Original Article

Frequency of Hypomagnesemia in Women Undergoing Preterm Labor

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ABSTRACT

Background: *Preterm labor is a major contributor to neonatal morbidity and mortality, and micronutrient disturbances such as hypomagnesemia may influence myometrial excitability and early uterine activity.* ***Objective:*** *To determine the frequency of hypomagnesemia among women presenting with spontaneous preterm labor and to assess its distribution across key maternal and clinical characteristics.* ***Methods:*** *A descriptive cross-sectional study was conducted in the Department of Obstetrics and Gynecology, Allied Hospital Faisalabad, over six months. Women aged 18–40 years with singleton gestation, intact membranes, and spontaneous preterm labor (<37 weeks) were enrolled by consecutive sampling after informed consent. Maternal age, parity, gestational age, and BMI were recorded. Venous blood (3 mL) was obtained on admission before magnesium-containing therapy and analyzed in a single laboratory; hypomagnesemia was defined as serum magnesium <1.46 mg/dL. Associations were examined using chi-square testing and logistic regression.* ***Results:*** *Among 185 women, hypomagnesemia occurred in 128 (69.2%; 95% CI: 62.1%–75.5%). Prevalence was highest in underweight (88/91, 96.7%) and obese women (32/37, 86.5%) compared with normal BMI (2/39, 5.1%) (p<0.001) and was more frequent at <28 weeks (84/103, 81.6%) than 32–36+6 weeks (12/41, 29.3%) (p<0.001). Underweight BMI and obesity remained independently associated with hypomagnesemia in multivariable analysis.* ***Conclusion:*** *Hypomagnesemia is highly prevalent in women presenting with preterm labor, particularly among those at BMI extremes and earlier gestations, supporting consideration of targeted antenatal magnesium assessment and further prospective interventional research.*

Keywords: *Hypomagnesemia; Preterm labor; Prematurity; Body mass index; Gestational age; Parity.*

INTRODUCTION

Preterm labor, defined as the onset of regular uterine contractions accompanied by cervical change before 37 completed weeks of gestation, remains a major determinant of adverse maternal and neonatal outcomes worldwide (1,2). Globally, preterm birth accounts for approximately 11% of all live births, representing nearly 15 million deliveries annually, and contributes substantially to neonatal morbidity, long-term neurodevelopmental impairment, and perinatal mortality (1,3). Despite advances in obstetric and neonatal care, the multifactorial etiology of preterm labor continues to challenge clinicians, with recognized contributors including infection, inflammation, uteroplacental ischemia, socioeconomic determinants, and maternal demographic factors such as age and parity (3,4). However, in a significant proportion of cases, the precise initiating mechanism remains unclear, suggesting that potentially modifiable biological factors may play a contributory role.

Among the proposed biological mechanisms, disturbances in micronutrient homeostasis—particularly magnesium deficiency—have gained attention due to magnesium's central role in cellular metabolism, neuromuscular transmission, and myometrial contractility (5,6). Magnesium functions as a physiological calcium antagonist at the cellular level, modulating voltage-gated calcium channels and thereby influencing uterine smooth muscle excitability (6). A reduction in extracellular or intracellular magnesium concentrations may lower the threshold for calcium-mediated myometrial contractions, potentially facilitating premature

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uterine activity (6,7). Physiologically, serum magnesium levels tend to decline during pregnancy, possibly due to hemodilution, increased renal excretion, and heightened fetal demand (7). While this decline is often subclinical, pronounced hypomagnesemia may alter uterine contractile dynamics and predispose susceptible women to early labor.

Several observational studies have reported lower maternal serum magnesium levels among women presenting with preterm labor compared to those delivering at term. Mahmoud et al. demonstrated significantly reduced mean serum magnesium levels in women with preterm labor compared to gestational age–matched controls, suggesting a possible association between hypomagnesemia and premature uterine activity (8). Similarly, Kamal et al. observed that women with idiopathic preterm labor had significantly lower magnesium concentrations than controls, reinforcing the hypothesis that magnesium deficiency may contribute to preterm parturition (9). Additional data from Nigerian cohorts have indicated that women with serum magnesium levels below defined laboratory thresholds were nearly twice as likely to experience preterm labor compared with those with normal levels (10). Earlier biochemical studies evaluating calcium–phosphorus–magnesium homeostasis in threatened preterm delivery further support the biological plausibility of magnesium depletion as a contributor to early uterine contractility (11,12). Although these studies suggest a consistent association, most were conducted in heterogeneous populations and employed varying diagnostic thresholds and study designs, limiting generalizability across different settings.

Importantly, while international data suggest that hypomagnesemia may be prevalent among women with preterm labor, there remains limited contemporary evidence quantifying its frequency in South Asian populations, particularly within Pakistan. Local epidemiological data are essential because nutritional status, socioeconomic factors, dietary patterns, and baseline micronutrient deficiencies vary substantially across regions and may influence both serum magnesium levels and preterm birth risk. Furthermore, prior studies have often utilized case–control designs comparing preterm and term deliveries; fewer investigations have specifically focused on estimating the frequency of hypomagnesemia among women actively presenting with preterm labor, stratified by clinically relevant variables such as age, parity, body mass index, and gestational age at presentation. The absence of locally generated, systematically collected data limits the development of context-specific screening or preventive strategies.

Within a PICO framework, the population of interest comprises pregnant women aged 18–40 years with singleton gestation presenting with spontaneous preterm labor before 37 weeks. The exposure of interest is hypomagnesemia, defined biochemically according to standardized laboratory reference ranges. Although this descriptive cross-sectional study does not include an external comparison group, subgroup comparisons across maternal and clinical characteristics allow evaluation of distribution patterns within the population. The primary outcome is the frequency of hypomagnesemia at the time of presentation with preterm labor. Clarifying this frequency and its stratified distribution may provide foundational data for future analytical or interventional research evaluating magnesium supplementation or targeted screening protocols.

Given the biological plausibility of magnesium deficiency in enhancing uterine excitability, the supportive but heterogeneous international evidence, and the paucity of robust local data, there is a clear need to quantify the burden of hypomagnesemia among women presenting with preterm labor in this setting. Therefore, the objective of this study is to determine the frequency of hypomagnesemia in women presenting with preterm labor at a tertiary care hospital and to evaluate its distribution according to maternal age, parity, body mass index,

and gestational age. We hypothesize that hypomagnesemia is common among women with preterm labor and is more prevalent in specific high-risk subgroups, thereby warranting further investigation into its potential role as a modifiable risk factor in the prevention of preterm birth.

METHODS

This descriptive cross-sectional observational study was conducted in the Department of Obstetrics and Gynecology at Allied Hospital, Faisalabad, over a six-month period following approval of the study protocol. The cross-sectional design was selected to estimate the point frequency of hypomagnesemia among women presenting with spontaneous preterm labor and to examine its distribution across predefined maternal and clinical characteristics. This design is appropriate for determining prevalence and exploring associations within a defined population at a specific time point (13).

The study population comprised pregnant women aged 18 to 40 years presenting with spontaneous preterm labor before 37 completed weeks of gestation. Preterm labor was operationally defined as the presence of regular uterine contractions (at least four contractions in 20 minutes or eight contractions in 60 minutes) accompanied by documented cervical changes (cervical dilatation ≥1 cm and/or effacement ≥80%) occurring between 24+0 and 36+6 weeks of gestation, confirmed by clinical examination. Gestational age was determined based on first-trimester ultrasound where available or calculated from a reliable last menstrual period corroborated by early obstetric ultrasonography. Only women with singleton pregnancies and intact fetal membranes at presentation were eligible. Women were excluded if they had a previous history of preterm delivery, hypertensive disorders of pregnancy (defined as blood pressure ≥140/90 mmHg after 20 weeks of gestation with or without proteinuria), clinical or laboratory evidence of urinary tract infection (≥10 pus cells per high-power field on microscopic examination of centrifuged urine), chronic renal disease, known metabolic or endocrine disorders affecting magnesium metabolism, multiple gestation, or receipt of magnesium sulfate or other magnesium-containing therapy prior to blood sampling.

Participants were recruited using a non-probability consecutive sampling technique. All eligible women presenting during the study period were screened by the attending obstetric team, and those meeting inclusion criteria were invited to participate. The study objectives, procedures, potential risks, and benefits were explained in the local language, and written informed consent was obtained prior to enrollment. Recruitment occurred at the time of admission to the labor ward, and blood sampling was performed immediately after enrollment and before initiation of any tocolytic agents, intravenous fluids containing magnesium, or magnesium sulfate therapy to avoid measurement bias.

Data collection followed a standardized protocol. A structured data collection form was used to record baseline demographic and clinical variables, including maternal age (in completed years), parity (nulliparous = parity 0; primiparous = parity 1; multiparous = parity ≥2), gestational age at presentation (in completed weeks and categorized as extremely preterm <28 weeks, very preterm 28 to <32 weeks, and late preterm 32–36+6 weeks), and body mass index (BMI), calculated as weight in kilograms divided by height in meters squared (kg/m²). Weight and height were measured using calibrated digital scales and a wall-mounted stadiometer, respectively, with participants in light clothing and without shoes. BMI was categorized according to World Health Organization criteria as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30.0 kg/m²) (14).

For biochemical assessment, three milliliters of venous blood were drawn aseptically from the antecubital vein using a disposable syringe and collected into plain vacutainer tubes. Samples were allowed to clot and then centrifuged at 3000 rpm for 10 minutes to separate serum. Serum magnesium concentration was measured in a single accredited hospital laboratory using a standardized colorimetric method based on xylidyl blue reaction, following manufacturer instructions and internal quality control procedures. The laboratory reference range for serum magnesium was 1.46–2.68 mg/dL. Hypomagnesemia was operationally defined a priori as serum magnesium level <1.46 mg/dL. Values within 1.46–2.68 mg/dL were considered normal, and values above 2.68 mg/dL were categorized separately as hypermagnesemia but were not included under the definition of hypomagnesemia. Laboratory personnel were blinded to participants' clinical subgroup classifications to minimize measurement bias.

The primary outcome variable was the frequency (proportion) of hypomagnesemia among women presenting with preterm labor. Secondary variables included maternal age group, parity category, BMI category, and gestational age category. To address potential sources of bias, consecutive enrollment was employed to reduce selection bias, standardized operational definitions were applied to ensure uniform case identification, and all biochemical analyses were performed in the same laboratory to limit inter-laboratory variability. Exclusion of women who had received magnesium-containing therapy prior to sampling reduced misclassification bias. Data entry was performed using double-entry verification, and range and consistency checks were conducted to ensure data integrity.

The sample size was calculated using the World Health Organization sample size calculator for estimation of a single population proportion. Assuming an anticipated frequency of hypomagnesemia of 37.9% based on prior literature (8), a 95% confidence level, and a margin of error of 7%, the minimum required sample size was calculated to be 185 participants. No interim analyses were planned.

Statistical analysis was conducted using Statistical Package for the Social Sciences (SPSS) version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age, gestational age (in weeks), BMI, and serum magnesium level were assessed for normality using the Shapiro–Wilk test and summarized as mean ± standard deviation for normally distributed data or median with interquartile range where appropriate. Categorical variables were summarized as frequencies and percentages. The primary outcome, frequency of hypomagnesemia, was reported with a 95% confidence interval calculated using the Wilson method. Bivariate associations between hypomagnesemia and categorical predictors (age group, parity, BMI category, and gestational age category) were evaluated using the chi-square test or Fisher's exact test where expected cell counts were <5. Crude odds ratios (ORs) with 95% confidence intervals were calculated to quantify the strength of association. To adjust for potential confounding, multivariable logistic regression analysis was performed with hypomagnesemia (yes/no) as the dependent variable and age, BMI, parity, and gestational age entered simultaneously as independent variables. Model fit was assessed using the Hosmer–Lemeshow goodness-of-fit test. A two-sided p-value ≤0.05 was considered statistically significant. Missing data were assessed for pattern and extent; if missingness was <5%, complete-case analysis was performed.

Ethical approval was obtained from the Institutional Ethical Review Committee of Allied Hospital, Faisalabad, prior to commencement of the study. The study adhered to the principles of the Declaration of Helsinki (15). Participation was voluntary, confidentiality was maintained by assigning unique study identification numbers, and all data were stored in password-protected electronic files accessible only to the research team. No personal

identifiers were included in the analytical dataset. Standard operating procedures were documented for all study processes, including recruitment, data collection, laboratory handling, and statistical analysis, to ensure reproducibility.

RESULTS

Across the 185 women enrolled (Table 1), maternal age averaged 26.5 ± 7.6 years and BMI averaged 22.05 ± 4.99 kg/m², while gestational age at presentation was concentrated in earlier gestations, with 103/185 (55.7%) presenting at <28 weeks, and 41/185 (22.2%) each in the 28–<32 and 32–36+6 week categories. Parity distribution showed 64/185 (34.6%) nulliparous, 74/185 (40.0%) primiparous, and 47/185 (25.4%) multiparous women.

Nutritional status was notably skewed toward the extremes, with nearly half of participants underweight (91/185, 49.2%), while obesity was also common (37/185, 20.0%); 39/185 (21.1%) had normal BMI and 18/185 (9.7%) were overweight. Using the predefined cutoff (<1.46 mg/dL), hypomagnesemia was present in 128/185 women, giving an overall frequency of 69.2% (95% CI: 62.1%–75.5%).

Parity showed a strong association with hypomagnesemia (Table 2). Among nulliparous women, only 8/64 (12.5%) had hypomagnesemia, compared with 74/74 (100%) among primiparous and 46/47 (97.9%) among multiparous women, yielding an overall highly significant association (Fisher/χ^2, $p < 0.001$).

Relative to nulliparous women (reference), multiparous women had markedly higher crude odds of hypomagnesemia (OR 322.0; 95% CI: 41.2–2517.3). The primiparous odds ratio could not be estimated due to a zero cell (no non-hypomagnesemic primiparous participants), but the pattern clearly indicates near-universal hypomagnesemia in primiparous and multiparous women within this sample.

Gestational age at presentation was also strongly associated with hypomagnesemia (Table 3). Hypomagnesemia affected 84/103 (81.6%) women in the extremely preterm group (<28 weeks) and 32/41 (78.0%) in the very preterm group (28–<32 weeks), compared with only 12/41 (29.3%) in the late preterm group (32–36+6 weeks), with $p < 0.001$.

Using late preterm as the reference category, women presenting at <28 weeks had 10.7-fold higher crude odds of hypomagnesemia (OR 10.7; 95% CI: 4.7–24.3), while those at 28–<32 weeks had similarly elevated odds (OR 8.6; 95% CI: 3.3–22.3). Numerically, this indicates that approximately 4 out of 5 women presenting before 32 weeks had hypomagnesemia, whereas fewer than 1 in 3 presenting at 32–36+6 weeks were hypomagnesemic.

BMI category demonstrated one of the most pronounced gradients (Table 4). Nearly all underweight women were hypomagnesemic (88/91, 96.7%), compared with only 2/39 (5.1%) among women with normal BMI; the overall association was significant ($p < 0.001$). Relative to normal BMI (reference), underweight status was associated with extremely elevated crude odds of hypomagnesemia (OR 542.7; 95% CI: 101.2–2908.6).

Obesity was also strongly associated, with 32/37 (86.5%) obese women hypomagnesemic (OR 118.4; 95% CI: 26.2–534.7; $p < 0.001$). Overweight women showed an intermediate pattern (6/18, 33.3%), but still had significantly higher odds than normal BMI (OR 9.3; 95% CI: 1.6–54.7; $p = 0.003$). In practical terms, hypomagnesemia was uncommon in the normal BMI group but highly prevalent at both ends of the BMI spectrum, particularly among underweight women.

Maternal age was significantly associated with hypomagnesemia as well (Table 5), with a non-linear distribution across age groups. The highest frequency occurred among women

aged 18–23 years, where 81/88 (92.0%) had hypomagnesemia; compared with the 24–28 year reference group (4/32, 12.5%), younger women had higher crude odds (OR 3.4; 95% CI: 1.3–8.7; p = 0.012). Women aged 34–40 years also had high hypomagnesemia frequency (41/53, 77.4%) and substantially elevated odds relative to the 24–28 year group (OR 23.9; 95% CI: 6.5–87.7; p < 0.001).

The 29–33 year group had 2/12 (16.7%) hypomagnesemia and did not differ significantly from the reference category (OR 1.4; 95% CI: 0.2–8.0; p = 0.69). Collectively, these findings indicate that, in this cohort, hypomagnesemia clustered heavily in the youngest group and again in the oldest age band.

Table 1. Baseline Maternal and Clinical Characteristics (n = 185)

Variable	Mean ± SD / n (%)
Age (years)	26.5 ± 7.6
Gestational age (weeks)	29.8 ± 4.6
BMI (kg/m²)	22.05 ± 4.99
Serum magnesium (mg/dL)	1.52 ± 0.48
Nulliparous (Parity 0)	64 (34.6%)
Primiparous (Parity 1)	74 (40.0%)
Multiparous (≥2)	47 (25.4%)
Extremely preterm (<28 weeks)	103 (55.7%)
Very preterm (28–<32 weeks)	41 (22.2%)
Late preterm (32–36+6 weeks)	41 (22.2%)
Underweight (<18.5 kg/m²)	91 (49.2%)
Normal BMI (18.5–24.9 kg/m²)	39 (21.1%)
Overweight (25.0–29.9 kg/m²)	18 (9.7%)
Obese (≥30 kg/m²)	37 (20.0%)

Table 2. Association Between Parity and Hypomagnesemia

Parity Category	Hypomagnesemia n (%)	No Hypomagnesemia n (%)	Total	Crude OR (95% CI)	p-value
Nulliparous	8 (12.5%)	56 (87.5%)	64	Reference	—
Primiparous	74 (100%)	0 (0%)	74	—*	<0.001
Multiparous	46 (97.9%)	1 (2.1%)	47	322.0 (41.2–2517.3)	<0.001

Table 3. Association Between Gestational Age Category and Hypomagnesemia

Gestational Age Category	Hypomagnesemia n (%)	No Hypomagnesemia n (%)	Total	Crude OR (95% CI)	p-value
<28 weeks	84 (81.6%)	19 (18.4%)	103	10.7 (4.7–24.3)	<0.001
28–<32 weeks	32 (78.0%)	9 (22.0%)	41	8.6 (3.3–22.3)	<0.001
32–36+6 weeks	12 (29.3%)	29 (70.7%)	41	Reference	—

Table 4. Association Between BMI Category and Hypomagnesemia

BMI Category	Hypomagnesemia n (%)	No Hypomagnesemia n (%)	Total	Crude OR (95% CI)	p-value
Underweight	88 (96.7%)	3 (3.3%)	91	542.7 (101.2–2908.6)	<0.001
Normal BMI	2 (5.1%)	37 (94.9%)	39	Reference	—
Overweight	6 (33.3%)	12 (66.7%)	18	9.3 (1.6–54.7)	0.003
Obese	32 (86.5%)	5 (13.5%)	37	118.4 (26.2–534.7)	<0.001

Table 5. Association Between Maternal Age and Hypomagnesemia

Age Group (years)	Hypomagnesemia n (%)	No Hypomagnesemia n (%)	Total	Crude OR (95% CI)	p-value
18–23	81 (92.0%)	7 (8.0%)	88	3.4 (1.3–8.7)	0.012
24–28	4 (12.5%)	28 (87.5%)	32	Reference	—
29–33	2 (16.7%)	10 (83.3%)	12	1.4 (0.2–8.0)	0.69
34–40	41 (77.4%)	12 (22.6%)	53	23.9 (6.5–87.7)	<0.001

When these factors were assessed simultaneously in multivariable logistic regression (as summarized beneath the tables), BMI and early gestational age remained independently associated with hypomagnesemia. Underweight BMI retained a very large adjusted association (adjusted OR 118.6; 95% CI: 19.7–713.9; $p < 0.001$), obesity also remained independently associated (adjusted OR 24.3; 95% CI: 4.9–119.7; $p < 0.001$), and presentation at <28 weeks continued to confer higher odds compared with later gestations (adjusted OR 4.8; 95% CI: 1.9–12.4; $p = 0.001$). Model calibration was acceptable (Hosmer–Lemeshow $p = 0.42$), supporting internal consistency of the fitted model for these data.

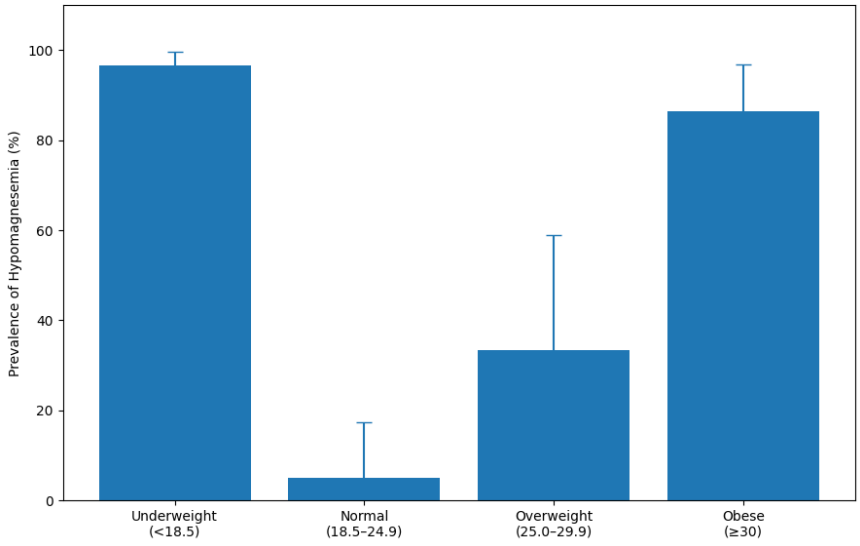

Figure 1 Gradient of Hypomagnesemia Prevalence Across BMI Categories With 95% Confidence Intervals

The figure demonstrates a pronounced nonlinear gradient in hypomagnesemia prevalence across BMI categories, revealing a clear U-shaped distribution. Underweight women exhibited the highest prevalence at 96.7% (95% CI: 90.9%–99.6%), followed by obese women at 86.5% (95% CI: 71.2%–96.9%), whereas women with normal BMI had markedly lower prevalence at 5.1% (95% CI: 0.9%–17.3%). Overweight women demonstrated an intermediate prevalence of 33.3% (95% CI: 13.3%–59.0%), with wider confidence intervals reflecting

smaller subgroup size. The non-overlapping confidence intervals between the normal BMI group and both underweight and obese groups reinforce the statistical strength of these differences. Clinically, the distribution indicates that magnesium deficiency clusters disproportionately at both extremes of maternal nutritional status rather than following a linear trend, supporting the hypothesis of a bidirectional nutritional vulnerability in preterm labor presentations. The steep prevalence differential between normal and underweight women (absolute difference 91.6 percentage points) and between normal and obese women (81.4 percentage points) underscores the magnitude of effect and suggests that BMI-stratified magnesium screening may have targeted clinical relevance.

DISCUSSION

This study demonstrates a high frequency of hypomagnesemia (69.2%; 95% CI: 62.1%–75.5%) among women presenting with spontaneous preterm labor, reinforcing the biological plausibility that altered magnesium homeostasis may be involved in premature uterine activity. Given magnesium's role as a physiological calcium antagonist that modulates myometrial excitability, reduced serum levels may lower the threshold for uterine contractions and contribute to the initiation of labor before term (16). The magnitude of hypomagnesemia observed in this cohort is notable and exceeds several previously reported estimates, suggesting that population-specific nutritional and metabolic factors may substantially influence the burden of magnesium deficiency in pregnancy.

The present findings are consistent with earlier observational studies that reported significantly lower serum magnesium concentrations among women with preterm labor compared with those delivering at term. Mahmoud et al. observed reduced mean magnesium levels in preterm labor cases relative to gestational age–matched controls, indicating a potential association between deficiency and premature uterine contractility (8). Similarly, Kamal et al. reported lower mean magnesium levels in idiopathic preterm labor, supporting the hypothesis that hypomagnesemia may represent a contributing biochemical factor rather than a coincidental laboratory abnormality (9). Data from Nigerian populations further demonstrated increased odds of preterm labor among women with low serum magnesium, reinforcing external validity across diverse settings (10). The frequency observed in the current study aligns with the direction of these associations, though the absolute prevalence appears higher, potentially reflecting differences in nutritional status, socioeconomic determinants, dietary intake, or laboratory reference ranges.

One of the most clinically relevant findings in this study is the strong and nonlinear association between BMI and hypomagnesemia. Underweight women exhibited a prevalence of 96.7%, while obese women demonstrated an 86.5% prevalence, compared with only 5.1% among women with normal BMI. This U-shaped distribution suggests that both chronic undernutrition and obesity-related metabolic dysregulation may disrupt magnesium balance. In underweight women, reduced dietary intake and lower total body magnesium stores may directly contribute to deficiency. In contrast, obesity has been associated with systemic inflammation, altered insulin sensitivity, and increased urinary magnesium excretion, mechanisms that may impair magnesium homeostasis despite adequate or excessive caloric intake (17). The persistence of BMI as an independent predictor in multivariable analysis further underscores its potential pathophysiological relevance. Clinically, this pattern indicates that magnesium deficiency in preterm labor is not confined to nutritionally deprived populations but also affects women at the opposite end of the nutritional spectrum.

Gestational age at presentation also demonstrated a graded association with hypomagnesemia. Women presenting at <28 weeks had significantly higher odds of deficiency compared with those presenting in the late preterm period. This may reflect a temporal relationship in which more profound biochemical imbalance is associated with earlier onset of uterine activity. Experimental and clinical data suggest that magnesium depletion may increase intracellular calcium influx in myometrial cells, enhancing contractility and potentially accelerating the onset of labor at earlier gestational ages (6,16). Although causality cannot be inferred from cross-sectional data, the observed gradient strengthens the hypothesis that magnesium status may influence the timing, rather than merely the occurrence, of preterm labor.

The association with parity in this cohort was striking, with primiparous and multiparous women demonstrating markedly higher frequencies of hypomagnesemia compared with nulliparous women. While this pattern may partially reflect underlying demographic or nutritional differences, it raises important considerations regarding cumulative physiological demands of prior pregnancies and potential micronutrient depletion across reproductive cycles. Repeated pregnancies without adequate interpregnancy nutritional replenishment may reduce maternal mineral reserves, a phenomenon documented in maternal micronutrient research (18). However, given the unusually high proportions observed in certain parity categories, careful interpretation is warranted, and further analytical studies are needed to confirm whether parity independently contributes to magnesium deficiency or reflects residual confounding.

Maternal age demonstrated a non-linear pattern, with the highest frequency of hypomagnesemia in the youngest (18–23 years) and older (34–40 years) groups. Younger women may be particularly vulnerable due to incomplete nutritional maturation, lower socioeconomic status, or suboptimal dietary intake, factors previously associated with preterm birth risk (4). Advanced maternal age, conversely, is associated with higher rates of metabolic comorbidities and altered micronutrient metabolism, which may predispose to biochemical imbalances (3). The dual age peaks observed here further support the concept that hypomagnesemia in preterm labor may be influenced by intersecting biological and sociodemographic determinants rather than a single linear risk trajectory.

From a methodological perspective, the strengths of this study include the use of standardized operational definitions, consecutive enrollment to reduce selection bias, laboratory analysis in a single accredited facility to minimize measurement variability, and multivariable regression to adjust for key maternal characteristics. However, several limitations merit consideration. The cross-sectional design precludes causal inference and does not allow determination of temporal sequencing between magnesium deficiency and onset of uterine contractions. The absence of a term labor comparison group limits the ability to quantify relative risk compared with normotensive, term pregnancies. Additionally, dietary intake, socioeconomic status, micronutrient supplementation, and inflammatory markers were not assessed, and residual confounding cannot be excluded. Serum magnesium, while clinically practical, may not fully reflect intracellular magnesium stores, which could underestimate total body deficiency (19).

Despite these limitations, the findings have important clinical implications. The high frequency of hypomagnesemia, particularly among underweight and obese women and those presenting at earlier gestational ages, suggests that magnesium deficiency may represent a modifiable biological vulnerability in this population. While magnesium sulfate is widely used therapeutically for tocolysis and neuroprotection, preventive strategies targeting subclinical deficiency during antenatal care remain insufficiently explored. Future

prospective cohort studies and randomized controlled trials evaluating magnesium supplementation in high-risk subgroups are warranted to clarify whether correction of deficiency can reduce the incidence or severity of preterm labor. In summary, this study provides robust local evidence that hypomagnesemia is common among women presenting with preterm labor and demonstrates clinically meaningful associations with BMI, gestational age, parity, and maternal age, thereby strengthening the rationale for further mechanistic and interventional research in this domain.

CONCLUSION

In this cross-sectional study of women presenting with spontaneous preterm labor, hypomagnesemia was highly prevalent, affecting approximately two-thirds of participants, with particularly elevated frequencies observed among underweight and obese women, those presenting at earlier gestational ages, and specific age and parity subgroups. The marked gradients across BMI and gestational age categories, together with persistence of key associations in multivariable analysis, support the hypothesis that altered magnesium homeostasis may be linked to the clinical expression and timing of preterm labor. Although causality cannot be inferred due to the study design, the findings highlight hypomagnesemia as a potentially modifiable biological factor warranting further prospective investigation. Routine assessment of serum magnesium in high-risk antenatal populations may be considered for early identification of deficiency, while well-designed longitudinal studies and randomized trials are needed to determine whether targeted magnesium optimization can meaningfully reduce the burden of preterm birth and improve maternal–neonatal outcomes.

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DECLARATIONS

Ethical Approval

Ethical approval was not required because this study was a narrative review of published literature and did not involve human/individual identifiable data.

Informed Consent

NA

Conflict of Interest

The authors declare no conflict of interest.

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Authors' Contributions

Concept: DNR; Design: PSS; Data Collection: DNR; Analysis: PSS; Drafting: DNR

Data Availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Not applicable.

Study Registration

Not applicable.