

Original Research Article

Incidence of hypoglycemia during the first 48 hours of life in small-for-gestational-age neonates in a tertiary care hospital

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Received: 03 September 2026

Revised: 16 September 2026

Accepted: 17 September 2026

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ABSTRACT

Background: To determine the incidence of hypoglycemia during the first 48 hours of life among small-for-gestational-age (SGA) neonates and compare its occurrence between term and preterm SGA neonates.

Methods: A prospective observational study was conducted among 100 SGA neonates admitted to a tertiary care neonatal intensive care unit. Blood glucose was measured at 1, 2, 4, 6, 12, 24, and 48 hours of life. Maternal and neonatal characteristics, risk factors, feeding practices, clinical manifestations, and outcomes were recorded. Associations with hypoglycemia were assessed using univariate and multivariable logistic regression.

Results: Of 625 newborns enrolled, 100 (16.0%) were SGA; 66 (66.0%) were term and 34 (34.0%) preterm. Hypoglycemia occurred in 26 (26.0%) neonates and was significantly more frequent in preterm than term SGA neonates (47.1% vs. 15.2%, $p < 0.001$). Among hypoglycemic neonates, 16 (61.5%) were symptomatic. The highest frequency of hypoglycemia occurred at 2 hours of life. Delayed initiation of enteral feeding was significantly associated with hypoglycemia. Blood glucose levels differed significantly between neonates weighing < 1.8 kg and those weighing ≥ 1.8 kg at 2 and 4 hours. Univariate analysis identified low birth weight, prematurity, anthropometric measures, caesarean delivery, and 1-minute Apgar score as significant factors. On multivariable analysis, head circumference, chest circumference, 1-minute Apgar score, and caesarean delivery remained significantly associated with hypoglycemia. Two neonates with concomitant sepsis died.

Conclusion: Hypoglycemia was common among SGA neonates, particularly those born preterm. Early glucose monitoring, especially during the first 4 hours, together with timely enteral feeding, may facilitate early detection and prevention of neonatal hypoglycemia.

Keywords: Neonatal hypoglycemia, Small-for-gestational-age neonates, Preterm neonates, Term neonates, Blood glucose, Early feeding, Birth weight, Apgar score, NICU

INTRODUCTION

Neonatal hypoglycemia is one of the most common metabolic disturbances during the early neonatal period and is particularly important among infants at increased risk because severe or recurrent episodes may be associated with neurological dysfunction and adverse neurodevelopmental outcomes.¹ The transition from

intrauterine to extrauterine life involves an abrupt interruption of the continuous placental glucose supply. During the first hours after birth, blood glucose concentrations normally decline as insulin secretion decreases and counter-regulatory mechanisms, including glucagon and catecholamines, stimulate glycogenolysis, gluconeogenesis, and utilization of alternative energy substrates.^{2,3} This physiological adaptation is usually

transient; however, infants with limited energy reserves or impaired metabolic adaptation may develop clinically important hypoglycemia. Preterm SGA neonates may have an additional risk because of greater metabolic immaturity and reduced energy reserves. Consequently, SGA infants are routinely considered a high-risk group for neonatal hypoglycemia and require appropriate postnatal glucose surveillance.^{4,5} The American Academy of Pediatrics recommended operational thresholds for at-risk infants, with intervention thresholds varying according to postnatal age.⁵ In the CHYLD cohort, neonatal hypoglycemia was not associated with overall neurosensory impairment at 4.5 years but was associated with poorer executive and visual-motor function, particularly following severe or recurrent episodes.⁶ A meta-analysis reported associations between neonatal hypoglycemia and later neurodevelopmental impairment, cognitive impairment, visual-motor impairment, and executive dysfunction, although the certainty of evidence was low or very low for most outcomes.⁷

Clinical manifestations of neonatal hypoglycemia are often nonspecific and may include jitteriness, poor feeding, lethargy, apnea, cyanosis, temperature instability, and seizures, while many affected neonates remain asymptomatic.⁸ This clinical variability makes targeted glucose screening important in high-risk infants, particularly SGA and preterm neonates. The timing of glucose monitoring is also relevant because the incidence of hypoglycemia is not uniform throughout the first postnatal day. Early feeding and appropriate nutritional support may help reduce the risk by providing an exogenous glucose source while endogenous metabolic adaptation occurs. Therefore, identifying the period of greatest vulnerability may help optimize monitoring and feeding practices.

Despite the recognized susceptibility of SGA neonates, data describing the incidence, temporal pattern, and associated factors of hypoglycemia during the first 48 hours of life remain variable across populations and clinical settings. Differences in gestational age, birth weight, feeding practices, screening schedules, and operational definitions of hypoglycemia may contribute to this variation. The present study was therefore undertaken to determine the incidence and temporal pattern of hypoglycemia among SGA neonates admitted to a tertiary care hospital and to compare glucose concentrations and the occurrence of hypoglycemia between term and preterm SGA neonates. The study also evaluated maternal, neonatal, feeding, and clinical factors associated with hypoglycemia during the first 48 hours of life.

METHODS

Study design

This was a prospective, hospital-based descriptive and observational study conducted among SGA neonates

admitted to the NICU of the Department of Pediatrics, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad. Study was conducted over a period of 18 months from February 2022 to July 2023.

Study population

The study included 100 term and preterm SGA neonates born at Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, and admitted to the NICU.

The study was conducted in the Department of Pediatrics, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad. The study was initiated after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from the parent or legally authorized guardian of each participating neonate before enrollment.

Sample size calculation

The sample size was calculated based on the reported incidence of neonatal hypoglycemia of 24% among newborns in the study by Shirley et al.³⁴

The sample size was estimated using the following formula.

$$n = DEFF \times Np(1-p) / (d^2 / Z_{1-\alpha/2}^2 \times (N-1) + p(1-p))$$

Based on the assumed prevalence, a 95% confidence level, and the specified precision, the calculated minimum sample size was 76 neonates. To improve the adequacy of the study sample, 100 neonates were ultimately included.

A purposive sampling technique with consecutive recruitment of eligible cases was used for participant selection.

Inclusion criteria

The study included term and preterm SGA neonates aged less than 48 hours who were admitted to the neonatal intensive care unit (NICU) and had a birth weight below the 10th percentile for gestational age according to the Lubchenco growth curve.

Exclusion criteria

Neonates aged more than 48 hours at enrollment, AGA or LGA neonates, and those with birth asphyxia, early-onset sepsis, active-phase illness, polycythemia, or infants born to mothers with diabetes mellitus were excluded from the study.

Data collection

A detailed prenatal, natal, and postnatal history was obtained for each enrolled neonate using a structured

proforma. Maternal and neonatal demographic characteristics, gestational age, birth weight, sex, mode of delivery, indications for neonatal interventions, immediate postnatal events, Apgar scores, relevant risk factors for neonatal hypoglycemia, feeding practices, clinical manifestations, and blood glucose measurements during the first 48 hours of life were recorded prospectively.

Blood glucose estimation

Capillary blood samples were obtained by heel prick. Initial blood glucose estimation was performed using a glucometer and compatible reagent strips according to the manufacturer's instructions. Capillary glucose values below 40 mg/dl obtained by the reagent-strip method were confirmed using a laboratory plasma glucose analyzer on a capillary blood sample.

Blood glucose concentrations were measured before feeding at predefined intervals during the first 48 hours of life. Measurements were obtained at 0, 1, 2, 3, 4, 6, 12, 24, and 48 hours of life. Neonates identified as having hypoglycemia were monitored and managed according to the institutional neonatal hypoglycemia protocol based on American Academy of Pediatrics recommendations.

Definition and clinical assessment of hypoglycemia

For the purpose of the study, hypoglycemia was defined according to the prespecified age-specific operational thresholds as a blood glucose concentration below 40 mg/dL during the first 4 hours of life and below 45 mg/dL from 4 to 24 hours of life. The occurrence, timing, frequency, and severity of hypoglycemic episodes were recorded during the first 48 hours.

Intravenous glucose therapy was initiated when hypoglycemia persisted or adequate correction was not achieved with enteral feeding, according to clinical assessment and institutional protocol.

The mean blood glucose concentration, timing and frequency of hypoglycemic episodes, symptomatic status, requirement for intravenous glucose therapy, clinical course, and neonatal outcome were prospectively recorded. All neonates received standard neonatal care throughout the study period.

Statistical analysis

Data were entered and analyzed using SPSS version 22.0 (SPSS Inc., Chicago, IL, USA). Continuous variables with an approximately normal distribution were expressed as mean±standard deviation (SD). Comparisons between neonates with and without hypoglycemia were performed using the Student's t-test for continuous variables and the Fisher's exact test for categorical variables, as appropriate.

RESULTS

Basic characteristics

Among 625 neonates assessed, 100 (16.0%) were SGA and 525 (84.0%) were AGA; no LGA neonates were identified. Of the 100 SGA neonates, 34 (34.0%) were preterm and 66 (66.0%) were term. Males predominated among both preterm and term SGA neonates (70.6% vs. 54.5%), with a significant difference in sex distribution ($\chi^2=5.71$, $p=0.017$). Maternal gravida also differed significantly between groups ($\chi^2=12.53$, $p=0.004$), with multigravida mothers predominating among preterm SGA neonates (70.6%), whereas primigravida mothers predominated among term SGA neonates (66.7%).

Hypoglycemia occurred in 26 (26.0%) SGA neonates during the first 48 hours, with a significantly higher incidence among preterm than term neonates (47.1% vs. 15.2%, $p<0.001$). Hypoglycemia was not significantly associated with maternal gravida (25.9% vs. 26.1%, $p=0.985$) or neonatal sex (26.7% in males vs. 25.0% in females, $p=0.850$).

Hypoglycemia occurred in 8 (17.4%) vaginally delivered and 18 (33.3%) caesarean-delivered neonates; however, the difference was not statistically significant ($p=0.070$). Among the 26 hypoglycemic SGA neonates, 12 (46.2%) experienced two episodes, 8 (30.8%) had a single episode, and 2 (7.7%) each had three, four, or five episodes. Overall, 16 (61.5%) experienced recurrent hypoglycemia (≥ 2 episodes), with two episodes being the most common pattern.

The number of hypoglycemic episodes did not differ significantly between preterm and term SGA neonates ($\chi^2=1.52$, $p=0.824$). Two episodes were most common, occurring in 8 (50.0%) preterm and 4 (40.0%) term neonates. Among hypoglycemic neonates, 16 (61.5%) were symptomatic and 10 (38.5%) were asymptomatic ($p<0.00001$). Hypoglycemic episodes occurred predominantly within the first 24 hours, with the highest frequency at 2 hours (21 episodes), followed by 1 and 6 hours (9 each) and 12 hours (7). Only 3 episodes occurred at 24 hours and 1 at 48 hours.

Table 3 shows that 78 (78.0%) SGA neonates received their first enteral feed within 1 hour, while 14 (14.0%) were fed at 1–2 hours and 8 (8.0%) after 2 hours. Hypoglycemia occurred in 15.4% of neonates fed within 1 hour compared with 64.3% and 62.5% among those fed at 1–2 hours and >2 hours, respectively. The association between delayed initiation of feeding and hypoglycemia was statistically significant ($\chi^2=20.77$, $p<0.001$).

Table 4 shows that low birth weight (1.501–2.500 kg) was the most common category among both term and preterm SGA neonates (75.8% and 64.7%, respectively), while extremely low birth weight was more frequent among preterm neonates (17.7% vs. 3.0%). Among

hypoglycemic SGA neonates, birth-weight distribution did not differ significantly between preterm and term groups ($p=0.176$). Mean blood glucose increased progressively from 40.5 ± 6.1 mg/dl at 2 hours to

61.6 ± 10.3 mg/dl at 24–48 hours, indicating lower glucose levels during the early neonatal period followed by a gradual increase.

Table 1: Association of neonatal hypoglycemia with mode of delivery.

Mode of delivery	Hypoglycemic, N (%)	Normoglycemic, N (%)	Total, N (%)	P value
Vaginal delivery	8 (17.39)	38 (82.61)	46 (100.0)	0.070
Caesarean delivery	18 (33.33)	36 (66.67)	54 (100.0)	
Total	26 (26.0)	74 (74.0)	100 (100.0)	

Table 2: Distribution of hypoglycemic episodes according to gestational maturity (n=26).

Number of episodes	Preterm SGA (n=16), N (%)	Term SGA (n=10), N (%)	χ^2	P value
1	4 (25.0)	4 (40.0)	1.5163	0.8237
2	8 (50.0)	4 (40.0)		
3	0 (0.0)	2 (20.0)		
4	2 (12.5)	0 (0.0)		
5	2 (12.5)	0 (0.0)		
Total	16 100.0)	3 100.0)		

Table 3; Timing of initiation of enteral feeding and association with hypoglycemia. Timing of first enteral feed among all SGA neonates.

Time of initiation (in hours)	Frequency	%
Within 1 hour	78	78.0
1–2	14	14.0
2–3	5	5.0
3–6	2	2.0
6–10	1	1.0
Total	100	100.0

Table 4: Timing of initiation of enteral feeding and association with hypoglycemia association between timing of feeding and hypoglycemia.

Time of initiation (in hours)	Total	Hypoglycemia, N (%)	No hypoglycemia, N (%)
<1	78	12 (15.39)	66 (84.61)
1–2	14	9 (64.29)	5 (35.71)
>2	8	5 (62.50)	3 (37.50)
Total	100	26 (26.0)	74 (74.0)

Table 5: Birth weight distribution according to gestational maturity.

Birth-weight category	Term SGA (n=66), N (%)	Preterm SGA (n=34), N (%)	P value
Extremely low birth weight (≤ 1.0 kg)	2 (3.03)	6 (17.65)	0.0385
Very low birth weight (1.001–1.500 kg)	14 (21.21)	6 (17.65)	
Low birth weight (1.501–2.500 kg)	50 (75.76)	22 (64.71)	
Total	66 (100.0)	34 (100.0)	

Table 5 compares blood glucose levels between preterm and term SGA neonates across different time intervals during the first 48 hours. At 2 hours, mean glucose was lower among preterm neonates than term neonates (38.2 ± 3.6 vs. 41.9 ± 6.7 mg/dl), although the difference

was not statistically significant ($p=0.07$). At 4 hours, glucose levels were also slightly lower in preterm neonates (51.7 ± 8.3 vs. 53.3 ± 6.5 mg/dl; $p=0.6$). Conversely, mean glucose was marginally higher in preterm neonates at 12–24 hours (58.3 ± 4.7 vs. 56.3 ± 8.3

mg/dl; $p=0.4$) and slightly lower at 24–48 hours (60.5 ± 6.8 vs. 62.3 ± 10.1 mg/dl; $p=0.59$). Table 6 shows that neonates weighing <1.8 kg had significantly lower mean blood glucose than those weighing ≥ 1.8 kg at 2

hours (39.6 ± 6.8 vs. 43.7 ± 8.2 mg/dl, $p=0.018$) and 4 hours (45.7 ± 5.9 vs. 60.1 ± 4.5 mg/dl, $p<0.0001$). No significant differences were observed at 12–24 hours ($p=0.190$) or 24–48 hours ($p=0.857$).

Table 6: Comparison of blood glucose levels between preterm and term SGA neonates.

Time interval	Preterm SGA (n=16), Mean $\pm$ SD (mg/dl)	Term SGA (n=10), Mean $\pm$ SD (mg/dl)	P value
2 hours	38.2 $\pm$ 3.6	41.9 $\pm$ 6.7	0.07
4 hours	51.7 $\pm$ 8.3	53.3 $\pm$ 6.5	0.60
12–24 hours	58.3 $\pm$ 4.7	56.3 $\pm$ 8.3	0.43
24–48 hours	60.5 $\pm$ 6.8	62.3 $\pm$ 10.1	0.59

Table 7: Blood glucose levels according to birth weight.

Birth weight	N	2 hours, Mean $\pm$ SD	4 hours, Mean $\pm$ SD	12–24 hours, Mean $\pm$ SD	24–48 hours, Mean $\pm$ SD
<1.8 kg	30	39.6 $\pm$ 6.8	45.7 $\pm$ 5.9	55.8 $\pm$ 7.2	63.0 $\pm$ 8.2
≥ 1.8 kg	70	43.7 $\pm$ 8.2	60.1 $\pm$ 4.5	57.6 $\pm$ 5.8	63.4 $\pm$ 10.9
P value		0.018	<0.0001	0.190	0.857

Table 8: Blood glucose distribution among preterm and term SGA neonates.

Blood glucose level (mg/dl)	Total SGA (n=100), N (%)	Preterm SGA (n=34), N (%)	Term SGA (n=66), N (%)
<30	4 (4.0)	4 (11.76)	0 (0.0)
30–39	16 (16.0)	6 (17.65)	10 (15.15)
40–49	14 (14.0)	6 (17.65)	8 (12.12)
50–59	56 (56.0)	16 (47.05)	40 (60.61)
≥ 60	10 (10.0)	2 (5.89)	8 (12.12)
Total	100 (100.0)	34 (100.0)	66 (100.0)

Table 9: Comparison of hypoglycemic and normoglycemic SGA neonates according to selected neonatal factors.

Neonatal factor	Hypoglycemic (n=26)	Normoglycemic (n=74)	OR (95% CI)	P value
Meconium aspiration	1	3	2.5 (0.75–8.5)	0.45
Transient tachypnea of newborn	4	1	1.2 (0.45–3.9)	0.37
Sepsis	2	0	—	—
Oral feeds by 1 hour	2	5	0.35 (0.15–0.80)	0.50
Oral feeds by 2 hours	3	8	0.64 (0.20–2.02)	0.42

Table 10: Blood glucose levels among hypoglycemic SGA neonates according to birth-weight category and gestational maturity.

Birth-weight category	Preterm SGA, Mean $\pm$ SD	Term SGA, Mean $\pm$ SD	P value
Extremely low birth weight (≤ 1.0 kg)	40.2 $\pm$ 3.6	42.9 $\pm$ 6.7	0.1924
Very low birth weight (1.001–1.500 kg)	53.7 $\pm$ 8.3	55.3 $\pm$ 6.5	0.6098
Low birth weight (1.501–2.500 kg)	57.3 $\pm$ 4.7	58.3 $\pm$ 8.3	0.6971

Early-fed neonates also had higher mean glucose levels than delayed-fed neonates (46.2 ± 7.9 vs. 42.0 ± 5.9 mg/dl, $p=0.0036$). Table 8 shows that the most common blood glucose range among SGA neonates was 50–59 mg/dl, observed in 56 (56.0%) neonates, followed by 30–39 mg/dl in 16 (16.0%) and 40–49 mg/dl in 14 (14.0%).

Glucose levels <30 mg/dl were observed in 4 (4.0%) neonates, all of whom were preterm, while glucose levels ≥ 60 mg/dl were observed in 10 (10.0%) neonates. Among preterm SGA neonates, 17.65% had glucose levels <30 mg/dl and 17.65% had levels of 30–39 mg/dl, compared with 0% and 15.15%, respectively, among term SGA neonates. Selected neonatal factors were not significantly

associated with hypoglycemia. Meconium aspiration, transient tachypnea, and feeding by 1 or 2 hours showed no statistically significant associations (all $p > 0.05$). Sepsis occurred in 2 hypoglycemic neonates and none of the normoglycemic neonates, precluding calculation of an OR and p value. Among hypoglycemic SGA neonates, NICU stay >3 days was observed in 70.0% of term and 75.0% of preterm neonates, with no significant difference ($p = 0.780$). Treatment duration was similar between term and preterm SGA neonates ($p = 0.922$). Among term neonates, 30.0% required treatment for <72 hours and 70.0% for 4–7 days, while among preterm neonates, 31.3%, 56.3%, and 12.5% required treatment for <72 hours, 4–7 days, and >7 days, respectively. Among hypoglycemic SGA neonates, discharge rates were 100% for low birth weight, 90.0% for very low birth weight, and 83.3% for extremely low birth weight, with no significant difference in outcome across birth-weight categories ($p = 0.885$). Overall, 24 (92.3%) were discharged and 2 (7.7%) died.

Similarly, outcomes did not differ significantly between preterm and term neonates (93.8% vs. 90.0% discharged; $p = 0.727$). Mean blood glucose was higher among discharged than deceased preterm neonates (59.2 ± 3.6 vs. 40.0 ± 0.0 mg/dl, $p < 0.0001$), whereas no significant difference was observed among term neonates (56.7 ± 8.3 vs. 52.0 ± 0.0 mg/dl, $p = 0.469$). Mean blood glucose increased with longer treatment duration in both term and preterm SGA neonates, from 46.5 ± 1.2 to 62.8 ± 10.9 mg/dl in term neonates and from 45.2 ± 2.1 to 67.8 ± 2.8 mg/dl in preterm neonates.

Among the 25 hypoglycemic SGA neonates assessed during follow-up, 24 (96.0%) had normal neurodevelopment at 3 months, with one preterm neonate showing an abnormal assessment. At 6 months, all 24 assessed neonates had normal neurodevelopment. These findings should be interpreted cautiously given the small follow-up sample and short follow-up duration.

DISCUSSION

In the present study, 625 neonates were assessed, of whom 100 (16.0%) were SGA. Hypoglycemia occurred in 26 (26.0%) SGA neonates during the first 48 hours of life. This finding is comparable with previous studies of SGA neonates. Narang et al reported hypoglycemia in 25.2% of 127 SGA neonates, while Shirley et al. reported an incidence of 24% among 100 SGA neonates. Behera et al also reported a similar incidence in a larger prospective cohort.^{9,10} The variation in reported incidence across studies may reflect differences in the operational definition of hypoglycemia, timing and frequency of glucose screening, gestational-age composition, feeding practices, and exclusion or inclusion of clinically ill neonates.

For example, an older study of term SGA infants reported an incidence of 14.7%, whereas Lubchenco et al and

Bard et al reported substantially higher rates, particularly among preterm SGA infants.¹¹ The incidence of hypoglycemia among term SGA neonates in the present study (15.2%) was close to the 14.7% reported by Holtrop et al and lower than the 25–26% reported in some earlier studies.¹² Differences may be attributable to variation in screening protocols, glucose thresholds, feeding practices, and study populations.

Hypoglycemia in the present study occurred predominantly during the early neonatal period, with the highest number of episodes at 2 hours of life, followed by 1 and 6 hours. Most affected neonates experienced hypoglycemia within the first 24 hours; only one preterm neonate had an episode during 24–48 hours. These findings are consistent with previous studies. Narang et al reported that approximately 98% of hypoglycemic episodes in SGA neonates occurred during the first 24 hours, while Behera et al reported approximately 94%. A study by Shirley et al similarly found the highest frequency at 2 hours of life, with progressively fewer episodes thereafter. Among the 26 hypoglycemic neonates in the present study, recurrent hypoglycemia was relatively common: 16 (61.5%) experienced two or more episodes, with two episodes being the most frequent pattern. However, the number of episodes did not differ significantly between preterm and term SGA neonates ($p = 0.824$). These findings emphasize that a single normal glucose measurement does not exclude subsequent hypoglycemia in an SGA infant.

In the present study, 16 (61.5%) of the 26 hypoglycemic neonates were symptomatic, whereas 10 (38.5%) were asymptomatic. The substantial proportion of asymptomatic cases supports the importance of glucose surveillance in SGA neonates because clinical manifestations of neonatal hypoglycemia are often nonspecific and may be absent. Previous studies have similarly demonstrated that a considerable proportion of neonatal hypoglycemia is clinically silent.

In contrast, Shirley et al reported a lower proportion of symptomatic cases, with 9% of the total SGA cohort being symptomatic.¹³ Early initiation of enteral feeding was an important finding in the present study. Seventy-eight percent of SGA neonates received their first enteral feed within 1 hour. Hypoglycemia occurred in 15.4% of those fed within 1 hour compared with 64.3% among those fed at 1–2 hours and 62.5% among those fed after 2 hours ($\chi^2 = 20.77$, $p < 0.001$). Similarly, mean blood glucose was significantly higher in the early-feeding group than in the delayed-feeding group (46.2 ± 7.9 vs. 42.0 ± 5.9 mg/dl, $p = 0.0036$).

These findings are consistent with Shirley et al who reported significantly lower hypoglycemia among SGA neonates whose enteral feeds were initiated within the first hour. Neonates weighing <1.8 kg had significantly lower mean glucose concentrations than those weighing ≥ 1.8 kg at 2 hours and 4 hours of life, whereas the

difference was no longer significant at 12–24 or 24–48 hours. This suggests that lower birth weight may be particularly relevant to glucose homeostasis during the early transitional period. The progressive increase in mean glucose concentration observed in the present study from 40.5 ± 6.1 mg/dl at 2 hours to 61.6 ± 10.3 mg/dl at 24–48 hours—is consistent with the expected postnatal transition toward more stable endogenous glucose production and enteral energy supply.

Hypoglycemia was more frequent among neonates delivered by caesarean section than vaginally (33.3% vs. 17.4%); however, this difference was not statistically significant ($p=0.070$). Therefore, the unadjusted findings do not establish a significant association between mode of delivery and hypoglycemia. Shirley et al. likewise found no significant association between mode of delivery and hypoglycemia in SGA neonates. Most hypoglycemic SGA neonates survived to discharge, with 24 (92.3%) discharged and 2 (7.7%) deaths. Mortality did not differ significantly according to gestational maturity or birth-weight category. The two deaths occurred in neonates with sepsis; therefore, the association between hypoglycemia and mortality should not be interpreted as causal, particularly given the very small number of deaths.

Among the 25 hypoglycemic SGA neonates assessed during follow-up, 24 (96.0%) had normal neurodevelopment at 3 months and one preterm neonate had an abnormal assessment. At 6 months, all 24 assessed neonates had normal neurodevelopmental findings. These results are reassuring but should be interpreted cautiously because of the small follow-up sample, short follow-up duration, absence of a suitable comparison group, and loss to follow-up. Existing evidence regarding the long-term neurodevelopmental effects of neonatal hypoglycemia is heterogeneous, and the relationship appears to depend on the severity, duration, and recurrence of hypoglycemia rather than on a single glucose value.

In the present study, univariate analysis identified low birth weight, prematurity, head circumference, chest circumference, length, Apgar score, and caesarean delivery as variables associated with hypoglycemia. In multivariable analysis, head circumference, chest circumference, 1-minute Apgar score, and caesarean delivery remained associated with hypoglycemia. These findings are broadly consistent with the study by Wang et al in which caesarean delivery, smaller head and chest circumference, and lower 1-minute Apgar score were identified as significant risk factors for early hypoglycemia among term and late-preterm SGA neonates.¹⁴

Limitations

The study has several limitations. The sample size was relatively small, the study was conducted at a single

tertiary-care center, and follow-up was limited to 3 and 6 months. The study therefore cannot determine long-term neurodevelopmental consequences of neonatal hypoglycemia. In addition, the observational design limits causal inference, particularly for feeding practices, mode of delivery, and clinical outcomes. The study evaluated glucose homeostasis during the first 48 hours; therefore, the findings should not be extrapolated to hypoglycemia occurring after this period. Differences in screening frequency and operational glucose thresholds also limit direct comparison with studies using different protocols.

CONCLUSION

Hypoglycemia was common among SGA neonates during the first 48 hours of life, with a significantly higher incidence among preterm than term SGA neonates. Most episodes occurred within the first 24 hours, with the highest frequency at 2 hours of life. Delayed initiation of enteral feeding and lower birth weight were associated with lower early glucose concentrations, while a substantial proportion of hypoglycemic neonates were asymptomatic. These findings support early and routine glucose monitoring, particularly in preterm and lower-birth-weight SGA neonates, along with timely initiation of enteral feeding.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Molugu CR, Reddy MH, Manikanta R. Incidence of hypoglycemia during the first 48 hours of life in small-for-gestational-age neonates in a tertiary care hospital. *Int J Contemp Pediatr* 2026;13:xxx-xx.