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Lipid Profiles and Blood Pressure among Worker Women, Its Correlation with Risk Factor of Coronary Heart Disease

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Abstract: Obesity lead to serious health consequences. Risk increases progressively as BMI increases to contributes an increased risk for chronic disease, including diabetes, cardiovascular diseases and certain types of cancer. The prevalence of obesity has increased in Indonesia. The cross sectional study was applied to 47 obese group and 97 normal weight women worker. We compared the lipid profiles and blood pressure of obese worker women (n = 47, mean age 38.89) to those with a normal weight (n = 97, mean age 37.09). The average body mass index was 30.13 kg/m^2 in the obese and 22.99 kg/m^2 in the normal group. Ratio LDL-cholesterol/HDL-cholesterol in obese group 2.62 ±1.03 higher than normal group 1.87±0.61. The obese group had significantly higher levels of total cholesterol, LDL-cholesterol, lower levels of HDL-cholesterol and higher blood pressure compared to the normal group. Obesity in worker women is associated with disturbances in lipid metabolism and a greater risk for cardiovascular disease.

Key words: Obesity, lipid profiles, blood pressure, worker women

INTRODUCTION

Obesity has become a serious public health problem throughout the world. Th World Health Organization states that the global trends of obesity keep on getting higher. Obesity is growing concern among middle aged adults as a result of its increasing prevalence and profound impact on health and quality of life. Weight gain often occurs in middle age. Women typically manifest additional gain in body fat during the perimenopausal period. BMI and weight are the strongest predictors of blood pressure in human (Dyer and Elliot, 1989).

The prevalence of obesity is increasing worldwide at an alarming rate in both developing and developed countries. It has become a serious epidemic health problem, estimated to be the fifth leading cause of mortality at global level. It is estimated that, worldwide, approximately 937 million adults are overweight and 396 million are obese (Kelly *et al*., 2008). In Indonesia, based on basic health research that was conducted by The Ministry of Health in (2007), the national prevalence of obesity (BMI = 27 kg/m^2) among man and woman the prevalence of obesity among people age more than 15 years is 10.38%, among man 13.9% and woman 23.8% (women were higher than men) (Ministry of Health Republic of Indonesia, 2008)

Obesity is a major risk factor for many disease, including cardiovascular disease, diabetes and certain types of cancer, resulting in an increased risk of death (Calle *et al*., 1999). Coronary Heart Disease (CHD) is leading cause disability and mortality in the world. There are many factors that has contribution to development CHD. Determinant factors of CHD were as follows stress, inactivity, diabetes mellitus, smoking, dyslipidemia, obesity and hypertension. Hypertension has associated with CHD, because of the presence of hypertension increases the risk by 6 times compared with who are not hypertensive (Michael and Jennifer, 2000).

In this study, we investigated the clinical and biochemical parameters of obese worker women to determine the metabolic abnormalities associated with obesity, its correlation with risk factor of coronary heart disease.

MATERIALS AND METHODS

Subjects: The cross sectional study was applied to 47 obese group and 97 normal weight women worker. Forty seven subjects older than 30 and younger 45 years of age with body mass index (BMI) over 25 kg/m^2 (obese group) and ninety seven subjects with between 18.5 and 24.9 kg/m^2 (normal weight group) were included in the study. None of the 144 subjects had evidence of healthy, had married, no pregnant and lactation, no smoking, no drink alcohol. Subjects who infectious disease were excluded from this study. The study protocol was approved by an ethical review committee at the Institute of Health Research and Development Indonesia (No. LB.02.01/5.2/KE.093/2013, 21 Maret, 2013). All subjects in the survey participated voluntarily and written informed consent was obtained from all subjects.

Anthropometric measurement: Anthropometric measurements which included weight and height.

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Height and weight were obtained using standardized techniques and calibrated equipment. Height was taken to the nearest 0.1 cm using the microtoise which was mounted to the wall. Subjects were asked to remove their shoes before measurement was taken. Weight was taken using TANAKA merk. Weight was measured to the nearest 0.1 kg with shoes, hand phone, coins and wallets removed. Body mass index (BMI) was calculated as body weight (kg) divided by squared body height (m^2).

Clinical and biochemical analysis: Clinical and biochemical indicators, which included blood pressure and full lipid profiles (i.e., total cholesterol, HDL-cholesterol, LDL-cholesterol and triglycerides) were determined. Blood pressure was measured using an automatic blood pressure monitor. The subjects were required to be seated while two measurements were taken using the subjects left arm with at least 5 min apart and the mean was used for analysis.

Venous blood was drawn for screening of full lipid profiles. Each subjects was required to fast for at least 10-12 h prior to drawing blood. Serum cholesterol, LDL-cholesterol, HDL-cholesterol were measured with clinical chemistry analizer selectra yunior. Serum triglyceride was analyzed using enzymatic-calorimetric method with clinical chemistry analizer selectra yunior.

Statistical analysis: Total cholesterol was classified as high (>230 mg/dL), borderline (200-229 mg/dL), normal (<200 mg/dL). LDL-cholesterol was categorized as high (>150 mg/dL), borderline (130-149 mg/dL), normal (<130 mg/dL). Low HDL-cholesterol as HDL-cholesterol (<50 mg/dL), normal HDL-cholesterol (>50 mg/dL). High triglyceride (>200 mg/dL), borderline (150-199 mg/dL), normal (<150 mg/dL). Blood pressure was define hipertensive as systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg, prehypertensive as systolic blood pressure 120-139 mmHg or diastolic blood pressure 80-89 mmHg, normal as systolic blood pressure <120 mmHg or diastolic blood pressure <80 mmHg.

The data were processed through descriptive and inferential analyses and are presented in tables. The descriptive analysis was used to describe the variables examined. Descriptive statistical results are presented in the tables as the Means±SD deviations. Relationships between categorical variables were analyzed using Chi-square test whereas Student's unpaired-t test was used to compare means of two groups. Significant level is at $p<0.05$.

RESULTS

Demographic and anthropometric characteristics of subjects: The demographic and anthropometric characteristics of test subjects are shown in Table 1. The mean age of the obese group was 37.09±4.03 years and that of normal weight group was 38.89±3.96 years. There was significant age difference between the two groups. BMI were significantly different between the obese and normal weight groups as we recruited subjects based on BMI (Table 1). Ratio total cholesterol/HDL-cholesterol and ratio LDL-cholesterol/HDL-cholesterol were significantly higher in the obese group ($p = 0.000$).

Table 1: Demographic and anthropometric characteristics of subjects

Number of subjects	Normal weight (n = 97)	Obese (n = 47)	Significance
Age (years)	37.09±4.03	38.89±3.96	p = 0.013
BMI (kg/m^2)	22.99±2.42	30.13±3.42	p = 0.000
Ratio chol/HDL-chol	3.20±0.70	4.17±1.30	p = 0.000
Ratio LDL-chol/HDL-chol	1.87±0.61	2.62±1.03	p = 0.000

Significance was test by a the Student's t-test. Value are Means±SD

Table 2: Clinical characteristics of subjects

Number of subjects	Normal weight (n = 97)	Obese (n = 47)	Significance
Total cholesterol (mg/dL)	161.69±34.77	184.89±39.90	p = 0.000
LDL-cholesterol (mg/dL)	94.04±29.62	115.66±35.12	p = 0.000
HDL-cholesterol (mg/dL)	51.39±9.57	46.32±9.55	p = 0.003
Triglyceride (mg/dL)	78.74±37.70	121.15±84.10	p = 0.002
Blood pressure (mmHg)			
Systolic	120.90±16.05	130.51±20.46	p = 0.003
Diastolic	79.21±9.16	85.72±11.82	p = 0.000

Significance was test by a the Student's t-test. Value are Means±SD

Lipid profiles and blood pressure: Blood pressure measurements and serum lipid profiles including total cholesterol, LDL-cholesterol, HDL-cholesterol and triglycerides are presented in Table 2. The obese group had significantly higher levels of total cholesterol ($p = 0.000$, 23.20 mg/dL higher), LDL-cholesterol ($p = 0.000$, 21.62 mg//dL higher), triglycerides ($p = 0.002$, 42.41 mg/dL higher) and significantly lower levels of HDL-cholesterol ($p = 0.003$, 5.07 mg/dL lower) as compared to the normal weight group. Although obese group's total and LDL-cholesterol levels and triglycerides were significantly higher than those of the normal weight group, their average values were still within the normal range (Table 2). Systolic ($p = 0.003$) and diastolic blood pressures ($p = 0.000$) were also significantly higher in the obese group. When the number of subjects with abnormal levels of clinical parameters counted, the obese group had a higher percentage of subjects with dyslipidemia (Table 3).

DISCUSSION

In this study, serum lipid profiles and blood pressure levels in our study confirmed that obesity is associated with an increased risk coronary heart disease. Obese subjects had significantly higher total cholesterol, LDL-cholesterol, triglycerides and blood pressure and significantly lower HDL-cholesterol than normal weight subjects.

Table 3: Number of subjects with abnormal lipid profiles and blood pressure

	Normal weight (n = 97)		Obese (n = 47)		
Criteria	N	%	N	%	Significance
Total cholesterol					p = 0.023
Normal: <200 mg/dL	84	86.6	32	68.1	
Borderline: 200-229 mg/dL	11	11.3	11	23.4	
High: >230 mg/dL	2	2.1	4	8.5	
LDL-cholesterol					p = 0.009
Normal <130 mg/dL	87	89.7	33	70.2	
Borderline: 130-149 mg/dL	6	6.2	6	12.8	
High: >150 mg/dL	4	4.1	8	17	
HDL-cholesterol					p = 0.003
Normal: > = 50 mg/dL	57	58.8	15	31.9	
Low: <50 mg/dL	40	41.2	32	68.1	
Triglyceride					p = 0.060
Normal : <150 mg/dL	92	94.8	40	85.1	
Borderline : 150-199 mg/dL	4	4.1	3	6.4	
High: >200 mg/dL	1	1	4	8.5	
Systolic blood pressure					p = 0.006
Normal: <120 mmHg	42	43.3	9	19.1	
Prehypertensive: 120-139 mmHg	41	42.3	23	48.9	
Hypertensive: >140 mmHg	14	14.4	15	31.9	
Diastolic blood pressure					p = 0.005
Normal: <120 mmHg	74	76.3	23	48.9	
Prehypertensive: 120-139 mmHg	4	4.1	4	8.5	
Hypertensive: >140 mmHg	19	19.6	20	42.6	
Dyslipidemia					p = 0.002
Without	47	48.5	10	21.3	
With	50	51.5	37	78.7	

Significance was test by a Chi-square test

Many clinical studies have reported that obesity is associated with incidence of cardiovascular disease. Wiyono *et al.* (2004) concluded that a change 1 unit BMI would increase 2.49 mg/dL total cholesterol (Wiyono *et al.*, 2004). Kromhout (1983), reported that a change in body weight of 1 kg was associated with a change in serum cholesterol of 2 mg/dl. Risk increases progressively as BMI increase (Levenson *et al.*, 2002). Harahap (2003) reported that the prevalence of hypertension, hypercholesterol and diabetes mellitus tends to increase with increasing BMI (Harahap, *et al.*, 2005).

Total cholesterol is the measure most widely used to describe blood cholesterol. Some study used ratio cholesterol total/HDL to know risk of cardiovascular disease. Ratio total cholesterol/HDL-cholesterol that recommended by Adult Treatment Panel were < 5:1. Low HDL-cholesterol and high total cholesterol will increase risk of cardiovascular disease. Ratio LDL-cholesterol/HDL-cholesterol <3.5: 1, ratio low HDL-cholesterol and high LDL-cholesterol increasing risk of cardiovascular disease (Levenson *et al.*, 2002).

Wiyono *et al.* (2004) conclude that associated BMI with LDL-cholesterol. A change in BMI of 1 unit was associated with a change in LDL-cholesterol of 1.65 mg/dL. Because LDL-cholesterol cause calcification and accumulation in the coronary arteries of LDL-cholesterol is considered a major risk factor of cardiovascular disease risk. Incidence of coronary heart disease increased by 1% for each increase of 1 mg/dL LDL-cholesterol (Wiyono *et al.*, 2004).

Triglyceride was not significantly different between obese and normal weight subjects. But triglyceride were one of risk factor for cardiovascular disease. Dietary effects on serum cholesterol or LDL alone do not provide an adequate basis on which to explain the relationship between diet and CHD within population. The effect of diet and in particular dietary fats, in modulating the clearance of triglyceride is of paramount importance in preventing the accumulation of atherogenic remnants and development of proatherogenic abnormalities in LDL and HDL (Gibney, 2002). When triglyceride borderline high (150-199 mg/dL), HDL-cholesterol levels begin to fall. When triglyceride levels are greater than 150 mg/dL, HDL-cholesterol concentrations frequently are 50 mg/dL in women. Thus, the term isolated low HDL can be reserved for HDL-cholesterol levels <50 mg/dL in the presence of serum triglycerides <150 mg/dL (Assmann and Schulte, 1992).

Although the obese group's averages were within normal ranges, it is clear that obesity is associated with abnormal lipid metabolism and a higher risk of dyslipidemia in worker women. Dyslipidemia is characterized by improper blood lipid profiles and people with dyslipidemia are at increased risk of coronary heart disease (Howard *et al.*, 2003).

Many studies show that the total cholesterol/ HDL cholesterol ratio is a powerful predictor of CHD risk. Some investigators propose that this "cholesterol ratio" is a simple approach for lipid risk assessment. This ratio reflects two powerful components of risk. A

high total cholesterol is a marker for atherogenic lipoproteins, whereas a low HDL cholesterol correlates with the multiple risk factors of the metabolic syndrome and probably imparts some independent risk (Schaefer, 2002).

Several studies PROCAM (Prospective Cardiovascular Munster Study) show that high triglyceride and ratio high LDL/HDL > 5 to increase risk of several degenerative diseases. The change in ratio of total cholesterol/HDL or LDL cholesterol/HDL-cholesterol is predictor of coronary heart disease (Alwi, 1996; Ayu, 2009).

The relationship between HDL and CHD risk is complex. First, a low HDL per se may directly promote the development of coronary atherosclerosis and predispose to CHD. Several mechanisms have been implicated; impaired reverse cholesterol transport, loss of protection against atherogenicity of LDL and reduction in HDL-carried, anti-atherogenic factors. Some persons with severe deficiency of HDL do not manifest premature CHD, this suggests that HDL is not uniquely involved in atherogenesis, as is LDL (Vega, 1996). Second, a low HDL commonly is a marker for atherogenic dyslipidemia raised triglycerides and remnant lipoproteins, small LDL particles and low HDL. Both remnants and small LDL may have independent atherogenic properties. Finally, a low HDL cholesterol can be a marker for the metabolic syndrome; many persons with isolated low HDL have the other risk factors characteristic of this syndrome. Besides atherogenic dyslipidemia, these persons often have hypertension and insulin resistance, the latter being indicated by the presence of obesity (Vega, 1996; Schaefer *et al*., 1994).

The high prevalence of hypertension worldwide has contributed to the present pandemic of cardiovascular disease. During the past century, such disease has changed from a minor cause of death and disability to one of the major contributors to the global burden of disease (Murray, 1997). Cardiovascular diseases are now responsible for 30% of all deaths worldwide (WHO, 2001; Yusuf *et al*., 2001). The rapid rise in the mortality of cardiovascular disease over a fairly short period is attributable mainly to change in environmental risk factors, such as diet and physical activity (Yusuf *et al*., 2001).

The relationship between weight loss and lowered blood pressure: The coexistence of obesity and hypertension provides a compelling rationale to examine specific mechanisms that contribute to obesity and evaluate whether these mechanisms explain the relationship between blood pressure and increased levels of body fat (Brown *et al*., 2000).

In conclusion, our study results suggests that obesity in worker women is associated with disturbances in lipid metabolism and a greater risk for cardiovascular disease.

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