ORIGINAL ARTICLE

Visceral Adiposity Index and Its Correlation with Sagittal Abdominal Diameter in Metabolic Syndrome

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Abstract

Introduction: Metabolic syndrome comprises a group of cardiometabolic risk factors and is highly prevalent worldwide. Visceral adiposity is a key element in the pathogenesis of metabolic syndrome.

Objective: To examine the relationship between the visceral adiposity index (VAI) and sagittal abdominal diameter (SAD) in individuals diagnosed with metabolic syndrome.

Materials and methods: A total of 150 participants aged 18 years or older who met the diagnostic criteria for metabolic syndrome were enrolled in the study. VAI and SAD measurements were obtained and analyzed in relation to various metabolic parameters.

Results: The findings indicated a weak positive association between SAD and VAI ($r = 0.1922$, $p = 0.01846$). In contrast, VAI demonstrated moderate to strong positive correlations with total cholesterol ($Rs = 0.5623$, $p < 0.001$), fasting blood glucose ($Rs = 0.5375$, $p < 0.001$), and systolic blood pressure ($Rs = 0.8666$, $p < 0.001$). On the other hand, SAD exhibited only weak correlations with these metabolic indicators.

Conclusion: VAI appears to be a more inclusive marker of visceral adiposity and metabolic risk than SAD alone. These findings emphasize the clinical value of utilizing VAI for evaluating metabolic risk, particularly in patients with metabolic syndrome.

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Introduction

Metabolic syndrome, defined by a combination of physiological and biochemical risk factors, markedly elevates the risk of developing cardiovascular disease, type 2 diabetes, and various other health complications.[1] It is estimated that 25–30% of adults worldwide have metabolic syndrome, with a prevalence of approximately 25% in urban India.[2] The incidence rises with age, especially after 40, and varies by gender, being more common in men than in premenopausal women.[3] Notably, developed countries face higher rates of metabolic syndrome due to obesity and sedentary lifestyles.[4] While insulin resistance is a key factor, the association between abdominal obesity and metabolic syndrome is particularly pronounced.[5] Elevated waist circumference (WC) and triglyceride levels serve as critical markers, though comprehensive risk assessment also requires consideration of traditional risk factors such as hypertension and dyslipidemia.[6] Numerous studies suggest that intra-abdominal visceral fat is more strongly associated with metabolic abnormalities than subcutaneous fat.[7] Indices that combine both anthropometric and biochemical factors, such as the visceral adiposity index (VAI), have demonstrated potential in predicting metabolic syndrome.[8] This study seeks to explore the relationship between VAI and sagittal abdominal diameter (SAD), aiming to deepen our understanding of visceral adiposity's role and contribute to more effective diagnostic and treatment approaches. This study was designed to measure several anthropometric parameters, including sagittal abdominal diameter, in individuals with metabolic syndrome. Our goals were to calculate and evaluate the correlation between the VAI and metabolic syndrome, and to examine the relationship between the VAI and sagittal abdominal diameter.

Materials and Methods

This observational study was conducted from December 2022 to December 2023 at the Outpatient Medicine Department of Government Medical College, Kota. It included 150 participants aged 18 years or older who met the ATP III criteria for metabolic syndrome. Individuals under 18, those who refused to provide consent, and patients with spinal deformities, abdominal tumors, or significant ascites were excluded from the study. The study carefully measured a range of anthropometric and clinical parameters, including height, weight, body mass index (BMI), WC, SAD, VAI, blood pressure, and laboratory values for high-density lipoprotein (HDL), triglycerides (TG), and fasting blood sugar (FBS), employing standardized methods. Metabolic syndrome was diagnosed if three or more of the ATP III criteria were present. SAD was measured to the nearest 0.1 cm after a normal exhalation, in a supine position with straight legs on a firm examination table. The measurement was taken without clothing over the measurement area at the level of the iliac crest (L4–5) using a locally made abdominal caliper. SAD represents the vertical distance between the examination table and the horizontal limb of the caliper. WC was measured according to WHO guidelines, standing after a normal exhalation, at the midpoint between the lower rib margins. Gender-specific cut-off values for WC (≥102 cm in males and ≥88 cm in females) and HDL–C (<40 mg/dL in males and < 50 mg/dL in females) were applied as per ATP III criteria.

The VAI was calculated using BMI (kg/m^2), WC (cm), triglycerides (TG; mg/dL), and high-density lipoprotein-cholesterol (HDL-C; mg/dL) values for both males and females, as follows:

- For males: VAI = {WC/[39.68 + (1.88 x BMI)]} x {TG/[1.03 x (1.31/HDL-C)]}
- For females: VAI = {WC/[36.58 + (1.89 x BMI)]} x {TG/[0.81 x (1.52/HDL-C)]}.[9]

Statistical analysis was performed after consultation with a statistician to ensure appropriateness of tests and accuracy of interpretation.

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Observation and Results

Study Population and Descriptive Statistics

In this study, the mean age of participants was 53.8 years, with a standard deviation of 14.69 years, a median of 55 years, and a range from 21 to 87 years. The first quartile (Q1) was 43 years, and the third quartile (Q3) was 63.75 years, resulting in an interquartile range (IQR) of 20.75 years. The age distribution was approximately symmetric, with a skewness of −0.03, and the 95% confidence interval for the mean age was between 51.42 and 56.18 years. Weight had a mean of 67.72 kg, with a standard deviation of 15.1 kg, a median of 66 kg, Q1 at 56.1 kg, and Q3 at 80 kg, resulting in an IQR of 23.9. The weight distribution showed a slight right skew (skewness = 0.18), with a 95% confidence interval for the mean weight between 65.28 and 70.17 kg. The mean height was 1.64 m, with a standard deviation of 0.11 m, Q1 at 1.56 m, and Q3 at 1.71 m (IQR = 0.15). The distribution showed a slight right skew (skewness = 0.28), and the 95% confidence interval for mean height ranged from 1.63 to 1.66 m. The mean BMI was 25.97 kg/m^2, with a standard deviation of 4.84 kg/m^2, Q1 at 22.63, and Q3 at 29.68 (IQR = 7.05). The distribution was slightly left-skewed (skewness = −0.19), with a 95% confidence interval for the mean BMI between 25.18 and 26.75. WC had a mean of 96.82 cm, a standard deviation of 12.09 cm, a median of 98 cm, Q1 at 89 cm, Q3 at 104 cm (IQR = 15), and was slightly left-skewed (skewness = −0.41), with a 95% confidence interval ranging from 94.86 to 98.78 cm. Sagittal abdominal diameter had a mean of 29.35 cm, a standard deviation of 5.07 cm, Q1 at 25.63 cm, Q3 at 33 cm (IQR = 7.38), and a slight right skew (skewness = 0.28). The 95% confidence interval for the mean sagittal abdominal diameter was between 28.54 and 30.18 cm. Triglycerides had a mean of 188.46 mg/dL, with a standard deviation of 58.57 mg/dL, Q1 at 149.75 mg/dL, and Q3 at 224 mg/dL (IQR = 74.25). The distribution was right-skewed (skewness = 0.73), with a 95% confidence interval for the mean triglyceride level ranging from 178.98 to 197.94 mg/dL. Serum total cholesterol had a mean of 175.5 mg/dL, with a standard deviation of 47.6 mg/dL, Q1 at 143 mg/dL, Q3 at 192.75 mg/dL (IQR = 49.75), and was right-skewed (skewness = 0.99), with a 95% confidence interval between 167.77 and 183.23 mg/dL. HDL had a mean of 46.72 mg/dL, a standard deviation of 12.65 mg/dL, Q1 at 38 mg/dL, Q3 at 55 mg/dL (IQR = 17), and was approximately symmetric (skewness = −0.02). The 95% confidence interval for the mean HDL level ranged from 44.67 to 48.77 mg/dL. Fasting blood sugar had a mean of 168.7 mg/dL, a standard deviation of 52.96 mg/dL, Q1 at 140.25 mg/dL, Q3 at 168 mg/dL (IQR = 27.75), and a strongly right-skewed distribution (skewness = 2.72). The 95% confidence interval for the mean fasting blood sugar level was between 160.11 and 177.32 mg/dL. Systolic blood pressure had a mean of 140.7 mm Hg, with a standard deviation of 8.45 mm Hg, Q1 at 134 mm Hg, Q3 at 146 mm Hg (IQR = 12), and a slight right skew (skewness = 0.31). The 95% confidence interval for the mean systolic blood pressure was between 139.39 and 142.13 mm Hg. Diastolic blood pressure had a mean of 82.3 mm Hg, with a standard deviation of 6.59 mm Hg, Q1 at 80 mm Hg, Q3 at 86 mm Hg (IQR = 6), was approximately symmetric (skewness = 0.16), and the 95% confidence interval for the mean diastolic blood pressure ranged from 81.23 to 83.37 mm Hg. The visceral adiposity index (VAI) had a mean of 3.51 (SD = 1.76), Q1 at 2.49, Q3 at 4.11 (IQR = 1.62), and was strongly right-skewed (skewness = 2.34). The 95% confidence interval for the mean VAI ranged from 3.23 to 3.8. The above descriptive statistics summarized in Table 1.

Demographic Characteristic of Study Population

The mean age was 53.8 years, with a standard deviation of 14.69 years. Table 2 presents the distribution of cases across three age groups. Individuals under the age of 40 accounted for 27 cases, which is 18% of the total. The 40–60 age group had the highest number of cases, with 74 cases, representing 49.3% of the total. Those aged 60 or older accounted for 49 cases, or 32.7% of the total. The chi-squared value of 22.12 and the *p*-value of 0.00001 indicate that the differences in case distribution across these age groups are statistically significant. This table also shows that 53% of the samples were male and 47% were female (among 150 individuals). A chi-square test yielded a nonsignificant *p*-value of 0.794, indicating that there is no statistically significant difference in the distribution of males and females in the sample. This suggests that the proportions of males and females are approximately equal. Additionally, the table shows that 29% were diabetic (43 individuals) and 71% were nondiabetic (107 individuals). Statistical analysis revealed no correlation ($R = 0.0$, $p = 1.0$) between diabetes status (diabetic or nondiabetic). Therefore, the presence or absence of diabetes does not appear to influence the variable under investigation in this sample.

Correlation Coefficient of Different Parameters of Study Population

Table 3 presents the correlation coefficients (*Rs*) between various metabolic and

Table 1: Descriptive data of study population

Variable	*Mean (± SD)*	*Median*	*Range*	*Skewness*	*95% CI*
Age (years)	53.8 (14.69)	55	21–87	−0.03	51.42–56.18
Weight (kg)	67.72 (15.1)	66	35–99	0.18	65.28–70.17
Height (m)	1.64 (0.11)	1.63	1.37–1.89	0.28	1.63–1.66
BMI (kg/m^2)	25.97 (4.84)	26.65	14.6–36.4	-0.19	25.18–26.75
WC (cm)	96.82 (12.09)	98	60–119	-0.41	94.86–98.78
SAD (cm)	29.35 (5.07)	29	20–39.6	0.28	28.54–30.18
TG (mg/dL)	188.4 (58.57)	183.5	53–450	0.73	178.98–197.94
T. cholesterol (mg/dL)	175.5 (47.6)	165.5	94–317	0.99	167.77–183.23
HDL-C (mg/dL)	46.72 (12.65)	46	18–70	-0.02	44.67–48.77
FBS (mg/dL)	168.7 (52.96)	152.2	112–492	2.72	160.11–177.32
SBP (mm Hg)	140.7 (8.45)	140	120–170	0.31	139.39–142.13
DBP (mm Hg)	82.3 (6.59)	80	70–100	0.16	81.23–83.37
VAI	3.51 (1.76)	3.32	0.89–14.38	2.34	3.23–3.8

Waist circumference and HDL-C values were interpreted using gender-specific cut-offs.

Table 2: Demographic characteristics of the study population

Variable	*No. of cases*	*Percentage*	*Statistical analysis*
Age			
< 40 years	27	18.0	$\chi2 = 22.12$, $p < 0.001$
40–60 years	74	49.3	
> 60 years	49	32.7	
Sex			
Male	79	53.0	$\chi2 = 0.068$, $p = 0.794$
Female	71	47.0	
Concomitant illness			
Diabetic	43	29.0	R = 0.0, $p = 1.0$
Noidiabetic	107	71.0	

Table 3: Correlation coefficient of different parameters of study population

		Correlation coefficient (Rs)	*p-value*
VAI	T. cholesterol	0.5623	<0.001
VAI	FBS (mg/dL)	0.5375	<0.001
VAI	SBP (mm Hg)	0.8666	<0.001
VAI	SAD (cm)	0.1922	0.018
SAD (cm)	T. cholesterol	0.0827	0.315
SAD (cm)	FBS (mg/dL)	0.139	0.089
SAD (cm)	SBP (mm Hg)	0.1945	0.017
SAD (cm)	WC (cm)	0.087	0.284
SAD (cm)	TG (mg/dL)	0.0508	0.537
SAD (cm)	HDL (mg/dL)	−0.1307	0.111

anthropometric variables, along with their corresponding *p*-values. The analysis focuses on the relationships between VAI, SAD, and a range of biomarkers, including total cholesterol (T. Cholesterol), FBS, systolic blood pressure (SBP), WC, TG, and HDL. VAI demonstrated strong positive correlations ($Rs > 0.5$) with total cholesterol, FBS, and SBP, all of which were statistically significant ($p < 0.001$). In contrast, weaker and less significant correlations are found between SAD and other variables, with only a few reaching significance ($p < 0.05$).

Discussion

Our study includes 150 subjects with metabolic syndrome, with 18% under 40 years, 49.3% between 40 and 60, and 32.7% over 60. The mean age is 53.8 years ($SD = 14.69$), indicating a predominantly middle-aged population. The chi-square result of 22.12 ($p = 0.00001$) shows a highly significant age distribution. The lower percentage in those under 40 may reflect healthier lifestyles and fewer risk factors, but the 18% of younger cases highlights the need for early prevention and intervention. Chen et al.[10] reported that, among participants aged ≤40 years, the prevalence of metabolic syndrome is lower among individuals engaged in nonsedentary occupations. Our findings indicate that the 40–60 age group had the highest proportion of individuals with metabolic syndrome, indicating a greater risk in this age range and highlighting the need for targeted health interventions. The higher incidence in this group may reflect survival bias, in which healthier individuals live longer, or it may suggest that metabolic syndrome leads to severe health consequences earlier. Zhao et al.[11] identify the 40–60 age group as a high-risk period for the development of metabolic syndrome. Critchley et al.[12] noted that survival bias can lead to an underestimation of the true impact of the condition. In our study, nearly one-third of cases are in the over-60 age group, underscoring the need for proactive metabolic health management during midlife to maintain quality of life and mitigate the risk of complications in later years. Mottillo et al.[13] reported that effective management of metabolic syndrome can significantly reduce the risk of serious complications, including cardiovascular disease, which remains a leading cause of morbidity and mortality in this population. Therefore, age emerges as a key determinant in the prevalence of metabolic syndrome, with Ford et al.[14] reporting a clear increase in prevalence with advancing age. In our study, males accounted for 53% and females for 47% of metabolic syndrome cases. The slight predominance of males may reflect factors such as lifestyle choices, genetic predispositions, and higher-risk health behaviors. The chi-square result of 0.0068 ($p = 0.794$) shows no statistically significant difference in the distribution of metabolic syndrome between males and females, indicating no substantial evidence that one sex is more likely to have the condition. Hirode et al.[15] also reported no significant difference in the prevalence of metabolic syndrome between males and females.

In our study, 29% of participants with metabolic syndrome are diabetic, while 71% are nondiabetic, highlighting that metabolic syndrome can occur without diabetes. This underscored that, while diabetes was a key risk factor, metabolic syndrome could also develop independently due to factors such as obesity, hypertension, and dyslipidemia. Grundy et al.[16] also demonstrated that metabolic syndrome can occur without diabetes, despite diabetes being a significant component and risk factor within the syndrome. In our study, the correlation analysis between diabetes status and metabolic syndrome yielded an *R*-value of 0.0 and a *p*-value of 1.0, indicating no statistically significant relationship.

In our study, total cholesterol levels exhibited a mean of 175.5 mg/dL and a median of 165.5 mg/dL, indicating a right-skewed distribution, with a subset of participants presenting elevated cholesterol levels. The Spearman's rank correlation coefficient between VAI and total cholesterol was 0.5623, demonstrating a statistically significant positive correlation ($p < 0.001$). This suggests that higher VAI values, which reflect increased visceral fat and elevated metabolic risk, are positively associated with higher cholesterol levels, a well-established risk factor for cardiovascular diseases. Zhao et al.[17] found that visceral fat accumulation is associated with unfavorable lipid profiles, including elevated cholesterol levels.

Eckel et al.[18] noted that VAI was a valuable predictor of cardiovascular risk factors, including elevated cholesterol. In contrast, the correlation between SAD and total cholesterol was weak ($Rs = 0.0827$), with a *p*-value of 0.315, indicating that the correlation was not statistically significant. Liu et al.[19] suggested that while central obesity is linked to metabolic disturbances, the correlation between SAD and cholesterol can be weak. Bouchard et al.[20] also found that SAD does not always strongly correlate with lipid profiles, suggesting that other factors, such as genetics, physical activity, and lifestyle, may influence cholesterol levels.

In our study, the FBS levels had a mean of 168.7 mg/dL and a median of 152.2 mg/dL,

indicating a right-skewed distribution with some participants having significantly higher levels. The Spearman's rank correlation coefficient between VAI and FBS was 0.5375, suggesting a moderate positive correlation ($p < 0.001$). This indicates that higher VAI values, reflecting greater visceral fat and metabolic risk, are associated with higher FBS levels. Kern et al.[21] found that visceral fat produces proinflammatory cytokines that impair insulin action, contributing to elevated blood sugar. Our findings align with this, suggesting that as visceral fat increases, FBS levels rise, a key marker for diabetes and metabolic health. Després[22] has been reported that greater visceral adiposity is associated with an increased risk of type 2 diabetes and elevated FBS levels. This supports the hypothesis that visceral fat, measured by VAI, was related to elevated fasting blood sugar in individuals with metabolic syndrome.[23] Among the nondiabetic participants, a subset fulfilled criteria for prediabetes based on fasting blood glucose values (100–125 mg/dL); however, these individuals were analyzed within the nondiabetic category, as subgroup-wise analysis was beyond the scope of the present study.

In our study, the Spearman correlation coefficient (Rs) between SAD and FBS levels was 0.139, suggesting a weak positive correlation. This suggests that as SAD increases, FBS levels tend to rise slightly. However, the weak correlation, along with a *p*-value of 0.089, indicates that the observed relationship is not statistically significant. Therefore, no meaningful association can be established between SAD and FBS levels, suggesting that other factors may have a more substantial influence on fasting blood sugar. Bouchard et al.[20] also found a very weak correlation ($\rho = 0.0827$, $p = 0.315$) between SAD and FBS, suggesting that SAD alone may not be a reliable indicator of FBS levels, which aligns with our findings. In the present study, SBP had a mean of 140.7 mm Hg and a median of 140 mm Hg, indicating an approximately symmetrical distribution. A Spearman's correlation analysis revealed a strong positive association between SBP and VAI ($Rs = 0.8666$, $p < 0.001$), indicating statistical significance.

This suggests that higher VAI values, indicating greater visceral fat and metabolic risk, are strongly associated with higher SBP, a critical cardiovascular health marker. Leite et al.[24] also reported that individuals with elevated VAI values exhibited higher blood pressure levels. In contrast, the Spearman correlation between SAD and SBP was 0.1945, indicating a weak positive relationship ($p = 0.017$) and suggesting a meaningful but weak association between central obesity (SAD) and SBP. Ohrvall et al.[25] reported stronger correlations between SAD and SBP, aligning with our findings.

Triglyceride levels in our study had a mean of 188.4 mg/dL and a median of 183 mg/dL. The correlation between SAD and TG levels was very weak ($Rs = 0.0508$, $p = 0.537$), indicating no statistically significant relationship. While Kobayashi et al.[26] reported a significant positive correlation between visceral adiposity and triglyceride levels in Japanese men, our findings indicate that SAD is not strongly associated with triglyceride levels.

High-density lipoprotein values in our study had a mean of 46.72 mg/dL and a median of 46 mg/dL, indicating a symmetrical distribution. The Spearman coefficient between SAD and HDL was −0.1307, indicating a weak negative relationship, suggesting that as SAD increases, HDL decreases, which could negatively impact cardiovascular health. However, a *p*-value of 0.111 indicates that the observed association is not statistically significant. Turcato et al.[27] also reported a negative correlation between SAD and HDL levels, consistent with our findings.

Waist circumference had a mean of 96.82 cm and a median of 98 cm, showing a relatively asymmetrical distribution. The correlation between SAD and WC was weak ($Rs = 0.087$, $p = 0.28495$), likely due to differences in what these measurements capture—SAD reflects visceral fat, while WC includes both visceral and subcutaneous fat. Firouzi et al.[28] found a high correlation between SAD and WC, which may not be replicable in our study due to our smaller sample size and WC's skewed distribution.

Visceral adiposity index in our study showed moderate skewness (mean = 3.51, median = 3.32), while SAD had a symmetrical distribution (mean = 29.35 cm, median = 29 cm). The weak positive correlation observed between SAD and VAI ($Rs = 0.1922$, $p = 0.01846$) indicates that VAI may serve as a more comprehensive marker of visceral adiposity and metabolic risk compared to SAD alone. Ferreira et al.[29] showed that VAI better predicts unhealthy metabolic phenotypes than conventional anthropometric measures. Our study supports VAI as a potentially more valuable tool for assessing metabolic risk, consistent with findings by Du et al.,[30] who reported that individuals with the highest VAI values exhibited the most unfavorable metabolic profiles

Conclusion

The study provided important insights into the associations between the VAI, SAD, and various metabolic parameters in individuals diagnosed with metabolic syndrome. Age distribution analysis revealed that the highest proportions of cases were observed in the 40–60 age group, underscoring a significant association between age and the prevalence of metabolic syndrome.

Sex distribution was nearly balanced, with no significant differences observed between males and females. Correlation analysis demonstrated that VAI is significantly associated with FBS, total cholesterol, and blood pressure levels, indicating that VAI is a robust indicator of metabolic disturbances in this population. Conversely, SAD showed weaker and often nonsignificant correlations with these parameters, except for a weak but statistically significant association with systolic blood pressure. Overall, these findings emphasize the critical role of visceral adiposity, as assessed by VAI, in the evaluation and management of metabolic syndrome, while also highlighting the relatively limited predictive value of SAD in identifying metabolic abnormalities.

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