J Pediatr Res 2026;13(2):141-9
DOI: 10.4274/jpr.galenos.2026.49207

Original Article

Blood Glucose ≤30 mg/dL as a Predictor of Symptomatic Hypoglycemia among Hypoglycemic Neonates in the First Four Hours of Life

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ABSTRACT

Aim: To determine predictors of symptomatic presentation among neonates diagnosed with hypoglycemia within the first four hours of life.

Materials and Methods: A retrospective cohort study was conducted using the medical records of hypoglycemic neonates evaluated within four hours postpartum at neonatology unit of the Hospital Nacional Docente Madre Niño San Bartolomé in Lima, Peru. Neonates with symptomatic hypoglycemia were compared to those with asymptomatic hypoglycemia. Multivariate logistic regression and receiver operating characteristic (ROC) analysis were performed.

Results: Among 95 hypoglycemic neonates, 48 (50.5%) exhibited symptoms. A blood glucose level ≤30 mg/dL was strongly associated with symptomatic presentation [adjusted odds ratio: 5.84; 95% confidence interval (CI): 1.58-21.5]. ROC analysis demonstrated fair discriminatory capacity (area under the curve: 0.671; 95% CI: 0.562-0.779). No significant associations were found for sex, birth weight, or gestational age.

Conclusion: Among hypoglycemic neonates, a glucose level ≤30 mg/dL is a predictor of symptomatic presentation within the first four hours. The model's modest discriminatory performance highlights the need for larger prospective studies to refine risk stratification.

Keywords: Newborn, neonatal disease, hypoglycemia, risk factors, neonatal screening

INTRODUCTION

Neonatal hypoglycemia is the most prevalent metabolic disorder in newborns, with reported incidence rates of 5-7% in at-term newborns and 3-14% in preterm populations (1-3). Its high frequency is largely attributable to risk factors such as prematurity (4), small-for-gestational-age (SGA) status (1,5), and maternal diabetes (6-13), collectively accounting for approximately 50% of neonatal hypoglycemia cases. In at-risk neonates, auxiliary evaluations, most notably capillary glucose measurements are recommended within the first four hours of life.

During the immediate postnatal period, neonates experience a physiological decrease in blood glucose levels, due primarily to the interruption of maternal

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Received: 26.12.2025 **Accepted:** 04.06.2026 **Publication Date:** 01.07.2026

Cite this article as: Barrientos Martel CJ, Gonzales RWO, Lavalle AC, Aguirre-Quispe W. Blood glucose ≤30 mg/dL as a predictor of symptomatic hypoglycemia among hypoglycemic neonates in the first four hours of life. J Pediatr Res. 2026;13(2):141-9

glucose flow at the moment of umbilical cord clamping (14). To maintain euglycemia, neonates rapidly activate endogenous regulatory pathways, including hepatic glycogenolysis and gluconeogenesis (14,15). Insufficiencies in these mechanisms can lead to a precipitous drop in serum glucose concentrations. Without appropriate and timely intervention, untreated hypoglycemia significantly increases the likelihood of clinical manifestations ranging from tremors, irritability, and lethargy to apnea, seizures, and coma and also heightens the risk of persistent neurological sequelae, such as psychomotor delays, cognitive impairments, or even epileptic syndromes (16-18).

The precise timing of symptomatic onset is critical for diagnosing and managing hypoglycemia. Existing guidelines suggest a broad temporal window, and studies of symptom sensitivity for hypoglycemia remain inconclusive (6,19). Some investigations have indicated that symptoms appear within the first 24 to 48 hours of life, whereas others have proposed that reduced serum glucose may persist until 72 hours before overt clinical signs manifest (2,20). Physiologically, however, glucose levels reach their nadir between 2 and 4 hours of life, following which homeostatic mechanisms become fully activated (21,22). As the current consensus on normal glycemia values for term, preterm, or high-risk neonates is lacking (4), the Spanish Society of Pediatric Endocrinology defines neonatal hypoglycemia as blood glucose <45 mg/dL while proposing additional thresholds based on postnatal age (23-25). This can be problematic, particularly since the lowest glucose concentrations occur during the initial hours of life, when diagnostic screenings are not typically administered unless symptoms are evident, and because screening devices show decreased accuracy at lower glucose levels (14,26).

Among neonates with established hypoglycemia (<45 mg/dL), the degree of glucose reduction is a key determinant of whether clinical symptoms develop. This relationship reflects the severity of metabolic disruption and the neonate's compensatory capacity. Thus, identifying a specific glucose threshold which predicts symptom onset within the hypoglycemic population is clinically relevant for guiding interventions.

Understanding the timing and predictors of symptomatic hypoglycemia may optimize the scheduling of glucose monitoring, reduce unnecessary interventions, and improve outcomes. This study aimed to identify predictors of symptomatic presentation among neonates diagnosed with hypoglycemia within the first four hours of life, to guide timely interventions and to minimize neurological complications.

Materials and Methods

This was a retrospective cohort study of neonates diagnosed with hypoglycemia (<45 mg/dL) within the first four hours of life. The study population was divided into two groups based on the presence or absence of clinical symptoms: symptomatic hypoglycemia (cases) and asymptomatic hypoglycemia (controls). The study design was therefore a comparative cohort analysis of hypoglycemic neonates, rather than a traditional case-control study of risk factors for developing hypoglycemia. This study was conducted at the neonatology unit of the Hospital Nacional Docente Madre Niño San Bartolomé, a national teaching and referral maternal-child hospital in Lima, Peru, from January 2022 to December 2023. The exposed group (symptomatic hypoglycemia) was defined as neonates with a capillary blood glucose level <45 mg/dL accompanied by at least one clinical sign such as tremors, jitteriness, lethargy, hypotonia, weak sucking, apnea, or seizures. The unexposed group (asymptomatic hypoglycemia) consisted of neonates with a capillary blood glucose level <45 mg/dL within the same timeframe but without any accompanying clinical signs.

The threshold of <45 mg/dL was selected based on guidelines from the Spanish Society of Pediatric Endocrinology and it aligns with operational thresholds recommended by the American Academy of Pediatrics for the first 4 hours of life. While other organizations such as the Canadian Paediatric Society use <47 mg/dL, our choice reflects a conservative approach commonly employed in our clinical setting.

Blood glucose measurement was performed within the first four hours of life as part of standard clinical protocol. Inclusion criteria for both groups were neonates of either sex within the first 4 hours of life with a diagnosis of hypoglycemia (capillary glucose <45 mg/dL) and complete medical record data for predefined risk factors. Exclusion criteria included neonates with congenital metabolic or genetic disorders such as congenital hypothyroidism, adrenal hyperplasia, phenylketonuria, galactosemia, or cystic fibrosis, as well as those from twin or multiple pregnancies.

Capillary blood glucose was measured using Accu-Chek® point-of-care glucometers calibrated according to the manufacturer's specifications. Quality control was performed daily per hospital protocol using standard control solutions. Confirmatory laboratory venous glucose testing was not routinely performed, as point-of-care measurements are standard practice for screening in the immediate care unit.

Data were retrospectively abstracted from medical records using a standardized data collection form. Capillary blood glucose was measured via a standardized heel prick procedure performed by trained nursing staff and recorded in mg/dL. The neonatal information and measurements collected included sex, birth weight, birth length, head circumference, and the precise time of glucose measurement. Gestational age was determined clinically using the Capurro B scale. Birth weight was categorized as low birth weight (<2,500 g), normal (2,500-4,000 g), or macrosomic. Weight for gestational age was classified as (SGA, <10th percentile), [appropriate for gestational age (AGA), 10th-90th percentile], or large for gestational age (LGA, >90th percentile) using established percentile charts. Maternal and pregnancy variables included advanced maternal age (>35 years), inadequate prenatal care (<6 visits), gestational diabetes, gestational hypertension (blood pressure ≥140/90 mmHg on two occasions 4 hours apart after 20 weeks), pregestational body mass index (BMI), maternal obesity (BMI≥30 kg/m^2), total gestational weight gain, excessive gestational weight gain according to Institute of Medicine guidelines, and mode of delivery.

The sample size was calculated *a priori* using G*Power software. Based on previous studies, we estimated a 30% prevalence of symptomatic hypoglycemia among neonates with hypoglycemia and expected to detect an odds ratio of 3.5 for the primary predictor. With a two-sided alpha of 0.05, 80% power, and 1:1 ratio of symptomatic to asymptomatic neonates, a minimum of 46 participants per group was required. A non-probabilistic convenience sampling method was employed, reviewing all eligible medical records until the target sample size was met, resulting in 95 neonates (48 symptomatic, 47 asymptomatic) for the final analysis (Figure 1).

The study protocol was approved by the Institutional Ethics Committees of Investigación de la Universidad Científica del Sur (CIEI-CIENTÍFICA) (approval no.: 537-CIEI-CIENTÍFICA-2024, date: 25.06.2024). The requirement for informed consent was waived due to the retrospective nature of this study using anonymized data. Data confidentiality was maintained through password-protected electronic files and the removal of identifying details prior to analysis, following the principles of the Declaration of Helsinki.

Statistical Analysis

Statistical analysis was performed using Stata Version 18. Normality of continuous variables was assessed with the Shapiro-Wilk test. Descriptive statistics included means with standard deviations for normally distributed variables, medians with interquartile ranges for non-normal variables, and frequencies with percentages for categorical variables. Bivariate analyses used Pearson's chi-square test (or Fisher's exact test) for categorical variables and Student's t-test (or the Mann-Whitney U test) for continuous variables. Variables for the multivariate logistic regression model were selected *a priori* based on clinical relevance and the

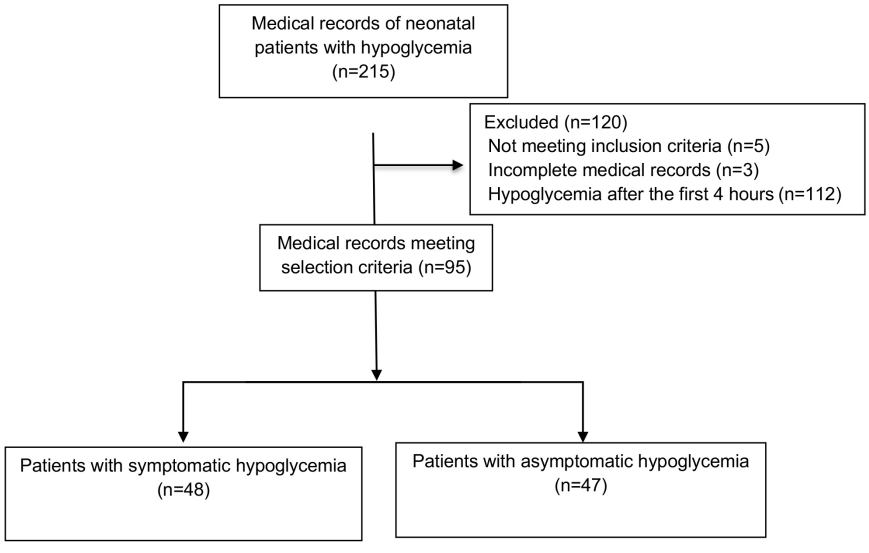

Figure 1. Study population (STROBE flow diagram)

established literature, including blood glucose level, sex, birth weight, birth length, gestational age, delivery type, maternal obesity, and time of glucose measurement. To minimize the risk of overfitting given the sample size (n=95), the model was limited to these 8 prespecified variables without automated stepwise selection. The results are presented as adjusted odds ratios with 95% confidence intervals. The model's discriminatory performance was assessed using receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) calculated to quantify predictive accuracy. The optimal probability cut-point was determined based on the maximum sum of sensitivity and specificity.

Results

A total of 95 neonates were analyzed: 48 (50.5%) with symptomatic hypoglycemia and 47 (49.5%) asymptomatic. No statistically significant differences were found between the groups regarding sex, birth weight, length, head circumference, or gestational age. The mean time of measurement was 2.2±0.7 hours in both groups (p=0.793), with no significant difference in sampling timing between the symptomatic and asymptomatic neonates.

Most neonates were classified as AGA (54.7%), with no significant group differences in weight categories. Maternal characteristics such as advanced maternal age (>35 years), inadequate prenatal care (<6 visits), gestational diabetes,

Table I. Baseline neonatal and maternal characteristics stratified by symptomatic hypoglycemia status

Neonatal characteristics	**Total (n=95)**	**Symptomatic hypoglycemia (n=48)**	**Asymptomatic hypoglycemia (n=47)**	**p value**
Male sex, n (%)	62 (65.3)	30 (62.5)	32 (68.1)	0.568
Birth weight, g, mean±SD	3,173±789	3,236±807	3,108±773	0.430
Birth length, cm, mean±SD	48.4±2.8	48.4±2.8	48.3±2.8	0.797
Head circumference, cm, mean±SD	33.6±2.0	33.8±1.9	33.4±2.2	0.344
Blood glucose (first 4h), mg/dL, mean±SD	36.8±7.0	35.2±8.3	38.5±5.1	0.022†
Time of glucose measurement, hours, mean±SD	2.2±0.7	2.2±0.7	2.2±0.7	0.793
Birth weight category, n (%)				
Normal	53 (55.8)	25 (52.1)	28 (59.6)	1.00 (Reference)
Low	23 (24.2)	12 (25.0)	11 (23.4)	0.688
Macrosomic	19 (20.0)	11 (22.9)	8 (17.0)	0.422
Weight for gestational age, n (%)				
Appropriate	52 (54.7)	23 (47.9)	29 (61.7)	1.00 (Reference)
Small	24 (25.3)	15 (31.3)	9 (19.1)	0.138
Large	19 (20.0)	10 (20.8)	9 (19.1)	0.529
Maternal & pregnancy characteristics				
Gestational age, weeks, mean±SD	38.3±1.7	38.4±1.7	38.2±1.6	0.553
Advanced maternal age (>35 years), n (%)	25 (26.3)	15 (31.3)	10 (21.3)	0.270
Inadequate prenatal care (<6 visits), n (%)	34 (35.8)	17 (35.4)	17 (36.2)	0.939
Gestational hypertension, n (%)	3 (3.2)	0 (0.0)	3 (6.4)	--
Gestational diabetes, n (%)	10 (10.5)	6 (12.5)	4 (8.5)	0.526
Pregestational BMI, kg/m^2, mean±SD	27.8±5.8	27.7±4.9	27.9±6.5	0.858
Maternal obesity (BMI≥30), n (%)	18 (19)	7 (14.6)	11 (23.4)	0.273
Total gestational weight gain, kg, mean±SD	10.6±6.5	11.4±6.9	9.8±6.2	0.407
Excessive gestational weight gain, n (%)	17 (17.9)	11 (22.9)	6 (12.8)	0.197
Cesarean delivery, n (%)	34 (35.8)	19 (39.6)	15 (31.9)	0.436

†Variable with statistically significant in the univariate and multivariate analysis. Bold indicates statistical significance (p<0.05)
SD: Standard deviation, BMI: Body mass index

and pregestational BMI were similarly distributed between the groups and they were without statistical significance. Gestational hypertension was observed in three cases, all in the asymptomatic group (Table I).

Upon analyzing specific neonatal factors, symptomatic newborns had a significantly lower mean blood glucose when compared to the asymptomatic ones (35.2±8.3 mg/dL vs. 38.5±5.1 mg/dL; p=0.022). Notably, nearly one-third (29.2%) of the symptomatic neonates had blood glucose levels ≤30 mg/dL, compared to only 8.5% in the asymptomatic group. Although it did not reach statistical significance, a trend towards a higher frequency of SGA infants was observed in the symptomatic group (31.3% vs. 19.1%; p=0.138).

Regarding maternal factors, excessive gestational weight gain was more than twice as frequent in the mothers of symptomatic neonates (22.9% vs. 12.8%), although this difference was not statistically significant (p=0.197). Maternal obesity, by contrast, showed an inverse trend, being less frequent in the symptomatic case group (14.6% vs. 23.4%).

Box plot analysis showed a lower and more variable glucose distribution in the symptomatic group, while the asymptomatic group had a narrower, more centralized distribution (Figure 2).

In the multivariate logistic regression analysis, only blood glucose level emerged as a statistically significant independent predictor of symptomatic hypoglycemia (coefficient: -0.079; p=0.021). Sex, birth weight, birth length, gestational age, delivery type, maternal obesity, and time of glucose measurement showed no significant associations (all p>0.05) (Table II). The overall model demonstrated poor fit (LR $\chi2$=8.26, p=0.220; pseudo R^2=0.063).

ROC curve analysis of the logistic regression model yielded an AUC of 0.671 [95% confidence interval (CI): 0.562-0.779], indicating fair discriminatory capacity for predicting symptomatic presentation among hypoglycemic neonates. The optimal probability threshold was 0.30, corresponding to a glucose value of approximately 32 mg/dL, with 62.5% sensitivity and 65.9% specificity. This threshold, while statistically derived, should be interpreted cautiously given the modest model performance. No cut-point achieved both high sensitivity (>80%) and high specificity (>80%) simultaneously (Figure 3).

The multivariate logistic regression analysis confirmed that a blood glucose level ≤30 mg/dL was the only independent and statistically significant predictor of symptomatic hypoglycemia (adjusted Odds ratio: 5.84; 95% CI: 1.58-21.50), even after adjusting for birth weight, gestational age, gestational diabetes, and other relevant maternal factors.

Among symptomatic neonates, the most common clinical sign was tremor (64.5%), followed by hypoactivity (39.6%) and weak sucking (16.7%). Less frequent symptoms included respiratory distress (4.1%) and distal coldness (2.1%) (Figure 4).

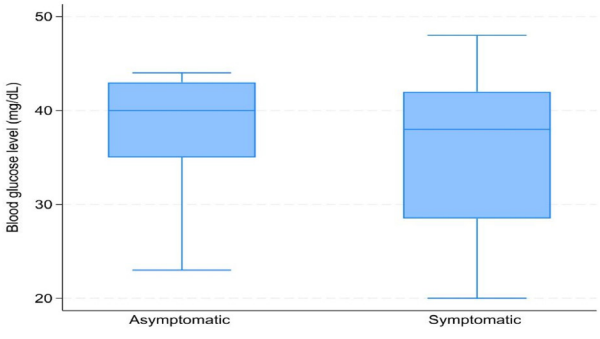

Figure 2. Distribution of blood glucose levels in symptomatic vs. asymptomatic neonates. *The box plot illustrates the lower and more variable blood glucose levels in the symptomatic group (n=48) compared to the asymptomatic group (n=47) within the first four hours of life. The central line within each box represents the median, the box encompasses the interquartile range (25th-75th percentiles), and the whiskers show the variability outside the upper and lower quartiles*

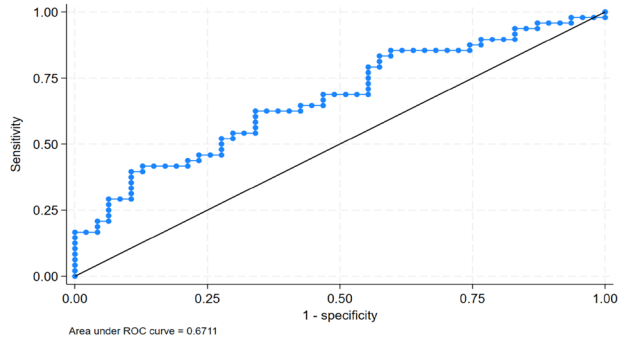

Figure 3. Receiver operating characteristic (ROC) curve for the prediction of symptomatic hypoglycemia. The area under the curve (AUC) is 0.671 (95% CI: 0.562-0.779), indicating fair discriminatory ability. The diagonal reference line represents no discriminatory power (AUC=0.5)
CI: Confidence interval

Table II. Crude and adjusted associations between neonatal/maternal factors and symptomatic hypoglycemia

Factor	Symptomatic n=48 n (%)	Asymptomatic n=47 n (%)	Crude OR (95% CI)	Adjusted OR* (95% CI)
Neonatal factors				
Male sex	30 (62.5)	32 (68.1)	0.78 (0.33-1.82)	0.85 (0.32-2.31)
Blood glucose ≤30 mg/dL	14 (29.7)	4 (8.51)	**4.42 (1.33-14.67)**	**5.84 (1.58-21.5)**
Birth weight category				
Normal	25 (52.1)	28 (59.6)	Reference	Reference
Low	12 (25.0)	11 (23.4)	1.22 (0.45-3.25)	1.04 (0.27-3.94)
Macrosomic	11 (22.9)	8 (17.0)	1.54 (0.53-4.43)	2.61 (0.21-31.1)
Weight for gestational age				
Appropriate	23 (47.9)	29 (61.7)	1.00 (Reference)	1.00 (Reference)
Small	15 (31.3)	9 (19.1)	2.10 (0.77-5.66)	2.12 (0.59-7.67)
Large	10 (20.8)	9 (19.1)	1.40 (0.48-4.01)	0.57 (0.05-6.02)
Maternal & pregnancy factors				
Advanced maternal age (>35 years)	15 (31.3)	10 (21.3)	1.68 (0.66-4.25)	1.96 (0.65-5.90)
Inadequate prenatal care (<6 visits)	17 (35.4)	17 (36.2)	0.96 (0.41-2.23)	1.05 (0.38-2.85)
Gestational diabetes	6 (12.5)	4 (8.5)	1.53 (0.40-5.83)	1.27 (0.27-5.95)
Maternal obesity (BMI≥30)	7 (14.6)	11 (23.4)	0.55 (0.19-1.59)	0.44 (0.13-1.48)
Excessive gestational weight gain	11 (22.9)	6 (12.8)	2.03 (0.68-6.03)	3.32 (0.92-11.96)
Cesarean delivery	19 (39.6)	15 (31.9)	1.39 (0.60-3.24)	0.87 (0.24-3.05)

*Adjusted for all other variables listed in the table. Bold indicates statistical significance (p<0.05).
OR: Odds ratio, CI: Confidence interval, BMI: Body mass index

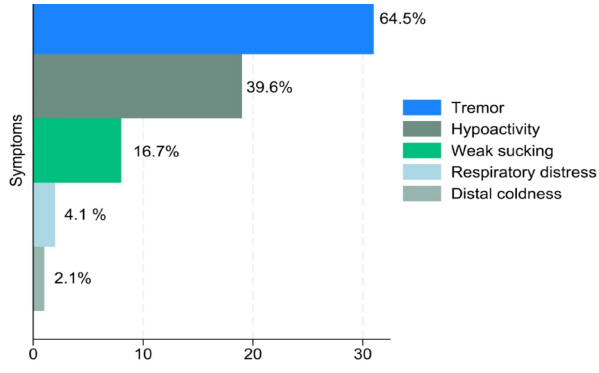

Figure 4. Prevalence of clinical manifestations among neonates with symptomatic hypoglycemia (n=48). The bar chart displays the frequency of clinical signs observed in the study cohort. Tremor was the most common symptom, present in 64.5% of symptomatic neonates, followed by hypoactivity (39.6%) and weak sucking (16.7%)

Discussion

Neonatal hypoglycemia represents a prevalent metabolic disorder in newborns, necessitating early recognition and intervention in order to mitigate associated complications. Identifying factors which predict symptomatic presentation among hypoglycemic neonates is essential in order to improve clinical outcomes and reduce long-term sequelae.

Our findings demonstrate that a blood glucose concentration of ≤30 mg/dL significantly increases the risk of symptomatic hypoglycemia. These results align with previous studies, such as those conducted in New Zealand and India, which showed that severe hypoglycemia (defined as glucose levels below 2 mmol/L or 35 mg/dL) correlates strongly with the onset of symptoms (27,28). One study demonstrated that lower glucose levels were significantly associated with the presence of hypoglycemia symptoms (29). Additional studies have reported mean glucose levels of 20±10 mg/dL in symptomatic neonates compared to 27.3±7 mg/dL in their asymptomatic counterparts, further corroborating our observations (30).

Severe hypoglycemia (≤30 mg/dL) has been implicated in neurodevelopmental impairments (31), particularly in cases of prolonged or recurrent episodes (32). Recurrent hypoglycemia in preterm neonates has been associated with lower scores on the Bailey scale, cognitive delays, and developmental deficits, reinforcing the necessity of prompt and aggressive management (31). The current clinical guidelines advocate for intravenous glucose administration in cases of symptomatic or severe hypoglycemia in order to prevent irreversible neuronal damage (33,34).

By contrast, our study did not find significant associations between low birth weight and symptomatic hypoglycemia. This contrasts with previous research from Japan (35), where low birth weight neonates frequently exhibited asymptomatic hypoglycemia within the first hour of life, without clear symptom development. Similarly, the classification as SGA did not emerge as a significant predictor of symptomatic hypoglycemia in our cohort. However, prior studies in India have reported symptomatic hypoglycemia rates of 10.3% and 31% in SGA neonates, a discrepancy which is potentially attributable to differences in study design and population characteristics (29,36).

The observed trend towards a higher frequency of SGA infants in the symptomatic group, while not statistically significant, warrants consideration. It is plausible that our study did not have the power to detect a moderate association. SGA neonates have limited hepatic glycogen stores and an impaired capacity for gluconeogenesis, theoretically rendering them more susceptible to symptomatic hypoglycemia. The discrepancy with those previous studies which reported a strong association could be attributed to differences in the definition of SGA, the timing of the glucose measurement, or the characteristics of the reference population (29,36). Future studies with a larger sample size are needed in order to clarify this relationship within the critical first four-hour window.

Our multivariate analysis established blood glucose level as the predominant predictor of symptomatic hypoglycemia in the immediate postnatal period, with a negative coefficient confirming the expected inverse relationship: decreasing glucose concentrations significantly increase the probability of symptomatic presentation. Notably, traditional risk factors such as birth weight and gestational age demonstrated limited predictive utility within this specific physiological window.

The model's discriminatory capacity, while statistically significant, was modest (AUC: 0.671), underscoring the complexity of symptomatic presentation and suggesting contributions from unmeasured variables. The inability to identify a probability cut-point with both high sensitivity and specificity further highlights the challenges in applying probabilistic models to individual clinical decision-making in this context.

The primary strength of this study lies in its focused investigation of the physiologically crucial first four hours of life, a period characterized by dynamic metabolic transitions but frequently understudied in the existing literature. Our identification of glucose ≤30 mg/dL as a strong, independent predictor provides clinicians with a clear, quantifiable threshold for targeted interventions during this vulnerable period.

Study Limitations

This study had several limitations. The retrospective design inherently limited data collection to previously documented medical records, potentially introducing information bias. The sample size, while adequate for detecting the primary association with glucose levels, may have constrained the statistical power in identifying more modest effects of other risk factors. Furthermore, being conducted at a single institution may affect generalizability to populations with differing demographic and clinical characteristics.

Additionally, point-of-care glucometers are known to have decreased accuracy at glucose levels <40 mg/dL, and confirmatory laboratory testing was not routinely performed. This may have introduced measurement bias, particularly in the severe hypoglycemia range, and should be considered when interpreting the threshold of ≤30 mg/dL.

Finally, the observed association between glucose ≤30 mg/dL and symptomatic presentation is physiologically expected; glucose level in this context functions more as a severity marker than as an independent risk factor. Our study quantifies this association but does not establish causality.

Conclusion

In this context, glucose ≤30 mg/dL should be viewed as a predictor of symptomatic presentation among hypoglycemic neonates within the first four hours of life. However, the modest discriminatory performance of our model (AUC: 0.671) and the limited sample size underscore the need for caution in clinical application. Larger prospective studies with standardized symptom assessment and confirmatory laboratory glucose measurement are needed in order to validate these findings and refine intervention thresholds.

Ethics

Ethics Committee Approval: The study protocol was approved by the Institutional Ethics Committees of Investigación de la Universidad Científica del Sur (CIEI-CIENTÍFICA) (approval no.: 537-CIEI-CIENTÍFICA-2024, date: 25.06.2024).

Informed Consent: The requirement for informed consent was waived due to the retrospective nature of this study using anonymized data.

Footnotes

Authorship Contributions

Surgical and Medical Practices: C.J.B.M., R.W.O.G., A.C.L., W.A.-Q., Concept: C.J.B.M., R.W.O.G., W.A.-Q., Design: C.J.B.M., R.W.O.G., W.A.-Q., Data Collection or Processing: C.J.B.M., R.W.O.G., A.C.L., Analysis or Interpretation: W.A.-Q., Literature Search: C.J.B.M., R.W.O.G., A.C.L., Writing: C.J.B.M., R.W.O.G., A.C.L., W.A.-Q.

Conflict of Interest: The authors declare that they have no competing interests.

Financial Disclosure: The authors received no financial support for the research, authorship, and/or publication of this article.

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