

# The Association of Visceral Adiposity Index with Insulin Resistance in Adults with Prediabetes

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**How to cite this paper:** Shahed-Morshed, Md., Haq, T., Fariduddin, Md., Parveen, S., Tohfa-E-Ayub, Mahbub, N. and Hasanat, M.A. (2021) The Association of Visceral Adiposity Index with Insulin Resistance in Adults with Prediabetes. *Open Journal of Endocrine and Metabolic Diseases*, 11, 12-23.

<https://doi.org/10.4236/ojemd.2021.111002>

**Received:** October 8, 2020

**Accepted:** January 19, 2021

**Published:** January 22, 2021

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## Abstract

Visceral adiposity mediates insulin resistance, but their association among adults with prediabetes is scarce in the literature. This study is aimed to determine the association of visceral adiposity index (VAI) with insulin resistance in adults with prediabetes. This cross-sectional study was done among 117 adults with newly detected prediabetes [m/f; 23/94; mean  $\pm$  SD: Age  $36.30 \pm 9.99$  years, BMI  $28.89 \pm 4.35$  kg/m<sup>2</sup>] based on American Diabetes Association 2018 criteria and 141 matched healthy controls [m/f: 28/113; mean  $\pm$  SD:  $35.30 \pm 6.88$  years, BMI  $25.03 \pm 4.58$ ]. Waist circumference, body mass index, fasting triglyceride, HDL cholesterol, fasting blood glucose and insulin were measured in each group to calculate VAI and homeostatic model assessment of insulin resistance (HOMA-IR). People with prediabetes had significantly higher median value of VAI {3.08 (2.26) vs. 1.86 (2.31);  $p < 0.001$ } with higher frequency of high VAI ( $>1$ ) (98.3% vs. 85.8%;  $p < 0.001$ ) than the control population. HOMA-IR level was significantly higher in prediabetes with high VAI (cut-off of 2.64) than control with normal VAI [2.78 (2.22, 4.15) vs. 2.20 (1.53, 3.36);  $p = 0.002$ ]. VAI was positively correlated with HOMA-IR in females with prediabetes ( $r = 0.299$ ,  $p = 0.003$ ). VAI had predictive association with prediabetes [OR (95% CI): 9.504 (2.173, 41.576);  $p = 0.03$ ] and high insulin resistance (HOMA-IR  $\geq 2.6$ ) in females with prediabetes [OR (95% CI) = 3.50 (1.476, 8.297);  $p = 0.004$ ] only. It could satisfactorily discriminate prediabetes in both sexes (male: AUC = 0.767,  $p = 0.001$ ; female: AUC = 0.641,  $p < 0.001$ ) and high insulin resistance in females with

prediabetes (AUC = 0.641;  $p = 0.019$ ) only. So, VAI was associated with prediabetes and insulin resistance only in females with prediabetes.

## Keywords

Visceral Adiposity Index, Insulin Resistance, Prediabetes, Homeostatic Model Assessment of Insulin Resistance

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## 1. Introduction

Bangladesh is now dealing with a huge burden of diabetes and prediabetes [1]. Prediabetes is the preceding stage of development of DM. It is associated with insulin resistance, metabolic syndrome and cardiovascular diseases [2]. These are mediated via several products including cytokines and free fatty acids secreted in the portal circulation from the visceral fat reaching directly to the liver [3]. Thus, measurement of visceral adiposity may give an early clue of insulin resistance and dysglycemia. High visceral adiposity is now considered as a cardiometabolic risk factor [4]. The gold standard imaging based assessment of visceral adiposity is expensive and impractical in daily practice. Visceral adiposity index (VAI) is a simple clinico-biochemical and gender-specific tool that represents adipose tissue dysfunction and ultimate insulin resistance. It is better than body mass index (BMI) and waist circumference (WC) alone to predict cardiovascular events. So, VAI can be used as an early indicator of cardiometabolic risk especially in patients before developing overt metabolic syndrome [5]. However, visceral adiposity is dependent on ethnicity [6]. So, VAI cut-off should be population specific. Previous study found an association of VAI with insulin resistance in adults with DM [7]. However, the association of VAI with insulin resistance in prediabetes has not been studied among Bangladeshi adults. Furthermore, data regarding this association among adults with prediabetes is scarce in the literature. Therefore, we decided to look at the association of VAI and insulin resistance in adults with prediabetes, and see whether VAI could predict insulin resistance and prediabetes.

## 2. Methodology

This cross-sectional study was done in the Department of Endocrinology, BSMMU and BIRDEM general hospital among 117 adults with newly detected prediabetes and 141 matched healthy control population over a period of two years (July 2017 to June 2019). People with overt cardiac, renal, hepatic, inflammatory and autoimmune diseases; women with pregnancy or lactation or taking any form of estrogen; people taking steroid or antilipid drugs were excluded from the study. Informed consent was taken from each participant. The study protocol was approved by the institutional review board of BSMMU and Bangladesh Diabetes Association (BADAS).

## 2.1. Study Protocol

The participants' age, sex and family history were documented and height, weight & WC were measured as per standard procedure [8]. BMI was calculated from weight and height {1)  $BMI = \text{weight in kg} \div (\text{height in meter})^2$ }. Venous blood was collected after 8 - 12 hours of fasting for measurement of fasting blood glucose, serum fasting insulin, serum triglyceride (TG) and serum high density lipoprotein cholesterol (HDL-C). Blood glucose was measured by glucose oxidase method. Rest of the blood was allowed to clot. After centrifugation for 10 minutes at a spun of 3000 rpm, separated plasma was stored at  $-20^{\circ}\text{C}$  until assay after proper labeling. Fasting insulin was measured by chemiluminescent microparticle immunoassay with ARCHITECT Insulin assay Abbott, USA with a coefficient of variability  $\leq 7\%$ . Assay of fasting TG and HDL-C were done by glycerol phosphate dehydrogenase-peroxidase and precipitating method respectively using total cholesterol enzymatic reagent [9].

## 2.2. Operational Definitions

### 2.2.1. Prediabetes

It was diagnosed according to American Diabetes Association 2018 criteria {any one from below: fasting blood glucose, FBG = 5.6 - 6.9 mmol/L (IFG), 2 hours after 75 gm OGTT = 7.8 - 11 mmol/L (IGT) or  $HbA_{1C} = 5.7\% - 6.4\%$ } and control was defined only by fasting blood glucose criteria [2].

### 2.2.2. Obesity and Central Obesity

The cut-off value to define obesity and central obesity were of BMI of  $25 \text{ Kg/m}^2$  and WC of 90 cm for males & 80 cm for females respectively [10].

### 2.2.3. Hypertriglyceridemia and Low HDL-C

They were defined by their metabolic syndrome criteria (TG  $\geq 150 \text{ mg/dl}$ ; HDL-C  $< 40 \text{ mg/dl}$  for males and  $< 50$  for females) [11].

### 2.2.4. Homeostatic Model Assessment of Insulin Resistance (HOMA-IR)

HOMA-IR is a validated tool for measuring insulin resistance in clinical practice that was calculated from the following formula.

$$2) \text{ HOMA-IR} = \{\text{fasting insulin } (\mu\text{IU/ml}) \times \text{FBG (mmol/L)}\} \div 22.5 \text{ [12].}$$

The cut-off value to define high insulin resistance and hyperinsulinemia were 2.6 and  $12 \mu\text{IU/ml}$  respectively for Bangladeshi population [13].

### 2.2.5. VAI

It was calculated from clinical (BMI, WC) and biochemical (TG, HDL-C) parameters with different formula for each sex.

$$3) \text{ Male: } VAI = \{\text{WC in cm} \div 39.68 + (1.88 \times \text{BMI in kg/m}^2)\} \\ \times (\text{TG in mmol/L} \div 1.03) \times (1.31 \div \text{HDL-C in mmol/L})$$

$$4) \text{ Female: } VAI = \{\text{WC in cm} \div 36.58 + (1.89 \times \text{BMI in kg/m}^2)\} \\ \times (\text{TG in mmol/L} \div 0.81) \times (1.52 \div \text{HDL-C in mmol/L})$$

VAI of 1 is considered as normal visceral adiposity. The cut-off value of VAI >

1 was considered as high in this study [4]. Another cut-off of VAI (2.64) was derived from receiver operating characteristics (ROC) curve to discriminate high insulin resistance from normal in people with prediabetes.

### 2.3. Statistical Analysis

All data were entered, edited and analyzed by computer-based SPSS program (version 22.0). The quantitative values were expressed in mean  $\pm$  standard deviation (SD) for normally distributed values or median (interquartile range, IQR) for skewed values (TG, fasting insulin, HOMA-IR and VAI). The qualitative values were expressed in frequency (percentages, %). To compare the mean value of groups (prediabetes vs. control, insulin resistance high vs. normal and high vs. normal VAI), independent-samples T test and for median value Mann Whitney U test or Kruskal-wallis test were done as appropriate. To test the association between categories (study groups, insulin resistance status and VAI status) Pearson's chi-square test or Fisher's exact test was done as applicable. ROC curves were analyzed to see whether VAI could discriminate presence of high insulin resistance and prediabetes. Logistic regression analyses were done to see whether high VAI could predict prediabetes and high insulin resistance.  $P < 0.05$  was taken as statistically significant.

### 3. Results

A total of 258 adults (117 with prediabetes and 141 healthy matched control) were included in this study. The study population was relatively young (mean age  $< 40$  years) with a female predominance ( $\sim 80\%$  female). The adults with prediabetes had a significantly higher family history of DM than the control population. The frequency of obesity, central obesity, low HDL-C, hypertriglyceridemia and high VAI ( $>1$ ) were significantly higher in people with prediabetes than the control population. Similarly, the mean or median value of BMI, WC, FBG, TG and VAI were significantly higher in the participants with prediabetes. However, the HDL-C was not found significantly lower in female with prediabetes. HOMA-IR level was significantly higher in prediabetes than control due to significant differences in fasting blood glucose (**Table 1(a)** & **Table 1(b)**).

VAI level was not associated with insulin resistance status (cut-off of 2.6) in the study population [normal vs. high insulin resistance {median (IQR)}: total population 2.23 (1.49, 3.67) vs. 2.79 (1.65, 4.27),  $p = 0.053$ ; control 1.70 (1.22, 3.37) vs. 1.96 (1.47, 3.89),  $p = 0.249$  and prediabetes: 2.49 (2.05, 4.11) vs. 3.48 (2.20, 5.01),  $p = 0.196$ ]. Similarly VAI status (cut-off of  $>1$ ) was not associated with insulin resistance status in the study population [normal vs. high insulin resistance {frequency (%)}: total population 132 (91.0%) vs. 104 (92.0%),  $p = 0.826$ ; control 73 (85.9%) vs. 48 (85.7%),  $p = 1.00$ ; prediabetes 59 (98.3%) vs. 56 (98.2%);  $p = 1.00$ ] (not shown).

Total study population was divided into four groups by considering glycemic status (prediabetes and control) and VAI status (high and normal by cut-off of 1

**Table 1.** (a) Characteristics [frequency (%)] of the study population (N = 258); (b) Clinical and biochemical variables [mean  $\pm$  SD or median (IQR)] of the study population (N = 258).

(a)				
Variables	Control (n = 141) Prediabetes (n = 117)		p	
	Frequency (%)			
Sex (female)	113 (80.1)	94 (80.3)	1.00	
Family history of diabetes mellitus	48 (34.0)	71 (60.7)	<0.001	
Obese (BMI ≥ 25 kg/m²)	60 (42.6)	94 (80.3)	<0.001	
Centrally obese (Male ≥ 90 cm, female ≥ 80 cm)	102 (72.3)	108 (92.3)	<0.001	
Low HDL-C (Male < 40 cm, female < 50 mg/dl)	84 (59.6)	87 (74.4)	0.017	
Hypertriglyceridemia (Serum TG ≥ 150 mg/dl)	30 (21.3)	60 (51.3)	<0.001	
Hyperinsulinemia (≥12 mIU/ml)	72 (51.1)	47 (40.2)	0.103	
High insulin resistance (HOMA-IR ≥ 2.6)	56 (39.7)	57 (48.7)	0.166	
High VAI (>1)	121 (85.8)	115 (98.3)	<0.001	
Pearson's chi-square test or Fisher's exact test was done as appropriate. IQR (interquartile range), BMI (body mass index), HDL-C (high density lipoprotein-cholesterol, TG (triglyceride), HOMA-IR (homeostatic model assessment of insulin resistance), VAI (visceral adiposity index).				
(b)				
Variables	Control (n = 141)	Prediabetes (n = 117)	p	
	mean ± SD or median (IQR)			
Age (years)	35.30 ± 6.88	36.30 ± 9.99	0.359	
BMI (kg/m²)	25.03 ± 4.58	28.89 ± 4.35	<0.001	
WC (cm)	Male	83.61 ± 12.38	94.77 ± 14.50	0.005
	Female	88.40 ± 10.64	98.33 ± 9.74	<0.001
FBG (mmol/L)	4.34 ± 0.89	5.79 ± 0.62	<0.001	
Serum HDL-C (mg/dl)	Male	50.36 ± 13.81	37.09 ± 7.43	<0.001
	Female	42.35 ± 16.87	42.94 ± 9.47	0.753
Serum TG (mg/dl)	103.90 (76.55, 143.25)	151.0 (114.50, 203.5.0)	<0.001	
Serum fasting insulin (µIU/ml)	12.0 (6.91, 17.59)	10.80 (7.25, 14.70)	0.172	
HOMA-IR	2.24 (1.27, 3.34)	2.56 (1.93, 3.69)	0.012	
VAI	1.86 (1.26, 3.57)	3.08 (2.07, 4.33)	<0.001	

Independent samples T test or Mann Whitney U test was done as appropriate. SD (standard deviation), IQR (interquartile range), BMI (body mass index), WC (waist circumference), HDL-C (high density lipoprotein-cholesterol, TG (triglyceride), HOMA-IR (homeostatic model assessment of insulin resistance), VAI (visceral adiposity index).

& 2.64). VAI > 1 indicates any abnormality in BMI, WC, TG or HDL-C. Similarly, the cut-off of 2.64 was derived from a ROC curve to discriminate high insulin resistance with a sensitivity and specificity of 50.4% and 62.8% respectively in the study population. There was no statistically significant association found between VAI and insulin resistance in prediabetes and control, except the me-

dian value of HOMA-IR when VAI cut off of 2.64 were used (**Table 2(a)** and **Table 2(b)**). Post hoc analysis (pairwise comparison) showed that HOMA-IR was significantly higher in prediabetes group with high VAI ( $\geq 2.64$ ) than normal VAI with both control ( $p = 0.002$ ) and prediabetes ( $p = 0.032$ ).

VAI was significantly and positively correlated with HOMA-IR in people with prediabetes ( $r = 0.223$ ,  $p = 0.015$ ) but not in control ( $r = 0.132$ ,  $p = 0.119$ ) population. Furthermore, the significant correlation was found only in female participants ( $r = 0.299$ ,  $p = 0.003$ ) with prediabetes (**Table 3**).

Linear regression analysis revealed that VAI level was not significantly associated with HOMA-IR level in the study population [Total:  $\beta = 0.09$ ,  $p = 0.150$ ; control:  $\beta = 0.057$ ,  $p = 0.499$  and prediabetes:  $\beta = 0.085$ ,  $p = 0.361$ ]. VAI  $> 1$  had a higher odds of developing prediabetes [OR (95% CI) = 9.504 (2.173, 41.576);  $p$

**Table 2.** (a) Association of HOMA-IR and VAI (cut-off of  $>1$ ) in the study population (N = 258); (b) Association of HOMA-IR and VAI (cut-off of 2.64) in the study population (N = 258).

(a)					
Study groups	VAI status	HOMA-IR	p	High IR	p
		median (IQR)		frequency (%)	
Control (n = 141)	Normal VAI (n = 20)	1.68 (0.78, 2.91)	0.069	8 (7.1)	0.533
	High VAI (n = 121)	2.25 (1.38, 3.36)		48 (42.5)	
Prediabetes (n = 117)	Normal VAI (n = 2 )	6.52 (1.25)		1 (0.9)	
	High VAI (n = 115 )	2.56 (1.94, 3.63)		56 (49.6)	
(b)					
Study groups	VAI status	HOMA-IR median	p	High IR	p
		(IQR)		frequency (%)	
Control (n = 141)	Normal VAI (n = 94)	2.22 (1.53, 3.36)	0.002	36 (38.3)	0.097
	High VAI (n = 47)	2.40 (1.51, 3.12)		20 (42.6)	
Prediabetes (n = 117)	Normal VAI (n = 52)	2.17 (1.57, 3.37)		20 (38.5)	
	High VAI (n = 65)	2.78 (2.22, 4.15)		37 (56.9)	

Kruskal-Wallis test and Pearson's chi-square test were done respectively; IQR (interquartile range), HOMA-IR (homeostatic model assessment of insulin resistance), VAI (visceral adiposity index).

**Table 3.** Correlation of HOMA-IR with VAI in the study population.

Study population		r	p
Total		0.186	0.003
Control (n = 141)		0.132	0.119
Prediabetes (n = 117)	Total	0.223	0.015
	Male	-0.183	0.393
	Female	0.299	0.003

Spearman's rho correlation test was done,  $r$  = correlation coefficient; HOMA-IR (homeostatic model assessment of insulin resistance), VAI (visceral adiposity index).

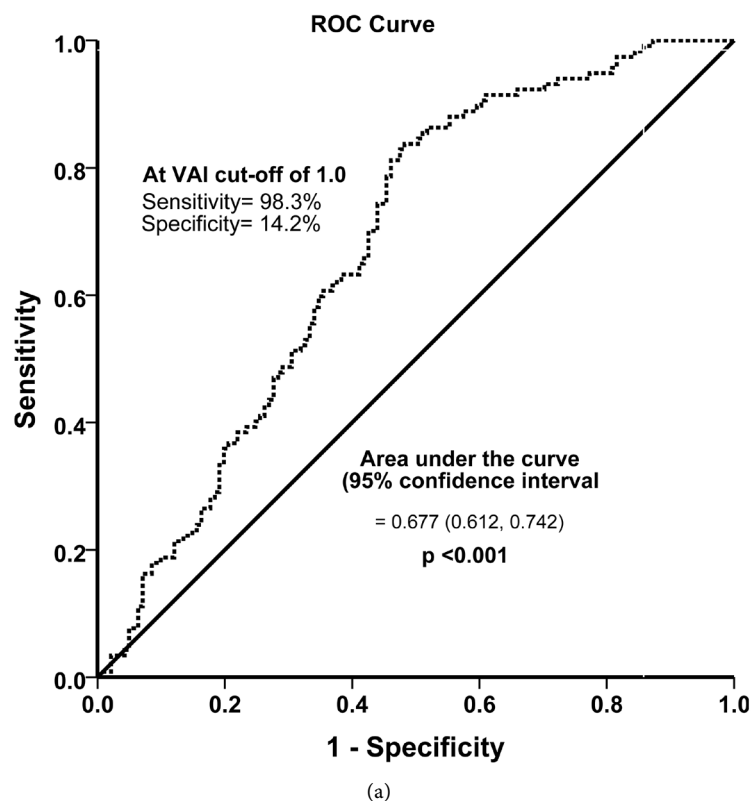
= 0.003] (not shown). Those with a VAI of  $\geq 2.64$  had predictive association with high insulin resistance ( $\geq 2.6$ ) in the study population [OR (95% CI) = 1.666 (1.012, 2.742);  $p = 0.045$ ] and also in the prediabetes group [OR (95% CI) = 2.114 (1.005, 4.448);  $p = 0.048$ ]. The association was only significant in the females with prediabetes [OR (95% CI) = 3.50 (1.476, 8.297);  $p = 0.004$ ] (**Table 4**).

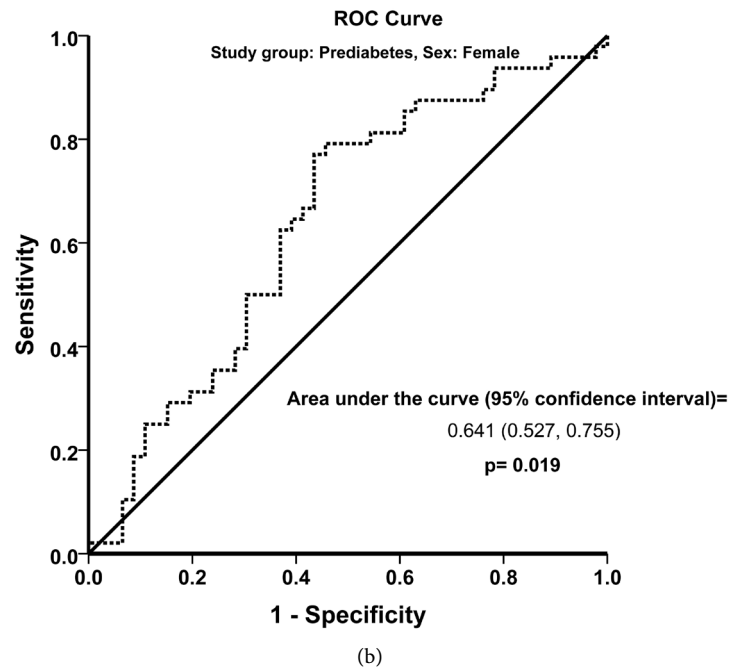
ROC curve analysis showed that VAI was an acceptable discriminator of prediabetes from control [area under the curve, AUC (95% confidence interval, CI) = 0.677 (0.612, 0.742);  $p < 0.001$ ] especially in males [AUC (95% CI) = 0.767 (0.637, 0.897);  $p = 0.001$ ] than females [AUC (95% CI) = 0.654 (0.580, 0.729);  $p < 0.001$ ]. With a VAI cut off of 1, the sensitivity and specificity were 98.3% and 14.2% respectively to discriminate prediabetes from control in the study population (**Figure 1(a)**). On the other hand, VAI was a less acceptable discriminator

**Table 4.** Logistic regression analysis for high VAI (cut-off of 2.64) in predicting high insulin resistance in the study population.

Study groups	OR (95% CI)	P
Total	1.666 (1.012, 2.742)	<b>0.045</b>
Control	1.193 (0.585, 2.433)	0.627
Prediabetes (n = 117)	2.114 (1.005, 4.448)	<b>0.048</b>
Male (n = 23)	0.214 (0.032, 1.425)	0.111
Female (n = 94)	3.50 (1.476, 8.297)	<b>0.004</b>

OR = Odds ratio; CI = confidence interval, VAI (visceral adiposity index).





**Figure 1.** VAI as discriminator of prediabetes (1a) and high insulin resistance in females with prediabetes (1b).

of high insulin resistance in the study population [AUC (95% CI) = 0.570 (0.050, 0.641);  $p = 0.05$ ] and not at all in people with prediabetes [AUC (95% CI) = 0.569 (0.464, 0.674);  $p = 0.196$ ]. However it was better in case of females [AUC (95% CI) = 0.641 (0.527, 0.755);  $p = 0.019$ ] than males [AUC (95% CI) = 0.214 (0.005, 0.423);  $p = 0.023$ ] in adults with prediabetes as a discriminator of high insulin resistance (**Figure 1(b)**).

#### 4. Discussion

This study is aimed to determine the level and status of VAI and its association with insulin resistance in adults with prediabetes. We found that VAI level and high VAI status were significantly higher in adults with prediabetes. Although high VAI status (cut off of  $>1$ ) was not associated with insulin resistance in the study population, HOMA-IR level was significantly higher in people with prediabetes with high VAI (cut off of 2.64) than control and prediabetes with low VAI. VAI was positively correlated with HOMA-IR in females with prediabetes. It had predictive association with prediabetes and high insulin resistance in females with prediabetes. It was also an acceptable discriminator of prediabetes in both sexes and insulin resistance only in females with prediabetes.

We found higher level of VAI and high VAI status in adults with prediabetes that were due to significantly higher BMI, WC, TG and lower HDL-C than the control population. So higher visceral obesity indicates poor glycemic status. Several studies also found similar findings [14] [15] [16] [17]. High VAI was associated with future development of DM in prospective studies [18] [19].

We found significantly higher HOMA-IR level in adults with prediabetes than



control despite an insignificant difference in fasting insulin level. Similar finding was also described by a study [14]. The significant association was due to the significant differences in fasting blood glucose level. However, the frequency of high insulin status and high insulin resistance status were not statistically different between the study groups. The people having prediabetes with high VAI had higher HOMA-IR than people with or without prediabetes with normal VAI. So, VAI mediates higher insulin resistance at a higher glycemic status. Lipid profile might further contribute in insulin resistance [20].

This study found a positive correlation of HOMA-IR with VAI in females with prediabetes. VAI was negatively correlated with insulin sensitivity measured by euglycemic hyperinsulinemic clamp method in IFG but not IGT reported in one study [21]. Another study did not find significant correlation between HOMA-IR and VAI in prediabetes [14].

This study showed that High VAI was associated with increase risk of development of prediabetes and high insulin resistance in females with prediabetes. Several studies also found association of VAI with prediabetes with various odds ratios either using a cut-off to define high VAI or comparing the extreme groups made by various percentile values [14] [15] [16] [17] [22] [23]. We also found an association of insulin resistance in females with prediabetes having high VAI. One study found the association of VAI with insulin resistance among subjects free of DM [19].

Our study found that VAI might be a useful discriminator of prediabetes from healthy population. Several studies also found a similar AUC value but different cut-offs for VAI to define high VAI status [17] [22] [23] [24]. Another study also found VAI as a discriminating index of prediabetes/diabetes (AUC = 0.69) but less good than lipid accumulation product and product of TG & glucose [25]. Another study failed to show VAI as a discriminator of prediabetes [16]. We also found, VAI could acceptably discriminate insulin resistance in females with prediabetes. As per our best knowledge, this is the first report to mention the usefulness of VAI as a discriminator of insulin resistance in people with prediabetes.

The main limitation of the study was small sample size. Besides, the selection of control by only FBG criteria might include participants with IGT or raised HbA<sub>1C</sub> in the control population in this study.

## 5. Conclusion

In conclusion, this study found a positive association of VAI with prediabetes. The association of VAI with insulin resistance was only found in females with prediabetes. VAI could discriminate prediabetes from healthy control and insulin resistance only in females with prediabetes.

## Ethics

The institutional review board of BSMMU and Bangladesh Diabetes Association (BADAS) approved the study protocol.

## Submission Declaration

This article is not under consideration for publication elsewhere. If accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder. All authors and responsible authorities where the work was carried out have approved its publication.

## Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## Conflicts of Interest

The authors declare that they have no competing interests.

## References

- [1] International Diabetes Federation (2019) IDF Diabetes Atlas. 9<sup>th</sup> Edition, Brussels. <https://www.diabetesatlas.org>
- [2] American Diabetes Association (2018) 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2018. *Diabetes Care*, **41**, S13-S27. <https://doi.org/10.2337/dc18-S002>
- [3] Hardy, O.T., Czech, M.P. and Corvera, S. (2012) What Causes the Insulin Resistance Underlying Obesity? *Current Opinion in Endocrinology & Diabetes and Obesity*, **19**, 81-87. <https://doi.org/10.1097/MED.0b013e3283514e13>
- [4] Amato, M.C., Giordano, C., Galia, M., Criscimanna, A., Vitabile, S., Midiri, M. and Galluzzo, A. (2010) Visceral Adiposity Index: A Reliable Indicator of Visceral Fat Function Associated with Cardiometabolic Risk. *Diabetes Care*, **33**, 920-922. <https://doi.org/10.2337/dc09-1825>
- [5] Štěpánek, L., Horáková, D., Cibičková, L., Vavrková, H., Karásek, D., Nakládalová, M. and Zapletalová, J. (2019) Can Visceral Adiposity Index Serve as a Simple Tool for Identifying Individuals with Insulin Resistance in Daily Clinical Practice? *Medicina*, **55**, 545. <https://doi.org/10.3390/medicina55090545>
- [6] Lear, S.A., Humphries, K.H., Kohli, S., Chockalingam, A., Frohlich, J.J. and Birmingham, C.L. (2007) Visceral Adipose Tissue Accumulation Differs According to Ethnic Background: Results of the Multicultural Community Health Assessment Trial (M-CHAT). *The American Journal of Clinical Nutrition*, **86**, 353-359. <https://doi.org/10.1093/ajcn/86.2.353>
- [7] Parveen, S., Ayub, T.E., Haq, T., Nahar, N., Manbub, N., Islam, F., Aktar, F. and Aziz, M. (2020) Association of Visceral Adiposity Index with Insulin Resistance in Adults with Diabetes Mellitus. *IMC Journal of Medical Science*, **14**, 5-12. <https://doi.org/10.3329/imcjms.v14i1.47382>
- [8] World Health Organization (2008) Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation. [https://apps.who.int/iris/bitstream/handle/10665/44583/9789241501491\\_eng.pdf?sequence=1](https://apps.who.int/iris/bitstream/handle/10665/44583/9789241501491_eng.pdf?sequence=1)
- [9] Kaplan, A. and Glucose, K. (1984) ClinChem. C.V. Mosby Co., St Louis, Toronto, Princeton, 436 p.

- [10] World Health Organization (2000) The Asia-Pacific Perspective: Redefining Obesity and Its Treatment. <https://apps.who.int/iris/handle/10665/206936>
- [11] Alberti, K.G.M.M., Eckel, R.H., Grundy, S.M., Zimmet, P.Z., Cleeman, J.I., Donato, K.A., Fruchart, J.-C., James, W.P.T., Loria, C.M. and Smith Jr., S.C. (2009) Harmonizing the Metabolic Syndrome: A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*, **120**, 1640-1645. <https://doi.org/10.1161/CIRCULATIONAHA.109.192644>
- [12] Matthews, D.R., Hosker, J.P., Rudenski, A.S., Naylor, B.A., Treacher, B.F. and Turner, R.C. (1985) Homeostasis Model Assessment: Insulin Resistance and  $\beta$ -Cell Function from Fasting Plasma Glucose and Insulin Concentrations in Man. *Diabetologia*, **28**, 412-419. <https://doi.org/10.1007/BF00280883>
- [13] Bhowmik, B., Siddiquee, T., Mujumder, A., Rajib, M.M.R., Das, C.K., Khan, M.I., Khan, A.K.A. and Hussain, A. (2016) Identifying Insulin Resistance by Fasting Blood Samples in Bangladeshi Population with Normal Blood Glucose. *Journal of Diabetes*, **7**, 4.
- [14] Yang, Y., Feng, Y., Ma, X., Chen, K., Wu, N., Wang, D., Li, P., Wang, M., Li, Q. and Zhang, J. (2015) Visceral Adiposity Index and Insulin Secretion and Action in First-Degree Relatives of Subjects with Type 2 Diabetes. *Diabetes/Metabolism Research and Reviews*, **31**, 315-321. <https://doi.org/10.1002/dmrr.2615>
- [15] Zheng, S., Shi, S., Ren, X., Han, T., Li, Y., Chen, Y., Liu, W., Hou, P.C. and Yaomin, H. (2016) Triglyceride Glucose-Waist Circumference, a Novel and Effective Predictor of Diabetes in First-Degree Relatives of Type 2 Diabetes Patients: Cross-Sectional and Prospective Cohort Study. *Journal of Translational Medicine*, **14**, Article No. 260. <https://doi.org/10.1186/s12967-016-1020-8>
- [16] Nusrianto, R., Ayundini, G., Kristanti, M., Astrella, C., Amalina, N., Muhadi, Riyadina, W., Tahapary, D.L. and Soewondo, P. (2019) Visceral Adiposity Index and Lipid Accumulation Product as a Predictor of Type 2 Diabetes Mellitus: The Bogor Cohort Study of Non-Communicable Diseases Risk Factors. *Diabetes Research and Clinical Practice*, **155**, Article ID: 107798. <https://doi.org/10.1016/j.diabres.2019.107798>
- [17] Kumpatla, S., Michael, C. and Viswanathan, V. (2011) Visceral Adiposity Index and Pattern of Dyslipidaemia at Different Stages of Glucose Intolerance—A Study from India. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, **5**, 173-178. <https://doi.org/10.1016/j.dsx.2012.05.003>
- [18] Chen, C., Xu, Y., Guo, Z.-R., Yang, J., Wu, M. and Hu, X.S. (2014) The Application of VAI in Identifying type 2 Diabetes Risks Based on a Prospective Cohort in China. *Lipids in Health and Disease*, **13**, Article No. 108. <https://doi.org/10.1186/1476-511X-13-108>
- [19] Brahimaj, A., Rivadeneira, F., Muka, T., Sijbrands, E.J., Franco, O.H., Dehghan, A. and Kavousi, M. (2019) Novel Metabolic Indices and Incident Type 2 Diabetes among Women and Men: The Rotterdam Study. *Diabetologia*, **62**, 1581-1590. <https://doi.org/10.1007/s00125-019-4921-2>
- [20] Fiorentino, T.V., Marini, M.A., Succurro, E., Andreozzi, F. and Sesti, G. (2019) Relationships of Surrogate Indexes of Insulin Resistance with Insulin Sensitivity Assessed by Euglycemic Hyperinsulinemic Clamp and Subclinical Vascular Damage. *BMJ Open Diabetes Research & Care*, **7**, e000911. <http://dx.doi.org/10.1136/bmjdr-2019-000911>

- [21] Liu, P.J., Ma, F., Lou, H.P. and Chen, Y. (2016) VAI Is Associated with Pre-Diabetes and Diabetes Mellitus in Chinese Adults Aged 20-50. *Annals of Nutrition and Metabolism*, **68**, 235-243. <https://doi.org/10.1159/000446121>
- [22] Samuel, V.T., Petersen, K.F. and Shulman, G.I. (2010) Lipid-Induced Insulin Resistance: Unravelling the Mechanism. *Lancet*, **375**, 2267-2277. [https://doi.org/10.1016/S0140-6736\(10\)60408-4](https://doi.org/10.1016/S0140-6736(10)60408-4)
- [23] Du, T., Yuan, G., Zhang, M., Zhou, X., Sun, X. and Yu, X. (2014) Clinical Usefulness of Lipid Ratios, Visceral Adiposity Indicators, and the Triglycerides and Glucose Index as Risk Markers of Insulin Resistance. *Cardiovascular Diabetology*, **13**, Article No. 146. <https://doi.org/10.1186/s12933-014-0146-3>
- [24] Gu, D., Ding, Y., Zhao, Y. and Qu, Q. (2018) Visceral Adiposity Index was a Useful Predictor of Prediabetes. *Experimental and Clinical Endocrinology & Diabetes*, **126**, 596-603. <https://doi.org/10.1055/s-0043-120440>
- [25] Ahn, N., Baumeister, S.E., Amann, U., Rathmann, W., Peters, A., Huth, C., Thorand, B. and Meisinger, C. (2019) Visceral Adiposity Index (VAI), Lipid Accumulation Product (LAP), and Product of Triglycerides and Glucose (TyG) to Discriminate Prediabetes and Diabetes. *Scientific Reports*, **9**, Article No. 9693. <https://doi.org/10.1038/s41598-019-46187-8>