Mesenteric Fat Thickness Is an Independent Determinant of Metabolic Syndrome and Identifies Subjects With Increased Carotid Intima-Media Thickness

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OBJECTIVE — Mesenteric fat, a reflection of visceral adiposity, may play an important role in the pathogenesis of metabolic syndrome and cardiovascular diseases (CVD). In this study, we examined the independent relationship between mesenteric fat thickness and metabolic syndrome and defined its optimal cutoff value to identify high-risk subjects for metabolic syndrome and CVD.

RESEARCH DESIGN AND METHODS — A total of 290 Chinese subjects had an ultrasound examination for measurements of thickness of mesenteric, preperitoneal, and subcutaneous fat as well as carotid intima-media thickness (IMT). Anthropometric measurements and metabolic risk profile were assessed by physical examination and blood taking.

RESULTS — Twenty (6.9%) subjects had metabolic syndrome according to the National Cholesterol Education Panel Adult Treatment Panel III criteria with Asian definitions for central obesity (waist circumference >80 cm in women and >90 cm in men). Mesenteric fat thickness had significant correlations ($P < 0.05$) with various metabolic variables. On multivariate regression, mesenteric fat thickness was an independent determinant of all components of metabolic syndrome after adjustment for age, sex, homeostasis model assessment of insulin resistance, and other fat deposits. The odds ratio of metabolic syndrome was increased by 1.35 (95% CI 1.10–1.66)-fold for every 1-mm increase in mesenteric fat thickness. On receiver-operating characteristic curve analysis, mesenteric fat thickness of ≥10 mm was the optimal cutoff value to identify metabolic syndrome, with sensitivity of 70% and specificity of 75%. Subjects with mesenteric fat thickness ≥10 mm had higher carotid IMT than those with thickness <10 mm (0.73 ± 0.19 vs. 0.64 ± 0.16 mm, $P = 0.001$).

CONCLUSIONS — Mesenteric fat thickness was an independent determinant of metabolic syndrome and identified subjects with increased carotid IMT.

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Metabolic syndrome is a constellation of multiple risk factors including hypertension, dysglycemia, dyslipidemia, and central obesity (1). Subjects with metabolic syndrome have a two- to threefold increased risk for cardiovascular diseases (CVDs) (2). There is ongoing debate regarding the relative roles of insulin resistance (1) and visceral adiposity in its pathogenesis (3). In this regard, there is a wealth of data showing the intimate relationships between visceral adiposity and adverse lipid profile (4), insulin sensitivity (5), elevated blood pressure (6), and impaired glucose tolerance (7).

Visceral adipose tissue, particularly mesenteric fat, is metabolically more active than subcutaneous or extraperitoneal fat (8). Carr et al. (3) recently reported independent relationships between intra-abdominal fat as measured by single-slice computed tomography and all components of metabolic syndrome. However, the relative contributions of different deposits of visceral fat remain uncertain. We hypothesized that these independent relationships were mainly attributable to mesenteric fat that directly drains into portal circulation. We have previously reported that sonographic measurement of mesenteric fat thickness was a good correlate with multiple risk factors (9) in a pilot study. In an expanded cohort, we found significant association between mesenteric fat thickness and carotid intima-media thickness (IMT) (10). In this study, we tested the hypothesis whether mesenteric fat thickness was an independent determinant of metabolic syndrome. We also evaluated its optimal cutoff value to identify subjects with metabolic syndrome and examined its association with carotid IMT.

RESEARCH DESIGN AND METHODS

— In a community-based health promotion program involving screening of >1,000 subjects of working age for metabolic diseases organized by our local hospital authority, 290 Chinese subjects (133 men and 157 women, mean BMI 23.7 kg/m^2, and mean age 41.8 years) with no known medical illnesses were invited to participate in the study. The same group of subjects had been studied for the association between abdominal fat thickness and IMT (10). The study was approved by the clinical research ethics committee of the Chinese University of Hong Kong, and all subjects gave informed written consent.

Ultrasound measurements of abdominal fat thickness and carotid IMT

An ultrasound machine ATL HDI 5000 (Bothell, CA) was used. Abdominal fat

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Abbreviations: CVD, cardiovascular disease; FPG, fasting plasma glucose; HOMA-IR, homeostasis model assessment of insulin resistance; IMT, intima-media thickness; ROC, receiver-operating characteristic curve.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

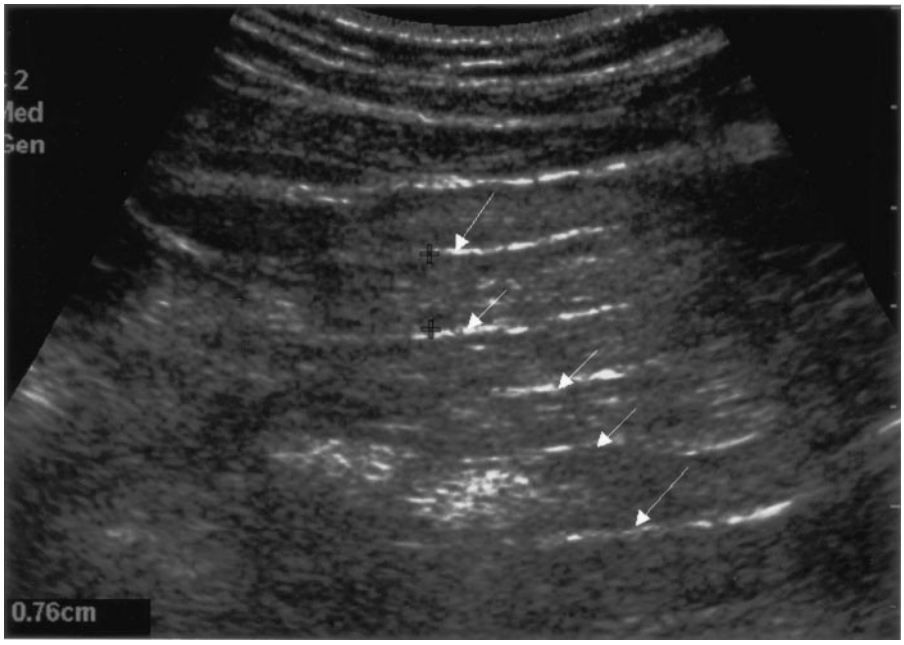

Figure 1—*Ultrasonogram of mesenteric leaves. Each mesenteric leaf is indicated by highly reflecting peritoneal surfaces (arrows). The maximum mesenteric thickness on the image was measured with the calipers (+).*

thickness was measured by a single operator, and the details of methodology were previously described (9). In brief, a complete survey of paraumbilical area was performed using a CL 7–4– or CL 5–2– MHz curvilinear transducer. The mesenteric leaves (Fig. 1) appeared as elongated structures with highly reflective peritoneal surfaces (11). The mean of the three thickest mesenteric leaves was used for the analysis. Both preperitoneal and subcutaneous fat thickness were measured with a L12–5–MHz linear transducer along the midline of the abdomen, between the xiphoid process and umbilicus. The maximum preperitoneal and subcutaneous fat thickness were each measured three times, and the mean value was taken. The interoperator and intraoperator reliability of various fat thickness measurements were within acceptable range (9), and the respective intraclass correlation coefficients for mesenteric fat thickness were 0.89 (95% CI 0.69–0.96; SE of measurement 0.11 cm) and 0.97 (0.93–0.99; SE of measurement 0.06 cm).

Carotid IMT was measured with a L12–5–MHz linear transducer using previously described methodology (10). All measurements of carotid IMT were made by a single operator. Three IMT measurements were made in the plaque-free section of both the right and left common carotid arteries, along the thickest point on the far wall and within ~1.5 cm proximal to the flow divider. The mean IMT was calculated by averaging six measurements from both sides. The intraclass correlation coefficients for inter- and intraoperator reliability for IMT measurement were 0.977 (95% CI 0.928–0.996) and 0.982 (0.909–0.994), respectively (10).

Clinical assessment

Body weight and height were measured in all subjects for calculation of BMI. Waist circumference was measured at the narrowest circumference between the xiphisternum and umbilicus. All subjects had blood taken after a 12-h fasting period for measurement of fasting plasma glucose (FPG), insulin, total cholesterol, HDL cholesterol, and triglycerides. Insulin resistance was estimated using the homeostasis model assessment of insulin resistance (HOMA-IR) equation, which simplified to the FPG and insulin product divided by 22.5. Before blood was taken, sitting blood pressure after 10 min of rest was measured.

Definition of metabolic syndrome

Metabolic syndrome was diagnosed according to the modified National Cholesterol Education Panel Adult Treatment Panel III definition for Asians (12). A subject was considered to have metabolic syndrome if three or more of the following criteria were met: *1*) waist circumference >90 cm in men and >80 cm in women, *2*) triglycerides ≥1.7 mmol/l, *3*) HDL cholesterol <1.0 mmol/l in men and <1.3 mmol/l in women, *4*) blood pressure ≥130/85 mmHg, and *5*) FPG ≥6.1 mmol/l.

Statistical analysis

All data are expressed as means ± SD or median (interquartile range). Student's *t* tests were used for between-group comparisons. Values of FPG, HOMA-IR, and triglycerides were logarithmically transformed due to skewed distributions. Pearson correlation was used to analyze associations between abdominal fat thickness and various metabolic variables. ANCOVA was used to compare the mean mesenteric fat thickness between men and women, with BMI as covariate. Multiple linear regression was used to identify the independent determinants for each component of the metabolic syndrome, using various abdominal fat thickness, HOMA-IR, sex, and age as independent variables. Multivariate logistic regression was used to examine whether abdominal fat thickness was an independent determinant of metabolic syndrome. Receiver-operating characteristic curve (ROC) analysis was performed to determine the optimal cutoff value of abdominal fat thickness to identify metabolic syndrome. This was followed by an analysis of the relationships between the cutoff value and carotid IMT. All analysis was performed using the Statistical Package for the Social Sciences (SPSS) for Windows, version 11.0.

RESULTS — Men had greater mesenteric and preperitoneal fat thickness, thinner subcutaneous fat thickness, and higher BMI than women (Table 1). Mesenteric fat thickness in this study ranged from 0.22 to 1.7 cm. The difference in mesenteric fat thickness between men and women remained significant ($P < 0.01$) after adjustment for BMI. Men had worse lipid and glucose profiles and higher blood pressure than women, but HOMA-IR and insulin concentration were similar between the two groups (Table 1).

Mesenteric fat thickness showed mild to moderate correlations with subcutaneous ($r = 0.35$) and preperitoneal ($r = 0.54$) fat thickness, respectively. Various abdominal fat thickness showed significant correlations with all metabolic variables (Table 2), except for a lack of association between subcutaneous fat

Table 1—*Clinical and biochemical characteristics of 290 Chinese study subjects*

Subjects' characteristics	Total	Men	Women	*P* values
n	290	134	157	—
Age (years)	41.8 ± 7.7	42.6 ± 8.3	41.1 ± 7.1	0.099
Mesenteric fat thickness (cm)	0.798 ± 0.32	0.911 ± 0.33	0.703 ± 0.28	<0.001
Preperitoneal fat thickness (cm)	1.32 ± 0.46	1.39 ± 0.47	1.26 ± 0.45	0.02
Subcutaneous fat thickness (cm)	2.30 ± 0.83	2.09 ± 0.72	2.48 ± 0.88	<0.001
Waist circumference (cm)	78.9 ± 8.9	83.5 ± 7.7	75.0 ± 8.0	<0.001
Waist-to-hip ratio	0.83 ± 0.06	0.87 ± 0.05	0.80 ± 0.05	<0.001
BMI (kg/m^2)	23.7 ± 3.30	24.6 ± 2.99	23.0 ± 3.38	<0.001
Systolic blood pressure (mmHg)	114.3 ± 16.0	120.3 ± 15.1	109.2 ± 15.0	<0.001
Diastolic blood pressure (mmHg)	74.8 ± 10.3	78.7 ± 9.49	71.5 ± 9.85	<0.001
Total cholesterol (mmol/l)	5.19 ± 0.94	5.34 ± 0.96	5.06 ± 0.90	0.011
LDL cholesterol (mmol/l)	3.08 ± 0.85	3.28 ± 0.87	2.91 ± 0.80	<0.001
HDL cholesterol (mmol/l)	1.58 ± 0.40	1.44 ± 0.35	1.69 ± 0.40	<0.001
Triglycerides (mmol/l)	1.02 (0.76–1.41)	1.14 (0.85–1.72)	0.88 (0.64–1.22)	<0.001
FPG (mmol/l)	4.9 (4.6–5.2)	5 (4.7–5.4)	4.8 (4.5–5.1)	0.041
Insulin concentration (μU/ml)	7.36 ± 4.5	7.86 ± 4.85	6.94 ± 4.16	0.082
HOMA (μU · ml^{-1} · mmol^{-1})	1.44 (0.92–2.13)	1.58 (1.07–2.39)	1.31 (0.87–1.94)	0.071

Data are means ± SD or median (interquartile range). Triglycerides, FPG, and insulin concentration were expressed as median (interquartile range) due to skewed distribution. *P* value: Students' *t* test between the men's and women's groups.

thickness and blood pressure and FPG. The mesenteric fat thickness showed the strongest correlations among the three abdominal fat deposits.

There were 20 (6.9%) subjects (11 men and 9 women) diagnosed to have metabolic syndrome according to the modified National Cholesterol Education Panel Adult Treatment Panel III for Asians (4.1% if original definition of central obesity of waist circumference >88 cm in women and >102 cm in men was used). Subjects with metabolic syndrome had greater median (interquartile range) mesenteric (10.9 [9.48–12.6] vs. 7.7 mm [5.25–9.93], $P < 0.001$), preperitoneal (14.9 [11.8–18.7] vs. 12.5 mm [9.74–15.7], $P = 0.032$), and subcutaneous (25.1 [17.8–31] vs. 22.6 mm [17.2–27.8], $P = 0.22$) fat thickness than those without.

On multiple regression (Table 3), mesenteric fat thickness was independently associated with all five components of the metabolic syndrome, while HOMA-IR was associated with waist circumference, triglycerides, and FPG. Preperitoneal and subcutaneous fat thickness had independent associations with diastolic blood pressure, triglycerides, and waist circumference.

On multivariate logistic regression, mesenteric fat thickness was an independent determinant of metabolic syndrome after adjustments for HOMA-IR, preperitoneal and subcutaneous fat thickness, sex, and age (Table 4). The odds ratio of metabolic syndrome was increased by 1.35 (95% CI 1.10–1.66)-fold for every 1-mm increase in mesenteric fat thickness. HOMA-IR was only marginally associated with metabolic syndrome ($P = 0.059$). Preperitoneal and subcutaneous fat thickness, sex, and age did not have independent associations with metabolic syndrome. To better assess the effect of sex on the association between mesenteric fat thickness and metabolic syndrome, an interaction term of (sex × mesenteric fat thickness) was used in the multivariate analysis. The interaction term did not reach statistical significance ($P > 0.05$).

The area under the ROC curve for mesenteric fat thickness to identify metabolic syndrome was 0.762 (95% CI 0.663–0.862, $P < 0.001$). A mesenteric fat thickness of 10 mm was the optimal cutoff value to define metabolic syndrome, with sensitivity of 70% and specificity of 75%. Subjects with mesenteric fat thickness ≥10 mm had higher carotid IMT than those with thickness <10 mm (0.73 ± 0.19 vs. 0.64 ± 0.16 mm, $P = 0.001$).

CONCLUSIONS — Subsequent to our pilot study, which aimed to explore the potential role of sonographic measurement of mesenteric fat thickness in obesity research (9), we have reported the association of mesenteric fat thickness with carotid IMT in an expanded cohort (10). In this study, we examined the interrelationships between mesenteric fat thickness, metabolic syndrome, and ca-

Table 2—*Correlation coefficients of abdominal fat thickness with various metabolic variables in 290 apparently healthy Chinese subjects*

	Mesenteric fat thickness	Preperitoneal fat thickness	Subcutaneous fat thickness
Total cholesterol	0.27*	0.15*	0.13†
HDL cholesterol	−0.38*	−0.26*	−0.12†
LDL cholesterol	0.32*	0.19*	0.13†
Triglycerides	0.45*	0.29*	0.19*
FPG	0.25*	0.13†	0.07
HOMA-IR	0.39*	0.32*	0.21*
Insulin concentration	0.34*	0.32*	0.20*
Systolic blood pressure	0.31*	0.17*	0.07
Diastolic blood pressure	0.38*	0.32*	0.13†

*$P < 0.01$; †$P < 0.05$.

Table 3—*Multiple linear regression analyses of individual components of the metabolic syndrome defined by the NCEP ATP III criteria with Asian definition for obesity (as continuous dependent variables) using mesenteric, preperitoneal, and subcutaneous fat thickness; insulin resistance; sex; and age as independent variables in 290 apparently healthy subjects*

	Mesenteric fat thickness	Preperitoneal fat thickness	Subcutaneous fat thickness	HOMA-IR	Sex	Age
Systolic blood pressure						
$R^2 = 0.22$, C = 91.7	0.84 (0.019)	−0.03 (0.90)	0.14 (0.28)	−0.21 (0.80)	−9.59 (<0.001)	0.45 (<0.001)
Diastolic blood pressure						
$R^2 = 0.25$, C = 59.1	0.58 (0.01)	0.33 (0.028)	0.049 (0.54)	−0.063 (0.90)	−5.7 (<0.001)	0.21 (0.004)
Triglycerides						
$R^2 = 0.32$, C = −0.33	0.022 (<0.001)	0.00016 (0.96)	0.0033 (0.043)	0.030 (0.003)	−0.077 (0.002)	0.002 (0.14)
HDL cholesterol						
$R^2 = 0.22$, C = 1.50	0.031 (<0.001)	0.0021 (0.72)	0.0044 (0.18)	−0.017 (0.41)	0.21 (<0.001)	0.0088 (0.002)
Fasting glucose						
$R^2 = 0.22$, C = 0.59	0.0029 (0.04)	0.0006 (0.53)	0.00015 (0.77)	0.020 (<0.001)	0.0082 (0.30)	0.0017 (<0.001)
Waist circumference						
$R^2 = 0.73$, C = 61.5	1.17 (<0.001)	0.29 (<0.001)	0.29 (<0.001)	0.72 (0.009)	−6.70 (<0.001)	−0.0018 (0.96)

Data are β (*P* value). β, regression coefficient; C, constant; R^2, total variances explained by the variable(s).

rotid IMT. To date, only a few studies (13,14) had examined the association of abdominal fat thickness measured by ultrasound scan with cardiovascular risk. Besides, these studies, only measured subcutaneous and preperitoneal fat thickness and their relative ratio or intraabdominal fat thickness as indicated by the distance between the rectus muscle and aorta.

In this analysis, we have confirmed mesenteric fat thickness as a major independent determinant of metabolic syndrome. In addition, on ROC analysis, a mesenteric fat thickness of 10 mm was found to be a discriminative value for metabolic syndrome, which was also associated with increased carotid IMT. Based on a pilot study of 37 Chinese subjects, we found good correlation between mesenteric fat thickness by ultrasound scan and total visceral adiposity ($r = 0.8$) as measured by MRI (K.H.L, Y.L.C., J.C.N.C., W.B.C., unpublished data). In agreement with other studies that used computed tomography scan to document visceral adiposity (3,4,6), we found that mesenteric fat thickness was the strongest correlate for all cardiovascular risk factors compared with preperitoneal and subcutaneous fat thickness.

Given the importance of metabolic syndrome as a predictor for future development of CVD (2), we observed that 4.1% of our subjects had this syndrome. This percentage increased to 6.9% if Asian definition for obesity was used, thus emphasizing the need to take ethnicity into consideration when defining obesity (12,15). This incidence was <17.9% reported in a community-based Asian study with similarly modified criteria (12). This difference is likely due to the younger age distribution and lack of known medical illnesses in the present cohort.

On multiple linear regression, mesenteric fat thickness was significantly associated with all components of the metabolic syndrome, while HOMA-IR or preperitoneal and subcutaneous fat thickness were only associated with two components in addition to waist circumference. These associations were further supported on multivariate logistic regression analysis, where mesenteric fat thickness was a determinant of metabolic syndrome independent of age, sex, HOMA-IR, and other abdominal fat thickness. Both insulin resistance and visceral adiposity are proposed to play critical roles in metabolic syndrome (16), although the causal relationships among

Table 4—*Multivariate logistic regression analysis to identify independent determinant(s) for metabolic syndrome (as categorical variable) using mesenteric, preperitoneal, and subcutaneous fat thickness; insulin resistance; sex; and age as independent variables in 290 Chinese apparently healthy subjects*

	β ± SE	*P* value	Odds ratio (95% CI)
Mesenteric fat thickness	0.30 ± 0.11	0.004	1.35 (1.10–1.66)
Preperitoneal fat thickness	−0.058 ± 0.07	0.41	0.94 (0.82–1.08)
Subcutaneous fat thickness	0.01 ± 0.034	0.77	1.01 (0.95–1.08)
HOMA-IR	0.33 ± 0.17	0.059	1.05 (1.00–1.10)
Sex	0.088 ± 0.61	0.885	1.09 (0.28–3.02)
Age	0.01 ± 0.034	0.775	1.01 (0.94–1.08)

β, regression coefficient.

these three variables remains to be elucidated. In this cross-sectional study, mesenteric fat thickness, a reflection of visceral adiposity, was the main explanatory variable for metabolic syndrome after adjustment of insulin resistance, suggesting that visceral fat may be the main culprit. While these findings will need independent replication, the causal relationship between insulin resistance and visceral fat will need to be examined in both prospective and interventional studies.

Nonetheless, it is now well recognized that visceral adipose tissue, notably mesenteric fat, drained by the portal circulation is metabolically more active than nonportal adipose tissues such as subcutaneous and preperitoneal fat deposits. The increased free fatty acids from these adipocytes can lead to reduced fat oxidation and ectopic fat deposition in liver and muscle, resulting in reduced glucose uptake (17). Furthermore, visceral adipocytes can secrete a large number of cytokines and vasoactive peptides, including interleukin-6, tumor necrosis factor α, angiotensin II, plasminogen activator inhibitor-1, etc., all of which can increase cardiovascular risks (18). In support of these hypotheses, mesenteric fat thickness was an independent determinant for all components of metabolic syndrome, and for every 1-mm increase in mesenteric fat thickness, the risk of metabolic syndrome was increased by 1.3-fold.

Several prospective studies have now confirmed that metabolic syndrome was associated with premature cardiovascular mortality and new onset of diabetes (19,20). Hence, to further explore the usefulness of mesenteric fat thickness as a diagnostic tool, we used ROC analysis to define the optimal cutoff point of this measurement. In support of its validity, the cutoff value of 10 mm had 70% sensitivity and 75% specificity, which was also associated with increased carotid IMT.

Compared with computed tomography or magnetic resonance imaging, sonographic method has the advantages of being relatively cheap, noninvasive, and reproducible. Our data also suggested that it is a more discriminative indicator for increased cardiovascular risk compared with other adipose tissues. Further prospective and interventional studies are required to validate the clinical use of these novel measurements.

In conclusion, mesenteric fat thickness was an independent determinant of metabolic syndrome in apparently healthy Chinese subjects, with an odds ratio of 1.35 for every 1-mm increase, at least within the observed range of mesenteric fat thickness. The discriminating cutoff point of 10 mm indicates the presence of metabolic syndrome and identifies subjects with increased IMT. Measurement of mesenteric fat thickness may potentially be developed into an alternative tool to identify subjects at risk for CVD.

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