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Association Between Visceral Fat and Monocyte Count in Type 2 Diabetes Mellitus: Evidence from a Referral Hospital Study

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Abstract

The majority of patients with type 2 diabetes mellitus (T2DM) exhibit central obesity. Central obesity is characterized by an abnormally high accumulation of visceral adipose tissue. Individuals with both obesity and T2DM often present with additional immune dysfunction, including significantly elevated monocyte counts compared to metabolically healthy obese individuals. A recent study has demonstrated a correlation between visceral fat and monocyte count in young adults with obesity; however, this association remains unclear in patients with T2DM. A cross-sectional observational study was conducted using a total sampling design from August to September 2022 at AMC Hospital, Indonesia. History taking, physical examination, and laboratory examination were conducted. Visceral fat, monocyte count, and confounders (age, gender, diagnosis duration of T2DM, glycemic control (using FBG), BMI, WC, number of comorbidities, and number of drug use) were observed. In the complete sample (n = 57), after adjusting for confounding variables, there was no statistically significant association between visceral fat and monocyte count (adjusted β = 86.635, 95% CI [–19.050, 192.321], P = 0.106). However, after excluding one influential subject and controlling for confounders, a significant association was observed (adjusted β = 110.023, 95% CI [11.111, 208.935], P = 0.030). There was a significant association between visceral fat and monocyte count in T2DM.

Keywords: Type 2 diabetes mellitus; visceral fat; monocyte count.

Abbreviations: T2DM: Type 2 Diabetes Mellitus; FBG: Fasting Blood Glucose; BMI: Body Mass Index; WC: Waist Circumference; CI: Confident Intervals

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a major global public health issue and one of the most common metabolic disorders (Galicia-Garcia et al., 2020; Wu et al., 2014). Worldwide, T2DM accounts for over 90% of all diabetes cases (IDF, 2021). In Indonesia, the number of people with T2DM is projected to rise from 8.4 million in 2000 to 21.3 million by 2030 (PERKENI, 2019). In West Java, Bandung Regency ranks 9th in terms of T2DM prevalence (Dinkes, 2019).

Central obesity, along with other metabolic disorders, contributes to a condition known as metabolic syndrome, which increases the risk of diabetes mellitus (DM), cardiovascular disease (CVD), and various other metabolic-related diseases (Błaszczyk-Bębenek et al., 2019; Owolabi et al., 2017). Central obesity plays a significant role in the development of chronic low-grade inflammation (van Greevenbroek et al., 2013). The excessive deposition of visceral fat in central obesity leads to both local and systemic immune dysfunction (de Frel et al., 2020). In the local adipose tissue, there is a shift from anti-inflammatory to pro-inflammatory immune cells, such as the transformation of macrophages from the M2 to the M1 phenotype (de Frel et al., 2020; Kaplan et al., 2015).

Chronic hyperglycaemia in T2DM leads to glucotoxicity, which promotes lipogenesis in the liver and increases the production of very low-density lipoprotein (VLDL) (Nesto, 2005). However, the production of lipoprotein lipase (LPL) is reduced or even inhibited, which may lead to a reduction in visceral fat (Nesto, 2005). Despite this, patients with T2DM typically have high levels of visceral fat, especially those with central obesity (van Greevenbroek et al., 2013). Furthermore, chronic hyperglycaemia also induces the expansion and proliferation of bone marrow myeloid progenitors, resulting in an increased number of monocytes in circulation (Nagareddy et al., 2013). As a consequence, patients with T2DM are more susceptible to infections (Berbudi et al., 2020).

MATERIALS AND METHODS

Study area

This was a cross-sectional observational study with total sampling design. The study was conducted from August 2022 to September 2022 at Annisa Medical Center (AMC) Hospital, Kabupaten Bandung, West Java, Indonesia. AMC is one of the private hospitals with secondary referral status in Kabupaten Bandung which is located in the east of Bandung City. The study protocol was approved by the Ethical Committee of Universitas Padjadjaran (Number: 054/UN6.KEP/EC/2022). The written informed consent was obtained from all participants.

All participants aged 20 to 79 years old who were diagnosed with T2DM were included in this study. Participants were excluded if they were diagnosed with Chronic Kidney Disease (CKD), malignancy, pregnancy, osteoarthritis, retinopathy, autoimmune disease, Chronic Obstructive Pulmonary Disease (COPD), taking anti-inflammatory medication, and had a known infection at the time of blood test. The study participant selection was conducted through history and medical records.

G*power version 3.1.9.7 for Windows was used to help calculate sample size for linear regression analysis (Faul et al., 2007). The probability type I and II errors were determined as 0.05 and 0.2. The number of predictors and the magnitude of the determination coefficient (R^2) were determined at 9 and 0.25. Based on the output of the software, it was obtained that the minimum sampe size were 56 participants.

Procedures

All eligible participants treated at AMC Hospital from August to September 2022 were recruited in this study. After the informed consents were obtained from the participants, data including age, duration of T2DM diagnosis, history of current illness leading to infection, pregnancy status (for women), and comorbidities were recorded during history taking. Anthropometric measurements included height, weight, and waist circumference (WC) were then conducted. The visceral fat was measured (Omron Karada Scan HBF-375) and venous blood were drawn for measurement of fasting plasma glucose (Agappe Mispa CCXL), routine blood test, and differential blood count (Sysmex XNL 330). The medical records were checked to confirm the participant's replies during history taking

Data analysis

The data were analysed using IBM SPSS (Statistical Package for Social Sciences) version 27.0 software for Windows. Numerical variables were presented as mean and standard deviation or median and interquartile range (IQR) depend on the normality of distribution, whereas categorical variables ware presented as frequency and percentages. A simple linear regression was conducted to analyse the association between visceral fat and monocyte count in T2DM. A Multiple linear regression was used for adjusting confounding variables (age, sex, gender, diagnosis duration of T2DM, glycemic control, body mass index (BMI), WC, number of comorbidities, and number of drug use). Diagnostic procedures for checking the assumptions of linear regression were performed. Linearity, residual constant variance, and normality of residual were met. No outliers with respect to outcome and predictor variables were observed. For observation with the largest residual (id 49: a male, 55 years old, had normal visceral fat, and had the 2nd highest monocyte count), it was observed that had large influence on the regression coefficient for visceral fat level. Hence, two linear regression models were constructed by including or excluding id 49. All statistical tests were performed at the significance level of 5%.

RESULTS AND DISCUSSION

A total of 120 participants were screened for the study. Of these, 63 were excluded for the following reasons: 31 had chronic kidney disease (CKD), 1 had ovarian cancer, 1 was pregnant, 5 had osteoarthritis, 2 had retinopathy, 8 showed symptoms suggestive of infection, and 15 had a neutrophil-to-lymphocyte count ratio (NCLR) greater than 3. As a result, 57 participants were deemed eligible for inclusion in the study. Baseline characteristics of the participants are presented in Table 1.

Table 1. Baseline characteristic of study participants.

Characteristics	n= 57
Visceral Fat (count), mean (SD)	11.62 (4.54)
Visceral fat, frequency (%)	
Normal	22 (38.6)
High-very high	35 (61.4)
Monocytes (cell count/μL), mean (SD)	523.11 (168.1)
Gender, frequency (%)	
Male	20 (35.1)
Female	37 (64.9)
Age (year), mean (SD)	54.54 (8.39)
Diagnosis duration of T2DM (months), median (IQR)	36 (54)
Diagnosis duration of T2DM, frequency (%)	
<5 years	35 (61.4)
≥5 years	22 (38.6)
Fasting blood glucose (mg/dL), median (IQR)	131 (39)
Glycemic control, frequency (%)	
Controlled	28 (49.1)
Uncontrolled	29 (50.9)
BMI (kg/m^2), mean (SD)	27.11 (4.15)
BMI category, frequency (%)	
Underweight	0 (0.0)
Normal	7 (12.3)
Overweight	9 (15.8)
Obese I	30 (52.6)
Obese II	11 (19.3)
WC male (cm), mean (SD)	89.31 (8.24)
WC female (cm), mean (SD)	86.30 (10.22)
Number of comorbidities (count), median (minimum,maksimum)	2.00 (0,4)
Number of drug use (count), median (minimum,maksimum)	4.00 (1,6)

Note: BMI, body mass index; Circumference; SD, standard deviation; IQR, interquartile range;
Comorbidities: hypertension, coronary artery disease, chest pain, arrhytmia, decompensated cardiomyopathy, asthma, neuropathy, dyslipidemia, stroke, congestive heart failure, cataract, cholelitihiasis.

Based on the bivariable analysis using complete observations, there was a statistically significant association between visceral fat and monocyte count. Participants with high to very high levels of visceral fat had an average monocyte count that was 106.54 units higher than those with normal visceral fat levels are presented in Table 2.

However, in the multivariable analysis using complete observations and controlling for potential confounding variables, the association between visceral fat and monocyte count was not statistically significant (adjusted β = 86.635, 95% CI: –19.050 to 192.321; P = 0.106). When one influential outlier (ID 49) was excluded from the analysis, and after adjusting for confounders, a significant association was observed (adjusted β = 110.023, 95% CI: 11.111 to 208.935; P = 0.030) are presented in Table 3 and Table 4.

Table 2. Association between visceral fat and monocyte count (crude analysis) with complete observation.

Effect	β	SE	T	P-value	95% CI for β
Constant	457.68	34.382	13.254	<0.001	(388.780,526.584)
Visceral fat (High - Very high vs Normal)	106.54	43.876	2.428	0.018	(18.617,194.477)

Note**:** β = Regression coefficients; SE= *Standard Error*; CI= Confidence Interval

Table 3. Association between visceral fat and monocyte count after controlling for age, gender, diagnosis duration of T2DM, glycemic control, number of comorbidities, and number of drug use with complete observation.

Effect	β	SE	T	P-value	95% CI for β
Constant	462.871	169.89	2.724	0.009	(121.457,804.284)
Visceral fat (High -Very high vs Normal)	86.635	52.59	1.647	0.106	(-19.050,192.321)
Age	-0.348	2.70	-0.129	0.898	(-5.777,5.080)
Gender (Male vs Female)	46.093	53.70	-0.145	0.395	(-61.828,154.015)
Diagnosis duration of T2DM (<5years vs ≥5 years)	26.796	47.44	0.569	0.572	(-68.370,122.316)
Glycemic control (Controlled vs Uncontrolled)	26.796	55.77	0.480	0.633	(-85.286,138.879)
Number of comorbidities	-16.607	27.78	-0.598	0.553	(-72.446,39.232)
Number of drug use	4.241	20.11	0.211	0.834	(-36.185, 44.668)

Note: β = Regression coefficients; SE= Standard Error; CI= Confidence Interval; BMI= Body Mass Index; WC= Waist Circumference

Table 4. Association between visceral fat and monocyte count after controlling for age, gender, diagnosis duration of T2DM, glycemic control, number of comorbidities, and number of drug use without id 49.

Effect	β	SE	T	P-value	95% CI for β
Constant	387.636	158.905	2.439	0.018	(68.137,707.135)
Visceral fat (High -Very high vs Normal)	110.023	49.194	2.236	0.030	(11.111,208.935)
Age	0.110	2.501	0.044	0.965	(-4.918, 5.138)
Gender (Male vs Female)	14.661	50.676	0.289	0.774	(-87.230,116.553)
Diagnosis duration of T2DM (<5years vs ≥5 years)	56.543	44.892	1.260	0.214	(-33.719,146.806)
Glycemic control (Controlled vs Uncontrolled)	16.346	51.652	0.316	0.753	(-87.509,120.200)
Number of comorbidities	-24.182	25.796	-0.937	0.353	(-76.048,27.684)
Number of drug use	15.668	18.960	0.826	0.413	(-22.455, 53.790)

Note: β = Regression coefficients; SE= Standard Error; CI= Confidence Interval; BMI= Body Mass Index; WC= Waist Circumference

Discussion

This study aimed to analyse the association between visceral fat and monocyte count in patients with Type 2 Diabetes Mellitus. The results revealed a difference in monocyte count between patients with high to very high levels of visceral fat and those with normal levels of visceral fat.

Visceral fat (epicardial fat and abdominal fat) is adipose tissue that surrounds the internal organs (myocardia and gastrointestinal) which is metabolically active, thereby capable of secreting bioactive molecules and hormones that affect normal and pathological processes in the human body (Chait & den Hartigh, 2020; Liang et al., 2018; Nauli & Matin, 2019). Each visceral adipose tissue (VAT) consists of adipocytes and the stromal vascular fraction (SVF) (Khan et al., 2020). SVF consists of an extracellular matrix (ECM) that binds together various cells including preadipocytes, fibroblast, stem cells, vascular endothelial cells, and immune cells. Under homeostatic condition, the VAT contains a group of macrophages known as Adipose Tissue Macrophages (ATMs), which comprises about 5-10% of the SVF (Khan et al., 2020).

The abnormal accumulation of VAT is known as visceral obesity while the excessive deposition of abdominal VAT is called central obesity (Nauli & Matin, 2019; Shuster et al., 2012). Both of them may lead to several pathological conditions such as impaired glucose metabolism and insulin resistance (Shuster et al., 2012). The first immune cell to be characterized inside VAT during obesity are macrophages (Khan et al., 2020). During obesity, there is an increase of accumulation of ATMs up to 40-50% of the SVF, displays an M1 phenotype, become metabolically activated, secrete pro-inflammatory mediators, including TNF-α, IL-1β, and inducible nitric oxide synthase (Khan et al., 2020; Nesto, 2005).

The accumulation of ATMs are the result through two main processes. Resident tissue macrophages and the adipocytes increases the levels of monocyte chemoattractant protein 1 (MCP1) which would promote recruitment of blood monocytes. Consequence of blood monocytes migration under inflammatory condition cause the accumulation of ATMs (Khan et al., 2020; Nesto, 2005). In addition, this accumulation of ATMs may also be due to local proliferation of macrophages. Thus, in obesity, both local and systemic immune dysfunction occur due to metabolic stress, such as in monocytes and macrophages which can lead to chronic low-grade inflammation that can interfere normal insulin function and thereby lead to insulin resistance and beta cell dysfunction (de Frel et al., 2020).

T2DM is chronic metabolic disorder characterised by persistent hyperglycaemia due to a combination of two main factors: β-cell dysfunction and tissue insulin resistance, including the adipose tissue, liver, and

skeletal muscle (Banday et al., 2020). Initially, decrease in insulin-stimulated glucose uptake triggers β-cells of pancreatic islet hyperfunction as a compensatory to maintain normoglycemia by increasing insulin secretion. However, when β-cells fail to compensate sufficiently for the insulin resistance, β-cell function begins to decline and lead to β-cell dysfunction which cause insulin deficiency and result in hyperglycaemia (Banday et al., 2020; Nesto, 2005).

This hyperglycaemic condition results in four main metabolic changes, including activation of protein kinase C isoforms, increased hexosamine and polyol pathway flux, and increased advanced glycation end-product formation. These four metabolic changes induce an increased production of superoxide, which activates inflammatory pathways and can lead to immune system dysfunction (Nesto, 2005).

Chronic hyperglycaemia is greatly associated with monocyte dysfunction, such as; increased monocyte circulation that results from hyperglycaemia induced proliferation and expansion of bone marrow myeloid progenitors, impaired chemotaxis, reduced glycolytic capacity and Fc gamma receptors on monocytes (Berbudi et al., 2020; Nagareddy et al., 2013). All these findings confirm the impaired functions of monocytes during the T2DM progression.

Chronic hyperglycaemia also alters function of neutrophil, suppression of cytokine production, dysfunction of natural killer cell (Błaszczyk-Bębenek et al.), suppression of regulatory T cells and inhibition of complement effector and antibody (Berbudi et al., 2020; Nesto, 2005). Thus, dysfunction of both innate and adaptive immunity occurs which then causes the impaired immune system to invade pathogens in T2DM (Aiello et al., 2019; Villar-Álvarez et al., 2022).

In general, innate immunity in older people is up-regulated in some functions and down-regulated in others such as increase in circulating monocytes and the number of NK cells, reduced function of neutrophils in bacteria phagocytosis and in the oxidative burst, reduced chemotaxis and phagocytosis of macrophage, and decreased cytokine production (Aiello et al., 2019). In addition, aging also alters the quality and quantity of the T and B cell responses such as decrease in the number of peripheral naïve T and B cells (Aiello et al., 2019).

Weight management remains challenging for patients with T2DM (Pi-Sunyer, 2009). Patients with T2DM are at risk for weight gain as a result of multiple factors, including high calorie diets, sedentary lifestyle, and diabetic medications (Siram et al., 2010). In addition, major therapeutic classes of medications used for T2DM, such as insulin, sulfonylureas, thiazolidinediones (TZD), and meglitinides have been associated with significant weight gain and worsening pre-existing obesity (Provilus et al., 2011; Siram et al., 2010).

The prevalence of both central obesity and T2DM are increasing in younger subjects, there is a long time for these chronic diseases develops and cause immune dysfunction (Berbudi et al., 2020; CDC, 2021; Wong et al., 2020). It is caused by a chronic exposure to an abnormal metabolic conditions such as adipose tissue inflammation and hyperglycaemic conditions which resulting in continued exposure to low grade inflammation, increased oxidative stress, and glucolipotoxicity (Nesto, 2005). Thus, these lead to persistent dysregulation in innate immunity such as monocyte function and adaptive immunity (Nesto, 2005).

That individuals with obesity and type 2 diabetes are associated with additional perturbations in the functionally immune system such as significantly higher monocytes compared to obese individuals that are metabolically healthy (Richard et al., 2017).

CONCLUSIONS

Visceral fat was significantly associated with elevated monocyte counts in T2DM, indicating immune dysfunction driven by chronic inflammation and hyperglycaemia. This may increase infection risk in centrally obese diabetic patients. Understanding these mechanisms is key to developing targeted strategies to prevent infection and improve patient outcomes

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