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Hepatic enzyme alterations as prognostic markers in neonates with hypoxic-ischemic encephalopathy: A hospital-based prospective cohort study.

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ABSTRACT

Background
Perinatal asphyxia remains a significant contributor to neonatal morbidity and mortality, with hypoxic-ischemic encephalopathy (HIE) being a major sequela. Liver injury is a common systemic complication, and hepatic enzymes may serve as potential biomarkers for the severity of hepatic encephalopathy (HIE).
Objectives: To assess the pattern of hepatic enzyme alterations in neonates with HIE and explore their prognostic value in stratifying disease severity.

Methods
This prospective cohort study enrolled 100 term neonates with perinatal asphyxia. HIE staging was performed using the modified Sarnat and Sarnat criteria. Liver function tests, including serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP), were measured on Days 1 and 3. Statistical analysis included ANOVA, post-hoc comparisons, and Chi-square tests to evaluate associations with clinical outcomes.

Results
Among the neonates, 33% had HIE Stage I, 39% Stage II, and 28% Stage III. Serial measurements showed significant increases in SGOT, SGPT, and ALP from Day 1 to Day 3 across all HIE stages ($p<0.001$). Enzyme elevations were most marked in Stage III. Cut-off values for predicting Stage III HIE were SGOT >77.4 U/L, SGPT >90.4 U/L, and ALP >257.1 U/L. Seizure activity ($p<0.001$) and mode of resuscitation ($p<0.001$) showed statistically significant associations with HIE severity, while maternal risk factors ($p=0.72$), mode of delivery ($p=0.64$), and place of delivery ($p=0.59$) did not.

Conclusion
Serial hepatic enzyme levels are reliable biochemical indicators for assessing the severity of HIE. Their prognostic relevance supports early stratification and targeted management in neonatal intensive care units.

Recommendations
Regular monitoring of hepatic enzymes in neonates with perinatal asphyxia can aid early detection and management of HIE severity.

Keywords: *Hypoxic-ischemic encephalopathy, Perinatal asphyxia, Serum glutamate oxaloacetate transaminase, Serum glutamate pyruvate transaminase, Alkaline phosphatase, Neonatal prognosis*
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Introduction

Perinatal asphyxia remains a significant contributor to neonatal illness and death, particularly in regions

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with limited healthcare resources. It refers to the inability of a newborn to initiate or maintain spontaneous breathing at birth, frequently resulting in multiple organ system dysfunction, including injury to the brain, liver, kidneys, lungs, and heart [1,2]. Among these complications, hypoxic-ischemic encephalopathy (HIE) is one of the most serious, characterized by abnormal neurological function due to inadequate oxygenation and cerebral blood flow during the perinatal period [4,5].

Globally, the incidence of HIE ranges from 1 to 8 per 1,000 live births, with higher rates reported in low- and middle-income countries. This is largely attributed to gaps in access to skilled birth attendants, timely obstetric intervention, and advanced neonatal resuscitation techniques [4]. Although the central nervous system is primarily affected, hypoxia also leads to systemic injury, commonly involving the liver, kidneys, and cardiovascular system [6]. The liver, due to its dual blood supply and high metabolic activity, is particularly vulnerable to hypoxic damage. As such, hepatic dysfunction may serve as an early marker of systemic oxygen deprivation [6].

While HIE is traditionally assessed using neurological criteria, increasing attention has been directed toward the role of biochemical indicators, such as serum glutamate oxaloacetate transaminase (SGOT/AST), serum glutamate pyruvate transaminase (SGPT/ALT), and alkaline phosphatase (ALP), in detecting hepatic injury in neonates with asphyxia [1,3]. However, there is limited data exploring their sequential changes and predictive value in grading the severity of HIE [2,7]. Additionally, most studies focus predominantly on neurological outcomes, with insufficient exploration of systemic biochemical markers for holistic clinical assessment.

The present study seeks to evaluate serial changes in hepatic enzyme levels in neonates diagnosed with HIE and determine their prognostic utility for clinical staging, thereby aiding early risk stratification and guiding management in neonatal intensive care settings.

Methodology

Study design and setting

This was a prospective cohort study conducted over 12 months, from January 2019 to December 2019. The study was carried out in the Neonatal Intensive Care Unit (NICU) of Niloufer Hospital for Children and Women, Hyderabad, Telangana. It is a major tertiary care referral center in South India, affiliated with Osmania Medical College. The hospital has a dedicated neonatal unit equipped with advanced resuscitation and intensive care facilities, catering to a high volume of perinatal admissions, particularly high-risk deliveries and complicated neonatal cases.

Study population

A total of 100 term neonates (≥37 weeks of gestation) diagnosed with perinatal asphyxia were enrolled. Perinatal asphyxia was defined as an Apgar score <3, delayed cry for 5 mins, need for resuscitation at birth, and/or presence of metabolic acidosis (pH <7.0). Neonates were included if they exhibited clinical features suggestive of hypoxic-ischemic encephalopathy (HIE) within the first 6 hours of life.

Study size

A total of 100 term neonates were enrolled based on consecutive sampling during the study period. The sample size was guided by feasibility considerations, incidence rates of perinatal asphyxia in the hospital, and previous literature reporting sufficient statistical power for detecting significant enzyme variations across HIE stages with a sample size of 80–100.

Exclusion criteria

Neonates with congenital anomalies, inborn errors of metabolism, neonatal sepsis, birth trauma, or pre-existing liver disorders were excluded to avoid confounding factors.

Staging of HIE

The severity of HIE was assessed and classified using the modified Sarnat and Sarnat scoring system, which evaluates consciousness, muscle tone, reflexes, and autonomic function. Participants were categorized into Stage I (mild), Stage II (moderate), or Stage III (severe) HIE.

Data collection and biochemical assessment

Demographic and clinical data, including maternal obstetric history, mode of delivery, Apgar scores, and need for resuscitation, were recorded. Liver function tests (LFTs) were performed on Day 1 (within 24 hours) and Day 3 of life. The following biochemical markers were measured: SGOT (AST), SGPT (ALT), Alkaline Phosphatase (ALP), Total Serum Bilirubin (TSB), Indirect and Direct Bilirubin. Blood samples were analyzed using standard automated chemistry analyzers in the hospital laboratory.

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Bias and minimization strategies

To minimize selection bias, consecutive term neonates meeting the inclusion criteria were enrolled prospectively. Diagnostic and staging assessments were standardized using the modified Sarnat and Sarnat scoring system by trained pediatricians blinded to biochemical results. Laboratory analyses were performed using automated equipment with quality control checks to reduce measurement bias. Confounding variables such as sepsis, metabolic disorders, and congenital anomalies were excluded through strict eligibility criteria.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as percentages. Differences in enzyme levels between HIE stages and over time were analyzed using ANOVA, followed by post-hoc Tukey's test for multiple comparisons. Associations between categorical variables were assessed using the Chi-square test. A p-value <0.05 was considered statistically significant.

Ethical consideration

Ethical approval was obtained from the Institutional Ethics Committee of Niloufer Hospital for Children and Women, Osmania Medical College, Hyderabad, before commencement. Written informed consent was obtained from the parents or legal guardians of all neonates included in the study.

RESULTS

Participant flow

A total of 118 term neonates with suspected perinatal asphyxia were admitted to the NICU during the study period (January–December 2019). Of these, 110 were examined for eligibility. Ten neonates were excluded, 3 due to congenital anomalies, 2 due to confirmed neonatal sepsis, 1 due to metabolic disorder, and 4 due to incomplete clinical or biochemical data. The remaining 100 neonates met the inclusion criteria and were enrolled in the study. All 100 participants completed both Day 1 and Day 3 hepatic enzyme assessments, and their data were included in the final analysis. No participants were lost to follow-up or withdrew consent.

A total of 100 term neonates diagnosed with perinatal asphyxia were enrolled in the study. Of these, 53 (53%) were male and 47 (47%) were female. According to the modified Sarnat and Sarnat classification, 33% of neonates were classified as Hypoxic-Ischemic Encephalopathy (HIE) Stage I, 39% as Stage II, and 28% as Stage III (Table 1).

Table 1: Demographic and HIE classification of study participants

Variable	Number (%)
Total Neonates	100 (100%)
Male	53 (53%)
Female	47 (47%)
HIE Stage I	33 (33%)
HIE Stage II	39 (39%)
HIE Stage III	28 (28%)

Demographic and clinical characteristics

Among the mothers, 70% were primigravida while 30% were multigravida. In terms of delivery mode, 66% of neonates were delivered vaginally, and 34% via lower segment cesarean section (LSCS). Pregnancy-induced hypertension was the most common antenatal risk factor, affecting 44% of mothers, followed by premature rupture of membranes (36%) and antepartum hemorrhage (20%) (Table 2).

Table 2: Maternal and delivery characteristics

Variable	Number (%)
Gravida - Primigravida	70 (70%)
Gravida - Multigravida	30 (30%)
Mode of Delivery - Vaginal	66 (66%)
Mode of Delivery - LSCS	34 (34%)
Pregnancy-Induced Hypertension	44 (44%)
Premature Rupture of Membranes	36 (36%)
Antepartum Hemorrhage	20 (20%)

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Resuscitation and seizure activity

Regarding immediate postnatal management, 58% of neonates responded to tactile stimulation alone, 34% required bag and mask ventilation, and 8% underwent intubation. Seizure activity within the first 24 hours of life was observed in 67% of neonates. Both the mode of resuscitation and seizure occurrence showed significant association with the severity of HIE ($p<0.001$) (Tables 3 and 4).

Table 3: Resuscitation and seizure activity

Variable	Number (%)
Tactile Stimulation	58 (58%)
Bag and Mask Ventilation	34 (34%)
Intubation	8 (8%)
Seizure Activity (within 24 hrs)	67 (67%)

Table 4: Association of variables with HIE severity

Variable	Association with HIE Severity
Mode of Resuscitation	Significant ($p<0.001$)
Seizure Activity	Significant ($p<0.001$)
Maternal Risk Factors	Not Significant
Mode of Delivery	Not Significant
Place of Delivery	Not Significant

Hepatic enzyme dynamics and stage-wise trends

Serial hepatic enzyme assessments revealed significant elevations in SGOT, SGPT, and ALP levels from Day 1 to Day 3 across all HIE stages. In Stage I, mean SGOT increased from 65.7 ± 6.3 to 71.8 ± 10.5 U/L, SGPT from 45.3 ± 4.6 to 49.6 ± 5.4 U/L, and ALP from 145.4 ± 6.7 to 155.7 ± 5.1 U/L. In Stage II, SGOT rose from 64.1 ± 12.1 to 131.1 ± 6.9 U/L, SGPT from 44.9 ± 6.4 to 126.2 ± 10.6 U/L, and ALP from 143.3 ± 6.7 to 343.7 ± 110.0 U/L. The most pronounced enzyme elevations were recorded in Stage III, where SGOT increased from 60.2 ± 8.5 to 177.3 ± 9.5 U/L, SGPT from 47.9 ± 7.4 to 154.5 ± 12.7 U/L, and ALP from 138.1 ± 18.8 to 600.5 ± 23.0 U/L (Table 5).

Table 5: Hepatic enzyme trends by HIE stage

HIE Stage	SGOT Day 1 (U/L)	SGOT Day 3 (U/L)	SGPT Day 1 (U/L)	SGPT Day 3 (U/L)	ALP Day 1 (U/L)	ALP Day 3 (U/L)
Stage I	65.7 ± 6.3	71.8 ± 10.5	45.3 ± 4.6	49.6 ± 5.4	145.4 ± 6.7	155.7 ± 5.1
Stage II	64.1 ± 12.1	131.1 ± 6.9	44.9 ± 6.4	126.2 ± 10.6	143.3 ± 6.7	343.7 ± 110.0
Stage III	60.2 ± 8.5	177.3 ± 9.5	47.9 ± 7.4	154.5 ± 12.7	138.1 ± 18.8	600.5 ± 23.0

Inter-stage enzyme comparisons

On Day 3, mean SGOT, SGPT, and ALP values were significantly different across HIE stages (ANOVA, $p<0.001$). Post-hoc analysis confirmed that neonates in HIE Stage III had significantly higher enzyme levels compared to those in Stages I and II ($p<0.001$).

Statistical correlations and diagnostic indicators

A strong statistical correlation was identified between the severity of HIE and elevated hepatic enzyme levels. SGOT, SGPT, and ALP yielded F-values of 637.2, 685.4, and 304.6, respectively, with all p-values <0.001. Diagnostic thresholds were established for predicting Stage III HIE: SGOT >77.4 U/L, SGPT >90.4 U/L, and ALP >257.1 U/L (Table 6).

Table 6: Statistical correlation summary and cut-off values

Parameter	F-value	p-value	Cut-off for Stage III
SGOT	637.2	<0.001	>77.4 U/L
SGPT	685.4	<0.001	>90.4 U/L
ALP	304.6	<0.001	>257.1 U/L

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Additional associations

While mode of resuscitation and seizure occurrence were significantly associated with the severity of HIE ($p<0.001$), maternal risk factors, mode of delivery, and place of delivery did not show statistically significant associations (**Table 4**)

DISCUSSION

This study investigated hepatic enzyme alterations as potential prognostic markers in term neonates diagnosed with hypoxic-ischemic encephalopathy (HIE), a significant cause of neonatal morbidity and mortality, particularly in low- and middle-income countries. The findings affirm that hepatic dysfunction is a consistent systemic manifestation of perinatal asphyxia and that elevated hepatic enzymes correlate strongly with the severity of HIE.

The distribution of HIE stages in the study population, 33% in Stage I, 39% in Stage II, and 28% in Stage III, is consistent with previous reports from tertiary care NICU settings that documented a higher prevalence of moderate to severe HIE among affected neonates [12,14]. Serial measurements of serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP) revealed significant elevations from Day 1 to Day 3 in all HIE stages, with the most pronounced increases observed in Stage III. These findings support the hypothesis that hepatic enzyme elevation reflects progressive hypoxic injury and provides an indirect measure of systemic compromise [12,14].

The statistical correlations between hepatic enzyme levels and HIE severity were robust, with SGOT, SGPT, and ALP showing highly significant F-values and p-values ($p<0.001$). The diagnostic thresholds identified in this study, SGOT >77.4 U/L, SGPT >90.4 U/L, and ALP >257.1 U/L, demonstrated strong discriminatory power for identifying Stage III HIE. These enzyme cut-offs are consistent with prior findings that suggest hepatic markers can be useful for early prognostic evaluation in neonates with perinatal asphyxia [9,10,13].

Seizure activity within the first 24 hours and the need for advanced resuscitation measures (bag and mask ventilation or intubation) were significantly associated with HIE severity ($p<0.001$), highlighting their utility as early bedside indicators of neurologic injury. These associations reinforce findings from previous clinical studies that demonstrated strong links between neurological signs and biochemical derangements in HIE [13,14]. In contrast, maternal risk factors, mode of delivery, and place of delivery did not show significant associations with HIE severity, an observation supported by systematic reviews that found limited predictive value of antenatal or peripartum variables in determining postnatal neurological outcomes [11].

The findings of this study also align with broader literature proposing liver enzymes as part of a multimodal approach to assessing HIE severity, particularly in neonates undergoing therapeutic hypothermia [8,10]. Although our study population did not receive therapeutic hypothermia, the biochemical trends remain relevant for early diagnosis and risk stratification.

Generalizability

The findings are generalizable to similar tertiary care neonatal intensive care settings in low- and middle-income countries, though extrapolation to rural or resource-limited centers should be cautious due to differences in infrastructure, case-mix, and diagnostic capabilities.

Conclusion

This study demonstrates that hepatic enzyme levels, specifically SGOT, SGPT, and ALP, rise significantly with increasing severity of hypoxic-ischemic encephalopathy in term neonates. The marked elevations observed, especially by Day 3, suggest progressive liver injury corresponding to neurological impairment. Enzyme thresholds identified in this study may serve as early, non-invasive prognostic indicators to assist clinicians in assessing disease severity and guiding management decisions. While seizure activity and mode of resuscitation showed significant associations with HIE staging, maternal and delivery-related factors were not predictive. Routine liver function monitoring in asphyxiated neonates may enhance early risk stratification, particularly in resource-constrained neonatal intensive care settings.

Strengths and limitations

A key strength of this study was the serial assessment of hepatic enzymes, allowing for time-sensitive trend analysis, which is often lacking in cross-sectional evaluations. However, the study is limited by the absence of long-term neurodevelopmental follow-up, which restricts the ability to correlate enzyme trends with cognitive or motor outcomes. Additionally, while liver function tests (LFTs) are economical and widely accessible, incorporating newer biomarkers

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such as lactate, LDH, or imaging modalities may enhance prognostic accuracy in future research.
Recommendations: It is recommended that hepatic enzyme levels, including SGOT, SGPT, and ALP, be routinely monitored in neonates diagnosed with perinatal asphyxia to assess the severity of hypoxic-ischemic encephalopathy (HIE). These enzyme markers can serve as valuable prognostic tools for early identification of severe HIE, allowing for timely interventions and better management strategies in neonatal intensive care units. Additionally, further research should focus on establishing standardized reference ranges for these enzymes across different HIE stages, enhancing their utility in clinical practice for early stratification and individualized care.

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List of abbreviations

HIE - Hypoxic-Ischemic Encephalopathy
SGOT - Serum Glutamate-Oxaloacetate Transaminase
SGPT - Serum Glutamate-Pyruvate Transaminase
ALP - Alkaline Phosphatase
TSB - Total Serum Bilirubin
Apgar - Appearance, Pulse, Grimace, Activity, and Respiration
NICU - Neonatal Intensive Care Unit
LSCS - Lower Segment Cesarean Section
pH - Potential of Hydrogen

Source of funding

The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

EM- Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript. RM-Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript, revision of the manuscript.DV-Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript.BV- Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of the manuscript

Data availability

Data is available on request.

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