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Original Article

Correlation Between Visceral Fat and Lipid Profile in Myocardial Infarction Patients

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Abstract

Background : Previous studies reported that visceral fat plays an important role in cardiovascular disease, even in non-obese individuals. Bioelectrical impedance analysis (BIA) is a non-invasive and radiation-free method for assessing visceral fat. Not much is known whether visceral fat correlates with lipid profile in myocardial infarction (MI) patients in Indonesian population. The purpose of this study was to analyze the correlation between visceral fat and serum lipid profile in MI patients.
Methods : This is a correlational study on 32 MI patients hospitalized at the ICCU of RSUP Dr. Kariadi Hospital recruited with consecutive sampling. Visceral fat was measured by BIA SECA mBCA 525 series, data regarding levels of total cholesterol, triglycerides, low density lipoprotein (LDL), and high density lipoprotein (HDL) were gathered from medical record. The data were normally distributed, then the hypothesis was tested with the Pearson.
Results : The mean age of the subjects was 55 ± 9.88 years, with 87.5% being male. As many as 81.3% of subjects experienced ST-elevation myocardial infarction (STEMI). The average body mass index (BMI) was 26.2 ± 3.68 kg/m², in which 40.6% of subjects were classified as grade 1 obesity. The majority of subjects (93.8%) had high visceral fat. As many as 68.8% of subjects had high LDL levels with an average of 120.5 ± 38.84 mg/dL. HDL average was 35±13.55 mg/dL with 62.5% of subjects having low HDL levels. More than half of the subjects (56.3%) experienced hypertriglyceridemia with an average of 157.4 ± 55.84 mg/dL. Visceral fat was significantly related to total cholesterol and triglycerides (r=0.40; p=0.02 and r=0.36; p=0.04).
Conclusion : There is a significant correlation between visceral fat and total cholesterol and triglycerides in MI patients.

Keywords : myocardial infarction, body composition, visceral fat, lipid profile.

INTRODUCTION

The main pathology of cardiovascular disease is the development of atherosclerosis. Several studies have reported that the accumulation of visceral fat plays an important role in the development of chronic inflammation in the arteries leading to atherosclerosis. Modifiable risk factors for MI include dyslipidemia and obesity as well as their interaction. Reducing and evaluating adiposity can be a promising intervention approach in the management of cardiometabolic risk. Body mass index (BMI) does not accurately describe fat distribution. Assessment of fat distribution, especially visceral fat is important in determining the risk of cardiovascular disease. A number of studies have reported that visceral fat and dyslipidemia are risk factors for cardiovascular disease but studies investigating correlation between visceral fat and lipid profiles in patients with a diagnosis of MI is rare.[1–3]

Diagnostic imaging using computed tomography (CT) and magnetic resonance imaging (MRI) can accurately measure body composition, visceral fat accumulation, and ectopic fat distribution, but these tests are expensive and involve radiation exposure, making them ineffective in everyday practice. Dual-X-ray absorptiometry (DXA) expose relatively low radiation, but visceral fat measurements tend to have lower values in individuals with normal weight and tend to have higher values in obese individuals compared to MRI. Visceral fat dysfunction contributes to metabolic syndrome and cardiovascular disease. Bioelectrical impedance analysis (BIA) is relatively simple, fast, and non-invasive which can provide a more reliable picture of body composition with minimal variability between examiners and fast examination results, with an examination error of <1% on re-examination.[4–6] BIA examination was reported in previous studies to have better results in estimating visceral fat despite differences in BMI, age group, and sex. A study in a population in Taiwan reported that visceral fat had a positive correlation with risk factors for cardiovascular disease (blood pressure, glucose levels, and lipid profile), after adjusting for age and abdominal circumference, with the strength of the relationship being stronger in women than in men. The purpose of this study was to analyze the correlation of visceral fat and lipid profile in IM patients.

METHODS

This is a correlational study with consecutive sampling. The sample size uses the following formula:

$$n = \left\{ \frac{(Z\alpha + Z\beta)}{0{,}5 \ln \{(1+r)/(1-r)\}} \right\}^2 + 3$$

It is determined that the magnitude of type I error (α) = 5%, then the value of Zα is 1.64. The type II error (β) is set at 20% (β=0.2), so the value of Zβ is 1.28. The minimum correlation that was considered significant (r) from the lipid profile in previous studies was obtained by a value of r = 0.475, so the minimum sample size was 32 people.[7]

Data is in the form of secondary data taken from ICCU patients at RSUP Dr. Kariadi as many as 32 subjects. The inclusion criteria are inpatients in the ICCU of RSUP Dr. Kariadi with a diagnosis of IM during the study period, age >18 years, not pregnant and complete medical record data. Exclusion criteria were patients with edema, pregnant patients, or patients with malignancy. The independent variable used is visceral fat. The dependent variables are total cholesterol, triglycerides, HDL, and LDL. The confounding variables that influenced the study were age, HbA1C, fasting glucose, and blood pressure. Data processing uses SPSS version 25. Data with a nominal or ordinal scale is displayed in the form of amounts (n) and percentages (%), data with a ratio or interval scale is displayed in the form of mean and standard deviation (SD). The normality test using Shapiro-Wilk and data distribution of variables was normal, then test the correlation was tested with the Pearson test (r values, p values with 95% confidence intervals). This research has gained permission from the Health Research Ethics Committee of RSUP Dr.Kariadi Semarang No.1111/EC/KEPK-RSDK/2022.

RESULTS

The subjects of this study were 32 patients with a diagnosis of MI treated at RSUP Dr. Kariadi Semarang according to the time of the study that met the inclusion and exclusion criteria. The basic characteristics of the research subjects are described in Table 1.

The youngest age of this research subject is 33 years and the oldest is 71 years. The independent variable in this study was visceral fat as measured by the BIA SECA mBCA 525 tool, visceral fat was grouped based on the cut-off value contained in the tool, namely the normal group <1.4L, the increased group <2.2L and the high group ≥2, 2L. Confounding variables include age, incidence of diabetes mellitus (DM), and incidence of hypertension. The characteristics of the research subjects are described in Table 2.

Variables are normally distributed, so the visceral fat correlation test with lipid profiles using the Pearson test can be seen in Table 3.

The scatter plot graph between visceral fat and lipid profile can be seen in Figure 1.

The correlation of visceral fat with lipid profile shown in Table 3 and Figure 1 shows that there is a unidirectional correlation between visceral fat and total cholesterol and triglycerides, but there is no significant correlation between visceral fat, HDL and LDL.

TABLE 1
Basic characteristics of research subjects

Subject Characteristics	Mean± SD	n (%)
Age (Years)	55 ± 9.9	–
<60 years	–	21 (65.6)
≥60 years	–	11 (34.4)
Gender	–	–
Male	–	28 (87.5)
Female	–	4 (12.5)
Miokardial infraction	–	–
STEMI	–	26 (81.2)
NSTEMI*	–	6 (18.8)

*non ST-elevation myocardial infarction

TABLE 2
Characteristics of research subjects

Subject Characteristics	Mean±SD/ Median	n (%)
BMI (kg/m^2)	26.2 ± 3.68	–
Underweight	–	–
Normoweight	–	5 (15.6)
Overweight	–	9 (28.1)
Obese 1	–	13 (40.6)
Obese 2	–	5 (15.6)
Viseral fat (L)	3.9 ± 1.34	–
Normal	–	1 (3.1)
Increased	–	1 (3.1)
High	–	30 (93.8)
DM	–	–
Yes	–	13 (40.6)
No	–	19 (59.4)
HbA1C (%)*	5.9	–
<6.5	–	19 (59.4)
>6.5	–	13 (40.6)
Fasting Glucose (mg/dL)	122	–
<126	–	18 (56.3)
>126	–	14 (43.8)
Hypertension	–	–
Yes	–	14 (43.5)
No	–	18 (56.3)

TABLE 2. Continued.

Subject Characteristics	Mean±SD/ Median	n (%)
Sistole (mmHg)	118.2 ± 17.91	–
<120	–	22 (68.8)
120–139	–	6 (18.8)
≥140	–	4 (12.5)
Diastole (mmHg)	75.5 ± 10.68	–
<80	–	20 (62.5)
80–89	–	8 (25)
≥90	–	4 (12.5)
Total cholesterol (mg/dL)	177.5 ± 45.97	–
Normal	–	20 (62.5)
Hypercholesterolemia	–	12 (37.5)
LDL (mg/dL)	120.50 ± 38.84	–
Normal	–	10 (31.3)
High	–	22 (68.8)
HDL (mg/dL)	35 ± 13.55	–
Low	–	20 (62.5)
Normal	–	12 (37.5)
Triglyseride (mg/dL)	157.6 ± 55.84	–
Normal	–	14 (43.8)
Hypertriglyseride	–	18 (56.3)

*Hemoglobin A1C

TABLE 3
Correlation of visceral fat with lipid profile

Variable	R	p value
Total cholesterol	0.400	0.023
Triglyseride	0.363	0.041
HDL	-0.272	0.133
LDL	0.326	0.068

The results of the visceral fat correlation test with confounding variables using the Pearson test can be seen in Table 4.

The scatter plot graph between visceral fat and confounding variables can be seen in Figure 2.

The correlation of visceral fat with all confounding variables shown in Table 4 and Figure 2 shows no significant correlation between visceral fat and all confounding variables.

DISCUSSION

Myocardial infarction is the most common cardiovascular disease, based on the results of th electrocardiogram, MI consists of STEMI and NSTEMI. RISKESDAS data in 2018 shows that the number of people with heart disease is more in men than women, as well as the British National Report in 2014, the prevalence of MI in men is 3x higher than that in women. Research in

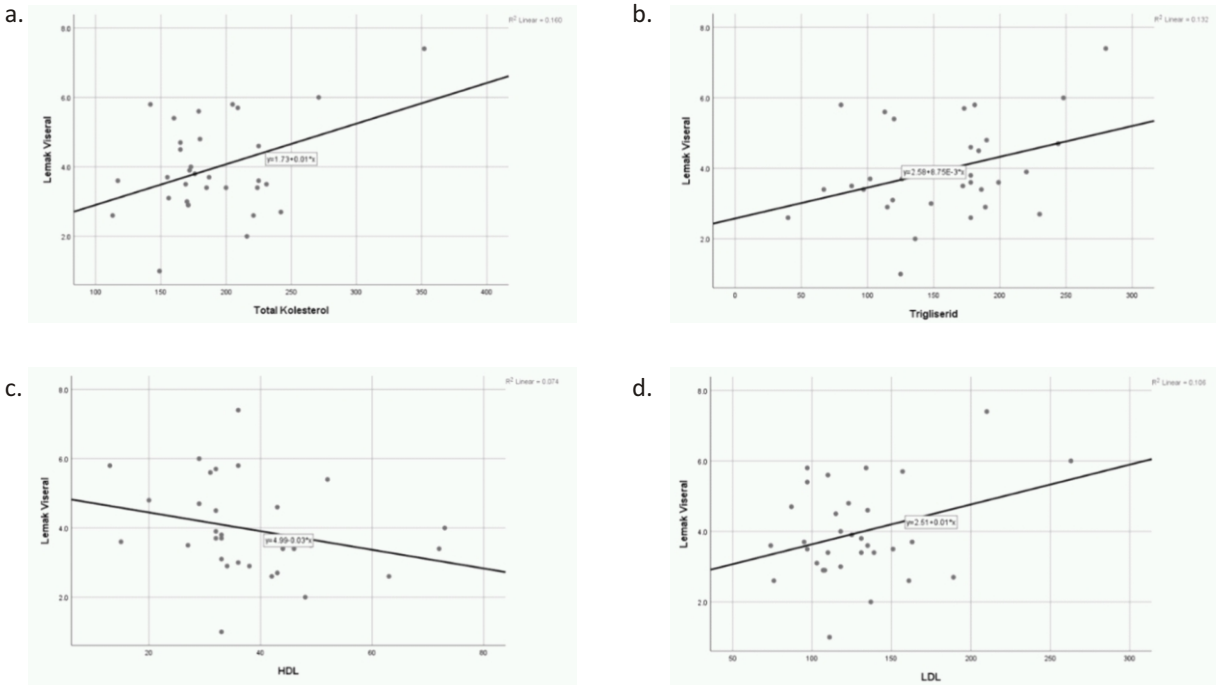

Figure 1. Scatter plot between visceral fat and (a) total cholesterol; (b) triglycerides; (c) HDL; and (d) LDL

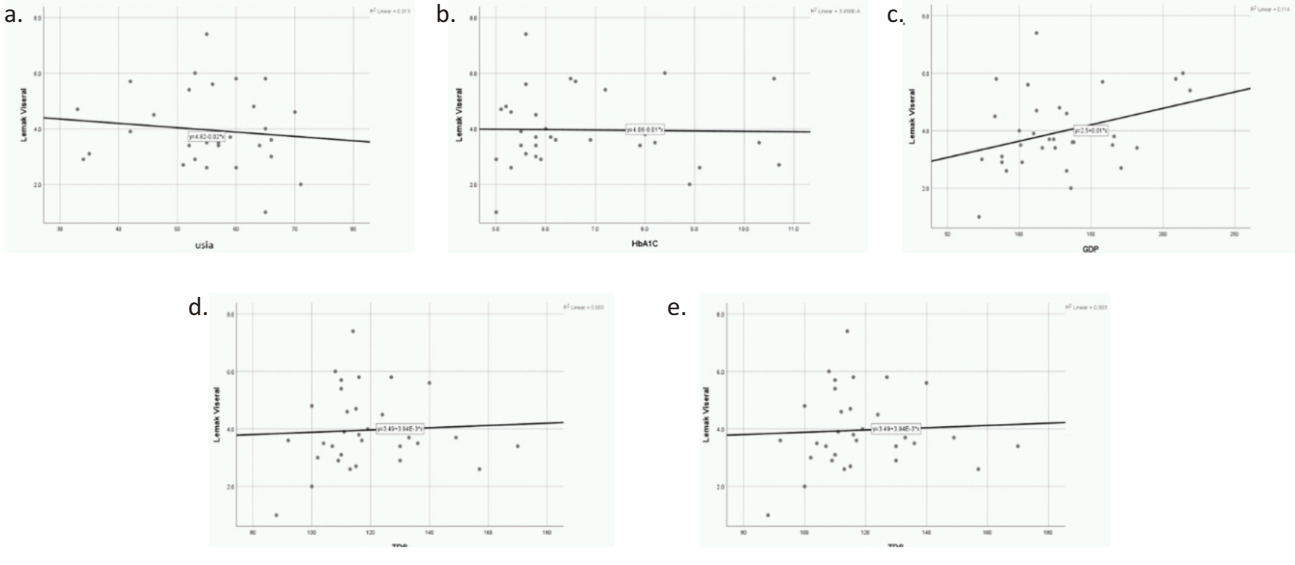

Figure 2. Scatter plot between visceral fat and (a) age; (b) HbA1C; (c) Fasting glucose; (d) diastole; and (e) systole

2020 at Dr. Kariadi General Hospital reported similar thing, 72.9% of MI patients were male. Sex hormones play an important role in the regulation, distribution and function of adipose tissue. Women have more total body fat and subcutaneous fat while men have more visceral fat. Population studies report that high testosterone concentrations are associated with increased visceral fat in women and decreased in men. In hypogonadal males, low testosterone concentrations are associated with accumulation of visceral fat.[8–10]

This study reported an average BMI of 26.22 ± 3.68 kg/m² and 40.6% of the study subjects were grade 1 obese. An increase in BMI was directly related to an increased risk of MI. Subjects in the normoweight group were 15.6%, this is in line with the Yajnik-Yudkin hypothesis (Y-Y paradox) stating that there is "thin fat", namely a significant difference in fat composition between Asian and Caucasian ethnic groups with the same BMI. The BMI anomaly of the Asian population is in the form of a slimmer physical appearance but the body

TABLE 4
Correlation of visceral fat with confounding variables

Variable	R	p value
Age	- 0.115	0.532
HbA1C	- 0.019	0.920
Fasting glucose	0.338	0.059
Diastole	0.204	0.262
Sistole	0.052	0.776

scan results show a high ratio of total fat to body weight.[11,12]

The mean visceral fat in this study was 3.9 ± 1.34 L where 93.8% of the subjects had high visceral fat. Fat distribution, especially visceral fat plays an important role in increasing the risk of cardiovascular disease. A number of studies indicate that obese patients with metabolic disorders such as insulin resistance and dyslipidemia indicate excess visceral fat. Fat distribution is very different in racial and ethnic groups and reflects differences in anthropometry of each ethnicity, Asian populations tend to have an "apple shape" body shape, which indicates a high prevalence of abdominal fat accumulation. Studies in Korea report important evidence that in individuals who appear normal and are assessed for cardiovascular risk only by anthropometry, there is a dramatic increase in cardiovascular disease without a significant increase in BMI, supporting the relevance of fat distribution. So the main target of therapy is reducing visceral fat with physical activity and healthy food. The Asian population has more visceral fat accumulation but lower BMI values.[11–13]

Visceral fat was significantly related to total cholesterol (r=0.400, p=0.023) and triglyceride (r=0.363, p=0.041), but visceral fat was not related to HDL (r= -272, p=0.133) and LDL levels (r=0.326, p=0.068). Accumulation of fat mass in obesity causes infiltration of marophages and becomes a site for the production of proinflammatory cytokines such as tumor necrosis factor (TNF) which has been shown to induce insulin resistance in adpository tissue and inhibit the synthesis and secretion of adiponectin. The decrease in adiponectin is associated with a decrease in HDL levels. The relationship between visceral fat and HDL is negative indicating that with an increase in visceral fat, HDL levels will be lower, this is in line with the results of this study, the average HDL level in this study was 35 ± 13.55 mg/dL with 62.5% subjects had low HDL levels (<40 mg/dL). HDL has anti-arterogenic effects in the form of the ability to remove cellular lipids and better cell survival. Decreased HDL levels and function are secondary to hormonal changes, inflammatory processes caused by food intake, smoking habits, and alcohol consumption were not examined in this study. Visceral fat accumulation has metabolic consequences and visceral fat may predict cardiovascular risk better than BMI even in non-obese individuals. The form of cholesterol circulation is bound to lipoproteins, the 4 main fractions of lipoproteins are divided based on density namely very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), LDL and HDL. Cholesterol and TG from the liver are carried by apolipoprotein B (ApoB) in the form of VLDL, which then undergo hydrolysis by lipase becoming IDL and LDL. VLDL cannot enter the tunica intima of the arteries, so lipase modifies it into smaller particles, so that it can enter the tunica intima of the arteries and is arteriogenic. Measurement of LDL levels is quite useful in evaluating the risk of cardiovascular disease, but this parameter only provides information about the amount of cholesterol bound to the LDL fraction, but does not provide information about the concentration of LDL particles. A number of studies highlight the missing link between LDL and cardiovascular events. A cohort study from the lipid research clinical prevalence study reports that men with LDL levels <100 mg/dl had an increased cardiovascular mortality compared to men with LDL levels of 100-130 mg/dl. A cohort study in patients with the metabolic syndrome shows that they had larger areas of visceral fat but lower LDL levels than patients without the metabolic syndrome. In line with this study, 93.8% of patients were in the high visceral fat group and the relationship between visceral fat and LDL was not significant (r=0.326, p=0.068). ApoB examination can accurately measure LDL concentrations, with this method patients with central obesity with LDL levels within normal limits but have metabolic abnormalities such as hypertriglycerides and decreased HDL levels, have 20–25% higher ApoB levels. This explains that eventhough LDL levels do not increase, patients still have a risk of cardiovascular disease because they still have high concentrations of LDL particles. The clinical practice of assessing LDL particle size is fasting triglyceride levels, which have shown to be negatively correlated with LDL particle size. This negative

correlation describes the activity of the cholesterol ester protein transfer enzyme due to hypertriglycerides, namely the exchange of triglyceride molecules from VLDL to LDL. This exchange results in a relative increase in LDL triglyceride content, which is further hydrolyzed by hepatic lipase. The end result is the formation of small particle LDL cholesterol. This explains that the accumulation of visceral fat is associated with increased TG levels and causes the formation of LDL which has small and dense particles, then develops into insulin resistance and cardiovascular disease. Assessment of cardiovascular disease risk in clinical practice can examine the levels of TG, HDL and the TK:HDL ratio.[9,14–21]

The age of the research subjects was dominated by <60 years old, which was 65.6%. This indicates a tendency for lipid profile disturbances to occur at a young age. This is in line with a study in Japan for 3 decades (1985–2014) which reported a significant increase in the age group ≤59 years. One of the most important health issues in Asia Pacific is cardiovascular disease, where there are increasing rates of dyslipidemia, DM, obesity and hypertension due to rapid urbanization, a shift in diet to a "western" diet, smoking, and decreased physical activity. Observational studies report that intake of fruit, dairy products, and fiber has a protective effect, whereas intake of fried and fatty foods, alcohol, red and processed meats, sugary drinks, processed flour and foods with a high glycemic index are associated with high visceral fat mass. Excess consumption of processed foods that contain high energy and low nutrient content combined with a sedentary lifestyle is a risk factor for cardiovascular disease. Dietary factors such as increased intake of fat and alcohol even in non-obese individuals will trigger the accumulation of visceral fat, which is characterized by high lipogenic activity and high lipolytic activity. Visceral fat circulates in the portal vein system and is directly related to the liver, where high lipolytic activity will cause insulin resistance due to increased levels of free fatty acids in the liver. Glucose intolerance is related to lipid peroxidation with consequent DNA damage. DNA damage can be used as a biological marker in the detection, monitoring and prognosis of degenerative diseases such as atherosclerosis. Increased oxidative stress causes endothelial dysfunction by increasing vascular superoxidation anion production, and in turn will decrease nitric oxide (NO). Endothelial dysfunction is associated with cardiovascular disease risk factors such as smoking, accumulation of visceral fat and fasting or postprandial hypertriglycerides. A study in England compared Asian and Caucasian ethnicity, found that the Asian population was diagnosed younger. Study in Korea on non-obese adult males reported that visceral fat was most significantly associated with impaired postprandial lipid response, lipid peroxidation, DNA damage and endothelial dysfunction, which associated with the risk of morbidity and mortality from cardiovascular disease.[13,22–26]

Visceral fat accumulation has been shown to cause glucose intolerance resulting in insulin resistance. Several studies have reported that the combination of visceral fat and lipid dysregulation is associated with high sugar intake. A study in South Korea showed a significant relationship between visceral fat and HbA1C in the pre-DM patient group. HbA1C is reported to be useful in assessing long-term glycemic control in DM patients. HbA1C levels are affected by the number of erythrocytes, so in anemic patients the HbA1C value cannot be used. There was no relationship between visceral fat and HbA1C in this study, this could be due to the subject being anemic. Anemia conditions and DM treatment history were not examined in this study. The high prevalence of DM in patients with cardiovascular disease among Asian population causes them to feel no any classic symptoms of MI, so that the percentage of "silent killers" is higher in the Asian population.[27,28]

Hypertrophy occurs in fat cells, especially visceral fat in obese individuals, causing a decrease in adiponectin and insulin sensitivity which develops into insulin resistance. Fasting blood sugar is one of the most sensitive parameters for detecting DM. Fasting glucose levels increase with age and visceral fat in overweight and obese individuals. In patients with acute myocardial infarction, there is a stress response in the form of excessive secretion of steroid hormones, adrenaline, glucagon and free fatty acids. Acute hyperglycemia can disrupt the prothrombotic phase, increase inflammation and oxidative responses, damage endothelial cells and microcirculatory function and eventually lead to massive myocardial infarction. There was no relationship between visceral fat and fasting glucose levels ($r=0.338$; $p=0.059$) in this study, possibly due to the use of insulin therapy in patients with high blood sugar levels at admission. In this study there was no data on the time of sampling for examination of fasting glucose levels, so it was not known whether it was the fasting glucose level after admission or the evaluation fasting glucose level.[29,30]

Visceral fat has been reported to be associated with blood pressure. Several mechanisms of visceral fat are associated with increased blood pressure, namely, adipokine secretion, free fatty acids derived from visceral fat circulating in the portal vein, cardiac sympathetic activity is higher in visceral obesity and visceral fat is associated with activation of the renin-angiotensin-aldosterone system. The results of this study found no relationship between visceral fat, TDS and TDD as no hypertension therapy before the subject experiencing MI or the time of blood pressure examination.[31,32]

Limitations of this study is no data on therapy either before treatment or during treatment is available, so that it affects the levels of lipid profiles, fasting blood sugar, and blood pressure. The research subjects were

ICCU patients where there was a change in the hypercatabolic state to be very different from the healing phase, so the time for data collection greatly influenced the results of the study. Future studies need a larger sample size using the cohort method.

CONCLUSION

This study shows that there is a significant correlation between visceral fat and total cholesterol and triglycerides in MI patients. All confounding variables (age, HbA1C, fasting glucose, and blood pressure) were not correlated with visceral fat in this study. Visceral fat assessment is very important as an effort to prevent non-communicable diseases, especially cardiovascular disease, with a relatively easy and fast examination with BIA, can describe metabolic risk. The BIA examination also evaluates more accurately than conventional anthropometric examinations, where BMI cannot describe the risk of fat distribution, thus causing underdiagnosis.

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