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The Association Between Platelet Mass Index and Morning Blood Pressure Surge in Newly Diagnosed Hypertensive Patients

Yeni Tanı Almış Hipertansif Hastalarda Platelet Mass İndeksi ile Sabah Kan Basıncı Dalgalanması Arasındaki İlişki

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ÖZ

Amaç: Bu çalışma, yeni tanı almış hipertansiyon hastalarında Platelet Mass İndeksi (PMI) ile Sabah Kan Basıncı Dalgalanması (SKBD) arasındaki ilişkiyi değerlendirmeyi amaçlamaktadır.
Araçlar ve Yöntem: Ocak 2025 ile Nisan 2025 tarihleri arasında bir üçüncü basamak sağlık merkezinin kardiyoloji polikliniğinde yürütülen bu kesitsel çalışmaya, 158 hipertansif ve 191 normotansif birey dahil edilmiştir. Kan basıncı ölçümleri Ayaktan Kan Basıncı İzleme (AKBİ) cihazı ile kaydedilmiş, sabah kan basıncı dalgalanması hesaplanmıştır. Tam kan sayımı parametrelerinden elde edilen Platelet Mass İndeksi, trombosit sayısının ortalama trombosit hacmi (MPV) ile çarpılmasıyla hesaplanmıştır. SKBD'nin öngörülmesinde PMI'nin performansı Alıcı İşletim Karakteristiği (AIK) eğrisi analizi ile değerlendirilmiştir.
Bulgular: Hipertansif grupta 24 saatlik, gündüz ve gece sistolik ve diyastolik kan basınçları anlamlı olarak yüksekti (hepsi $p<0.001$). SKBD, hipertansif bireylerde anlamlı şekilde daha fazlaydı (medyan: 28.2 mmHg; $p=0.007$). SKBD değeri >28.2 mmHg olan hastalarda trombosit sayısı, PMI, Trombosit-Lenfosit Oranı (PLO) ve nötrofil düzeyleri anlamlı olarak yüksekti ($p<0.001$). PMI ile PLO arasında güçlü pozitif bir korelasyon gözlenirken ($r=0.518$, $p<0.001$), PMI ile gece sistolik kan basıncı arasında zayıf ancak anlamlı negatif bir ilişki saptandı ($r=–0.167$, $p=0.036$). AIK analizine göre, PMI'nin SKBD'yi öngörmedeki AUC değeri 0.749 olup, %67.1 duyarlılık ve %67.1 özgüllük ile en yüksek ayırt edici güce sahipti.
Sonuç: Platelet Mass İndeksi'nin artmış SKBD ile anlamlı ilişkili olduğu gösterilmiştir. Bulgular, PMI'nin inflamasyon aracılı vasküler stresin bir yansıması olabileceğini ve erken dönemde kardiyovasküler risk belirteci olarak kullanılabileceğini düşündürmektedir.

Anahtar Kelimeler: basınç değişkenliği; basınç paterni; sirkadiyen ritim; trombosit aktivasyonu; vasküler enflamasyon

ABSTRACT

Purpose: This study aimed to evaluate the relationship between Platelet Mass Index (PMI) and Morning Blood Pressure Surge (MBPS) in patients with newly diagnosed hypertension.
Materials and Methods: A cross-sectional study was conducted between January and April 2025 at a tertiary cardiology outpatient clinic, including 158 hypertensive and 191 normotensive individuals. Blood pressure was monitored using Ambulatory Blood Pressure Monitoring (ABPM), and MBPS was calculated. PMI was derived by multiplying the platelet count by the mean platelet volume (MPV). The predictive ability of PMI for identifying elevated MBPS was assessed via receiver operating characteristic (ROC) curve analysis.
Results: Systolic and diastolic blood pressure values across 24-hour, daytime, and nighttime measurements were significantly higher in the hypertensive group (all $p<0.001$). MBPS was also significantly increased in hypertensive patients (median: 28.2 mmHg; $p=0.007$). Patients with MBPS >28.2 mmHg showed significantly higher platelet count, PMI, PLR, and neutrophil levels ($p<0.001$). PMI was strongly correlated with PLR ($r=0.518$, $p<0.001$), and inversely associated with nighttime systolic BP ($r=–0.167$, $p=0.036$). ROC analysis revealed that PMI had the highest discriminatory power with an AUC of 0.749, sensitivity of 67.1%, and specificity of 67.1%.
Conclusion: PMI was significantly associated with exaggerated MBPS, suggesting that platelet activation may contribute to vascular stress and circadian BP dysregulation. PMI may represent a useful early biomarker for cardiovascular risk stratification in hypertensive patients.

Keywords: blood pressure pattern; blood pressure variability; circadian rhythm; platelet activation; vascular inflammation

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INTRODUCTION

Hypertension (HT) continues to be a major global public health challenge, representing one of the most common chronic medical conditions encountered in clinical practice. It is a key determinant of cardiovascular morbidity and mortality, substantially contributing to the development of ischemic heart disease, heart failure, stroke, and chronic kidney disease.[1] Blood pressure (BP) is known to exhibit a distinct circadian variation, typically decreasing during nighttime sleep and rising sharply in the early morning hours—a phenomenon described as the morning blood pressure surge (MBPS).[2] This early-morning elevation in BP is believed to result from the activation of the sympathetic nervous system, increased plasma catecholamine levels, and heightened vascular tone. Importantly, an exaggerated MBPS has been independently linked to a heightened risk of subclinical organ damage as well as major cardiovascular complications, including cerebrovascular accidents, acute coronary syndromes, and sudden cardiac death. Therefore, understanding the clinical significance of MBPS and identifying its determinants may have important implications for risk stratification and management in hypertensive individuals.[3,4]

Although the underlying mechanisms of MBPS are multifactorial, recent research has highlighted the role of inflammation and platelet activation in its pathogenesis. In this context, various hematological indices derived from routine blood counts—such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR)—have been investigated as surrogate markers of subclinical inflammation and BP variability.[5-7] However, these parameters may not fully capture the combined effects of platelet number and function.

Platelet Mass Index (PMI), calculated as the product of platelet count and mean platelet volume (MPV), has emerged as a novel marker reflecting total platelet mass and functionality. Previous studies have linked elevated PMI to pro-inflammatory and pro-thrombotic states in various clinical scenarios, including acute ischemic stroke, cardiovascular surgery, and autoimmune conditions.[8,9] Moreover, PMI has shown superior predictive performance compared to MPV or platelet count alone in assessing systemic inflammation and vascular risk.[10,11]

Despite growing evidence of the interplay between platelet function and blood pressure variability, the association between PMI and MBPS in newly diagnosed, untreated hypertensive patients remains unexplored. Considering the growing understanding of platelets' roles in vascular inflammation and circadian blood pressure regulation, this study sought to explore the possible link between PMI and MBPS. We hypothesized that elevated PMI values might be associated with exaggerated MBPS, potentially providing a simple, cost-effective marker for early vascular risk stratification in essential HT.

MATERIALS and METHODS

This cross-sectional observational study was conducted at the cardiology outpatient clinic of a tertiary healthcare center between January 2025 and April 2025. This study has been reviewed and approved by the Health Sciences Ethics Committee of Kırşehir Ahi Evran University Faculty of Medicine (dated 07.01.2025, and numbered 2025-01/01).

Study Population and Sample Size

Based on a power analysis (effect size =0.50, α =0.05, power =0.95) performed with GPower (version 3.1.9.4), 88 participants were needed per group, resulting in a total of 176 subjects. Inclusion criteria encompassed newly diagnosed, treatment-naïve patients with essential HT aged between 18 and 75 years. HT was diagnosed based on current European Society of HT guidelines using 24-hour ambulatory blood pressure monitoring (ABPM).[12] Exclusion criteria were as follows: known history of secondary hypertension, coronary artery disease, diabetes mellitus, heart failure, chronic kidney disease (eGFR<60 mL/min/1.73 m²), active infection, current use of antibiotics, systemic inflammatory or autoimmune disorders, hematologic disease, malignancy, and recent use (within the past 4 weeks) of antihypertensive, antiplate-

let, anti-inflammatory, or anticoagulant medications. Additional exclusion criteria included pregnancy, known sleep disorders such as obstructive sleep apnea, morbid obesity (BMI>40 kg/m²), and significant anemia (hemoglobin<10 g/dL), as these conditions may influence MBPS and hematologic indices. Individuals with irregular sleep-wake patterns or incomplete ABPM data were also excluded.

ABPM and MBPS Calculation

Ambulatory blood pressure measurements were obtained using a validated oscillometric device programmed to record BP at 20-minute intervals during the day (07:00–22:00) and at 30-minute intervals overnight (22:00–07:00). The MBPS was determined by subtracting the lowest nighttime systolic blood pressure from the average systolic blood pressure during the first two hours after waking, using the mean of three consecutive readings with the lowest values recorded during sleep. Wake and sleep times were based on self-reported diaries and confirmed via device programming.

The ABPM device (The Mobil-O-Graph manufactured by I.E.M. GmbH, Stolberg, Germany) used in this study is clinically validated according to the European Society of Hypertension International Protocol. Calibration procedures were performed prior to each monitoring session as per manufacturer guidelines. A minimum of 70% valid readings over 24 hours, with at least 14 daytime and 7 nighttime measurements, was required for inclusion in the final analysis.

Laboratory Measurements and Platelet Mass Index Calculation

Venous blood samples were collected in the early morning hours after an overnight fast. Complete blood count parameters including platelet count and MPV were analyzed using an automated hematology analyzer. The PMI was calculated by multiplying the platelet count (×10³/μL) by MPV (fL), yielding a composite marker that reflects total circulating platelet mass. Additional indices such as NLR and PLR were derived to assess systemic inflammatory burden.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 29.0 (Armonk, NY, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Variables with normal distribution were expressed as mean ± standard deviation and compared using the independent samples t-test. Non-normally distributed variables were summarized as medians with interquartile ranges (25th–75th percentiles) and compared using the Mann–Whitney U test. Categorical variables were presented as frequencies and percentages and compared using the chi-square test. To explore the relationship between PMI and MBPS, correlation analysis was performed using Spearman's rank correlation coefficient.

Furthermore, Receiver Operating Characteristic (ROC) curve analysis was employed to assess the discriminative performance of PMI, NLR, and PLR in identifying patients with MBPS values exceeding the median threshold of 28.2 mmHg. Optimal cutoff values, along with sensitivity, specificity, and area under the curve (AUC) statistics, were reported to evaluate diagnostic accuracy.

RESULTS

A total of 349 participants were included in the study, comprising 191 normotensive individuals and 158 newly diagnosed, untreated hypertensive patients. Demographic and laboratory parameters are summarized in Table 1. No statistically significant differences were observed between the groups in terms of age, sex distribution, lipid profile, hemoglobin, or leukocyte counts (all p>0.05). However, platelet counts were significantly higher in the hypertensive group compared to controls (median:269 vs. 253×10³/μL, p=0.021). Similarly, the PMI and PLR were significantly elevated in hypertensive patients (p=0.039 and p=0.006, respectively), whereas MPV and NLR values showed no significant group differences.

Ambulatory blood pressure data revealed markedly higher systolic and diastolic values across all measurement periods (24-hour, daytime, and nighttime) in the hypertensive cohort

(p<0.001 for all). Furthermore, the MBPS was significantly greater in hypertensive patients (median:28.2 mmHg) compared to controls (median:24.75 mmHg, p=0.007).

Table 1. Comparison of demographic and laboratory characteristics of Controls and Hypertensive groups.

Variable	Control Group (n=191)	Hypertensive (n=158)	p-value
Age, year	54 (44-63)	57.5 (45.75-65)	0.098
Gender (male), n (%)	84 (44)	83 (52.5)	0.111
LDL, mg/dl	112.69±35.42	122.37±50.11	0.069
HDL, mg/dl	48.54±13.18	47.68±12.96	0.599
Triglyceride, mg/dl	139 (95.25-214)	161.5 (101.5-223.75)	0.151
Hgb, g/dl	14.3 (13.2-15.53)	14.4 (13.15-15.68)	0.452
White Blood Cell, $10^3/\mu l$	7.93±2.10	8.63±3.49	0.232
Platelet, $10^3/\mu l$	253 (217-298)	269 (225-315)	**0.022**
Neutrophil, $10^3/\mu l$	2.38 (1.93-2.9)	2.31 (1.85-2.99)	0.087
Lymphocyte, $10^3/\mu l$	2.59±1.13	2.57±1.20	0.896
MPV	10.4 (9.9-11.1)	10.45 (9.6-11.13)	0.277
PMI	24.04 (20.58-28.79)	25.7 (21.22-29.89)	**0.039**
PLR	105.73 (82.7-130.14)	116.41 (86.35-155.18)	**0.006**
NLR	1.79 (1.43-2.3)	1.99 (1.43-2.65)	0.094
Creatinine, mg/dl	0.75 (0.67-0.89)	0.79 (0.7-0.90)	0.062
24-h SBP, mmHg	111 (106-116)	130 (124-137)	**<0.001**
24-h DBP, mmHg	63 (59-68)	77 (71-82)	**<0.001**
Daytime SBP, mmHg	113 (108-120)	132 (123.75-140)	**<0.001**
Daytime DBP, mmHg	66 (61-71)	78 (71-85)	**<0.001**
Nighttime SBP, mmHg	105 (99-112)	126 (120-134)	**<0.001**
Nighttime DBP, mmHg	59 (54-64)	73 (67-79)	**<0.001**
MBPS, mmHg	24.75 (16-33.5)	28,2 (19.94-37.49)	**0.007**

Values are presented as n (%), median (25th–75th percentiles), or mean ± standard deviation, as appropriate. Parametric tests (Student's t-test) were used for normally distributed variables (LDL, HDL, WBC, and lymphocyte counts), while non-parametric tests (Mann–Whitney U test) were applied to non-normally distributed variables. Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein; Hgb, hemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; MBPS, morning blood pressure surge; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PMI, platelet mass index; MPV, mean platelet volume

Among hypertensive individuals, stratification by MBPS median value (28.2 mmHg) identified distinct hematologic differences (Table 2). Patients with MBPS>28.2 mmHg exhibited significantly higher platelet counts, PMI, PLR, and neutrophil levels compared to those with lower MBPS values (p<0.001, p<0.001, p=0.004, and p=0.002, respectively). MPV and NLR did not significantly differ between the two subgroups.

Table 2. Comparison of demographic and laboratory characteristics according to the median value of MBPS in hypertensive patients.

Variable	MBPS<28.20 (n=79)	MBPS>28.20 (n=79)	*p-value*
Age, year	58 (48-66)	57 (45-64)	0.397
Platelet, $10^3/\mu l$	239 (202-274)	295 (261-372)	**<0.001**
Neutrophil, $10^3/\mu l$	4.14 (3.57-5.19)	5.28 (3.82-6.7)	**0.002**
Lymphocyte, $10^3/\mu l$	2.21 (1.75-2.84)	2.34 (1.99-3.06)	0.188
MPV	10.5 (9.7-11.3)	10.1 (9.4-11)	0.211
PMI	23.48 (18.11-26.64)	28.44 (23.82-35.05)	**<0.001**
PLR	103.18 (78.7-136.07)	127.66 (99.23-163.09)	**0.004**
NLR	1.93 (1.36-2.39)	2.08 (1.44-2.88)	0.258

Values are presented as median (25th–75th percentiles). All variables were analyzed using non-parametric tests (Mann–Whitney U test). Abbreviations: MBPS: Morning Blood Pressure Surge, NLR: Neutrophil to lymphocyte ratio, PLR: Platelet to lymphocyte ratio, PMI: Platelet Mass Index, MPV: Mean Platelet Volume

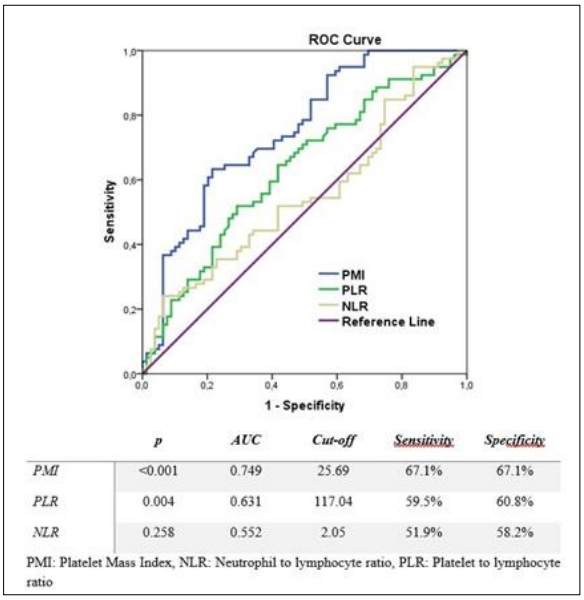
Correlation analysis demonstrated a strong positive association between PMI and PLR (r=0.518, p<0.001), while a weak but significant inverse correlation was observed between PMI and nighttime systolic blood pressure (r=-0.167, p=0.036) (Table 3).

ROC curve analysis was performed to evaluate the predictive capacity of PMI, PLR, and NLR for identifying patients with MBPS>28.2 mmHg (Figure 1). PMI yielded the highest diagnostic accuracy with an AUC of 0.749 (95% CI not shown), demonstrating a cutoff value of 25.69, with 67.1% sensitivity and specificity. PLR showed moderate predictive value (AUC=0.631), while NLR had limited discriminative ability (AUC=0.552).

Table 3. Bivariate correlation analysis according to PMI in Hypertensive Patients.

PMI	Coefficient (r)	p-value
PLR	0.518	**<0.001**
NLR	0.070	0.382
Age	-0.120	0.134
Nighttime SBP, mmHg	-0.167	**0.036**
Daytime DBP, mmHg	0.155	**0.052**
LDL-C, mg/dl	0.157	0.089

PMI: Platelet Mass Index; PLR: Platelet-to-Lymphocyte Ratio; NLR: Neutrophil-to-Lymphocyte Ratio; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; LDL-C: Low-Density Lipoprotein Cholesterol.



	p	AUC	Cut-off	Sensitivity	Specificity
PMI	<0.001	0.749	25.69	67.1%	67.1%
PLR	0.004	0.631	117.04	59.5%	60.8%
NLR	0.258	0.552	2.05	51.9%	58.2%

PMI: Platelet Mass Index, NLR: Neutrophil to lymphocyte ratio, PLR: Platelet to lymphocyte ratio

Figure 1. ROC analysis depicting sensitivity and specificity of ~~of~~ the Platelet Mass Index (PMI), Neutrophil-to-Lymphocyte Ratio (NLR), and Platelet-to-Lymphocyte Ratio (PLR) for predicting a Morning Blood Pressure Surge (MBPS) exceeding 28.2 in hypertensive patients.

DISCUSSION

In this study, we investigated the association between PMI and MBPS in newly diagnosed, treatment-naïve hypertensive patients. Our research revealed that individuals with an overactive MBPS system showed substantial increases in PMI levels, indicating that the cumulative platelet count could be a key factor in the development of irregular circadian blood pressure fluctuations. These results contribute to the expanding body of evidence linking platelet-related inflammatory processes to cardiovascular risk phenotypes, especially those associated with BP variability.

PMI, calculated as the product of platelet count and MPV, serves as a composite index integrating both platelet number and size, thereby reflecting total platelet mass and activity. Unlike MPV or platelet count alone, PMI provides a broader perspective on platelet-driven pro-inflammatory and prothrombotic potential.[13] Prior research has demonstrated that platelet activation is intricately involved in systemic inflammation, vascular endothelial dysfunction, and early atherosclerosis.[14,15] Platelets release a variety of bioactive molecules, such as platelet factor 4, thromboxane A2, and CD40 ligand, which actively modulate leukocyte recruitment, cytokine release, and endothelial permeability. Thus, elevated PMI may serve as a surrogate marker for heightened vascular immune reactivity.[16,17]

Our findings support the hypothesis that platelet activation may be a key contributor to the exaggerated MBPS phenotype. It is well-established that morning hours are associated with increased sympathetic nervous system activity, augmented cortisol release, and heightened vascular tone, all of which can predispose to surges in blood pressure.[2] Kario and colleagues have previously shown that MBPS represents not just a hemodynamic variation, but also a pathophysiological state that may precipitate cardiovascular events, particularly in the elderly and in those with preexisting target organ damage.[18] The dynamic interaction between the circadian autonomic balance and vascular inflammation appears to be central to this phenomenon.

Several studies have proposed that MBPS is not merely a reflection of BP rhythm, but rather a manifestation of vascular vulnerability. Shimizu et al. observed that hypertensive patients with elevated MBPS and concurrent high-sensitivity C-reactive protein (hsCRP) levels had significantly increased rates of silent cerebral infarctions and clinical stroke events.[4] Similarly, Saylik et al. demonstrated that systemic immune-inflammation index (SII) was significantly correlated with MBPS and that SII outperformed NLR and PLR in predicting exaggerated morning surges.[19] In this context, our study extends the concept of MBPS as an inflammatory-prone phenotype by introducing PMI as a novel, yet simple, hematologic indicator of platelet-driven vascular stress.

Interestingly, in our study, MPV alone did not significantly differ between MBPS subgroups. This observation emphasizes the value of using combined indices such as PMI to

capture more subtle shifts in platelet kinetics. Indeed, MPV values may remain within normal limits even in the presence of activated platelets, particularly when bone marrow production compensates for peripheral platelet consumption with smaller, immature cells.[14] By multiplying platelet count and MPV, PMI provides a more integrative measure of circulating platelet mass, which may better mirror the cumulative inflammatory and thrombotic potential of the blood compartment.[20,21]

On a cellular level, platelets interact with endothelial cells and leukocytes to propagate inflammation. Activated platelets express P-selectin, enabling adhesion to neutrophils and monocytes.[21] This interaction can induce the formation of platelet-leukocyte aggregates, which serve as mediators of vascular injury. Moreover, platelets release microparticles enriched in inflammatory lipids and cytokines, further amplifying endothelial dysfunction.[14,16,17,21] It is plausible that patients with elevated PMI harbor a microvascular environment primed for inflammatory responses, which may contribute to enhanced early-morning vasoreactivity and exaggerated BP surges.

Our correlation analysis showed a significant inverse relationship between PMI and nighttime systolic blood pressure. This suggests a potential compensatory interplay between nocturnal BP depression and morning platelet activation. It may be speculated that individuals with lower nighttime BP experience relative ischemia in susceptible vascular territories, leading to subtle endothelial activation and platelet priming during the late night and early morning hours.[20] This aligns with prior work indicating that the transition from night to morning is a period of increased oxidative stress, catecholamine release, and vascular tone.[16]

The ability of PMI to distinguish patients with high MBPS was further supported by ROC curve analysis, which demonstrated superior diagnostic performance compared to NLR and PLR. The AUC value for PMI (0.749) suggests moderate to good discriminative capacity, with acceptable sensitivity and specificity. Given that PMI is readily obtainable from routine hemograms, it offers an accessible and cost-effective option for risk stratification in hypertensive patients, particularly in settings where advanced imaging or inflammatory assays are not feasible.

In cardiovascular medicine, there has been growing interest in identifying biomarkers that reflect both inflammation and hemodynamic burden.[19,22] While hsCRP, IL-6, and TNF-α have been studied extensively, they are not always available in daily clinical practice. In contrast, hematological indices such as PMI, SII, and derived ratios (e.g., NLR, PLR) are increasingly being recognized as pragmatic tools to assess vascular inflammatory status.[5-7,19] Our findings suggest that among these, PMI may offer superior specificity for BP-related inflammation, particularly in the early stages of essential HT.

From a clinical standpoint, our data suggest that patients with elevated PMI might benefit from more aggressive monitoring of circadian BP patterns, and possibly from interventions targeting platelet reactivity. This may include lifestyle modifications, antiplatelet agents, or therapies that modulate neurohumoral tone.[23] However, prospective trials are needed to determine whether targeting elevated PMI can improve cardiovascular outcomes or attenuate the impact of MBPS on end-organ damage.

Despite the promising findings, this study has some limitations. First, the cross-sectional nature of the analysis prevents causal inference. Second, we did not evaluate circulating inflammatory cytokines or endothelial markers, which could have strengthened the mechanistic underpinnings of the observed associations. Third, the relatively small sample size and single-center design may limit generalizability, although our population was well-characterized and homogenous. Lastly, PMI was assessed at a single time point; serial measurements could provide insight into its temporal relationship with BP variability. Additionally, several potential confounding factors—such as dietary sodium intake, physical activity levels, sleep quality, and psychosocial stress—were not assessed and may have influenced MBPS or platelet indices. These variables warrant consideration in future prospective studies.

Looking forward, future research should aim to validate our findings in larger, multi-ethnic cohorts with longitudinal follow-up. It would also be valuable to investigate whether integrating PMI with ambulatory BP parameters and imaging-based vascular assessments (e.g., carotid intima-media thickness or pulse wave velocity) enhances risk prediction. In addition, mechanistic studies exploring the role of platelet-derived cytokines and microparticles in the pathogenesis of MBPS would offer deeper insights into the biological plausibility of our observations.

Conclusion

Our study identifies a novel link between PMI and exaggerated MBPS in newly diagnosed hypertensive patients. The findings suggest that PMI may serve as a convenient and meaningful biomarker reflecting the interplay between platelet activity and circadian blood pressure regulation. Given the emerging evidence connecting platelet-driven inflammation with cardiovascular events, PMI deserves further consideration as part of an integrated risk assessment strategy in clinical HT management.

Conflict of Interest

The authors declare that there is not any conflict of interest regarding the publication of this manuscript.

Ethics Committee Permission

This study was approved by the Health Sciences Ethics Committee of Kırşehir Ahi Evran University Faculty of Medicine (date: 07.01.2025, number: 2025-01/01).

Authors' Contributions

Concept/Design: AY, MSA. Data Collection and/or Processing: AY, MSA. Data analysis and interpretation: AY, MSA. Literature Search: AY, MSA. Drafting manuscript: AY, MSA. Critical revision of manuscript: AY, MSA.

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