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Original Article

Relationship Between Visceral Adiposity Index and Early Microvascular Changes in Type 2 Diabetes

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ABSTRACT

Background: Visceral adipose dysfunction may contribute to diabetic microvascular injury through insulin resistance, dyslipidaemia, inflammation, oxidative stress, and endothelial dysfunction. Objective: To determine the association between Visceral Adiposity Index and early retinal or renal microvascular involvement among adults with type 2 diabetes mellitus. Methods: This hospital-based cross-sectional analytical study included 180 adults with type 2 diabetes. Demographic, anthropometric, biochemical, urinary albumin-to-creatinine ratio, and funduscopic data were collected. Visceral Adiposity Index was calculated using sex-specific equations incorporating waist circumference, body mass index, triglycerides, and high-density lipoprotein cholesterol. The primary outcome was the presence of early diabetic retinopathy, microalbuminuria, or both. Group comparisons, correlation analysis, and multivariable logistic regression were performed. Results: Participants had a mean age of 52.4 ± 8.7 years, and 95 (52.8%) were male. Early diabetic retinopathy was present in 43 (23.9%), microalbuminuria in 51 (28.3%), and at least one microvascular outcome in 70 (38.9%). Mean Visceral Adiposity Index was 4.68 ± 1.62 among participants with microvascular involvement and 2.99 ± 1.18 among those without involvement, corresponding to a mean difference of 1.69 (95% CI: 1.25–2.13) and Hedges' g of 1.23. Each one-unit increase in the index was associated with 48% higher adjusted odds of microvascular involvement. Conclusion: Higher Visceral Adiposity Index was associated with early retinal and renal microvascular abnormalities, particularly microalbuminuria. It may complement, but not replace, standard renal and retinal assessment. Keywords: Type 2 diabetes mellitus; Visceral Adiposity Index; diabetic retinopathy; microalbuminuria; diabetic kidney disease; visceral obesity.

INTRODUCTION

Type 2 diabetes mellitus is a major and rapidly expanding public-health challenge that contributes substantially to cardiovascular disease, kidney dysfunction, visual impairment, neuropathy, disability, and premature mortality. A large pooled analysis of population-representative studies demonstrated substantial worldwide increases in diabetes prevalence between 1990 and 2022, with Pakistan among the countries carrying a particularly high disease burden (1). The International Diabetes Federation has similarly projected a continuing increase in the number of adults living with diabetes, particularly in low- and middle-income countries where preventive care and complication screening remain unevenly accessible (2). Evidence from systematic reviews, meta-analyses, and national surveys indicates that diabetes and prediabetes affect a considerable proportion of the adult population in Pakistan, creating

an increasing demand for affordable strategies that can identify patients at elevated risk of complications before irreversible organ damage occurs (3-6).

Microvascular complications result from progressive injury to small blood vessels and commonly affect the retina and kidneys. Diabetic retinopathy may initially manifest as microaneurysms, small retinal haemorrhages, exudates, or mild non-proliferative changes before the development of clinically apparent visual impairment. Systematic reviews have demonstrated a substantial prevalence of diabetic retinopathy among people with type 2 diabetes in Pakistan, while evidence from diabetic centres in Lahore confirms that retinal involvement is also common in the local clinical population (7-9). Diabetic kidney disease may similarly remain clinically silent during its initial stages. Increased urinary albumin excretion can precede a detectable reduction in renal function or an increase in serum creatinine, making urinary albumin-to-creatinine ratio an important marker of early renal microvascular involvement. Pakistani studies have reported associations of microalbuminuria with hyperuricaemia, dyslipidaemia, poor glycaemic control, and other metabolic abnormalities among patients with type 2 diabetes (10,11). Local evidence also indicates that both diabetic retinopathy and microalbuminuria are frequently encountered in routine diabetic practice, supporting the need for timely retinal and renal assessment (12,13).

Although general obesity is an established risk factor for type 2 diabetes, body mass index does not fully characterize body-fat distribution or adipose-tissue dysfunction. Individuals with similar body mass index values may differ substantially in abdominal fat accumulation, lipid metabolism, insulin sensitivity, inflammatory activity, and vascular risk. Visceral adipose tissue is metabolically active and contributes to insulin resistance, oxidative stress, endothelial dysfunction, altered adipokine secretion, and chronic low-grade inflammation. These mechanisms may adversely affect the retinal and glomerular microvasculature and may therefore contribute to the development of early diabetic complications. The Visceral Adiposity Index was developed as a sex-specific surrogate measure of visceral adipose dysfunction using waist circumference, body mass index, serum triglycerides, and high-density lipoprotein cholesterol (14,15). Because these measurements are routinely available in diabetes clinics, VAI may offer a practical assessment of metabolic risk without requiring computed tomography, magnetic resonance imaging, or specialized body-composition equipment. Previous studies have demonstrated relationships between VAI, insulin resistance, metabolic syndrome, and the development of type 2 diabetes, suggesting that the index captures metabolic information not represented by body mass index alone (16-18).

Emerging evidence has extended the potential relevance of visceral adiposity indices to diabetic microvascular complications. Prospective research has identified associations between visceral obesity indices and incident diabetic retinopathy, while analyses of diabetic cohorts have linked higher VAI with nephropathy and diabetic kidney disease (19-22). Studies evaluating urinary albumin excretion have also found that VAI is independently associated with microalbuminuria and reduced renal function (23-25). These observations provide a plausible basis for examining VAI as an associated metabolic marker of early retinal and renal injury. However, most available studies have been conducted outside Pakistan. Differences in body-fat distribution, dietary practices, glycaemic control, access to preventive services, medication use, and health-seeking behaviour may limit the direct applicability of international findings to Pakistani patients. Local evidence assessing the relationship of VAI with both retinal and renal microvascular abnormalities remains limited.

The present study was therefore conducted among adults with type 2 diabetes mellitus in Lahore, Pakistan, to determine whether VAI was associated with early microvascular involvement. The primary objective was to evaluate the relationship between VAI and a composite outcome defined as the presence of early diabetic retinopathy, microalbuminuria, or both. Secondary objectives were to examine the relationships of VAI with early diabetic retinopathy and microalbuminuria separately and to evaluate its correlation with urinary albumin-to-creatinine ratio. It was hypothesized that participants with early

retinal or renal microvascular involvement would have higher VAI values than those without these complications and that VAI would remain associated with the composite outcome after accounting for relevant demographic and clinical covariates.

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted among adult patients with previously diagnosed type 2 diabetes mellitus attending medical and diabetic outpatient departments in Lahore, Pakistan. The hospital-based setting enabled the recruitment of patients undergoing routine diabetes follow-up, clinical examination, and laboratory assessment. Participants were enrolled through non-probability consecutive sampling until the required sample of 180 patients was achieved. All patients presenting during the recruitment process were assessed against the eligibility criteria, and those who fulfilled the criteria and provided written informed consent were included.

Adults of either sex aged 30-70 years who had been diagnosed with type 2 diabetes mellitus for at least one year were eligible to participate. A minimum diabetes duration of one year was used to exclude very recently diagnosed patients in whom established microvascular abnormalities would be less likely to have developed. Patients were excluded if they had type 1 diabetes, pregnancy, an acute systemic illness, active urinary tract infection, severe anaemia, heart failure, chronic liver disease, or known chronic kidney disease attributable to a non-diabetic cause. Patients with advanced kidney disease or urinary albumin excretion above the range defined for microalbuminuria were also excluded because the study focused on early renal involvement. Additional ophthalmic exclusions included previous retinal surgery, known non-diabetic retinal disease, and ocular-media opacity that prevented adequate fundus examination.

Ethical approval was obtained from the relevant institutional ethical review committee before participant recruitment and data collection. The study purpose and procedures were explained to each eligible patient in understandable language, and written informed consent was obtained before enrolment. Participation was voluntary, and patients were informed that they could withdraw without affecting their clinical care. Personal identifiers were kept confidential, and the collected information was used exclusively for research purposes.

Data were recorded using a predesigned proforma. Demographic and clinical information included age, sex, residence, occupation, smoking status, family history of diabetes, duration of diabetes, diabetes treatment, history of hypertension, and use of lipid-lowering medication. Blood pressure was measured using a standard sphygmomanometer after the participant had remained seated and rested for at least five minutes. Repeat measurements were obtained when required, and the average value was recorded for analysis.

Anthropometric measurements were performed by trained staff using standardized procedures. Body weight was measured in kilograms with a calibrated weighing scale while participants wore light clothing and no shoes. Height was measured in metres using a stadiometer. Body mass index was calculated as weight in kilograms divided by height in metres squared and expressed as kg/m^2. Waist circumference was measured in centimetres at the midpoint between the lower margin of the last palpable rib and the upper border of the iliac crest. The measurement was obtained at the end of normal expiration using a non-stretchable measuring tape positioned horizontally without compressing the skin.

Venous blood samples were collected after an overnight fast of 8-12 hours. Fasting blood glucose, glycated haemoglobin, serum triglycerides, high-density lipoprotein cholesterol, serum creatinine, and other routine biochemical parameters were measured in the hospital laboratory using standard laboratory procedures. Triglyceride and HDL cholesterol concentrations reported in mg/dL were converted to mmol/L before calculation of VAI. Triglycerides were converted by multiplying the mg/dL value by 0.01129, while HDL cholesterol was converted by multiplying the mg/dL value by 0.02586.

VAI was calculated separately for male and female participants using the original sex-specific equations (14,15). For male participants, the equation was:

$$VAI = [WC \div \{39.68 + (1.88 \times BMI)\}] \times (TG \div 1.03) \times (1.31 \div HDL)$$

For female participants, the equation was:

$$VAI = [WC \div \{36.58 + (1.89 \times BMI)\}] \times (TG \div 0.81) \times (1.52 \div HDL)$$

In these equations, WC represented waist circumference in centimetres, BMI represented body mass index in kg/m², and TG and HDL represented serum triglyceride and high-density lipoprotein cholesterol concentrations in mmol/L, respectively. VAI was analysed as a continuous exposure variable, with higher values indicating greater visceral adipose dysfunction.

Renal microvascular involvement was assessed using urinary albumin-to-creatinine ratio measured from a spot urine specimen. A UACR of 30-300 mg/g was classified as microalbuminuria and was used to define early renal microvascular involvement. Patients with urinary albumin excretion above this range or established advanced kidney disease were not included in the early renal outcome category. UACR was evaluated both categorically, according to the presence or absence of microalbuminuria, and continuously when examining its relationship with VAI. Albuminuria assessment was selected because increased urinary albumin excretion may identify diabetic kidney involvement before a substantial decline in glomerular filtration or a marked increase in serum creatinine becomes evident (26-29).

Retinal microvascular involvement was assessed by fundus examination through the ophthalmology service. Pupillary dilatation was performed where required to permit adequate visualization of the retina. The fundus was examined for features of diabetic retinopathy, including microaneurysms, small retinal haemorrhages, hard exudates, cotton-wool spots, and mild non-proliferative changes. Participants were classified as having no diabetic retinopathy or early diabetic retinopathy according to the recorded funduscopic findings. Severe or advanced retinal disease was documented separately and was not included in the early-retinopathy category used for the principal analysis. Retinal screening and classification were undertaken in accordance with the clinical importance of detecting diabetic retinopathy before the development of advanced sight-threatening disease (30).

The primary outcome was a composite indicator of early microvascular involvement, defined as the presence of early diabetic retinopathy, microalbuminuria, or both. Participants without either condition were classified as having no early microvascular involvement. Secondary outcomes were the presence of early diabetic retinopathy, the presence of microalbuminuria, and UACR analysed as a continuous renal marker. VAI was the principal independent variable. Age, sex, duration of diabetes, body mass index, HbA1c, hypertension, waist circumference, triglycerides, HDL cholesterol, smoking status, and relevant treatment history were evaluated as clinical or metabolic characteristics. Standardized measurement procedures, calibrated equipment, fasting biochemical sampling, consecutive participant enrolment, and review of completed data forms were used to improve measurement consistency and data quality.

After completion of clinical, anthropometric, ophthalmic, and laboratory assessments, the data were entered into statistical software and checked for missing, implausible, or incorrectly entered values. Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequency and percentage. Distributional characteristics were assessed before inferential analysis. Pearson correlation was used to assess relationships between approximately normally

distributed continuous variables, whereas Spearman rank correlation was used when distributional assumptions were not satisfied. VAI and other continuous characteristics were compared between participants with and without the composite microvascular outcome and between participants with and without each individual outcome using an independent-samples test selected according to the distribution of the data. Categorical variables were compared using the chi-square test.

Binary logistic regression analysis was performed to examine the association between VAI and the composite early microvascular outcome. VAI was entered as a continuous variable so that the estimated odds ratio represented the change in the odds of early microvascular involvement associated with each one-unit increase in VAI. The adjusted model included age, sex, duration of diabetes, body mass index, HbA1c, and hypertension, consistent with their clinical relevance and the reported analytical framework. Associations were summarized using crude and adjusted odds ratios with 95% confidence intervals. All statistical tests were two-sided, and a p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 180 adults with type 2 diabetes mellitus were included. Their ages ranged from 32 to 70 years, with a mean of 52.4 ± 8.7 years. The study population comprised 95 men (52.8%) and 85 women (47.2%), and the mean duration of diabetes was 7.2 ± 4.1 years. The mean HbA1c was 8.3 ± 1.4%, indicating generally suboptimal glycaemic control. Mean body mass index was 28.1 ± 4.2 kg/m², mean waist circumference was 96.7 ± 10.5 cm, and mean VAI was 3.65 ± 1.58.

Early diabetic retinopathy was identified in 43 participants (23.9%), while 51 (28.3%) had microalbuminuria. Twenty-four participants had both conditions. Consequently, 19 participants had diabetic retinopathy without microalbuminuria, 27 had microalbuminuria without diabetic retinopathy, and 70 (38.9%) had at least one early microvascular abnormality. The remaining 110 participants (61.1%) had neither outcome.

Table 1. Demographic, metabolic, and microvascular characteristics of the participants

Variable	Value
Total participants	180
Age, years	52.4 ± 8.7
Male	95 (52.8)
Female	85 (47.2)
Duration of diabetes, years	7.2 ± 4.1
BMI, kg/m²	28.1 ± 4.2
Waist circumference, cm	96.7 ± 10.5
HbA1c, %	8.3 ± 1.4
Triglycerides, mg/dL	184.6 ± 61.3
HDL cholesterol, mg/dL	39.5 ± 8.2
Visceral Adiposity Index	3.65 ± 1.58
Early diabetic retinopathy	43 (23.9)
Microalbuminuria	51 (28.3)
Retinopathy without microalbuminuria	19 (10.6)
Microalbuminuria without retinopathy	27 (15.0)
Both retinopathy and microalbuminuria	24 (13.3)
Any early microvascular involvement	70 (38.9)
No early microvascular involvement	110 (61.1)

Values are presented as mean ± standard deviation or n (%). The mutually exclusive microvascular categories were derived from the reported retinal, renal, and composite outcome totals. BMI: body mass index; HbA1c: glycated haemoglobin; HDL: high-density lipoprotein.

Participants with early microvascular involvement were older and had a longer duration of diabetes than participants without involvement. The mean age difference was 4.1 years (95% CI: 1.49–6.71), while the difference in diabetes duration was 3.2 years (95% CI: 2.00–4.40). The microvascular-involvement group

also had greater body mass index, waist circumference, HbA1c, triglycerides, VAI, and UACR, together with lower HDL cholesterol.

The largest standardized group differences were observed for UACR, VAI, triglycerides, HbA1c, HDL cholesterol, and diabetes duration. Mean VAI was 4.68 ± 1.62 in participants with microvascular involvement compared with 2.99 ± 1.18 in those without involvement. The resulting mean difference was 1.69 units (95% CI: 1.25–2.13), with a Hedges' g of 1.23, indicating a large standardized difference.

Table 2. Comparison of participants according to composite early microvascular involvement

Variable	No microvascular involvement, n=110	Microvascular involvement, n=70	Mean difference (95% CI)	p-value	Hedges' g
Age, years	50.8 ± 8.2	54.9 ± 8.9	4.10 (1.49 to 6.71)	0.002	0.48
Duration of diabetes, years	5.9 ± 3.4	9.1 ± 4.3	3.20 (2.00 to 4.40)	<0.001	0.84
BMI, kg/m²	27.4 ± 3.9	29.2 ± 4.4	1.80 (0.53 to 3.07)	0.006	0.44
Waist circumference, cm	93.8 ± 9.4	101.1 ± 10.6	7.30 (4.23 to 10.37)	<0.001	0.74
HbA1c, %	7.8 ± 1.2	9.0 ± 1.4	1.20 (0.80 to 1.60)	<0.001	0.93
Triglycerides, mg/dL	162.5 ± 48.7	219.3 ± 65.8	56.80 (38.71 to 74.89)	<0.001	1.01
HDL cholesterol, mg/dL	42.1 ± 7.9	35.4 ± 7.1	−6.70 (−8.94 to −4.46)	<0.001	−0.88
Visceral Adiposity Index	2.99 ± 1.18	4.68 ± 1.62	1.69 (1.25 to 2.13)	<0.001	1.23
UACR, mg/g	18.4 ± 8.9	76.2 ± 48.5	57.80 (46.12 to 69.48)	<0.001	1.86

Values are presented as mean ± standard deviation. Mean differences were calculated as the microvascular-involvement group minus the group without involvement. Confidence intervals were estimated from the reported means, standard deviations, and group sizes using unequal-variance standard errors. Hedges' g was calculated using the pooled standard deviation with small-sample correction. BMI: body mass index; HbA1c: glycated haemoglobin; HDL: high-density lipoprotein; UACR: urinary albumin-to-creatinine ratio.

When the retinal and renal outcomes were considered separately, VAI remained higher among participants with each complication. Participants with early diabetic retinopathy had a mean VAI of 4.58 ± 1.61, compared with 3.21 ± 1.34 among those without retinopathy. The mean difference was 1.37 units (95% CI: 0.83–1.91), with a Hedges' g of 0.97. Participants with microalbuminuria had a mean VAI of 4.74 ± 1.66, compared with 3.18 ± 1.29 among those without microalbuminuria. This corresponded to a mean difference of 1.56 units (95% CI: 1.04–2.08) and a Hedges' g of 1.11. The magnitude of the standardized difference was therefore slightly greater for microalbuminuria than for diabetic retinopathy.

Table 3. Visceral Adiposity Index according to retinal and renal microvascular outcomes

Outcome	Outcome absent, mean ± SD	Outcome present, mean ± SD	Mean difference (95% CI)	p-value	Hedges' g
Early diabetic retinopathy	3.21 ± 1.34, n=137	4.58 ± 1.61, n=43	1.37 (0.83 to 1.91)	<0.001	0.97
Microalbuminuria	3.18 ± 1.29, n=129	4.74 ± 1.66, n=51	1.56 (1.04 to 2.08)	<0.001	1.11
Composite microvascular involvement	2.99 ± 1.18, n=110	4.68 ± 1.62, n=70	1.69 (1.25 to 2.13)	<0.001	1.23

Mean differences were calculated as outcome present minus outcome absent. Confidence intervals and p-values were derived from the reported group means, standard deviations, and sample sizes using unequal-variance independent-group comparisons. Hedges' g was calculated with small-sample correction.

VAI was positively correlated with UACR, indicating that greater visceral adipose dysfunction was associated with greater urinary albumin excretion ($p<0.05$). In the multivariable logistic regression model, VAI remained associated with the composite microvascular outcome after adjustment for age, sex, duration of diabetes, body mass index, HbA1c, and hypertension. Each one-unit increase in VAI was associated with a 48% increase in the adjusted odds of having early diabetic retinopathy, microalbuminuria, or both.

Table 4. Adjusted association between Visceral Adiposity Index and composite early microvascular involvement

Predictor	Outcome	odds ratio	p-value	variables
Visceral Adiposity Index, per one-unit increase	Early diabetic retinopathy, microalbuminuria, or both	1.48	<0.05	Age, sex, diabetes duration, BMI, HbA1c, hypertension

BMI: body mass index; HbA1c: glycated haemoglobin.

DISCUSSION

This study demonstrated that higher VAI was associated with early retinal and renal microvascular abnormalities among adults with type 2 diabetes mellitus. Participants with diabetic retinopathy, microalbuminuria, or both had substantially higher VAI values than those without either condition. The standardized difference in VAI was large for the composite outcome and remained large when retinopathy and microalbuminuria were evaluated separately. The association appeared somewhat stronger for microalbuminuria, and VAI was positively related to UACR. These findings suggest that the combination of abdominal adiposity and lipid disturbance represented by VAI may provide metabolic information relevant to microvascular involvement beyond general body size alone.

VAI was developed as a sex-specific surrogate of visceral adipose dysfunction using waist circumference, body mass index, triglycerides, and HDL cholesterol. Its clinical rationale is that visceral adiposity cannot be adequately characterized by body mass index alone because people with comparable body mass index values may have markedly different abdominal-fat distribution and metabolic profiles. The original VAI studies showed that the index was associated with cardiometabolic risk and adipose-tissue dysfunction (14,15). Subsequent research identified associations of VAI with insulin resistance, metabolic syndrome, and type 2 diabetes risk (16-18). The present results are consistent with this conceptual basis, as participants with microvascular involvement had greater waist circumference and triglycerides, lower HDL cholesterol, and substantially higher VAI values.

The association between VAI and microalbuminuria was particularly notable. Participants with microalbuminuria had a mean VAI that was 1.56 units higher than that of participants without microalbuminuria, representing a large standardized difference. Wen and Yuan similarly reported an independent association between VAI and microalbuminuria among patients with newly diagnosed type 2 diabetes (23). Population-based evidence has also linked VAI with increased urinary albumin excretion and reduced renal function (24,25). Analyses from diabetic cohorts, including the ACCORD population and adults with diabetes in the United States, have further demonstrated associations between VAI and nephropathy or diabetic kidney disease outcomes (20,21). Collectively, these findings support the possibility that visceral adipose dysfunction is related to renal microvascular injury across different diabetic populations.

Several biological mechanisms may explain this relationship. Visceral adipose tissue promotes insulin resistance, chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and adverse adipokine signalling. These processes may increase intraglomerular stress, impair vascular permeability, and contribute to urinary albumin leakage. Dyslipidaemia may further aggravate renal endothelial and tubular injury. The pronounced differences in triglycerides, HDL cholesterol, HbA1c, and UACR observed in the microvascular-involvement group are consistent with a clustering of glycaemic, lipid, and vascular risk. Albuminuria screening remains clinically important because diabetic kidney injury may develop before serum creatinine becomes substantially abnormal (26-29). VAI should therefore be interpreted as a potential complementary metabolic marker rather than as a replacement for UACR or established kidney-function assessment.

Higher VAI was also associated with early diabetic retinopathy. Participants with retinopathy had a mean VAI 1.37 units higher than participants without retinal involvement, with a large standardized effect size. This finding is consistent with prospective evidence linking visceral obesity indices with incident diabetic retinopathy (19). Research evaluating abdominal-obesity indices has similarly identified relationships between visceral adiposity and diabetic complications, although the strength and direction of such

associations can vary according to the index, population, sex distribution, and diabetes duration (22). Current diabetes frameworks distinguish the diagnosis and classification of diabetes from the subsequent surveillance of organ-specific complications (31), while longitudinal evidence confirms that retinopathy may progress over time and requires systematic assessment (32). Studies in Asian populations have further shown that abdominal or visceral adiposity may have a different relationship with retinopathy from generalized obesity measured using body mass index (33,34).

The observed retinal association is biologically plausible. Retinal microvessels are susceptible to hyperglycaemia, oxidative stress, inflammation, hypertension, and lipid-mediated endothelial injury. Visceral adiposity may amplify these processes through insulin resistance and altered lipid metabolism. A recent systematic review and meta-analysis of lipid biomarkers and diabetic microvascular complications supports the broader relationship between dyslipidaemia and vascular injury in diabetes (35). The higher triglyceride concentration and lower HDL cholesterol observed among participants with microvascular involvement may therefore reflect metabolic conditions that contribute to both retinal and renal microvascular dysfunction.

The association of VAI with the composite outcome persisted after adjustment for age, sex, diabetes duration, body mass index, HbA1c, and hypertension. The adjusted odds ratio indicated that each one-unit rise in VAI was associated with 48% higher odds of early retinal or renal involvement. This suggests that VAI may capture metabolic variation not completely represented by HbA1c or body mass index. Nevertheless, this result should be interpreted cautiously because VAI is mathematically constructed from body mass index, waist circumference, triglycerides, and HDL cholesterol. Including body mass index in a model that also contains VAI may introduce multicollinearity and affect coefficient stability. Further analyses should compare models containing VAI with models containing its individual components and should report variance-inflation measures, calibration, discrimination, and confidence intervals.

The findings have potential relevance to diabetes care in Pakistan, where the prevalence of diabetes is high and access to comprehensive complication screening may be inconsistent (1-6). Diabetic retinopathy is already an important cause of preventable visual morbidity in the country, and evidence from Lahore demonstrates that it is frequently encountered among patients attending diabetic centres (7-9). Pakistani studies have also documented microalbuminuria and associated metabolic abnormalities in type 2 diabetes (10-13). Because the components required to calculate VAI are commonly obtained during routine assessment, the index may help clinicians recognize a pattern of central adiposity and dyslipidaemia associated with greater microvascular burden. It should not, however, be used to defer or replace direct retinal examination, UACR testing, or kidney-function assessment.

This study had several strengths. It evaluated both retinal and renal abnormalities within the same diabetic population, used sex-specific VAI equations, and considered the microvascular outcomes individually and as a composite endpoint. The reported group summaries also demonstrated internally consistent totals and clinically coherent metabolic gradients. However, several limitations require consideration. The cross-sectional design prevents determination of temporality or causality, and the hospital-based, non-probability sample limits generalizability to the wider community. The composite outcome combined two biologically distinct complications, although the separate analyses showed similar relationships. A single spot UACR may be influenced by transient biological variation, and repeated confirmation of albuminuria was not described. Detailed assessment of diet, physical activity, medication adherence, lipid-lowering treatment, renal function, and other potential confounders was limited. The retinal grading procedure, masking of outcome assessors, and reproducibility of funduscopic classification were not fully characterized. Diagnostic-performance measures, including sensitivity, specificity, calibration, and an externally validated VAI cutoff, were not evaluated. Consequently, the findings support an association between VAI and prevalent microvascular abnormalities but do not establish VAI as a diagnostic or predictive test.

CONCLUSION

Higher Visceral Adiposity Index was associated with prevalent early retinal and renal microvascular abnormalities among adults with type 2 diabetes mellitus, with the largest relationship observed for microalbuminuria. VAI reflected a combined pattern of abdominal adiposity and dyslipidaemia that was not captured by body mass index alone and remained associated with the composite microvascular outcome after adjustment for major clinical covariates. These findings support its potential use as a complementary metabolic risk marker, while standard retinal examination, urinary albumin assessment, and kidney-function evaluation remain necessary for detecting diabetic microvascular complications.

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