

# Relationship between Serum Parathyroid Hormone Levels and 24-Hour Ambulatory Blood Pressure Monitoring Parameters in Hypertensive Patients

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## Abstract

**Background:** We conducted this study to investigate and evaluate the correlation between the serum parathyroid hormone (PTH) concentrations and 24-hour ABPM parameters in hypertensive patients

**Methods and Results:** A cross-sectional descriptive study was conducted in the Cardiology Department of Hue Central Hospital on 74 hypertensive patients. All patients underwent 24-hour ABPM. Serum PTH was measured using a sandwich electrochemiluminescence immunoassay. The mean values of daytime systolic blood pressure (SBP), daytime diastolic blood pressure (DBP), nighttime SBP, and nighttime DBP were  $127.74 \pm 14.44$  mmHg,  $77.24 \pm 9.01$  mmHg,  $122.86 \pm 16.57$  mmHg, and  $76.92 \pm 11.69$  mmHg, respectively. The median value of serum PTH concentration was 68.6 pg/mL (39.83 pg/mL – 87.46 pg/mL).

All patients were divided into two groups. Group 1 included 40 AH patients with serum PTH  $> 65$  pg/mL, and Group 2 included 34 AH patients with serum PTH  $\leq 65$  pg/mL. The mean values of 24-hour SBP, nighttime SBP, nighttime DBP, and nighttime mean blood pressure (MBP) in Group 1 were significantly higher than in Group 2 ( $P < 0.05$ ). Meanwhile, the patients of Group 1 had a statistically significantly higher rate of nocturnal blood pressure dipping and early morning blood pressure surge than the patients of Group 2 ( $P < 0.01$ ). We found a moderate positive correlation between serum PTH concentration and nighttime SBP ( $r = 0.595$ ;  $P < 0.001$ ), nighttime DBP ( $r = 0.666$ ;  $P < 0.001$ ), and nighttime MBP ( $r = 0.560$ ;  $P < 0.001$ ). In multivariate regression analysis, only nighttime SBP and nighttime DBP were two independent factors that significantly predicted the PTH index.

**Conclusion:** The serum PTH concentration shows a moderate positive correlation with nocturnal DBP, nocturnal SBP, and nocturnal MBP. Only nocturnal DBP and nocturnal SBP are independent factors significantly predicting PTH levels. (**International Journal of Biomedicine. 2024;14(4):545-550.**)

**Keywords:** arterial hypertension • parathyroid hormone • ambulatory blood pressure monitoring

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## Abbreviations

AH, arterial hypertension; ABPM, ambulatory blood pressure monitoring; BP, blood pressure; BMI, body mass index; CKD, chronic kidney disease; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; MBP, mean BP; MBPS, morning blood pressure surge; PHPT, primary hyperparathyroidism; PTH, parathyroid hormone; SBP, systolic blood pressure.

## Introduction

Arterial hypertension is a leading cause of morbidity and mortality, afflicting more than 1.2 billion people across

the globe.<sup>1</sup> The heart, kidney, brain, retina, and arterial blood vessels are prime targets of hypertensive damage. Uncontrolled and untreated hypertension accelerates the damage to these organs and could cause their failure. Damage

to these organs could also manifest as coronary heart disease, cognitive impairment, retinopathy, or optic neuropathy. For a better understanding, it is important to analyze molecular factors in the pathogenesis of hypertension and hypertension-related target organ damage.<sup>2</sup>

Depending on whether a clear cause can be found, hypertension is divided into two categories: essential (primary) hypertension without a definite cause and secondary hypertension with a definite cause. Essential hypertension accounts for more than 90% of all hypertensive patients.<sup>3</sup> There are many reported causes of secondary hypertension, such as renal parenchymal disease, endocrine disorders, renal artery stenosis, and aortic coarctation.<sup>4</sup> Thyroid disorders make up the common treatable causes of secondary hypertension.<sup>5</sup> In addition, parathyroid hormone (PTH) has been related to the risk of hypertension, but the matter remains controversial.<sup>6</sup> The association between PTH and hypertension remains unclear. Still, it has been attributed to the combined effect of persistently high serum levels of calcium, PTH, and other vasoactive, calcium-regulating hormones and/or renal function impairment. Furthermore, excessive occurrence of cardiovascular complications and mortality has been reported in patients with primary hyperparathyroidism (PHPT).<sup>7</sup>

Ambulatory blood pressure monitoring (ABPM) allows the collection of blood pressure (BP) readings several times an hour across 24 hours. The various BP categorizations afforded by ABPM are valuable for the clinical management of high BP since they increase the accuracy of diagnosis and prediction of cardiovascular risk. Patients with nocturnal hypertension are strongly associated with increased cardiovascular morbidity and mortality.<sup>8</sup>

The data on the correlation between thyroid hormones and 24-hour ABPM in hypertensive patients is still limited. We conducted this study to investigate and evaluate the correlation between the serum PTH concentrations and 24-hour ABPM parameters in hypertensive patients.

## Materials and Methods

This cross-sectional descriptive study was conducted in the Cardiology Department of Hue Central Hospital on hypertensive patients from February 2022 to April 2023. The study included patients aged 18 and above who met the criteria for AH according to the 2018 ESC/ESH guidelines, with a diagnosis of essential hypertension or newly diagnosed hypertension upon admission.<sup>2</sup> We excluded cases with existing thyroid or parathyroid disease or parathyroid surgery, patients with a history of CKD, and patients using drugs that affect metabolism, bone, and vitamin D in the last four weeks, such as anticonvulsants, isoniazid, lithium, rifampin, and phosphate-containing drugs. Patients with less than 70% adherence to 24-hour ABPM were also excluded from the study.

Blood samplings were performed during the morning (7 a.m. – 11 a.m.) after 12 hours of fasting. Before blood sampling, patients remained seated for at least 10 minutes. All blood-derived parameters were determined at least 4 hours after collection. Before the analyses, all samples

were kept at room temperature, except for PTH, which was kept at 4°C. Serum PTH was measured using a sandwich electrochemiluminescence immunoassay on a Cobas 8000. The patients underwent 24-hour ABPM using the ScottCare ABP320 device (the United States). The data were analyzed using the ABPCare software following standard procedures, with automatic updates of parameters. Daytime BP was measured from 6 a.m. to 9.59 p.m. (automatically measured every 30 minutes), and nighttime BP was measured from 10 p.m. to 5.59 a.m. (automatically measured every 60 minutes). Patients with a success rate of  $\geq 70\%$  of the total number of measurements and no disruptions for a consecutive 2 hours or more were considered to have successful ambulatory monitoring. The study also collected demographic variables such as age, gender, body mass index (BMI), and clinical variables, including a history of hypertension, vital signs, and laboratory variables including glucose, creatinine, ionized calcium, total calcium, and lipid. The estimated glomerular filtration rate (eGFR) is predicted based on blood creatinine concentration and estimated using the CKD-EPI 2021 formula.

Statistical analysis was performed using the statistical software package SPSS version 20.0 (SPSS Inc, Armonk, NY: IBM Corp). The normality of the distribution of continuous variables was tested by a one-sample Kolmogorov-Smirnov test. Baseline characteristics were summarized as frequencies and percentages for categorical variables. Continuous variables with normal distribution were presented as mean (standard deviation [SD]); non-normal variables were reported as median (interquartile range [IQR]). Means of 2 continuous normally distributed variables were compared by independent samples Student's t-test. The Mann-Whitney U test was used to compare the means of 2 groups of variables that were not normally distributed. The frequencies of categorical variables were compared using the chi-square test. Pearson's correlation coefficient ( $r$ ) was used to determine the strength of the relationship between the two continuous variables. We stratified PTH levels according to tertiles to present the baseline characteristics of the cohort. In case of a significant bivariate correlation, we performed multivariate linear regression analyses with PTH for the respective ABPM reading. A value of  $P < 0.05$  was considered significant.

## Ethical Considerations

The study protocol was reviewed and approved by the Ethics Committee at the Hue University of Medicine and Pharmacy. Written informed consent was obtained from all the participants.

## Results

During the study period, 74 patients with hypertension were included. All patients were divided into two groups. Group 1 included 40 AH patients with serum PTH  $> 65$  pg/mL, and Group 2 included 34 AH patients with serum PTH  $\leq 65$  pg/mL. The average age in the study was  $70.12 \pm 13.84$  years, and men accounted for 50%. The mean values of daytime SBP, daytime DBP, nighttime SBP, and nighttime DBP

were  $127.74 \pm 14.44$  mmHg,  $77.24 \pm 9.01$  mmHg,  $122.86 \pm 16.57$  mmHg, and  $76.92 \pm 11.69$  mmHg, respectively. The median value of serum PTH concentration was 68.6 pg/mL (39.83 pg/mL – 87.46 pg/mL).

Office SBP, the ionized calcium and total calcium concentrations in Group 1 were higher than in Group 2. Other indicators had no difference between the study groups (Table 1).

The mean values of 24-hour SBP, nighttime SBP, nighttime DBP, and nighttime MBP in Group 1 were significantly higher than in Group 2 ( $P < 0.05$ ). Other BP indices and the heart rate showed no significant differences between the two groups. Meanwhile, the patients of Group 1 had a statistically significantly higher rate of nocturnal BP dipping and early morning BP surge than the patients of Group 2 ( $P < 0.01$ ) (Table 2).

The serum PTH concentration showed a moderate positive correlation with ionized calcium levels ( $r = 0.479$ ;  $P < 0.001$ ) and total calcium levels ( $r = 0.553$ ;  $P < 0.001$ ). The serum PTH concentration weakly correlated with 24-hour MBP ( $r = 0.299$ ;  $P = 0.01$ ) and daytime MBP ( $r = 0.231$ ;  $P = 0.047$ ). Additionally, there was a moderate positive correlation between serum PTH concentration and nighttime SBP ( $r = 0.595$ ;  $P < 0.001$ ), nighttime DBP ( $r = 0.666$ ;  $P < 0.001$ ), and nighttime MBP ( $r = 0.560$ ;  $P < 0.001$ ) (Table 3, Figure 1).

Multivariate regression analysis has a common correlation coefficient of  $R = 0.645$ . Parameters on 24-hour ABPM explain about 41.7% of the variation in PTH. Only nighttime SBP and nighttime DBP were two independent factors that significantly predicted the PTH index (Table 4).

**Table 1.**

**Clinical and laboratory characteristics of the study patients.**

	Total patients (n=74)	Group 1 (n=40)	Group 2 (n=34)	P-value
Age, year	70.12 ± 13.84	68.55 ± 13.97	71.97 ± 13.65	>0.05
Male, no (%)	37 (50)	22 (55)	15 (44.1)	>0.05
BMI, kg/m <sup>2</sup>	22.33 ± 2.87	22.09 ± 3.08	22.62 ± 2.62	>0.05
Hypertension history >5 years, n (%)	30 (40.5)	19 (47.5)	11 (32.4)	0.186
Office SBP, mmHg	140 (130 – 160)	150 (130 – 160)	140 (127.5 – 150)	0.047
Office DBP, mmHg	80 (77.5 – 90)	90 (80 – 90)	80 (70 – 90)	>0.05
PTH, pg/mL	68.60 (39.83 – 87.46)	86.24 (73.31 – 115.6)	38.84 (25.15 – 45.28)	-
FBG, mmol/L	5.89 (5.09 – 7.20)	5.75 (4.87 – 6.69)	6.15 (5.40 – 8.24)	>0.05
Creatinine, μmol/L	88.95 (74.38 – 118.63)	91.1 (76.05 – 128.83)	87.25 (64.84 – 113.38)	>0.05
eGFR, ml/min/1.73m <sup>2</sup>	68.20 ± 24.35	70.5 (46.5 – 88.5)	72.5 (45.75 – 91)	>0.05
Ionized calcium, mmol/L	1.2 (1.11 – 1.37)	1.36 (1.13 – 1.43)	1.15 (1.09 – 1.23)	<0.001
Total calcium, mmol/L	2.37 ± 0.18	2.53 (2.29 – 2.58)	2.31 (2.26 – 2.36)	0.001
LDL-C, mmol/L	2.82 ± 1.12	2.80 ± 1.02	2.85 ± 1.25	>0.05
Triglycerides, mmol/L	1.39 (1.08 – 2.12)	1.39 (0.87 – 2.17)	1.38 (1.11 – 1.95)	>0.05

**Table 2.**

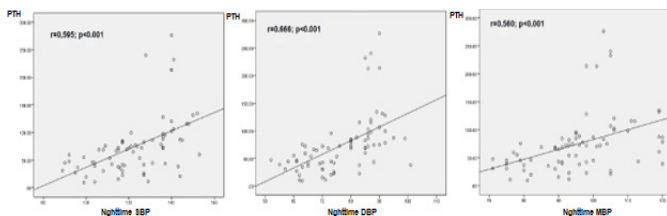
**The 24-hour ABPM indicators in the study groups.**

	Total patients (n=74)	Group 1 (n=40)	Group 2 (n=34)	P-value
Average 24-h SBP, mmHg	127.58 ± 13.38	129.38 ± 12.66	125.47 ± 14.08	>0.05
Average 24-h DBP, mmHg	77.51 ± 9.35	78.48 ± 9.54	76.38 ± 9.13	>0.05
Average 24-h MBP, mmHg	97 ± 11.18	101.5 (90.5 – 106.5)	92 (82.75 – 103)	0.026
Average daytime SBP, mmHg	127.74 ± 14.44	129.7 ± 13.92	125.44 ± 14.90	>0.05
Average daytime DBP, mmHg	77.24 ± 9.01	77.95 ± 9.30	76.41 ± 8.72	>0.05
Average daytime MBP, mmHg	98.65 ± 11.2	100.13 ± 10.69	96.91 ± 11.70	>0.05
Average nighttime SBP, mmHg	122.86 ± 16.57	130.35 ± 12.57	114.06 ± 16.53	<0.001
Average nighttime DBP, mmHg	76.92 ± 11.69	83.65 ± 8.14	69.0 ± 10.22	<0.01
Average nighttime MBP, mmHg	95.66 ± 12.71	101.03 ± 9.90	89.35 ± 12.87	<0.001
“Non-dipper” n (%)	52 (70.3)	35 (87.5)	17 (50)	<0.001
MBPS, n (%)	31 (41.9)	26 (65)	5 (14.7)	<0.001

Table 3.

**Bivariate correlations between serum PTH level and 24-hour ABPM parameters.**

	Serum PTH, pg/mL	
	r	P-value
Age, y	-0.144	0.221
BMI, kg/m <sup>2</sup>	-0.174	0.139
Creatinine, μmol/L	0.133	0.258
Ionized calcium, mmol/L	0.479	<0.001
Total calcium, mmol/L	0.533	<0.001
Average 24-h SBP, mmHg	0.216	0.064
Average 24-h DBP, mmHg	0.165	0.159
Average 24-h MBP, mmHg	0.299	0.010
Average daytime SBP, mmHg	0.183	0.119
Average daytime DBP, mmHg	0.160	0.175
Average daytime MBP, mmHg	0.231	0.047
Average nighttime SBP, mmHg	0.595	<0.001
Average nighttime DBP, mmHg	0.666	<0.001
Average nighttime MBP, mmHg	0.560	<0.001



**Fig. 1.** The correlations between serum PTH concentration and 24-hour ABPM parameters

Table 4.

**Relationship between serum PTH level and 24-hour ABPM parameters for 74 hypertensive patients in a multivariate linear regression model.**

	Serum PTH			
	B	P-value	R	R <sup>2</sup>
Constant	-93.649	0.047	0.645	0.417
Average 24-h MBP, mmHg	-1.707	0.108		
Average daytime MBP, mmHg	0.481	0.564		
Average nighttime SBP, mmHg	2.216	0.002		
Average nighttime DBP, mmHg	2.698	0.002		
Average nighttime MBP, mmHg	-2.020	0.118		

## Discussion

Thus, the mean values of 24-hour SBP, nighttime SBP, nighttime DBP, and nighttime BP in AH patients with increased PTH were higher than those without increased PTH. At the same time, the AH patients with increased PTH had a statistically significant higher rate of nocturnal BP depression and early morning BP surge than the AH patients without increased PTH.

Several studies have found a relationship between hypertension and PTH and vitamin D levels in the body. Research by Nicolas and colleagues indicated that PTH increased in patients with elevated nocturnal DBP and in patients with average nocturnal SBP.<sup>10</sup> A study by Letizia et al.,<sup>11</sup> through the 24-hour ABPM of 53 patients with PHPT compared to 100 hypertensive patients and 31 healthy subjects, reveals that the average 24-hour SBP and average 24-hour DBP were higher in the group of patients with PHPT and hypertension ( $P<0.05$ ), as well as in the hypertensive group ( $P<0.05$ ), than in the group of patients with PHPT without hypertension and the healthy subject group. A study by Morfis et al.<sup>12</sup> on the correlation between PTH and 24-hour ABPM parameters in 123 elderly individuals reveals a strong association between serum PTH concentration and 24-hour BP, particularly nocturnal SBP, which is less related to nocturnal DBP, daytime SBP, daytime DBP, and average 24-hour SBP. Daytime DBP and average 24-hour DBP are unrelated to serum PTH concentration. Ballegooijen and colleagues<sup>13</sup> studied 3002 men and women without prevalent cardiovascular disease and hypertension, aged 45 to 84 years. Over an average follow-up period of 9 years, the results showed that a PTH concentration  $\geq 65$  pg/mL was significantly associated with an increased risk of hypertension after adjusting for potential confounding factors.

Primary hyperparathyroidism is associated with an increased risk of hypertension, with prevalence rates ranging from 40% to 65%.<sup>14</sup> Parathyroid hormone is a principal regulator of calcium balance in physiological and pathological conditions associated with cardiovascular disorders and plays a major physiological role in bone homeostasis. PTH receptors presenting in the cardiovascular system, including blood vessels and the heart, suggest that secreted PTH may play a role in the pathophysiology of cardiovascular diseases beyond its role in mineral and bone metabolism.<sup>15</sup> Clinically, patients with PHPT are at an increased risk of cardiovascular-related mortality and exhibit various unfavorable cardiac disorders, such as coronary artery dysfunction, subclinical aortic valve calcification, increased arterial stiffness, endothelial dysfunction, and elevated BP.<sup>16</sup> The research conducted by Hagström and colleagues<sup>17</sup> emphasizes that PTH is a strong predictive risk factor for both clinical and subclinical atherosclerosis. This study involved two population groups in the Swedish community, comprising over 1000 individuals aged 70 years or older. The first prospective study group (PIVUS) revealed that PTH is independently associated with the burden of atherosclerosis, even after adjusting for cardiovascular risk factors and metabolic variables. In the second independent cohort study (ULSAM) with a follow-up

period of 16.7 years, PTH was linked to a long-term increased risk of non-fatal atherosclerosis in both peripheral and large arteries, contributing to 11.6% of the overall risk in the population. PTH level in patients with stages 3 and 4 CKD is associated with increased incidence of cardiovascular events independent of calcium-phosphorous level.<sup>18</sup>

The suspicion for the existence of a pathophysiological relationship between hyperparathyroidism and BP increase has a long history. In 1996, Goldsmith et al.<sup>19</sup> published the results of a study conducted on 21 hypertensive hemodialysis patients with tertiary hyperparathyroidism who had undergone parathyroidectomy. Interestingly, BP values, mean plasma calcium levels, and heart rate were significantly reduced after the surgical procedure. However, post-surgery PTH decrease was not followed by a prompt BP normalization, which was observed a few months later. Consequently, Goldsmith et al.<sup>19</sup> hypothesized that this effect was linked to a long-term corporeal re-arrangement of calcium distribution. This hypothesis has been sustained and confirmed by other authors describing a mechanism by which, following post-surgery PTH lowering, the removal of calcium from the vessel walls might explain a BP improvement related to reduced vessel stiffness. Several recent studies investigating this important issue also reported direct iPTH effects on vascular stiffness. Specifically, PTH stimulates PTH2 receptors expressed on vascular smooth muscle cells with a consequent increase in collagen production.<sup>20</sup> Another important study suggested that the mechanism underlying hypertensive disease in PHPT involves the complex interaction between PTH and the renin-angiotensin-aldosterone system (RAAS). There is increasing evidence of a bidirectional relationship between PTH and RAAS, whereby increased PTH leads to subsequent activation of RAAS and vice versa.<sup>21</sup> Several epidemiological and prospective studies have indicated a higher prevalence of hypertension in patients with PHPT. In a longitudinal observational study by Yao and colleagues, elevated PTH levels in Black individuals were associated with an increased risk of developing new-onset hypertension over an average follow-up period of 6 years.<sup>22</sup>

Furthermore, there is a correlation between PTH and nighttime BP, which may be attributed to the circadian rhythm of PTH secretion. PTH reaches its highest levels during the night, typically between 2 a.m. and 4 a.m. A study on 292 hypertensive patients in the Styrian Hypertension Study<sup>10</sup> demonstrated that higher PTH levels were associated with elevated nighttime BP rather than daytime BP indices.

Our study also has some limitations. It is a single-center study conducted at a hospital, which may introduce selection bias related to the study's single-center nature and small sample size. Another limitation is that we allowed patients with pre-existing hypertension to continue using antihypertensive medications. This could potentially diminish the relationship between BP and PTH levels.

## Conclusion

Our study reveals that among the hypertensive patients investigated, the serum PTH concentration shows a moderate

positive correlation with nocturnal DBP, nocturnal SBP, and nocturnal MBP. Only nocturnal DBP and nocturnal SBP are independent factors significantly predicting PTH levels.

## Competing Interests

The authors declare that they have no competing interests.

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