Correlation between Obesity and Lipid Profile in Type 2 Diabetes Mellitus Patients at the Endocrine and Metabolic Polyclinic in General Hospital Pirngadi Medan

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Abstract

BACKGROUND: Obesity is a multifactorial disease that is dangerous and is a factor in the emergence of serious diseases such as dyslipidemia, stroke, coronary heart disease and others. In Type 2 Diabetes Mellitus (T2DM) patients there is a disorder of lipid metabolism, namely dyslipidemia. Changes in lipid profile that occurred were an increase in total cholesterol levels, Low-Density Lipoprotein (LDL), and triglycerides, and decreased levels of high-density lipoprotein (HDL). The phenomenon of an increase in T2DM patients in Indonesia caused double mortality in recent decades.

AIM: This study was to determine the relationship between obesity and lipid profile in T2DM patients at Pirngadi Medan Hospital in 2018.

METHODS: This study was conducted in an observational analytic with a cross-sectional study approach. Fifteen obese patients with T2DM who were treated at the Endocrine and Metabolic Polyclinic in Pirngadi General Hospital Medan from January to December 2018 were recruited into the study sample.

RESULT: Based on the analysis using the results of a one-way correlative analytical test showing that there was a positive correlation between obesity and total cholesterol levels ($r = 0.209$; $p = 0.455$) and LDL levels ($r = 0.335$; $p = 0.222$) but not significant. There was a negative correlation between obesity and HDL levels ($r = -0.072$; $p = 0.798$) and triglyceride levels ($r = -0.025$; $p = 0.930$) but not significant. There was no significant relationship between obesity and blood glucose levels ($r = 0.463$; $p = 0.082$). This study concluded that there was no significant relationship between obese patients and lipid profiles in T2DM patients.

CONCLUSION: Obesity positively correlates with blood glucose level, but its correlation with a lipid profile is not reliable.

Introduction

Diabetes Mellitus (DM) is a group of metabolic diseases characterised by hyperglycemia due to defects insulin work in the liver and peripheral tissues, insulin secretion from pancreatic beta cells, or both [1]. Nutritional status influences the incidence of Type 2 DM (T2DM). High BMI (Body Mass Index) has a 2 times greater risk of developing T2DM compared to low BMI. The results showed that general obesity had a risk of 2.24 times while abdominal obesity had a risk of 2.44 times for the occurrence of DM [2].

In T2DM patients, abnormalities of lipid metabolism can be found in the form of dyslipidemia. Dyslipidemia is a disorder of lipid metabolism characterised by an increase or decrease in lipid fraction in the plasma. The main lipid fraction abnormalities include increases in total cholesterol, triglycerides, Low-Density Lipoprotein (LDL), and decreased High-Density Lipoprotein (HDL). Dyslipidemia caused by DM is secondary dyslipidemia [3], [4].

Since 1980 the incidence of obesity in the epidemic has begun to increase. Where currently, more than 30% of the US population is obese with a

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Body Mass Index (BMI) more than 30 kg/m² and almost two-thirds are overweight (BMI between 25 and 29.9 kg/m²) [4], [5].

In Indonesia, the incidence of obesity continues to increase. Where in adult men there was an increase from 13.9% in 2007 to 19.7% in 2013? Whereas in adult women there was a very high increase reaching 18.1%, from 14.8% in 2007 to 32.9% in 2013 [6].

Obesity is a risk factor for developing insulin resistance and type 2 diabetes. In 2015, an estimated 2.3 billion adults will experience overweight, and 700 million of them will be obese. Significant relationship exists between per cent body fat and body weight in DM, besides that the prevalence of diseases associated with insulin resistance increases together with increasing BMI because an increase in adipose tissue is characterised by decreased HDL and increased triglycerides [7].

Obesity is a multifactorial disease that rises very sharply throughout the world which reaches dangerous levels and is a factor for the emergence of serious diseases including hypertension, stroke, dyslipidemia, coronary heart disease, and type 2 diabetes [8]. Based on the background described, the researchers were interested in examining the relationship between obesity and lipid profile in patients with type 2 diabetes.

Material and Methods

This study was conducted in an observational analytic study with a cross-sectional approach. In this study, no follow-up was carried out on the measurements taken. This study was conducted on a set of objects, within a certain period aimed at knowing the relationship of obesity and lipid profile in patients with type 2 diabetes mellitus. Fifteen obese patients with type 2 DM who were treated at the Endocrine and Metabolic Polyclinic in Pirngadi General Hospital Medan from January to December 2018 were recruited to become a research sample.

Inclusion criteria were m en and women who suffer from obesity (BMI > 25 kg/m² or obesity I and obesity II), adults over 25 years, and willing to give written permission after being given informed consent to take part in this research

Exclusion criteria were patients who are not cooperative, patients suffering chronic diseases, the patient's health condition is not possible to take part in this study, Patients are outside the city so they cannot take part in the study according to the set schedule

The variables examined in this study were the independent variable was Obesity (Obesity I and Obesity II). The dependent variables were lipid profiles (total cholesterol, HDL, LDL and triglycerides).

In this study, the patient's lipid profile data were obtained from the report of the attached laboratory examination results and the method of measurement by looking at the lipid profile data from the laboratory examination in units of mg/dl. Obesity is measured by the measurement body Mass Index (BMI) (body weight kg/body height m^2).

Statistical analysis

Data were analysed through statistical calculations to test hypotheses using the Pearson correlation test method. If the normality test was found that the data is not normally distributed, the hypothesis test could be done using the Spearman correlation test method. P value considered significant if it is less than 0.05

Results

The 15 diabetic patients who were divided into 4 men and 11 women in the Internal Medicine Department at the Endocrine and Metabolic Polyclinic in General Hospital Pirngadi Medan, the characteristics in Table 1.

Table 1: Basic characteristics of research subjects

Characteristics	Mean ± SD
Age (year)	56.733 ± 10.271
Body weight (kg)* (Md, Min, Max)	77.533 (75,65,106)
Body height (cm)	154.00 ± 7.156
Cholesterol total	230.2 ± 45.896
HDL	53.6 ± 13.69
LDL	128.6 ± 38.52
Triglycerides	240.86 ± 219.10
IMT	32.83 ± 3.05
Blood Glucose	209.06 ± 84.11
Systole	138 ± 14.735
Diastole	86.66 ± 9.75

Md: Median, Min: minimum, Max: maximum.

Based on the results obtained from the correlative analytic test found a positive correlation between obesity and Total Cholesterol levels (r = 0.209; p = 0.455) and LDL levels (r = 0.335; p = 0.222) but not significant. There was a negative correlation between obesity and HDL levels (r = -0.072; p = 0.798) and triglyceride levels (r = -0.025; p = 0.930) but not significant. There was no significant relationship between obesity and blood glucose levels (r = 0.463; p = 0.082), (Table 2).

Table 2: The Correlation between obesity and lipid profiles

Variable	r value	P value
Cholesterol total	0.209	0.455
HDL	-0.072	0.798
LDL	0.335	0.222
Triglycerides	-0.025	0.930
Glucose	0.463	0.082

P value considered significant if it is less than 0.05.

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In Table 3 there was a positive relationship between obesity and systolic blood pressure (r = 0.213) but not diastole (r = -0.226).

Table 3: Correlation of obesity with blood pressure

Group	Obesity	
	r value	P value
Sistole	0.213	0.446
Diastole	-0.226	0.418

Discussion

In this study, 15 samples were obtained that met the inclusion and exclusion criteria. The sample consisted of 4 men (10%) and 11 women (90%). This finding is by the opinion of Brunner and Suddart (2002) who stated that women suffer from diabetes mellitus more than men. This is triggered by the presence of a greater percentage of body fat in women compared to men which is one of the factors that can reduce sensitivity to the workings of insulin in the muscles and liver [9].

Fluctuations in estrogen levels can affect blood glucose levels. When estrogen levels increase, the body can become resistant to insulin [10]. Irawan (2010) stated that menopause causes body fat distribution to be easily accumulated due to these hormonal processes so that women are at risk of suffering from T2DM [11].

The average BMI of the patient is 32.83. The average total cholesterol level was 230.2 gr/dl, 53.6 gr/dl HDL levels, 128.6 gr/dl LDL levels, average triglycerides 240.86 gr/dl and the average blood glucose level 209.06 mg/dl (Table 1).

To find out the relationship between obesity and lipid profile in patients with type 2 diabetes, a one-way correlative analytic test was performed. The correlative analytic test results between obesity and lipid profile (total cholesterol, HDL, LDL and triglyceride) and blood glucose in table 2. The results obtained showed a positive correlation between obesity levels and total cholesterol levels (r = 0.209; p = 0.455) and LDL (r = 0.335; p = 0.222). There was a positive relationship between the two but not significant. There was a negative correlation of HDL levels (r = -0.072; p = 0.798) and triglyceride levels (r = -0.025; p = 0.930) but not significant. With a p-value considered significant, it is less than 0.05. This is not by the theory that the most frequent picture of dyslipidemia in type 2 DM is a decrease in HDL levels and an increase in triglyceride levels by the theory [12].

In this study, there was a positive correlation between obesity and blood glucose levels (r = 0.463; p = 0.082). The results of this study are not much different from previous studies conducted by Jin Ook Chung, Dong Hyeok Cho, Dong Jin Chung, and Min Young Chung (2012) in the Associations among Body Mass Index, Insulin Resistance, and Pancreatic-Cell Function in Korean Patients with New-Onset Type 2 Diabetes. This study showed that there was a significant relationship between BMI and the occurrence of insulin resistance which caused an increase in fasting blood glucose levels, p < 0.05 [13].

The results of other studies were also conducted by Ninh T. Nguyen, Xuan-Mai T Nguyen, John Lane, and Ping Wang (2011) in Relationship Between Obesity and Diabetes in US Adult Population: Findings from the National Health and Nutrition Examination Survey, 1999-2006 showed a significant relationship between obesity and the occurrence of type 2 diabetes mellitus [14]. The results of this study meant that the greater body mass index value, the greater the fasting blood glucose value. The greater body mass index value means the patient leads to obesity. This was accordance with the theory of Suyono (2011), that the risk factors of type 2 diabetes mellitus are overweight/obesity factors which include lifestyle changes from traditional to the western lifestyle, overeating, and relaxed life (lack of motion) [15].

In the digestive system, food is broken down into the basic ingredients of the food itself. Carbohydrates become glucose, proteins become amino acids, and fats become fatty acids. The three food substances will be absorbed by the intestine and then enter the blood vessels and circulated throughout the body to be used by organs as fuel. To function as a fuel, food cells, especially glucose, must be metabolised first. In the metabolic process, insulin plays an important role, which is to enter glucose into cells, which can then be used as fuel [15], [16].

In normal circumstances, it means that insulin levels are enough and sensitive, insulin will be captured by insulin receptors that are on the cell surface, and then open the cell entrance, so that glucose can enter the cell and then burned into energy. As a result, blood glucose levels become normal [15]. This is different in the state of obesity, an increase in mRNA Lipopolysaccharides (LPS) - induced TNF-α factor (LITAF) and protein levels along with increased BMI indicate a parallel relationship between LITAF and metabolic disorders.

According to the study, LITAF is activated in obese patients and contributes to the development of obesity which induces inflammation and insulin resistance, since LITAF plays a role in the inflammatory process in regulating the expression of TNF-α, IL-6 and MCP-1 which results in insulin resistance, and TLR4. One of the LITAF receptors in macrophages can also be stimulated by free fatty acids which can cause an inflammatory process in obese patients. LITAF is a TNF-α description of the regulator that should play a role in the immune mechanism against infection. The LITAF gene is

located at 16p13.13 which is significantly present in lymph, lymph nodes, and peripheral blood leukocytes. TNF-α is a strong trigger for proinflammatory cytokines such as IL-6, MCP-1, leptin and PAI-1. It is very involved in the inflammatory process in obese patients. The increase in TNF-α observed in fat tissue in obese patients shows a direct relationship to the emergence of insulin resistance in obese patients [17]. The occurrence of insulin resistance causes glucose circulating in the blood to be unable to enter the cell, so the sugar level in the blood becomes higher than normal [15].

Hyperglycemia in people with diabetes mellitus is also closely related to fat metabolism. Fat has the main task of storing energy in the form of triglycerides through the process of lipogenesis which occurs in response to excess energy and mobilises energy through the process of lipolysis in response to energy shortages. Under normal circumstances, these two processes are strictly regulated [18].

The condition of obesity is caused by excessive intake of nutrients continuously causing fat deposits to become excessive. Deposits of fatty acids in the form of chemical compounds in the form of triacylglycerol contained in adiposity cells can protect the body from the toxic effects of fatty acids. Freeform fatty acids can circulate in blood vessels throughout the body and cause oxidative stress which we are familiar with lipo-toxicity. The emergence of lipo-toxicity effects caused by several free fatty acids released by the triacylglycerol to compensate for the destruction of excessive fat deposits affects the adipose and non-adipose tissue and plays a role in the pathophysiology of diseases in various organs such as the liver and pancreas. This release of free fatty acids from excessive triacylglycerol can also inhibit fat synthesis and reduce the clearance of triacylglycerol. This can increase the tendency of hypertriglyceridemia.

The release of free fatty acids by endothelial lipoprotein from triglycerides which increases in the increase of lipoprotein β causes lipo-toxicity which also interferes with the function of insulin receptors. The consequence of insulin resistance is hyperglycemia, which is compensated by glucose synthesis from the liver (gluconeogenesis), which contributes to aggravating hyperglycemia.

Free fatty acids also contribute to hyperglycemia by reducing glucose use from insulin-stimulated muscles. Lipotoxicity due to excess free fatty acids also decreases insulin secretion from pancreatic β cells, which ultimately β cells will experience fatigue [19]. The weakness of this study is a cross-sectional study that has limitations: 1. Not all confounding variables can be controlled properly so that better follow-up research is needed with appropriate research designs. 2. This study is based on local state hospitals so that to generalise to the population of type 2 diabetes mellitus with obesity globally should be wiser. 3. The number of samples is limited so that more samples are needed. Further research is needed with better research designs and with more and varied samples to prove the relationship between Obesity and lipid profiles in patients with type 2 DM.

Acknowledgement

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