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Nocturnal Hypertension and its Relationship with Vitamin D in Older Hypertensive Adults

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Abstract

Objective: Nocturnal hypertension (HT) predicts HT-related end-organ damage and cardiovascular mortality, with a complex pathophysiology involving multiple factors. Vitamin D is considered an emerging contributor. This study examined the relationship between vitamin D level and nocturnal HT in older adults with HT.

Materials and Methods: This cross-sectional study examined 219 patients aged ≥60 years, who underwent ambulatory blood pressure (BP) monitoring. An average nighttime systolic BP≥120 mm Hg and/or diastolic BP≥70 mm Hg was diagnosed as nocturnal HT. Vitamin D insufficiency was defined as serum 25 (OH) vitamin D levels 30 ng/mL.

Results: The prevalence of nocturnal HT was 69.9% among older hypertensive adults. In the group with nocturnal HT, there was a significantly higher percentage of patients with vitamin D insufficiency than those without (89.5% vs. 72.7%; p=0.002). A reverse linear relationship was noted between the quartiles of 25 (OH) vitamin D and the occurrence of nocturnal HT. The percentage of individuals with nocturnal HT declined as the quartiles of 25 (OH) vitamin D increased (p-value for trend = 0.015). In the multivariate logistic regression analysis, after accounting for age, Charlson's comorbidity index, and average daytime systolic BP values, vitamin D insufficiency was linked to a significantly higher likelihood of nocturnal HT (OR=4.92, 95% CI=1.66-14.61, p=0.004).

Conclusion: Vitamin D insufficiency may contribute to the development of nocturnal hypertension in older hypertensive adults.

Keywords: 25-Hydroxyvitamin D 2, aged, ambulatory blood pressure monitoring, hypertension, blood pressure

Introduction

Nocturnal hypertension (HT) can be diagnosed through ambulatory blood pressure monitoring (ABPM) and is defined as having a mean systolic blood pressure (SBP) ≥120 mm Hg and/or diastolic blood pressure (BP) of ≥70 mm Hg during the night (1). BP follows a circadian rhythm throughout the day, marked by the reduction of sympathetic activity and increase in parasympathetic activity during nighttime, leading to decreased BP during sleep. Consequently, daytime BP tends to be higher than nighttime BP. Normally, the decrease in nighttime BP should range between 10-20%, indicating a typical dipping BP pattern. The physiological dipping pattern of BP is lost or insufficient in nocturnal HT, leading to a nondieting or reverse dipping pattern, where night BP dipping is 10% or even increases instead of decreasing, respectively (2). Nocturnal HT emerges as a stronger predictor of future cardiovascular mortality than daytime HT and holds more significance than dipping status concerning HT-related end organ damage (proteinuria, left ventricular hypertrophy, arterial stiffness and retinopathy) and cardiovascular events (angina pectoris, myocardial infarction, heart failure, stroke) (3-6). Nocturnal HT is observed in approximately 40-60% of the general population

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(7). However, various factors, such as age, ethnicity, and the presence of comorbid conditions, particularly those causing autonomic dysfunction, water retention, or sleep disturbances, significantly influence its occurrence (8,9). Due to the high prevalence and serious health consequences of nocturnal HT, there is a growing interest in identifying new factors that contribute to its development and treatment. This effort involves exploring alternative therapeutic approaches beyond conventional lifestyle modifications, dietary interventions, and antihypertensive medications, which may contribute to improving BP management. Vitamin D is one of these emerging factors under investigation and has gained considerable attention. Serum vitamin D levels in older adults are influenced by their residential status, whether in a nursing home or within the community, along with factors such as specific comorbidities, medication usage, or even sex and body mass index. Vitamin D deficiency is defined as levels below 20 ng/mL, while insufficiency is defined as levels between 21 and 29 ng/mL (10). The prevalence of vitamin D insufficiency among community-living older adults above 60 years was reported as 31.7% (11). Although the optimal serum vitamin D level for extraskeletal function remains unknown, the American geriatrics society recommends maintaining vitamin D concentrations exceeding 30 ng/mL in older adults to support bone health (12). Several studies have investigated the impact of low vitamin D levels on BP, suggesting that they may contribute to the development of HT through various mechanisms and could be linked to a non-dipping BP pattern. However, the precise clinical implications of vitamin D levels on nocturnal HT, particularly among older adults, remain inadequately defined in clinical practice. In this study, our goal was to investigate the prevalence of nocturnal HT among hypertensive older adults and examine its correlation with vitamin D levels, along with other clinical factors.

Materials and Methods

Patient Selection

In this cross-sectional study, patients who were treated at the geriatrics outpatient clinic of Ankara University Faculty of Medicine between January 2016 and December 2018 and who underwent 24-hour ABPM were evaluated. Among these patients, those who i) were aged 60 and over, ii) were receiving antihypertensive treatment, or iii) were diagnosed with HT according to the ABPM results were included in the study. Patients meeting the following criteria were excluded from the study: i) those receiving Vitamin D treatment when serum levels were measured, ii) those without a diagnosis of HT, iii) those with 24 hours of ABPM recording or 70% successful systolic and diastolic BP recordings during a 24-hour period, and iv) those receiving antihypertensive drugs in the evening or before bedtime. This study adhered to the Declaration of Helsinki's principles and obtained ethical approval from Ankara University's ethical committee (approval number:17-1121-18, date: 22.10.2018). This study was conducted using patient files, and informed consent was waived due to the retrospective nature of the data analysis, in line with institutional and ethical guidelines.

Medical History and Laboratory Assessments

The ages, sexes, weights, heights, smoking and alcohol habits, comorbidities, and antihypertensive medications of the patients were recorded from their medical records. Additionally, the Charlson Comorbidity Index (CCI) of the patients was calculated to evaluate the burden of comorbidities (13). To assess patients' functionality, Katz activities of daily living scores (maximum score of 8 points indicating full attendance to activities), Lawton instrumental activities of daily living scores (maximum score of 17 points indicating full attendance to activities), handgrip strengths (kg), 4-meter walking speeds (m/sec), and fall histories in the last year were also recorded (14,15). Handgrip strength was measured using an automated hand dynamometer (Takei scientific instruments, Niigata, Japan), and walking speeds were determined by measuring the time taken to walk 4 meters, followed by calculating the corresponding speed in meters per second. In men, handgrip measurements 32 kg and in women, measurements 22 kg indicate low muscle strength (16). A gait speed of 0.8 m/s defines low physical performance (16). Laboratory parameters, including creatinine, lipid profile, inflammatory markers, hemoglobin, calcium, intact parathyroid hormone (PTH), and 25 (OH) vitamin D levels, were recorded from patients' electronic medical records, all of which were performed within 1 month of the ABPM. These parameters were chosen based on their known associations with HT and were recorded from the patients' electronic medical records. High-performance liquid chromatography (Immuchrom GmBH, Heppenheim, Germany) was used to measure serum 25 (OH) vitamin D levels. The intra-assay and inter-assay variability percentages were 3% and 5%, respectively. Serum levels below 30 ng/mL indicate vitamin D insufficiency.

Ambulatory Blood Pressure Monitoring

The ABPM results of the patients were obtained from the electronic medical records. Patients' 24-hour ambulatory BP was measured using a validated automated non-invasive oscillometric device (Mobil-O-Graph Monitor; IEM GmbH, Stolberg, Germany) (17). The device was programed to measure BP at 20-minute intervals during the day and at 30-minute intervals during night time over 24 hours. The patients' sleeping and waking hours were recorded either by the patients themselves or by their relatives and then entered the software before downloading the BP recordings. Consequently, the device can calculate the percentage decrease in nighttime systolic BP. Based on this percentage decrease, patients were classified into four categories: reverse dippers (with a BP drop of less than 0%),

non-dipper (with a BP drop of less than 10%), dippers (with a BP drop between 10% and 20%), and extreme dippers (with a BP drop of more than 20%). The diagnosis of HT is established based on the use of at least one antihypertensive medication or according to the results of ABPM. ABPM results were interpreted in line with the 2023 European society of hypertension guidelines, which define HT as having an average daytime SBP ≥135 and/or diastolic blood pressure (DBP)≥85 mmHg and/or 24hour SBP≥130 and/or DBP≥80 mmHg. Nocturnal HT was defined as an average nighttime SBP≥120 mm Hg and/or DBP≥70 mm Hg (1).

Statistical Analysis

Statistical analyses were conducted using SPSS Statistics (version 29.0.2.0, Armonk, NY: IBM Corp.). Categorical variables were analyzed using count and percentage distributions, whereas continuous variables were assessed using medians and interquartile ranges (IQR). The patients were categorized based on the presence (+) or absence (-) of nocturnal HT. Categorical variables were compared using either the chi-square test or Fisher's exact test, while continuous variables were assessed using the Wilcoxon rank-sum test. Multivariate logistic regression analysis was performed to adjust for potential confounding factors associated with the presence of nocturnal HT. Odds ratios were calculated. Statistical significance was set at $p<0.05$.

Results

The study included 219 patients. Table 1. Delineated patient features, organized according to the presence of nocturnal HT. Among the 219 patients, 153 (69.9%) had nocturnal HT, whereas 66 (30.1%) patients did not have nocturnal HT. The patients had a median age of 76.3 (70.3-82.8) years. There was no significant difference in the median age between patients with (76.3) and without (77.5) nocturnal HT (p=0.449), and age categories were similarly distributed between the groups (p=0.899). Of the patients, 83 (37.9%) were male and 136 (62.1%) were female, indicating comparable sex distributions in the groups with and without nocturnal HT (p=0.547). In Table 2, the ABPM results of the groups with and without nocturnal HT are presented. The average daytime SBP and DBP, average nighttime SBP and DBP, and 24-h SBP and DBP values were significantly higher in the group with nocturnal HT ($p<0.001$). Moreover, the percentage decrease in SBP during sleep was significantly lower in the group with nocturnal HT (0.7% vs. 5.8%; $p<0.001$). The prevalence of non-dipper was the highest, with 111 cases (51.4%). However, when comparing dipping patterns between patients with and without nocturnal HT, the prevalence of reverse dippers (65 cases, 42.5% vs. 12 cases, 19%) is higher in the group with nocturnal HT, and the prevalence of dippers is lowest (13 cases, 8.5% vs. 15 cases, 23.8%) ($p<0.001$).

In Table 3, laboratory results were presented according to the presence of nocturnal HT. Hemoglobin levels, lipid profiles, creatinine levels, calcium levels, and inflammation markers among the groups showed no significant differences. However, the median 25 (OH) vitamin D level was significantly lower in the group with nocturnal HT compared to those without (15.5 vs. 21.1; p=0.013). Furthermore, the percentage of patients with 25 (OH) vitamin D levels below 30 ng/mL was significantly higher in the group with nocturnal HT (137 cases, 89.5%) than in those without nocturnal HT (48 cases, 72.7%), with a p-value of 0.002. A negative linear correlation was found between the quartiles of 25 (OH) vitamin D and the occurrence of nocturnal HT. The percentage of patients with nocturnal HT declined as quartiles of 25 (OH) vitamin D increased (p for trend=0.015). Additionally, intact PTH levels were significantly higher in the nocturnal HT group (median 87.1 pg/mL vs. 62.5 pg/mL; p=0.048). Vitamin D insufficiency significantly increased the odds of nocturnal HT, even after accounting for age, CCI, and average daytime SBP values in the multivariate logistic regression. (OR=4.92, 95% CI 1.66-14.61, p=0.004) (Table 4).

Discussion

This study explored the prevalence of nocturnal HT and its relationship with vitamin D levels in older adults with HT. Our study revealed a prevalence of nocturnal HT of 69.9% among older hypertensive adults, in line with other studies reporting rates between 40% and 60%, and reaching 70% in poorly controlled hypertensive patients (7,18). Furthermore, we observed a negative association between vitamin D levels and nocturnal HT, with the percentage of patients experiencing nocturnal HT increasing as 25 (OH) vitamin D levels decreased. This correlation was independent of advanced age, CCI, and average daytime systolic BP values which are commonly associated with nocturnal HT. Although the underlying mechanism of nocturnal HT is not clear, stimulation of the renin-angiotensin-aldosterone system, renal dysfunction, and elevated sympathetic activation are thought to be the main mechanisms (19). Thus, conditions that induce these states, such as chronic kidney disease, diabetes mellitus, or other diseases causing autonomic dysfunction, as well as conditions like obstructive sleep apnea syndrome (OSAS), chronic pain, and restless leg syndrome that can disrupt sleep patterns and cause increased sympathetic activation at night, may contribute to nocturnal HT. Additionally, aging, anxiety disorders, and obesity are commonly cited as risk factors for nocturnal HT (19). In our study, aside from the significant correlation of vitamin D and PTH levels with nocturnal HT, we did not observe any other significant differences in the presence of nocturnal HT among individuals with age, diabetes mellitus, renal insufficiency, and adiposity. Vitamin D receptors (VDRs) are present in nearly all nucleated cells, and 3% of the

Table 1. Basic characteristics of the patients according to their nocturnal hypertension status

	All (n=219)		Nocturnal hypertension (+) (n=153, 69.9%)		Nocturnal hypertension (-) (n=66, 30.1%)		p
Median age, years (IQR)	76.7	(70.3-82.8)	76.3	(70.3-82.5)	77.5	(72-84.1)	0.449
Age group, n (%)							0.899
60-69 years	43	(19.6)	30	(19.6)	13	(19.7)	
70-79 years	91	(41.6)	65	(42.5)	26	(39.4)	
≥80 years	85	(38.8)	58	(37.9)	27	(40.9)	
Sex, n (%)							0.547
Male	83	(37.9)	56	(36.6)	27	(40.9)	
Female	136	(62.1)	97	(63.4)	39	(59.1)	
BMI, n (%)							0.417
<18.5	3	(1.4)	3	(2)	0	(0)	
18.5-24.9	57	(26)	39	(25.7)	18	(27.3)	
25-29.9	74	(33.8)	48	(31.6)	26	(39.4)	
≥30	84	(38.4)	62	(40.8)	22	(33.3)	
Active smokers	23	(10.5)	16	(10.5)	7	(10.6)	0.974
Functional status							
Katz ADL, median (IQR)	6	(5-6)	6	(5-6)	6	(5-6)	0.154
Lawton IADL, median (IQR)	14	(7.5-17)	14	(6.5-17)	14	(8-17)	0.384
Fall in the last year, n (%)	71	(32.4)	52	(34)	19	(28.8)	0.451
Muscle strength of female subjects (kg), median (IQR)	15.7	(10.8-20)	14.5	(9.5-19.4)	16.6	(13-20.5)	0.125
Muscle strength of males (kg), median (IQR)	24.9	(19.9-31.8)	25.2	(20-33.1)	23.7	(16.5-28.3)	0.185
Physical performance (m/sec), median (IQR)	0.57	(0.40-0.66)	0.50	(0.38-0.66)	0.57	(0.44-0.66)	0.282
Comorbidities, n (%)							
Diabetes mellitus	113	(51.6)	85	(55.6)	28	(42.4)	0.074
Chronic kidney disease*	88	(40.2)	62	(40.5)	26	(39.4)	0.876
Depression	64	(29.2)	47	(42.3)	17	(38.6)	0.673
Coronary artery disease	51	(23.3)	35	(22.9)	16	(24.2)	0.826
Dementia	26	(11.9)	20	(13.1)	6	(9.1)	0.403
Cerebrovascular disease	22	(10)	14	(9.2)	8	(12.1)	0.502
Heart failure	15	(6.8)	11	(7.2)	4	(6.1)	0.762
CCI, median (IQR)	5	(4-7)	5	(4-7)	5	(4-6)	0.420
Antihypertensive drugs, n (%)							
ARB	109	(48.2)	74	(48.4)	35	(53)	0.526
Diuretics	107	(47.3)	75	(49)	32	(48.5)	0.942
CCB	95	(42)	66	(43.1)	29	(43.9)	0.912
β-blockers	88	(38.9)	62	(40.5)	26	(39.4)	0.876
ACE inhibitors	46	(20.4)	29	(19)	17	(25.8)	0.257
α-agonists	19	(8.4)	14	(9.2)	5	(7.6)	0.704
MRA	7	(3.1)	5	(3.3)	2	(3)	0.927
Number of antihypertensive drugs, median (IQR)	2	(1-3)	2	(1-3)	2	(1.75-3)	0.407

*Patients with an estimated glomerular filtration rate of 60 mL/min,
ADL: Activities of daily living, ACE: Angiotensin-converting enzyme, ARB: Angiotensin receptor blockers, BMI: Body mass index, CCB: Calcium channel blockers, CCI: Charlson comorbidity index, IADL: Instrumental activities of daily living, IQR: Interquartile range, kg: Kilograms, m: Meter, MRA: Mineralocorticoid receptor antagonists, n: Number of cases, sec: Second

Table 2. Ambulatory blood pressure results according to nocturnal hypertension status

	All (n=219)		Nocturnal hypertension (+) (n=153, 69.9%)		Nocturnal hypertension (-) (n=66, 30.1%)		p
Average daytime SBP (mmHg)	128	(119-141)	136	(126-146)	116	(110-121.2)	<0.001
Average daytime DBP (mmHg)	75	(68-81)	78	(73-84)	67	(64-73)	<0.001
Average night SBP (mmHg)	125	(114-139)	134	(124-144)	111	(105-114.2)	<0.001
Average night DBP (mmHg)	70	(64-79)	75	(70-82)	62	(57-65)	<0.001
Average 24-h SBP (mmHg)	128	(118-141)	136	(126-145)	114	(110-120)	<0.001
Average 24-h DBP (mmHg)	73	(68-80)	77	(72.5-83)	66	(63-69)	<0.001
SBP drop during sleep (%)	2	(-3.3-7.3)	0.7	(-4.85-5.55)	5.8	(0.9-10.35)	<0.001
SBP dipping status*							<0.001
Reverse dippers, n (%)	77	(35.6)	65	(42.5)	12	(19)	
Non-dippers, n (%)	111	(51.4)	75	(49)	36	(57.1)	
Dippers, n (%)	28	(13)	13	(8.5)	15	(23.8)	

Reported values are medians (IQR) or counts (percent), *Extreme dipping category was excluded due to only 3 cases,
BP: Blood pressure, DBP: Diastolic blood pressure, n: Number of cases, SBP: Systolic blood pressure, IQR: Interquartile range

Table 3. Laboratory results according to the nocturnal hypertension status

	Nocturnal hypertension (+) (n=153, 69.9%)		Nocturnal hypertension (-) (n=66, 30.1%)		p
Hemoglobin, g/dL (n=219)	12.7	(11.5-13.8)	12.3	(11.4-13.7)	0.473
Total cholesterol, mg/dL (n=216)	187.5	(157.7-221)	195	(164.2-220.2)	0.525
LDL, mg/dL (n=218)	116	(88.2-139.7)	115	(94.5-136.7)	0.794
Triglyceride, mg/dL (n=217)	127	(95-169)	130	(95.5-170)	0.525
HDL, mg/dL (n=216)	46	(38-54)	47.5	(39-56)	0.426
Creatinine, mg/dL (n=219)	0.96	(0.76-1.19)	0.93	(0.77-1.18)	0.755
Albumin, g/dL (n=219)	4.2	(3.9-4.4)	4.1	(3.8-4.4)	0.915
CRP, mg/L (n=200)	3.87	(1.6-10.8)	5.8	(1.8-14.7)	0.224
Sedimentation rate, mm/hr (n=199)	19.5	(12-30.7)	23	(13-34)	0.232
25 (OH) D, ng/mL (n=219)	15.5	(9.47-23.3)	21.1	(12.1-30.6)	0.013
25 (OH) D groups, n (%)					
<30 ng/mL	137	(89.5)	48	(72.7)	0.002
25 (OH) D quartiles, n (%)					
1st quartile	43	(28.1)	12	(18.2)	0.015*
2nd quartile	41	(26.8)	14	(21.2)	
3rd quartile	38	(24.8)	17	(25.8)	
4th quartile	31	(20.3)	23	(34.8)	
Intact PTH, pg/mL (n=88)	87.1	(61.6-127.7)	62.5	(39.6-96.3)	0.048
Calcium, mg/dL (n=219)	9.5	(9.2-9.9)	9.5	(9.1-9.7)	0.374

Reported values are medians (IQR) or counts (percent), *p-value for trend across quartiles,
CRP: C-reactive protein, dL: Deciliter, HDL: High density lipoprotein, IQR: Interquartile range, g: Gram, LDL: Low density lipoprotein, n: Number of cases, mg: Milligram, ng: Nanogram, PTH: Parathormone, IQR: Interquartile range

Table 4. Results of univariate and multivariate analyses of predictors of nocturnal hypertension

	Univariate			Multivariate		
Variable	**OR**	**95% CI**	**p**	**OR**	**95% CI**	**p**
Age (continuous)	0.98	0.94-1.02	0.422	1.02	0.97-1.09	0.273
CCI (continuous)	1.08	0.92-1.27	0.297	0.92	0.73-1.17	0.926
25 (OH) vitamin D (<30 vs. ≥30 ng/mL	3.21	1.51-6.79	0.002	4.97	1.68-14.71	0.004
Average daytime systolic blood pressure (continuous)	1.15	1.10-1.19	<0.001	1.16	1.11-1.21	<0.001

CCI: Charlson comorbidity index, CI: Confidence interval, OR: Odds ratio

human genome is regulated by vitamin D, indicating a wider-ranging role for vitamin D beyond its traditional functions in regulating calcium and bone homeostasis (20). Moreover, cardiomyocytes, endothelial cells, and vascular smooth muscle cells have VDRs (21-23). This implies that vitamin D deficiency could potentially result in vascular dysfunction through these cardiovascular cells, trigger arterial atherogenesis, and play a role in HT by promoting arterial stiffness (24). In advanced age, independent of serum 25 (OH) vitamin D levels, a decrease in VDR expression has also been noted (25). Additionally, in mice with deactivated VDR or 1a-hydroxylase gene, an increase in renin expression and plasma angiotensin 2 production, leading to HT and cardiac hypertrophy, was observed (26,27). Vitamin D deficiency can lower blood calcium levels, leading to PTH release. In a study involving older adults, although no relationship was observed between HT and vitamin D levels, an association was noted with high PTH levels (28). This correlation was further supported by the administration of PTH, which resulted in elevated BP in healthy adults (29). Despite numerous studies examining the relationship between BP and vitamin D levels, the findings remain contradictory even across several recent studies (30,31). Moreover, conflicting results have also emerged from vitamin D supplementation trials investigating its potential for reducing BP. While studies focusing solely on the impact of vitamin D on HT often fail to demonstrate significant reductions, combining vitamin D supplementation with other standard antihypertensive medications appears to have more promising outcomes (32).

When investigating studies that evaluated the relationship between vitamin D levels and nocturnal BP decrease, it was observed that the number of studies was limited, and once again, the results were inconsistent. Although a few studies have indicated an association between low vitamin D levels and nondieting BP status, all of these studies were conducted with relatively younger patients (with mean ages≤50 years) compared with our study, and none of them directly evaluated nocturnal HT (33-35). However, another study showed that except for nocturnal diastolic BP decrease, there was no association between vitamin D levels and dipping status in hypertensive adults (36). Additionally, in a different study, which involved relatively older patients (mean age 61.8 years) compared with other studies, the relationship between the severity of autonomic dysfunction and dipping status with vitamin D levels in patients with Parkinson's Disease was explored. However, no correlation was found between any of these factors (37). Nocturnal HT is a stronger predictor of HT-related end organ damage and future cardiovascular mortality than daytime HT and dipping status.(4) Therefore, unlike other studies that primarily focused on evaluating dipping status, this study investigated the prevalence of nocturnal HT and its association with vitamin D and other clinical factors in older patients with hypertension. Furthermore, we provided a comprehensive evaluation of various patient factors, including functionalities, physical performance, and muscle strengths, together with comorbidities, antihypertensive medications, and related laboratory parameters.

Study Limitations

Our study has several limitations. This was a cross-sectional investigation, so it was incapable of determining a direct cause-and-effect relationship between vitamin D levels and nocturnal HT. Additionally, we lacked information on patients' sleep patterns or conditions that could cause sleep disturbances, such as OSAS, anxiety disorders, chronic pain conditions, or other undiagnosed conditions that may affect the sleep-wake cycle. Furthermore, we did not have data on whether the participants had any form of autonomic dysfunction that could contribute to nocturnal HT.

Conclusion

In conclusion, our study suggests a correlation between vitamin D insufficiency and nocturnal HT in older hypertensive adults, independent of age, comorbidity burden, or average daytime SBP values. This relationship appears to be mediated by various complex pathogenetic mechanisms and is influenced by multiple factors. The extensive occurrence of vitamin D insufficiency in older adults with nocturnal HT underscores the importance of screening serum 25 (OH) vitamin D levels when necessary. This practice appears to be beneficial for not only bone health but also BP regulation. Although the results of vitamin D supplementation trials on HT are conflicting, the advantages of vitamin D supplementation for both the skeletal and extraskeletal systems are notable. Additionally, its minimal side effects at appropriate dosages and cost-effectiveness

suggest that it may still be considered alongside traditional antihypertensive treatment, especially to sustain serum 25 (OH) vitamin D levels above 30 ng/mL in older adults. Nevertheless, further research is required to explore its potential protective or therapeutic effects on nocturnal HT through prospective studies.

Ethics

Ethics Committee Approval: This study adhered to the Declaration of Helsinki's principles and obtained ethical approval from Ankara University's ethical committee (approval number:17-1121-18, date: 22.10.2018).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.M.S., T.T., R.B., H.S.Ö., M.V., Concept: D.M.S., M.V., Design: D.M.S., M.V., Data Collection or Processing: D.M.S., T.T., R.B., H.S.Ö., Analysis or Interpretation: D.M.S., M.V., Literature Search: D.M.S., T.T., R.B., H.S.Ö., Writing: D.M.S., M.V.

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