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Research Article

Correlation between Mean Platelet Volume, Body Mass Index, and Waist-Hip Ratio in Young Adults: A Cross-Sectional Study

Short title: Mean platelet volume and waist-hip ratio in young adults

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Abstract

Introduction: Central adiposity promotes inflammation that may increase the production of larger, more reactive platelets. This study aimed to assess whether mean platelet volume (MPV) correlates more closely with waist-hip ratio (WHR) than with body mass index (BMI) in apparently healthy young adults.

Methods: This cross-sectional analytical study at a tertiary care teaching hospital in Tamil Nadu, India (October 2025 to March 2026), included 200 apparently healthy adults aged 18–25 years (100 males, 100 females). Anthropometry was standardized, and fasting K_2EDTA samples were analyzed for MPV within 2 hours. Pearson correlations were compared using Steiger's Z test, and MPV was compared across sex-specific WHR strata by independent-samples *t* test.

Results: Mean age was 20.4 ± 1.6 years, BMI 23.1 ± 4.2 kg/m², WHR 0.84 ± 0.08, and MPV 9.8 ± 1.1 fL. MPV correlated strongly with WHR ($r = 0.68$; 95% CI, 0.60–0.75; $P < .001$) and weakly with BMI ($r = 0.18$; 95% CI, 0.04–0.31; $P = .011$); the MPV–WHR correlation was significantly stronger (Steiger's $Z = 7.75$; $P < .001$). MPV was 2.2 fL higher in males with WHR ≥0.95 (11.6 vs 9.4 fL) and in females with WHR ≥0.85 (11.4 vs 9.2 fL) (both $P < .001$). The highest MPV (12.0 fL) occurred in normal-weight participants with central adiposity.

Conclusion: In young adults, MPV is associated far more closely with central adiposity than with BMI. Assessing WHR together with routinely reported MPV may help identify young individuals with adverse fat distribution for early cardiometabolic risk assessment.

Keywords: Mean Platelet Volume; Waist-Hip Ratio; Body Mass Index; Obesity, Abdominal; Intra-Abdominal Fat; Blood Platelets; Young Adult

1. Introduction

Obesity prevalence in adults has risen in almost every country since 1990,[1] and in India abdominal obesity (39.5%) is now more common than generalized obesity (28.6%).[2] Expanded adipose tissue acts as an endocrine organ that

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secretes pro-inflammatory cytokines, sustaining chronic low-grade inflammation, endothelial dysfunction, and a prothrombotic state.[3,4] Platelets are central to this thrombo-inflammatory milieu, and platelet activation is enhanced in obesity.[5] Mean platelet volume (MPV), the average size of circulating platelets reported with every complete blood count, reflects platelet production and reactivity: larger platelets are younger, contain and release more granular content, express more glycoprotein VI, and aggregate and form thrombi more readily.[6,7] Elevated MPV has been associated with myocardial infarction, adverse cardiovascular events, and mortality.[8,9]

Body mass index (BMI), the conventional measure of obesity, does not distinguish fat from lean mass or show how fat is distributed, and it can misclassify health risk in individuals.[10] Waist-based measures are practical surrogates of visceral adiposity and provide risk information beyond BMI.[11] Central adiposity at a normal BMI is already associated with excess cardiovascular risk in young adults,[12] and abdominal obesity and hyperinsulinemia are common even among normal-weight Indian adolescents and young adults.[13] However, most studies linking MPV with adiposity have examined older adults or patients with metabolic disease and have relied mainly on BMI-defined obesity.[14-16] Whether MPV follows fat distribution rather than overall body mass in healthy young adults therefore remains unclear.

This study aimed to assess the correlation of MPV with waist-hip ratio (WHR) and with BMI in apparently healthy young adults aged 18–25 years, to compare the strength of these two correlations, and to examine MPV according to sex-specific WHR thresholds.

2. Materials and Methods

2.1 Study design and setting

This cross-sectional analytical study was conducted in the Department of Pathology of a tertiary care teaching hospital in Tamil Nadu, India, from October 2025 to March 2026.

2.2 Participants

Two hundred apparently healthy young adults (100 males and 100 females) aged 18–25 years were recruited from individuals attending routine health check-ups. Individuals were excluded if they had hypertension, diabetes mellitus, cardiovascular disease, an active infection, or a bleeding or coagulation disorder; were current smokers or consumed alcohol; or were taking antiplatelet agents (including aspirin), anticoagulants, oral contraceptives, or other drugs known to affect hematopoiesis.

2.3 Anthropometric measurements

Weight and height were measured using a calibrated digital weighing scale and a stadiometer, respectively. BMI was calculated as weight in kilograms divided by the square of height in meters and was classified according to both the World Health Organization (WHO) international classification and the Asia-Pacific classification.[17] Waist circumference was measured midway between the lower rib margin and the anterior superior iliac crest at the end of normal expiration, and hip circumference at the maximum circumference of the buttocks. WHR was calculated as waist circumference divided by hip circumference, and abdominal obesity was defined by the WHO cut-offs of WHR ≥0.90 in males and ≥0.85 in females.[18]

2.4 Blood sampling and MPV measurement

After an overnight fast, 2 mL of venous blood was collected into K_2EDTA tubes between 08:00 and 10:00 hours. To limit time-dependent, EDTA-induced platelet swelling, all samples were analyzed within 2 hours of collection on an automated hematology analyzer (Sysmex Corporation, Kobe, Japan) that sizes platelets by electrical impedance. The analyzer was calibrated according to the manufacturer's instructions, and internal quality control was performed daily before samples were run.

2.5 Statistical analysis

Data were entered in Microsoft Excel and analyzed using IBM SPSS Statistics, version 25.0 (IBM Corp, Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD) with range, and categorical variables as number (percentage). Pearson

correlation coefficients (r) were calculated for MPV with WHR, MPV with BMI, and BMI with WHR. The 95% confidence intervals (CIs) were derived by Fisher's z-transformation, and r^2 was reported as the proportion of variance explained. Correlation strength was graded as weak ($|r|$ 0.10–0.29), moderate (0.30–0.49), or strong (≥0.50).

Because the MPV–WHR and MPV–BMI correlations share MPV and were estimated in the same participants, they were compared with Steiger's Z test for dependent correlations, which accounts for the correlation between BMI and WHR;[19] the 95% CI for the difference between the two coefficients was also calculated. For the sex-stratified analysis, the WHR levels at which pronounced MPV elevation was observed were identified from the sex-specific data (≥0.95 in males and ≥0.85 in females). MPV was compared between participants below and at or above these thresholds with the independent-samples t test, and the mean difference with its 95% CI was reported. Seven participants with contrasting combinations of BMI and WHR were profiled individually, and their MPV was expressed in SDs from the cohort mean. A two-sided $P < .05$ was considered statistically significant.

2.6 Ethical considerations

The study was approved by the Institutional Human Ethics Committee (approval number withheld for anonymous peer review; provided on the title page) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment.

3. Results

3.1 Characteristics of the study population

A total of 200 apparently healthy young adults were analyzed, comprising 100 males (50.0%) and 100 females (50.0%). Participants were aged 18–23 years, with a mean age of 20.4 ± 1.6 years. BMI ranged from 16.1 kg/m² (underweight) to 37.4 kg/m² (WHO class II obesity), with a mean of 23.1 ± 4.2 kg/m². Mean waist and hip circumferences were 74.6 ± 10.8 cm and 88.3 ± 9.5 cm, respectively, giving a mean WHR of 0.84 ± 0.08 (range 0.68–1.15). MPV averaged 9.8 ± 1.1 fL and ranged from 8.4 to 12.0 fL (Table 1).

Table 1. Baseline demographic, anthropometric, and hematological characteristics of the study population (N = 200)

Parameter	Minimum	Maximum	Mean ± SD / n (%)
Demographics			
Male, n (%)	–	–	100 (50.0)
Female, n (%)	–	–	100 (50.0)
Age (years)	18	23	20.4 ± 1.6
Anthropometry			
Height (cm)	143	184	164.2 ± 9.1
Weight (kg)	40	96	62.8 ± 12.4
Body mass index (kg/m²)	16.1[a]	37.4[b]	23.1 ± 4.2
Waist circumference (cm)	32	102	74.6 ± 10.8
Hip circumference (cm)	38	113	88.3 ± 9.5
Waist-hip ratio	0.68	1.15	0.84 ± 0.08
Hematology			
Mean platelet volume (fL)	8.4	12.0	9.8 ± 1.1

Continuous variables are presented as mean ± standard deviation (SD) with observed range; categorical variables as n (%). [a]Underweight (<18.5 kg/m²). [b]WHO obesity class II (35.0–39.9 kg/m²).

3.2 Correlation of MPV with central and generalized adiposity

MPV showed a strong, positive, and statistically significant correlation with WHR ($r = 0.68$; 95% CI, 0.60–0.75; $P < .001$), with WHR accounting for approximately 46% of the variance in MPV ($r^2 = 0.46$). The correlation between MPV and BMI was weak, although statistically significant ($r = 0.18$; 95% CI, 0.04–0.31; $P = .011$), with BMI explaining only about 3% of the variance in MPV ($r^2 = 0.03$) (Table 2; Figure 1). BMI and WHR were moderately correlated with each other ($r = 0.40$; 95% CI, 0.28–0.51; $P < .001$).

The 95% CIs of the MPV–WHR and MPV–BMI coefficients did not overlap, and WHR explained approximately 14 times as much of the variance in MPV as BMI did. On formal comparison of the two dependent correlations, the MPV–WHR correlation was significantly stronger than the MPV–BMI correlation (difference in r, 0.50; 95% CI, 0.37–0.63; Steiger's Z = 7.75; $P < .001$).

MPV rose progressively with increasing WHR and was highest among participants with WHR between 0.95 and 1.15, in whom mean MPV was ≥11.7 fL. Conversely, participants with BMI >30 kg/m² but proportionate fat distribution retained MPV values within 9.1–9.5 fL.

Table 2. Pearson correlation of mean platelet volume with waist-hip ratio and body mass index (N = 200)

Variable pair	n	*r*	95% CI	r^2 (variance explained)	Strength	*P* value
MPV vs WHR	200	0.68	0.60 to 0.75	0.46 (46.2%)	Strong	<.001
MPV vs BMI	200	0.18	0.04 to 0.31	0.03 (3.2%)	Weak	.011
BMI vs WHR	200	0.40	0.28 to 0.51	0.16 (16.0%)	Moderate	<.001

BMI, body mass index; CI, confidence interval; MPV, mean platelet volume; r, Pearson correlation coefficient; r^2, coefficient of determination; WHR, waist-hip ratio. 95% CIs were derived by Fisher's z-transformation. Strength of correlation: $|r|$ 0.10–0.29 weak, 0.30–0.49 moderate, ≥0.50 strong. The MPV–WHR and MPV–BMI correlations differed significantly (Steiger's Z test for dependent correlations sharing one variable: Z = 7.75, $P < .001$).

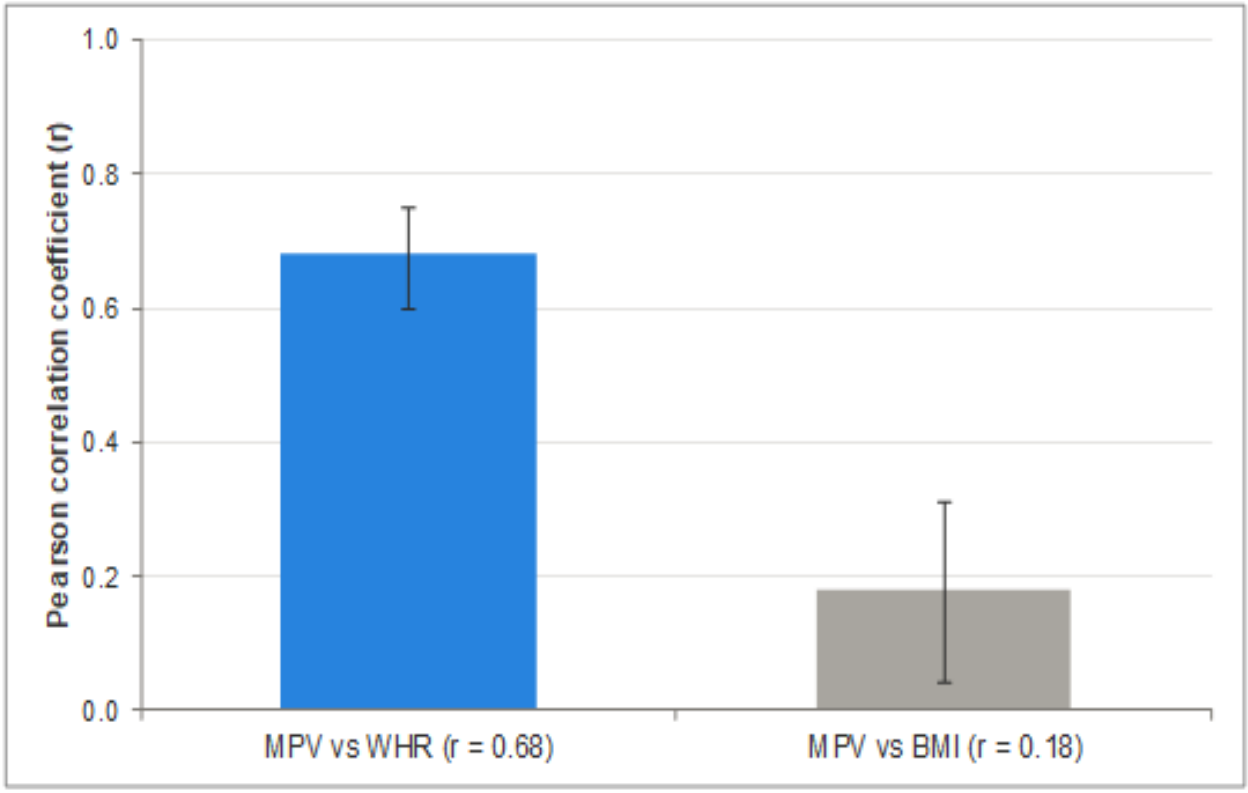


Figure 1. Strength of correlation of mean platelet volume (MPV) with waist-hip ratio (WHR) and body mass index (BMI) in the study population (N = 200). Bars show Pearson correlation coefficients; error bars show 95% confidence intervals (Fisher's z-transformation).

3.3 Discordance between generalized and central adiposity: individual profiles

Seven individually profiled participants illustrated the dissociation between BMI and WHR in relation to platelet size (Table 3; Figures 2 and 3).

Table 3. Anthropometric classification and mean platelet volume of individually profiled participants (n = 7)

Subject (sex, age)	BMI, kg/m²	BMI class: WHO / Asia-Pacific[a]	WHR	WHO cut-off[b]	Study threshold[c]	MPV, fL	MPV, SD from mean[d]
Group A: normal BMI with central adiposity							
042 (M, 19 y)	24.3	Normal / Overweight	0.95	≥ cut-off	≥ threshold	12.0	+2.00
118 (F, 19 y)	20.3	Normal / Normal	0.95	≥ cut-off	≥ threshold	12.0	+2.00
Group B: high BMI with discordant central adiposity							
002 (M, 19 y)	37.4	Obese II / Obese II	0.92	≥ cut-off	< threshold	9.9	+0.09
011 (M, 18 y)	29.6	Overweight / Obese I	0.92	≥ cut-off	< threshold	9.1	−0.64
157 (F, 19 y)	31.2	Obese I / Obese II	0.85	≥ cut-off	≥ threshold	11.9	+1.91
Group C: proportionate fat distribution							
104 (F, 19 y)	24.6	Normal / Overweight	0.82	< cut-off	< threshold	8.4	−1.27
112 (F, 21 y)	18.0	Underweight (both)	0.80	< cut-off	< threshold	9.0	−0.73

F, female; M, male; BMI, body mass index; MPV, mean platelet volume; WHR, waist-hip ratio. [a]WHO: underweight <18.5, normal 18.5–24.9, overweight 25.0–29.9, obese I 30.0–34.9, obese II 35.0–39.9 kg/m²; Asia-Pacific: underweight <18.5, normal 18.5–22.9, overweight 23.0–24.9, obese I 25.0–29.9, obese II ≥30.0 kg/m². [b]WHO abdominal obesity: ≥0.90 in males, ≥0.85 in females. [c]Study-derived threshold for pronounced MPV elevation: ≥0.95 in males, ≥0.85 in females. [d](MPV − 9.8)/1.1, using the cohort mean and SD.

Group A: normal BMI with central adiposity. The highest MPV in the cohort (12.0 fL; 2.0 SD above the cohort mean) was observed in two participants of normal BMI with elevated WHR: a 19-year-old male (Subject 042; BMI 24.3 kg/m², WHR 0.95) and a 19-year-old female (Subject 118; BMI 20.3 kg/m², WHR 0.95).

Group B: high BMI with discordant central adiposity. Despite class II obesity, a 19-year-old male (Subject 002; BMI 37.4 kg/m²) whose WHR of 0.92 lay below the study-derived male threshold of 0.95 had an MPV of 9.9 fL, close to the cohort mean. Similarly, an overweight 18-year-old male (Subject 011; BMI 29.6 kg/m², WHR 0.92) had an MPV of 9.1 fL. In contrast, a 19-year-old female with class I obesity whose WHR reached the female threshold (Subject 157; BMI 31.2 kg/m², WHR 0.85) had an MPV of 11.9 fL.

Group C: proportionate fat distribution. Two females with WHR below 0.85 (Subject 104: BMI 24.6 kg/m², WHR 0.82; Subject 112: BMI 18.0 kg/m², WHR 0.80) had MPV values of 8.4 fL (the cohort minimum) and 9.0 fL, respectively.

Across the seven profiles, the three participants whose WHR was at or above the sex-specific threshold had MPV values of 11.9–12.0 fL (mean 12.0 fL), whereas the four below the threshold had MPV values of 8.4–9.9 fL (mean 9.1 fL). BMI overlapped widely between the two sets (20.3–31.2 vs 18.0–37.4 kg/m²).

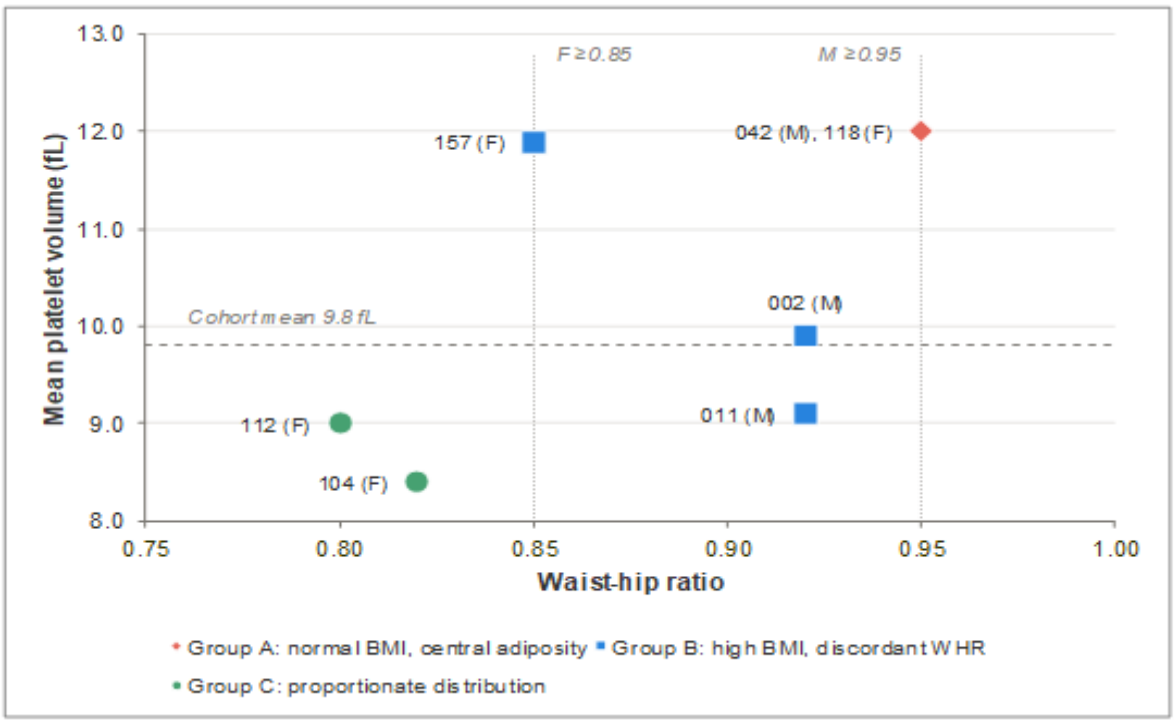


Figure 2. Mean platelet volume plotted against waist-hip ratio for the seven individually profiled participants (labels give subject number and sex). Dashed line, cohort mean MPV (9.8 fL); dotted lines, study-derived WHR thresholds for females (0.85) and males (0.95). Subjects 042 and 118 share identical values.

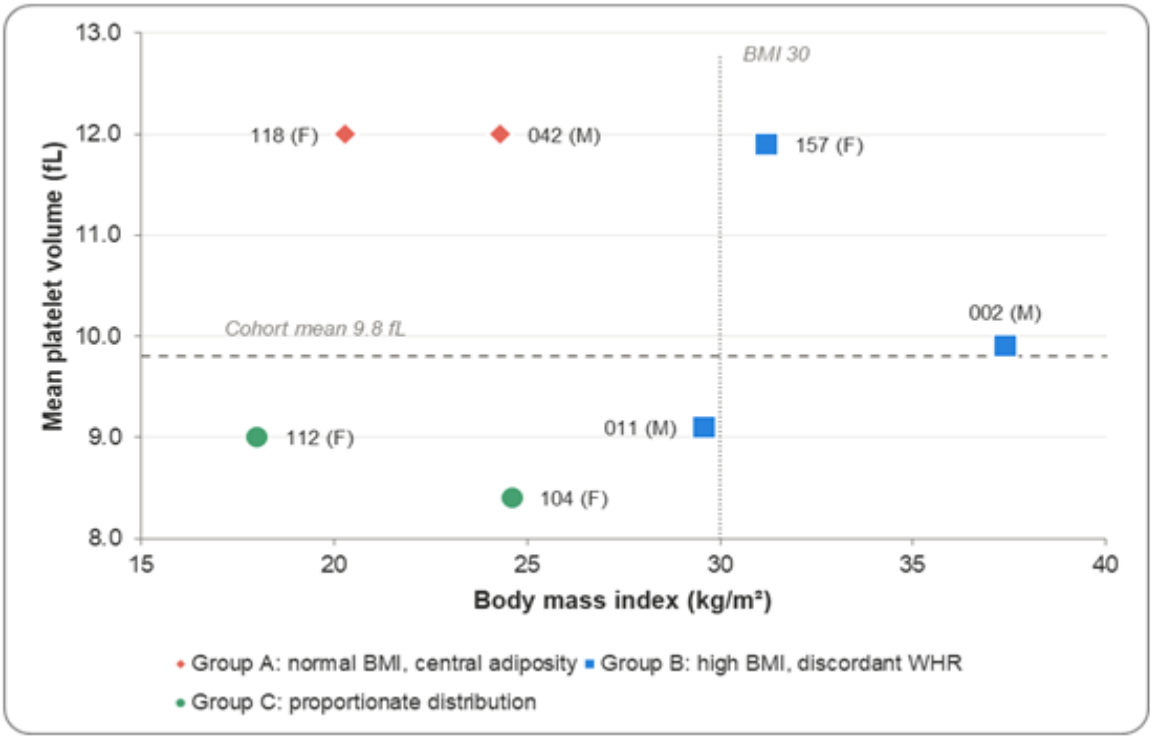


Figure 3. Mean platelet volume plotted against body mass index for the same seven participants. Dashed line, cohort mean MPV (9.8 fL); dotted line, WHO obesity cut-off (30 kg/m²). The highest MPV values occurred at normal BMI (Subjects 042 and 118), whereas the participant with the highest BMI (Subject 002) had an MPV close to the cohort mean.

3.4 Sex-specific WHR thresholds for MPV elevation

On sex-stratified analysis, pronounced elevation of MPV was observed at WHR ≥0.95 in males and ≥0.85 in females (Table 4; Figure 4). The female threshold coincides with the WHO cut-off for abdominal obesity (≥0.85), whereas the male threshold lies above the corresponding WHO cut-off (≥0.90). Twenty-two males (22.0%) and 28 females (28.0%) had a WHR at or above their sex-specific threshold.

In males, mean MPV was 11.6 ± 0.6 fL at WHR ≥0.95 compared with 9.4 ± 0.9 fL below this threshold (mean difference, 2.2 fL; 95% CI, 1.8–2.6; $P < .001$). In females, mean MPV was 11.4 ± 0.7 fL at WHR ≥0.85 compared with 9.2 ± 0.8 fL below it (mean difference, 2.2 fL; 95% CI, 1.9–2.5; $P < .001$). The mean difference was the same in both sexes, although the threshold WHR was lower in females.

Table 4. Mean platelet volume according to sex-specific waist-hip ratio thresholds

Sex	WHR stratum	n (%)	MPV, fL (mean ± SD)	Mean difference, fL (95% CI)	*P* value
Male (n = 100)	<0.95	78 (78.0)	9.4 ± 0.9	Reference	–
	≥0.95	22 (22.0)	11.6 ± 0.6	2.2 (1.8 to 2.6)	<.001
Female (n = 100)	<0.85	72 (72.0)	9.2 ± 0.8	Reference	–
	≥0.85	28 (28.0)	11.4 ± 0.7	2.2 (1.9 to 2.5)	<.001

CI, confidence interval; MPV, mean platelet volume; SD, standard deviation; WHR, waist-hip ratio. Thresholds are the study-derived values for pronounced MPV elevation. Mean differences are for the upper vs the lower WHR stratum within each sex, compared by independent-samples *t* test (males $t = 10.8$, females $t = 12.8$; df = 98).

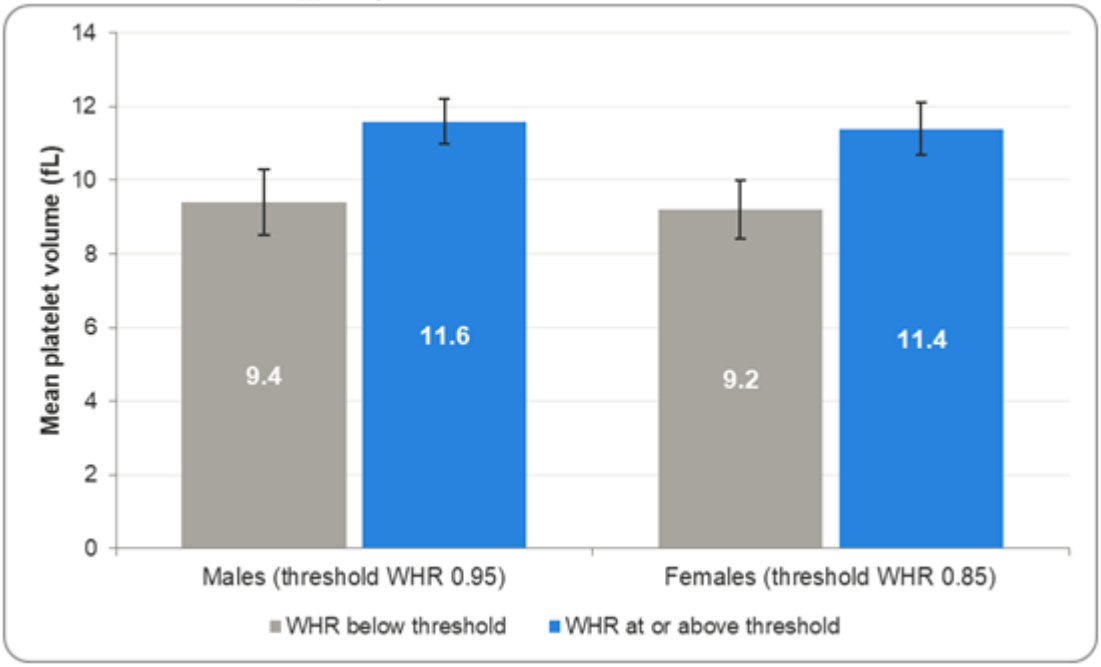


Figure 4. Mean platelet volume below and at or above the sex-specific waist-hip ratio thresholds. Bars show means; error bars show ±1 SD. In both sexes, MPV was 2.2 fL higher in the upper WHR stratum ($P < .001$). Males: n = 78 vs 22; females: n = 72 vs 28.

4. Discussion

In this cross-sectional study of apparently healthy young adults, MPV correlated strongly with WHR but only weakly with BMI. WHR accounted for 46% of the variance in MPV, compared with 3% for BMI, and the difference between the two correlations was statistically significant. By conventional benchmarks, a

correlation of 0.68 represents a large effect.[20] MPV was about 2.2 fL higher above the sex-specific WHR thresholds in both sexes. The highest values occurred in normal-weight participants with central adiposity, whereas participants with obesity but a WHR below the male threshold had MPV values close to the cohort mean. Together, these findings indicate that in this age group platelet size is related to the distribution of body fat rather than to overall body mass.

These results are consistent with epidemiological evidence that fat distribution carries prognostic information that BMI does not. In the UK Biobank, WHR had the strongest and most consistent association with all-cause mortality of the adiposity measures studied, irrespective of BMI.[21] In pooled cohorts of more than 200 000 individuals followed for a median of 20 years, 18% of normal-weight individuals had a high WHR, and a high waist measure was associated with a 15% to 50% greater risk of most cardiovascular outcomes among those of normal weight or overweight.[22] Normal-weight central obesity has likewise been associated with a higher risk of heart disease and stroke,[23] and with myocardial infarction in young adults without traditional risk factors.[12] This discordance may be particularly relevant in Indian populations, in whom greater visceral fat and insulin resistance are present at a BMI similar to that of Caucasians.[24] In our profiled participants, the highest MPV values occurred at a BMI of 20.3 and 24.3 kg/m², which illustrates the same pattern at the level of platelet indices.

Studies of MPV and adiposity have yielded inconsistent results. MPV was higher in adults with obesity than in non-obese controls (10.3 vs 9.0 fL),[14] and body fat was an independent determinant of MPV in an elderly population.[15] In patients with metabolic syndrome and abdominal obesity, however, MPV correlated only weakly with both waist circumference and BMI ($r = 0.14$ for each).[16] Other studies found no difference in MPV by metabolic syndrome status among individuals with obesity,[25] an inverse association between metabolic syndrome and MPV in women only,[26] and, in a meta-analysis of 170 000 patients, lower rather than higher MPV in metabolic syndrome.[27] In a large Thai population, the independent correlates of MPV included female sex, diabetes, metabolic syndrome, triglycerides, and hypertension.[28] This heterogeneity probably reflects differences in age, comorbidity, treatment, analyzer, and sample handling, as well as the use of BMI-defined obesity. In our young cohort, which was free of most of these confounders, the weak correlation with BMI alongside the strong correlation with WHR suggests that BMI-based classification can dilute an underlying association with central fat.

A biological explanation is plausible. Visceral fat drains into the portal circulation, and in obese individuals portal vein interleukin-6 (IL-6) concentrations are about 50% higher than arterial concentrations and correlate with systemic C-reactive protein.[29] Visceral and ectopic fat, rather than total fat mass, are the adipose depots most closely linked to atherosclerosis and cardiometabolic disease.[30] IL-6 stimulates hepatic thrombopoietin production and thrombopoiesis,[31] and inflammatory signals alter megakaryopoiesis and platelet production more broadly.[32] Tumor necrosis factor-α–driven inflammation has also been shown to reprogram megakaryocytes and generate hyperreactive platelets.[33] An adiposity-related inflammatory profile, with higher leptin and IL-6 and lower adiponectin, is already detectable in young adults with overweight or obesity.[34] Because larger platelets are more reactive,[6,7] a pathway from visceral fat through cytokines to megakaryocytes could explain why MPV followed WHR more closely than BMI. This pathway was not measured in the present study and remains a hypothesis.

The female threshold for MPV elevation (WHR ≥0.85) coincided with the WHO cut-off for substantially increased metabolic risk, whereas the male threshold (≥0.95) exceeded the WHO cut-off of 0.90.[18] Sex differences in MPV and in its relationship with metabolic risk have been

described,[26,28] and because these thresholds were derived from our own data, they require external validation before clinical use. All MPV values in our cohort (8.4–12.0 fL) were within the reference interval reported for healthy Indian adults (8.1–13.9 fL).[35] The association therefore describes a shift within the reference range rather than overt abnormality, and MPV should be interpreted as a continuous risk correlate rather than as a diagnostic test. Nevertheless, MPV is reported with every complete blood count, and WHR requires only a measuring tape. Reading the two together could help identify young adults with an adverse fat distribution, including those with a normal BMI, for lifestyle counseling and cardiometabolic follow-up. MPV also appears to be modifiable: short-term tirzepatide treatment lowered MPV by about 0.9 fL in patients with obesity.[36]

This study has several strengths: a homogeneous young cohort with an equal sex distribution, exclusion of conditions and drugs known to affect platelet indices, standardized fasting morning sampling with analysis within 2 hours, and formal comparison of dependent correlations. It also has limitations. The cross-sectional design precludes causal inference, and a single-center sample may not represent other populations. Visceral fat was inferred from WHR rather than measured by imaging, and inflammatory markers, thrombopoietin, and platelet function were not assessed. MPV in K_2EDTA increases with time after venipuncture,[37] and values differ between analyzers and measuring principles,[38] so absolute MPV values and thresholds may not transfer directly to other laboratories. The analyses were unadjusted, and potential confounders such as physical activity, diet, and platelet count were not modeled. The sex-specific thresholds were identified in the same data set and may overestimate the true difference, and the individual profiles are illustrative. Longitudinal studies should combine imaging-based measurement of visceral fat, inflammatory markers, and multivariable models, and should validate WHR thresholds in independent cohorts.

5. Conclusion

In apparently healthy young adults, MPV correlated strongly with WHR and only weakly with BMI, and the MPV–WHR correlation was significantly stronger. MPV was about 2.2 fL higher in males with a WHR ≥0.95 and in females with a WHR ≥0.85. The highest MPV values occurred in participants with a normal BMI but central adiposity, whereas a high BMI with a lower WHR was not accompanied by elevated MPV. Body fat distribution, rather than overall body mass, therefore appears to be the anthropometric correlate of platelet size in young adults. Measuring WHR alongside routinely reported MPV may support early cardiometabolic risk assessment in primary care, pending validation in longitudinal studies.

Declarations

Ethical approval: Approved by the Institutional Human Ethics Committee (IHEC), Government Sivagangai Medical College Hospital, Tamil Nadu, India. The study was conducted in accordance with the Declaration of Helsinki.

Informed consent: Written informed consent was obtained from all individual participants before enrolment.

Conflict of interest: The authors declare that they have no financial or non-financial competing interests regarding the publication of this manuscript.

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Author contributions (CRediT taxonomy)

- **Vijayalakshmi A:** Conceptualization, Methodology, Data collection, Formal analysis, Writing - Original Draft Preparation.
- **N. Sharmila Thilagavathy:** Resources, Supervision, Protocol validation, Critical revisions for intellectual content.

- **Vijayalakshmi M:** Data curation, Writing - Review & Editing, Laboratory analysis, Investigation.

Use of Artificial Intelligence: A generative artificial intelligence tool (Notion AI) was used to assist with drafting and language editing of the manuscript and with retrieving and formatting references. The authors reviewed, edited, and verified all content, data, and references and take full responsibility for the manuscript.

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