Journal of Chemical Health Risks

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JCHR (2025) 15(1), 1020-1026 | ISSN:2251-6727

Incidence and Clinical Correlates of Vitamin D Deficiency in Hypertensive Pregnant Women

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Received date: 06/01/2025, ***Revised Date: 20/01/2025*** ***Accepted Date: 03/02/2025***

KEYWORDS

Vitamin D deficiency, Hypertensive disorders of pregnancy, Preeclampsia.

ABSTRACT:

Background: Hypertensive disorders of pregnancy (HDP) are major contributors to maternal and neonatal morbidity and mortality. Emerging evidence suggests that vitamin D deficiency may play a role in their pathogenesis through its effects on vascular endothelial function, inflammation, and placental development.

Aim: To determine the incidence and clinical correlates of vitamin D deficiency in hypertensive pregnant women.

Materials and Methods: A cross-sectional observational study was conducted on 99 hypertensive pregnant women admitted to the Department of Obstetrics and Gynaecology at a tertiary care center. Clinical evaluation, obstetric history, and biochemical assessment of serum 25-hydroxyvitamin D [25(OH)D] levels were performed using a quantitative ELISA method. Vitamin D deficiency was defined as serum 25(OH)D <20 ng/mL. Associations between vitamin D levels, HDP subtypes, and maternal-neonatal outcomes were analyzed using chi-square tests, t-tests, and ANOVA, with $p < 0.05$ considered statistically significant.

Results: Vitamin D deficiency was observed in 76.5% of the participants (95% CI: 68.1-84.9%). The mean serum 25(OH)D concentration was 14.51 ± 7.06 ng/mL. The mean maternal age was 27.31 ± 4.47 years, and the mean birth weight was 2.35 ± 0.72 kg. The prevalence of deficiency was highest among women with superimposed preeclampsia (90.9%) and non-superimposed preeclampsia (89.5%) compared to gestational hypertension (67.4%). ANOVA revealed a significant difference in mean vitamin D levels across HDP types ($F(3,95)=5.34$, $p=0.002$). However, no significant association was found between vitamin D deficiency and mode of delivery ($p=0.636$) or NICU admission ($p=0.213$).

Conclusion: Vitamin D deficiency is highly prevalent among hypertensive pregnant women, with lower serum levels observed in more severe hypertensive subtypes. These findings suggest a potential association between vitamin D status and HDP severity, emphasizing the need for routine screening and preventive supplementation strategies in antenatal care.

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) are among the most common complications encountered during gestation, contributing significantly to maternal and perinatal morbidity and mortality worldwide. The global burden of HDP ranges between 5% and 10% of all pregnancies, and conditions such as gestational hypertension and preeclampsia are recognized precursors

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of future cardiovascular disease in women. In recent years, vitamin D deficiency has emerged as a potentially modifiable factor associated with the pathophysiology of preeclampsia and other hypertensive disorders during pregnancy. Beyond its classical role in calcium homeostasis and bone metabolism, vitamin D exerts pleiotropic effects on immune regulation, angiogenesis, and vascular endothelial function-all of which are critical in maintaining placental health and normal gestational progression.[1]

Vitamin D deficiency during pregnancy is widespread, with reported prevalence ranging from 18% to 84%, depending on geographic location, sunlight exposure, skin pigmentation, and cultural practices that limit sun exposure. In India, where vegetarian diets and conservative clothing are prevalent, vitamin D deficiency among pregnant women has been reported as high as 70%-90%.[2] The deficiency is exacerbated by increased maternal demand and altered metabolism during pregnancy, resulting in reduced circulating 25-hydroxyvitamin D [25(OH)D] levels. Several studies have reported that vitamin D plays a role in modulating placental development by influencing the expression of angiogenic and antiangiogenic factors such as VEGF, PlGF, and soluble fms-like tyrosine kinase-1 (sFlt-1). Deficiency of vitamin D may, therefore, contribute to the endothelial dysfunction and vasospasm characteristic of preeclampsia.[3]

Mechanistically, 1,25-dihydroxyvitamin D acts through genomic and non-genomic pathways to suppress pro-inflammatory cytokines (IL-6, TNF-α), enhance regulatory T-cell function, and reduce renin-angiotensin system activation-mechanisms that collectively help maintain normal vascular tone and placental perfusion. Observational studies have linked low serum 25(OH)D levels with an increased risk and severity of preeclampsia. *et al.*(20)[4] reported a twofold increased risk of preeclampsia in vitamin D-deficient women, while *et al.*(20)[5] demonstrated that mid-gestation deficiency was associated with severe forms of the disease. Conversely, vitamin D supplementation during pregnancy has shown potential benefits, such as improved endothelial function and reduced need for antihypertensive therapy, though results remain inconsistent.

Aim

To determine the incidence and clinical correlates of vitamin D deficiency in hypertensive pregnant women.

Objectives

1. To estimate the prevalence of vitamin D deficiency among pregnant women diagnosed with hypertensive disorders.
2. To evaluate the association between serum vitamin D levels and the severity/type of hypertensive disorder.
3. To correlate maternal vitamin D status with obstetric and neonatal outcomes.

MATERIAL AND METHODOLOGY

Source of Data: The study was conducted among pregnant women diagnosed with hypertensive disorders of pregnancy who attended the Department of Obstetrics and Gynaecology at a tertiary care teaching hospital.

Study Design: A cross-sectional observational study.

Study Location: Department of Obstetrics and Gynaecology, a tertiary care centre catering to urban and rural populations.

Study Duration: The study was conducted over 18 months, from January 2023 to June 2024.

Sample Size: 99 pregnant women meeting the inclusion criteria were enrolled.

Inclusion Criteria:

- Pregnant women aged 18-40 years.
- Diagnosed with hypertensive disorders of pregnancy (gestational hypertension, preeclampsia, or eclampsia).
- Gestational age ≥28 weeks.
- Provided informed consent.

Exclusion Criteria:

- Women with pre-existing chronic renal, hepatic, or metabolic disorders.
- Women already on vitamin D supplementation or calcium therapy.
- Multiple pregnancies.

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- Known endocrine disorders (e.g., thyroid or parathyroid diseases).

Procedure and Methodology: All participants were evaluated clinically and biochemically at admission. Detailed obstetric and medical history was recorded, including age, parity, gestational age, BMI, and type of hypertensive disorder. Blood pressure was measured using a validated sphygmomanometer, and HDP was classified as per ACOG guidelines. Blood samples (5 mL) were collected under aseptic conditions and centrifuged for serum separation. Serum 25-hydroxyvitamin D [25(OH)D] concentrations were measured using a quantitative ELISA kit (Calbiotech, USA). Levels <20 ng/mL were classified as deficient, 20-30 ng/mL as insufficient, and >30 ng/mL as sufficient.

Sample Processing: Serum samples were processed within two hours of collection and stored at −20°C until analysis. Internal quality control was ensured using standard reference samples.

Statistical Methods: Data were analyzed using SPSS version 25. Descriptive statistics (mean ± SD, frequencies, and percentages) were calculated. The association between vitamin D status and categorical variables (e.g., type of HDP, mode of delivery, NICU admission) was assessed using chi-square or Fisher's exact test. Continuous variables were compared using t-test or ANOVA as appropriate. A p-value <0.05 was considered statistically significant.

Data Collection: Data were collected using a structured proforma, including demographic details, obstetric history, vitamin D levels, type of hypertension, maternal complications, and neonatal outcomes. Confidentiality was maintained throughout the study, and ethical clearance was obtained from the institutional ethics committee.

OBSERVATION AND RESULTS

Table 1: Incidence and clinical correlates of vitamin D deficiency in hypertensive pregnant women

Variable	n/N (%) or Mean ± SD	95% CI	Test of significance
Vitamin D deficiency (<20 ng/mL)	75/98 (76.53%)	68.1-84.9%	
Serum 25(OH)D (ng/mL)	14.51 ± 7.06 (N=99)	13.12-15.90	
Maternal age (years)	27.31 ± 4.47 (N=99)	26.42-28.20 (reported)	
NICU admission (any)	27/99 (27.27%)	18.4-36.1%	
Birth weight (kg)	2.35 ± 0.72 (N=99)	2.21-2.49	
Deficiency vs. PIH type	-	-	χ^2=7.45, df=3, *P*=0.059 (NS)
Deficiency vs. mode of delivery	-	-	χ^2=4.30, df=6, *P*=0.636 (NS)

Notes: Incidence row uses N=98 due to one missing vitamin-D value. CI for deficiency uses a binomial approximation; CIs for means use mean ± 1.96·SE.

Table 1 presents the incidence and principal clinical correlates of vitamin D deficiency among 99 hypertensive pregnant women. The overall incidence of deficiency (serum 25(OH)D < 20 ng/mL) was high, affecting 76.53% of the study cohort (95% CI: 68.1-84.9%), confirming that three-quarters of women with hypertensive disorders were vitamin D-deficient. The mean serum 25(OH)D concentration was 14.51 ± 7.06 ng/mL (95% CI: 13.12-15.90), which is well below the sufficiency threshold. The mean maternal age was 27.31 ± 4.47 years (95% CI: 26.42-28.20), reflecting a relatively young obstetric population. Neonatal intensive care unit (NICU) admission was required in 27.27% of cases (95% CI: 18.4-36.1%), and the mean birth weight was 2.35 ± 0.72 kg (95% CI: 2.21-2.49), indicating a predominance of low-birth-weight neonates. When vitamin D deficiency status was compared with the type of hypertensive disorder, a mild, non-significant trend was observed ($\chi^2 = 7.45$, df = 3, $p = 0.059$). Similarly, no statistically significant relationship was found between

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JCHR (2025) 15(1), 1020-1026 | ISSN:2251-6727

vitamin D status and the mode of delivery (χ^2 = 4.30, df = 6, p = 0.636).

Table 2: Prevalence of vitamin D deficiency among women with hypertensive disorders (overall and by PIH type)

Group	Deficient n/N (%)	95% CI	Test of significance
Overall	75/98 (76.53%)	68.1-84.9%	
Impending eclampsia (IE)	9/14 (64.3%)	38.8-83.7%	χ^2 across 4 groups = 7.45, df=3, P=0.059 (NS)
Superimposed pre-eclampsia (SPE)	20/22 (90.9%)	72.2-97.5%	
Non-superimposed pre-eclampsia (NSPE)	17/19 (89.5%)	66.6-97.3%	
Gestational hypertension (GHTN)	29/43 (67.4%)	52.3-79.3%	

Table 2 details the prevalence of vitamin D deficiency among the different clinical subtypes of hypertensive disorders of pregnancy (HDP). The overall prevalence remained at 76.53% (95% CI: 68.1-84.9%). Sub-categorization revealed that deficiency was most frequent in women with superimposed pre-eclampsia (90.9%), followed closely by non-superimposed pre-eclampsia (89.5%), whereas gestational hypertension (67.4%) and impending eclampsia (64.3%) demonstrated relatively lower frequencies. The inter-group comparison yielded a non-significant chi-square statistic (χ^2 = 7.45, df = 3, p = 0.059), indicating that while vitamin D deficiency tended to be more pronounced in severe forms of HDP, statistical evidence for a graded association across categories was borderline.

Table 3: Association between serum vitamin D levels (ng/mL) and PIH severity/type (ANOVA)

PIH type	n	Mean ± SD	95% CI for mean
Impending eclampsia (IE)	14	15.77 ± 10.36	10.34-21.20
Superimposed pre-eclampsia (SPE)	23	9.95 ± 6.54	7.29-12.61
Non-superimposed pre-eclampsia (NSPE)	19	14.18 ± 5.67	11.63-16.73
Gestational hypertension (GHTN)	43	16.68 ± 5.50	15.03-18.33
Overall	99	14.51 ± 7.06	13.12-15.90

Test of significance: One-way ANOVA: F(3,95)=5.34, P=0.002 (significant difference in 25(OH)D across PIH categories).

Table 3 summarizes the association between mean serum vitamin D levels and the severity/type of hypertensive disorder, analyzed by one-way ANOVA. The lowest vitamin D levels were observed in superimposed pre-eclampsia (9.95 ± 6.54 ng/mL), followed by non-superimposed pre-eclampsia (14.18 ± 5.67 ng/mL) and impending eclampsia (15.77 ± 10.36 ng/mL), whereas women with gestational hypertension had relatively higher mean levels (16.68 ± 5.50 ng/mL). The overall mean for the cohort was 14.51 ± 7.06 ng/mL. Statistical analysis revealed a significant difference in mean 25(OH)D levels across these four groups (F(3, 95) = 5.34, p = 0.002), confirming that lower vitamin D concentrations are associated with greater disease severity.

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Table 4: Maternal vitamin D status vs. obstetric and neonatal outcomes

Outcome / Comparison	Group 1	Group 2	Effect (95% CI)	Test of significance
Mode of delivery vs. deficiency	7-level mode distribution in deficient (n=75)	Same in non-deficient (n=23)	-	χ^2=4.30, df=6, *P*=0.636 (NS)
NICU admission	Yes: 27/99 (27.27%)	No: 72/99 (72.73%)	-	
Vitamin D level by NICU admission	NICU Yes: 12.77 ± 9.09 (n=27)	NICU No: 15.17 ± 6.08 (n=72)	Mean diff = −2.40 (approx. −5.55 to +0.75)	*t*(97)=−1.52, *P*=0.133 (NS); unequal-variance *P*=0.213.
Birth weight (kg)	-	-	2.35 ± 0.72 (95% CI 2.21-2.49)	

Table 4 examines maternal vitamin D status in relation to obstetric and neonatal outcomes. No significant relationship was observed between vitamin D deficiency and mode of delivery ($\chi^2 = 4.30$, df = 6, $p = 0.636$). NICU admission was required for 27.27% of neonates, but the mean maternal vitamin D level among these women (12.77 ± 9.09 ng/mL) was only slightly lower than that of mothers whose infants did not require NICU care (15.17 ± 6.08 ng/mL), and this difference was statistically non-significant ($t(97) = -1.52$, $p = 0.133$; unequal-variance $p = 0.213$). The overall mean birth weight of 2.35 ± 0.72 kg further reflects the adverse perinatal milieu associated with hypertensive disorders. In summary, although vitamin D deficiency was prevalent and showed a declining trend with increasing severity of HDP, its independent association with delivery outcomes and neonatal morbidity did not reach statistical significance in this cohort.

DISCUSSION

In cohort of hypertensive pregnancies, three-quarters of women were vitamin-D deficient (75/98; 76.5%, 95% CI 68.1-84.9), and the mean 25(OH)D concentration was low (14.51 ± 7.06 ng/mL), underscoring a substantial deficiency burden in this clinical group. These findings are directionally consistent with Indian and South-Asian reports where antenatal hypovitaminosis-D frequently exceeds 60-70% in routine obstetric populations, and may be yet higher in high-risk groups Lv J *et al.*(2022)[6]. The deficiency prevalence we observed aligns with global estimates from meta-analyses indicating widespread antenatal insufficiency and deficiency, though absolute percentages vary with latitude, skin pigmentation, diet, and clothing practices Fogacci S *et al.*(2020)[7]. Data therefore reinforce that vitamin-D scarcity is common among women with hypertensive disorders of pregnancy (HDP).

When deficiency status was cross-tabulated against HDP subtypes, we noted high proportions of deficiency in superimposed pre-eclampsia (SPE, 90.9%) and non-superimposed pre-eclampsia (NSPE, 89.5%), with relatively lower proportions in gestational hypertension (GHTN, 67.4%) and impending eclampsia (IE, 64.3%). The overall χ^2 across categories was borderline (χ^2=7.45, df=3, *p*=0.059), suggesting a trend toward more deficiency in the pre-eclampsia phenotypes without achieving conventional significance-likely reflecting limited power for between-group proportion tests Zhang H *et al.*(2022)[8]. Notably, however, mean vitamin-D levels differed significantly across HDP types on ANOVA ($F(3,95)=5.34$, $p=0.002$): the lowest values occurred in SPE (9.95 ± 6.54 ng/mL), intermediate values in NSPE (14.18 ± 5.67 ng/mL) and IE (15.77 ± 10.36 ng/mL), and the highest in GHTN (16.68 ± 5.50 ng/mL). This graded pattern coheres with biological models positing that lower 25(OH)D could amplify angiogenic imbalance and endothelial dysfunction-prominent in pre-eclampsia-more than in milder hypertensive phenotypes Caccamo D *et al.*(2020)[9].

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Despite strong between-group differences in mean 25(OH)D, deficiency per se did not show a significant association with mode of delivery (seven-level comparison; χ^2=4.30, df=6, p=0.636). Likewise, maternal 25(OH)D was only modestly lower among infants requiring NICU care (12.77 ± 9.09 ng/mL) than among those who did not (15.17 ± 6.08 ng/mL), and this difference was statistically non-significant (equal-variance $t(97)$=−1.52, p=0.133; Welch p=0.213). These null associations could reflect confounding by indication (e.g., decision pathways for urgent delivery), multifactorial determinants of neonatal morbidity in HDP (placental insufficiency, iatrogenic prematurity), and sample-size constraints for outcome contrasts Das B *et al.*(2021)[10]. Still, the overall mean birth weight in the cohort was low (2.35 ± 0.72 kg), consistent with the well-described growth-restriction milieu in hypertensive pregnancies, wherein vitamin-D status is one of several contributors rather than a sole driver Mokhtari E *et al.*(2022)[11].

CONCLUSION

The present study demonstrated a high incidence of vitamin D deficiency among hypertensive pregnant women, with over three-fourths of the study population exhibiting serum 25(OH)D levels below the sufficiency threshold. The mean vitamin D concentration was markedly low, indicating that deficiency is a common comorbidity in hypertensive disorders of pregnancy (HDP). Although no statistically significant correlation was observed between vitamin D deficiency and the type of hypertensive disorder, mean serum vitamin D levels were found to decrease progressively with increasing disease severity, being lowest among women with superimposed preeclampsia. This suggests a potential contributory role of vitamin D in the pathophysiology of endothelial dysfunction and placental insufficiency seen in HDP. Neonatal outcomes, including birth weight and NICU admissions, did not differ significantly across vitamin D status categories, implying that other clinical factors may mediate perinatal risk. Overall, these findings highlight the high burden of hypovitaminosis D in hypertensive pregnancies and underscore the need for early screening and possible supplementation strategies to optimize maternal and fetal outcomes.

LIMITATIONS OF THE STUDY

1. The study was conducted at a single tertiary care center, limiting generalizability to other populations with differing sunlight exposure, dietary habits, and ethnic profiles.
2. The cross-sectional design precludes establishing causal relationships between vitamin D deficiency and hypertensive disorders.
3. The sample size (n=99) may have been inadequate to detect smaller intergroup differences or associations with neonatal outcomes.
4. Potential confounding factors such as dietary calcium intake, BMI, seasonal variation, and socioeconomic status were not controlled for.
5. Vitamin D status was assessed only once during pregnancy, and longitudinal variations in serum levels were not evaluated.

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