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Original Article

Incidence of Asymptomatic Hypoglycemia in Full-Term Low-Birth-Weight Babies Versus Full-Term Normal-Birth-Weight Babies

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ABSTRACT

Background: Early neonatal hypoglycemia is a common metabolic problem and may remain clinically silent, particularly in neonates with reduced metabolic reserves. Full-term low-birth-weight neonates may be vulnerable despite term gestation, but local comparative data remain limited. Objective: To compare the incidence of asymptomatic hypoglycemia during the first four hours of life between full-term low-birth-weight and full-term normal-birth-weight neonates. Methods: This prospective comparative cohort study was conducted in the Department of Pediatrics, Ali Fatima Hospital, Lahore, Pakistan, from September 12, 2024, to March 12, 2025. Sixty full-term neonates were enrolled by consecutive sampling and divided into low-birth-weight and normal-birth-weight groups, with 30 neonates in each group. Blood glucose was measured by heel-prick capillary sampling during the first four hours of life. Asymptomatic hypoglycemia was defined as blood glucose <55 mg/dL. Data were analyzed using SPSS version 25. Results: The mean gestational age was 37.73 ± 0.75 weeks, and the mean birth weight was 2.59 ± 0.41 kg. Overall, asymptomatic hypoglycemia occurred in 21 neonates (35.0%). Hypoglycemia was more frequent in low-birth-weight neonates than in normal-birth-weight neonates, occurring in 16/30 (53.3%) and 5/30 (16.7%), respectively. The crude relative risk was 3.20, with an absolute risk difference of 36.7 percentage points. Conclusion: Full-term low-birth-weight neonates had a higher incidence of early asymptomatic hypoglycemia than normal-birth-weight neonates, supporting early glucose screening in this risk group. Keywords: Low Birth Weight; Normal Birth Weight; Full-Term Neonates; Neonatal Hypoglycemia; Asymptomatic Hypoglycemia; Blood Glucose Screening.

INTRODUCTION

Low birth weight remains a major public health concern worldwide and is an important determinant of neonatal survival, growth, and neurodevelopment, particularly in low- and middle-income countries where preventable neonatal morbidity remains high (1,2). In Pakistan, the reported frequency of low birth weight among term neonates varies considerably across local settings, ranging from 8.9% to 26.9%, reflecting differences in maternal health, antenatal care, socioeconomic status, nutritional status, and perinatal service quality (3–5). Although low birth weight is often discussed alongside small-for-gestational-age status, the two are not identical; low birth weight is defined by an absolute birth weight below 2500 g, whereas small-for-gestational-age status requires comparison with gestational-age-specific birth-weight percentiles. This distinction is clinically important because full-term neonates with low birth weight may have limited metabolic reserve even when classified using an absolute weight threshold, placing them at higher risk of early neonatal metabolic instability.

Glucose is the principal energy substrate for the fetus and newborn, and fetal glucose homeostasis depends largely on continuous transplacental transfer from the mother during intrauterine life (6–8). At birth, clamping of the umbilical cord abruptly interrupts this continuous maternal glucose supply, requiring the neonate to maintain glucose levels through glycogenolysis, gluconeogenesis, lipolysis, ketogenesis, and early enteral feeding (7–9). This metabolic transition may be challenging in neonates with low birth weight because they often have reduced glycogen stores, lower fat reserves, immature counter-regulatory responses, and higher vulnerability to feeding delay or perinatal stress. Consequently, hypoglycemia may occur during the early postnatal period, and because many affected neonates remain asymptomatic, clinical recognition based on signs alone may be insufficient.

Neonatal hypoglycemia is one of the most frequently encountered metabolic problems in neonatal care and may be associated with adverse neurological outcomes when prolonged, recurrent, or severe (6,10,11). However, the operational definition of neonatal hypoglycemia remains debated, and different thresholds have been used across studies and clinical guidelines (11). Despite this variation, early glucose monitoring is commonly recommended for neonates with recognized risk factors, including term and late-preterm small-for-gestational-age infants, neonates with low birth weight, infants of diabetic mothers, and newborns exposed to perinatal stress (10). Previous studies have reported a meaningful association between low birth weight or growth restriction and neonatal hypoglycemia, but reported incidence varies according to study population, timing of glucose measurement, feeding practices, diagnostic threshold, and whether glucose was measured once or serially (12–18).

Local evidence comparing asymptomatic hypoglycemia between full-term low-birth-weight and normal-birth-weight neonates remains limited, particularly in hospital-based neonatal populations where early screening practices may influence timely recognition and management. Establishing the magnitude of this association in a local clinical setting is important for refining neonatal screening protocols, identifying high-risk neonates during the first hours of life, and preventing avoidable complications of unrecognized hypoglycemia. Therefore, this study aimed to compare the incidence of asymptomatic hypoglycemia during the first four hours of life between full-term low-birth-weight neonates and full-term normal-birth-weight neonates admitted to a tertiary-care hospital setting.

MATERIAL AND METHODS

This prospective comparative cohort study was conducted in the Department of Pediatrics, Ali Fatima Hospital, Lahore, Pakistan, over a six-month period from September 12, 2024, to March 12, 2025. The study compared full-term low-birth-weight neonates with full-term normal-birth-weight neonates to determine the incidence of asymptomatic hypoglycemia during the early neonatal period. A cohort approach was used because exposure status was defined by birth-weight category before assessment of the outcome, and neonates were subsequently evaluated for hypoglycemia during the first four hours of life.

A total of 60 full-term neonates were enrolled using a non-probability consecutive sampling technique from the labor room, nursery, and relevant neonatal care areas of Ali Fatima Hospital. Neonates were eligible if they were delivered at a gestational age of 37 completed weeks to less than 42 weeks, as determined from antenatal records and obstetric assessment, and were clinically asymptomatic at the time of glucose assessment. Full-term neonates with birth weight from 1500 g to less than 2500 g were classified as the exposed group, representing low-birth-weight neonates, whereas full-term neonates with birth weight greater than 2500 g were classified as the non-exposed group, representing normal-birth-weight neonates. Thirty neonates were included in each group. Infants of diabetic mothers, neonates with respiratory distress syndrome, sepsis, birth asphyxia, congenital anomalies, twin pregnancy, post-term birth beyond 42 weeks, and very-low-birth-weight neonates weighing less than 1500 g were excluded to reduce confounding from conditions independently associated with altered neonatal glucose homeostasis.

After screening for eligibility, written informed consent was obtained from the parents or legal guardians before enrollment. Demographic and clinical data were recorded on a predesigned proforma, including neonatal sex, gestational age at birth, birth weight, mode of delivery, duration of labor, and maternal glycemic status. Birth weight was measured in kilograms and used as the primary exposure variable. The primary outcome variable was asymptomatic hypoglycemia, operationally defined as blood glucose level below 55 mg/dL during the first four hours of life. Blood glucose was assessed using a heel-prick capillary sample obtained under aseptic precautions. A drop of blood was applied to a glucometer for estimation of blood glucose concentration. Neonates identified with hypoglycemia were managed according to the institutional neonatal management protocol.

To reduce selection bias, all eligible neonates meeting the predefined criteria during the study period were enrolled consecutively until the required sample size was completed. Eligibility criteria were applied before outcome assessment to minimize classification bias. Potential confounding was addressed at the design stage by excluding neonates with major clinical conditions known to influence glucose levels, including maternal diabetes, sepsis, respiratory distress syndrome, birth asphyxia, congenital anomalies, multiple pregnancy, post-term birth, and very-low-birth-weight status. Stratified analysis was planned for gestational age, sex, and mode of delivery to assess whether the association between birth-weight category and asymptomatic hypoglycemia differed across clinically relevant subgroups.

The sample size was calculated using the WHO sample size calculator with 90% statistical power, equal allocation between exposed and non-exposed groups, and assumptions based on previously reported frequencies of hypoglycemia among neonates with low and normal birth weight. Data were entered and analyzed using IBM SPSS Statistics version 25. Continuous variables, including gestational age, birth weight, and blood glucose level, were summarized as mean ± standard deviation with range where appropriate. Categorical variables, including sex, mode of delivery, birth-weight group, and hypoglycemia status, were summarized as frequencies and percentages. The incidence of asymptomatic hypoglycemia was compared between exposed and non-exposed groups using the chi-square test or Fisher's exact test where expected cell counts were small. Relative risk with 95% confidence interval was used to estimate the strength of association between low birth weight and asymptomatic hypoglycemia. Stratified analyses were performed for gestational age, sex, and mode of delivery. A p-value of less than 0.05 was considered statistically significant. Missing data were checked before analysis, and only complete records for the required exposure and outcome variables were included in the final analysis.

The study was conducted after institutional approval, and all procedures followed ethical principles for research involving human participants. Parental informed consent was obtained before enrollment, confidentiality of participant information was maintained, and data were used only for research purposes. Data were recorded on standardized proformas, checked for completeness before entry, and reviewed for consistency after entry into SPSS to support data integrity and reproducibility.

RESULTS

A total of 60 full-term neonates were included, with 30 neonates in the low-birth-weight group and 30 neonates in the normal-birth-weight group. The mean gestational age was 37.73 ± 0.75 weeks, with a reported range of 37.00 to 39.00 weeks. The mean birth weight was 2.59 ± 0.41 kg, ranging from 1.90 to 3.80 kg, and the mean blood glucose level during the first four hours of life was 61.07 ± 12.22 mg/dL, ranging from 38.0 to 99.0 mg/dL. Male neonates constituted 33 (55.0%) of the sample, while 27 (45.0%) were female. Cesarean section was the most frequent mode of delivery, recorded in 35 (58.3%) neonates, while 25 (41.7%) were delivered vaginally.

Table 1. Baseline Characteristics and Blood Glucose Profile of Included Neonates

Variable	Mean ± SD	Range	n (%)
Gestational age, weeks	37.73 ± 0.75	37.00–39.00	—
Birth weight, kg	2.59 ± 0.41	1.90–3.80	—

Variable	Mean ± SD	Range	n (%)
Blood glucose level, mg/dL	61.07 ± 12.22	38.0–99.0	—
Male	—	—	33 (55.0)
Female	—	—	27 (45.0)
Vaginal delivery	—	—	25 (41.7)
Cesarean section	—	—	35 (58.3)

Overall, asymptomatic hypoglycemia was identified in 21 of 60 neonates, giving an incidence of 35.0%. The remaining 39 neonates (65.0%) had blood glucose levels at or above the operational threshold for hypoglycemia.

Table 2. Incidence of Asymptomatic Hypoglycemia by Birth-Weight Group

Hypoglycemia Status	Low Birth Weight, n (%)	Normal Birth Weight, n (%)	Total, n (%)
Present	16 (53.3)	5 (16.7)	21 (35.0)
Absent	14 (46.7)	25 (83.3)	39 (65.0)
Total	30 (100.0)	30 (100.0)	60 (100.0)

Asymptomatic hypoglycemia was more frequent among low-birth-weight neonates than among normal-birth-weight neonates, with risks of 53.3% and 16.7%, respectively. The absolute risk difference was 36.7 percentage points, and the crude risk ratio was 3.20, indicating that low-birth-weight neonates had approximately three times the risk of early asymptomatic hypoglycemia compared with normal-birth-weight neonates.

Table 3. Association Between Birth-Weight Group and Asymptomatic Hypoglycemia

Comparison	Low Birth Weight	Normal Birth Weight	Risk Difference	Relative Risk	95% CI	p-value
Hypoglycemia risk	16/30 (53.3)	5/30 (16.7)	36.7	3.20	1.34–7.62	0.003

CI, confidence interval. Risk difference is reported in percentage points. p-value was derived using the chi-square test. Relative risk was calculated directly from the displayed group risks.

Stratified analysis showed that the difference in hypoglycemia frequency was most prominent among neonates with gestational age ≤38 weeks, female neonates, and neonates delivered by cesarean section. Among neonates with gestational age ≤38 weeks, hypoglycemia occurred in 15 of 27 low-birth-weight neonates compared with 2 of 22 normal-birth-weight neonates, corresponding to a risk difference of 46.5 percentage points. Among female neonates, hypoglycemia occurred in 8 of 13 low-birth-weight neonates and in none of the 14 normal-birth-weight neonates. Among neonates delivered by cesarean section, hypoglycemia occurred in 13 of 20 low-birth-weight neonates compared with 5 of 15 normal-birth-weight neonates.

Table 4. Stratified Incidence of Asymptomatic Hypoglycemia by Birth-Weight Group

Stratum	Low Birth Weight, n/N (%)	Normal Birth Weight, n/N (%)	Risk Difference	p-value
≤38 weeks	15/27 (55.6)	2/22 (9.1)	46.5	0.001
>38 weeks	1/3 (33.3)	3/8 (37.5)	-4.2	1.000
Male	8/17 (47.1)	5/16 (31.3)	15.8	0.481
Female	8/13 (61.5)	0/14 (0.0)	61.5	0.001
Vaginal delivery	3/10 (30.0)	0/15 (0.0)	30.0	0.052
Cesarean section	13/20 (65.0)	5/15 (33.3)	31.7	0.092

p-values were derived using Fisher's exact test because several subgroup cells were small. Risk difference is reported in percentage points.

The stratified findings should be interpreted cautiously because several strata had small denominators and zero-cell counts. The female and vaginal-delivery strata included zero events in the normal-birth-weight group, making conventional risk-ratio estimation unstable without continuity correction. Therefore, subgroup results are best interpreted as exploratory patterns rather than definitive effect estimates.

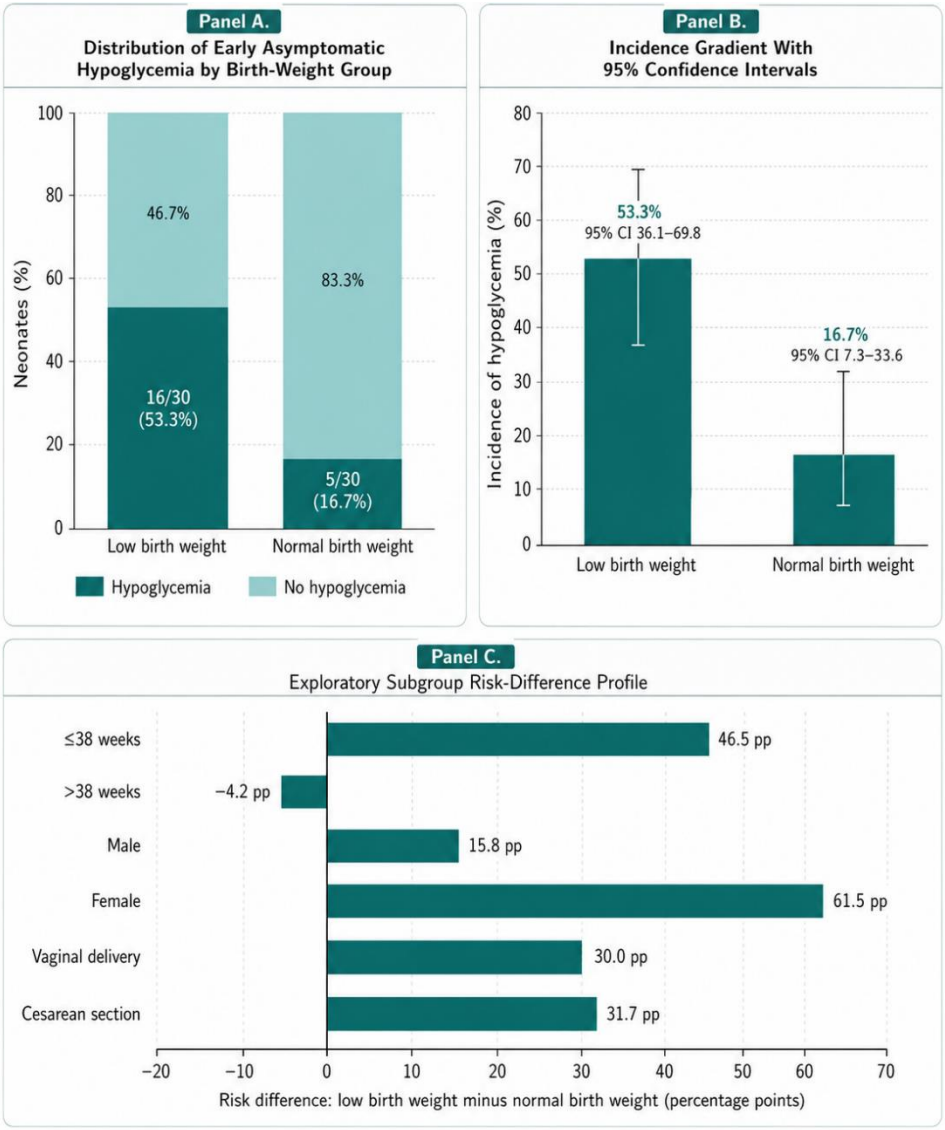

Figure 1 Birth-Weight Category and Early Asymptomatic Hypoglycemia in Full-Term Neonates

The panelled figure demonstrates a clear incidence gradient for early asymptomatic hypoglycemia by birth-weight category. Low-birth-weight neonates had a hypoglycemia incidence of 53.3% compared with 16.7% among normal-birth-weight neonates, corresponding to an absolute risk difference of 36.7 percentage points and a crude relative risk of 3.20. The stratified risk-difference profile showed the largest gradients among female neonates, where the difference was 61.5 percentage points, and among neonates with gestational age ≤38 weeks, where the difference was 46.5 percentage points. The cesarean-delivery subgroup also showed a 31.7 percentage-point higher incidence among low-birth-weight neonates, although the small subgroup denominators require cautious interpretation.

DISCUSSION

This prospective comparative cohort study found that early asymptomatic hypoglycemia was substantially more frequent among full-term low-birth-weight neonates than among full-term normal-birth-weight neonates during the first four hours of life. Overall, asymptomatic hypoglycemia was identified in 21 of 60 neonates, corresponding to an incidence of 35.0%. The incidence was 53.3% in the low-birth-weight group compared with 16.7% in the normal-birth-weight group, giving an absolute risk difference of 36.7 percentage points and a crude relative risk of 3.20. This finding supports the clinical view that full-term neonates with low birth weight remain metabolically vulnerable despite being born at term, and that birth weight alone may help identify neonates who require early glucose surveillance.

The higher incidence of hypoglycemia among low-birth-weight neonates is biologically plausible. During fetal life, glucose is supplied continuously through placental transfer, but after delivery the neonate must rapidly transition to intermittent enteral intake and endogenous glucose production (7–9). Low-birth-weight neonates may have reduced hepatic glycogen stores, limited adipose reserves, reduced availability of alternative substrates, and impaired adaptation to early feeding intervals. These mechanisms may increase susceptibility to hypoglycemia even in the absence of symptoms. Because neonatal hypoglycemia may remain clinically silent, reliance on symptoms alone may delay recognition, particularly in neonates who appear well during the immediate postnatal period (6,10,11).

The findings are broadly consistent with previous literature reporting increased hypoglycemia risk among neonates with low birth weight or impaired fetal growth. Thirumalaikumarasamy et al. reported asymptomatic hypoglycemia among term neonates and observed a significant association between hypoglycemia and birth weight, supporting birth weight as an important early metabolic risk marker (14). Saini et al. also reported hypoglycemia among low-birth-weight neonates and highlighted the role of growth status and neonatal characteristics in determining hypoglycemia patterns (12). Similarly, Mejri et al. observed hypoglycemia among term small-for-gestational-age neonates, reinforcing the vulnerability of neonates with restricted growth or low birth weight during early metabolic transition (15). Although small-for-gestational-age status and low birth weight are not interchangeable, both concepts identify neonates who may have reduced metabolic reserves and therefore require closer early monitoring.

The subgroup findings suggested that the association between low birth weight and hypoglycemia was more prominent among neonates with gestational age ≤38 weeks, female neonates, and those delivered by cesarean section. In neonates with gestational age ≤38 weeks, hypoglycemia occurred in 55.6% of low-birth-weight neonates compared with 9.1% of normal-birth-weight neonates. Among female neonates, hypoglycemia occurred in 61.5% of low-birth-weight neonates and in none of the normal-birth-weight neonates. Among cesarean-delivered neonates, the incidence was 65.0% in the low-birth-weight group and 33.3% in the normal-birth-weight group. These patterns may reflect differences in physiological maturity, feeding initiation, perinatal stress, and early postnatal adaptation; however, the subgroup denominators were small, and some strata included zero events, so these analyses should be interpreted as exploratory rather than confirmatory.

The clinical implication of this study is that full-term status should not be considered protective against early hypoglycemia when birth weight is low. Screening protocols in neonatal units often emphasize prematurity, maternal diabetes, birth asphyxia, or overt clinical signs, but the present findings support routine early glucose assessment among full-term low-birth-weight neonates as a practical risk-based approach. Early identification is important because untreated or recurrent neonatal hypoglycemia has been associated with neurological risk, although the precise threshold, duration, and causal relationship remain debated (6,11,17,18). Therefore, the results should be used to strengthen early screening and timely feeding or treatment protocols rather than to infer long-term neurological outcomes from a single early glucose measurement.

This study has several limitations. It was conducted at a single hospital with a small sample size and non-probability consecutive sampling, which limits generalizability. Blood glucose was measured once during the first four hours of life, so recurrent or delayed hypoglycemia could not be assessed. Capillary glucometer testing was used, and laboratory confirmation of low glucose values was not described, which may introduce measurement variability. Feeding status, exact timing of first feed, interval since last feed, and maternal nutritional or obstetric factors were not fully analyzed. The study excluded several important confounders, including infants of diabetic mothers and neonates with sepsis, respiratory distress, birth asphyxia, congenital anomalies, and very-low-birth-weight status, but residual confounding may still remain. Finally, subgroup analyses were limited by small denominators and zero-cell counts; therefore, they should be considered hypothesis-generating.

In summary, the study provides useful local evidence that full-term low-birth-weight neonates have a higher incidence of early asymptomatic hypoglycemia than full-term normal-birth-weight neonates. The corrected crude estimate suggests an approximately threefold higher risk based on the reported group counts. Future studies should use larger multicenter samples, serial glucose monitoring, standardized feeding documentation, laboratory confirmation of hypoglycemia, and adjusted statistical models to better define the independent contribution of low birth weight to neonatal hypoglycemia risk.

CONCLUSION

Full-term low-birth-weight neonates had a higher incidence of early asymptomatic hypoglycemia than full-term normal-birth-weight neonates during the first four hours of life. Based on the reported group counts, hypoglycemia occurred in 53.3% of low-birth-weight neonates compared with 16.7% of normal-birth-weight neonates, corresponding to a crude relative risk of 3.20 and an absolute risk difference of 36.7 percentage points. These findings support early blood glucose screening for full-term low-birth-weight neonates in routine neonatal care, although larger studies with serial glucose monitoring and adjusted analysis are needed to confirm the magnitude of risk and refine screening protocols.

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