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VISCERAL ADIPOSITY INDEX AND RESISTENSI INSULIN IN DIABETES MELLITUS TYPE 2

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ABSTRACT

Background: Diabetes mellitus (DM) has been increasing worldwide. Obesity, especially central obesity, is a sign of increased visceral fat in various diseases, especially DM. Individuals with visceral compromise insulin resistance and metabolic disorders and develop diabetes. The gold standard for assessing insulin resistance is the hyperinsulinemic-euglycemic clamp (HEC). However, this method is invasive and expensive, so the homeostasis model assessment-insulin resistance (HOMA-IR) and the quantitative insulin sensitivity check index (QUICKI) are more straightforward, more practical, and invasive measurement methods. Visceral adiposity index (VAI) was developed as a new indicator of visceral adipose tissue dysfunction that can be used for CT examination as a marker of visceral adiposity.
Objective: To determine the correlation between visceral adiposity index and insulin resistance in patients with type 2 diabetes mellitus
Methods: The study was conducted as an analytical observational study using a cross-sectional design approach at the Diponegoro National Hospital Semarang from March to April 2022. Data analysis using the Pearson test (meaningful if $p < 0.05$)
Results: From the 70 samples examined, there was a correlation between VAI and HOMA-IR (r=0,480; p=<0,001), VAI and QUICKI (r=-0,475; p=<0,001), and a strong correlation between HOMA-IR and QUICKI (r=-0,892; p=<0,001)
Conclusion: There was a moderate positive correlation between VAI and HOMA-IR and a moderate negative correlation between VAI and QUICKI. Meanwhile, the results of the correlation test between HOMA-IR and QUICKI show that there is a robust negative correlation.

Keyword : Diabetes mellitus, HOMA-IR, QUICKI, VAI, Insulin resistance

INTRODUCTION

In recent decades, diabetes mellitus (DM) has increased worldwide. In 2015, Indonesia had the seventh-highest prevalence of DM worldwide, after China, India, the United States, Brazil, Russia, and Mexico. According to the 2018 Health Research Study by the Ministry of Health of the Republic of Indonesia, Central Java province has the highest prevalence of DM. [1].

Diabetes mellitus is a significant risk factor for premature death in the general population and imposes a substantial social and economic burden on health systems worldwide. Obesity is closely associated with a higher incidence of type 2 diabetes. It is involved in the development of diabetic complications.[2] Visceral fat in the obese state is associated with a wide range of diseases. Previous studies have reported that individuals with high visceral fat have an increased risk of insulin resistance and metabolic disorders and are more likely to develop DM.[3]

Insulin resistance is a metabolic disorder characterized by the failure of fat storage into subcutaneous adipose tissue, leading to ectopic fat deposition into visceral fat tissue and insulin-sensitive tissues such as liver and skeletal muscle. These tissues progress to lipotoxicity status, interfering with insulin signaling and action, resulting in insulin resistance.[4]

Assessment of insulin resistance is using a hyperinsulinemic-euglycemic clamp (HEC), but this method is invasive, time-consuming, and expensive to implement in clinical practice. Current assessment of insulin resistance using the homeostatic model (HOMA-IR) and quantitative insulin sensitivity check index (QUICKI) are more straightforward, practical, and minimally invasive measurement methods. HOMA-IR and QUICKI can be calculated using fasting blood glucose (GDP) levels and insulin levels.[5]

Magnetic resonance imaging (MRI) and computerized tomography scan (CT) measure visceral fat tissue and body fat distribution. However, these techniques are costly, have radiation side effects, and are unavailable in every health service. There is a need for a simple alternative parameter to measure visceral fat.[6]

The visceral adiposity index (VAI) was developed as a new visceral adipose tissue dysfunction indicator. Zhang M et al. reported that VAI can be used to replace visceral CT scan examination as a marker of visceral adiposity. The visceral adiposity index itself is an indirect measurement method based on gender that measures a combination of anthropometric examinations based on waist circumference (WC), body mass index (BMI), triglyceride (TG) levels, and high-density lipoprotein (HDL) cholesterol levels using the following equation[7]:

Man : VAI = (WC/(39.68 + (1.886*BMI)))*(TG/1.03)* (1.31/HDL)

Woman : VAI = (WC/(36.58 + (1.896*BMI)))* (TG/0.81)* (1.52/ HDL)

Several studies have proven a correlation between VAI and HOMA-IR in various populations. Ji et al. found a correlation between VAI and homeostatic model assessment of insulin resistance (HOMA-IR) in participants with normal weight.[8] Another study by Praveen et al. showed an increase in VAI values in type 2 DM patients, and VAI has a positive correlation with HOMA-IR in DM.[3] The quantitative insulin sensitivity check index is an empirical math-transformed

calculation of fasting glucose and plasma insulin levels that has been shown to provide better predictive power consistently and precisely. QUICKI is a variation of the HOMA equation, according to Gutch et al. QUICKI has a better correlation in patients with diabetes and obesity.[5] A cut-off value of HOMA-IR > 2.5 indicates insulin resistance while QUICKI <0.328.[9]

Based on the explanation above, we want to analyze the correlation between VAI and insulin resistance assessed by the HOMA-IR and QUICKI methods in patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

Several studies have proven a correlation between VAI and HOMA-IR in various populations. Ji et al. found a correlation between VAI and homeostatic model assessment of insulin resistance. The research method is analytic observational with a cross-sectional approach, adult research subjects. This study was conducted from March to April 2022 at Diponegoro National Hospital Semarang, and 70 people participated. Subjects with a history of hepatic, renal, and thyroid disease were excluded from this study. Ethical clearance was obtained from the Health Research Ethics Commission (KEPK) of the Faculty of Medicine, Diponegoro University, with No: 62/EC/KEPK/FK-UNDIP/III/2022.

Consecutive sampling according to the research criteria until the number of samples is met. Fasting glucose was examined using the hexokinase method, while HDL-C and TG used the colorimetric enzymatic method with an automated clinical chemistry device. Fasting insulin was tested using the Chemiluminescent Microparticle Immunoassay (CMIA) method. Body mass index (BMI) examination using the calculation of body weight in kg divided by height in meters squared (Kg/m2). VAI was calculated based on the combination of (WC), (BMI), triglyceride (TG), and (HDL) examinations with the formula,

Male: VAI = (WC/(39.68 + (1.886*BMI))) * (TG/1.03) * (1.31/HDL),

female: VAI = (WC/ (36.58 + (1.896*BMI)) * (TG/0.81) * (1.52/HDL)

Insulin resistance was calculated with HOMA-IR and QUICKI with the following formula :

$$\text{HOMA-IR} = (\text{glucose mg/dL} \times \text{insulin } \mu\text{U/L})/405$$

$$\text{QUICKI} = 1/[\log(\text{Insulin } \mu\text{U/mL}) + \log(\text{Glucose mg/dL})]$$

RESULT

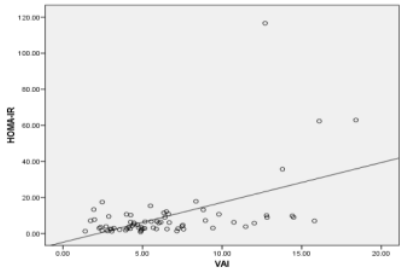
A total of 70 patients who met the criteria participated in the study. Seventy samples were obtained, consisting of 36 (51,4%) men and 34 (48,6%) women.The distribution of subject characteristics is presented in Table 1.

Table 1. Data characteristics of research subjects

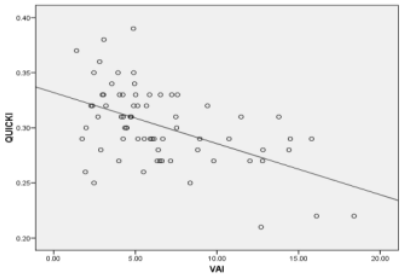
Variable	Mean ± SD	Median (min – max)
Year	56,91 ± 10,16	59 (34 – 79)
WC (cm)	92,37 ± 11,65	91,5 (66 – 120)
BMI (kg/m$^{2)}$	26,29 ± 4,39	25,55 (16,4 – 39,7)
Cholesterol (mg/dL)	200,87 ± 45,50	200,5 (122 – 346)
LDL (mg/dL)	130,04 ± 52,07	123,5 (60 – 388)
Triglycerides (mg/dL)	158,15± 74,69	139,0 (58 - 407)
HDL (mg/dL)	43,9 ± 13,78	41,0 (26 -90)
Triglycerides (mmol/L)	8,41 ± 3,78	7,42 (2,39 – 19)
HDL (mmol/L)	2,54 ± 1,25	2,25 (1,44 – 10,84)
FBS (mg/dL)	155,30 ± 77,55	127,5 (12 – 369)
Insulin (µU)	24,05 ± 37,80	14,21 (3,7 – 263,02)

WC waist circumference; BMI, body mass index; HDL, high-density lipoprotein-cholesterol; FBS fasting blood sugar; HOMA - IR homeostasis assessment of insulin resistance; QUICKI quantitative insulin sensitivity assessment index; VAI, visceral adiposity index; SD (standard deviation); min (minimum); max (maximum).

The results of data analysis with the Pearson test showed a moderate positive correlation between the number of VAI and HOMA-IR (p=<0.001; r=0.480) and a moderate negative correlation between VAI and QUICKI (p=<0.001; r=-0.475). The distribution of VAI data with HOMA-IR and QUICKI can be seen in Figure 1.



Correlation VAI and HOMA-IR



Correlation VAI and QUICKI

Figure 1. Scatter plot graph of VAI correlation with HOMA-IR and QUICKI

DISCUSSION

This study provides the results of a moderate positive correlation between VAI and HOMA-IR and a moderate negative correlation between VAI and QUICKI. The results of the correlation test between HOMA-IR and QUICKI show that there is a robust negative correlation.

This correlation between VAI and HOMA-IR aligns with a previous study on participants with average abdominal circumference, which showed that VAI is

closely related to HOMA-IR and is an independent risk factor in increasing HOMA-IR in both men and women.[8]

Aside from being an anthropometric parameter, VAI is also an indicator of fat, so it can be a helpful indicator in fat loss management (with lifestyle intervention or medication) to prevent and treat cardiometabolic diseases. The association of obesity to insulin resistance is more prominent, where populations with BMI >30kg/m2 will experience 4-fold more excellent insulin resistance. Elevated TG is associated with decreased insulin sensitivity, and both these abnormalities and low HDL-C are associated with insulin resistance.[10]

Another study by Mazzuca et al with a population of 528 reported that increased VAI was associated with insulin resistance.[11] Parven et al in their study of 439 Bangladeshi population, reported VAI had a positive correlation with HOMA-IR in patients with Type 2 DM but did not show a significant correlation in controls.[3]

Previous study by Yi et al. showed that visceral fat has a link with insulin resistance calculated by HOMA IR and QUICKI and metabolic syndrome, which has a better correlation in pre-diabetic 33 men and patients with type 2 DM population.[12] Another study by Vizzuso et al. reported a significant correlation between VAI, HOMA-IR, and QUICKI in a population of Caucasian children aged 8-15 years with metabolic syndrome.[13] Hernandez et al concluded a moderate correlation between VAI, HOMA- IR, and QUICKI in 396 obese children in Mexico.[14]

Several previous studies have 30 monstrated the correlation between visceral adipose tissue and insulin resistance. A meta-analysis study by Zhang et al. r 29 rted a significant positive association between adipose tissue build-up and insulin resistance as measured by HOMA-IR. The visceral fat mass is closely correlated with HOMA-IR, followed by total fat mass, BMI, and WC.[6]

Sun et al. said there is a correlation between visceral fat in several measurement methods and the risk of diabetes and insulin resistance.[2] Borel et al. showed that changes in visceral adipose tissue were associated with improved insulin sensitivity after one year of lifestyle intervention.[15] An increased risk of diabetes may result from excess visceral fat. Visceral fat has more excellent endocrine activity than subcutaneous fat and is a marker of adipose tissue dysfunction and ectopic fat deposition. These circumstances cause lipotoxicity and insulin resistance in muscle cells, liver, and pancreatic cells that inhibit glucose uptake. Therefore, visceral fat contributes as one of the risk factors for diabetes.[12]

Insu 2 n resistance measurement with HOMA-IR was done more than 15 years ago.[5] HOMA-IR has been observed to have a linear correlation with glucose clamp and minimal model in estimating insulin sensitivity/resistance in 2 arious studies in different populations. In contrast, QUICKI is the logarithm of HOMA-IR, which explains its almost perfect correlation with HOMA.[16] This is consistent with this study, where a very strong correlation was found between HOMA-IR and QUI 31 I. Katz et al., in their study, also mentioned that HOMA-IR and QUICKI have a better correlation than HOMA-IR 14 th glucose clamp.[17]

Insulin resistance occurs due to impaired insulin action in metabolically active tissues and organs, including skeletal muscle, liver, and fat tissue.[12] In insulin

resistance, the effect on adipose tissue is an increased hepatic free fatty acid flow that tends to increase hepatic very low-density lipoprotein (VLDL) production. At the same time, ketogenesis remains suppressed due to compensatory hyperinsulinemia.[8] Fat-induced insulin resistance, where there is decreased fat storage capacity within the subcutaneous adipose tissue, will lead to ectopic fat deposition into visceral fat tissue and insulin-sensitive tissues such as liver and skeletal muscle. These tissues will progressively develop to lipotoxicity status, disrupt insulin signaling and action, and cause insulin resistance and decreased glucose tolerance. Insulin resistance increases as BMI, WC, and especially waist-hip ratio increases. This reflects an increase in adiposity and incredibly visceral adipose tissue.[2,18]

The visceral adiposity index was developed as a novel indicator of visceral adipose tissue dysfunction that proved to be a good indicator of endocrine dysfunction and low-grade inflammation of adipose tissue in a state referred to as adipose tissue dysregulation.[19] Adipose tissue dysregulation is a state characterized by altered fat distribution and function and is believed to be a cornerstone in the pathogenesis of insulin resistance through altered adipocytokine production, increased lipolytic activity, and inflammation.[20]

CONCLUSION

There was a moderate positive correlation between the number of VAI and HOMA-IR and a moderate negative correlation between VAI and QUICKI. The results of the correlation test between HOMA-IR and QUICKI show that there is a very strong negative correlation. Further studies are needed to evaluate the association between VAI and insulin resistance and visceral fat accumulation by CT or MRI

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