THE DIFFERENCE OF HEART RATE RECOVERY AMONG OBESE SUBJECTS WITH AND WITHOUT METABOLIC SYNDROME

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ABSTRAK

*Obesitas sering disertai dengan sindrom metabolik sebagai komplikasi dari obesitas. Abnormalitas pemulihan denyut jantung (HRR) sering ditemukan pada subyek obesitas dengan sindrom metabolik. Penelitian ini bertujuan untuk menganalisis perbedaan HRR antara subyek obesitas dan obesitas dengan tanpa sindrom metabolik. Metode penelitian ini adalah penelitian observasional analitik dengan desain cross -sectional. HRR diperoleh dari 68 pria obesitas tanpa sindrom metabolik dan 68 pria obesitas dengan sindrom metabolik setelah test 1 menit treadmill dilakukan. Subyek yang memiliki riwayat stroke, penyakit sendi (arthritis), penyakit ginjal kronis, penyakit jantung koroner, gagal jantung dan penyakit paru-paru kronis. HRR pada subyek obesitas tanpa sindrom metabolik lebih cepat dibandingkan subyek obesitas dengan sindrom metabolik. Variabel lain pada subyek obesitas tanpa sindrom metabolik seperti laju jantung puncak, HDL, kapasitas cardiopulmonary/Mets secara signifikan lebih tinggi dibandingkan subyek obesitas dengan sindrom metabolik. Tetapi variabel lain seperti BMI, TG, glukosa puasa, SBP beristirahat, DBP beristirahat dan HR beristirahat secara signifikan lebih rendah pada subyek obesitas tanpa sindrom metabolik yang dibandingkan dengan subyek obesitas dengan sindrom metabolik. Penelitian ini menyimpulkan bahwa tidak signifikan HRR perbedaan antara subyek obesitas dengan dan tanpa sindrom metabolik. HRR berpotensi menjadi penanda tambahan dalam keberhasilan pelatihan dan stratifikasi risiko pasien yang menjalani rehabilitasi jantung.**(FMI 2013;49:268-271)***

***Kata kunci**: pemulihan denyut jantung, obesitas, sindrom metabolik, kapasitas cardiopulmonary*

ABSTRACT

*Obesity was often accompanied by metabolic syndrome as a complication of obesity. Abnormality of heart rate recovery (HRR) was rather frequently found in obese subjects with metabolic syndrome. This research aimed to analyze the differences of HRR between obese subjects with and obesity without metabolic syndrome. The research method of this study was an observational analytic study using cross-sectional design. HRR was obtained from 68 obese men without metabolic syndrome and 68 obese men with metabolic syndrome after 1 minute treadmill test was done. Subjects who had a history of stroke, joint disease (arthritis), chronic kidney disease, coronary heart disease, heart failure and chronic lung disease. The HRR on obese subjects without metabolic syndromes was faster than obese subjects with metabolic syndrome. Other variables on obese subjects without metabolic syndrome such as peak heart rate, HDL, cardiopulmonary capacity/Mets were significantly higher than obese subjects with metabolic syndrome. But another variables such as BMI, TG, fasting glucose, resting SBP, resting DBP and resting HR were significantly lower in obese subjects without metabolic syndrome that compared to obese subjects with metabolic syndrome. This research concluded that there was significantly HRR difference between obese subject with and without metabolic syndrome. HRR potentially can become an additional markers in the succeed of training and risk stratification of patients undergoing cardiac rehabilitation.**(FMI 2013;49:268-271)***

***Keywords**: heart rate recovery, obesity, metabolic syndrome, cardiopulmonary capacity*

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INTRODUCTION

Obesity was a health problem and its prevalence has continued to increase almost become an epidemic in the world (Wilding 1997). Basic Health Research survey data in 2010 estimated that 11.7% of adults were obese and 29.5% were overweight/overweight (Ministry of Health, Republic of Indonesia 2010). Obesity closely associated with the prevalence of heart disease (Hubert et al 1983). Obesity was associated with preclinical structural and functional changes in the heart, which can develop into heart failure. Obesity-related chronic increase in left ventricular mass and increased cardiovascular morbidity and mortality (Kathrotia et al 2010, Levy et al 1990). Obesity was often accompanied by metabolic syndrome as one of the complication of obesity. Metabolic syndrome is a group of symptoms that include insulin resistance, central obesity, dyslipidemia, and hypertension. Recently, the clinical evaluation of heart rate recovery (HRR) as a diagnostic

and prognostic tool for cardiovascular disease (CVD) becomes an interesting topic. HRR has been identified as a strong and independent predictor of cardiovascular disease risk and mortality from other causes in healthy adults, in patients with CVD, and diabetes. HRR has also been reported to be a supplementary examination of individual physical fitness (Dimkpa 2009). The study was expected to show how a person's body composition will influence one indicator of cardiac fitness, which is HRR. This study aims to determine the difference of HRR on obese subjects with metabolic syndrome and obese subjects without metabolic syndrome.

MATERIALS AND METHODS

This research was an observational analytic with cross sectional design in 136 medical check up participants at LNG Badak Hospital KSO Badak-Pertamedika Bontang since April to Mei 2012. Sampling was conducted in consecutive sampling study, all subjects who were come and appropriated with the inclusion criterias in the study until the required sample size was obtained. Inclusion criterias were obesity, willing participated in the study and signed an informed consent, men aged 20-60 years, not smoking, and minimum three of metabolic syndrome criterias on obesity with metabolic syndrome group. Exclusion criterias were arthritis patient, people who suffered kidney disease, coronary heart disease, heart failure and chronic lung disease, people who comsumed beta blocker and calcium channel blockers drugs, an angina, or significant ST segment change was occurred during the test, and subjects who was intended intensive training exercise.

The research procedures started with applied the inform consent for subjects whom appropriated with inclusion criteria. Sample allocation was done into 2 group which were obesity with metabolic syndrome group and obesity without metabolic syndrome group. Data collections of primary data which include body height, body weight, resting heart rate, laboratory examination were done before the treadmill test began. Peak heart rate was obtained from QRS waves per minute maximum number of ECG recordings during the treadmill test was doing. Recovery heart rate was measured from the difference in peak heart rate with the heart rate 1 minute after recovery, in which the treadmill still running slowly. Descriptive data are shown as mean ± SD which is displayed as a percentage. Categorical data was analyzed using non-parametric test (Mann-Whitney test). Numeric data was analyzed using parametric test (independent t test). Differences were considered significant if the obtained value of $p < 0.05$.

RESULTS

The study that was included by 68 participants of each group. The descriptive data among obesity without metabolic syndrome group versus obesity with metabolic syndrome group were mean of age 45.4 vs 46.1 years; mean of BMI 27.5 vs 29.3; mean of HDL 47.3 vs 44.78 mg/dl; mean of fasting glucose 100.1 vs 130.8 mg/dl; mean of TG 129.2 vs 244.1 mg/dl; mean of resting systolic BP 122.6 vs 125.7 mmHg; mean of resting diastolic BP 81.3 vs 84.2 mmHg; mean of resting HR 79 vs 83.7 beats/min; mean of peak HR 168.9 vs 164.1 x/min, mean of cardiopulmonary capacity 11.73 vs 10.2 METs, and mean of HRR 23.2 vs 17.96 x/min (table 1).

Table 1. Descriptive data of the samples

	Groups of Obesity Without Metabolic Syndrome (mean ± SD)	Groups of Obesity With Metabolic Syndrome (mean ± SD)
N	68	68
Age	45.4 ± 8.82	46.1 ± 8.0
BMI	27.5 ± 2.08	29.3 ± 2.81
Resting systolic BP	122.6 ± 7.65	125.7 ± 9.51
Resting diastolic BP	81.3 ± 3.41	84.2 ± 5.20
HDL	47.3 ± 6.63	44.78 ± 8.21
Fasting Glucose	100.1 ± 10.46	130.8 ± 36.23
TG	129.2 ± 57.27	244.1 ± 104.07
Resting HR	79.0 ± 3.93	83.7 ± 4.35
Peak HR	168.9 ± 8.05	164.1 ± 7.18
METS	11.73 ± 1.38	10.2 ± 1.18
HRR	23.2 ± 2.27	17.96 ± 2.21

Table 2. Test of normality using the One-Sample Kolmogorov-Smirnov Test

Variables	*Asymp. Sig. (2-tailed)*
Age	0.00
BMI	0.03
Resting systolic BP	0.00
Resting diastolic BP	0.00
TG	0.41
HDL	0.24
Fasting Glucose	0.00
METS	0.01
Resting HR	0.00
Peak HR	0.16
HRR	0.22

Those data were analyzed using Kolmogorov-Smirnov Test to establish the normality of distribution. The data of peak HR and HRR were normally distribution. The data of age, BMI, resting systolic BP, resting diastolic BP, TG, HDL, fasting glucose, METs, and resting HR were not normally distribution (Table 2). There were significant differences in HRR and peak HR variables among obesity with metabolic syndrome group and

obesity without metabolic syndrome group (independent t-test, p <0.05) (Table 3)

Table 3. Independent t-test for HRR and peak HR variables

	Variables	Sig. (2-tailed)
HRR	Equal variances assumed	0.00
Peak HR	Equal variances assumed	0.00

There were significant differences in BMI, TG, HDL, Fasting Glucose, METs, resting systolic BP, resting diastolic BP, and resting HR variables among obesity with metabolic syndrome group and obesity without metabolic syndrome group (Mann-Whitney test, p <0,05). But there were no significant difference in age among obesity with metabolic syndrome group and obesity without metabolic syndrome group (p> 0.05) (Table 4)

Table 4. Mann-Whitney test for BMI, TG, HDL, GDP, METs, and resting HR variables.

Variables	*Asymp. Sig. (2-tailed)*
Age	0.81
BMI	0.01
TG	0.00
HR break	0.00
TD systolic	0.02
TD diastolic	0.03
HDL	0.00
GDP	0.00
METS	0.00

DISCUSSION

This study concluded that the main results of this study was obtained value of HRR higher (faster) on the obesity without the metabolic syndrome (23.2 ± 2.27 compared 17.96 ± 2.21, p = 0.000). This is consistent with research Sung et al (2006) that examined the association of metabolic syndrome with delayed HRR. The research was conducted in 1434 participants of medical chec-up in Samsung medical centre Korea' 248 of them with the metabolic syndrome. The results showed that the metabolic syndrome associated with delayed HRR and vagal reactivation weak (impaired vagal reactivation). HRR in subjects with metabolic syndrome obtained lower than subj oak without the metabolic syndrome (10, 3 ± 11, 6 vs. 13.6 ± ± 9, 7 per minute). It shows that metabolic syndrome was associated with decreased HRR.

In this research found that resting HR was higher in the obese group with metabolic syndrome. This is consistent with research Sung et al (2006) found that HR at rest was higher in subjects with metabolic syndrome than subjects without metabolic syndrome (64.3 ± 10.3 vs 61.6 ± 9.1 per minute). Effect of metabolic syndrome on cardiovascular system may be mediated by vagal reactivation failure due to excessive sympathetic activity (overactivity symphatetic) (Sung et al 2006). Moreover, in obese subjects without metabolic syndrome had a peak HR (168.9 ± 164.1 vs 8.05 ± 7.18, p = 0.000), HDL (47.3 ± 6.63 vs 44.78 ± 8, 21, p = 0.000), cardiopulmonary capacity/Mets (11.73 ± 1.38 vs 10.2 ± 1.18, p = 0.000) were significantly higher and BMI (27.5 ± 2.08 vs 29, 3 ± 2.81, p = 0.009), TG (129.2 ± 57.27 vs 244.1 ± 104.07, p = 0.000), systolic BP (122.6 ± 125.7 ± 7.vs 9.51, p = 0.014), diastolic BP (81.3 ± 3.41 vs 84.2 ± 5.20, p = 0.025), and GDP (100.1 ± 10.46 vs 130.8 ± 36.23, p = 0.000) were significantly lower than obese subjects with metabolic syndrome.

Believed mechanisms lead to the occurrence of the metabolic syndrome was insulin resistance and central obesity (visceral) (Alberti & Zimmet 1998). Visceral fat is more metabolically active than peripheral fat. Buildup of fat cells will increase the free fatty acids from lipolysis that will result in a lower insulin sensitivity (Williams & Pickup 1999). Insulin resistance related and mostly found along with other cardiovascular risks, such as hypertension, dyslipidemia, which are atherogenic (Stein & Colditz 2004, DeFronzo & Ferrannini 1991). Several studies have shown that weight improvements can improve blood pressure, risk of diabetes, risk of sleep apnea, hyperlipidemia, sympathetic and parasympathetic tone (Lara et al 2005)

Dyslipidemia commonly found in insulin resistance or diabetes mellitus (DM) type 2. Although the blood sugar controlled. Specific features of dyslipidemia in insulin resistance is increased TG, decreased HDL, increased small dense LDL although total LDL is sometimes normal. Dyslipidemia is also allegedly associated with hyperinsulinemia. Insulin resistance increased lipolysis, resulting in an increase in plasma free fatty acids, which in turn will increase the uptake of free fatty acids into the liver. Besides an increase in de novo synthesis of TG in the liver due to hyperinsulinemia stimulates expression of sterol regulation element binding protein (SREBP1c). Protein serves as a factor that activates gene transcription involved in lipogenesis in the liver. Cholesterol ester transferase protein and hepatic lipase also increased, resulting in an increase in VLDL1 which later became small dense LDL. Increased levels of VLDL1 increased catabolism of HDL. This leads to low HDL. Some of the above mechanisms explain the low HDL, high TG and small dense LDL in type 2 diabetes. Dyslipidemai pattern is often referred to a diabetic dyslipidemia or type B which are closely related to cardiovaskular disease in the general population (Rohman 2007).

Based on epidemiological studies, low HDL and high TG closely associated with the incidence of coronary heart disease than total cholesterol and LDL in metabolic syndrome (Adiels et al 2006). As a protective lipoproteins, HDL shown to inhibit LDL oxidation and adhesion molecules, which can inhibit foam cell formation, and in turn inhibits atherosclerosis progression. With low HDL, the protective effect was substantially reduced (Olsson et al 2005). Higher cardiopulmonary capacity in obese subjects without metabolic syndrome, which is also obtained higher HRR, consistant with previous research. Tuppo et al showed that poor physical fitness is associated with reduced cardiac vagal function during exercise (Dimkpa 2009). HRR was potential to be additional markers in success training and stratification risk patients who underwent cardiac rehabilitation (Myers et al 2007).

CONCLUSION

There was significantly HRR difference between obese subject with and without metabolic syndrome. HRR potentially can become an additional markers in the success of training and risk stratification of patients undergoing cardiac rehabilitation.

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