ORIGINAL ARTICLE

VISCERAL ADIPOSITY INDEX: A SIMPLE TOOL FOR ASSESSING RISK OF TYPE 2 DIABETES MELLITUS

Adnan Zar, Sobia Sabir Ali
MTI, Lady Reading Hospital, Peshawar-Pakistan

Background: The prevalence of type 2 diabetes mellitus (T2DM) has increased recently in Pakistan. Visceral adiposity index (VAI) appears to be a better predictor for metabolic syndrome associated with insulin resistance. VAI has been shown to be linearly and positively associated with diabetes mellitus (DM) in many populations. It is, however, uncertain whether VAI could be associated with T2DM in the Pakistani adult population. **Methods:** This is a cross-sectional study of 300 outpatients with a newly diagnosed T2DM. Subjects were recruited from Lady Reading Hospital, Pakistan, during the period from April, 2020 to January, 2021. For all study subjects, anthropometric measurements were performed. Blood samples were collected for the assessment of high-density lipoproteins (HDL-C), triglycerides (TGs), glycated hemoglobin (HbA1c), and random blood glucose. **Results:** Participants with high VAI showed poor glycemic control. The number of patients with poor glycemic control increased across the VAI quartiles. VAI showed significant correlations with TGs (r=0.715, *p*=<0.001), total cholesterol (TC) (r=0.256, *p*=<0.001), low density lipoprotein (LDL-C) (r=0.154, *p*=0.007), uric acid (r=0.205, *p*=0.019), duration of diabetes (r=0.171, *p*=0.033), TSH (r=0.163, *p*=0.007), and random blood glucose (r=0.195, *p*=0.019). **Conclusion:** Our data suggest that VAI is significantly and positively correlated with the risk factors of DM such as random blood glucose, uric acid and TSH. The findings of the study do not imply a significant direct association between VAI and DM among the Pakistani adult population. Prospective-large scale studies can help inform an effectiveness of VAI for the prediction of the risk of T2DM among Pakistani population.

Keywords: Visceral adiposity; Anthropometric; Random blood glucose; Diabetes mellitus

Citation: Zar A, Ali SS. Visceral Adiposity Index: A simple tool for assessing risk of Type 2 Diabetes Mellitus. J Ayub Med Coll Abbottabad 2022;34(2):345–50.

INTRODUCTION

Globally, diabetes mellitus (DM) is a major risk factor for morbidity and mortality in terms of cardiovascular outcomes.[1,2] Obesity is considered as one of the leading risk factors for the development of type 2 Diabetes mellitus (T2DM) and its complications.[3] Body mass index (BMI) is considered an indicator of obesity in clinical settings. A high BMI is a risk factor for the development of metabolic syndrome including insulin resistance and T2DM. Studies have been done on the importance of BMI in predicting body adiposity but there were limitations in the significance of BMI alone.[4,5]

Visceral adiposity is an important public health challenge and an independent risk factor for the development of T2DM, insulin resistance and glucose intolerance.[6] In this regard, many anthropometric measurements have been proposed to measure visceral adiposity and predict the presence of insulin resistance and development of T2DM.[7] Previous study reported that the waist circumference (WC) is relatively better correlated with visceral fat than BMI particularly in men.[8] There is a significant correlation of metabolic syndrome including T2DM with deranged high-density lipoproteins (HDL-C) and triglycerides (TGs).[9] These entire correlations such as WC, BMI, HDL-C and TGs lead to an idea of a new index called visceral adiposity index (VAI).[10]

The VAI suggested by Amato et al is a reliable indicator of visceral fat distribution and visceral adiposity dysfunction.[10] VAI is a mathematical formula that consists of laboratory parameters and the core elements of anthropometry such as BMI and WC, TGs, and HDL-C.[11] Sigina *et al* suggested that VAI can be considered as a potential tool for assessing cardiometabolic risk.[12] There is a significant association of VAI with the different components of metabolic syndrome in older men and women including hyperglycemia, hypertriglyceridemia, low HDL-cholesterol, and abdominal obesity.[13] Previous study revealed that VAI can provide an estimation of sub-clinical atherosclerosis than the calculation of Homeostatic Model Assessment of Insulin resistance (HOMA-IR) in individuals prone to T2DM.[14] The VAI was proposed by Amato et al using anthropometric measurements and biochemical data separately in men and women for cardio-metabolic risk in Caucasian Sicilian population.[15] They determined the cut-off values of VAI in different age groups in their population having visceral adipose dysfunction. Following are the optimal VAI cut-off points based on groups: 2.52 (<30 years), 2.23 (≥30 and <42 years), 1.92 (≥42 and <52 years), 1.93 (≥52 and <66 years), and 2.00 (≥66 years).[15] According to the International Diabetes Federation (IDF), about 425 million people worldwide have diabetes, a number that is projected to reach 640 million by 2040.[16] In Pakistan, a

national survey reported that approximately 26.3% of population aged more than 19 years have diabetes.[17] If the current situation continues, Pakistan is expected to achieve a high prevalence of diabetes globally. Likewise, it will put burden on healthcare system and quality of life. Although VAI can predict the risk of T2DM, it is not known whether it is better predictor of T2DM in Pakistani adults. Therefore, the aim of this study was to examine the effectiveness of VAI in predicting the risk of T2DM in the Pakistani adult population. The results of this study will help taking preventive measures in patients with T2DM, thus alleviating the burden of the disease.

MATERIAL AND METHODS

It was a cross-sectional study conducted during April, 2020 to January, 2021 at the Department of Endocrinology, Lady Reading Hospital, which is a tertiary care setting with a 1691-bed capacity in Peshawar, Pakistan. This setting serves a wide range of patients drawn from a large population, many of whom present with complex medical comorbidities and are referred from different regions of the country. We included both male and female patients aged 18 years and above, and outpatients with newly diagnosed T2DM with duration <1 year. We excluded patients with type 1 DM, and other endocrine disorders such as polycystic ovarian syndrome, Cushing's syndrome and acromegaly, besides that pregnancy, lactation, acute illness within 3 months, including COVID 19 and major surgery were excluded. Patients with recent use of steroids and/or statins and malignant diseases undergoing chemo or radiotherapy were also excluded.

For this study, a convenience sample of study subjects was recruited from our study setting. The data were collected from the patient files and the hospital's electronic health records. They were organized on data collection sheets, and the following variables were examined: age, gender, marital status, education, occupation, duration of diabetes in newly diagnosed cases, family history of diabetes, weight, height, BMI, WC, hip circumference, systolic and diastolic blood pressure, glycated hemoglobin (HbA1c), TGs, and HDL-C. Diagnosis of T2DM was based on the diagnostic criteria suggested by American Diabetes Association.[18] BMI was calculated using the following equation: weight (kg)/ height (m^2). WC was measured in centimeters (cm) with a measuring tape kept at mid-point between lower rib and iliac crest. For all included patients, 10 ml blood was collected under aseptic technique and sent to the laboratory on the same day for investigations of TG, HDL-c, HbA1c and random blood glucose. All tests were done under the supervision of a senior hospital pathologist.

VAI was calculated using the following formulas[14]:

$$\text{Male: VAI}=\left(\frac{\text{WC}}{39.68+(1.88\times\text{BMI})}\right)\times\left(\frac{\text{TG}}{1.03}\right)\times\left(\frac{1.31}{\text{HDL}}\right)$$

$$\text{Female: VAI}=\left(\frac{\text{WC}}{36.58+(1.89\times\text{BMI})}\right)\times\left(\frac{\text{TG}}{0.81}\right)\times\left(\frac{1.52}{\text{HDL}}\right)$$

This study was conducted according to the ethical principles of the Declaration of Helsinki. All study subjects were briefed about the objective of the study and were also provided study information sheet. A consent statement was given to each study subject before his/her recruitment in the study. We did not recruit any potential research subject without obtaining verbal consent. This study was commenced after the approval (Reference number: 488/LRH/MTI) from Institutional Review Board of Lady Reading Hospital.

The sample size was determined using an online calculator. It is based on the prevalence (26.3%) of T2DM in Pakistani population reported by a national survey[16] with 5% margin of error, a 95% confidence interval, which yielded a sample size of 300. Data analysis was performed with the Statistical Package for the Social Sciences software, version 22.0 (IBM, Armonk, New York). Descriptive statistics were used to report the data, and normally distributed data were presented as mean ± standard deviation. Independent samples' t-test and analysis of variance (ANOVA) were applied for two and more than two means difference, respectively. A non-parametric Mood's median test was used for non-normally distributed data. Pearson correlation was used to explore any associations between VAI, age, anthropometric and laboratory investigations. A p-value of <0.05 was taken as the level of significance between responses.

RESULTS

A total of 300 Pakistani patients with T2DM (mean age 45.3±11.6 years; 52.0% females) were enrolled in this study. Table 1 shows study characteristics of the study sample. Young patients of 18-45 years and females had significantly higher VAI (p<0.01). About 5.8% of the participants had a high education level. Seventy percent of the patients were unskilled, and half of the total patients reside in rural area. More than 80% patients had duration of diabetes up to 6 months. Ten percent of T2DM patients had HbA1c levels within the American Diabetes Association recommendations for good glycemic control (<7%). Approximately 60% of patients had higher HbA1c levels (>9%). Two-thirds of patients had a family history of DM. The prevalence of comorbidities varied from 0.70% to 42.3%, with fatty liver having the highest prevalence, followed by hypertension (29.3%), dyslipidemia (6.7%), and gout (6.7%). Table-2 represents the distribution of age, anthropometric and laboratory investigations by VAI quartiles. Majority of the variables were greater in higher VAI quintiles. Height and HDL-C were inversely associated with VAI quartiles. The

percentage of patients with poor glycemic control (>7%) also increased across the VAI quartiles. Table-3 shows the correlations between VAI and different clinical and anthropometric parameters. VAI had a weak correlation with TC (r=0.256, *p*=<0.001), LDL-C (r=0.154, *p*=0.007), and TSH (r=0.163, *p*=0.007). In contrast, VAI was negatively correlated to height (r=-0.141, *p*=0.015) and HDL-C (r=-0.402, *p*=<0.001). VAI had a good correlation with TGs (r=0.715, p=<0.001). VAI also had a weak correlation with uric acid (r=0.205, *p*=0.019) and random blood glucose (r=0.195, *p*=0.019) in male patients, whereas duration of diabetes (r=0.171, *p*=0.033) and TSH (r=0.234, *p*=0.004) in female patients.

Table-1: Study Characteristics (n=300)

Variables	Descriptive	Visceral Adiposity Index	P-value
Age, years; mean ±SD	45.3 ±11.6		
Age groups			**0.005**
18-45 years	167 (55.7)	5.0 ±3.4	
46-75 years	133 (44.3)	4.1 ±2.3	
Gender			**<0.001**
Male	144 (48)	3.9 ±2.7	
Female	156 (52)	5.2 ±3.2	
Education			0.464
Uneducated	147 (49)	4.9 ±3.1	
Primary	24 (8)	5.0 ±3.1	
Middle	37 (12.3)	4.4 ±3.6	
Secondary	22 (7.3)	3.7 ±2.4	
Higher-secondary	31 (10.3)	3.9 ±2.1	
Graduation	22 (7.3)	4.1 ±2.1	
Post-graduation	17 (5.8)	4.6 ±4.2	
Occupation			**0.023**
Unskilled	211 (70.3)	4.8 ±3.2	
Skilled	59 (19.7)	3.6 ±1.8	
Professional	30 (10)	4.4 ±3.5	
Area of Living			0.799
Urban	96 (32)	4.6 ±2.8	
Peri-urban	53 (17.7)	4.8 ±3.1	
Rural	151 (50.3)	4.5 ±3.1	
Duration of T2DM			0.801
1week – 6 months	241 (80.3)	4.5 ±2.9	
7 months – 1 year	59 (19.7)	4.6 ±3.6	
HbA1c levels			0.307
< 7%	31 (10.3%)	3.9 ±2.3	
7%-9%	90 (30%)	4.4 ±2.8	
> 9%	179 (59.7%)	4.7 ±3.2	
Associated Conditions			
HTN	88 (29.3)	4.6 ±3.1	0.819
IHD	3 (1)	5.0 ±3.8	0.804
Dyslipidemia	20 (6.7)	6.5 ±3.6	0.003
Hypothyroidism	5 (1.7)	4.6 ±2.2	0.999
Gout	20 (6.7)	6.8 ±5.2	**0.004**
Fatty Liver	127 (42.3)	5.5 ±3.8	**<0.001**
HBS	2 (0.7)	4.5 ±1.6	0.999
HCV	8 (2.7)	5.1 ±5.5	0.442
Family History of Diabetes	201 (67)	4.7 ±3.2	0.400
Meal Frequency			**0.009**
Twice a day	17 (5.7)	4.2 ±2.2	
Thrice a day	248 (82.7)	4.5 ±2.9	
Thrice a day within b/w 1 snack	23 (7.7)	4.1 ±3.0	
Thrice a day within b/w 2 snacks	12 (4)	7.4 ±4.9	
Exercise			0.728
Very Limited	26 (8.7)	4.1 ±1.8	
Limited	249 (83)	4.6 ±3.1	
Somewhat	7 (2.3)	3.8 ±1.7	
Regular	18 (6)	4.8 ±3.9	
Depression			0.513
No	164 (54.7)	4.4 ±2.8	
Mild	76 (25.3)	5.0 ±3.6	
Moderate	59 (19.7)	4.3 ±2.7	
Severe	1 (0.3)	3.4	
Sleep duration, hours			0.405
Upto 3	36 (12)	5.3 ±4.2	
4-6	67 (22.3)	4.3 ±2.3	
7-9	191 (63.7)	4.5 ±2.9	
10 or more	6 (2)	5.3 ±3.9	
Addiction (Smoker/Alcohol)	19 (6.3)	3.7 ±2.6	0.220

SD=standard deviation, HTN=hypertension, IHD=ischemic heart disease, HBS=Hepatitis B Surface Antigen, HCV=hepatitis C virus. All numerical data presented in mean ±SD. All categorical data presented in n (n%).

Table-2: Distribution of age, anthropometric and laboratory investigations by VAI quartiles.

Variables	Visceral Adiposity Index Quartiles				
	VAI <2.56 n=75	VAI= 2.56-3.74 n=74	VAI=3.75-5.58 n=76	VAI >5.58 n=75	*p*-value
Age (years)	46.2 ±12.4	45.9 ±11.8	44.8 ±10.7	44.1 ±11.8	0.679
DM Duration (years)*	2 (4.8)	2 (5)	1.5 (3.3)	2 (5.5)	0.650
Height (cm)	163.7 ±8.9	161.4 ±9.1	159.9 ±8.3	158.6 ±8.5	**0.003**
Weight (kg)	82.1 ±17.3	81.2 ±16.3	80.8 ±20.6	75.4 ±14.9	0.087
BMI (kg/m^2)	30.8 ±7.3	31.2 ±6.1	31.6 ±8.1	29.9 ±5.4	0.448
WC (cm)	105.4 ±13.3	109.2 ±9.8	108.3 ±14.1	106.2 ±11.9	0.218
HC (cm)	103.1 ±13.6	107.8 ±9.5	108.5 ±13.3	106.4 ±13.9	0.052
SBP (mmHg)	129.5 ±19.3	129.9 ±16.2	130.9 ±18.4	133.6 ±18.1	0.503
DBP (mmHg)	83.4 ±13.6	84.3 ±10.8	82.2 ±13.2	83.4 ±12.2	0.802
Hb (g/dL)	12.8 ±1.6	12.6 ±1.3	12.8 ±1.5	12.9 ±1.5	0.513
TLC (x10^3)	8.1 ±2.3	8.1 ±1.8	8.2 ±2.9	8.5 ±3.2	0.824
Platelet (x10^5)*	2.6 (1.01)	2.5 (1.04)	2.8 (1.04)	2.8 (1.30)	0.093
Total Bilirubin*	0.3 (0.4)	0.3 (0.4)	0.3 (0.3)	0.3 (0.4)	0.770
ALT (U/L)*	30 (18)	30 (20.5)	33.5 (23.8)	35 (36)	0.609
ALP (U/L)	123.0 ±40.4	119.4 ±36.2	136.4 ±62.0	147.6 ±105.5	**0.041**
Creatinine	0.8 ±0.3	0.8 ±0.2	0.8 ±0.3	0.8 ±0.2	0.307
TC (mg/dL)	178.1 ±36.6	188.7 ±38.0	193.1 ±52.5	206.1 ±45.0	**0.001**
TGs (mg/dL)	134.7 ±35.3	182.6 ±62.8	251.3 ±139.9	345.7 ±180.9	**<0.001**
LDL (mg/dL)	120.4 ±33.9	135.4 ±32.2	131.6 ±41.8	144.5 ±41.4	**0.001**
HDL-C (mg/dL)	49.0 ±15.2	44.3 ±10.7	38.7 ±9.7	33.5 ±7.9	**<0.001**
HbA1c (%)	9.6 ±2.1	9.6 ±2.1	10.1 ±2.1	10.1 ±2.3	0.412
FPG (mg/dL)	186.5 ±42.7	192.3 ±53.2	197.1 ±103.7	193.3 ±44.5	0.805
RBG (mg/dL)	288.6 ±87.3	298.8 ±86.1	300.8 ±84.5	303.9 ±86.6	0.723
TSH (mIU/mL)	1.6 ±0.8	1.8 ±1.0	1.6 ±0.8	2.1 ±3.0	0.200
Uric Acid (mg/dL)	4.2 ±1.2	4.2 ±1.1	4.4 ±1.3	4.3 ±1.4	0.845

All data presented in mean ±standard deviation. *data presented in median (interquartile range). DM=diabetes mellitus, BMI=body mass index, WC=waist circumference, HC=hip circumference, SBP=systolic blood pressure, DBP=diastolic blood pressure, TGs=triglycerides, HDL-C=high density lipoprotein, LDL=low density lipoprotein, TC=total cholesterol, FPG=fasting plasma glucose, RBG=random blood glucose, Hb=hemoglobin, TLC=total leukocyte count, ALT=alanine transaminase, ALP=alkaline phosphatase, TSH=thyroid stimulating hormone.

Table-3: Correlation of VAI with age, anthropometric and laboratory investigations.

Variables	Overall		Male		Female	
	r	p-value	r	p-value	r	p-value
Age (years)	-0.103	0.076	-0.133	0.112	-0.069	0.393
DM Duration (years)	0.035	0.550	-0.079	0.347	0.171	**0.033**
Height (cm)	-0.141	**0.015**	0.006	0.944	-0.022	0.782
Weight (kg)	-0.072	0.218	0.039	0.642	-0.104	0.198
BMI (kg/m^2)	-0.016	0.788	0.018	0.830	-0.106	0.189
WC (cm)	0.026	0.649	0.127	0.129	-0.087	0.282
HC (cm)	0.094	0.106	0.137	0.102	-0.012	0.884
SBP (mmHg)	0.073	0.206	0.032	0.706	0.048	0.555
DBP (mmHg)	0.007	0.907	-0.069	0.411	0.026	0.746
Hb (g/dL)	0.106	0.067	0.022	0.797	0.246	**0.002**
TLC (x10^3)	0.022	0.700	0.034	0.687	-0.044	0.584
Platelet (x10^5)	0.107	0.064	-0.030	0.724	0.162	**0.044**
Total Bilirubin	0.050	0.387	0.102	0.223	-0.019	0.811
ALT (U/L)	0.066	0.255	0.067	0.422	0.090	0.263
ALP (U/L)	0.112	0.053	0.146	0.081	0.119	0.139
Creatinine	-0.073	0.207	0.024	0.775	-0.128	0.111
TC (mg/dL)	0.256	**<0.001**	0.184	**0.027**	0.319	**<0.001**
TGs (mg/dL)	0.715	**<0.001**	0.877	**<0.001**	0.638	**<0.001**
LDL (mg/dL)	0.154	**0.007**	-0.030	0.725	0.287	**<0.001**
HDL-C (mg/dL)	-0.402	**<0.001**	-0.339	**<0.001**	-0.492	**<0.001**
HbA1c (%)	0.053	0.363	0.079	0.346	0.063	0.437
FPG (mg/dL)	0.031	0.587	0.150	0.072	-0.026	0.743
RBG (mg/dL)	0.043	0.457	0.195	**0.019**	-0.058	0.473
TSH (mIU/mL)	0.163	**0.007**	-0.126	0.158	0.234	**0.004**
Uric Acid (mg/dL)	0.093	0.119	0.205	**0.019**	0.084	0.308

r=Pearson correlation, BMI=body mass index, WC=waist circumference, HC=hip circumference, SBP=systolic blood pressure, DBP=diastolic blood pressure, TGs=triglycerides, HDL-C=high density lipoprotein, LDL=low density lipoprotein, TC=total cholesterol, FPG=fasting plasma glucose, RBG=random blood glucose, Hb=hemoglobin, TLC=total leukocyte count, ALT=alanine transaminase, ALP=alkaline phosphatase, TSH=thyroid stimulating hormone.

DISCUSSION

To the best of our knowledge, this is the first study to examine the role of VAI in T2DM in the Pakistani adult population. This study found that patients with increased VAI showed poor glycemic control. The number of patients with poor glycemic control increased across the VAI quartiles. In addition, VAI showed a good correlation with TGs and weak correlations with TC, TGs, LDL-C, uric acid, random blood glucose, TSH, and duration of diabetes.

Visceral adiposity is regarded as the vital cornerstone of metabolic derangement since it affects metabolism through different pathway and releases increased amount of non-esterified fatty acids, adipocytokines, glycerol, and pro-inflammatory cytokines such as interlukin-6.[18] The relationship between visceral fat accumulation and poor glycemic control can be elucidated at least to a certain extent by the unwarranted release of free fatty acids from the visceral adipose tissue. Moreover, visceral fat has higher rates of lipolysis than subcutaneous fat and the lipolysis of adipose tissue demonstrates less suppression by insulin. It leads to increased transport of free fatty acid to the liver by the portal circulation, thereby stimulating hepatic glucose production by fatty acids. Likewise, the removal of hepatic insulin is recognized to be interfered by fatty acids, which may further enhance the insulin resistance.[19] The findings of this study are not consistent with the findings of previous reports that showed a direct association between VAI and DM.[6,20,21] There is a likelihood that cut-off points of the VAI based on the European population are not appropriate for the Pakistan population. However, we found associations with strong risk factors of T2DM such as random blood glucose (in males), uric acid (in males), and TSH (both male and females).[22,23] According to the International Diabetes Federation, patients with a random blood glucose value 100 to 199 mg/dl (5.6 to 11.0 mmol/L) are required to undergo a formal diabetes testing.[22] Previous study reported a strong association of random blood glucose with an undiagnosed DM.[22]

Chaker *et al* found that the risk of DM is 1.16 times higher with higher TSH levels.[23] Given the recommendations made by Amalo *et al*[10], a VAI may be useful in determining the risk of DM in patients with abnormal thyroid function. DM and thyroid diseases are the two most frequent endocrine disorders, often co-existing in patients.[24] Thyroid hormone regulates metabolic processes and energy expenditure and is directly involved in the control of insulin secretion and glucose homeostasis.[25,26] There are numerous studies on the association between DM and thyroid dysfunction; however, some studies reported associations of both hyperthyroidism and hypothyroidism with DM.[23] Thyroid disease meddles with the action and metabolism of insulin and induces insulin resistance.[27–29] A nation-wide cohort study in Taiwan reported a higher risk of T2DM in patients with thyroid disease.[30] Nonetheless, there is currently no explicit consensus regarding whether individuals with thyroid disease should be screened for DM.[23] In this study, a VAI was also significantly and positively associated with uric acid particularly in male diabetic patients. This result was consistent with other studies.[31,32] Uric acid appears to be an important risk factor that increases the risk of DM.[31,33] It directly inhibits the trigger of insulin signaling pathway at receptor level.[34] Similarly, the levels of serum uric acid increases during the early stages of impaired glucose metabolism.[35]

The strengths of our study include sample of genders and direct assessments of the anthropometric indices rather than self-reported assessment. There were also some limitations to this study. First, this study was cross-sectional and limited in its capacity to inform about causality. Based on the findings of a single setting, the results cannot be generalized to the entire Pakistani population. Therefore, these results need to be validated by a multicenter longitudinal study across healthcare settings in Pakistan.

CONCLUSION

In conclusion, our results suggest that VAI is significantly and positively correlated with the risk factors of DM such as random blood glucose, uric acid, and TSH. The findings of the study do not imply a significant direct association between VAI and DM among the Pakistani adult population. Even though the number of study subjects with poor glycemic control increased across the visceral adiposity index (VAI) quartiles. Prospective-large scale studies can help inform an effectiveness of VAI for the prediction of the risk of T2DM among Pakistani population.

Statement of Ethics:

The study was approved by the Ethical Research Committee (Reference no: 488/LRH/MTI) of MTI, Lady Reading Hospital, Peshawar. All subjects have given their written informed consent.

AUTHORS' CONTRIBUTIONS

SSA: (Principal Investigator and Corresponding Author): Study Concept, Design, Literature Search, Data Collection, Data Analysis, Manuscript Writing. AZ: Data Collection, Data Entry, Edited and Approved the manuscript

Data availability

Data available on reasonable request from the authors.

Acknowledgement

The authors wish to acknowledge SA for his assistance in statistical analysis, manuscript editing and formatting.

REFERENCES

1. Stamler J, Vaccaro O, Neaton JD, Wentworth D. Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. Diabetes Care 1993;16(2):434–44.
2. Tancredi M, Rosengren A, Svensson AM, Kosiborod M, Pivodic A, Gudbjörnsdottir S, *et al*. Excess Mortality among Persons with Type 2 Diabetes. N Engl J Med 2015;373(18):1720–32.
3. Tobias DK, Pan A, Jackson CL, O'Reilly EJ, Ding EL, Willett WC, *et al*. Body-mass index and mortality among adults with incident type 2 diabetes. N Engl J Med 2014;370(3):233–44.
4. Nevill AM, Metsios GS. The need to redefine age- and gender-specific overweight and obese body mass index cutoff points. Nutr Diabetes 2015;5(11):e186.
5. Rothman KJ. BMI-related errors in the measurement of obesity. Int J Obes (Lond) 2008;32(Suppl 3):S56–9.
6. Bozorgmanesh M, Hadaegh F, Azizi F. Predictive performance of the visceral adiposity index for a visceral adiposity-related risk: type 2 diabetes. Lipids Health Dis 2011;10:88.
7. Cornier MA, Després JP, Davis N, Grossniklaus DA, Klein S, Lamarche B, *et al*. Assessing adiposity: a scientific statement from the American Heart Association. Circulation 2011;124(18):1996–2019.
8. Schulze MB, Thorand B, Fritsche A, Häring HU, Schick F, Zierer A, *et al*. Body adiposity index, body fat content and incidence of type 2 diabetes. Diabetologia 2012;55(6):1660–7.
9. Huang PL. A comprehensive definition for metabolic syndrome. Dis Model Mech 2009;2(5-6):231–7.
10. Amato MC, Giordano C. Visceral Adiposity Index: An Indicator of Adipose Tissue Dysfunction. Int J Endocrinol 2014;2014:730827.
11. Oh JY, Sung YA, Lee HJ. The visceral adiposity index as a predictor of insulin resistance in young women with polycystic ovary syndrome. Obesity (Silver Spring) 2013;21(8):1690–4.
12. Gârgavu SR, Clenciu D, Rosu MM, Tenea Cojan TS, Costache A, Vladu IM, *et al*. Visceral Adiposity Index (VAI) – a potential marker of cardiometabolic risk. Arch Balk Med Union 2018;53(2):246–51.
13. Goldani H, Adami FS, Antunes MT, Rosa LH, Fassina P, Quevedo Grave MT, *et al*. Applicatility of the visceral adiposity index (vai) in the prediction of the components of the metabolic syndrome in elderly. Nutr Hosp 2015;32(4):1609–15.
14. Randrianarisoa E, Lehn-Stefan A, Hieronimus A, Rietig R, Fritsche A, Machann J, *et al*. Visceral Adiposity Index as an Independent Marker of Subclinical Atherosclerosis in Individuals Prone to Diabetes Mellitus. J Atheroscler Thromb 2019;26(9):821–34.
15. Amato MC, Giordano C, Pitrone M, Galluzzo A. Cut-off points of the visceral adiposity index (VAI) identifying a visceral adipose dysfunction associated with cardiometabolic risk in a Caucasian Sicilian population. Lipids Health Dis 2011;10:183.
16. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, *et al*. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9(th) edition. Diabetes Res Clin Pract 2019;157:107843.
17. Basit A, Fawwad A, Qureshi H, Shera AS. Prevalence of diabetes, pre-diabetes and associated risk factors: second National Diabetes Survey of Pakistan (NDSP), 2016-2017. BMJ Open 2018;8(8):e020961.
18. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. Nature 2006;444(7121):840–6.
19. Frayn KN. Visceral fat and insulin resistance--causative or correlative? Br J Nutr 2000;83(Suppl 1):S71–7.
20. Cao YY, Tang X, Sun KX, Liu ZK, Xiang X, Juan J, *et al*. [Relationship between glycemic control and visceral adiposity index among the patients with type 2 diabetes mellitus]. Beijing Da Xue Xue Bao Yi Xue Ban 2017;49(3):446–50.
21. Alkhalaqi A, Al-Naimi F, Qassmi R, Shi Z, Ganji V, Salih R, *et al*. Visceral adiposity index is a better predictor of type 2 diabetes than body mass index in Qatari population. Medicine 2020;99(35):e21327.
22. Bowen ME, Xuan L, Lingvay I, Halm EA. Random blood glucose: a robust risk factor for type 2 diabetes. J Clin Endocrinol Metab 2015;100(4):1503–10.
23. Chaker L, Ligthart S, Korevaar TIM, Hofman A, Franco OH, Peeters RP, *et al*. Thyroid function and risk of type 2 diabetes: a population-based prospective cohort study. BMC Med 2016;14(1):150.
24. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. Arch Intern Med 2000;160(4):526–34.
25. Bertrand C, Blanchet E, Pessemesse L, Annicotte JS, Feillet-Coudray C, Chabi B, *et al*. Mice lacking the p43 mitochondrial T3 receptor become glucose intolerant and insulin resistant during aging. PLoS One 2013;8(9):e75111.
26. Crunkhorn S, Patti ME. Links between thyroid hormone action, oxidative metabolism, and diabetes risk? Thyroid 2008;18(2):227–37.
27. Maratou E, Hadjidakis DJ, Kollias A, Tsegka K, Peppa M, Alevizaki M, *et al*. Studies of insulin resistance in patients with clinical and subclinical hypothyroidism. Eur J Endocrinol 2009;160(5):785–90.
28. Shah JH, Motto GS, Papagiannes E, Williams GA. Insulin metabolism in hypothyroidism. Diabetes 1975;24(10):922–5.
29. Stanická S, Vondra K, Pelikánová T, Vlcek P, Hill M, Zamrazil V. Insulin sensitivity and counter-regulatory hormones in hypothyroidism and during thyroid hormone replacement therapy. Clin Chem Lab Med 2005;43(7):715–20.
30. Chen RH, Chen HY, Man KM, Chen SJ, Chen W, Liu PL, *et al*. Thyroid diseases increased the risk of type 2 diabetes mellitus: A nation-wide cohort study. Medicine (Baltimore) 2019;98(20):e15631.
31. Dong H, Xu Y, Zhang X, Tian S. Visceral adiposity index is strongly associated with hyperuricemia independently of metabolic health and obesity phenotypes. Sci Rep 2017;7(1):8822.
32. Kim TH, Lee SS, Yoo JH, Kim SR, Yoo SJ, Song HC, *et al*. The relationship between the regional abdominal adipose tissue distribution and the serum uric acid levels in people with type 2 diabetes mellitus. Diabetol Metab Syndr 2012;4(1):3.
33. Xiong Q, Liu J, Xu Y. Effects of Uric Acid on Diabetes Mellitus and Its Chronic Complications. Int J Endocrinol 2019;2019:9691345.
34. Tassone EJ, Cimellaro A, Perticone M, Hribal ML, Sciacqua A, Andreozzi F, *et al*. Uric Acid Impairs Insulin Signaling by Promoting Enpp1 Binding to Insulin Receptor in Human Umbilical Vein Endothelial Cells. Front Endocrinol (Lausanne) 2018;9:98.
35. Katsiki N, Papanas N, Fonseca VA, Maltezos E, Mikhailidis DP. Uric acid and diabetes: Is there a link? Curr Pharm Des 2013;19(27):4930–7.

Submitted: August 15, 2021	*Revised: August 24, 2021*	*Accepted: September 4, 2021*

Address for Correspondence:

Sobia Sabir Ali, Assistant Professor & Head of Division of Diabetes & Endocrinology, MTI, Lady Reading Hospital, Peshawar-Pakistan

Cell: +92 333 924 7104. **Email:** drsobias@hotmail.com