CHAPTER IV

RESULTS

Three hundred and forty seven healthy adults were enrolled in this study and the ages of participants ranged from 40 to 80 years old. Fifty-five percent (191/347) were male. Age and biochemical measurements were shown in median and 95 % confidence interval. The median of age, glucose, TC, TG, HDL-C, LDL-C and hs-CRP concentration were 51 (48 – 71), 88 (75 – 116), 211 (137 – 317), 114 (85 – 466), 51 (36 – 84), 131 (90 – 233), and 1.33 (0.20 – 5.38) respectively. Baseline characteristics of all subjects were shown in Table 7.

There were 60 % (207/347) of normal weight (BMI<25.00), 37 % (130/347) of overweight (BMI ≥ 25.00 - 29.99), and 3 % (10/347) of obesity (BMI ≥ 30). There were 45 % (156/347) of central obesity and 55 % (191/347) of non-central obesity (normal waist circumference).

The comparison of anthropometric data between non-central obesity (normal waist circumference) and central obesity participants indicated that the mean of BMI, median of LDL-C and hs-CRP among non-central obesity participant were significant lower than those among central obesity participants ($p < 0.0001$, $p = 0.0001$, $p < 0.0001$ respectively). Whereas, the median of HDL-C among non-central obesity participants was significant higher than those among central obesity group ($p = 0.0002$) (Table 8).

In all participants, if defined risk of CVDs which classified by hs-CRP concentrations, there were 56.2 % (195/347) of low risk, 29.1 % (101/347) of moderate risk, and 14.7 % (51/347) of high risk as shown in Table 9.

The concentration of serum hs-CRP > 3 mg/L was found in central obesity 21 % higher than those found in normal waist circumference when the Chi square statistical methods was applied for data analysis as shown in Table 10. This may due to central obesity had been associated with adipose mass and expression of the proinflammatory gene tumor necrosis factor-α (Hotamisligil, G.S., 1993)

Table 11 classified peripheral vascular types by using PWV and ABI into 4 type; Peripheral vascular type I is a normal type of peripheral vascular defied by normal PWV and normal ABI. The feature of this type is normal without harder and occlusion vascular.

Peripheral vascular type II is an abnormal type of peripheral vascular defied by slightly increase PWV and slightly decrease ABI. The feature of this type is slightly harder, possible occlusion vascular or slightly harder, slightly occlusion vascular.

Peripheral vascular type III is an abnormal type of peripheral vascular defied by increase PWV and normal ABI. The feature of this type is stiffness vascular.

Peripheral vascular type IV is an abnormal type of peripheral vascular defied by increase PWV and slightly or moderate decrease ABI. The feature of this type is stiffness, possible occlusion vascular or stiffness, moderate occlusion.

There was 40.9 % (142/347) of normal vascular and 59.1 % (205/347) of abnormal vascular. The abnormal features were 32.9 % (114/205) of slightly harder with slightly occlusion, 23.9 % (83/205) of stiffness without occlusion, and 2.3 % (8/205) stiffness with occlusion as show in Table 12.

Peripheral vascular type IV was found in central obesity 5 % higher than those found in normal waist circumference when the Chi square statistical methods was applied for data analysis as shown in Table 13.

Stepwise regression analysis among variables, hs-CRP, BMI, WC, SBP, DBP, PWV, ABI, GLU, TC, TG, HDL, and LDL was shown in Table 14. There were three final fitted models to predict hs-CRP, PWV, and ABI ($P<0.05$). Serum hs-CRP was associated with PWV, ABI, WC, and TG. Decreasing in 0.1 of ABI and increasing in 10 cm of WC resulted in increasing of hs-CRP by 0.4 and 0.2 mg/L, respectively. In addition, the stepwise regression analysis also shows that increasing in 1 mg/L of hs-CRP will increase PWV by 19 cm/s and will decrease ABI by 0.012.

Medians of hs-CRP were compared among non-central and central obesity adults with and without abnormal peripheral vascular as shown in Figure 9. The hs-CRP in non-central obesity (0.62 mg/L) was not significant different ($P>0.05$) to central obesity (0.72 mg/L). However, there was significant difference ($P<0.001$) of

hs-CRP between non-central obesity (0.95 mg/L) and central obesity (2.31 mg/L) with presented peripheral vascular atherosclerosis.

Table 7 Anthropometric data of all participants

Variable	Min	Max	Mean or Median*	SD or 95 %CI**
Age, years	40	80	51*	48 - 71**
BMI, kg/m^2	17	30	24	3
WC, cm	60	106	83	10
SBP, mmHg	88	159	123	12
DBP, mmHg	58	101	74	7
RPWV, cm/s	1113	2193	1461	193
LPWV, cm/s	1094	1984	1419	191
RABI	0.83	1.28	1.06	0.08
LABI	0.83	1.28	1.07	0.08
GLU, mg/dL	68	126	88*	75 - 116**
mmol/L	3.8	7.0	4.9*	4.2 – 6.4**
TC, mg/dL	97	376	211*	137 - 317**
mmol/L	2.5	9.7	5.5*	3.5 – 8.2**
TG, mg/dL	40	638	114*	85 - 466**
mmol/L	0.5	7.2	1.3*	1.0 – 5.3**
HDL-C, mg/dL	20	97	51*	36 - 84**
mmol/L	0.5	2.5	1.3*	0.9 – 2.2**
LDL-C, mg/dL	61	270	131*	90 - 233**
mmol/L	1.6	7.0	3.4*	2.3 – 6.0**
hs-CRP, mg/L	0.12	6.74	1.33*	0.20-5.38**

* Data is showing in median

** 95% of confidential interval

Table 8 Comparison of anthropometric data between non-central obesity and central obesity

Variable	Non-central obesity (n = 191)		Central obesity (n = 156)		p-value
	Mean or Median*	SD or 95 %CI**	Mean or Median*	SD or 95 %CI**	
Age, years	51	47 - 71	51*	48 - 71**	NS
BMI, kg/m^2	21	3	27	3	<0.0001
SBP, mmHg	124	13	122	10	NS
DBP, mmHg	73	8	74	7	NS
RPWV, cm/s	1463	193	1461	192	NS
LPWV, cm/s	1419	196	1421	190	NS
RABI	1.07	0.07	1.06	0.08	NS
LABI	1.08	0.07	1.07	0.08	NS
GLU, mg/dL	92*	74 - 110**	90*	77 - 120**	NS
mmol/L	5.1*	4.1 – 6.1**	5.0*	4.3 – 6.7**	
TC, mg/dL	200*	135 - 316**	223*	139 - 320**	NS
mmol/L	5.2*	3.5 – 8.2**	5.8*	3.6 – 8.3**	
TG, mg/dL	104*	80 - 469**	125*	84 - 470**	NS
mmol/L	1.2*	0.9 – 5.3**	1.4*	0.9 – 5.3**	
HDL-C, mg/dL	55*	40 - 91**	48*	35 - 80**	0.0002
mmol/L	1.4*	1.0 – 2.4**	1.2*	0.9 – 2.1**	
LDL-C, mg/dL	124*	71 - 220**	138*	92 - 241**	0.0001
mmol/L	3.2*	1.8 – 5.7**	3.6*	2.4 – 6.3**	
hs-CRP, mg/L	0.64*	0.12 – 5.01**	1.09*	0.22-5.42**	<0.0001

* Data is showing in median

** 95% of confidential interval

Table 9 The risk of CVDs and hs-CRP levels in all participants

Subject	Low risk* hs-CRP < 1 mg/L n (%)	Moderate risk* hs-CRP 1 – 3 mg/L n (%)	High risk* hs-CRP > 3 mg/L n (%)
Healthy adults (n = 347)	195 (56.2)	101 (29.1)	51 (14.7)

* CVDs risk defined by CDC/AHA, The hs-CRP results were excluded if the concentration was more than 10 mg/L

Table 10 The risk of CVDs and hs-CRP levels in non-central obesity and Central obesity

Subject	Low risk* hs-CRP < 1 mg/L n (%)	Moderate risk* hs-CRP 1 – 3 mg/L n (%)	High risk* hs-CRP > 3 mg/L n (%)	Chi-sq (p-value)
Non-central obesity (n = 191)	119 (62)	54 (28)	18 (10)	10.96 (0.004)
Central obesity (n = 156)	76 (49)	47 (30)	33 (21)	

* CVDs risk defined by CDC/AHA

Table 11 Classifications and characteristics of peripheral vascular type

PWV	ABI	Classification*	Characteristic	Interptetation
Normal	Normal	Type I	Normal without harder and occlusion	Normal
Slightly increase	Slightly decrease	Type II	Slightly harder, possible occlusion Slightly harder, slightly occlusion	Abnormal
Increase	Normal	Type III	Stiffness	Abnormal
Increase	Slightly or moderate decrease	Type IV	Stiffness and moderate or possible occlusion	Abnormal

* Vascular types were classified by using PWV and ABI

Table 12 Peripheral vascular types

Subject	Peripheral vascular types*			
	Type I n (%)	Type II n (%)	Type III n (%)	Type III n (%)
Healthy adults (n = 347)	142 (40.9)	114 (32.9)	83 (23.9)	8 (2.3)

* Type I, normal; type II, slightly harder, possible occlusion, slightly harder and slightly occlusion; type III, stiffness; type IV, stiffness and moderate or possible occlusion

Table 13 Peripheral vascular types in non-central obesity and central obesity

Subject	Peripheral vascular type*				(P-value) †
	Type I n (%)	Type II n (%)	Type III n (%)	Type IV n (%)	
Non-central obesity (n = 191)	87 (46)	65 (43)	39 (20)	0 (0)	0.0015
Central obesity (n = 156)	55 (35)	49 (32)	44 (28)	8 (5)	

* Type I, normal; type II, slightly harder, possible occlusion, slightly harder and slightly occlusion; type III, stiffness; type IV, stiffness and moderate or possible occlusion

† P-value is analyzed by Fisher's Exact test

Table 14 Stepwise regression analysis

Dependent variable	Regression model	R^2	Adjusted R^2	P-value
hs-CRP	1.52 + 0.002 (PWV) – 4.000 (ABI) + 0.020 (WC) + 0.002 (TG)	0.106	0.097	0.013
PWV	520 + 6.250 (SBP) -2.540 (GLU) + 18.500 (hs-CRP) + 332.000 (ABI)	0.230	0.221	0.008
ABU	1.02 + 0.0002 (TG) - 0.012 (hs-CRP) + 0.00006 (PWV) – 0,0002 (TC)	0.126	0.116	0.004

hs-CRP, high sensitivity C-reactive protein; PWV, pulse wave velocity; ABI, ankle brachial index; WC, waist circumference; TG, triglyceride; SBP, systolic blood pressure; GLU, glucose; TC, Total cholesterol; R^2, coefficient of multi-regression; adj R^2, coefficient of multi-regression after adjusted.

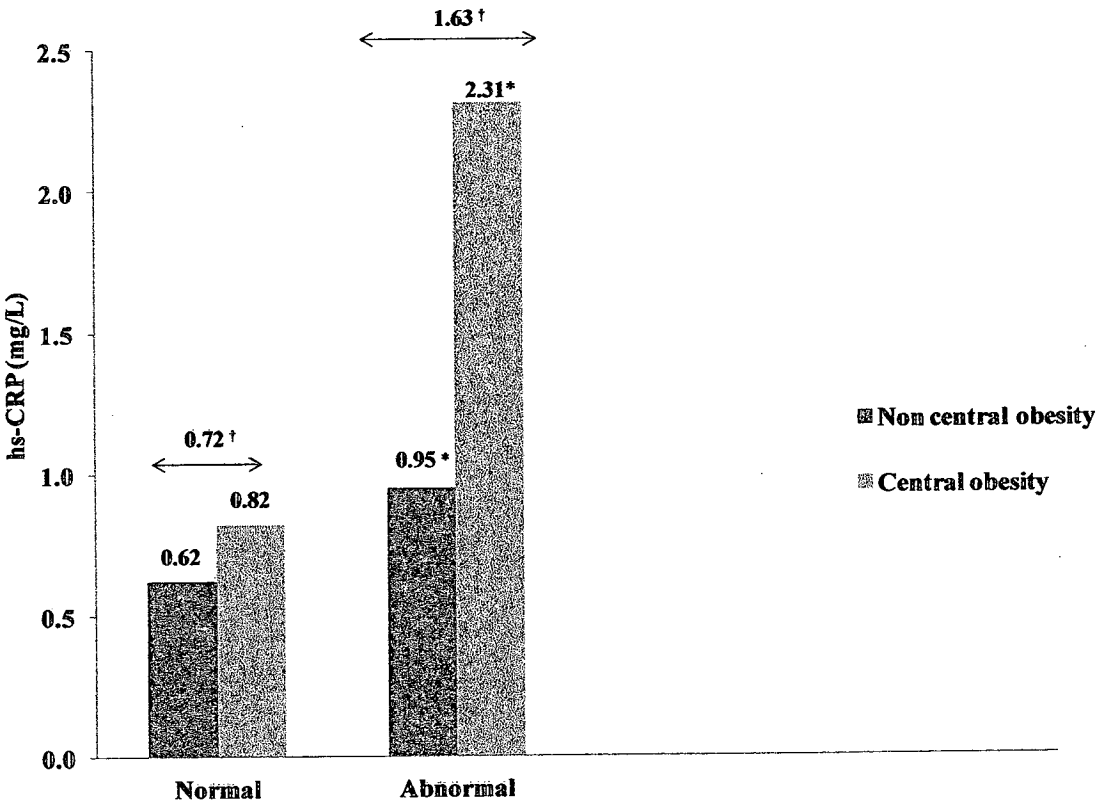

Figure 9 Median of high sensitivity C-reactive protein (hs-CRP) in normal and abnormal peripheral vascular characteristics in non-central obesity and central obesity. Peripheral vascular types were categorized by using angle brachial index (ABI) and pulse wave velocity (PWV). High sensitivity C-reactive protein concentration was determined in mg/L. Normal and abnormal were indicated normal and abnormal peripheral vascular atherosclerosis. *Significant differences between non-central and central obesity groups, P< 0.001; †Significant differences between normal and abnormal types, P< 0.001