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Research Article

Differences between Lipid Profile and Blood Pressure in Diabetes Mellitus Type 2

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ABSTRACT

Dyslipidemia can occur together with diabetes mellitus type 2. Diabetes or impaired glucose tolerance with hypertension was characterized by low HDL, high LDL, and high VLDL levels. The purpose of this study was to determine whether there were differences in lipid profiles with blood pressure in type 2 diabetes mellitus patients. This study was an observational analytic study with a cross-sectional approach conducted at general hospital Pirngadi Medan. The total sample was 15 patients with diabetes mellitus type 2. The research sample was taken step by step, namely *purposive sampling*, followed by *incidental sampling*. The research instrument used medical records and lipid profile data from laboratory examinations of diabetes mellitus type 2 from patients with hypertension and without hypertension. Based on the analysis using the results of a one-way correlative analytic test, it showed that there was a positive correlation between systolic blood pressure and total cholesterol levels ($p = 0.267$), glucose levels ($p = 0.967$), HDL levels ($p = 0.117$), LDL levels ($p = 0.634$) and triglyceride levels ($p = 0.361$) and negatively correlated with HbA1c ($p = 0.370$) but not significant. There was a positive correlation between diastolic blood pressure and total cholesterol levels ($p = 0.114$), HDL levels ($p = 0.174$), LDL levels ($p = 0.485$) and triglyceride levels ($p = 0.073$) and negatively correlated with HbA1C ($p = 0.248$), Glucose content ($p = 0.699$), but not significant. There was no relationship between HbA1C levels and the degree of hypertension, $p = 0.600$. This study concluded that there was no significant relationship between hypertensive patients and lipid profiles in people with T2DM, and there was no relationship between HbA1C levels and the degree of hypertension.

Keywords: Diabetes mellitus type 2, lipid profile, hypertension, dyslipidemia

INTRODUCTION

Diabetes mellitus (DM) is a severe health problem in Indonesia and the world. It continues to increase every year, causing high morbidity and mortality rates. In 2019, there were 463 million people in the world suffering from diabetes mellitus.[1] In Indonesia, Basic Health Research (Riskesmas) of the Health Ministry of the Republic of Indonesia reported an increase in the prevalence of DM sufferers in 2018, by 2% from 2013. DM patients mostly occurred in the age range of 55-64 and 65-74 years old. There were more women than men, and more often, there was more DM patient among those who live in urban areas than in rural areas.[2] Diabetes occurred together with hypertension, obesity, and hyperlipidemia is characterized by low levels of high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL).[3] Hyperlipidemia due to insulin resistance occurs because the liver produces a lot of apolipoprotein B (apoB) and VLDL. The Apolipoprotein CIII (apoCIII) will later cause more VLDL production.[4]

Insulin resistance plays an essential role in the pathogenesis of DM type 2, in the increase of pro-inflammation and dyslipidemia. It will disrupt insulin signaling, cause blood vessel endothelial disorders, and trigger arteriosclerosis as a risk factor for stroke and myocardial infarction.[5][6] Blood vessel dilation disorders are related to glucose delivery limitation to target organs due to the damaged endothelium and the aggravation of insulin resistance.[7][8] It is also common to find dyslipidemia in DM patients experiencing hypertension. However, Indonesia's latest data still lacks local incidents, especially the city of Medan, North Sumatra, Indonesia. This paper aims to explore the lipid profile and blood pressure of patients with DM type 2.

MATERIALS AND METHOD

This study is an analytical epidemiological study with a cross-sectional study approach. A cross-sectional is a momentary study in which the subject is observed briefly or only once for each respondent. The research was carried out in the

endocrine polyclinic of General Hospital Pirngadi Medan with a patient of diabetes mellitus type 2. The location of the examination was at the Clinical Laboratory of Doctor's Lab Medan.

This research aims to determine the relationship between lipid profiles and blood pressure in diabetes mellitus type 2 patients. It was conducted on a group of objects, within a certain period. The sample size was a minimum sample of fifteen hypertensive patients with diabetes mellitus type 2. Patients were treated in the Endocrine and Metabolic Polyclinic and were recruited to be the research sample at General Hospital Pirngadi Medan from December 2017 to January 2018.

The inclusion criteria were (1) men and women suffered from diabetes mellitus together with hypertension, (2) men and women suffered from diabetes mellitus without hypertension. Patients have been informed about the study before research initiation. Patients have been willing to participate and have given written permission (informed consent). The exclusion criteria of this study were (a) the patients were not in a healthful condition that allows them to participate in this study, (b) the patient was outside the city so that they could not follow the study according to the schedule that being set, (c) diabetes mellitus

patients with a history of kidney disease, (d) diabetes mellitus patients with a history of liver disease.

The independent variable of this study was diabetes mellitus patients with hypertension and without hypertension, while the dependent variable was the lipid profile (total cholesterol, HDL, LDL, triglycerides). In this study, the patient's lipid profile data were obtained from the accompanying laboratory examination report. Meanwhile, blood pressure in hypertensive patients was measured using a tensiometer. Data were analyzed through statistical calculations to test the hypothesis with the *Pearson correlation* test method. If the normality test shows that the data were not normally distributed, the hypothesis testing was conducted using the *spearman correlation* test method.

RESULTS AND DISCUSSION

From the fifteen diabetic patients, there were four men and eleven women who were outpatients in the Internal Medicine Section at the General Hospital Pirngadi Medan Endocrine and Metabolic Polyclinic. Outpatients characteristics data are presented in Table 1.

Table 1: Basic characteristics of research subjects

Characteristics	Mean ± SD
Age (year)	57.900±11.985
Weight (kg)* (Md, Min, Max)	70.00 (106.00)
Height (cm) * (Md, Min)	145.00 (165.00)
Total cholesterol	220.400±46.210
HbA1C	9.800±2.451
Glucose	223.800±78.605
HDL	54.200±12.532
LDL	130.300±32.225
Triglyceride	179.400±82.579
Systole	137.000±13.374
Diastole	86.000±9.660

Md: median, Min: minimum, Max: maximum

In this study, the data from Table 1 showed that characteristics of samples were elderly patients, mostly female, patients with uncontrolled glycemic status, dyslipidemia, and hypertension. The results showed that the age of 57 was suffered from diabetes mellitus type 2. This finding is in accordance with the data from Riskesda, which mentioned that Indonesian with an age range of 55-64 is suffered more often from diabetes mellitus type 2.[2] The risk factors for the elderly is more prone to diabetes mellitus type 2. It is

because of impaired insulin secretion in response to the incretin hormone, namely Gastric inhibitory polypeptide (GIP), increased insulin resistance, and impaired pancreatic islet function. Aging interferes with metabolism due to increased levels of pro-inflammation such as interleukin (IL) 1, IL-6, IL-8, IL-13, IL-18, C-reactive protein, interferon α, and β, transforming growth factor β (TGF-β), tumor necrosis factor α (TNF-α), and serum amyloid which causes oxidative stress, DNA damage, mitochondrial disorders, cell aging, and

tissue dysfunction. This situation is exacerbated due to changes in body composition following aging. Fat mass increase, especially in the visceral, decreases preadipocyte replication, while the number of old cells in adipose tissue causes lipotoxicity and aggravates inflammation. [9][10] This study shows that diabetic patients were more common in women, and most were obese. It is different from the global data that showed 240.1 million males DM patients and 222.9 million female DM patients.[1] In Indonesia, according to

epidemiological research conducted by the Health Research and Development Agency of the Ministry of Health of the Republic of Indonesia, the percentage of diabetes mellitus sufferers according to gender was 1.8% female and 1.2% male. [2] This situation is because most women are more obese than men. The decrease in estrogen in women will also lead to visceral obesity. Women are also more likely to be less active and consume more carbohydrates.[11]

Table 2: Correlation of systolic blood pressure with glucose, HbA1c, and lipid profiles

Variable	r Value	p-Value
Total cholesterol	0.389	0.267
HbA1c	-0.319	0.370
Fasting Blood Glucose	0.015	0.967
HDL	0.528	0.117
LDL	0.172	0.634
Triglyceride	0.324	0.361

** Correlation is significant at the least 0.01 level

Based on the results obtained from Table 2, the correlative analytical test found a positive correlation between systolic blood pressure and total cholesterol levels ($r = 0.389$; $p = 0.267$), glucose levels ($r = 0.015$; $p = 0.967$), HDL levels

($r = 0.528$; $p = 0.117$), LDL ($r = 0.172$; $p = 0.634$), Triglycerides ($r = 0.324$, $p = 0.361$) but not significant. There was a negative correlation between systolic blood pressure and HbA1c levels ($r = -0.319$; $p = 0.370$).

Table 3: Correlation of Diastolic Blood Pressure with Glucose, HbA1c and lipid profiles

Variable	r Value	p-Value
Total cholesterol	0.532	0.114
HbA1c	-0.403	0.248
Fasting Blood Glucose	-0.140	0.699
HDL	0.466	0.174
LDL	0.251	0.485
Triglyceride	0.590	0.073

** Correlation is significant at the least 0.01 level

In Table 3, there is a positive correlation between diastolic blood pressure and total cholesterol levels ($r = 0.532$; $p = 0.114$), HDL levels ($r = 0.466$; $p = 0.174$), LDL ($r = 0.251$; $p = 0.485$), triglycerides ($r = 0.590$, $p = 0.073$) but not significant. There is a negative correlation between systolic blood pressure with HbA1c levels ($r = -0.403$; $p = 0.248$) and glucose levels ($r = -0.140$; $p = 0.699$). In this study, there was no significant correlation between systolic and diastolic blood pressure on blood sugar levels, HbA1C, and lipid profiles. There is also no correlation between HbA1c and the degree of

hypertension. The blood pressure and blood glucose levels also showed no significant positive correlation, which means that hypertension damages blood vessels' endothelium through oxidative stress, failing blood vessels to experience vasodilation. Insulin also accelerates the absorption of water and salt in the distal renal tubule, increasing fluid volume. Besides, insulin plays a role in stimulating the sympathetic nerves to cause peripheral vasoconstriction, causing hypertension. Dyslipidemia, hypertension, and diabetes predispose factors for arteriosclerosis.[12][13].

Table 4: Relationship of HbA1C with Blood Pressure Degrees

Variable	Mean ± SD	p-Value
Pre-hypertension	11.066 ± 1.556	0.600
1st-degree hypertension	9.420 ± 2.960	
2nd-degree hypertension	8.850 ± 2.616	

** Correlation is significant at the least 0.05 level

Based on Table 4, there is no significant relationship between HbA1C and the degree of blood pressure. Hyperglycemia that is not controlled continuously will stimulate the emergence of oxidative stress and pro-inflammation that damages the pancreatic β islet cells resulting in apoptosis and disrupting insulin production, secretion, and performance.[14] Hyperglycemia and the performance effects of insulin change the composition of plasma lipoproteins to hypertriglyceridemia and convert LDL to Small dense LDL cholesterol (sdLDL-c), which is more atherogenic.[15] Insulin resistance is also a potential factor causing hypertension and diabetes in addition to other pathways such as obesity, inflammation, oxidative stress, and psychological factors.[16]

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CONFLICTS OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

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