Effects of Plasma Lipids and Abdominal Obesity on Heart Rate Variability in Thai Overweight Dyslipidemic Individuals at Khon Kaen, Northeast Thailand

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Background: *Reduced heart rate variability (HRV), a marker of autonomic imbalance, is associated with increased cardiovascular disease (CVD) risk.*

Objective: *To investigate dyslipidemia effects on HRV and HRV correlations with plasma lipids and adiposity measures in Thai overweight dyslipidemic individuals.*

Material and Method: *HRV of 84 dyslipidemic subjects (age 48.7±6.7 years, BMI 25±2.3 kg/m^2) were compared with 20 normolipidemic subjects (56±2.3 years, BMI 26±1.7 kg/m^2). Then, HRV correlations with lipid levels and adiposity were assessed in 84 dyslipidemic subjects. HRV was determined from 10-minute continuous resting electrocardiography. Anthropometry, body composition, and plasma lipid profile, measured in blood samples after overnight fasting, were also measured.*

Results: *Total power ($p<0.01$), low-frequency power (LF) ($p<0.01$), high-frequency power (HF) ($p<0.01$), LF/HF ratio ($p<0.05$), standard deviation of R-R intervals ($p<0.01$), root mean square of successive differences ($p<0.01$), and percentage difference between adjacent normal R-R intervals >50 ms ($p<0.01$) were significantly lower in dyslipidemic than normolipidemic subjects. HF (nu) in these dyslipidemic subjects negatively correlated with triglycerides (TG), TG/high-density lipoprotein cholesterol (HDL-c) ratio, and waist-to-hip (W/H) ratio whereas, LF (nu) and LF/HF ratio positively correlated with TG, TG/HDL-c ratio, and W/H ratio.*

Conclusion: *Autonomic imbalance correlated with CVD risk factors in Thai overweight dyslipidemic individuals, suggesting increased CVD risk.*

Keywords: *Lipid profile, Autonomic imbalance, Cardiovascular disease risk*

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Dyslipidemia has been shown to increase the risk of cardiovascular disease (CVD) by causing the impairment of endothelial function[1]. Endothelial dysfunction is the initial step in the clinical manifestation of atherosclerosis and subsequent adverse cardiovascular events. This manifestation is closely linked with increased sympathetic drive in mice[2]. The sympathetic drive is a determinant of vascular tone and an indicator of adverse cardiovascular outcomes[3] such as essential hypertension[4], diabetes[5], and obesity[6]. The previous study in obesity was done in male subjects. The extent of sympathetic activation has often been correlated with the severity and prognosis of CVD[3].

The rate of cardiovascular events and mortality were shown to be associated with the responsiveness of cardiac autonomic function to dynamic environmental changes[7]. Cardiac autonomic function is measured by heart rate variability (HRV), which is a noninvasive and practical diagnostic method[7]. Published recommendations indicate that short-term HRV are suitable for analysis of cardiac autonomic function[8,9].

It has been long recognized that the cardiovascular mortality rate is increased by hypercholesterolemia[10,11] and abdominal obesity[6]. It is possible that high plasma lipid and cholesterol levels are correlated with an increased risk of CVD due to

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an autonomic imbalance. To date, only one study has examined the correlation between HRV, plasma lipid profiles, and adiposity measure in overweight dyslipidemic individuals[12]. No study has investigated this event in Thai overweight dyslipidemic individuals. Therefore, the present study aimed to investigate the effect of dyslipidemia on HRV and the correlation between HRV and plasma lipid profiles and adiposity measures in Thai overweight dyslipidemic individuals. The authors hypothesized that the Thai overweight dyslipidemic individuals had impaired HRV which was correlated with plasma lipids and adiposity.

Material and Method

Eighty-four overweight dyslipidemic (42 men and 42 women) and 20 normolipidemic subjects were enrolled. Dyslipidemia was defined as high total cholesterol (TC) levels (≥240 mg/dL), triglyceride (TG) levels (≥200 mg/dL), low-density lipoprotein cholesterol (LDL-c) levels (≥160 mg/dL), or low high-density lipoprotein cholesterol (HDL-c) levels (≤40 mg/dL)[33]. Normolipidemic subjects were healthy and had normal level of all lipid profile. All subjects were voluntarily recruited from the staff working in Khon Kaen University who participated in an annual health check-up program performed at Faculty of Associated Medical Sciences, Khon Kaen University Thailand. The overweight dyslipidemic subjects did not receive any medication. All females were post-menopausal. The present study was conducted according to the guidelines for Human Research Protection and was approved by the Ethical Committee of our university (HE 510254). Written and verbal informed consent was obtained from each subject. All subjects were screened by interview, physical and blood examinations, and health questionnaires including CVD risk factors and physical activity. Subjects with underlying CVD, hypertension, diabetes mellitus, respiratory diseases, hepatic or renal disease, cancer, or impaired physical mobility were excluded. The sample size of dyslipidemic subjects in the present study was calculated using the WINPEPI program according to the previous report of Kepez et al[37] on the evaluation of association between hyperlipidemia and HRV in subjects without apparent CVD. The decision was made to require 80% power with a significant level of 0.05. Thus, having 84 subjects was required to finish this study.

All subjects rested in supine position for 20 minutes followed by 10-minute electrocardiogram (ECG) recording (FE132 Bio Amp; ML206 Gas Analyser, ADInstruments, NSW, Australia) with controlled breathing (10 to 12 breaths/minute) to determine HRV[13]. ECG recording took place in a quiet air-conditioned room in supine position with room humidity and temperature of 58±1.9% and 25±1.1°C, respectively. HRV was determined from the time domain and frequency domain variables[13]. In the frequency domain, the total spectral power (TP, approximately ≤0.40 Hz), high-frequency spectral power (HF 0.15 to 0.40 Hz), low-frequency spectral power (LF 0.04 to 0.15 Hz), and very-low-frequency spectral power (VLF ≤0.04 Hz) were analyzed from the power spectral density curve. The normalized HF (HFnu = HF/(TP - VLF) x 100), normalized LF (LFnu = LF/(TP - VLF) x 100), and LF/HF ratio were calculated according to the procedures of the Task Force on HRV research[7]. Time-domain analysis including standard deviation of R-R intervals (SDNN), which represents total variability, the root mean square of successive differences (RMSSD), which represents the beat-to-beat variability, and the percentage difference between adjacent R-R intervals >50 ms (pNN50) were also calculated.

The height (Ht) and body weight (BW) of all subjects dressed in minimal clothing were measured using a balanced laboratory scale (Detecto, MO, USA). Body mass index (BMI) was calculated as the ratio of BW in kilograms to height in meters squared. The body fat percentage (%BF) was calculated based on biceps, triceps, subscapular, and suprailiac skinfold thickness measured using a Harpenden skinfold caliper (British Indicators Ltd., St Albans, Herts, England). Fat mass (FM) and fat-free mass (FFM) were then calculated from %BF. Waist circumference (W) was measured midway between the costal margin and iliac crest during full inspiration, and hip circumference (H) was measured around the buttocks at the level of maximal dimension in a free-standing position using a tape. W/Ht ratio was also calculated.

Blood samples were obtained from an antecubital vein after a 12-hour fast. Concentrations of TC, TG, and HDL-c were measured using a chemistry analyzer (Beckman Synchron CX4, Beckman Coulter Inc., MA, USA), and LDL-c was calculated by the Friedewald equation (LDL-c = TC - HDL-c - [0.20 x TG])[14]. These parameters were analyzed by the medical laboratory of our institution.

The Kolmogorov-Smirnov test was used to test the normality of data distribution. Normally distributed parameters were analyzed by an independent t-test and expressed as mean ± SD, mean difference,

and 95% confidence interval. Mean differences of HRV parameters were analyzed by the Mann-Whitney test and expressed as mean ± SD. After multivariate adjustment, Spearman's rho correlation coefficient was used to analyze the correlation between HRV parameters, blood parameters, and adiposity measures. Significant differences were defined as the *p*-value <0.05. All statistical analyses were performed using SPSS statistical software version 16.0 (IBM SPSS statistics, USA).

Results

Comparison of Thai overweight normolipidemic and dyslipidemic subjects

Table 1 described the characteristics of Thai overweight normolipidemic and dyslipidemic subjects. TC ($p<0.01$), TG, and TG/HDL-c ($p<0.05$) were significantly higher in Thai overweight dyslipidemic subjects than their normolipidemic counterparts. However, there were no significant differences in age, anthropometry and body composition, BP, TG, HDL-c, or LDL-c between the groups.

Overweight dyslipidemic subjects had significantly lower TP (ms^2) ($p<0.01$), LF (ms^2) ($p<0.01$), HF (ms^2) ($p<0.01$), LF/HF ratio ($p<0.05$), SDNN (ms) ($p<0.01$), RMSSD (ms) ($p<0.01$), and pNN50 (%) ($p<0.01$) than normolipidemic subjects.

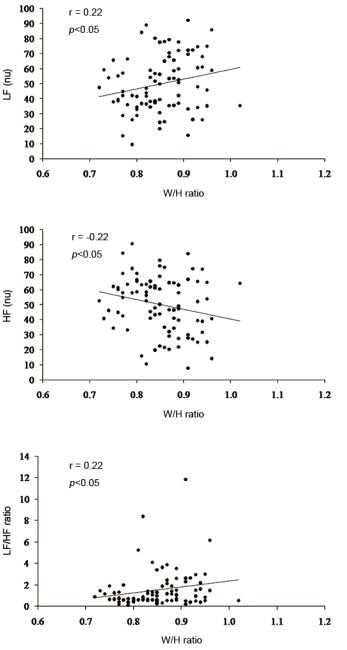

Fig. 1 Correlation between frequency domain HRV parameters and W/H ratio in dyslipidemic individuals.

Table 1. Baseline characteristics of normolipidemic and dyslipidemic individuals

	Normolipidemic (n = 20)	Dyslipidemic (n = 84)	*p*-value
Age (years)	56.1±2.3	48.7±6.7	0.49
BW (kg)	65.4±12.1	62.8±10.3	0.61
Height (cm)	160.2±9.1	159.7±10.3	0.86
BMI (kg/m^2)	25.3±2.3	25.2±3.2	0.36
%BF	32.8±6.9	30.9±4.6	0.62
FM (kg)	20.9±3.5	19.5±5.1	0.15
FFM (kg)	44.5±11.7	43.2±6.6	1.00
W (cm)	83.9±8.4	81.3±9.0	0.68
W/H ratio	0.9±0.1	0.9±0.1	0.13
W/Ht ratio	0.5±0.1	0.5±0.1	0.61
Resting HR (/minute)	68.7±13.4	67.8±7.5	0.79
TC (mg/dL)	206.0±25.8	252.1±46.6**	<0.01
TG (mg/dL)	120.2±35.4	213.7±170.2*	<0.05
HDL-c (mg/dL)	58.5±11.8	57.3±11.4	0.91
LDL-c (mg/dL)	122.6±22.1	159.0±46.2	0.33
TG/HDL-c	2.2±1.0	3.8±3.2*	<0.05

BW = body weight; BMI = body mass index; %BF = body fat percentage; FM = fat mass; FFM = fat free mass; W = waist circumference; W/H ratio = waist-to-hip ratio; W/Ht ratio = waist-to-height ratio; FM = fat mass; HR = heart rate; TC = total cholesterol; TG = triglyceride; HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol

Values are expressed as mean ± SD.

Normolipidemic individuals had normal lipid profile levels. Dyslidemia was defined as high TC levels (≥240 mg/dL), TG levels (≥200 mg/dL), LDL-c levels (≥160 mg/dL), or low HDL-c levels (≤40 mg/dL)[33]. Dyslidemic individuals in this study had high TC levels (≥240 mg/dL) and TG levels (≥200 mg/dL).

Significant differences from normolipidemic subjects were tested (* $p<0.05$, ** $p<0.01$).

However, normalized LF and HF did not significantly differ between the groups (Table 2).

Correlation between HRV variables and adiposity measures in Thai overweight dyslipidemic subjects

Spearman's rho correlation coefficient analysis indicated that among HRV variables in the frequency domain, HF (nu) were negatively correlated with the W/H ratio (p<0.05), whereas LF (nu) and LF/HF ratio were positively correlated with the W/H ratio (p<0.05) in Thai overweight dyslipidemic subjects (Fig. 1). However, there was no significant correlation between frequency domain variables and BMI, W, W/Ht ratio, %BF, or FM. There was no significant correlation between time domain variables with BMI, W, W/H ratio, W/Ht ratio, %BF, and FM (Table 3).

Correlation between HRV variables and lipid profiles in Thai overweight dyslipidemic subjects

In the frequency domain, HF (nu) was negatively correlated with TG (mg/dL) (p<0.01) and TG/HDL-c (p<0.01), whereas LF (nu) and LF/HF ratio were positively correlated with TG (mg/dL) (p<0.01) and TG/HDL-c (p<0.01) in Thai overweight dyslipidemic subjects (Fig. 2). However, there was no significant correlation between time domain and lipid profile variables (Table 4).

Discussion

The present study is the first demonstration of a reduction in the time and frequency domains of HRV in Thai overweight dyslipidemic subjects. Interestingly, after multivariate adjustment, the

Table 2. HRV parameters in normolipidemic and dyslipidemic individuals

	Normolipidemic (n = 20)	Dyslipidmia (n = 84)	Z	p-value
Total power (ms^2)	3,406±1,441	2,786±1,306**	-2.9	<0.01
LF (ms^2)	991±358	527±360**	-3.4	<0.01
LF (nu)	51.7±17.7	49.9±18.7	-1.2	0.33
HF (ms^2)	842±234	485±259**	-3.5	<0.01
HF (nu)	45.9±18.5	50.1±18.7	-1.4	0.22
LF/HF ratio	1.2±1.1	1.0±1.2*	-2.4	<0.05
SDNN (ms)	128±21	64.3±22**	-3.3	<0.01
RMSSD (ms)	25.9±9.1	17.4±13.6**	-3.8	<0.01
pNN50 (%)	5.1±4.3	3.0±2.5**	-3.7	<0.01

LF = low-frequency power; HF = high-frequency power; SDNN = standard deviation of normal R-R intervals; RMSSD = root mean square of successive differences; pNN50 = percentage difference between adjacent normal R-R intervals >50 ms
Values are expressed as mean ± SD.
Normolipidemic individuals had normal lipid profile levels. Dyslidemia was defined as high TC levels (≥240 mg/dL), TG levels (≥200 mg/dL), LDL-c levels (≥160 mg/dL), or low HDL-c levels (≤40 mg/dL)[33]. Dyslidemic individuals in this study had high TC levels (≥240 mg/dL) and TG levels (≥200 mg/dL).
Significant differences from normolipidemic subjects were tested (* p<0.05, ** p<0.01).

Table 3. Correlation between heart rate variability (HRV) parameters and adiposity measures in dyslipidemic individuals

Variables	BMI (kg/m^2)	W (cm)	W/H ratio	W/Ht ratio	%BF	FM (kg)
Total power (ms^2)	-0.07	-0.06	0.03	-0.08	-0.00	0.00
LF (ms^2)	-0.00	0.04	0.20	0.04	-0.02	0.04
LF (nu)	-0.03	0.08	0.22*	0.09	-0.14	-0.07
HF (ms^2)	-0.03	-0.06	-0.03	-0.08	0.03	0.04
HF (nu)	0.03	-0.08	-0.22*	-0.09	0.14	0.07
LF/HF ratio	-0.03	0.06	0.22*	0.11	-0.15	-0.09
SDNN (ms)	-0.06	-0.06	0.03	-0.07	0.00	0.01
RMSSD (ms)	-0.00	-0.01	0.05	-0.02	0.01	0.03
pNN50 (%)	0.02	-0.03	0.01	-0.09	-0.03	0.02

BMI = body mass index; W = waist circumference; W/H ratio = waist-to-hip ratio; W/Ht ratio = waist-to-height ratio; %BF = body fat percentage; W/Ht ratio = waist to height ratio; FM = fat mass; LF = low-frequency power; HF = high-frequency power; SDNN = standard deviation of normal R-R intervals; RMSSD = root mean square of successive differences; pNN50 = percentage difference between adjacent normal R-R intervals >50 ms
n = 84, * p<0.05
Values are expressed as Spearman's rho correlation coefficient (r).

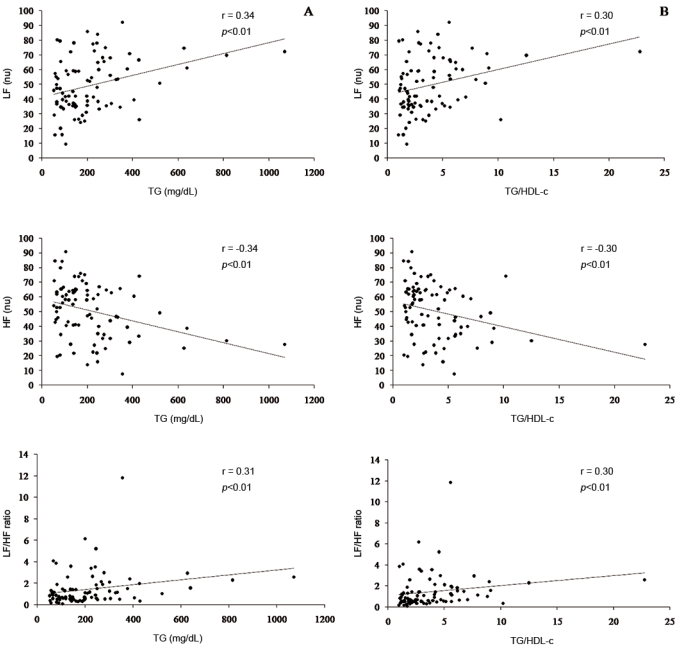

Fig. 2 Correlation between frequency domain HRV parameters and TG (A), and frequency domain HRV parameters and TG/HDL-c (B) in dyslipidemic individuals.

reduction in HRV in Thai dyslipidemic adults was correlated with increases in plasma TG, TG/HDL-c, and W/H ratio.

A few human studies have previously investigated HRV in dyslipidemic subjects, all involving Western populations[12,15,16]. Therefore, the present study is the first to show the presence of HRV in Thai dyslipidemic individuals. Although race has been reported to affect HRV[17], the present study found the similar evidence as other races showing that Thai dyslipidemic individuals had lower vagal tone, as measured by HF power, and RMSSD than normolipidemic Thai individuals. In addition, the present study also found that sympathovagal balance as measured by the SDNN and pNN50 was also decreased in overweight dyslipidemic individuals compared to normolipidemic individuals. Moreover, even in healthy subjects, cardiac autonomic nervous system activity was stimulated in the presence of elevated plasma fatty acid levels[18]. A previous animal study in hypercholesterolemic mice also reported that sympathetic activation of the vascular system resulted

Table 4. Correlation between HRV parameters and lipid profiles in dyslipidemic individuals

Variables	TC (mg/dL)	TG (mg/dL)	HDL-c (mg/dL)	LDL-c (mg/dL)	TG/HDL-c
Total power (ms^2)	-0.03	-0.21	-0.16	0.08	-0.17
LF (ms^2)	-0.10	-0.06	-0.09	-0.08	-0.03
LF (nu)	-0.05	0.34*	0.06	-0.20	0.30*
HF (ms^2)	-0.06	-0.06	-0.15	0.07	-0.18
HF (nu)	0.05	-0.34*	-0.06	0.20	-0.30*
LF/HF ratio	-0.06	0.31*	0.05	-0.23	0.30*
SDNN (ms)	-0.05	-0.17	-0.16	0.06	-0.13
RMSSD (ms)	-0.11	-0.16	-0.18	0.02	-0.12
pNN50 (%)	-0.15	-0.07	-0.17	-0.10	-0.03

TC = total cholesterol; TG = triglyceride; HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol; LF = low-frequency power; HF = high-frequency power; SDNN = standard deviation of normal R-R intervals; RMSSD = root mean square of successive differences; pNN50 = percentage difference between adjacent normal R-R intervals >50 ms

n = 84, * $p<0.01$

Values are expressed as Spearman's rho correlation coefficient (r).

in cardiac modulation[2]. This finding is consistent with the previous study[16] who observed reduced HF power in hypercholesterolemic patients of normal weight. In fact, the only previous study performed in overweight, dyslipoproteinemic individuals reported that TC was inversely correlated with the time and frequency domains of HRV[12]. However, this previous study also found that these individuals had inverse correlation between LDL-c and the time and frequency domains of HRV. The reason explaining the lack of correlation between HRV and LDL-c in the present study may be due to the normal LDL-c level in the patients in this study.

A tentative mechanism underlying this relationship could be that the sympathetic nervous activity might modulate lipoprotein metabolism[19,20]. Experimental data suggest that elevated plasma catecholamines, which include noradrenaline from the sympathetic nervous system, within the pathophysiological range increased TG and TC in cholesterol-fed rabbits[21]. This finding is consistent with the results of TG and TG/HDL-c ratio in the present study. Catecholamines are known to downregulate the LDL receptor[22] and enhance the activity of rate-limiting enzymes in cholesterol synthesis[23]. Thus, it is possible that overweight dyslipidemic individuals have relatively increased sympathetic activity, which negatively correlates with HRV. Increased sympathetic nervous system activity has been associated with endothelial dysfunction and may lead to an exaggerated pro-coagulant state, which increases the risk of atherosclerosis. Epidemiological data have established that high levels of TG, in combination with raised LDL-c levels, have an atherogenic effect that has been reported to greatly increase the risk of CVD[11].

The other mechanism that may explain the results in the present study is a reduction in baroreflex sensitivity induced by high plasma free fatty acid concentration (FFA)[24]. Although the present study did not measure FFA, high TG concentration may reflect a high FFA concentration, since TG is hydrolyzed to yield FFA and glycerol. High FFA concentration was accompanied by increased caveolar sequestration of cardiac myocardial muscarinic receptor (M2-mAChR) without changing M2-mAChR protein expression. The sequestration of M2-mAChR reduces baroreflex sensitivity. Therefore, the increased TG in the present study may reduce the baroreflex sensitivity resulting in reduced HRV in Thai overweight dyslipidemic individuals. A previous study in healthy subjects also showed that FFA induced a reduction in HRV[25]. Furthermore, TG and TG/HDL-c ratio are the most useful metabolic markers in identifying insulin-resistant individuals[34]. The presence of abnormal HRV with elevated TG/HDL-c ratio is associated with an increased risk of cardiovascular events. Taken together, the present study suggests that abnormal HRV, may identify a group of individuals with insulin resistance who have a prognostically important impairment of parasympathetic tone. However, the measurement of parameters involved in this mechanism is necessary in Thai overweight subjects to further investigate its possible involvement.

Interestingly, the reduction in HRV in overweight dyslipidemic subjects was correlated with increases in the W/H ratio. Similar findings have been reported in studies with Asians and data suggest that abdominal adiposity is more closely associated with HRV than BMI or percent body fat in Asians[35,36]. Moreover, this result is consistent with studies by Poliakova et al[26] and Indumathy et al[27], who reported that abdominal obesity was associated with specific HRV parameters indicating sympathovagal imbalance in the form of increased sympathetic and decreased parasympathetic cardiac drive[26,27]. Based on their reduction in cardiac parasympathetic activity, the subjects in the present study may have had subclinical atherosclerosis, particularly in the coronary arteries[28]. A previous study was done in post-menopausal women with psychological stress which is different group of subjects causing the impairment of endothelial function[1]. Thus, sympathovagal imbalance has been associated with a worse prognosis and increased risk of cardiovascular events[4,5].

A possible limitation of the present study is that HRV was measured from 10-minute ECG recording, while a previous study measured 24-hour ECG[29]. However, the HRV from 5-minute ECG recording is accepted as the standard method for HRV measurement[8,9,30]. The present study employed controlled breathing within 10 to 12 breaths per minute during the 10-minute ECG recording, and environmental conditions such as light, noise, and temperature during the measurement were also controlled. Therefore, the HRV data from the present study should be valid enough to measure the sympathovagal imbalance. As another possible limitation, the subjects were not asked to complete a mental health assessment, although dysfunction of the autonomic nervous system and lipid metabolism have been associated with psychiatric disorders[31,32]. However, the medical screening indicated that no subjects in the present study had

anxiety or depression. Therefore, the interpretation of HRV in these subjects was unlikely to have been affected by psychiatric factors. A major strength of the present study is that Thai normolipidemic subjects matched for age, sex, and BMI served as a control group.

Of note, HRV had inversely correlated with only W/H ratio not with other anthropometric parameters (such as BMI or W) or body composition (such as %BF or FM). This may be due to a greater positive correlation between visceral obesity (indicated by W) and sympathetic neural activity[38] and atherogenic metabolites than peripheral FM[4] (indicated by %BF or FM)[39]. In addition, though in 2010 W/Ht ratio (which is another important indicator of MetS[40] was reported to be more related with mortality than BMI[41], a cohort study in 2011 showed that W/H ratio (when adjusted for BMI) predicted cardiovascular death better than W/Ht ratio[42].

The present study is helpful by providing knowledge warning that even having overweight status, which seems to be less severe than obesity, Thai dyslipidemic individuals have impaired cardiac autonomic activity. This increases risk of CVD. Moreover, regarding quite high W/Ht ratio (0.5)[43], normolipidemic individuals in the present study have high CVD risk though they are healthy. This information should be announced to the public to help prevent both groups of people from being cardiovascular patients.

Conclusion

The present study reported that low HRV parameters were correlated with atherogenic lipid levels in Thai overweight dyslipidemic individuals. Based on the knowledge that autonomic imbalance is an independent predictor for CVD, the findings suggest that these overweight dyslipidemic individuals have high CVD risk.

What is already known on this topic?

In overweight Western dyslipidemic individuals, cardiac autonomic imbalance indicated by impaired HRV is associated with physical stress, elevated serum lipids, overweight, and risk of coronary artery disease.

What this study adds?

The present study showed that Thai overweight dyslipidemic individuals had low HRV parameters, which were correlated with atherogenic lipid levels. Together with the knowledge that autonomic imbalance is an independent predictor for CVD, high W/Ht ratio shows that these overweight individuals with dyslipidemia have a high CVD risk. Moreover, regarding quite high W/Ht ratio (0.5)[43], normolipidemic individuals in the present study have high CVD risk though they are healthy.

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Potential conflicts of interest

None.

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ผลของไขมันในเลือดและภาวะอ้วนลงพุงต่อความผันแปรของอัตราการเต้นของหัวใจในคนน้ำหนักเกินที่มีระดับไขมันในเลือดผิดปกติ จังหวัดขอนแก่น ภาคตะวันออกเฉียงเหนือ ประเทศไทย

จตุพร เพิ่มทรัพย์ทวี, สุพรรณิการ์ ลดาวัลย์, นงนุช เศรษฐเสถียร, นฤมล ลีลายุวัฒน์

ภูมิหลัง: *การลดลงของความผันแปรของอัตราการเต้นของหัวใจที่เป็นตัวบ่งชี้ของการเสียสมดุลของระบบประสาทอัตโนมัติ มักพบร่วมกับความเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดที่สูงขึ้น*

วัตถุประสงค์: *เพื่อประเมินผลของความผิดปกติของระดับไขมันในเลือดต่อความผันแปรของอัตราการเต้นของหัวใจ และเพื่อศึกษาความสัมพันธ์ระหว่างความผันแปรของอัตราการเต้นของหัวใจกับระดับไขมันในเลือด และตัวแปรของภาวะอ้วนในคนอ้วนที่มีระดับไขมันในเลือดผิดปกติ*

วัสดุและวิธีการ: *ความผันแปรของอัตราการเต้นของหัวใจของอาสาสมัครที่มีระดับไขมันในเลือดผิดปกติ 84 คน (48.7±6.7 ปี, ดัชนีมวลกาย 25±2.3 กก./ม.^{2}) ถูกนำมาเปรียบเทียบกับอาสาสมัครที่มีระดับไขมันในเลือดปกติ 20 คน (56±2.3 ปี, ดัชนีมวลกาย 26±1.7 กก./ม.^{2}) จากนั้นความผันแปรของอัตราการเต้นของหัวใจถูกนำมาหาความสัมพันธ์กับระดับไขมันในเลือดและภาวะความอ้วนในอาสาสมัครที่มีระดับไขมันในเลือดผิดปกติ 84 คน ความผันแปรของอัตราการเต้นของหัวใจวัดได้จากการบันทึกคลื่นไฟฟ้าหัวใจขณะพักนาน 10 นาที นอกจากนี้อาสาสมัครยังได้รับการตรวจวัดโครงสร้างร่างกาย องค์ประกอบของร่างกาย และระดับไขมันในเลือดหลังจากอดอาหารข้ามคืน*

ผลการศึกษา: *อาสาสมัครที่มีระดับไขมันในเลือดผิดปกติมีค่า total power (p<0.01), low-frequency power (LF) (p<0.01), high-frequency power (HF) (p<0.01), LF/HF ratio (p<0.05), standard deviation of R-R intervals (p<0.01), root mean square of successive differences (p<0.01), และ percentage difference between adjacent normal R-R intervals >50 ms (p<0.01) น้อยกว่าอาสาสมัครที่มีระดับไขมันในเลือดปกติ นอกจากนี้พบความสัมพันธ์เชิงลบระหว่าง HF (nu) กับ triglyceride (TG), TG/high-desity lipoprotein cholesterol (HDL-c) ratio และอัตราส่วนของเส้นรอบเอวกับสะโพก และพบความสัมพันธ์เชิงบวกระหว่าง LF (nu) และ LF/HF ratio กับ TG, TG/HDL-c ratio และอัตราส่วนของเส้นรอบเอวกับสะโพก*

สรุป: *การเสียสมดุลของระบบประสาทอัตโนมัติอาจจะมีความสัมพันธ์กับปัจจัยเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดในคนน้ำหนักเกินที่มีระดับไขมันในเลือดผิดปกติ แสดงให้เห็นถึงการมีความเสี่ยงต่อการเกิดโรคหัวใจและหลอดเลือดเพิ่มขึ้น*