



Association Between Vitamin D3 Status, Blood Pressure, and Lipid Profiles in Hypertensive Patients

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Abstract: Hypertension is a major global health burden that is associated with millions of people across the globe. Epidemiological research indicates that vitamin D3 deficiency appears to be associated with hypertension, although extensive quantitative analyses have not been conducted. This research analyzed the correlation among serum Cholecalciferol levels, blood pressure parameters, and lipid profiles among individuals with hypertension. The study was an observational study that included 138 participants (80 hypertensive and 58 controls). A chemiluminescence assay was employed to evaluate participants' blood pressure status and categorize vitamin D3 levels as deficient (less than 20 ng/mL), insufficient (20-30 ng/mL), or sufficient (30 or higher). Blood pressure indices, lipid profiles, and biochemical parameters were measured. Pearson's correlation was utilized to appraise the relationships among variables. Compared to controls, hypertensive patients showed a highly reduced level of vitamin D3 (18.32 ± 1.03 vs. 30.33 ± 1.06 ng/mL, $p < 0.001$), a high systolic blood pressure (146.9 ± 2.31 vs. 119.0 ± 1.34 mmHg, $p < 0.001$), and a higher diastolic blood pressure (99.13 ± 2.31 vs. 77.93 ± 1.03 mmHg, $p < 0.001$). Vitamin D3 showed significant inverse relationships regarding the systolic blood pressure ($r = -0.5981$, $p = 0.001$), the diastolic blood pressure ($r = -0.4077$, $p = 0.001$), total cholesterol ($r = -0.5938$, $p = 0.001$), triglycerides ($r = -0.3161$, $p = 0.05$), and with atherogenic index ($r = -0.3630$, $p < 0.01$). The results reflect deficiency of vitamin D3 is significantly linked to high blood pressure and dyslipidemia in hypertensive patients and thus it could be considered to be used as a cardiovascular risk biomarker.

Keywords: Vitamin D3; hypertension; lipid profile; atherogenic index; cardiovascular risk

1. Introduction

Hypertension is regarded as one of the significant healthcare issues affecting the global population, and it causes a significant cardiovascular morbidity and mortality [1, 2]. As a known risk factor in peripheral vascular disorder, heart failure, cerebral stroke, myocardial infarction, and coronary artery disorder [3, 4], hypertension has a pathophysiology of factors that interact to cause it, like gender, age, familial background, obesity, smoking, behavioral pattern, intake of sodium in the diet, and stress [5, 6]. Recent findings indicate that vitamin D3 insufficiency is another person-alterable risk factor that is negatively linked to the emergence of hypertension [6]. Vitamin D3 is an essential lipid-soluble vitamin involved in many bodily processes, and reduced Cholecalciferol levels are attributed to higher death rate, as they are tied to cardiovascular disease, metabolic syndrome, type-2 diabetes, and several types

of malignancy [7,8]. Several investigations have demonstrated the link between Cholecalciferol status and cardiovascular disorder risk factors, including coronary artery disorder, stroke, heart failure, elevated vascular resistance, atherosclerosis, and hypertension [8, 9].

Mechanistically, vitamin D3 can cause a direct vascular effect on blood pressure via vitamin D receptors whose expression can be located in vascular endothelial cells [10, 11], in the renin-angiotensin-aldosterone system (RAAS) [12, 13], and in regulating intracellular calcium homeostasis to regulate renin secretion [11]. Deficiency of vitamin D3 stimulates RAAS and increases plasma renin and angiotensin II levels, which, in turn, worsens hypertension [14, 15]. Also, a vitamin D3 deficiency may be linked to insulin resistance, which further increases the risk of hypertension [16]. In addition to cardiovascular disease, vitamin D3 deficiency also affects cardiovascular risk factors, such as dysregulation of lipid metabolism, atherosclerosis, and other cardiovascular complications [17, 18]. Several studies have found correlations between vitamin D3 levels and lipid levels [19,20], where deficiency results in metabolic disorders of cholesterol and lipoproteins [16,21]. Such lipid changes are significant contributors to the accelerated development of cardiovascular disease and atherosclerosis [16, 22, 21]. Changes that are associated with lipid metabolism often accompany hypertension, and increased serum lipids are often observed among hypertensive subjects [23, 24]. Nevertheless, there is limited evidence on the subject, notwithstanding the growing existing literature, and the analysis of the simultaneous links between vitamin D3 status and the blood pressure parameters and lipid profiles of hypertensive patients has not been conducted comprehensively. This research project was conducted to measure the relationships among vitamin D3 deficiency, high blood pressure, and lipid profile changes in hypertensive individuals.

2. Materials and Methods

2.1. Research Framework and Study Subjects

The individuals involved in this descriptive observational research were enrolled at the Specialized Cardiovascular Health Center, Al-Diwaniyah Teaching Hospital, Iraq, and numbered 138. A total of 80 hypertensive patients (42 men and 38 women) and 58 healthy controls (36 men and 22 women) were enrolled in this research population. The subjects were between 29 and 76 years old. Exclusion criteria were arthritis, non-hypertensive cardiovascular disease, neoplastic diseases, and renal disease. Each participant completed a full questionnaire that included population characteristics (sex, age, marital status, education, employment), lifestyle characteristics (smoking, nutrition), and medical variables (vitamin D3 supplementation, disease status). The institution's ethics committee approved the research protocol, and all respondents provided their authorization.

2.2. Blood Pressure Measurements

The audit measure was performed after a 10-minute rest interval, using the standard protocol for quantifying blood pressure. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were assessed, and the mean was calculated for analysis. Pulse pressure (PP) was determined as $PP = SBP - DBP$. The equation used to determine mean arterial pressure (MAP) was $MAP = DBP + (PP/3)$ [25]. Hypertension was described as $SBP > 140$ mmHg and $DBP > 90$ mmHg.

2.3. Sample Handling and Biochemical Testing

Each of the participants had about 3 mL of venous blood taken away after staying overnight without consuming food. Centrifugation of blood samples was performed at 3000 rpm for 10 minutes, separating the serum, which was then preserved at -80°C before biochemical profiling. The concentration of serum 25-hydroxyvitamin D3 [25(OH)D3] was assayed using a chemiluminescent immunoassay (Monobind Kit, USA) based on the manufacturer's guidelines. Vitamin D3 levels were categorized as deficient (<20 ng/mL), insufficient (20-30 ng/mL), or sufficient (> 30 ng/mL). Photometric analysis was performed on the lipid profile, which consisted of total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C) with a photometric system of an automated analyzer (Hitachi, Germany) and suitable reagent kits. The atherogenic index of plasma (AIP) was computed as $AIP = \log_{10}(TG/HDL-C)$ [27]. Ratios of TC/HDL-C and LDL-C/HDL-

C were derived as additional atherogenic markers. Serum calcium, phosphorus, urea, and creatinine were computed employing an automated analyzer (Reflotron, Germany) with specific reagent kits.

2.4. Statistical Analysis

The analysis of the findings was done with GraphPad Prism (USA), and the results were illustrated as Mean $\pm$ SE. Between-group contrasts were performed using a Student t-test for two groups and a one-way analysis of variance (ANOVA), and, finally, the post hoc Tukey test for multiple groups. Pearson correlation coefficient was employed to test relationships among variables. Findings were deemed meaningful at $p \leq 0.05$. The post-hoc power assessment validated that the sample sizes of $n=80$ (hypertensive) and $n=58$ (Controls) provided power greater than 99 to detect a between-group difference in vitamin D3 levels at the 0.05 level. Nevertheless, the unequal design and the lack of a priori sample-size calculation are recognized as disadvantages, and the interpretations of subgroup analyses are to be taken with an appropriate degree of caution.

3. Results

3.1. Sociodemographic and Clinical Features

The research group included 138 individuals aged 29-76 years. The age mean of hypertensive patients was 49.88 ± 2.90 years, and that of the controls was 42.62 ± 3.43 years ($p > 0.05$). Hypertensive patients had extremely high SBP (146.9 ± 2.31 vs. 119.0 ± 1.34 mmHg, $p < 0.001$), DBP (99.13 ± 2.31 vs. 77.93 ± 1.03 mmHg, $p < 0.01$), PP (48.54 ± 2.19 vs. 41.11 ± 1.62 mmHg, $p < 0.01$) and MAP (115.6 ± 2.67). The mean of serum vitamin D3 was 18.32 ± 1.03 ng/mL lower than hypertensive patients (30.33106 ng/mL, $p < 0.01$), which was a 40% reduction relative to controls. Serum calcium was significantly lower in hypertensive patients (4.86 ± 0.67 vs. 6.86 ± 0.70 mg/dL, $p < 0.05$), while phosphorus (3.30 ± 0.08 vs. 1.16 ± 0.07 mg/dL, $p < 0.05$), creatinine (1.15 ± 0.06 vs. 0.87 ± 0.05 mg/dL, $p < 0.01$), and urea (6.77 ± 0.37 vs. 2.02 ± 0.20 mg/dL, $p < 0.01$) were significantly elevated (Table 1). A number of biochemical parameters require further interpretation. The level of serum calcium was lower than the traditional reference range in both groups, which was likely due to the analyzer's calibration; this finding should be approached with caution and justified by a standard laboratory procedure. Serum urea values stated in mmol/L correspond to 40.6 ± 2.2 and 12.1 ± 1.2 mg/dL in the hypertensive and control groups, respectively, which are within the standard reference range. Low serum phosphorus in the reference group may be due to dietary factors in this population, and this should be investigated.

Table 1. Comparison of blood pressure, vitamin D3, demographic, and biochemical indicators of hypertensive patients and healthy controls.

Parameter	Controls (n=58)	Hypertensive (n=80)
Age (years)	42.62 ± 3.43	49.88 ± 2.90
Systolic BP (mmHg)	119.0 ± 1.34	$146.9 \pm 2.31^{***}$
Diastolic BP (mmHg)	77.93 ± 1.03	$99.13 \pm 2.31^{**}$
Pulse Pressure (mmHg)	41.11 ± 1.62	$48.54 \pm 2.19^{**}$
Mean Arterial Pressure (mmHg)	92.31 ± 1.034	$115.6 \pm 2.67^{***}$
Vitamin D3 (ng/mL)	30.33 ± 1.06	$18.32 \pm 1.03^{**}$
Calcium (mg/dL)	6.86 ± 0.70	$4.86 \pm 0.67^*$
Phosphorus (mg/dL)	1.16 ± 0.07	$3.30 \pm 0.08^*$
Creatinine (mg/dL)	0.87 ± 0.05	$1.15 \pm 0.06^{**}$
Urea (mg/dL)	2.02 ± 0.20	$6.77 \pm 0.37^{**}$

Data presented as Mean $\pm$ SEM. BP: blood pressure. Significance vs. Controls: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

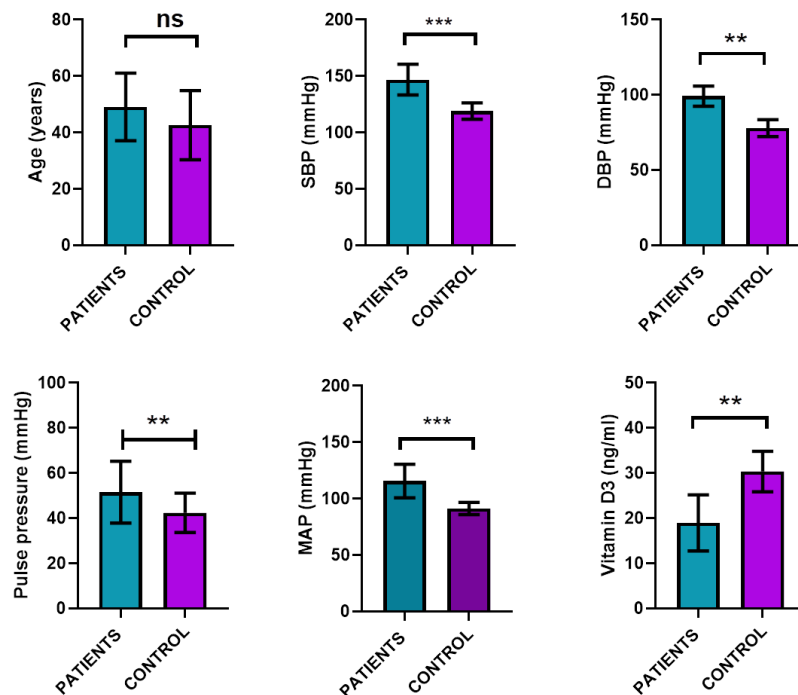


Figure 1. Comparison of age, blood pressure indexes, and vitamin D3 levels between high blood pressure patients and control groups.

3.2 Lipid Profile Comparison Between Groups

Hypertensive patients showed notable changes in lipid profile compared with controls (Table 2). Total cholesterol was elevated by 17% (175.3 ± 7.06 vs. 150.2 ± 10.28 mg/dL, $p < 0.05$), triglycerides by 22% (169.4 ± 9.97 vs. 139.1 ± 4.76 mg/dL, $p < 0.05$), LDL-C by 66% (94.14 ± 9.68 vs. 56.57 ± 5.95 mg/dL, $p < 0.05$), and VLDL-C by 22% (33.89 ± 1.99 vs. 27.82 ± 0.95 mg/dL, $p < 0.05$). Conversely, HDL-C was reduced by 30% (20.25 ± 0.93 vs. 29.01 ± 1.45 mg/dL, $p < 0.01$). There were significant increases in hypertensive patients by atherogenic indices. 33% (0.92 ± 0.02 vs. 0.69 ± 0.05 , $p < 0.001$), 59% (8.87 ± 0.64 vs. 5.59 ± 1.03 , $p < 0.05$), 109% (4.90 ± 0.57 vs. 2.35 ± 0.29 , $p < 0.01$) increases in plasma atherogenic index, TC/HDL-C ratio, and LDL-C/HDL-C ratio.

Table 2. Comparison of atherogenic indices and lipid profile of hypertensive patients and healthy controls.

Parameter	Controls (n=58)	Hypertensive (n=80)
Total Cholesterol (mg/dL)	150.2 ± 10.28	$175.3 \pm 7.06^*$
Triglycerides (mg/dL)	139.1 ± 4.76	$169.4 \pm 9.97^*$
HDL-C (mg/dL)	29.01 ± 1.45	$20.25 \pm 0.93^{**}$
LDL-C (mg/dL)	56.57 ± 5.95	$94.14 \pm 9.68^*$
VLDL-C (mg/dL)	27.82 ± 0.95	$33.89 \pm 1.99^*$
Atherogenic Index of Plasma	0.69 ± 0.05	$0.92 \pm 0.02^{***}$
TC/HDL-C Ratio	5.59 ± 1.03	$8.87 \pm 0.64^*$
LDL-C/HDL-C Ratio	2.35 ± 0.29	$4.90 \pm 0.57^{**}$

Data presented as Mean ± SEM. Significance vs. Controls: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

3.3 Blood Pressure and Lipid Parameters Stratified by Vitamin D3 Status

In hypertensive patients, blood pressure and lipid parameters worsened, and vitamin D3 status decreased with vitamin D3 stratification (Table 3). The low group had the oldest mean age (45.86 ± 2.15 years), then came the insufficient (43.27 ± 4.36 years) and sufficient (40.00 ± 13.08 years) groups. Measurements of blood pressure showed strong status-dependence of vitamin D3. Deficient patients had the highest SBP (142.7 ± 2.47 mmHg) and DBP (98.95 ± 1.50 mmHg), an intermediate result in insufficient patients (SBP: 138.3 ± 2.07

mmHg; DBP: 84.17 ± 4.16 mmHg), and the lowest result in sufficient patients (SBP: 131.7 ± 5.42 mmHg; DBP: 81.25 ± 3.50 mmHg). Likewise, pulse pressure and MAP rose gradually with the deficiency of vitamin D3. The vitamin D3 status declined, and the lipid profile worsened. Deficient and insufficient groups demonstrated elevated TC (161.6 ± 5.21 and 171.7 ± 9.54 mg/dL) and TG (171.8 ± 3.78 and 175.3 ± 6.68 mg/dL) compared to sufficient patients (TC: 131.0 ± 2.64 mg/dL; TG: 141.0 ± 6.00 mg/dL). HDL-C was progressively reduced across deficient (18.21 ± 0.28 mg/dL), insufficient (21.75 ± 0.73 mg/dL), and sufficient (29.25 ± 0.75 mg/dL) groups. Atherogenic indices (AIP, TC/HDL-C, LDL-C/HDL-C) showed a consistent pattern, with the highest values in vitamin D3-deficient patients. It should be noted that the subgroup of patients with sufficient vitamin D3 within the hypertensive cohort was small. Accordingly, comparisons involving this subgroup are exploratory and should be interpreted with caution, given the risk of inflated Type I error rates in Tukey's post hoc analyses.

Table 3. Blood pressure and lipid parameters in hypertensive patients stratified by vitamin D3 status

Parameter	Deficient (<20)	Insufficient (20-30)	Sufficient (≥ 30)
Vitamin D3 (ng/mL)	14.99 ± 0.88^a	24.92 ± 1.04^b	31.50 ± 1.92^c
Systolic BP (mmHg)	142.7 ± 2.47^a	138.3 ± 2.07^b	131.7 ± 5.42^c
Diastolic BP (mmHg)	98.95 ± 1.50^a	84.17 ± 4.16^b	81.25 ± 3.50^c
Total Cholesterol (mg/dL)	161.6 ± 5.21^a	171.7 ± 9.54^a	131.0 ± 2.64^b
HDL-C (mg/dL)	18.21 ± 0.28^a	21.75 ± 0.73^a	29.25 ± 0.75^b
AIP	0.94 ± 0.03^a	0.85 ± 0.01^b	0.82 ± 0.02^c

Data presented as Mean $\pm$ SEM. Different superscript letters (^a, ^b, ^c) highlight meaningful differences between groups ($p < 0.05$, Tukey's post-hoc test). AIP: atherogenic index of plasma.

3.4 Correlation Analysis of Vitamin D3 with Blood Pressure and Lipid Parameters

Pearson correlation analysis indicated significant correlations between vitamin D3 levels and multiple cardiovascular parameters in hypertensive patients (Table 4). Vitamin D3 demonstrated strong inverse correlations with age ($r = -0.4618$, $p < 0.001$), SBP ($r = -0.5981$, $p < 0.001$), and DBP ($r = -0.4077$, $p < 0.001$). MAP exhibited moderate inverse correlation ($r = -0.4251$, $p < 0.001$), while pulse pressure showed weaker inverse association ($r = -0.1788$, $p > 0.05$). Lipid parameters illustrated notable correlations with vitamin D3 status. Total cholesterol ($r = -0.5938$, $p < 0.001$), triglycerides ($r = -0.3161$, $p < 0.05$), LDL-C ($r = -0.3883$, $p < 0.01$), and VLDL-C ($r = -0.1161$, $p > 0.05$) exhibited inverse correlations, while HDL-C illustrated a positive linkage ($r = 0.1896$, $p > 0.05$). Atherogenic indices established meaningful associations: AIP ($r = -0.3630$, $p < 0.01$), TC/HDL-C ratio ($r = -0.3987$, $p < 0.01$), and LDL-C/HDL-C ratio ($r = -0.4472$, $p < 0.001$). Biochemical parameters showed variable correlations with vitamin D3. Positive associations were observed with calcium ($r = 0.1004$), phosphorus ($r = 0.1587$), and creatinine ($r = 0.3091$), while urea exhibited a negative correlation ($r = -0.1396$).

Table 4. Pearson correlation coefficients between vitamin D3 levels and cardiovascular parameters in hypertensive patients

Parameter	Correlation (r)	Significance
Age	-0.4618	$p \leq 0.001$
Systolic Blood Pressure	-0.5981	
Diastolic Blood Pressure	-0.4077	
Mean Arterial Pressure	-0.4251	
Total Cholesterol	-0.5938	
Triglycerides	-0.3161	$p \leq 0.05$
LDL-C	-0.3883	$p \leq 0.01$
Atherogenic Index of Plasma	-0.3630	
TC/HDL-C Ratio	-0.3987	
LDL-C/HDL-C Ratio	-0.4472	

Data show Pearson correlation coefficients (r) and statistical significance $n = 80$ hypertensive patients.

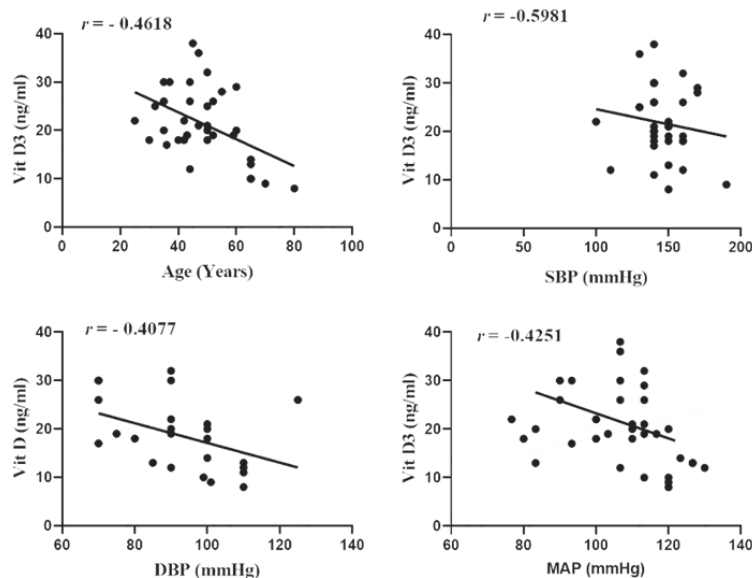


Figure 2. The scatter plot showed the correlation coefficient between the blood pressure index and vitamin D3 level in hypertensive patients.

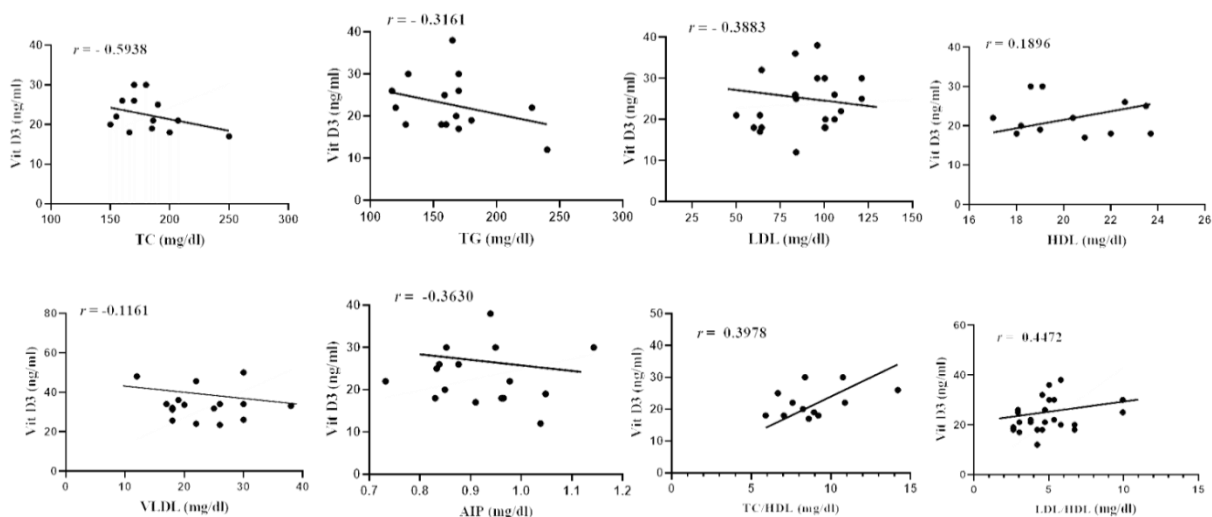


Figure 3. The scatter plot showed the correlation between lipid index and vitamin D3 in patients with hypertension.

4. Discussion

This paper reveals a great deal of correlation between vitamin D3 deficits, high blood pressure, and dyslipidemia among hypertensive individuals. The 40 percent decrease in vitamin D3 serum levels in hypertensive patients (18.32 ± 1.03 vs. 30.33 ± 1.06 ng/mL, $p < 0.01$) is a clinically significant hypovitaminosis D, in agreement with earlier studies [28, 29]. This hypothesis that vitamin D3 status plays a significant role in blood pressure regulation is supported by the strong negative correlation between vitamin D3 and systolic blood pressure ($r = -0.5981$, $p < 0.001$). The negative age-vitamin D3 correlation ($r = -0.4618$, $p = 0.001$) is also consistent with age-related decreases in cutaneous vitamin D3 synthesis [30, 31]. Age-related decreases in 7-dehydrocholesterol levels and the declining efficiency of the skin to produce vitamin D3 in the dermal layer are factors that lead to progressive vitamin D3 deficiency in the elderly [30]. Such an outcome presupposes a specific clinical interest given the increased prevalence of hypertension in the aging population [32, 33]. Vitamin D3 status stratification showed that as vitamin D3 levels decreased, blood pressure rose continuously, and deficient patients had systolic blood pressure 8% higher than that of adequate patients (142.7 ± 2.47 vs.

131.7 ± 5.42 mmHg). This dose-response correlation enhances the blood pressure-regulating effect of vitamin D3 [34, 35]. The results are similar to those of earlier investigations that reported negative relationships between vitamin D3 and blood pressure among hypertensive individuals [28, 29, 36]. These findings should be placed in the context of the broader literature. Randomized controlled trials and Mendelian randomization studies have produced conflicting results in large-scale studies. The VITAL trial, one of the largest RCTs to date, found that a vitamin D3 supplement (2000 IU/day) did not show meaningful differences in cardiovascular events or blood pressure in the general public. In the same manner, Mendelian randomization studies [36] have yielded weak indications of the impact of vitamin D status on blood pressure, adjusting for genetic confounding. Such discrepancies may indicate a divergence between observational relationships and true causal relationships, and highlight the complexity of the vitamin D-blood pressure relationship. We should then be interpreted as hypothesis-generating rather than confirmatory of a causal mechanism, with reference to our findings.

Several processes can be attributed to vitamin D3's impact on BP. Vitamin D3 regulates the angiotensin-aldosterone-renin system by directly transcriptionally regulating expression of renin genes [12, 37]. Deficiency of vitamin D3 increases RAAS activation, leading to higher plasma renin and angiotensin II levels and, in turn, elevating blood pressure [14, 15]. Also, vitamin D3 affects endothelial vascularity via vitamin D receptor-mediated mechanisms that stimulate nitric oxide synthesis and vascular relaxation [10,11]. Calcium homeostasis is also controlled by vitamin D3; hypocalcemia due to deficiency triggers parathyroid hormone release, which enhances calcium deposition in the intracellular smooth musculature of the vasculature and may raise vascular resistance [38, 39]. In patients with hypertension, dyslipidemia is characterized by increases in total cholesterol (17%), triglycerides (22%), LDL-C (66%), and HDL-C (30%), indicating severe dysregulation of lipid metabolism. Vitamin D3 had serious negative associations with atherogenic lipid fractions (TC: $r=-0.5938$; TG: $r=-0.3161$; LDL-C: $r=-0.3883$), which are consistent with the control of lipid metabolism in vitamin D3 [19,20,40]. Vitamin D3 can affect lipid metabolism in a variety of mechanistic ways. Vitamin D3 facilitates intestinal calcium absorption, thereby decreasing the release of hepatic triglycerides into the blood and the absorption of intestinal fatty acids [41, 42]. High serum calcium increases fecal lipid and bile acid excretion, which may reduce cholesterol levels [43, 44]. Also, in vitamin D3 deficiency, parathyroid hormone levels are increased, which activate lipogenesis, suppress lipolysis, and impair triglyceride clearance [45]. A clear link exists between vitamin D3 insufficiency and beta-cell dysfunction and insulin resistance, leading to low HDL-C levels and augmented triglycerides [46]. A known predictor of cardiovascular risk, the atherogenic index of plasma, was significantly elevated in hypertensive patients (33 percent) and strongly inversely linked to vitamin D3 ($r = -0.3630$, $p < 0.01$). This result indicates that vitamin D3 deficiency facilitates atherogenic lipoprotein phenotypes [27, 47]. Equally, elevated TC/HDL-C and LDL-C/HDL-C levels among hypertensive patients, which were negatively associated with vitamin D3 ($r = -0.3987$ and $r = -0.4472$, correspondingly), suggested that cardiovascular risk was considerably high [48]. The changes in the renal functions (increased urea and creatinine) noticed among hypertensive patients indicate that the kidney dysfunction is early, and probably due to hypertension-related nephropathy [6]. The reduced serum calcium in hypertensive patients is consistent with the effects of vitamin D3 deficiency on calcium homeostasis.

Study limitations are the cross-sectional model that does not permit drawing any causal conclusions, the lack of multivariate regression analysis to eliminate the possible confounding factors contributing to the results, such as renal functionality, body mass index, medications, smoking, and diet, along with the small and unbalanced sample size and the recruitment of volunteers in one place, which makes it challenging to apply the results broadly. The higher creatinine and urea levels in hypertensive patients are a sign of subclinical renal injury that can independently affect the processes of vitamin D3 hydroxylation and lipid metabolism, and the reported associations are thus to be regarded with due caution. Another serious limitation is that the covariates of primary importance, such as BMI, physical activity, sun exposure, vitamin D intake, antihypertensive medication use, statin therapy, and diabetes mellitus status, are not statistically controlled. These unmeasured confounders could have had an independent impact on both vitamin D3 levels and

lipid/blood pressure outcomes, and the fact that they were not included in the analytical model significantly undermines the possibility of concluding that these associations are independent.

5. Conclusions

This paper shows that vitamin D3 insufficiency is strongly associated with high blood pressure and atherogenic dyslipidemia in hypertensive patients. The vitamin D3 level was found to be 40% low in hypertensive individuals, and there was an inverse correlation between vitamin D3 and systolic blood pressure ($r = -0.5981$), diastolic blood pressure ($r = -0.4077$), total cholesterol ($r = -0.5938$), and the atherogenic indices (AIP; $r = -0.3630$). The stratification of vitamin D3 showed that the blood pressure and lipid profiles steadily worsened as the vitamin D3 status declined. The results provide evidence of the potential usefulness of vitamin D3 as a cardiovascular risk biomarker in hypertensive individuals, and suggest that the ideal vitamin D3 level may be a promising treatment intervention for the prevention of hypertension and cardiovascular disease. Prospective and randomized controlled trials should be conducted in the future to determine whether correction of vitamin D3 deficiency is attributed to enhancements in blood pressure and lipid profiles in hypertensive individuals.

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