ORIGINAL ARTICLE

AHI EVRAN MEDICAL JOURNAL
E-ISSN: 2619 - 9203

Evaluation of the Relationship Between Systemic Immune-Inflammatory Index and Morning Blood Pressure Surge in Newly Diagnosed Essential Hypertension Patients

Yeni Tanı Almış Esansiyel Hipertansiyon Hastalarında Sistemik İmmün İnflamatuar İndeksi ve Sabah Kan Basıncı Dalgalanması Arasındaki İlişkinin Değerlendirilmesi

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ÖZ

Amaç: Bu çalışma, yeni tanı konmuş esansiyel hipertansiyon hastalarında Sistemik İmmün-İnflamatuar İndeks (Sİİ) ile Sabah Kan Basıncı Yükselmesi (SKBY) arasındaki ilişkiyi araştırmayı amaçlamaktadır.
Araçlar ve Yöntem: Nisan ve Haziran 2024 tarihleri arasında, kardiyoloji polikliniğinden 217 kontrol ve 188 hipertansif hastadan oluşan 405 katılımcıyı içeren kesitsel bir çalışma yapılmıştır. Katılımcıların kan basıncı, Ayaktan Kan Basıncı İzleme (AKBİ) kullanılarak izlenmiş ve Sİİ dahil inflamatuar belirteçler değerlendirilmiştir. Sİİ, platelet sayısının nötrofil sayısıyla çarpılması ve lenfosit sayısına bölünmesiyle hesaplanmıştır. İstatistiksel analizler, SKBY'yi öngörmede Sİİ'nin değerini değerlendirmek için ROC eğrisi analizini içermektedir.
Bulgular: Hipertansif hastalar, kontrol grubuna kıyasla 24 saatlik, gündüz ve gece sistolik ve diyastolik kan basıncı değerlerinin anlamlı derecede yüksek olduğunu göstermiştir (hepsi $p<0.001$). SKBY ve Sİİ de hipertansif grupta anlamlı derecede yükselmiştir (her ikisi de $p=0.003$). ROC analizi, Sİİ'nin 27.3 mmHg'nin üzerinde SKBY 'yi öngörmede 577.38 kesim değeri ile %56.4 duyarlılık ve %67 özgüllükle 0.645 ($p=0.001$) AUC'ye sahip olduğunu göstermiştir. Yüksek nötrofil ve platelet sayıları, daha yüksek kan basıncı seviyeleri ve kardiyovasküler risk ile ilişkilendirilmiştir.
Sonuç: Çalışma, hipertansif hastalarda Sİİ ve SKBY arasında anlamlı bir ilişki olduğunu belirlemiş, bu da sistemik inflamasyonun kan basıncının düzenlenmesinde ve hipertansiyonun patogenezinde rol oynayabileceğini önermektedir. Sİİ, SKBY ve ilgili kardiyovasküler riskleri öngörmede değerli bir biyomarker olarak kullanılabilir ve daha erken ve daha hedeflenmiş müdahaleleri kolaylaştırabilir.

Anahtar Kelimeler: enflamatuar belirteçler; kan basıncı değişkenliği; kardiyovasküler hastalık; kardiyovasküler risk

ABSTRACT

Purpose: This study aims to explore the relationship between the Systemic Immune-Inflammatory Index (SII) and Morning Blood Pressure Surge (MBPS) in patients with newly diagnosed essential hypertension.
Materials and Methods: A cross-sectional study was conducted between April and June 2024, involving 405 participants, 217 controls, and 188 hypertensive patients, recruited from a cardiology outpatient clinic. Participants' blood pressure was monitored using Ambulatory Blood Pressure Monitoring (ABPM), and inflammatory markers, including SII, were assessed. SII was calculated by multiplying platelet count by neutrophil count and dividing by lymphocyte count. Statistical analysis included ROC curve analysis to evaluate SII's predictive value for MBPS.
Results: Hypertensive patients exhibited significantly higher 24-hour, daytime, and nighttime systolic and diastolic blood pressure values compared to controls (all $p<0.001$). MBPS and SII were also significantly elevated in the hypertensive group (both $p=0.003$). ROC analysis demonstrated that SII had an AUC of 0.645 ($p=0.001$) with a sensitivity of 56.4% and specificity of 67% at a cut-off value of 577.38 for predicting MBPS greater than 27.3 mmHg. Elevated neutrophil and platelet counts were associated with higher blood pressure levels and cardiovascular risk.
Conclusion: The study identified a significant association between SII and MBPS in hypertensive patients, suggesting that systemic inflammation may play a role in the regulation of blood pressure and the pathogenesis of hypertension. SII could serve as a valuable biomarker for predicting MBPS and associated cardiovascular risks, facilitating earlier and more targeted interventions.

Keywords: blood pressure variability; cardiovascular disease; cardiovascular risk; inflammatory markers

Received: 30.07.2024; Accepted: 04.11.2024

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How to cite: Ateş MS, Keleş F. Evaluation of the relationship between systemic immune-inflammatory index and morning blood pressure surge in newly diagnosed essential hypertension patients. Ahi Evran Med J. 2025;9(1):24-29. DOI: 10.46332/aemj.1525095

INTRODUCTION

Hypertension remains a pervasive cardiovascular risk factor, significantly contributing to the global burden of heart disease, cerebrovascular accidents, and renal dysfunction.[1] Among the various forms of hypertension, essential hypertension is particularly prevalent, affecting a large segment of the adult population.[2] This condition is characterized by elevated blood pressure levels in the absence of a discernible secondary cause, making its management a primary focus of clinical practice and public health initiatives. The pathophysiology of essential hypertension is multifaceted, involving a complex interplay of genetic, environmental, and lifestyle factors that culminate in sustained hypertension and end-organ damage.[3]

Recently, there has been increasing recognition of the role systemic immune and inflammatory mechanisms play in the etiology and progression of essential hypertension.[4,5] Chronic low-grade inflammation and immune system dysregulation have been implicated in vascular remodeling, endothelial dysfunction, and heightened vascular resistance-all key features of hypertension. The identification of reliable biomarkers that can capture the extent of immune and inflammatory activity is crucial for advancing our understanding of hypertension and improving prognostic assessments.

The Systemic Immune-Inflammatory Index (SII) is an innovative biomarker that encapsulates the systemic inflammatory status by integrating three readily available hematological parameters: platelet count, neutrophil count, and lymphocyte count. This composite index has garnered attention for its prognostic value across a spectrum of diseases, including oncological, infectious, and cardiovascular conditions.[6,7] In the context of hypertension, elevated SII levels may reflect an ongoing inflammatory process that exacerbates vascular dysfunction and contributes to poor clinical outcomes. Understanding the relationship between SII and hypertension could offer novel insights into the disease's pathophysiology and potential therapeutic targets.

Adding to the complexity of hypertension management is the phenomenon of the morning blood pressure surge (MBPS), a transient but significant rise in blood pressure occurring during the early morning hours. This surge has been associated with an increased risk of cardiovascular events; stroke and myocardial infarction, underscoring the need for effective monitoring and intervention strategies.[8,9] The pathogenesis of MBPS is thought to involve a combination of neurohormonal fluctuations, autonomic nervous system activity, and vascular reactivity. Investigating the interplay between SII and MBPS may reveal critical links between inflammation, circadian rhythms, and cardiovascular risk in hypertensive patients.

This study aims to investigate the relationship between SII and MBPS in patients with newly diagnosed essential hypertension. By examining these associations, we hope to delineate the potential role of systemic inflammation in the initial stages of hypertension and its impact on circadian blood pressure patterns.

MATERIALS and METHODS

Study Design and Participants

This cross-sectional study was conducted between June and July 2024, involving patients who were newly diagnosed with essential hypertension. Participants were recruited from the cardiology outpatient clinic. Inclusion criteria encompassed adults aged 18-75 years with newly diagnosed essential hypertension, while exclusion criteria involved patients with secondary hypertension, malignancies, hyperthyroidism, chronic renal disease, rheumatologic conditions, inflammatory diseases, active infections, those on antiplatelet or antibiotic therapy, and those on antihypertensive medication. A power analysis using G*Power (version 3.1.9.4) with an effect size of 0.50 determined a required sample size of 88 participants per group, totaling 176 participants. This study was approved by the Scientific Ethics Committee of Kırşehir Ahi Evran University Faculty of Medicine, Health Sciences (dated 11.06.2024 and numbered 2024-12/98).

Ambulatory Blood Pressure Monitoring (ABPM)

ABPM was utilized to obtain accurate blood pressure measurements over a 24-hour period. An appropriately sized cuff was placed on the non-dominant arm of each

participant. ABPM devices were calibrated every six months to ensure accuracy and reliability of the measurements. Blood pressure was measured every half hour at night and every 15 minutes during the day. Participants were instructed to adhere to their usual daily routines and remain still during measurements. The recorded data was transferred to a computer for analysis.

Measurement of Blood Pressure and Calculation of MBPS

The data collected encompassed systolic blood pressure (SBP) and diastolic blood pressure (DBP) values over 24 hours, as well as during daytime and nighttime periods. Hypertension was classified based on a 24-hour average SBP exceeding 130 mmHg and/or DBP exceeding 80 mmHg. Additionally, an average daytime SBP over 135 mmHg and/or DBP over 85 mmHg, and an average nighttime SBP above 120 mmHg and/or DBP above 70 mmHg were also indicative of hypertension.[10] MBPS was determined by subtracting the mean SBP one hour before waking from the mean SBP two hours after waking.[11]

Inflammatory Markers

Blood samples were obtained the following morning after an overnight fast to assess inflammatory markers. SII was calculated by multiplying the platelet count by the neutrophil count and then dividing this product by the lymphocyte count.[12] Other markers were also assessed, including the platelet-lymphocyte ratio (PLR) and neutrophil-lymphocyte ratio (NLR).

Statistical Analysis

The statistical analyses were performed using IBM SPSS version 29.0 for Windows (Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess the normality of continuous variables. Descriptive statistics were presented as median (25th-75th percentiles) and frequencies (n %). In the study, the comparisons between two groups for continuous quantitative variables, where the assumption of normality was not met, were conducted using the Mann-Whitney U test. Categorical variables were analyzed using chi-square. A receiver operating characteristic (ROC) curve analysis was conducted, with particular emphasis on establishing a diagnostic threshold for a MBPS greater than 27.3 mmHg.

RESULTS

A total of 405 participants were enrolled in the study, comprising 217 individuals in the control group and 188 in the hypertensive group. Age differences between the control and hypertensive groups were not statistically significant (p=0.099). The gender distribution was similar across both groups, with males representing 46.1% of the control group and 50% of the hypertensive group (p=0.431).

Lipid profiles, hematological parameters, and lymphocyte counts showed no significant differences between the control and hypertensive groups, though neutrophil counts were marginally higher in the hypertensive group (Table 1).

Blood pressure measurements indicated significantly higher values in the hypertensive group across all periods, including 24-hour, daytime, and nighttime SBP and DBP (all p<0.001). The MBPS and the SII were also significantly elevated in the hypertensive group (p=0.003 for both). Additionally, the PLR and NLR were higher in the hypertensive group (p=0.034 and p=0.023, respectively). For comprehensive data, refer to Table 1.

Hypertensive patients were divided into two groups based on the median MBPS value of 27.3, with the median serving as the cutoff point. Age and gender distribution were similar between hypertensive patients with MBPS below and above 27.3 mmHg. However, platelet counts were significantly higher in the group with MBPS above 27.3 mmHg (p=0.002). Neutrophil counts were significantly elevated in the group with higher MBPS (p<0.001).

The SII was notably higher in hypertensive patients with MBPS above 27.3 mmHg compared to those with MBPS below this threshold (p=0.001). In contrast, the PLR and NLR did not show significant differences between these two groups, with p-values of 0.147 and 0.216, respectively.

ROC curve analysis evaluated the predictive value of SII, NLR, and PLR for MBPS above 27.3 mmHg in hypertensive patients. The SII had an AUC of 0.645 (p=0.001), a

cut-off of 577.38, 56.4% sensitivity, and 67% specificity. PLR showed an AUC of 0.561 (p=0.147), with a cut-off of 115.39, 55.3% sensitivity, and 59.6% specificity. NLR had an AUC of 0.552 (p=0.217), a cut-off of 2.05, 51.1% sensitivity, and 61.7% specificity.

Table 1. Comparison of demographic and laboratory characteristics of Controls and Hyperten-sive groups.

Variable	Control Group (n=217)	Hypertensive (n=188)	p-value
Age, year	54 (45.5-64)	57 (47-65)	p=0.099
Gender (male), n (%)	100 (46.1)	94 (50)	p=0.431
LDL, mg/dl	110 (89-134)	116.5 (93-142)	p=0.215
HDL, mg/dl	46 (39-56)	46 (39-55)	p=0.940
Triglyceride, mg/dl	142 (96-214)	160 (104.5-233)	p=0.098
Hgb, g/dl	14.3 (13.2-15.5)	14.3 (13.1-15.6)	p=0.778
White Blood Cell, 103/µl	7.60 (6.53-8.99)	7.74 (6.45-9.69)	p=0.194
Platelet, 103/µl	264 (225-302)	270 (225-304)	p=0.595
Neutrophil, 103/µl	4.46 (3.65-5.19)	4.84 (3.75-5.90)	p=0.055
Lymphocyte, 103/µl	2.43 (1.97-2.88)	2.34 (1.86-2.84)	p=0.296
Glucose, mg/dl	101 (91-110)	104 (90-142)	p=0.145
Creatinine, mg/dl	0.80 (0.67-0.91)	0.82 (0.69-0.98)	p=0.073
24-h SBP, mmHg	111 (106-116)	130 (123-137)	p<0.001
24-h DBP, mmHg	63 (59-68)	76.5 (70.25-82)	p<0.001
Daytime SBP, mmHg	113 (108-120)	132 (124-140)	p<0.001
Daytime DBP, mmHg	66 (61-71)	78 (71-85)	p<0.001
Nighttime SBP, mmHg	104 (99-112)	126 (120-134)	p<0.001
Nighttime DBP, mmHg	59 (54-63.5)	73 (67-79)	p<0.001
MBPS, mmHg	25 (16.46-32.35)	27.30 (21.1-36.45)	p=0.003
SII	494.5 (379.5-593.5)	545.7 (381.5-697.9)	p=0.003
PLR	110.9 (87.7-130.4)	113.5 (89.6-144.4)	p=0.034
NLR	1.85 (1.48-2.23)	1.96 (1.51-2.61)	p=0.023

Values are n (%),median (25th and 75th percentiles). 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, SII: Systemic immune-inflammation index.

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

Variables	MBPS<27.30 (n=94)	MBPS>27.30 (n=94)	p-value
Age, year	57.5 (47-66)	57 (45.75-65)	p=0.775
Gender (male), n (%)	48 (51.1)	46 (48.9)	p=0.770
Platelet, 103/µl	254 (212-286)	275.5 (234.5-343.2)	p=0.002
Neutrophil, 103/µl	4.25 (3.59-5.15)	5.22 (4.02-6.86)	p<0.001
Lymphocyte, 103/µl	2.31 (1.74-2.70)	2.37 (2.01-3.02)	p=0.082
SII	508.9 (350.5-610.8)	614.54 (403.6-888.2)	p=0.001
PLR	110.9 (89.9-137.2)	118.4(89.4-148.3)	p=0.147
NLR	1.96 (1.58-2.35)	2.07 (1.50-2.91)	p=0.216

Values are n (%), median (25th and 75th percentiles). MBPS: Morning Blood Pressure Surge, NLR: Neutrophil to lymphocyte ratio, PLR: Platelet to lymphocyte ratio, SII: Systemic immune-inflammation index.

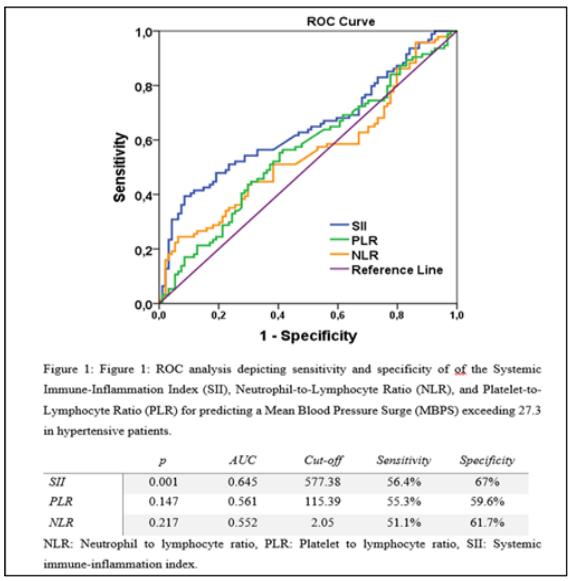

Figure 1: Figure 1: ROC analysis depicting sensitivity and specificity of of the Systemic Immune-Inflammation Index (SII), Neutrophil-to-Lymphocyte Ratio (NLR), and Platelet-to-Lymphocyte Ratio (PLR) for predicting a Mean Blood Pressure Surge (MBPS) exceeding 27.3 in hypertensive patients.

	p	AUC	Cut-off	Sensitivity	Specificity
SII	0.001	0.645	577.38	56.4%	67%
PLR	0.147	0.561	115.39	55.3%	59.6%
NLR	0.217	0.552	2.05	51.1%	61.7%

NLR: Neutrophil to lymphocyte ratio, PLR: Platelet to lymphocyte ratio, SII: Systemic immune-inflammation index.

Figure 1. ROC analysis depicting sensitivity and specificity of the Systemic Immune-Inflammation Index (SII), Neutrophil-to-Lym phocyte Ratio (NLR) and Platelet-to Lymphocyte Ratio (PLR) for predicting a mean Blood Pressure Surge (MBPS) exceed-ing 27.3 in hypertensive patients.

DISCUSSION

In this study, we observed that hypertensive patients exhibited significantly higher 24-hour systolic and diastolic blood pressure values compared to controls, alongside an elevated MBPS. Our findings suggest a significant association between the SII and MBPS, indicating that higher SII values are linked with greater MBPS. These results highlight the potential role of systemic inflammation in the regulation of blood pressure and the pathogenesis of hypertension.

Earlier research has shown a connection between inflammation and elevated blood pressure. For instance, Saylik et al. reported that higher SII levels were independently associated with exaggerated morning surge in newly diagnosed, treatment-naive hypertensive patients.[13] Our study

corroborates these findings, showing a significant correlation between SII and MBPS, and further extends the understanding of how systemic inflammation might contribute to blood pressure variability.

In our study, the significant differences in neutrophil and platelet counts observed between the hypertensive and control groups support the association between systemic inflammation and BP variability. Elevated neutrophil counts have been implicated in the pathogenesis of hypertension through mechanisms involving oxidative stress and endothelial dysfunction.[14] Similarly, increased platelet counts, and activity have been associated with higher blood pressure levels and cardiovascular risk.[15] These findings underscore the importance of inflammatory pathways in the development and progression of hypertension.

Besides SII, we assessed other inflammatory markers like NLR and PLR. Both NLR and PLR were significantly elevated in hypertensive patients compared to controls, consistent with previous research identifying these ratios as predictors of hypertension and cardiovascular events.[16,17] The inclusion of these markers alongside SII provides a comprehensive assessment of the inflammatory status in hypertensive patients and their potential impact on blood pressure regulation.

Given the strong association between SII and MBPS, SII could serve as a valuable biomarker for identifying hypertensive patients at higher risk for cardiovascular events. The ability to predict MBPS through a simple blood test could facilitate earlier intervention and more personalized treatment strategies, improving patient outcomes. Furthermore, targeting systemic inflammation through lifestyle modifications and pharmacological interventions may offer a novel approach to managing hypertension and reducing cardiovascular risk.

Despite the strengths of our study, including a large sample size and the use of ABPM for accurate blood pressure measurement, there are limitations that must be acknowledged. The exclusion of patients on antihypertensive treatment limits the generalizability of our findings to all hypertensive patients. Additionally, the reliance on self-reported waking and sleeping times for calculating MBPS may introduce variability. Future studies should incorporate objective measures such as actigraphy to validate these periods and provide more accurate assessments.

Another limitation is the cross-sectional design of our study, which precludes conclusions about causality between systemic inflammation and MBPS. Longitudinal studies are required to establish the temporal relationship between these factors and to determine whether anti-inflammatory interventions can effectively reduce MBPS and related cardiovascular events. Moreover, our study did not evaluate other potential contributors to blood pressure variability, such as genetic factors, stress, and lifestyle habits, including diet and physical activity, which should be considered in future research.

The clinical importance of our study lies in the identification of SII as a potential biomarker for predicting exaggerated MBPS. Given that MBPS has been associated with increased cardiovascular events, our findings suggest that SII could be used as a simple, cost-effective marker to identify high-risk hypertensive patients. This could facilitate earlier and more targeted interventions to mitigate cardiovascular risk. Moreover, the use of SII in clinical practice could enhance risk stratification and personalized treatment approaches for hypertensive patients.

Conclusion

Our study demonstrates that hypertensive patients have significantly higher SII and MBPS compared to controls. The strong association between SII and MBPS highlights the potential role of systemic inflammation in the pathogenesis of hypertension and suggests that SII could serve as a valuable biomarker for predicting MBPS and associated cardiovascular risks. These findings underscore the importance of integrating inflammatory markers into the management of hypertension to improve patient outcomes and reduce the burden of cardiovascular disease.

Conflict of Interest

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

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Ethics Committee Permission

This study was approved by the Scientific Ethics Committee of Kırşehir Ahi Evran University Faculty of Medicine, Health Sciences (dated 11.06.2024 and numbered 2024-12/98).

Authors' Contributions

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

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