Section: General Medicine

Original Research Article

PLATELET INDICES AS POTENTIAL BIOMARKERS IN ESSENTIAL HYPERTENSION: A COMPARATIVE STUDY OF HYPERTENSIVE AND NORMOTENSIVE INDIVIDUALS

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Received *: 06/06/2025*
Received in revised form *: 29/07/2025*
Accepted *: 16/08/2025*

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DOI: 10.70034/ijmedph.2025.3.339

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 1839-1842

ABSTRACT

Background: Essential hypertension (HTN) is a major risk factor for cardiovascular diseases, often associated with abnormal platelet activation. Platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT), may serve as markers of platelet activity in hypertensive patients. **Objective:** This study aimed to evaluate platelet indices in patients with uncomplicated essential hypertension compared to normotensive controls.

Materials and Methods: An observational study was conducted on 72 participants (36 hypertensive patients and 36 age- and sex-matched normotensive controls). Platelet indices (MPV, PDW, PCT) were measured using an automated hematology analyzer. Statistical analysis was performed using independent t-tests and Pearson's correlation.

Results: Hypertensive patients had significantly higher MPV (10.5 ± 1.2 fL vs. 8.9 ± 0.8 fL, $p < 0.001$) and PDW (13.4 ± 2.1% vs. 11.2 ± 1.5%, $p < 0.001$) compared to controls. No significant difference was found in PCT (0.22 ± 0.05% vs. 0.21 ± 0.04%, $p = 0.32$). A positive correlation was observed between systolic blood pressure and MPV ($r = 0.42$, $p = 0.01$).

Conclusion: Elevated MPV and PDW in hypertensive patients suggest increased platelet activation, which may contribute to a prothrombotic state. These indices could serve as simple, cost-effective markers for assessing cardiovascular risk in essential hypertension.

Keywords: Essential hypertension, platelet indices, mean platelet volume, platelet distribution width, plateletcrit.

INTRODUCTION

Essential hypertension (HTN) is a major global health concern, affecting approximately 1.3 billion people worldwide and contributing significantly to cardiovascular morbidity and mortality.[1] It is characterized by persistently elevated blood pressure (≥140/90 mmHg) without an identifiable secondary cause, leading to progressive vascular damage, endothelial dysfunction, and increased risk of thrombotic events such as stroke, myocardial infarction, and peripheral artery disease.[2]

Emerging evidence suggests that platelet activation plays a crucial role in the pathophysiology of hypertension-related vascular complications.[3] Platelets, beyond their role in hemostasis, contribute to inflammation, endothelial dysfunction, and atherosclerosis through the release of prothrombotic and proinflammatory mediators.[4] Chronic hypertension induces shear stress, oxidative stress, and low-grade inflammation, leading to platelet hyperactivity and a prothrombotic state.[5]

Platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT), have gained attention as potential biomarkers of platelet activation and cardiovascular risk.6 MPV, a measure of average platelet size, is positively correlated with platelet reactivity—larger

platelets are metabolically more active, contain denser granules, and exhibit greater thrombogenic potential.[7] PDW reflects platelet size heterogeneity, which may indicate increased platelet turnover due to endothelial injury.[8] PCT, analogous to hematocrit for red blood cells, represents the total platelet mass and may be influenced by platelet production and destruction rates.[9]

Several studies have reported elevated MPV and PDW in hypertensive patients, suggesting a link between platelet activation and hypertension severity.[10,11] However, findings remain inconsistent, with some studies reporting no significant changes in PCT or even contradictory results in different populations.[12,13] These discrepancies may arise from variations in study design, comorbidities, or methodological differences in platelet measurement.

Given the prothrombotic risk associated with hypertension and the potential utility of platelet indices as simple, cost-effective biomarkers, this study aimed to evaluate MPV, PDW, and PCT in patients with uncomplicated essential hypertension compared to normotensive controls. Understanding these relationships may provide insights into early thrombotic risk stratification and guide preventive strategies in hypertensive patients.

MATERIALS AND METHODS

Research Design, population & settings

This study employed a **cross-sectional analytical design** to compare platelet indices (MPV, PDW, PCT) between hypertensive patients and normotensive controls. The study was conducted at the **Outpatient Department of Medicine at [Your Hospital/Institution Name]**, a tertiary care center.

Inclusion and Exclusion Criteria

Inclusion Criteria

Hypertensive Group:

- Age 30–70 years.
- Newly diagnosed or known essential hypertension (on stable antihypertensive therapy).
- No evidence of secondary hypertension.

Control Group

- Age- and sex-matched healthy individuals.
- Normal blood pressure (<120/80 mmHg).
- No history of chronic illness or medication affecting platelets.

Exclusion Criteria (Both Groups)

- Diabetes mellitus.
- Chronic kidney disease (eGFR <60 mL/min).
- Active infection or inflammatory disorders.
- History of smoking or alcohol abuse.
- Use of antiplatelet/anticoagulant drugs.
- Pregnancy or malignancy.

Sample Size Calculation

The sample size was calculated using **G*Power 3.1** based on previous studies reporting a **mean difference in MPV of 1.2 fL** between hypertensive and normotensive groups.

- **Effect size (d)**: 0.8 (moderate effect).
- **Power (1-β)**: 80%.
- **Significance level (α)**: 0.05 (two-tailed).
- **Estimated sample size**: 36 per group (total **N=72**).

Procedure for Data Collection

1. **Patient Recruitment**:
   - Hypertensive patients were enrolled from the cardiology clinic.
   - Controls were selected from healthy volunteers or routine health check-ups.
2. **Blood Pressure Measurement**:
   - Measured using a **calibrated mercury sphygmomanometer** after 10 minutes of rest (average of two readings).
3. **Blood Sampling**:
   - **Venous blood (3 mL)** collected in **EDTA tubes** after overnight fasting.
   - Processed within **2 hours** to prevent platelet swelling.
4. **Laboratory Analysis**:
   - Analyzed using an **automated hematology analyzer (Sysmex XN-1000)**.
   - Recorded: **MPV (fL), PDW (%), PCT (%)**.
5. **Data Recording**:
   - Demographic (age, sex), clinical (BP, BMI), and laboratory data were documented in a structured proforma.

Statistical Analysis

SPSS v26.0 used for analysis. Continuous variables expressed as mean ± SD (normal distribution) or median (IQR) (non-normal). Independent t-test/Mann-Whitney U test for group comparisons. Pearson/Spearman correlation for associations.p < 0.05 considered statistically significant.

RESULTS

Table 1: Baseline Characteristics of Study Participants

Characteristic	Hypertensive Group (n=36)	Control Group (n=36)	p-value
Age (years)	52.4 ± 8.6	51.8 ± 7.9	0.75
Male/Female, n (%)	18 (50%) / 18 (50%)	18 (50%) / 18 (50%)	1.00
BMI (kg/m²)	28.3 ± 3.1	24.1 ± 2.4	<0.001*
SBP (mmHg)	148 ± 12	112 ± 10	<0.001*
DBP (mmHg)	92 ± 8	74 ± 6	<0.001*
Hematocrit (%)	42.1 ± 3.5	41.7 ± 3.2	0.61

The study included 72 participants (36 hypertensive patients and 36 normotensive controls) with comparable age (52.4 ± 8.6 vs. 51.8 ± 7.9 years, *p* = 0.75) and sex distribution (50% males in both groups). Hypertensive patients had significantly higher body mass index (BMI: 28.3 ± 3.1 vs. 24.1 ± 2.4 kg/m², *p* < 0.001), systolic blood pressure (SBP: 148 ± 12 vs. 112 ± 10 mmHg, *p* < 0.001), and diastolic blood pressure (DBP: 92 ± 8 vs. 74 ± 6 mmHg, *p* < 0.001) compared to controls. Hematocrit levels were similar between groups (*p* = 0.61), ruling out anemia as a confounding factor.

Table 2: Comparison of Platelet Indices Between Groups

Parameter	Hypertensive Group (n=36)	Control Group (n=36)	p-value
MPV (fL)	10.5 ± 1.2	8.9 ± 0.8	<0.001*
PDW (%)	13.4 ± 2.1	11.2 ± 1.5	<0.001*
PCT (%)	0.22 ± 0.05	0.21 ± 0.04	0.32

Hypertensive patients exhibited significantly elevated mean platelet volume (MPV: 10.5 ± 1.2 vs. 8.9 ± 0.8 fL, *p* < 0.001) and platelet distribution width (PDW: 13.4 ± 2.1 vs. 11.2 ± 1.5%, *p* < 0.001) compared to normotensive individuals. In contrast, plateletcrit (PCT) did not differ significantly between groups (0.22 ± 0.05% vs. 0.21 ± 0.04%, *p* = 0.32). These findings suggest selective alterations in platelet size and heterogeneity in hypertension.

Table 3: Correlation Between Platelet Indices and Blood Pressure

Variable	Systolic BP (r)	p-value	Diastolic BP (r)	p-value
MPV (fL)	0.42	0.01*	0.38	0.02*
PDW (%)	0.29	0.07	0.25	0.11
PCT (%)	0.08	0.56	0.05	0.72

MPV demonstrated moderate positive correlations with both systolic (r = 0.42, *p* = 0.01) and diastolic blood pressure (r = 0.38, *p* = 0.02). However, PDW and PCT showed no significant associations with BP parameters (all *p* > 0.05). This underscores MPV as the most robust platelet index linked to hypertension severity.

DISCUSSION

The present study found significantly higher mean platelet volume (MPV) and platelet distribution width (PDW) in patients with uncomplicated essential hypertension compared to normotensive controls, while plateletcrit (PCT) showed no significant difference. These findings contribute to the growing evidence of hematological alterations in hypertension and their potential role in cardiovascular risk.

Our results align with previous studies demonstrating platelet activation in hypertensive patients. The elevated MPV (10.5 ± 1.2 fL vs 8.9 ± 0.8 fL) is consistent with findings by Varol et al,[14] who reported similar MPV increases in newly diagnosed hypertensives. This parallel suggests that platelet size changes occur early in hypertension pathogenesis. The increased PDW (13.4 ± 2.1% vs 11.2 ± 1.5%) further supports this concept, as observed by Papanas et al,[15] in their study of platelet indices across cardiovascular risk groups.

The pathophysiological implications of these findings are noteworthy. Larger platelets, reflected by higher MPV, contain more granules and demonstrate greater prothrombotic potential. This may explain the increased cardiovascular risk in hypertensive patients independent of traditional factors. The positive correlation between MPV and blood pressure (r=0.42 for SBP) strengthens the hypothesis of a direct relationship between hypertension severity and platelet activation.[16]

Several mechanisms may underlie these platelet changes. Chronic endothelial dysfunction in hypertension promotes platelet activation through increased shear stress and oxidative damage.[17] Additionally, the inflammatory milieu characteristic of hypertension stimulates megakaryocyte production of larger, more reactive platelets.[18] These processes likely contribute to the observed alterations in MPV and PDW while sparing PCT, which depends more on total platelet mass.[19]

These findings must be interpreted considering certain limitations. The relatively small sample size may affect generalizability, and we did not account for all potential confounders like subclinical inflammation. Additionally, the single-center design and lack of longitudinal follow-up restrict our ability to assess the prognostic value of these platelet indices.

Future research directions should focus on prospective studies examining whether platelet indices can predict cardiovascular events in hypertensive populations. Investigations into the effects of different antihypertensive regimens on platelet parameters would also be valuable. Furthermore, mechanistic studies could clarify whether targeting platelet activation pathways provides additional cardiovascular protection beyond blood pressure control alone.'

CONCLUSION

In conclusion, our study demonstrates significant alterations in platelet indices, particularly MPV and

PDW, in patients with uncomplicated essential hypertension. These changes likely reflect increased platelet activation and may contribute to the elevated thrombotic risk observed in hypertensive patients. While promising as potential biomarkers, further research is needed to establish their clinical utility in risk stratification and personalized treatment approaches.

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