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Association of Maternal Vitamin D Deficiency with Severity of Hypertensive Disorders of Pregnancy

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KEYWORDS

Vitamin D deficiency; Hypertensive disorders of pregnancy; Preeclampsia.

ABSTRACT:

Background: Vitamin D deficiency has been implicated in the pathogenesis of hypertensive disorders of pregnancy (HDP) through its effects on placental function, vascular health, and immune modulation. However, its relationship with disease severity remains inadequately characterized in Indian populations.

Aim: To assess the association of maternal vitamin D deficiency with the severity of hypertensive disorders of pregnancy.

Materials and Methods: This cross-sectional analytical study included 99 pregnant women diagnosed with HDP at a tertiary care hospital. Participants were categorized into gestational hypertension (GHTN), non-superimposed preeclampsia (NSPE), superimposed preeclampsia (SPE), and impending eclampsia (IE). Serum 25-hydroxyvitamin D levels were measured using chemiluminescent immunoassay (CLIA). Vitamin D deficiency was defined as <20 ng/mL. Maternal and fetal outcomes, including mode of delivery, NICU admission, and birth weight, were recorded. Data were analyzed using chi-square, ANOVA, and t-tests with significance set at $p < 0.05$.

Results: The overall prevalence of vitamin D deficiency was 76.5% (95% CI 67.2-83.8), with mean serum 25(OH)D = 14.51 ± 7.06 ng/mL. Deficiency was most frequent in SPE (90.9%) and NSPE (89.5%) compared to GHTN (67.4%). One-way ANOVA revealed a significant difference in vitamin D levels across HDP categories ($F = 5.34$, $p = 0.002$), with the lowest levels observed in severe preeclampsia. Maternal and neonatal outcomes showed no statistically significant correlation with vitamin D status ($p > 0.05$), although lower vitamin D tended to associate with preterm birth and NICU admission.

Conclusion: Vitamin D deficiency is highly prevalent among women with hypertensive disorders of pregnancy and is significantly associated with disease severity, particularly in preeclampsia. Screening and correction of vitamin D deficiency during antenatal care may serve as a cost-effective adjunct in reducing HDP-related morbidity.

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) are among the leading causes of maternal and perinatal morbidity and mortality worldwide, encompassing gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia. These disorders complicate approximately 5-10% of all pregnancies and are major contributors to preterm delivery, intrauterine growth restriction (IUGR), and perinatal death. Although the exact pathogenesis of HDP is multifactorial and not yet completely elucidated, endothelial dysfunction, abnormal placentation, and exaggerated systemic inflammatory response play pivotal roles in their development.[1]

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Vitamin D, traditionally known for its role in calcium homeostasis and skeletal health, has been increasingly recognized as an immunomodulatory, anti-inflammatory, and vasculoprotective agent influencing multiple physiological systems. During pregnancy, adequate maternal vitamin D levels are crucial for implantation, placental development, and fetal growth. Active vitamin D [$1,25(OH)_2D_3$] modulates angiogenesis by upregulating vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), while suppressing anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1). Deficiency in vitamin D may thus disrupt this angiogenic balance, leading to impaired trophoblastic invasion, placental hypoperfusion, and systemic endothelial injury-hallmarks of preeclampsia.[2]

Several studies have demonstrated an association between low maternal serum 25-hydroxyvitamin D levels and the occurrence and severity of hypertensive disorders. *et al.* (20)[3] reported that women with vitamin D deficiency were twice as likely to develop preeclampsia compared to those with sufficient levels. Similarly, *et al.* (20)[4] observed that midgestation vitamin D deficiency was significantly correlated with severe preeclampsia. In contrast, a few Mendelian randomization studies have failed to establish causality, suggesting that vitamin D deficiency might act as a marker rather than a direct etiologic factor. However, emerging evidence supports the role of vitamin D in regulating the renin-angiotensin system, modulating immune tolerance at the maternal-fetal interface, and reducing systemic inflammation, thereby implicating it as a modifiable determinant of adverse pregnancy outcomes.

Vitamin D deficiency during pregnancy is widespread, affecting 40-80% of women globally, particularly in South Asia, where factors such as limited sunlight exposure, darker skin pigmentation, and vegetarian diets compound the risk. In India, the prevalence of hypovitaminosis D among pregnant women is alarmingly high (70-90%), despite abundant sunlight. This deficiency has been correlated not only with HDP but also with gestational diabetes mellitus, bacterial vaginosis, low birth weight, and neonatal rickets. Given the physiological upsurge in vitamin D demand during pregnancy and its potential role in mitigating placental dysfunction, assessing maternal vitamin D status in HDP becomes essential for early identification and preventive intervention.[5]

Aim

To assess the association of maternal vitamin D deficiency with the severity of hypertensive disorders of pregnancy.

Objectives

1. To determine the prevalence of vitamin D deficiency among pregnant women diagnosed with hypertensive disorders.
2. To correlate serum vitamin D levels with the severity of hypertensive disorders of pregnancy.
3. To evaluate maternal and fetal outcomes in relation to maternal vitamin D status.

MATERIAL AND METHODOLOGY

Source of Data: This study included all antenatal women diagnosed with hypertensive disorders of pregnancy attending the Department of Obstetrics and Gynaecology at a tertiary care teaching hospital.

Study Design: A cross-sectional analytical study.

Study Location: Department of Obstetrics and Gynaecology, a tertiary care centre in Western India.

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

Sample Size: A total of 99 pregnant women fulfilling the inclusion criteria were enrolled.

Inclusion Criteria:

- Pregnant women ≥20 weeks of gestation diagnosed with hypertensive disorders of pregnancy (gestational hypertension, preeclampsia, eclampsia, chronic hypertension with superimposed preeclampsia).
- Singleton pregnancies.
- Patients who provided written informed consent.

Exclusion Criteria:

- Patients with pre-existing renal, hepatic, or cardiovascular disease.
- Known metabolic bone disease or chronic steroid therapy.
- Multifetal gestation.
- Women on prior vitamin D supplementation.

Procedure and Methodology: Eligible women were recruited after clinical evaluation and confirmation of diagnosis based on the ACOG guidelines for hypertensive disorders of pregnancy. Detailed obstetric, medical, and nutritional histories were obtained. Blood pressure measurements were taken using calibrated sphygmomanometers in the sitting position after adequate rest. Severity of preeclampsia was determined

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by blood pressure levels, proteinuria (measured by dipstick and 24-hour urine protein), and the presence of end-organ involvement.

Venous blood samples (5 mL) were collected under aseptic precautions. Serum was separated by centrifugation and stored at -20°C until analysis. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using chemiluminescent immunoassay (CLIA) kits. Based on the Endocrine Society's criteria, vitamin D status was classified as: sufficient (>30 ng/mL), insufficient (20-30 ng/mL), and deficient (<20 ng/mL). Relevant laboratory investigations such as liver and renal function tests, complete blood counts, serum calcium, and uric acid were also performed.

Sample Processing: All samples were processed in the institutional biochemistry laboratory following standard operating procedures. Internal quality controls and calibration protocols were maintained throughout the study.

Data Collection: Clinical, biochemical, and obstetric data were recorded in predesigned proformas, including maternal age, parity, gestational age at delivery, type and severity of HDP, mode of delivery, neonatal birth weight, NICU admission, and perinatal outcomes.

Statistical Methods: Data were entered in Microsoft Excel and analysed using SPSS version 25. Descriptive statistics (mean, SD, frequencies, percentages) were computed. Categorical variables were compared using Chi-square or Fisher's exact tests. Continuous variables were compared using Student's t-test or ANOVA, as appropriate. Correlation between vitamin D levels and severity of HDP was assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

OBSERVATION AND RESULTS

Table 1: Association of maternal vitamin D deficiency with severity of hypertensive disorders of pregnancy (HDP)

HDP category	n	Vitamin D deficient n (%)	95% CI for %
Gestational Hypertension (GHTN)	43	29 (67.4)	52.5-79.5
Non-superimposed Preeclampsia (NSPE)	19	17 (89.5)	68.6-97.1
Superimposed Preeclampsia (SPE)	22	20 (90.9)	72.2-97.5
Impending Eclampsia (IE)	14	9 (64.3)	38.8-83.7
Total	**98***	**75 (76.5)**	**67.2-83.8**

Test of association: $\chi^2(3)=7.45$, **p=0.059** (not statistically significant at 0.05).

Table 1 demonstrates the distribution of vitamin D deficiency across the four major categories of hypertensive disorders in pregnancy. Among 98 women whose vitamin D levels were available, the overall prevalence of deficiency was 76.5 % (95 % CI 67.2-83.8). Within sub-groups, deficiency was noted in 67.4 % of women with gestational hypertension (GHTN), 89.5 % with non-superimposed pre-eclampsia (NSPE), 90.9 % with superimposed pre-eclampsia (SPE), and 64.3 % with impending eclampsia (IE). The chi-square test for association between vitamin D deficiency and HDP type yielded χ^2 (3)= 7.45, p = 0.059, indicating a trend toward higher prevalence of deficiency in severe forms of HDP, although the result did not reach conventional statistical significance ($p < 0.05$). When pre-eclampsia cases (SPE + NSPE) were pooled and compared with GHTN, the odds of vitamin-D deficiency were about 4.5-fold higher (OR = 4.47; 95 % CI 1.33-15.02)

Table 2: Prevalence of vitamin D deficiency among women with HDP

Measure	Value
Deficient (<20 ng/mL) - n/N (%)	75/98 (76.5%)
95% CI for prevalence	67.2% to 83.8%
Serum 25(OH)D (ng/mL), mean (SD)	14.51 (7.06)
95% CI for mean	13.10 to 15.92

Table 2 quantifies the overall burden of vitamin D deficiency in the cohort. Of the 98 women assessed, 75 were deficient (< 20 ng/mL), corresponding to a prevalence of 76.5 % (95 % CI 67.2-83.8). The mean serum 25(OH)D concentration was 14.51 ± 7.06 ng/mL (95 % CI 13.10-15.92), well below the accepted sufficiency threshold. These results confirm a high frequency of maternal hypovitaminosis D among women with hypertensive disorders, consistent with previous Indian studies reporting a similar pattern in late-gestation HDP populations.

Table 3: Correlation of serum vitamin D levels with severity of HDP

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HDP category	n	Mean 25(OH)D (ng/mL)	SD	95% CI for mean
GHTN	43	16.68	5.50	14.99-18.37
NSPE	19	14.18	5.67	11.45-16.91
SPE	23	9.95	6.54	7.12-12.78
IE	14	15.77	10.36	9.79-21.75
Overall	**99**	**14.51**	**7.06**	**13.10-15.92**

One-way ANOVA: F(3,95)=5.34, p=0.002.

Table 3 explores mean 25(OH)D levels according to the severity and type of hypertensive disorder. The average serum 25(OH)D level progressively declined with increasing disease severity-from 16.68 ± 5.50 ng/mL in GHTN to 14.18 ± 5.67 ng/mL in NSPE, reaching the lowest mean of 9.95 ± 6.54 ng/mL in SPE. Women with impending eclampsia (IE) had a mean level of 15.77 ± 10.36 ng/mL, showing wide inter-individual variation. The overall mean (14.51 ± 7.06 ng/mL) reinforces the deficient status of the cohort. One-way ANOVA demonstrated a statistically significant difference among groups (F = 5.34, df = 3,95, p = 0.002), confirming that serum vitamin D concentrations differed significantly by HDP category, with the lowest levels observed in severe pre-eclampsia.

Table 4: Maternal & fetal outcomes in relation to maternal vitamin D status

A. Mode of delivery by vitamin-D status

Mode of delivery	Deficient (n=75)	Non-deficient (n=23)	Total
Emergency LSCS	40	12	52
Full-term vaginal delivery	17	9	26
Preterm vaginal delivery	12	2	14
Preterm Emergency LSCS	2	0	2
Elective LSCS	2	0	2
Vacuum-assisted vaginal delivery	1	0	1
Full-term normal delivery (FTND)†	1	0	1
Total	**75**	**23**	**98***

Chi-square: $\chi^2(6)=4.30$, **p=0.636** (no association).

B. Neonatal NICU admission vs maternal vitamin-D (continuous)

Outcome	n	Mean 25(OH)D (ng/mL)	SD
NICU admission - Yes	27	12.77	9.09
NICU admission - No	72	15.17	6.08

Two-sample t test: equal-variances t(97)=-1.52, p=0.133 (Cohen's d=0.34). Welch t also non-significant: t(35.1)=-1.27, p=0.213.

C. Birth weight (overall)

Mean (SD) birth-weight at delivery: **2.35 (0.72) kg**.

Table 4 summarises obstetric outcomes and neonatal parameters stratified by maternal vitamin D status. In the deficient group (n = 75), emergency lower-segment caesarean section (LSCS) was the predominant mode of delivery (53.3 %), compared with 52.2 % in the non-deficient group. Full-term vaginal delivery occurred in 22.7 % of deficient versus 39.1 % of non-deficient women, while preterm deliveries and operative interventions (preterm LSCS or vacuum-assisted births) were slightly more frequent in the deficient cohort. However, the association between vitamin D status and mode of delivery was not statistically significant (χ^2 (6)= 4.30, p = 0.636).

Regarding neonatal outcomes, 27 newborns (27.3 %) required NICU admission. Their mothers had lower mean vitamin D levels (12.77 ± 9.09 ng/mL) compared with mothers of non-admitted infants (15.17 ± 6.08 ng/mL), though the difference did not achieve statistical significance (t(97)= -1.52, p = 0.133; 95 % CI -6.24 to +1.44). The mean birth weight across the cohort was 2.35 ± 0.72 kg, denoting a predominance of low-birth-weight infants.

DISCUSSION

Table 1 (vitamin-D deficiency across HDP categories). In cohort, vitamin-D deficiency (<20 ng/mL) was common (overall 76.5%, 95% CI 67.2-83.8) and most frequent in pre-eclampsia phenotypes-NSPE 89.5% and

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SPE 90.9%-versus GHTN 67.4% and IE 64.3%. Although the overall association narrowly missed conventional significance ($\chi^2(3)=7.45$, p=0.059), the directionality is consistent with biologic plausibility and prior reports linking low 25(OH)D to placental angiogenic imbalance and more severe disease.

Pooled exploratory contrast (PE vs GHTN) showed 4.5-fold higher odds of deficiency (OR 4.47, 95% CI 1.33-15.02), reinforcing the gradient toward lower vitamin-D in clinically more severe HDP. This accords with Osman OM *et al.* (2020)[6], who observed higher pre-eclampsia risk with deficiency, and Das B *et al.* (2021)[7], who reported mid-gestation deficiency associated with severe pre-eclampsia. Guo Y *et al.* (2023)[8] found low 25(OH)D in early-onset severe PE, and Alanazi M *et al.* (2022)[9] linked maternal vitamin-D status to angiogenic markers and PE risk. By contrast, Mendelian-randomization work Lv J *et al.* (2022)[10] has not confirmed causality, suggesting vitamin-D may be a marker of upstream placental/immune perturbations rather than a sole driver. Indian cohorts report similar or higher deficiency burdens in HDP: Suárez-Varela MM *et al.* (2022)[11] documented markedly lower 25(OH)D in hypertensive vs normotensive pregnancies, and Olapeju B *et al.* (2020)[12] reported an ≈88% deficiency rate among HDP cases.

Table 2 (prevalence of deficiency). Prevalence was 76.5% (75/98) with a mean 25(OH)D of 14.51 ± 7.06 ng/mL, underscoring a high background of hypovitaminosis D in HDP at centre.

This mirrors South-Asian data and aligns with reports of widespread antenatal deficiency despite abundant sunlight exposure [6,7]. Such a high baseline may attenuate detectable between-group contrasts (a "floor effect") and partly explain the borderline p-value in Table 1.

Table 3 (vitamin-D by HDP severity). Mean 25(OH)D differed significantly across HDP categories (ANOVA $F(3,95)=5.34$, p=0.002), with the lowest levels in SPE (9.95 ± 6.54 ng/mL), intermediate in NSPE (14.18 ± 5.67) and IE (15.77 ± 10.36), and highest in GHTN (16.68 ± 5.50).

This gradient maps onto mechanistic pathways-vitamin-D's immunomodulatory and vasculoprotective actions, including down-regulation of sFlt-1/soluble endoglin and up-regulation of VEGF/PlGF-described by Dahma G *et al.* (2022)[13] and earlier immunologic/placental work. Together with Table 1's effect-direction, these data support an association between lower vitamin-D and more severe HDP phenotypes.

Table 4 (maternal & fetal outcomes). Mode of delivery did not differ by vitamin-D status ($\chi^2(6)=4.30$, p=0.636), despite numerically more emergency LSCS in the deficient group (53% vs 52%).

For neonatal outcomes, mothers of NICU-admitted newborns had lower mean 25(OH)D (12.77 ± 9.09) than those without NICU admission (15.17 ± 6.08), but differences were non-significant ($t(97)=-1.52$, p=0.133; mean diff -2.40 ng/mL, 95% CI -6.24 to +1.44).

Cohort birth-weight averaged 2.35 ± 0.72 kg, reflecting a high LBW burden typical of HDP populations.

Null findings here may reflect sample size/power and confounding by gestational age at delivery and disease severity, both of which strongly determine delivery mode and NICU need irrespective of vitamin-D status. Prior studies variably link low maternal 25(OH)D to adverse perinatal endpoints, but effect sizes often attenuate after adjustment for HDP severity and prematurity Giourga C *et al.* (2023)[14].

CONCLUSION

The present study demonstrated a high prevalence of vitamin D deficiency among women with hypertensive disorders of pregnancy (HDP), with 76.5% of participants showing deficient serum 25(OH)D levels. Although the overall association between vitamin D deficiency and HDP subtype narrowly missed statistical significance, women with preeclampsia-both superimposed and non-superimposed-showed markedly lower vitamin D concentrations compared to those with gestational hypertension. Mean serum vitamin D levels were significantly lower in severe cases, particularly in superimposed preeclampsia (9.95 ± 6.54 ng/mL), suggesting a dose-response relationship between declining vitamin D status and disease severity. Maternal and neonatal outcomes, such as mode of delivery, NICU admission, and low birth weight, did not show statistically significant associations with vitamin D status, although trends toward poorer outcomes in the deficient group were observed. These findings support the hypothesis that vitamin D deficiency may play an important contributory role in the pathophysiology and severity of HDP through its influence on endothelial function, angiogenesis, and placental development. Routine screening and targeted supplementation of vitamin D during antenatal care could be considered as a preventive measure, particularly for women at high risk of preeclampsia.

LIMITATIONS OF THE STUDY

1. **Sample size constraint:** The study included 99 participants, which may have limited the power

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to detect small but clinically meaningful differences between HDP subgroups.

2. **Single-center design:** Conducted at one tertiary care hospital, the results may not be generalizable to populations with different nutritional, genetic, or environmental profiles.
3. **Cross-sectional nature:** As serum vitamin D was assessed after disease onset, causal inference between vitamin D deficiency and HDP severity cannot be established.
4. **Confounding factors:** Potential confounders such as dietary intake, sunlight exposure, BMI, socioeconomic status, and seasonality were not fully controlled.
5. **Lack of supplementation data:** Information on prior vitamin D or calcium supplementation during early pregnancy was limited.
6. **Single-point measurement:** Serum 25(OH)D was measured once during pregnancy, which might not accurately represent longitudinal vitamin D status throughout gestation.

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