Original Article

Abdominal Fat Distribution and Serum Lipids in Patients with and without Coronary Heart Disease

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Abstract

Objective: To investigate the association between obesity indices, abdominal fat distribution, and lipid profile in patients with stable angina (SA).

Methods: Body weight, height, waist circumference (WC), body mass index (BMI), and waist /height ratio (WHtR) of 123 patients with SA who underwent coronary angiography were measured. Fasting blood samples were taken to measure the levels of fasting blood sugar (FBS), total cholesterol (TC), low- and high-density lipoprotein cholesterol (LDL-C, HDL-C), apolipoproteins A and B (apo A and apo B), and triglycerides (TG). According to angiography reports, the participants were divided into patients with or without coronary heart disease (CHD). All patients underwent an abdominal computerized tomography (CT) scan to measure the visceral, superficial, and deep subcutaneous fat.

Results: The mean ages of the patients with CHD (n = 73) and without CHD (n = 50) were 50.5 ± 7.6 and 53.7 ± 7.6 years, respectively (P = 0.03). The patients with CHD had significantly higher levels of TC, TG, and superficial subcutaneous fat, while the patients without CHD had higher levels of apo A (P ≤ 0.05). Multivariate analyses showed a significant association of visceral fat with TC, LDL-C, TG, and apo B, in the patients without CHD, while significant inverse associations were found between WC and HDL-C, WHtR, and apo A as well as visceral fat and LDL-C in the patients with CHD.

Conclusions: Among anthropometrics and imaging indices of obesity, WC and WHtR have shown better association between central obesity with dyslipidemia in the patients with CHD, while CT-measured visceral adipose tissue area was the best correlate of dyslipidemia in the patients without CHD.

Keywords: Body fat distribution, body mass index, coronary heart disease, intra-abdominal fat, waist circumference

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Introduction

Coronary heart disease (CHD) has emerged as a global health-threatening problem.[1] Many metabolic and physical risk factors have been identified for this chronic condition, including dyslipidemia, obesity, and their interactions.[2–6]

Obesity level has been assessed using various methods. Body mass index (BMI) has a confirmed value in predicting overall obesity and was used by the World Health Organization (WHO) to define obesity categories.[7] However, in various national studies and populations, the predictive value of BMI has become ambiguous in recent years.[8–10] On the other hand, in a number of studies, patients with normal BMI values have shown excessive central fat accumulations, a finding which underscores limitations of this index as a predictive tool for obesity-related metabolic alterations.[10–11]

Body composition and the distribution of adipose tissue have now emerged as critical issues in assessing the link between obesity and metabolic adverse outcomes;[12–14] for instance, in some studies waist circumference (WC) and the waist/height ratio (WHtR) were found to be imprecise measures of fat distribution in CHD patients.[15–16] Moreover, there is a controversy about the respective contribution of visceral and subcutaneous body fat, and their relative impact on the development of CHD.[17–18]

Previous studies established a link between lipid profile and CHD; however recent studies reported the different effect of various kinds of dyslipidemia and apolipoproteins on CHD based on different obesity indices. Furthermore, the dependent or independent role of lipid profile in developing CHD is questionable.[19–21] According to the existing controversies about the relationship between the body fat distribution and CAD, this study was designed to investigate the association of different indices of obesity (BMI, WC, WHtR), fat distribution indicators (visceral and subcutaneous fat), and lipid profile in patients with stable angina (SA) with and without CHD.

Subjects and Methods

Study population

This cross-sectional study was performed in Isfahan Cardiovas-

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cular Research Center (a WHO-Collaborating Center) on 123 patients with SA who underwent coronary angiography. SA is a typical angina pectoris brought on by exertion and relieved by rest or the use of nitroglycerin.[22] The patients were 35 – 75 years old and were consecutively referred for the evaluation of their chest pain in 2008. Before performing the angiography, the patients who agreed to participate signed an informed consent. Demographic data as well as the medical and drug consumption history of the patients were collected using a questionnaire. Patients with a history of chronic renal failure, chronic or acute hepatitis, congenital and valvular heart diseases, myocardial infarction, heart failure, and pregnant women were excluded. Participation to any weight reduction program (including diet) was also considered among exclusion criteria. This study was reviewed and approved by the Ethical Committee of Isfahan Cardiovascular Research Center.

Anthropometrics and laboratory data measurements

The patients' weight and height were measured while they were wearing light indoor clothing without shoes. BMI was calculated as weight (in kg) divided by the square of height (in meter); horizontal tape measures at the umbilical level (1cm above the navel) were used for determining waist circumference (WC).[15]

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured twice with five minutes intervals using a standard mercury sphygmomanometer in a quiet and comfortable room; the mean of both SBP and DBP measurements were used for analyses.

On the morning of catheterization, blood samples were taken after 12 hours of fasting for measuring total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL-C), low- density lipoprotein (LDL-C), and apolipoproteins A and B (apo A, and apo B). Measurement of TC, TG, HDL-C, and LDL-C levels was carried out by the enzymatic method. Immunoturbidimetric method was used to measure the levels of apo A and apo B. Pars Azmoon Kits accredited by Bioactiva Dignostica (Germany) was used for all the above- mentioned biochemical measures.

Hypertension was defined as a SBP and/or DBP at or above 140/90mmHg or receiving antihypertensive drugs. LDL-C $\geq$ 100 mg/dL or HDL-C $\leq$ 40 mg/dL in male and $\leq$ 50mg/dL, in female, or TG $\geq$ 150mg/dL were considered as dyslipidemia for both sexes.[23]

Determination of coronary artery involvement and body composition

Coronary angiography was carried out by left-heart catheterization and arteriography using Judkins method,[24] and then two cardiologists separately reviewed the angiography films and if they were agreed on the stenosis $\geq$ 50% of any of coronary arteries, the patient was considered as CHD-positive. According to angiography reports, the patients were divided into two groups; patients with (n = 73) or without CHD (n = 50).

Three days after undergoing coronary angiography, each patient underwent abdominal CT scan using a Philips Medical Systems CT (TOMO SCAN AV). With a collimation of 5mm and no overlap, a scout view and four cuts tangentially at the distal of the L4 inferior end-plate were taken. Scan parameters were: 120kV, 250 mA, slice thickness: 5mm, field of view: 500 mm, window width: 500, window center: 40. Images were transferred to the DICOM (Digital Imaging and Communications in Medicine) workstation, and analyzed with freeware ImageJ software version 1.36. Adipose tissue area was measured in mm^2 in different compartments. For each patient, the measurements were performed on four images and the averages were used for analysis.[25]

Statistical Analyses

Statistical analyses were carried out using SPSS software (version 15.0, Chicago, IL, USA). Unpaired Student t-tests were used for comparing continuous variables. To evaluate the possible correlation between body fat distribution indicators and lipid profile measures, Pearson correlation coefficients were used as the association of different measures of body fat distribution (A) was separately evaluated with TC , LDL-C, HDL-C, TG, apo B, and apo A in age and sex adjusted models, as well as full adjusted models(hypertention [history of high blood pressure {SBP $\geq$ 140 or DBP $\geq$ 90} and/or using any hypertensive drugs), dyslipidemia (history of dyslipidemia and/or TC $\geq$ 200, and/or TG $\geq$ 150 and/or LDL-C $\geq$ 100 and/or HDL-C $\leq$ 40 in men and $\leq$ 50).

Results

One hundred and twenty- three patients with SA who underwent angiography (age 35 – 75 years) were enrolled in the study. Among the participants, 55% and 58.8% were females in the CHD- positive and -negative groups, respectively. The mean age of the CHD- positive and -negative groups were 50.5 ± 7.6 and 53.7 ± 7.6 years, respectively (P = 0.03). Characteristics of the study population are presented in Table 1. The patients with CHD had significantly higher levels of TC (P $\leq$ 0.001), TG (P = 0.02), and superficial subcutaneous fat (P $\leq$ 0.001). The patients without CHD had a significantly higher levels of apo A (P = 0.03).

As shown in Table 2, in patients without CHD, visceral fat correlated positively with LDL-C, TG, and apo B levels, and inversely with HDL-C levels. In patients with CHD, WC was inversely correlated with HDL-C and apo A levels. Furthermore, visceral fat inversely correlated with apo B level. In patients with CHD, CT-measured visceral adipose tissue area was significantly and positively associated with TC and LDL-C , TG, and apo B levels. A negative association was observed between visceral adipose tissue area and HLD-C levels.

Adjusted correlations between adiposity measures and blood lipid profile variables are shown in Table 3. Visceral fat area was associated with TC, LDL-C, TG, and apo B levels and inversely associated with HDL-C level in patients without CHD independent of age and sex. Furthermore, in this group, superficial subcutaneous fat was associated with LDL-C level after adjustment for age, sex, and dyslipidemia. Also, WHtR was inversely associated with HDL-C only in the second step of data analysis.

In patients with CHD, WC was inversely associated with HDL-C and apo A levels; in addition WHtR was inversely associated with apo A level, and visceral fat was inversely associated with LDL-C level in both data analysis steps. Visceral fat was inversely associated with HDL-C level, when the effects of age and sex were adjusted. WHtR was inversely associated with TC, and visceral fat inversely associated with apo A only in the second step of data adjusting.

Discussion

Among the body fat distribution indicators, superficial subcutaneous fat level was higher in patients with CHD compared to those without CHD. In addition, TC and TG levels were higher in this group. WC and WHtR were the only indicators of body fat

Table 1. Background characteristics of the study population

Variable	Coronary heart disease-negative	Coronary heart disease-positive	Total	P- value
BMI*	27.81 ± 0.39	28.87 ± 0.44	28.39 ± 0.30	0.740
Waist/height	0.619 ± 0.006	0.632 ± 0.007	0.626 ± 0.005	0.221
Waist(cm)	100.44 ± 0.94	101.48 ± 0.93	101.01 ± 0.66	0.436
Visceral fat	11538.46 ± 734.70	11612.21 ± 563.95	11584.91 ± 445.90	0.937
Deep SQ fat**	14112.81 ± 816.399	15279.005 ± 695.53	14847.36 ± 532.499	0.292
Superficial SQ fat	1111.97 ± 873.80	13620.63 ± 730.33	12694.32 ± 569.01	0.033
Total SQ fat	25230.79 ± 1543.64	28899.63 ± 1278.08	27541.68 ± 994.43	0.075
Total cholesterol (mg/dL)	180.56 ± 4.92	200.79 ± 4.54	191.29 ± 3.41	0.003
Triglycerides (mg/dL)	160.48 ± 10.05	196.89 ± 12.38	179.79 ± 8.18	0.024
HDL-C (mg/dL)	35.77 ± 1.15	32.89 ± 1.21	34.25 ± 0.84	0.366
LDL-C (mg/dL)	107.23 ± 3.42	115.83 ± 3.26	111.79 ± 2.37	0.710
Apo A(mg/dL)	148.31 ± 2.87	158.96 ± 2.74	153.96 ± 2.02	0.008
Apo B(mg/dL)	98.75 ± 3.09	106.51 ± 2.74	102.86 ± 2.07	0.610

*BMI: body mass index; **SQ: subcutaneous.

Table 2. Association of lipid profile with different measures of fat distribution in the participants with or without CHD

Variable	BMI		Waist		Waist / Height		Visceral fat		Total SQ** fat		Sup SQ fat		Deep SQ fat	
	¶CHD positive	CHD Negative	CHD positive	CHD Negative	CHD positive	CHD Negative	CHD positive	CHD Negative	CHD positive	CHD Negative	CHD positive	CHD Negative	CHD positive	CHD Negative
Total cholesterol(mg/dL)	0.002	0.10	-0.17	0.10	0.66	0.12	-0.12	0.34*	0.04	0.26	0.09	0.21	-0.01	0.26
LDL-C(mg/dL)	-0.03	0.06	-0.14	0.04	0.08	0.04	-0.19	0.35*	0.03	0.17	0.04	0.22	0.02	0.09
HDL-C(mg/dL)	-0.08	-0.01	-0.23*	-0.04	0.09	0.03	-0.20	-0.39*	-0.09	0.20	-0.04	0.23	-0.12	0.14
Triglycerides(mg/dL)	0.02	0.18	-0.01	0.16	0.02	0.19	0.15	0.37*	-0.01	0.10	0.00	-0.03	-0.04	0.23
Apo A (mg/dL)	0.002	0.01	-0.28*	-0.01	-0.03	0.06	-0.05	-0.11	0.09	0.19	0.20	0.14	-0.05	0.20
Apo B (mg/dL)	0.01	0.12	0.11	0.13	0.06	0.15	-0.07*	0.53*	0.03	0.21	0.05	0.19	-0.006	0.20

*P- value ≤ 0.05; **Subcutaneous; ¶Coronary heart disease.

Table 3. Association of coronary heart disease, physical, and metabolic risk factors with different measures of body fat distribution

		BMI		Waist		Waist/Height		Visceral fat		Total SQ fat		Superficial SQ fat		Deep SQ fat	
		¶CHD Positive	CHD Negative	CHD Positive	CHD Negative	CHD Positive	CHD Negative	CHD Positive	CHD Negative	CHD Positive	CHD Negative	CHD Positive	CHD Negative	CHD Positive	CHD Negative
Total cholesterol (mg/dL)	Adjusted**	-.072	.063	106*	.063	-.221	.066	-.230	.202	- .025	.363	.051	.336	-.064	.266
	Adjusted†	-.113	.051	146*	.050	-.251*	.047	-.258	.216	-.016	.352	.050	.347	-.055	.255
LDL-C (mg/dL)	Adjusted**	-.085	.057	-.172	.027	-.204	.026	-.303*	.352*	-.013	.324	-.019	.552*	-.005	.106
	Adjusted†	-.108	.064	-.195	.045	-.221	.049	-.310*	.373*	.006	.368	-.019	.563*	.021	.152
HDL-C (mg/dL)	Adjusted **	-.148	-.158	-.265*	-.138	-.233	-.239	-.281*	-.437*	-.180	.040	-.130	.047	-.154	.024
	Adjusted†	-.106	-.192	-.242*	-.196	-.234	-.334*	-.230	-.425*	-.148	-.041	-.121	.052	-.119	-.084
Triglycerides (mg/dL)	Adjusted**	.033	.174	-.017	.158	-.016	.227	.192	.344*	.028	.113	.099	-.250	-.025	.267
	Adjusted†	-.016	.165	-.055	.152	-.038	.226	.148	.342*	.0005	.081	.094	-.242	-.055	.246
Apo B (mg/dL)	Adjusted**	-.022	.102	-.127	.107	-.149	.145	-.136	.501*	-.016	.265	.029	.273	-.039	.180
	Adjusted†	-.072	.105	-.172	.118	-.177	.165	-.182	.503*	-.024	.296	.025	.272	-.049	.226
Apo A (mg/dL)	Adjusted**	-.147	-.087	-.330*	-.085	-.309*	-.108	-.223	-.182	-.175	.054	-.026	-.103	-.212	.119
	Adjusted†	-.163	-.115	-.352*	-.133	-.330*	-.177	-.234	-.192	-.164	-.015	-.018	-.102	-.209	.039

*P- value ≤ 0.05; ¶Coronary heart disease; Adjusted"**: age and sex adjusted; Adjusted † history of high blood pressure and / or using any hypertensive medication, and / or SBP ≥ 140 and / or DBP ≥ 90, history of dyslipidemia and / or TC ≥ 200 and/or TG ≥ 150 and/orLDL-C ≥ 100 and / or HDL C ≤ 40 in men and ≤ 50 in women was used.

distribution that were associated with serum lipids in the patients with CHD. Serum lipid levels of the patients without CHD were only associated with CT-measured visceral fat.

In many studies such as the survey carried out by Lamon-Fava, et al.[26] BMI was considered as one of the CHD risk factors. However, more recent studies such as that of Romero-Corral, et al.[11] showed that this index of obesity was not an independent CHD predictor. In the current study, although the patients with CHD had slightly higher BMI values compared to non-CHD patients, this difference did not reach statistical significance and both groups were in the overweight category. This finding is consistent with the results obtained by Romero-Corral, et al.[11] Other findings of the present study showed that BMI is not associated with serum lipids, which is in accordance with the study that reported that BMI variation is independent of serum lipid levels in patients with CHD.[27] Other indicators of specific types of obesity, including WC and WHtR were introduced as CHD risk factors have also been considered.[15] WC and WHtR of the patients with CHD were associated with lipid profile variables in our study. This finding is in agreement with the study by Thomas, et al.[28] which suggested that obesity measurements which indicate visceral and subcutaneous abdominal obesity[29] possibly interact with serum lipids in CHD patients.

Our results show that serum lipid levels are associated with visceral fat in the patients without CHD in line of findings obtained in the studies carried out by Rosito, et al.[30] and Fax, et al.[31] These outcomes observed in their studies were obtained in normal populations. Nevertheless, we showed this association in the patients without coronary artery involvement. In the patients with CHD, visceral fat did not associate with the levels of serum lipids. By reviewing other literature which evaluated this relationship in patients with acute MI[32] and in patients with higher burden of artery plaques,[33] it was found that visceral fat was associated with the occurrence of acute MI only in female patients and this association is independent of lipid profiles. Although in the current study we did not evaluate the burden of plaque or coronary artery lesions, our findings did not conflict with the results of the aforementioned studies and showed that visceral fat probably does not associate with coronary artery involvement in patients with CHD; this result is in accordance with those of two other studies which reported intrathoracic artery calcification as one of the atherosclerosis indicators which is associated with visceral fat and metabolic risk factors.[17,18]

It is quite interesting that visceral fat was not different between the CHD and non-CHD groups. It Could be because both subgroups are already at high risk of developing CHD when they present with SA. The fact that both groups of patients were overweight and had a relatively high WC seems to support this notion. The difference of having CHD or not in this case would be predicted, as we mention, by other factors not considered in the analyses. Another interesting finding is that the CHD group, despite having similar (or non-significantly higher) visceral fat, does have higher triglycerides. Thus, it could mean that the CHD group has hypertrophic, hyperlipolytic adipose tissue compared to the non-CHD group despite a similar (visceral) fat mass, suggesting inability of their adipose tissues to handle daily lipid fluxes. This would in turn lead to high TG levels.

After the adjustment for history of hypertension and dyslipidemia, visceral fat was inversely associated with LDL-C level in the patients with CHD. This may be due to statin consumption of the patients in the present study who were not excluded.

Our results showed higher levels of superficial subcutaneous fat in the patients with CHD, which is in agreement with the results reported by Fox, et al. in 2009 and 2007.[18,31] Nonetheless, lipid profile values in the patients with CHD in the current study were not associated with superficial subcutaneous fat area. Hence, the relationship between superficial subcutaneous fat and CHD may be attributable to other risk factors, which we did not evaluate in the present study, e.g., fasting blood glucose and blood pressure, rather than the lipid profile. The correlation between subcutaneous fat area and fasting glucose or blood pressure has been reported in other studies.[21]

Limitations of our study include its small sample size, the cross-sectional nature of the study, and the fact that patients who took statins were not excluded from the study.

Among anthropometric measures and imaging indices of obesity, WC and WHtR measures of abdominal fat were related to lipid profile alterations in the patients with CHD, while CT-measured visceral fat predicted lipid profile values in non-CHD patients. Simple anthropometric indicators must be considered in the clinical evaluation of patients with coronary artery disease.

References

1. Chrysochou C, Kalra PA. Epidemiology and natural history of atherosclerotic endovascular disease. *Prog Cardiovasc Dis*. 2009; **52(3):** 184 – 195.
2. Palma Gamiz JL, Conget DI, Bertomeu GV, Ascas Gimilio JF, Gonzalez Jr, Alegria EE, et al. Prevalence of the metabolic syndrome in Spanish patients with established cardiovascular disease: CLYDIA study. *Med Clin (Barc).* 2007; **128(11):** 407 – 413.
3. Couffinhal T, Boulon C. Metabolic syndrome with a predisposition to coronary diseases and type 2 diabetes. *Nephrol Ther.* 2006; **2(1):** 39 – 48.
4. Haddad FG, Brax H, Zein E, Abou EH. Obesity and related diseases in a Lebanese medical center. *J MedLiban.* 2006; **54(3):** 152 – 155.
5. Onat A, Sari I, Hergenc G, Yazici M, Uyarel H, Can G, et al. Predictors of abdominal obesity and high susceptibility of cardio- metabolic risk to its increments among Turkish women: a prospective population-based study. *Metabolism.* 2007; **56(3):** 348 – 356.
6. Curb JD, Sharp DS. Associations of adiposity with prevalent coronary heart disease among elderly men: the Honolulu Heart Program. *Int J Obes Relat Metab Disord.* 1997; **21(5):** 340 – 348.
7. Pourmoghaddas Z, Sadeghi M, Hekmatnia A, Sanei A, Tavakoli B, Roohafza H, et al. Different measurements of the obesity, adiponectin, and coronary heart disease: a single center study from Isfahan. *Journal of Research in Medical Sciences.* 2012; **17(4):** 218 – 222.
8. Chumlea WC, Guo SS, Kuczmarski RJ. Body composition estimates from NHANES III bioelectrical impedance data. *Int J Obes Relat Metab Disord*. 2002; **12:** 1596 – 1609.
9. Deurenberg-Yap M, Chew SK, Lin VF, Tan BY, van Staveren WA, Deurenberg P. Relationships between indices of obesity and its co-morbidities in multi-ethnic Singapore. *Int J Obes Relat Metab Disord*. 2001; **25(10):** 1554 – 1562.
10. Deurenberg-Yap M, Chew SK, Deurenberg P. Elevated body fat percentage and cardiovascular risks at low body mass index levels among Singaporean Chinese, Malays, and Indians. *Obes Rev*. 2002; **3(3):** 209 – 215.
11. Romero-Corral A, Somers VK, Sierra-Johnson J, Korenfeld Y, Boarin S, Korinek J, et al. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. *Eur Heart J*. 2010; **31(6):** 737 – 746.
12. Hara M, Saikawa T, Kurokawa M, Sakata T, Yoshimatsu H. Leg fat percentage correlates negatively with coronary atherosclerosis. *Circ J*. 2004; **68(12):** 1173 – 1178.
13. Thompson CJ, Ryu JE, Craven TE, Kahl FR, Crouse JR. Central adipose distribution is related to coronary atherosclerosis. *Arteriosclerosis and Thrombosis.* 1991; **11:** 327 – 333.

14. Banerji MA, Faridi N, Atluri R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, leptin, and insulin resistance in Asian Indian men. *J Clin Endocrinol Metab*. 1999; **84(1):** 137 – 144.
15. Siavash M, Sadeghi M, Salarifar F. Comparison of body mass index and waist/height ratio in predicting definite coronary artery disease. *Ann Nutr Metab*. 2008; **53:** 162 – 166.
16. Schneider HJ, Glaesmer H, Klotsche J, Bohler S, Lehnert H, Zeiher AM, et al. Accuracy of anthropometric indicators of obesity to predict cardiovascular risk. *J Clin Endocrinol Metab*. 2007; **92:** 589 – 594.
17. Rosito GA, Massaro JM, Hoffmann U, Ruberg FL, Mahabadi AA, Vasan RS, et al. Pericardial fat, visceral abdominal fat, cardiovascular disease risk factors, and vascular calcification in a community-based sample: the Framingham Heart Study. Circulation. 2008; **117(5):** 605 – 613.
18. Fox CS, Hwang SJ, Massaro JM, Lieb K, Vasan RS, O'Donnell CJ, et al. Relation of subcutaneous and visceral adipose tissue to coronary and abdominal aortic calcium (from the Framingham Heart Study). *Am J Cardiol*. 2009; **104(4):** 543 – 547.
19. Karthikeyan G, Teo KK, Islam S, McQueen MJ. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: an analysis from the INTERHEART Study. *J Am Coll Cardiol*. 2009; **53(3):** 244 – 253.
20. Ingelsson E, Schaefer EJ, Contois JH. Clinical utility of different lipid measures for prediction of coronary heart disease in men and women. *AMA*. 2007; **15(7):** 776 – 785.
21. Arsenaul B, Rana J, Stroes E. Beyond low-density lipoprotein cholesterol. *J Am Coll Cardiol*. 2010; **55:** 35 – 41.
22. Morrow DA, Gersh BA. Chronic Coronary Artery Disease. Braunwald's Heart Disease, 8th edition, Philadelphia: W.B. Saunders Company 2008: 1353 – 1354.
23. Janus ED, Watt NM, Lam KS. The prevalence of diabetes, association with cardiovascular risk factors, and implications of diagnostic criteria (ADA 1997 and WHO 1998) in a 1996 community-based population study in Hong Kong Chinese. Hong Kong Cardiovascular Risk Factor Steering Committee. American Diabetes Association. *Diabet Med*. 2000; **17(10):** 741 – 745.
24. Bush CA, van Fossen DB, Kolibash AJ . Cardiac catheterization and coronary angiography using 5 French preformed (Judkins) catheters from the percutaneous right brachial approach: a comparative analysis with the femoral approach. *Cathet Cardiovasc Diagn*. 1993; **29(4):** 267 – 272.
25. Deschenes D, Couture P, Dupont P, Tchernof A. Subdivision of the subcutaneous adipose tissue compartment and lipid-lipoprotein levels in women. *Obesity Res.* 2003; **11:** 469 – 476.
26. Lamon-Fava S, Wilson P, Schaefer E. Impact of body mass index on coronary heart disease risk factors in men and women. the Framingham offspring study. *Arterioscler Thromb and Vasc Biol*. 1996; **16:** 1509 – 1515.
27. Bogers RP, Bemelmans WJ, Hoogenveen RT, Boshuizen HC, Woodward M, Knekt P. Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a meta-analysis of 21 cohort studies including more than 300 000 persons. *Arch Intern Med*. 2007; **167(16):** 1720 – 1728.
28. Thomas GN, Ho SY, Lam KS, Janus ED, Hedley AJ, Lam TH. Impact of obesity and body fat distribution on cardiovascular risk factors in Hong Kong Chinese. *Obes Res*. 2004; **12(11):** 1805 – 1813.
29. Mahabadi AA, Massaro JM, Rosito GA, Levy D, Morabito JM, Wolf PA, et al. Association of pericardial fat, intrathoracic fat, and visceral abdominal fat with cardiovascular disease burden: the Framingham Hrart Study. *European Heart J*. 2009; **30(7):** 850 – 856.
30. Rosito GA, Massaro JM, Hoffmann U, Ruberg FL, Mahabadi AA, Vasan RS, et al. Pericardial fat, visceral abdominal fat, cardiovascular disease risk factors, and vascular calcification in a community-based sample: the Framingham Heart Study. *Circulation*. 2008; **117(5):** 605 – 613.
31. Fox CS, Massaro JM, Hoffmann U. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation*. 2007; **116(1):** 39 – 48.
32. Nicklas BJ, Penninx BW, Cesari M. Association of visceral adipose tissue with incident myocardial infarction in older men and women: the Health, Aging, and Body Composition Study. *Am J Epidemiol*. 2004; **160(8):** 741 – 749.
33. Kim DJ, Bergstrom J, Barrett-Connor E, Laughlin GA. Visceral adiposity and subclinical coronary artery disease in older adults: Rancho Bernardo Study. *Obesity (Silver Spring)*. 2008; **16(4):** 853 – 858.