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Original Research Article

Beyond Body Mass Index: Association of Visceral Adiposity Index with Breast Cancer

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Abstract:

Background: Body mass index (BMI) is widely used to define obesity but does not describe the distribution or metabolic behaviour of adipose tissue. Visceral fat is more metabolically active than subcutaneous fat and may be more biologically relevant to breast carcinogenesis than overall body mass. The visceral adiposity index (VAI), a sex-specific composite of waist circumference (WC), BMI, triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C), is a validated surrogate marker of visceral fat dysfunction.

Objective: To determine whether VAI is associated with breast cancer, and whether this association is independent of BMI, in a hospital-based case–control study of Indian women.

Materials and Methods: We studied 156 women with newly diagnosed, histologically confirmed breast cancer and 156 age-matched healthy controls attending a tertiary-care teaching hospital. Anthropometric and fasting biochemical variables were recorded, and VAI was calculated using the Amato formula. Group comparisons used the independent-samples t-test or Mann–Whitney U test as appropriate; logistic regression (crude and adjusted for age and BMI) and receiver operating characteristic (ROC) curve analysis were used to evaluate the association and discriminatory performance of VAI.

Results: VAI was significantly higher in cases than controls (median 4.69 [interquartile range, IQR 3.85–5.94] vs 3.43 [IQR 2.78–4.37]; $p<0.001$), as were BMI, WC, TG and VLDL-cholesterol, while HDL-cholesterol, total cholesterol, serum albumin and total lymphocyte count were lower (all $p<0.05$). Women in the highest VAI quartile had markedly higher odds of breast cancer than those in the lowest quartile (odds ratio [OR] 12.18, 95% confidence interval [CI] 5.70–26.01; p for trend <0.001). Each unit increase in VAI remained independently associated with breast cancer after adjustment for age and BMI (adjusted OR 2.16, 95% CI 1.73–2.68; $p<0.001$). VAI showed acceptable discrimination for breast cancer (area under the curve [AUC] 0.748, 95% CI 0.694–0.802), broadly comparable to WC (AUC 0.788) and BMI (AUC 0.713).

Conclusion: In this case–control study, higher VAI was associated with breast cancer independently of BMI, supporting the hypothesis that indices capturing visceral fat function may carry information beyond simple body mass. These observational findings require confirmation in prospective, multi-centre studies before any diagnostic or risk-stratification use can be recommended.

Keywords: Breast Neoplasms; Obesity, Abdominal; Body Mass Index; Waist Circumference; Triglycerides; Case-Control Studies.

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Introduction

Breast cancer is the most frequently diagnosed cancer among women worldwide, accounting for an estimated 2.3 million new cases in 2020 [1]. In India, the disease has overtaken cervical cancer as the leading cancer site in most urban population-based registries, with a rising incidence trend documented over successive decades [2]. Despite advances in screening and systemic therapy, breast cancer remains a major cause of cancer-related mortality, and identification of modifiable, biologically informative risk markers continues to be a priority for both prevention and risk stratification.

Obesity has long been recognised as a modifiable risk factor for several malignancies. A landmark meta-analysis of prospective observational studies demonstrated that increasing BMI is associated

with a graded increase in the risk of a number of cancers, including postmenopausal breast cancer [3]. Subsequent dose-response meta-analyses have shown that measures of central or abdominal adiposity, such as waist circumference, are independently associated with breast cancer risk across both pre- and postmenopausal women, sometimes more consistently than BMI itself [4]. This discrepancy reflects a fundamental limitation of BMI: as a simple ratio of weight to height, it cannot distinguish fat mass from lean mass, nor can it describe how adipose tissue is distributed across visceral and subcutaneous depots. Two individuals with an identical BMI may therefore carry markedly different amounts of metabolically active visceral fat and correspondingly different cardiometabolic and oncological risk [5].

Visceral adipose tissue differs biologically from subcutaneous fat: it is more lipolytically active, drains directly into the portal circulation, and secretes a distinct profile of adipokines and inflammatory mediators. To capture this dimension of adiposity without resorting to imaging, Amato and colleagues proposed the visceral adiposity index (VAI), an empirically derived, sex-specific formula combining waist circumference, BMI, fasting triglycerides and HDL-cholesterol, that correlated with directly measured visceral fat and cardiometabolic risk in an Italian cohort [6]. Because VAI incorporates both an anthropometric and a lipid/functional component, it has been proposed as a more integrated marker of "adipose tissue dysfunction" than either BMI or WC alone, although its performance appears to vary across ethnic groups and clinical populations [7].

Several biologically plausible pathways link visceral adiposity to breast carcinogenesis. Insulin resistance, a hallmark of visceral fat dysfunction, results in compensatory hyperinsulinaemia and increased bioavailability of insulin-like growth factor-1, a recognised mitogen for breast epithelium, while also reducing hepatic synthesis of sex-hormone-binding globulin and thereby raising bioavailable oestrogen [8,9]. Dysfunctional visceral fat is further characterised by chronic low-grade inflammation [10] and by an altered adipokine profile, with relatively higher leptin and lower adiponectin concentrations that favour breast epithelial proliferation and reduced apoptosis [11,12]. Adipose tissue is also the principal site of peripheral aromatisation of androgens to oestrogens after menopause, and obesity-related breast inflammation has been shown to further upregulate local aromatase expression in breast tissue [13,14].

Only a small number of case–control studies, predominantly from Latin America, have directly examined VAI in relation to breast cancer [15,16], and data from South Asian populations – in whom fat distribution and cardiometabolic risk at a given BMI are known to differ from Western cohorts – remain scarce. We therefore conducted this hospital-based case–control study to compare VAI and its constituent variables between women with newly diagnosed breast cancer and age-matched healthy controls, to examine whether VAI is associated with breast cancer independently of BMI, and to evaluate its discriminatory performance using ROC curve analysis.

Materials and Methods

Study Design and Setting: This was a hospital-based, case–control study conducted in the Department of Biochemistry in conjunction with the Departments of Surgery and Oncology of a tertiary-care teaching hospital in Jaipur, India. The present analysis is a focused, secondary statistical examination of the visceral adiposity index within an existing case–control clinical-biochemical dataset that had originally been assembled to compare lipid profiles between breast cancer patients and healthy controls; VAI was not the parent study's primary hypothesis, and this should be borne in mind when interpreting the findings (see Limitations).

Study Population: The dataset comprised women with newly diagnosed, histologically confirmed breast cancer (cases), recruited from the outpatient and inpatient services of the Surgery and Oncology departments, and apparently healthy women (controls) drawn from patient attendants, hospital staff and students, frequency-matched for age. During data cleaning, two entries in each group were identified as row-level summary statistics (standard-deviation rows) rather than individual patient records and were excluded. The final analytic sample comprised 156 cases and 156 controls (total N=312).

Inclusion and Exclusion Criteria: Women aged 30–65 years with a new histological diagnosis of breast cancer who provided written informed consent were eligible as cases; age-matched healthy women without a personal history of malignancy were eligible as controls. Women with known diabetes mellitus, hypertension, thyroid disorder, renal disease, or congenital/acquired cardiac disease; those taking anti-hyperlipidaemic medication; those with a prior diagnosis of breast cancer already receiving chemotherapy; and those with a history of oral-contraceptive use or current pregnancy were excluded from both groups.

Anthropometric Assessment: Height and weight were measured in light clothing without footwear, and BMI was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the lower costal margin and the iliac crest, at the end of gentle expiration, using a non-stretchable tape.

Biochemical Analysis: Venous blood samples were collected after an overnight fast of at least 8 hours. Serum was separated by centrifugation, and fasting blood sugar, urea, creatinine, SGOT, SGPT, triglycerides, total cholesterol, HDL-cholesterol, LDL-cholesterol and VLDL-cholesterol were measured on a fully automated clinical chemistry analyser using standard enzymatic/colorimetric methods.

Calculation of visceral adiposity index: VAI was calculated for each woman using the sex-specific formula of Amato et al. [6]:

VAI = [WC / (36.58 + 1.89 × BMI)] × [TG(mmol/L) / 0.81] × [1.52 / HDL-C(mmol/L)]

Because triglycerides and HDL-cholesterol were recorded in mg/dL, values were converted to mmol/L before calculation using the factors 1 mmol/L triglycerides = 88.5 mg/dL and 1 mmol/L HDL-cholesterol = 38.6 mg/dL.

Statistical analysis: Data were analysed using Python (SciPy and scikit-learn libraries). The distribution of each continuous variable was assessed with the Shapiro–Wilk test. Normally distributed variables (BMI, waist circumference, serum albumin, total lymphocyte count) are presented as mean ± SD and compared using the independent-samples t-test; non-normally distributed variables, including VAI, are presented as mean ± SD together with median and interquartile range and were compared using the Mann–Whitney U test. Spearman's rank correlation was used to examine the relationship of VAI with its constituent variables. Women were also categorised into sample-based quartiles of VAI, and unadjusted odds ratios for breast cancer in each quartile relative to the lowest were calculated with 95% confidence intervals, with a trend test performed by entering quartile rank as an ordinal covariate in logistic regression. Binary logistic regression (maximum-likelihood estimation) was used to estimate the crude and adjusted association of VAI, as a continuous variable, with breast cancer status, with sequential adjustment for age and for age plus BMI. Receiver operating characteristic curve analysis was performed for VAI, WC and BMI, and the area under the curve with 95% confidence interval was estimated using the Hanley–McNeil method [17]; the optimal cut-off was identified using the Youden index. A two-sided p-value <0.05 was considered statistically significant throughout.

Ethical considerations: The parent study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment. The present analysis used de-identified data and did not involve any additional contact with participants.

Results

A total of 312 women were analysed: 156 with newly diagnosed breast cancer and 156 age-matched controls. Age did not differ significantly between groups (44.50 ± 8.09 vs 44.04 ± 8.62 years; p=0.563), confirming adequate age matching between cases and controls.

Table 1. Demographic, Anthropometric, and Biochemical Characteristics of Breast Cancer Patients and Controls

Variable	Cases (n=156) Mean ± SD	Controls (n=156) Mean ± SD	Test statistic	p-value
Age (years)	44.50 ± 8.09	44.04 ± 8.62	MWU	0.563
Haemoglobin (g/dL)	10.27 ± 1.61	10.46 ± 1.90	MWU	0.099
Fasting blood sugar (mg/dL)	92.72 ± 14.04	95.77 ± 12.86	MWU	0.075
Blood urea (mg/dL)	24.83 ± 10.13	22.24 ± 6.75	MWU	**0.040**
Serum creatinine (mg/dL)	0.92 ± 0.25	0.88 ± 0.21	MWU	0.729
SGOT (U/L)	26.56 ± 10.65	27.96 ± 11.56	MWU	0.294
SGPT (U/L)	21.79 ± 9.61	21.41 ± 10.79	MWU	0.342
Triglycerides (mg/dL)	223.02 ± 58.80	191.47 ± 58.57	MWU	**<0.001**
Total cholesterol (mg/dL)	191.25 ± 42.31	203.30 ± 47.40	MWU	**0.009**
HDL-cholesterol (mg/dL)	39.20 ± 5.30	42.91 ± 8.40	MWU	**<0.001**
LDL-cholesterol (mg/dL)	107.46 ± 43.70	122.10 ± 47.71	MWU	**0.003**
VLDL-cholesterol (mg/dL)	44.60 ± 11.76	38.29 ± 11.71	MWU	**<0.001**
BMI (kg/m²)	26.92 ± 3.74	24.09 ± 2.99	t-test	**<0.001**
Waist circumference (cm)	91.52 ± 9.76	80.78 ± 9.07	t-test	**<0.001**
Serum albumin (g/dL)	3.68 ± 0.46	4.20 ± 0.37	t-test	**<0.001**
Total lymphocyte count (/mm³)	1929.56 ± 526.27	2210.90 ± 542.64	t-test	**<0.001**

MWU = Mann–Whitney U test (used for variables not normally distributed on Shapiro–Wilk testing); t-test = independent-samples t-test (used for normally distributed variables). SD = standard deviation. p<0.05 considered statistically significant (bold).

Table 1 summarises the demographic, anthropometric and biochemical characteristics of the two groups. Compared with controls, women with breast cancer had significantly higher BMI, waist circumference, triglycerides and VLDL-cholesterol, and significantly lower HDL-cholesterol, total cholesterol, serum albumin and total lymphocyte count (all p<0.05). Blood urea was marginally higher in cases (p=0.040), while fasting blood sugar, haemoglobin, creatinine and liver enzymes (SGOT, SGPT) did not differ significantly between groups. These findings indicate that cases and controls were well matched for age and renal/hepatic function but differed systematically in adiposity- and lipid-related parameters.

Table 2: Comparison and Association of Visceral Adiposity Index and Related Adiposity Markers with Breast Cancer

A. **Group comparison of VAI and its anthropometric components (n=156 per group)**

Variable	Cases	Controls	p-value
VAI (units) – Mean ± SD	4.99 ± 1.62	3.72 ± 1.38	**<0.001**
VAI (units) – Median [IQR]	4.69 [3.85–5.94]	3.43 [2.78–4.37]	**<0.001**
BMI (kg/m²)	26.92 ± 3.74	24.09 ± 2.99	**<0.001**
Waist circumference (cm)	91.52 ± 9.76	80.78 ± 9.07	**<0.001**

B. **Spearman correlation of VAI with its constituent variables (total sample, n=312)**

Component	Spearman ρ	p-value	Interpretation
BMI (kg/m²)	−0.025	0.662	No correlation
Waist circumference (cm)	0.436	**<0.001**	Moderate, positive
Triglycerides (mg/dL)	0.771	**<0.001**	Strong, positive
HDL-cholesterol (mg/dL)	−0.455	**<0.001**	Moderate, inverse

C. **Distribution of breast cancer across sample-based VAI quartiles, with unadjusted odds ratios**

VAI quartile (range)	Cases / Controls (n)	% Cases	Odds ratio (95% CI)
Q1 (0.75–3.12)	15 / 63	19.2%	1.00 (reference)
Q2 (3.14–4.08)	35 / 43	44.9%	**3.42 (1.67–7.01)**
Q3 (4.08–5.21)	48 / 30	61.5%	**6.72 (3.26–13.87)**
Q4 (5.24–11.65)	58 / 20	74.4%	**12.18 (5.70–26.01)**

VAI quartiles were derived from the distribution of VAI in the total analytic sample (n=312; 78 women per quartile). CI = confidence interval. p for linear trend across quartiles <0.001 (logistic regression with quartile rank entered as an ordinal covariate).

Table 2 presents the comparison of VAI and its correlation with its constituent variables, together with the distribution of breast cancer across VAI quartiles. VAI was significantly higher among cases than controls, as were its anthropometric components BMI and WC. Within the total sample, VAI correlated strongly with triglycerides (ρ=0.771) and inversely with HDL-cholesterol (ρ=−0.455), moderately with WC (ρ=0.436), but showed essentially no correlation with BMI (ρ=−0.025), reflecting the compensatory role of BMI within the VAI formula. Breast cancer prevalence increased progressively across VAI quartiles, from 19% in the lowest to 74% in the highest, with a significant dose-response trend (p<0.001).

Table 3: Regression and Diagnostic Performance Analysis of Visceral Adiposity Index for Breast Cancer

A. **Binary logistic regression: association of VAI (continuous, per unit) with breast cancer**

Model	Variable	Odds ratio (95% CI)	p-value
Model 1 – Crude	VAI	**1.83 (1.52–2.21)**	**<0.001**
Model 2 – Adjusted for age	VAI	**1.84 (1.52–2.22)**	**<0.001**
	Age	1.01 (0.98–1.04)	0.414
Model 3 – Adjusted for age + BMI	VAI	**2.16 (1.73–2.68)**	**<0.001**
	BMI	1.39 (1.27–1.53)	**<0.001**
	Age	1.01 (0.98–1.05)	0.390
VAI per 1-SD increase (SD=1.63) – crude	VAI	**2.69 (1.98–3.65)**	**<0.001**

Logistic regression estimated by maximum likelihood (iteratively re-weighted least squares). CI = confidence interval; BMI = body mass index; SD = standard deviation.

B. **ROC curve analysis of VAI, waist circumference and BMI for breast cancer**

Variable	AUC (95% CI)	Optimal cut-off	Sensitivity	Specificity
VAI	**0.748 (0.694–0.802)**	3.61	84.0%	56.4%
Waist circumference (cm)	**0.788 (0.737–0.838)**	87.2	67.9%	77.6%
BMI (kg/m²)	**0.713 (0.656–0.770)**	27.3	44.9%	87.2%

AUC = area under the receiver operating characteristic curve, estimated by the Hanley–McNeil method (all $p<0.001$ vs AUC=0.5). Optimal cut-off identified by the Youden index.

Table 3 shows the logistic regression and ROC analyses. Each unit increase in VAI was associated with approximately 83% higher unadjusted odds of breast cancer (OR 1.83, 95% CI 1.52–2.21), equivalent to an approximately 2.7-fold higher odds per one-standard-deviation increase in VAI. This association was materially unchanged after adjustment for age and was strengthened, not attenuated, after additional adjustment for BMI (adjusted OR 2.16, 95% CI 1.73–2.68), indicating that the relationship between VAI and breast cancer in this dataset was not explained by BMI. VAI showed acceptable discriminatory performance for breast cancer (AUC 0.748), numerically similar to WC (AUC 0.788) and higher than BMI alone (AUC 0.713), although the 95% confidence intervals for these three AUC estimates overlapped substantially.

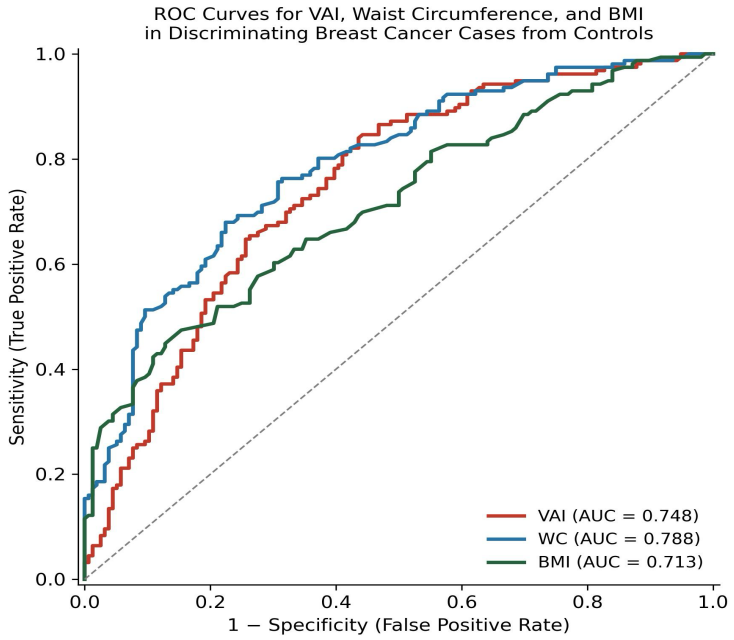

Figure 1: Receiver operating characteristic (ROC) curves for VAI, waist circumference (WC) and BMI in discriminating breast cancer cases from controls. The diagonal reference line represents an area under the curve (AUC) of 0.5 (no discrimination).

Figure 1 shows the receiver operating characteristic curves for VAI, waist circumference and BMI in discriminating breast cancer cases from controls. All three curves lay above the line of no discrimination, confirming that each variable carried some discriminatory information for breast cancer status in this dataset. VAI and WC showed visibly similar curves through most of the sensitivity range, both lying above the BMI curve in the low false-positive-rate region, while all three curves converged at higher false-positive rates. This pattern is consistent with WC and VAI capturing overlapping information related to central/visceral adiposity that is only partially reflected by BMI.

Discussion

In this hospital-based case–control study, women with breast cancer had significantly higher VAI than age-matched healthy controls, and this association persisted – and indeed strengthened – after adjustment for BMI. Women in the highest VAI quartile had over twelve-fold higher unadjusted odds of breast cancer than those in the lowest quartile, with a clear dose-response gradient. These findings add to a small body of literature

suggesting that indices of visceral adiposity may capture information about breast cancer that is not fully explained by overall body mass.

Our results are broadly consistent with two previous case–control studies that examined VAI specifically in relation to breast cancer. Godinho-Mota and colleagues, in a Brazilian hospital-based case–control study of 116 cases and 226 controls, reported that higher VAI was independently associated with breast cancer after adjustment for BMI and age (OR 1.91, 95% CI 1.17–3.13) [15]. Similarly, Cardoso-Peña and colleagues found VAI to be significantly higher in Mexican breast cancer survivors than in matched controls [16]. The magnitude of association observed in our cohort was numerically larger than in these prior reports; this may reflect differences in population characteristics, the underlying distribution of VAI, or the tighter age-matching of our control group, and direct comparison across studies is limited by differences in menopausal status, ethnicity and analytic approach.

Several interlinked biochemical mechanisms may plausibly underlie the association we observed. Visceral adipose tissue dysfunction promotes insulin resistance and compensatory hyperinsulinaemia, increasing the bioavailability of insulin-like growth factor-1 and reducing hepatic synthesis of sex-hormone-binding globulin, thereby raising bioavailable oestrogen available to stimulate breast epithelial proliferation [8,9]. Dysfunctional adipocytes also secrete a pro-tumorigenic adipokine profile, with relatively increased leptin and decreased adiponectin, both shown in experimental models to favour breast epithelial proliferation and inhibit apoptosis [11,12]. Chronic low-grade inflammation of adipose tissue, histologically evidenced by macrophage-rich crown-like structures surrounding necrotic adipocytes, has been shown to upregulate local aromatase expression in the breast tissue of obese women, increasing local oestrogen synthesis independent of circulating hormone concentrations [10,14]. The dyslipidaemic component of VAI – elevated triglycerides and low HDL-cholesterol – is itself independently associated with breast cancer risk in several cohort studies and meta-analyses, lending biological plausibility to the lipid terms embedded within the VAI formula [18,19,20]; a broader link between metabolic dysregulation and cancer risk has also been described for the metabolic syndrome as a composite entity [21].

The principal appeal of VAI lies in its low cost and ease of calculation from routine anthropometry and a fasting lipid profile, without additional imaging. In our data, VAI performed similarly to, but not clearly better than, waist circumference alone in discriminating breast cancer status, and neither measure achieved the performance expected of a standalone diagnostic test; any future utility is more plausible as a component of composite risk assessment than as an isolated screening tool. Notably, the association between VAI and breast cancer strengthened rather than attenuated after adjustment for BMI, in contrast to some cardiometabolic studies in which VAI and BMI carry substantially overlapping information [3,5]; this divergence may reflect biology specifically linking visceral fat dysfunction, rather than fat mass per se, to breast carcinogenesis, but could equally represent a chance finding in a moderately sized case–control sample and requires replication in larger, prospective cohorts.

This study has several limitations. Its case–control, hospital-based, single-centre design is susceptible to selection bias and cannot establish a temporal or causal relationship between VAI and breast cancer; elevated VAI could conceivably be a consequence, rather than a cause or correlate, of early tumour-related metabolic change. The sample size, while adequate for the primary comparison, limited statistical power for subgroup analyses by menopausal status or tumour subtype, factors known to modify the association between adiposity and breast cancer risk [22], and these were not available in this dataset. VAI is a surrogate, formula-derived marker validated primarily in non-Indian populations and was not corroborated here with direct imaging-based measurement of visceral fat (computed tomography, magnetic resonance imaging or dual-energy X-ray absorptiometry). Residual confounding by unmeasured factors such as parity, breastfeeding history, physical activity and dietary pattern cannot be excluded. For transparency, we also note that two row-level summary entries per group, rather than individual patient records, were identified in the source spreadsheet and excluded during data cleaning.

Conclusion

In this case–control study of Indian women, visceral adiposity index was significantly higher in those with breast cancer than in age-matched controls, and this association was independent of BMI, with a clear dose-response relationship across VAI quartiles. VAI showed discriminatory performance for breast cancer that was comparable to waist circumference and modestly better than BMI alone. These findings support the biological plausibility that markers of visceral fat dysfunction, rather than overall adiposity, may be relevant to breast cancer, but their observational, cross-sectional derivation means they cannot establish causation. Prospective, multi-centre studies are needed before VAI can be considered for risk-stratification or clinical use.

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