11 (22)
Grade I	10 (20)
Grade II	13 (26)
Grade III	16 (32)
Any HIE	39 (78)

Duration of resuscitation and severity of HIE

There was a significant association between longer duration of IPPR and higher HIE grade. Neonates requiring more than 120 seconds of IPPR had a greater proportion of severe HIE compared with those requiring shorter resuscitation. The reported p value was 0.0012.

Duration of NICU stay

The duration of NICU admission increased with increasing severity of HIE. The reported mean duration of stay was 7.25 ± 4.67 days among neonates without HIE, 7.6 ± 1.6 days among those with grade I HIE, 16 ± 4.43 days among those with grade II HIE and 12 ± 9.6 days among those with grade III HIE.

Mortality

Among 50 neonates with severe birth asphyxia, 21 (42%) died and 29 (58%) survived. Among neonates with HIE, 20/39 (51.28%) died. The highest mortality was observed among neonates with severe HIE.

Neurological outcome

Among survivors available for follow-up at 3 months, 29 had normal neurological development while 12 had abnormal neurological findings. The study identified higher-grade HIE, persistent

neurological abnormalities during the first week of life and low Apgar score at 10 minutes as important predictors of adverse neurological outcome.

The source study reported that all infants with HIE grade III had abnormal neurological outcomes, whereas infants without HIE and those with grade I HIE had comparatively favourable neurological outcomes.

Predictors of adverse neurological outcome

Multivariable analysis showed that abnormal neurological findings at day 7, Apgar score <6 at 10 minutes and HIE grade II or higher were significantly associated with adverse neurological outcome, with the reported overall significance of p=0.01.

Predictors of mortality

Difficult-to-control seizures, multiorgan dysfunction and Apgar score <4 at 10 minutes were identified as clinical risk factors associated with mortality. The original summary reports statistical significance for these factors.

DISCUSSION

Perinatal asphyxia continues to represent an important cause of neonatal mortality and neurological morbidity. In the present cohort of full-term neonates with severe birth asphyxia, HIE developed in 78% of infants and mortality was substantial. Among survivors, a considerable proportion had abnormal neurological findings at 3 months. These findings highlight the serious short-term consequences of severe neonatal hypoxic-ischaemic injury. Previous studies have similarly demonstrated that perinatal asphyxia is associated with significant neonatal mortality and neurological morbidity.[22–25]

The incidence of severe birth asphyxia reported in this study was 34 per 1,000 live births, with an incidence of approximately 27 per 1,000 full-term live births. Although the incidence of perinatal asphyxia varies considerably between populations depending on case definition, obstetric services, referral patterns and availability of neonatal resuscitation, the burden remains higher in many low- and middle-income settings.[26–28]

In the present study, 78% of neonates developed clinically evident HIE. This high proportion is understandable because the study specifically enrolled neonates with severe birth asphyxia rather than an unselected population of all newborns. Among affected neonates, grade III HIE constituted the largest single category, accounting for 32% of the entire cohort. The concentration of severe encephalopathy in this population has important prognostic implications. Previous studies have demonstrated a strong relationship between the severity of HIE and subsequent neurological outcome.[29–31]

The relationship between HIE severity and outcome was particularly striking. The study demonstrated that severe grades of HIE were associated with adverse neurological outcomes and mortality. Grade III HIE was associated with particularly poor outcome. This observation is consistent with the established prognostic value of Sarnat staging, where increasing severity of encephalopathy corresponds to increasing risks of mortality and neurodevelopmental impairment.[29–31]

Low Apgar scores at 10 minutes were another important predictor of poor outcome. In the present study, Apgar <6 at 10 minutes was associated with adverse neurological outcome, while Apgar <4 at 10 minutes was associated with mortality. The Apgar score should not be interpreted as an isolated diagnostic test for intrapartum hypoxia; however, persistent severe depression despite resuscitation may provide useful prognostic information when considered together with neurological examination and other clinical findings.[32–34]

Duration of resuscitation was also associated with severity of HIE. Neonates requiring longer IPPR had a greater likelihood of severe encephalopathy, and the association was statistically significant (p=0.0012). Prolonged need for assisted ventilation may reflect the severity and duration of the initial insult and therefore serves as an easily available clinical marker of risk.[35,36]

Seizures were observed in 54% of neonates. Difficult-to-control seizures were associated with mortality in the present cohort. Neonatal seizures are clinically important because they may reflect significant cerebral injury and can contribute to secondary neuronal injury. Previous studies have similarly reported an association between neonatal seizures and neurological outcome.[37]

Multiorgan dysfunction was observed in 32% of neonates and was associated with mortality. Severe hypoxia-ischaemia is a systemic insult that can affect the brain, kidneys, cardiovascular system, lungs, liver and coagulation system.[38,39] These findings emphasize that management of severe birth asphyxia must extend beyond neurological monitoring to comprehensive multisystem supportive care.

The present study also found that 70% of mothers had not received adequate antenatal care according to the study's predefined criterion. Although the study was not designed to establish a causal relationship between inadequate antenatal care and birth asphyxia, this finding highlights the potential importance of antenatal surveillance and identification of high-risk pregnancies. Previous studies have identified inadequate antenatal care and obstetric risk factors among potentially preventable contributors to neonatal encephalopathy.[40,41]

The duration of NICU stay increased among neonates with HIE, particularly among those with moderate or severe encephalopathy. Prolonged hospitalization represents both a marker of disease severity and an important healthcare burden. Severe HIE was accompanied by increased need for

intensive supportive care, including ventilation and management of systemic complications.[38,39]

At 3 months, 29 surviving infants had normal neurological assessments, whereas 12 had abnormal findings. The presence of abnormal neurological findings at the seventh day of life was independently associated with adverse neurological outcome. This observation is clinically relevant because neurological examination remains a low-cost and readily available method of identifying infants requiring closer follow-up. Previous studies have demonstrated the prognostic value of early neurological assessment in term infants with hypoxic-ischaemic injury.[30,42]

The findings have important implications for neonatal care in tertiary hospitals. Early identification of neonates with persistent low Apgar scores, prolonged resuscitation, higher-stage HIE, seizures and multiorgan dysfunction can help clinicians identify infants at increased risk of mortality or neurological impairment. Such infants may require closer neurological monitoring, appropriate respiratory and cardiovascular support, early seizure management, developmental surveillance and structured high-risk follow-up.[29–31,37]

Therapeutic hypothermia is an important neuroprotective intervention for appropriately selected term and near-term infants with moderate-to-severe HIE when initiated within the therapeutic window. Evidence from randomized trials has demonstrated that therapeutic hypothermia improves survival without disability and reduces neurodevelopmental injury in appropriately selected infants.[43–45]

However, the current study primarily evaluated clinical prognostic variables and did not systematically incorporate cord blood pH, base deficit, continuous EEG or MRI findings. The absence of these parameters limits the ability to compare clinical predictors with biochemical, electrophysiological and neuroimaging markers. MRI and EEG can provide additional prognostic information in neonates with HIE.[46–48]

The study has several strengths. It was prospective, included consecutive eligible neonates and followed infants beyond discharge for early neurological assessment. The use of clinically relevant parameters such as Apgar scores, resuscitation duration, HIE staging, seizures, multiorgan dysfunction and neurological examination makes the findings applicable to routine neonatal practice.

There are also important limitations. Only full-term neonates with birth weight ≥1,800 g were included, limiting generalizability to preterm and very-low-birth-weight infants. Severe birth asphyxia was defined using the clinical Apgar-based criterion without universal confirmation by cord blood gas analysis. EEG and MRI were not systematically performed, and follow-up was limited to 3 months. Therefore, the neurological findings should be interpreted as early neurological outcomes rather than definitive long-term neurodevelopmental outcomes. A further issue requiring correction before publication is the discrepancy in sample size between the detailed results (n=50) and the summary section (n=45). This discrepancy should be reconciled against the original database and case records before submission. The final manuscript, abstract, tables, statistical analysis and flow diagram should all report the same verified sample size.

CONCLUSION

Severe birth asphyxia among full-term neonates was associated with a high frequency of hypoxic-ischaemic encephalopathy, substantial multisystem morbidity and considerable mortality. Higher-grade HIE, persistent neurological abnormalities during the first week of life and low Apgar scores at 10 minutes were important predictors of adverse neurological outcome. Difficult-to-control seizures and multiorgan dysfunction were associated with increased mortality.

Routine clinical assessment, including serial neurological examination, documentation of Apgar scores and duration of resuscitation, and early identification of seizures and organ dysfunction, may assist in identifying neonates at high risk of adverse outcomes. These infants require intensive monitoring during hospitalization and structured neurodevelopmental follow-up after discharge.

Future multicentre prospective studies incorporating cord blood gases, EEG, MRI and longer-term neurodevelopmental assessment are needed to improve prognostic accuracy and determine the long-term burden of hypoxic-ischaemic injury.

Limitations

1. The study included only full-term neonates and therefore the findings cannot be generalized to preterm infants.
2. Infants with birth weight <1,800 g were excluded.
3. Severe birth asphyxia was defined primarily using the Apgar score rather than a combination of clinical and biochemical criteria.
4. Cord blood pH and base deficit were not systematically available.
5. EEG and MRI findings were not available for all neonates.
6. The follow-up period was limited to 3 months and therefore does not establish long-term neurodevelopmental outcome.
7. The study was conducted at a single tertiary-care centre, which may limit generalizability.
8. There is an inconsistency between the reported sample size of 45 in the original summary and 50 in the detailed results; this must be resolved from the original dataset before submission.

Ethics approval

The study was approved by the Institutional Ethics Committee of the study institution.

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