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RESEARCH ARTICLE

Correlation of LV Mass Index and Its Parameters with Vitamin D Levels in Essential Hypertension

Dr. Manjul Chopra[1], Dr. Savita Kumari[2], Dr. Kashish Narula[3] and Dr. Devanshi Thakkar[4]

[1]Junior Resident-III, Department of General Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Haryana, India
[2]Professor and Head of Unit, Department of General Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Haryana, India
[3]Assistant Professor, Department of General Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research (MMIMSR), Mullana, Haryana, India
[4]Junior Resident-II, Department of Pharmacology, King George's Medical University (KGMU), Lucknow, Uttar Pradesh, India

*Corresponding Author
Dr. Manjul Chopra

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Abstract: ***Background***: Essential hypertension is a leading risk factor for cerebrovascular, renal and cardiovascular morbidity, left ventricular hypertrophy (LVH) is a key marker of adverse outcomes. Emerging evidence suggests vitamin D deficiency contributes to cardiac remodeling via modulation of the renin–angiotensin–aldosterone system, calcium balance, and endothelial function. ***Objective***: To evaluate the correlation between serum vitamin D levels with left ventricular mass index (LVMI) and its parameters in patients with essential hypertension. ***Methods***: A hospital-based observational study was conducted on 100 patients with essential hypertension at MMIMSR, Ambala (January 2023–March 2025). Demographic, clinical, and biochemical data were collected. Serum 25(OH)D was measured and classified as deficient (<20 ng/mL), insufficient (20–30 ng/mL), or sufficient (≥30 ng/mL). Echocardiography was performed to assess LVMI, interventricular septal thickness (IVST), posterior wall thickness (PWD), and end-diastolic diameter (EDD). Statistical analyses included ANOVA, Chi-square, and Pearson's correlation. ***Results***: Vitamin D deficiency was highly prevalent (62%), with an additional 20% insufficient. Abnormal LVMI was found in 66% overall, with higher prevalence in the deficient group (71%) compared to insufficient (60%) and sufficient (55.6%), though not statistically significant (p = 0.391). Abnormal PWD was significantly associated with vitamin D deficiency (96.8% vs. 77.8%, p = 0.031). IVST and EDD showed no significant associations. Proteinuria was more frequent in deficient patients (p = 0.038), reflecting renal target organ damage. ***Conclusion***: In India Vitamin D deficiency is widespread in general population, however it is significantly more among hypertensive patients and is associated with adverse echocardiographic changes, particularly posterior wall thickening. While LVMI and IVST trends indicated higher values in deficient groups, statistical significance was not reached. These findings highlight vitamin D as a potentially modifiable factor in reducing hypertensive target organ damage and cardiovascular risk.

Keywords: Essential hypertension, Vitamin D deficiency, Left ventricular hypertrophy, LV mass index, Echocardiography.

INTRODUCTION

Hypertension, defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg (JNC-7, 2003) is a major global health burden. Prehypertension (SBP 120–139 mmHg, DBP 80–89 mmHg), stage 1 (140–159/90–99 mmHg), and stage 2 (≥160/≥100 mmHg) indicate progressive risk.[1] Essential hypertension arises from both genetic and environmental influences, with incidence increasing with age. Based on plasma renin activity (PRA), patients may show vasoconstrictive hypertension (high renin) or volume-dependent hypertension (low renin).[2]

Hypertension predisposes to cardiovascular disease, stroke, renal failure, peripheral arterial disease, and hypertensive retinopathy.[3] cardiovascular disease is the leading cause of death in hypertensives.[4] Structural changes such as left ventricular hypertrophy (LVH), atrial enlargement, arrhythmias, and heart failure are frequent findings in patients with long standing essential hypertension.[5] The risk of cardiovascular events doubles with every 20 mmHg rise in SBP and 10 mmHg in DBP.[6] LVH is an increase in left ventricular mass that may be physiological in athletes or pathological due to pressure/volume overload. It is initially compensatory but later maladaptive, with myocardial fibrosis, coronary microvascular dysfunction (CMD), and heart failure progression.[7] LV remodelling involves cardiomyocytes, fibroblasts, endothelial and immune cells.[8]

Non-invasive imaging (ECG, 2D/3D echocardiography, speckle-tracking, cardiac MRI) allows LVH detection. The 2018 ESC/ESH guidelines recognise LVH as a high-risk factor and marker of diastolic dysfunction.[8] Prevalence of LVH is 15–20% in the general population, 23–48% in hypertensives, and 58–77% in high-risk groups[9]. Obesity doubles the risk, with eccentric 6hypertrophy more common than concentric.[10] Echocardiographic LV mass is derived from LVEDD, IVSd, and PWd, indexed to body surface area.[11,12] Regression of LVH with antihypertensive therapy reduces cardiovascular morbidity.[11]

Micronutrient deficiencies also influence cardiac function. Deficits of L-carnitine, thiamine, and taurine are linked to cardiomyopathies, suggesting supplementation may benefit heart failure patients.[13]

Vitamin D deficiency (25(OH)D <20 ng/mL) is widespread, affecting nearly 490 million in India.[14] It reduces calcium and phosphorus absorption, elevates PTH, and disrupts mineral balance, contributing to cardiovascular dysfunction. Vitamin D receptors are abundant in heart, kidney, and vascular tissue, supporting its role in cardiovascular metabolism.[15] Low vitamin D is associated with hypertension, insulin resistance, metabolic syndrome, and dyslipidemia.[15]

The renin–angiotensin–aldosterone system (RAAS) plays a central role in hypertension and heart failure. While ACE inhibitors, ARBs, and aldosterone antagonists reduce morbidity, elevated PRA remains an adverse prognostic factor.[16] Vitamin D suppresses RAAS, regulates remodelling, improves endothelial function,[17,18] reduces oxidative stress,[19] and enhances prostacyclin-mediated vasodilation.[20] It prevents vascular calcification, maintains calcium homeostasis, and supports normal vascular tone.[21]

Animal studies show calcitriol improves contractility and lowers natriuretic peptides in deficiency states.[22] Vitamin D also inhibits renin synthesis, lowers inflammatory cytokines, and reduces atherosclerosis.[23] Clinical studies confirm deficiency and hyperparathyroidism are frequent in heart failure, often exacerbated by renal dysfunction and diuretic use.[24,25]

Thus, hypertension-induced LVH is a major predictor of adverse cardiovascular outcomes. Given the high prevalence of vitamin D deficiency in India, its role in modulating RAAS, calcium balance, and endothelial function highlights it as a potentially modifiable factor to reduce LVH and cardiovascular risk.

MATERIALS AND METHODS

Study Setting and Design

This observational study was conducted in the Department of Medicine, MMIMSR Medical College and Hospital, Mullana, Ambala. Data was collected from patients attending the outpatient department (OPD), admitted to medical wards, including intensive care unit (ICU) between January 2023 to March 2025.

Sample Size

The sample size was calculated using the formula $n = Z^2 \times P \times (1 - P) / d^2$, where Z = 1.96 (95% CI), P = 24.4% (Lokhandwala & Damle, 2004)[26], and d = allowable error. The final sample size was 100 patients with essential hypertension.

Inclusion and Exclusion Criteria

Patients >18 years of age with a diagnosis of essential hypertension were included. Exclusion criteria were: pregnancy, secondary causes of hypertension and patients on regular ACEIs/ARBs for ≥6 months.

Ethics and Consent

Ethical clearance was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants or their attendants.

Data Collection and Clinical Evaluation

Demographic details (age, sex, BMI), comorbidities, smoking/alcohol status, medications, duration of hypertension, and treatment history were recorded. Clinical assessment included general and systemic examination including fundus examination.

Investigations

- Routine baseline blood investigations including Complete Blood Count (CBC), Renal Function Tests (RFT), and Liver Function Tests (LFT) were performed for all patients.
- Serum 25(OH)D: Venous blood (2 ml) was collected, and vitamin D levels were classified as deficient (<20 ng/mL), insufficient (20–30 ng/mL), sufficient (30–40 ng/mL), and toxic (>100 ng/mL).
- Thyroid Profile: TSH, free T3, free T4 were estimated using electrochemiluminescence immunoassay.
- Blood sugar levels were assessed using Random Blood Sugar (RBS) estimation by Trinder's method, and glycated hemoglobin (HbA1c) was also measured.
- Other specialized investigations such as urinary VMA was estimated using spectrophotometry, renal artery Doppler, serum aldosterone, catecholamines, and CT angiography were performed when clinically indicated.

Echocardiography

All echocardiograms were performed on the same machine by a single cardiac sonographer to reduce inter-observer variability. Measurements included interventricular septal thickness (IVSd), posterior wall thickness (PWd), and LV end-diastolic diameter (LVEDD). LV mass was calculated using:

$$LV\ mass = 0.8 \times \{1.04 \times [(LVEDD + PWd + IVSd)^3 - (LVEDD)^3]\} + 0.6\ g$$

LV mass index (LVMI) was derived as LV mass divided by body surface area. Normal values were ≤95 g/m² for females and ≤115 g/m² for males; LVH was diagnosed when values exceeded these cut-offs.

Statistical Analysis

Data were analysed using SPSS version 26. Continuous variables were expressed as mean ± SD and categorical variables as frequencies or percentages. Group comparisons were made using Student's *t*-test or ANOVA, while categorical variables were analysed

using Chi-square test. Correlation between vitamin D levels and LVMI was assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

RESULTS AND OBSERVATIONS:

Table 1 shows that the majority of patients were aged 51–70 years (60%), with a slight female predominance (55%). Overweight and obesity were highly prevalent (66%). Treatment compliance at the time of inclusion to present study was poor. Among the patient prescribed treatment for hypertension, only 3.2% were compliant with the therapy. Vitamin D deficiency was widespread (62%). Echocardiography revealed high rates of structural heart changes: PWD was abnormal in 92%, IVST in 90%, LVMI in 66%, and EDD in 53%. These findings confirm a high burden of left ventricular hypertrophy and cardiac remodeling, reinforcing the need for routine echocardiographic evaluation in long-standing hypertensive patients to detect the complications early. (figure 1)

Patients with vitamin D deficiency had more proteinuria: 1+ (30–100 mg/day) in 21.0%, 2+ (100–300 mg/day) in 4.8%, and 3+ (>300 mg/day) in 11.3%. In contrast, insufficiency showed only 1+ in 5.0% and 2+ in 5.0%, while sufficiency showed 2+ in 11.1% with no 1+ or 3+ cases. Negative proteinuria was most common in insufficiency (90.0%) and sufficiency (88.9%) compared to deficiency (62.9%). The association was statistically significant ($\chi^2 = 13.354$, df = 6, **p = 0.038**).(table 2)

Abnormal LVMI was common in all groups, seen in 71.0% of vitamin D–deficient, 60.0% of insufficient, and 55.6% of sufficient patients, though the difference was not significant. Abnormal IVST was also frequent, occurring in 93.5% of deficient, 85.0% of insufficient, and 83.3% of sufficient cases, with no significant difference(Figure 2)

For PWD, abnormalities were most frequent in deficient cases (96.8%) and least in sufficient children (77.8%), showing a significant association with vitamin D status (p = 0.031).

Abnormal EDD was found in 59.7% of deficient, 35.0% of insufficient, and 50.0% of sufficient cases, but this was not statistically significant.

Table 1. Baseline Characteristics of Patients with Essential Hypertension (n = 100)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	≤30	3	3.0
	31–60	47	47.0
	>60	50	50.0
Gender	Male:Female	45:55	45%(males,55%female
BMI	Normal	34	34
	Overweight	50	50
	Obese	16	16
Treatment Status(n=62)	Non-compliant	**60**	**96.8**
	Compliant	**2**	**3.2**
Personal Habits	Alcohol intake	17	17
	Smoking	18	18
Comorbidities	**Diabetes**	19	19%
	Hypothyroidism	3	3%
	Hyperthyroidism	1	1%

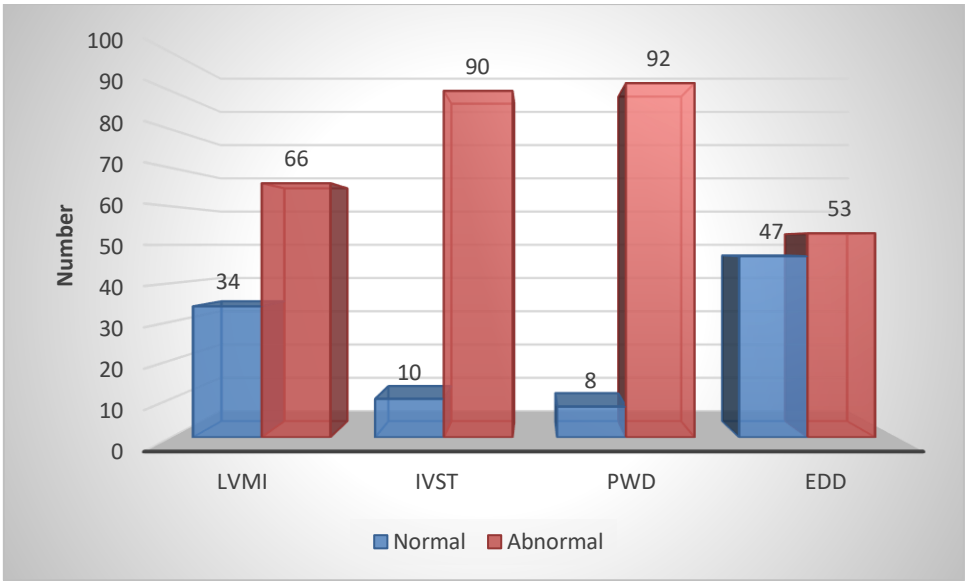


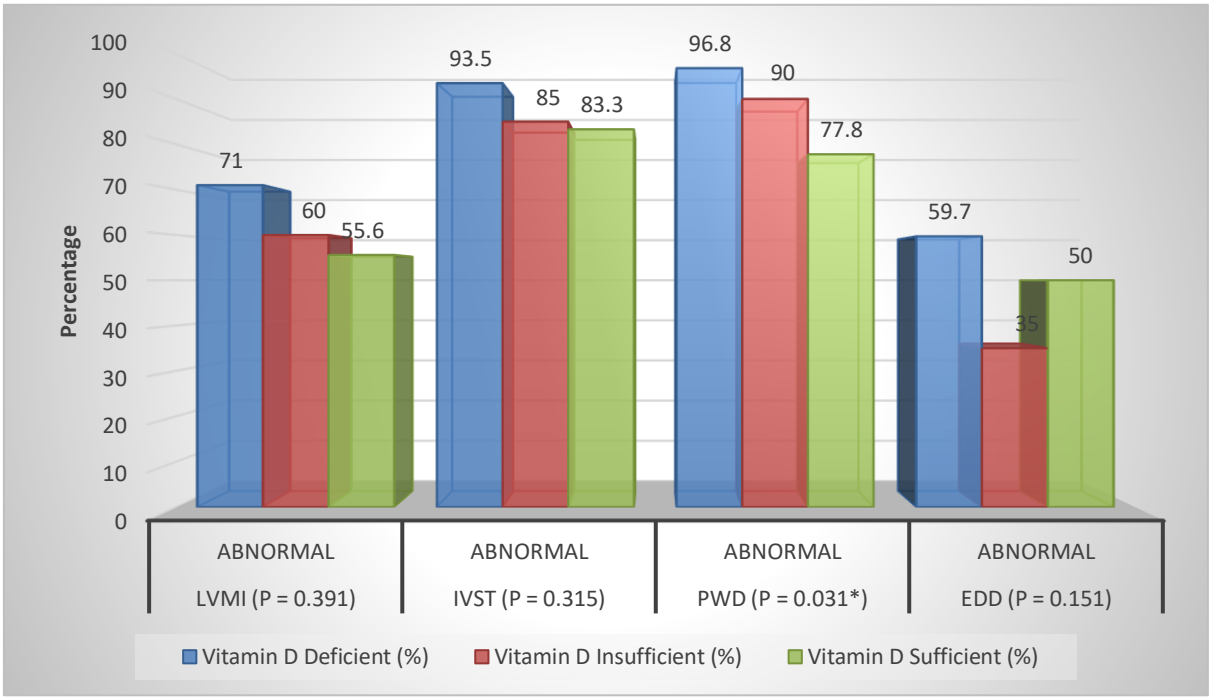
Figure 1: Frequency of LVMI and its parameters in Patients with Essential Hypertension (n = 100)

Table 2: Association between Proteinuria and Vitamin D Categories

Proteinuria	Deficiency (n=62)	Insufficiency (n=20)	Sufficiency (n=18)	Total (n=100)
Negative (–)	39 (62.9%)	18 (90.0%)	16 (88.9%)	73 (73.0%)
1+	13 (21.0%)	1 (5.0%)	0 (0.0%)	14 (14.0%)
2+	3 (4.8%)	1 (5.0%)	2 (11.1%)	6 (6.0%)
3+	7 (11.3%)	0 (0.0%)	0 (0.0%)	7 (7.0%)
Total	**62 (100.0%)**	**20 (100.0%)**	**18 (100.0%)**	**100 (100.0%)**

Chi-square test: $\chi^2 = 13.354$, df = 6, **p = 0.038** (significant

Figure 2: Association of Vitamin D Levels with Echocardiographic Parameters in Patients with Essential Hypertension (n = 100)



*Significant at $p < 0.05$

Table 3 : Mean Comparison of LVMI and its parameters with Vitamin D Levels in Patients with Essential Hypertension (n = 100)

Parameter	Vitamin D Deficient (n=62) Mean ± SD	Vitamin D Insufficient (n=20) Mean ± SD	Vitamin D Sufficient (n=18) Mean ± SD	p-value
End-diastolic diameter index (cm)	4.38 ± 0.63	4.37 ± 0.50	4.43 ± 0.61	0.945
Interventricular septal thickness at end diastole (cm)	1.37 ± 0.27	1.30 ± 0.30	1.32 ± 0.30	0.542
Posterior wall thickness at end diastole (cm)	1.38 ± 0.27	1.32 ± 0.29	1.31 ± 0.28	0.476
LV Mass Index (g/m²)	110.37 ± 26.36	103.29 ± 22.45	102.76 ± 41.70	0.473

DISCUSSION

In the present study, the mean age of hypertensive patients was 59.77 ± 12.71 years. Most patients were older, with 50% above 60 years, 47% between 31–60 years, and only 3% aged ≤30 years, showing predominance in the elderly. A slight female predominance (55%) was noted. In the present study, a statistically significant association was found between LVMI and sex (p = 0.004), with males showing a higher prevalence of increased LVMI compared to females.

Overweight/obesity was common (66%), emphasizing the role of excess body weight in cardiac remodeling and increased LVMI. Lifestyle factors included alcohol use in 17% and smoking in 18%, both contributing to disease progression but the no. of patients were too small to calculate its significant correlation with all the parameters. In the present study, the majority of patients had low Vitamin D levels, with 82% {showing either deficiency (62%) or insufficiency (20%) }, while only 18% had sufficient levels.

Proteinuria was detected in 27% of patients, reflecting renal involvement, a target organ damage. A significant inverse association was noted between Vitamin D status and proteinuria in vitamin D-deficient patients . Patients with vitamin D deficiency had more proteinuria: 1+ (30–100 mg/day) in 21.0%, 2+ (100–300 mg/day) in 4.8%, and 3+ (>300 mg/day) in 11.3%. In contrast, insufficiency showed only 1+ in 5.0% and 2+ in 5.0%, while sufficiency showed 2+ in 11.1% with no 1+ or 3+ cases. Negative proteinuria was most common in insufficiency (90.0%) and sufficiency (88.9%) compared to deficiency (62.9%). The association was statistically significant ($\chi^2 = 13.354$, df = 6, p = 0.038).Proteinuria is a key indicator of renal target organ damage in essential hypertension, reflecting the effects of disease duration, severity, and blood pressure control. Sustained hypertension raises intraglomerular pressure, leading to endothelial dysfunction, basement membrane sclerosis, podocyte injury, and abnormal protein leakage[27]. Mild proteinuria denotes early reversible changes, moderate indicates ongoing structural damage, and severe reflects advanced nephrosclerosis. Its presence marks progression toward hypertensive nephropathy and predicts higher cardiovascular risk.[27]

Yang Liu et al.[28] found a significant inverse correlation between serum 25(OH)D and albuminuria (r = -0.120, p = 0.026) in hypertensive children. Microalbuminuria prevalence varies widely depending on patient profile—from as low as 6.7% in untreated mild hypertensives[29] to as high as 40% in long-standing disease[30]. An Indian study found microalbuminuria in ~43% of male and 33% of female hypertensives, with prevalence increasing to 75% in those with >8 years of hypertension[31]. Our findings are therefore consistent with existing literature, highlighting proteinuria and the potential influence of vitamin D deficiency in hypertension.

A striking finding in this study was the high prevalence of low vitamin D levels (62% deficient, 20% insufficient). This mirrors data from South Asian populations. Singla et al.[32] reported 70.7% deficiency in hypertensives, while Siddiqi et al.[33] found significantly lower vitamin D3 levels in patients with essential hypertension (22.84 ± 5.10 ng/mL) compared to healthy controls (without essential hypertension) (47.09 ± 7.65 ng/mL). Komal Bharti et al.[34] also observed lower levels of vitamin D in hypertensives (20.25 ± 3.28 ng/mL) as compared with normotensives, with strong inverse correlations with blood pressure and ventricular mass. Similar results have been shown by Krishnasamy Prasad et al.[35] and Fallo et al.[36]

In this study, raised LVMI(>95 g/m² for females ; >115 g/m² for males) was seen in 71.0% of vitamin D–deficient, 60.0% of insufficient, and 55.6% of sufficient group patients, though the difference was not significant ($\chi^2 = 1.87$, p = 0.391). But Fallo et al.[36] demonstrated an inverse correlation between 25(OH)D and LVMI (r = -0.366, p < 0.003), with LVH prevalence of 57.1% in deficient versus 17.6% in sufficient groups. Similar results were observed by Komal Bharti et al.[34] and Magurno et al.[37]

For interventricular septal thickness (IVST), 93.5% of deficient patients had abnormal values, compared to 83.3% of sufficient patients with no significant difference.($\chi^2 = 2.31$, p = 0.315). Though IVST was higher in the vitamin D deficient group (1.37 cm) compared to the insufficient (1.30 cm) and sufficient groups (1.32 cm), the difference was not statistically significant (p = 0.542). Posterior wall thickness (PWD) showed an inverse association with vitamin D levels in significant percentage of hypertension patients. A significant percentage of hypertension patients with vitamin D deficiency had abnormal PWD (96.8%) as compared to patients with normal vitamin D levels

(77.8%) (χ^2 = 6.97, p = 0.031). However there was no significant difference in terms of absolute values of PWD (1.38 cm vs. 1.31 cm, p = 0.476). In contrast Siddiqui et al.[33], reported LVH(mean septal thickness and PWD) of 1.23 ± .18 cm and a strong inverse correlation with vitamin D (r = -0.774, p = 0.0001).

End-diastolic diameter (EDD) did not show significant associationswith vitamin D levels. Abnormal EDD was more frequent in the vitamin D deficient group (59.7%) compared to the insufficient (35.0%) and sufficient groups (50.0%), but the difference was not statistically significant (p = 0.151). Mean EDD values were nearly identical across groups (4.38 vs. 4.37 vs. 4.43 cm, p = 0.945). Vitamin D levels in patients with abnormal and normal EDD were also virtually identical. This suggests that EDD, a volume-related parameter, is less sensitive to vitamin D status. Liu et al.[28] reported a weak correlation between vitamin D and LVMI in children (r = -0.110, p = 0.041), but adult studies do not consistently support EDD as vitamin D–sensitive.

Overall, this study found that low vitamin D levels was common in patients of essential hypertension (82%), which was more than in the general population. In the present study, vitamin D deficiency was significantly associated with echocardiographic changes, with a higher percentage of patients showing abnormal posterior wall thickness (PWD) compared to those with sufficient levels (p = 0.031). This indicates a potential protective role of adequate vitamin D in maintaining posterior wall integrity. Although the percentages of abnormal LVMI and IVST were also higher among vitamin D–deficient patients, these differences did not reach statistical significance, though the trends pointed in the same direction.

CONCLUSION

While not all associations reached statistical significance, this study demonstrates a clear trend linking vitamin D deficiency with adverse cardiac remodeling in essential hypertension, especially posterior wall thickness. Together with existing literature, these findings suggest that vitamin D deficiency may accelerate hypertensive target organ damage. Monitoring and correction of vitamin D status could therefore represent a simple, low-cost adjunctive measure in the comprehensive management of hypertensive patients.

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