Original Research Article

Relationship between leptin levels and lipid profile in obesity adult in Puskesmas Nanggalo Padang working area

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ABSTRACT

Background: Obesity is a condition with abnormal or excessive fat accumulation in adipose tissue so that it can interfere with health. Leptin is an adipokine produced by adipose tissue that acts as the main regulator in regulating energy balance and weight. High leptin levels inhibits lipid synthesis, fatty acid synthesis and acetyl CoA carboxylase which contributes to lipid accumulation. Leptin and lipid profiles were found to be elevated in obese subjects. The purpose of this study was to determine differences in leptin levels and lipid profiles between obese and non-obese adults, to know the association between leptin levels and lipid profiles in obese adults in the Nanggalo Padang health center work area.

Methods: Comparative cross sectional research design. The research was carried out in the Nanggalo Padang Health Center, Biomedical and Biochemistry Laboratory, Medical Faculty of Andalas University in December 2017 until August 2018. The sample was 22 obese adults and 22 non-obese adults. The sample was taken by consecutive sampling technique. Serum leptin levels was measured using the ELISA method and lipid profile using the CHOD-PAP and GPO-PAP methods. Data analysis using data normality test with Shapiro Wilk, mann whitney T-Test and correlation.

Results: There was a significant difference between obesity and non-obesity leptin ($p<0.05$), there was no significant difference between total cholesterol ($p>0.05$), triglycerides ($p>0.05$), LDL ($p>0.05$), HDL ($p>0.05$), there is a weak association between leptin levels and lipid profile.

Conclusions: There was significant difference in leptin levels between obese adult and non-obese adults, there was no significant difference in lipid profiles between obese adults and non-obese adults, and there was a weak association between leptin levels and lipid profiles in obese adults.

Keywords: Leptin level, Lipid profile, Obesity

INTRODUCTION

The flow of times and changes in trends and unhealthy lifestyles, lead people to suffer from obesity. Obesity is considered as the first signal of the emergence of a group of non-infectious diseases (non communicable diseases) that occur in many developed and developing countries. This phenomenon is often named new world syndrome and this has caused a very large socio-economic and public health burden in developing countries including Indonesia.[1]

Obesity is one of the nutritional problems in Indonesia. Based on the 2013 basic health research report RISKESDAS, obesity is also one of the nutritional problems in Indonesia and the prevalence of obesity continues to increase annually. Based on BMI, obesity prevalence in population ≥18 years of age is as much as

15.4%, in 2013 the prevalence of obese adult males was 19.7%, the prevalence increased from 2007 which was 13.9% and decreased in 2010 to 7.8%. In 2013, the prevalence of obesity in adult women >18 years of age was 32.9%, an increase of 18.1% from 2007, which was 13.9%, and an increase of 17.5% from 2010 which was 15.5%.[2]

The prevalence of obesity in West Sumatra is also quite high at 13.5% and the city of Padang is the top 5 districts/cities with the highest prevalence (Ministry of Health, 2013). The prevalence of obesity for examinations by sex, sub districts, and Health Center in Padang in 2014 was 21.0%, in 2015 amounted to 39.49%. In 2016 the Nanggalo Health Center Padang had the highest prevalence of obesity, which was 6.96% (City Health Service Padang, 2016).[3] Leptin works mainly in the arcuate nucleus. Leptin stimulates the release of melanocortin signals (appetite suppressants) by proopiomelanocortin/cocain and amphetamine-regulated transcript (POMC/CART) neurons and at the same time inhibits the signal of neuropeptide Y (NPY) in the hypothalamus thereby suppressing appetite and increasing energy expenditure.[4,5]

High leptin levels inhibit lipid synthesis, fatty acid synthesis and acetyl CoA carboxylase which contribute to lipid accumulation.[6,7] Obesity is generally caused by excessive intake of fatty foods into the body. This can cause an increase in blood cholesterol levels. Increased blood cholesterol is one of the symptoms of dyslipidemia which is a disorder of lipid metabolism and is associated with risk factors for coronary heart disease (CHD).

Cholesterol in the blood cannot dissolve, therefore, to dissolve, together with triglycerides, bind to a specific protein, namely apoprotein and lipoprotein. This consists of High Density Lipoprotein-Cholesterol (HDL-C) and Low Density Lipoprotein-Cholesterol (LDL-C) complex. Cholesterol carried by LDL, in higher levels is associated with CHD, while cholesterol carried by HDL, in lower levels is associated with CHD.[8]

METHODS

The design of this research was cross sectional comparative, where the independent variables and dependent variables were examined at the same time. This research was carried out from December 2017 to November 2018 in the Nanggalo Health Center Padang.

Examination of total cholesterol, triglycerides, LDL and HDL was carried out in the Biochemical Laboratory of the Medical Faculty, Andalas University and examination of leptin levels was done at the Biomedical Laboratory of the Medical Faculty, Andalas University. Height measurement, weighting, waist circumference measurement and blood sampling were carried out at the sampling location. Serum leptin levels was measured

using the ELISA method and lipid profile using the CHOD-PAP and GPO-PAP methods.

The population in this study were obese adults and as non-obese adult controls in the Nanggalo Padang Health Center Working Area. The samples of this study were obese adults and non-obese adults in the Nanggalo Padang Community Health Center Working Area who fulfilled the criteria for exclusion and exclusion.

Inclusion criteria

Willing to be involved in the study and sign a letter of consent to be a respondent, age 35-45 years, obese adults and non-obese adults as evidenced by BMI measurements and waist circumference.

Examination criteria

Suffering from cancer, hepatitis, kidney and diabetes, taking certain drugs regularly, including vitamin supplements, using hormone therapy (estrogen, testosterone, insulin), having smoking habits and consuming alcoholic beverages.

The sampling technique in this study was consecutive sampling. In consecutive sampling, all subjects who came and met the selection criteria were included in the study until the required number of subjects was fulfilled.

This study had obtained ethical approval from the Research Ethics Committee at the Medical Faculty, Andalas University. The data obtained were tested for normality by the Shapiro-Wilk test then processed by the Independent T-Test and Correlation test with a significance level of p = 0.05.

RESULTS

Research and examination resulted that each group consisted of 22 obese and 22 non-obese with a total sample of 44 people.

The table 1 showed that the mean leptin levels in the obese group were (46.54±29.86μg/ml) and non-obese were (21.54±18.13μg/ml). The mean cholesterol level in the obese group were (224.41±30.44mg/dl) and non-obese group were (211.85±49.02mg/dl). The mean triglyceride levels in the obese group were (179.60±79.03mg/dl) and non-obese group were (146.07±50.32mg/dl). The mean LDL levels in the obese group were (134.88±39.90mg/dl) and non-obese group were (124.39±51.87mg/dl).

Based on Figure 1, it can be concluded that there is a weak relationship between leptin levels and total cholesterol in obese respondents. The results showed that the higher the level of leptin, the higher the total cholesterol level because it has a value of p>0.05.

Based on Figure 2, it can be concluded that there is a weak correlation between leptin levels and triglycerides in obese respondents. The results showed that the higher

the level of leptin, the lower the triglyceride level because it has a value of p>0.05.

Table 1: Average leptin levels between groups.

Variable	Group	n	Mean±SD	P value
Leptin	Obesity	22	(46.54±29.86 µg/ml)	0.002
	Non Obesity	22	(21.54±18.13 µg/ml)	
Cholesterol	Obesity	22	(224.41±30.44 mg/dl)	0.313
	Non Obesity	22	(211.85±49.02 mg/dl)	
TG	Obesity	22	(179.60±79.03 mg/dl)	0.304
	Non Obesity	22	(146.07±50.32 mg/dl)	
LDL	Obesity	22	(134.88±39.90 mg/dl)	0.456
	Non Obesity	22	(124.39±51.87 mg/dl)	
HDL	Obesity	22	(53.60±23.51 mg/dl)	0.532
	Non Obesity	22	(58.19±24.82 mg/dl)	

Based on Figure 3, it can be concluded that there is a weak relationship between leptin levels and LDL levels in obese respondents. The results show that the higher the leptin level, the higher the LDL level because it has a value of p>0.05.
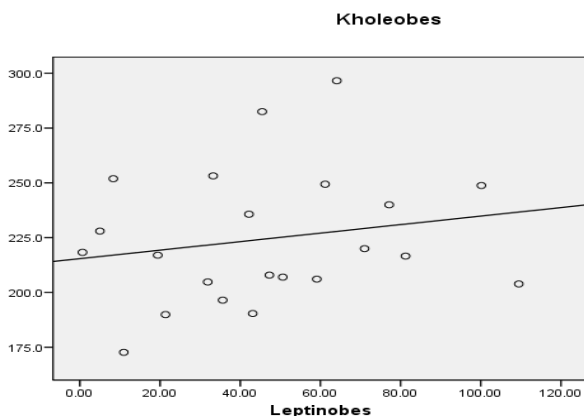

Figure 3: Association between leptin levels with LDL.

Based on Figure 4, it can be concluded that there is a weak relationship between leptin levels and HDL levels in obese respondents. The results show that the higher the leptin level, the higher the HDL level because it has a value of p>0.05.

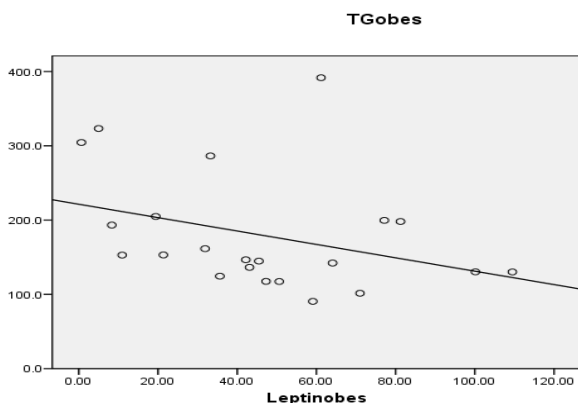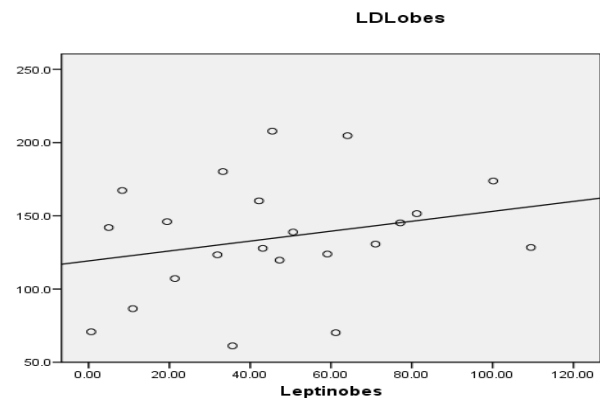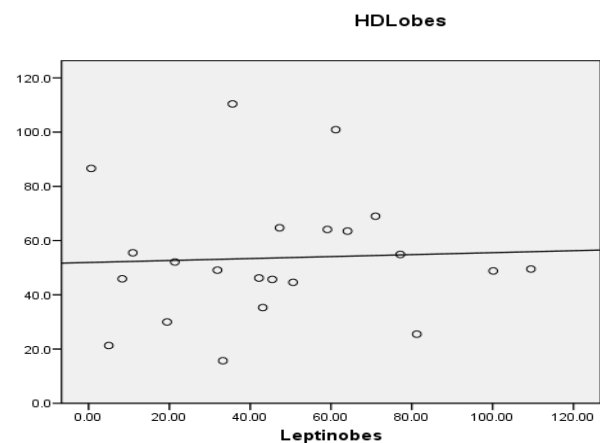**Figure 1: Association between leptin levels and cholesterol.**

Figure 2: Association between leptin levels and TG.

Figure 4: Association between leptin levels with HDL.

DISCUSSION

Relationship of leptin levels with total cholesterol in obese adults in Nanggalo Health Center Work Area Padang

From the results of the study, it was found that the average leptin level in obese adults was higher than non-obese adults. Statistical tests showed that there were significant differences between obesity and non-obesity leptin levels.

Most leptin is produced by adipose tissue which acts as the main regulator in regulating energy balance and weight.[4] Cholesterol can be found in almost all body cells, especially in the liver and intestine cells. Cholesterol functions as a bile salt precursor, acts in the process of digestion and absorption of fat, and as a steroid hormone precursor.[9] In obese patients, high levels of leptin inhibit lipid synthesis, fatty acid synthesis and acetyl CoA carboxylase action which contribute to lipid accumulation.[6,7] Acetyl CoA which inhibited in turn will inhibit cholesterol synthesis because acetyl CoA is a precursor for cholesterol synthesis, which is formed from amino acids or fatty acids and glucose.[9]

The results of this study are in line with the research of Sonia et al, concerning the association of hyperleptinemia with low levels of systemic inflammation and metabolic dysfunction in obese people in Mexico. It showed that leptin had a positive association with elevated triglyceride levels (r = 0.4135, p = 0.0001), but not with total cholesterol.[10] Different results were stated by Hiba et al, about the effects of obesity and the components of the metabolic syndrome with leptin levels in women in Saudi Arabia. It showed that there was a significant association between leptin levels and total cholesterol p<0.05.[11]

Relationship between leptin levels and tg in adults obesity in the Nanggalo Health Center Work Area Padang

The results showed that there was a weak relationship between leptin levels and triglyceride.

In obesity, high triglyceride levels are caused by excessive fat accumulation resulting in an increase in the amount of free fatty acids in adipose tissue. Leptin is produced mostly by adipose tissue which acts as the main regulator in regulating energy balance and weight.[4] An increase in adipose tissue number is accompanied by increase in leptin levels. Leptin stimulates lipolysis in adipose tissue, where chylomicrons are immediately broken down into fatty acids and glycerol. Furthermore, these fatty acids and glycerol are reformed into triglyceride deposits. At any time, if we need energy from fat, triglycerides are broken down into fatty acids and glycerol, transported into cells to be oxidized into energy. Free fatty acids stimulate β oxidation in the mitochondria to produce energy in the β oxidation process.[12] High

levels of leptin inhibit the expression of acetyl CoA carboxylase, fatty acid synthesis and lipid synthesis, and any biochemical reactions that contribute to lipid accumulation.[6,7] Then high levels of fat or triglycerides inhibit leptin transport through the blood brain barrier to the hypothalamus, resulting in the inability of leptin to bind to its receptors in the hypothalamus. This causes leptin unable to suppress incoming food intake, causing high appetite, high food intake and low energy use.[13]

The results of this study are in line with the research of Mohmoud et al, about the association between serum leptin, BMI, waist circumference and cholesterol level in adolescents in Gaza. It showed that there was no significant association between serum leptin and triglycerides p>0.05.[14]

Different result was discovered by Sonia et al, regarding the association between hyperleptinemia and low systemic inflammatory parameters and metabolic dysfunction in obese people in Mexico. It showed that leptin had a positive association with elevated triglyceride levels (r = 0.4135, p = 0.0001).[10]

Association between leptin levels and ldl in obese adults in the Nanggalo Health Center Work Area Padang

The results of the study showed that in the obese respondents, the higher the leptin level, the LDL level will be higher. There was a weak association between leptin levels and LDL levels.

Obesity and excessive fat diet can cause high levels of fat in the body. Increased fat content is stored in the body in the form of triglyceride acid. Increased fat transport, especially cholesterol in the blood will cause a significant increase in LDL. This is because LDL is the lipoprotein containing the most cholesterol. High leptin levels inhibit lipid synthesis, fatty acid synthesis and Acetyl CoA Carboxylase which contribute to lipid accumulation.[6,7] In obese people, inhibition of fatty acid synthesis by leptin, will cause an uncontrolled increase in free fatty acid (FFA) produced by adipose through lipolysis, which can lead to increased FFA uptake to the liver. This increase in FFA causes an increase in triglyceride (TG) production and synthesis of very low density lipoprotein (VLDL) in the liver, accompanied by inhibition of lipoprotein lipase in adipose tissue and skeletal muscle, resulting in hypertriglyceridemia. This condition is caused by the accumulation of fat in a state of obesity which cause inability of adipose cells to store triglycerides adequately, which will trigger an increase in triglycerides and eventually increase LDL levels. The state of increased triglycerides in the blood will promotes metabolism of VLDL into LDL, which is easily oxidized.[15]

The results of this study are in line with the research of Erkin et al, about lipids and leptin levels in the native population of Kyrgyzstan. It showed that there was no

significant association between leptin levels and LDL with p>0.05.[16]

Different research results were found by Kismat et al, about the effect of obesity on serum leptin and lipid profiles in Turkey. It shows that there was a significant association between positive leptin levels and LDL (r = 0.366, P<0.05). The difference between this study and the previous research may be due to differences in research methods and characteristics of the sample in the study.[17]

Relationship between leptin levels and hdl in obese adults in Nanggalo Health Center Work Area Padang

The results of the study stated that in the obese respondents, the higher the level of leptin, the HDL level will be higher. There was a weak association between leptin levels and HDL levels.

Most leptin is produced by adipose tissue which acts as the main regulator in the regulation of energy balance and weight.[6,7] HDL comes from the liver. HDL can restore excess cholesterol by transporting the accumulated cholesterol in the tissues and walls of blood vessels back to the liver. When transported, cholesterol is facilitated by lecithin cholesterol acytransferase (LCAT). HDL also has less fat content and has higher density so it can carry excess bad cholesterol in the arteries to be processed and disposed. HDL also prevents cholesterol accumulation in the arteries, thus preventing atherosclerosis.[9] High leptin levels inhibit lipid synthesis, fatty acid synthesis and acetyl CoA carboxylase which contribute to lipid accumulation.[6,7] So it is harder for HDL to prevent the deposition of cholesterol in the arteries, thus preventing atherosclerosis will be harder.

The results of this study are in line with the research of Erkin et al, about lipids and leptin levels in the native population of Kyrgyzstan. It showed that there was no significant association between leptin levels with HDL with p>0.05.[16]

A different study was carried out by Mohmoud et al, regarding the association between serum leptin, BMI, waist circumference and cholesterol levels among adolescents in Gaza. It shows that there was a significant association between serum leptin with HDL with p<0.05. The difference between this study and previous study may be due to differences in research methods and characteristics of the sample in the study.[14]

CONCLUSION

There was significant difference in leptin levels between obese and non-obese, there was no significant difference in the lipid profile levels between obese and non-obese, there was a weak association between leptin and lipid profile (triglycerides, cholesterol, LDL, HDL) levels in obese adults in the Nanggalo Health Center Work Area Padang in 2018.

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