

Aortomesenteric Fat Thickness With Ultrasound Predicts Metabolic Diseases in Obese Patients

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Abstract: *Background:* The relation between visceral fat accumulation and development of cardiovascular and metabolic disorders has been demonstrated. The aim of this study was to determine the relationship between a new ultrasound visceral fat thickness (VFT) measurement and clinical and anthropometric data in a consecutive series of obese patients. *Methods:* Fifty-five consecutive male obese patients underwent ultrasound evaluation and metabolic and anthropometric parameters determination at baseline and after 3 weeks of a very low-calorie diet (VLCD) therapy. The new ultrasound measurement, the thickness of the fat between the aorta and the superior mesenteric artery (AMFT), was determined along with the maximum thickness of preperitoneal fat and the global VFT. *Results:* AMFT showed a better correlation than VFT and preperitoneal fat with all anthropometric and metabolic parameters, both at baseline and after VLCD regimen. At baseline, patients in the middle and high AMFT and VFT tertiles had a significantly higher prevalence of metabolic diseases with respect to AMFT and VFT low tertile patients, whereas after VLCD period, AMFT only showed significant difference within tertiles. The odds ratios for the various metabolic diseases were higher in the middle and high AMFT tertiles than those in the middle and high VFT tertiles, remaining significant after adjustment for age, body mass index and VLCD regimen only in the middle and high AMFT tertiles. *Conclusions:* The ultrasonographic AMFT evaluation is strongly correlated to the presence of metabolic syndrome and could be a valuable tool to predict metabolic diseases and associated cardiovascular risks in men.

Key Indexing Terms: Metabolic syndrome; Cardiovascular risk; Visceral fat. [Am J Med Sci 2014;347(1):8-13.]

Obesity is a serious medical condition that has an adverse effect on health, leading to increased health problems and reduced life expectancy. Moreover, in obese patients, the presence of problems related to metabolic diseases, such as glucose intolerance, hypertension, dyslipidemia and hyperinsulinemia, may increase the risk of occurrence of cardiovascular diseases (CVDs).^{1,2} In these patients, the distribution of fat may be compartmentalized, and it has been demonstrated that different fat compartments are associated with differential metabolic risk factors.^{3,4} Reliable methods for measurement of body fat and fat distribution are therefore of paramount importance. In particular, the visceral adipose tissue (VAT) compartment may be a unique pathogenic fat depot, and recent studies have demonstrated the relation between visceral fat accumulation and development of both CVD and metabolic disorders.⁵⁻⁷ VAT

has been termed an endocrine organ because it secretes adipocytokines and other vasoactive substances that can influence the risk of developing metabolic traits.⁸⁻¹⁰ Simple and noninvasive methods to assess fat visceral accumulation are anthropometric index, such as body mass index (BMI), waist circumference (WC), and waist-to-hip circumference. Although anthropometric measurements have been extensively studied, their reliability is still debated.¹¹⁻¹³ Methods for direct assessment of abdominal fat include magnetic resonance imaging (MRI) and computed tomography (CT),^{14,15} usually considered as gold standard.¹⁶ However, these methods are expensive and, in the case of CT, the subjects are exposed to ionizing radiation. Direct assessment of visceral fat, moreover, can be obtained by ultrasound imaging that has been proposed as a suitable technique to accurately estimate intra-abdominal fat.^{17,18} So far, several studies have found a good correlation between thickness of intra-abdominal fat measured by ultrasound and the amount of fat measured by CT, but the use of these ultrasonographic measures has been criticized because of their presumed low reproducibility.¹⁹⁻²¹

To overcome the limitations of CT and/or MRI measurements, we have developed an ultrasound protocol for the assessment of VAT by measuring the thickness of fat between the abdominal aorta and the superior mesenteric artery (SMA), and we have called this measurement the aortomesenteric fat thickness (AMFT). To precisely validate the relationship between AMFT and anthropometric and metabolic parameters, we conducted a prospective, randomized, blinded study in a consecutive series of obese patients at baseline and after dietetic treatment.

MATERIALS AND METHODS

Fifty-five consecutive male patients, referred to Clinical Nutrition Unit because of obesity, were enrolled in the study. Our study includes only men because of the significant difference in body fat distribution between the sexes.²² Exclusion criteria were the presence of chronic renal insufficiency, defined as a serum creatinine >1.5 mg/dL; a previously diagnosed type 1 diabetes; the presence of cardiac insufficiency, defined as a left ventricular ejection fraction ≤40% and the presence of chronic active hepatitis, defined as a stable serum increase of transaminases (2-fold as compared with the normal values). Inclusion criteria were the presence of class I to III obesity, defined as a BMI ≥30.0 kg/m², according to World Health Organization classification²³ and of metabolic syndrome. According to International Diabetes Federation definition,²⁴ metabolic syndrome was defined as the presence of central obesity (defined as WC ≥94 cm for European men) along with ≥2 of the following conditions: raised triglyceride levels: ≥150 mg/dL, or specific treatment for this lipid abnormality; reduced high-density lipoprotein (HDL)-cholesterol: <40 mg/dL, or specific treatment for this lipid abnormality; raised blood pressure (BP): systolic BP ≥130 or diastolic BP ≥85 mm Hg, or treatment of previously diagnosed hypertension; raised plasma

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fasting glucose ≥ 110 mg/dL, or previously diagnosed type 2 diabetes. Written informed consent after a detailed description of the procedure was obtained from all patients, and the study was approved by the Ethics Committee.

All patients underwent the same study protocol: ultrasound evaluation, metabolic and anthropometric parameters were determined at baseline and after 3 weeks of a very low-calorie diet (VLCD), defined as a dietetic regimen of 600 to 800 kcal/diet, according to calculated ideal body weight of patients.^{25,26}

Ultrasound Echotomography Technique

All patients underwent echographic evaluation at baseline and after 2 weeks at completion of dietetic regimen. All ultrasound examinations were performed by the same investigator, using an ultrasonographic system (Hitachi EUB-8500; Hitachi Medical Systems America, Inc, Twinsburg, OH), with the use of a 3.5-MHz convex or a 7.5-MHz linear probe, as was appropriate. The patient setting was the same for all measurements. All patients underwent a fasting period of at least 8 hours before the ultrasonographic evaluation. The measurements were done without distortion (by compression) of the abdominal cavity, with subjects in the supine position, after a forced expiration and 1 cm above the umbilicus. The most widely used ultrasound measurements were determined¹⁸⁻²¹: the maximum thickness of preperitoneal fat (PFT), defined as the thickness of the fat tissue between the liver surface and the linea alba, and the visceral fat thickness (VFT), defined as the distance between the anterior wall of the aorta and the internal face of the rectoabdominal muscle, perpendicular to the aorta. A 7.5-MHz linear probe was used to determine the PFT, whereas a 3.5-MHz convex probe was used to determine the VFT.

The thickness of the fat between the aorta and the SMA, namely AMFT, was also measured using a 3.5-MHz convex probe. To precisely assess the origin of the SMA from the abdominal aorta and its course, we performed longitudinal and transverse scanning with color-flow imaging support. We measured in longitudinal scanning the distance between the anterior wall of the abdominal aorta and the posterior wall of SMA at its maximum convexity, and we also measured in transverse scanning, the distance between the abdominal aorta and the SMA when it appears to be perfectly rounded with equal anteroposterior and laterolateral diameters. These distances, obtained by frozen images immediately after a forced expiration to remove bowel interference, were expressed in millimeters (Figures 1 and 2). All these measurements, performed by the same operator who was blinded about the study,

were performed 3 times, and the mean value was taken for analyses.

Biochemical and Anthropometric Analyses

All anthropometric data were obtained by the same investigator at baseline visit and at the day after the completion of dietetic regimen. BMI was calculated as weight (kilograms) divided by height (meters squared) and the WC was measured halfway between the lower rib and the iliac crest.

After sitting for at least 10 minutes, blood pressure was measured using a standard mercury sphygmomanometer and the Korotkoff sound V was taken as the diastolic blood pressure. Blood samples for determination of plasma values of metabolic parameters were obtained from each patient by venous puncture into EDTA blood collection tube and were collected at baseline and on the day after the completion of dietetic regimen. All subjects had at least 12 hours of fasting before blood was taken for serum measurements. Samples were centrifuged (3000g for 20 minutes), the supernatant was carefully removed and stored at -80°C and then thawed at room temperature just before the assays. Plasma concentrations of metabolic parameters were measured by using the appropriate test, according to the manufacturer's guidelines, and the operators were blinded about the ultrasound results.

Statistical Analyses

All data are presented as mean \pm standard deviation. A 2-tailed *P* value of <0.05 was considered significant. Pearson correlation coefficients, which were adjusted for age and BMI, were calculated for the associations of the different measures of intra-abdominal fat with the metabolic risk factors. For continuous variables, differences between groups were assessed using univariate ANOVA, and for categorical variables, differences between groups were assessed using post hoc analysis performed by Bonferroni's test or a χ^2 test with the Fisher's exact test and odds ratio (OR) with 95% confidence intervals.

Logistic regression, which was adjusted for age, VLCD therapy and BMI, was used to analyze the associations between the tertiles of the AMFT and VFT and the presence of metabolic diseases. A low tertile of AMFT and VFT was used as the reference category (OR = 1.00). Results are presented as ORs (and 95% confidence intervals), which are regarded as approximations of relative risks. The use of log-transformed variables did not significantly change the associations. All analyses were performed with SPSS 12.01 for Windows (SPSS Inc, Chicago, IL).

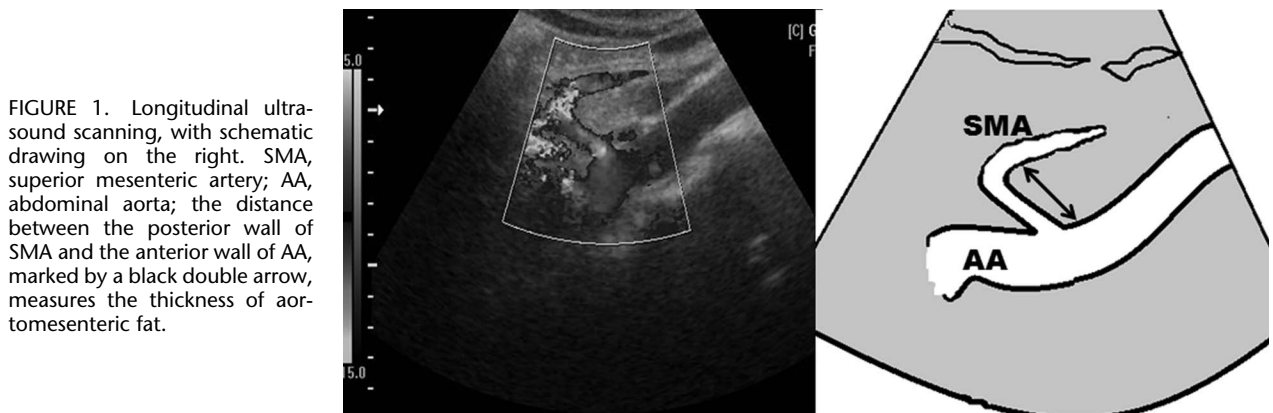


FIGURE 1. Longitudinal ultrasound scanning, with schematic drawing on the right. SMA, superior mesenteric artery; AA, abdominal aorta; the distance between the posterior wall of SMA and the anterior wall of AA, marked by a black double arrow, measures the thickness of aortomesenteric fat.

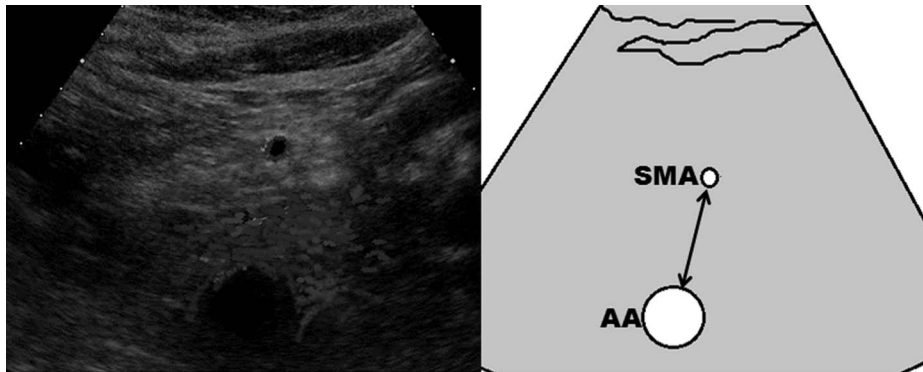


FIGURE 2. Transverse ultrasound scanning, with schematic drawing on the right. SMA, superior mesenteric artery; AA, abdominal aorta; the distance between the posterior wall of SMA and the anterior wall of AA, marked by a black double arrow, measures the thickness of aortomesenteric fat.

RESULTS

Three patients were lost because of nonadherence to VLCD therapy. The remaining 52 patients fulfilled the study protocol. The main anthropometric, imaging and clinical characteristics of the patients are summarized in Table 1: mean values of ultrasound, anthropometric and biochemical parameters were significantly higher at baseline than those after the 3 weeks of VLCD regimen. Interestingly, considering 5 of the most significant parameters (anthropometric: BMI; ultrasound: VFT and AMFT and clinical: total cholesterol and triglycerides), AMFT (22.2% of reduction with respect of baseline values) and triglycerides (28.7% of reduction) were consistently the most significant values.

In Pearson correlation analysis, at baseline, AMFT was significantly related to all anthropometric and metabolic parameters showing excellent correlation with BMI, plasma fasting glucose and insulin levels ($P < 0.001$) (Table 2). AMFT was also significantly related to diastolic blood pressure value and low-density lipoprotein (LDL)-cholesterol and triglycerides plasma levels ($P < 0.01$). VFT also showed significant correlation with all anthropometric and metabolic parameters considered, whereas PFT failed to show correlation with systolic blood pressure and HDL-cholesterol levels. However, the correlation coefficients between AMFT and the considered parameters were higher than those between VFT and PFT and the considered parameters. After the 3 weeks of VLCD regimen, AMFT was still significantly related to BMI and plasma fasting insulin levels ($P < 0.001$), to LDL-cholesterol and triglycerides plasma levels ($P < 0.01$) and to total cholesterol and fasting glucose levels ($P < 0.05$). VFT showed significant correlation with BMI ($P < 0.01$) and fasting insulin and LDL-cholesterol levels ($P < 0.05$), whereas PFT was related only to BMI and fasting insulin values ($P < 0.05$). After the dietetic regimen, the correlation coefficients between AMFT and the considered parameters showed a better correlation than those between VFT and PFT and the considered parameters.

Subjects were classified by the tertiles on the basis of their AMFT and VFT. The mean VFT values were <44.1 and <36 mm for the low tertile, 44.1 to 58.0 and 36.0 to 47.0 mm for the middle tertile and >58.0 and >47.0 mm for the high tertile, at baseline and after VLCD period, respectively. The mean AMFT values are <14.5 and <10.0 mm for the low tertile, 15.0 to 25.0 and 10.5 to 20.0 mm for the middle tertile, and >25.0 and >20.0 mm for the high tertile, at baseline and after VLCD period, respectively. The prevalence of metabolic syndrome was compared according to AMFT and VFT tertiles. At baseline, patients in the middle and high AMFT and VFT tertiles had a significantly higher prevalence of hypertriglycer-

idemia, low HDL-cholesterolemia, diastolic hypertension and high fasting glucose in AMFT and VFT low tertile patients, whereas after VLCD period, AMFT only showed significant difference within tertiles, Table 3.

The logistic regression results showing the ORs for the various metabolic diseases in the AMFT and VFT tertiles are listed in Table 4. The ORs for hypertension, hypertriglyceridemia, low HDL-cholesterolemia and hyperglycemia were higher in the middle and high AMFT tertiles than those in the middle and high VFT tertiles. These observations remained significant after adjustment for age, BMI and VLCD regimen in the middle and high AMFT tertiles, with the only exception of hypertension in the middle tertile, whereas after adjustment, hypertriglyceridemia only remained significant in middle and high VFT tertiles.

DISCUSSION

Our study has shown that AMFT measured by ultrasonography has an excellent relation to the prevalence of metabolic diseases and maintains this relation also after a VLCD

TABLE 1. Anthropometric, ultrasound imaging and cardiovascular data

Parameter	Baseline	After VLCD	P
Age (yr)	48.6 ± 9.8	NA	NA
WC (cm)	106.3 ± 8.2	101.1 ± 7.6	0.001
BMI (kg/m ²)	35.9 ± 5.2	33.1 ± 4.9	0.001
VFT (mm)	52.3 ± 9.7	47.6 ± 8.9	0.01
PFT (mm)	15.3 ± 1.7	14.4 ± 1.9	0.01
AMFT (mm)	18.1 ± 6.3	14.2 ± 5.4	0.0001
Syst BP (mm Hg)	144.7 ± 14.7	135.6 ± 13.9	0.001
Diast BP (mm Hg)	88.0 ± 8.3	81.6 ± 8.9	0.002
Glucose (mg/dL)	123.7 ± 18.2	112.5 ± 13.9	0.0006
Insulin (IU/mL)	15.7 ± 9.5	11.6 ± 8.9	0.02
Total cholesterol (mg/dL)	248.8 ± 57.1	212.5 ± 48.8	0.0007
LDL-C (mg/dL)	162.2 ± 35.4	145.6 ± 30.9	0.01
HDL-C (mg/dL)	44.3 ± 12.4	49.2 ± 10.9	0.03
Triglycerides (mg/dL)	250.6 ± 62.4	179.3 ± 46.8	<0.0001
Uric acid (mg/dL)	6.4 ± 2.1	6.8 ± 1.9	0.4

Data are presented as mean ± standard deviation.

Syst BP, systolic blood pressure; diast BP, diastolic blood pressure; glucose, fasting glucose; insulin, fasting insulin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; NA, not applicable.

TABLE 2. Pearson correlation coefficients between various intra-abdominal fat thickness measures by ultrasound and anthropometric and metabolic data

Parameter	Baseline			After VLCD		
	AMFT, r	VFT, r	PFT, r	AMFT, r	VFT, r	PFT, r
BMI	0.83 ^a	0.61 ^b	0.36 ^c	0.78 ^a	0.61 ^b	0.45 ^c
Glucose	0.77 ^a	0.59 ^b	0.31 ^c	0.39 ^c	0.30 ^d	0.16 ^d
Chol	0.45 ^c	0.33 ^c	0.33 ^c	0.33 ^c	0.30 ^d	0.18 ^d
LDL-C	0.51 ^b	0.43 ^c	0.28 ^c	0.42 ^b	0.36 ^c	0.23 ^d
HDL-C	-0.39 ^c	-0.2 ^c	0.08 ^d	-0.30 ^d	0.17 ^d	0.14 ^d
TG	0.46 ^b	0.41 ^c	0.30 ^d	0.48 ^c	0.25 ^d	0.11 ^d
Insulin	0.63 ^a	0.54 ^b	0.42 ^b	0.58 ^a	0.40 ^c	0.31 ^c
Syst BP	0.37 ^c	0.39 ^c	0.36 ^d	0.15 ^d	0.09 ^d	0.08 ^d
Diast BP	0.40 ^b	0.4 ^c	0.39 ^c	0.11 ^d	0.07 ^d	0.10 ^d

^a P < 0.001.

^b P < 0.01.

^c P < 0.05.

^d Not significant.

Glucose, fasting glucose; Chol, total serum cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; insulin, fasting insulin; syst BP, systolic blood pressure; diast BP, diastolic blood pressure.

regimen. VAT accumulation has been found to be a major correlate of a cluster of diabetogenic, atherogenic, prothrombotic and proinflammatory metabolic abnormalities referred to as the metabolic syndrome.⁶ Evidence suggests that this dysmetabolic profile is predictive of a substantially increased risk of CVD even in the absence of classical risk factors. The mechanism of increased metabolic risk is hypothesized to be related to the metabolically active adipose tissue found in the visceral region. Many studies have demonstrated that the visceral fat compartment is metabolically active, secreting many vasoactive substances (inflammatory markers, adipocytokines, markers of hemostasis and fibrinolysis and growth factors, including vascular endothelial growth factor) that may contribute to its role in cardiometabolic risk-factor manifestation.²⁷ This also applies to the epicardial fat that is a metabolically active organ, which generates various bioactive molecules, as free fatty acids and

a number of bioactive molecules such as adiponectin, resistin and inflammatory cytokines that could affect both the coronary artery response and the cardiac function.²⁸ This metabolic activity was also demonstrated for the periaortic, pericardial²⁹ and perirenal fat depot.³⁰ Variation in the association of levels of circulating vascular growth factors (and their soluble receptors) with distinct body fat compartments may explain differences in the systemic pathogenicity of regional fat depots.

Several methods of assessing the amount of visceral fat accumulation have been investigated. A simple and noninvasive method of assessing regional adiposity is the use of an anthropometric index, such as BMI, WC, abdominal sagittal diameter³¹ or neck circumference.³² However, these indexes do not directly quantify the real amount of fat and do not discriminate between visceral and subcutaneous fat. Moreover, substantial variations in the visceral fat content may be observed among persons with a similar anthropometric index.

Accordingly, alternative and reliable methods are needed to overcome limitations of anthropometric indexes. Imaging techniques, like CT and MRI, allow a precise and reliable measurement of visceral fat. However, these imaging techniques are expensive, not generally available and, in the case of CT, expose subjects to ionizing radiations. Therefore, ultrasonographic measurements have been developed as an emerging method to carefully estimate visceral adiposity, with the same reliability and reproducibility of MRI and CT.³³⁻³⁵ A noninvasive technique to assess the amount of intra-abdominal fat to quantify metabolic syndrome risk may be useful in specifically targeting preventive actions. Especially in a hospital setting, with suitable equipment and trained technicians, ultrasound may be such a noninvasive technique. The most frequent ultrasonographic measures reported in previous studies were PFT and VFT, the latter better correlated with the risk of CVDs and with the presence of various metabolic diseases. Furthermore, a recent study showed that the measurement of perirenal fat thickness could be applied as an easy and reliable imaging indicator of visceral obesity and cardiovascular risk factors in the metabolic syndrome.³⁰ But, despite encouraging results, ultrasonographic measurements can be operator dependent and lack standard measuring sites, thereby resulting in a high degree of irreproducibility. To overcome these shortcomings, our approach considers 2 precise and operator-independent anatomical landmarks: the anterior wall of the abdominal aorta and

TABLE 3. Global, and within the AMFT and VFT tertiles, prevalence of metabolic syndrome, at baseline and after VLCD regimen

Prevalence	Baseline				After VLCD				
	BP, n (%)	TG, n (%)	HDL-C, n (%)	Gluc, n (%)	Prevalence	BP, n (%)	TG, n (%)	HDL-C, n (%)	Gluc, n (%)
Global, n = 52	36 (69.2)	42 (80.7)	34 (65.4)	32 (61.5)	Global, n = 52	16 (30.8)	12 (23.1)	15 (28.8)	15 (28.8)
AMFT tertiles					AMFT tertiles				
Low, n = 8	1 (12.5)	2 (25.0)	2 (25.0)	1 (12.5)	Low, n = 35	6 (17.1)	7 (20.0)	5 (14.3)	6 (17.1)
Middle, n = 17	13 (76.4) ^a	15 (88.2) ^a	14 (82.3) ^a	13 (76.4) ^a	Middle, n = 13	7 (53.8) ^b	8 (61.5) ^a	6 (46.2) ^b	6 (46.2) ^b
High, n = 27	23 (85.2) ^a	25 (92.6) ^a	24 (88.1) ^a	22 (81.5) ^a	High, n = 4	3 (75.0) ^b	4 (100.0) ^a	3 (75.0) ^b	3 (75.0) ^b
VFT tertiles					VFT tertiles				
Low, n = 9	3 (33.3)	3 (33.3)	2 (22.2)	3 (33.3)	Low, n = 20	4 (20.0)	5 (25.0)	6 (30.0)	5 (25.0)
Middle, n = 15	11 (73.3) ^c	12 (80.0) ^b	10 (66.6) ^b	11 (73.3) ^c	Middle, n = 19	4 (21.1) ^c	4 (21.1) ^c	5 (26.3) ^c	6 (31.6) ^c
High, n = 28	21 (75.0) ^b	25 (89.3) ^a	21 (75.0) ^a	23 (60.7) ^b	High, n = 13	4 (30.8) ^c	3 (23.1) ^c	4 (30.8) ^c	4 (30.8) ^c

Significantly different from the low tertile (Fisher's exact test): ^aP < 0.01; ^bP < 0.05; ^cnot significant.

BP, systolic blood pressure ≥130 or diastolic blood pressure ≥85 mm Hg; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; Gluc, fasting glucose.

TABLE 4. Odds ratios and 95% CIs of metabolic diseases by tertile of AMFT and VFT

Ultrasound technique	Low tertile	Unadjusted			Adjusted ^a		
		Middle tertile	High tertile	P for trend	Middle tertile	High tertile	P for trend
AMFT							
Hyperglycemia	1.00	2.83 (1.20–12.23) ^b	3.93 (1.90–8.14) ^c	0.01	4.48 (1.29–5.51) ^b	2.04 (1.06–32.94) ^b	0.016
Hypertension	1.00	3.85 (1.89–7.21) ^c	6.19 (3.01–12.74) ^c	<0.001	1.36 (0.90–2.05) ^d	2.87 (1.41–5.86) ^b	0.003
Hypertriglyceridemia	1.00	6.00 (1.72–20.89) ^c	8.89 (2.27–34.79) ^c	0.014	1.91 (1.24–2.95) ^b	3.38 (1.61–7.10) ^b	0.018
Low-HDL cholesterolemia	1.00	4.73 (2.24–8.78) ^c	11.14 (4.92–25.23) ^c	<0.001	3.27 (1.28–12.53) ^b	1.95 (1.16–3.27) ^b	0.003
VFT							
Hyperglycemia	1.00	1.45 (0.41–3.21) ^d	0.84 (0.31–2.26) ^d	0.30	0.95 (0.30–3.03) ^d	0.67 (0.36–41.28) ^d	0.35
Hypertension	1.00	3.69 (1.37–10.79) ^b	4.22 (1.52–11.73) ^b	0.003	3.30 (0.33–33.48) ^d	1.71 (0.95–3.09) ^d	<0.05
Hypertriglyceridemia	1.00	2.22 (1.16–4.26) ^b	2.28 (1.18–4.40) ^b	0.014	5.32 (1.48–19.09) ^b	2.81 (1.33–5.97) ^b	<0.05
Low-HDL cholesterolemia	1.00	4.43 (1.35–16.54) ^b	4.83 (1.73–15.92) ^c	<0.001	1.25 (0.91–5.04) ^d	0.74 (0.32–17.83) ^d	0.06

^a Adjusted for body mass index, age and very low-calorie diet therapy.

^b $P < 0.05$.

^c $P < 0.01$.

^d Not significant.

the posterior wall of the SMA. SMA normally arises from the anterior wall of abdominal aorta and travels in an anteroinferior direction to the left forming with the aorta an angle, the aortomesenteric angle, of 35 to 55 degrees. Retroperitoneal fat fills this angle and determines a precise and a well-defined echogenic zone. The narrowing of the aortomesenteric angle less than 25 degrees is generally associated to the aortomesenteric syndrome.³⁶ With our approach, we perform 2 perpendicular scans, longitudinal and transverse, and we consider the maximum convexity and the perfect circularity of SMA as ideal landmarks for AMFT measurements. The measurement of fat between these 2 fix structures is independent from the operator and may be standardized, thus making it easily reproducible.

Visceral fat usually shows greater responses than subcutaneous fat to interventional therapy, such as changes in caloric intake or physical exercise.³⁷ Despite the correlation between ultrasonographic determination of visceral fat and cardiovascular and metabolic diseases, as has been thoroughly studied, the persistence of this correlation after a strict dietetic therapy, which significantly improves metabolic and anthropometric data, has not yet been demonstrated. We enrolled a consecutive series of obese patients with metabolic syndrome and put them on a VLCD regimen. In these patients, the AMFT measurement has shown, both at baseline and after 3 weeks of VLCD regimen, a significant relation to metabolic and anthropometric parameters, whereas VFT and PFT measurements failed to obtain a significant correlation. That AMFT predicted visceral obesity better than VFT and PFT in this study suggests that the detection of AMFT may be an improvement of ultrasound measurements of visceral fat and a new and reliable method for visceral obesity prediction. The identification of vascular structure (abdominal aorta and SMA) is simple and not affected by personal interpretation, and the mesenteric fat will not be distorted by the pressure of a transducer. The ultrasound measurement of AMFT, as a relatively cheap noninvasive and technically less demanding ultrasound method with good reproducibility, could potentially become a useful imagine tool for metabolic syndrome research. Moreover, considering

that visceral fat usually shows greater responses than subcutaneous fat to interventional therapy, such as changes in caloric intake, it can be also applied in evaluating the efficiency of dietetic treatment both in reducing weight and metabolic risk factors.

In conclusion, our technique of ultrasonographic AMFT evaluation is strongly correlated to the presence of metabolic syndrome, and AMFT measurement could be a valuable tool to predict metabolic diseases with its associated risks in men. A study performing a comparative investigation with CT scan or MRI, together with intra- and interobserver analysis, could be mandatory to assess both reliability and reproducibility of our methods, so that the AMFT measurement could be the only ultrasound method to evaluate visceral abdominal fat accumulation.

REFERENCES

1. Dekker JM, Girman C, Rhodes T, et al. Metabolic syndrome and 10-year cardiovascular disease risk in the Hoorn Study. *Circulation* 2005;112:666–673.
2. Grundy SM. Pre-diabetes, metabolic syndrome, and cardiovascular risk. *J Am Coll Cardiol* 2012;59:635–43.
3. Larsson B, Svardsudd K, Welin L, et al. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13-year follow-up of participants in the study of men born in 1913. *Br Med J* 1984;288:1401–4.
4. Bemelmans RH, van der Graaf Y, Nathoe HM, et al; SMART Study Group. Increased visceral adipose tissue is associated with increased resting heart rate in patients with manifest vascular disease. *Obesity* (Silver Spring). 2012;20:834–41.
5. von Eyben FE, Mouritsen E, Holm J, et al. Intra-abdominal obesity and metabolic risk factors: a study of young adults. *Int J Obes Relat Metab Disord* 2003;27:941–949.
6. Després JP. Is visceral obesity the cause of the metabolic syndrome? *Ann Med* 2006;38:52–63.
7. Cannon CP. Mixed dyslipidemia, metabolic syndrome, diabetes mellitus, and cardiovascular disease: clinical implications. *Am J Cardiol* 2008;102:5L–9L.

8. **Bjorntorp P.** Body fat distribution, insulin resistance, and metabolic diseases. *Nutrition* 1997;13:795–803.
9. **Hutley L, Prins JB.** Fat as an endocrine organ: relationship to the metabolic syndrome. *Am J Med Sci* 2005;330:280–289.
10. **Ahima RS, Flier JS.** Adipose tissue as an endocrine organ. *Trends Endocrinol Metab* 2000;11:327–332.
11. **Hill AM, LaForgia J, Coates AM, et al.** Estimating abdominal adipose tissue with DXA and anthropometry. *Obesity (Silver Spring)* 2007;15: 504–10.
12. **Barreira TV, Staiano AE, Harrington DM, et al.** Anthropometric correlates of total body fat, abdominal adiposity, and cardiovascular disease risk factors in a biracial sample of men and women. *Mayo Clin Proc* 2012;87:452–60.
13. **Jacobs EJ, Newton CC, Wang Y, et al.** Waist circumference and all-cause mortality in a large US cohort. *Arch Intern Med.* 2010;170: 1293–301.
14. **Ohsuzu F, Kosuda S, Takayama E, et al.** Imaging techniques for measuring adipose-tissue distribution in the abdomen: a comparison between computed tomography and 1.5-tesla magnetic resonance spin-echo imaging. *Radiat Med* 1998;16:99–107.
15. **Seidell JC, Bakker CJG, van der Kooy K.** Imaging techniques for measuring adipose-tissue distribution – a comparison between computed tomography and 1.5-T magnetic resonance. *Am J Clin Nutr* 1990;51:953–957.
16. **Smith JD, Borel AL, Nazare JA, et al.** Visceral adipose tissue indicates the severity of cardiometabolic risk in patients with and without type 2 diabetes: results from the INSPIRE ME IAA study. *J Clin Endocrinol Metab.* 2012;97:1517–25.
17. **Bazzocchi A, Filonzi G, Ponti F, et al.** Accuracy, reproducibility and repeatability of ultrasonography in the assessment of abdominal adiposity. *Acad Radiol.* 2011;18:1133–43.
18. **De Lucia Rolfe E, Sleigh A, Finucane FM, et al.** Ultrasound measurements of visceral and subcutaneous abdominal thickness to predict abdominal adiposity among older men and women. *Obesity (Silver Spring).* 2010;18:625–31.
19. **van der Kooy K, Seidell JC.** Techniques for the measurement of visceral fat: a practical guide. *Int J Obes Relat Metab Disord* 1993; 17:187–196.
20. **Hirooka M, Kumagi T, Kurose K, et al.** A technique for the measurement of visceral fat by ultrasonography: comparison of measurements by ultrasonography and computed tomography. *Intern Med* 2005; 44:794–799.
21. **Stolk RP, Wink O, Zelissen PM, et al.** Validity and reproducibility of ultrasonography for the measurement of intra-abdominal adipose tissue. *Int J Obes Relat Metab Disord* 2001;25:1346–1351.
22. **Hu G, Qiao Q, Tuomilehto J, et al; DECODE Study Group.** Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic European men and women. *Arch Intern Med* 2004;164:1066–1076.
23. **WHO Consultation.** Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Geneva (Switzerland): World Health Organization; 1999.
24. **Alberti KG, Zimmet P, Shaw J.** Metabolic syndrome: a new worldwide definition. A consensus statement from the International Diabetes Federation. *Diabet Med* 2006;23:469–480.
25. **Poirier P, Giles TD, Bray GA, et al.** Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss. *Arterioscler Thromb Vasc Biol* 2006;26:968–76.
26. **Pi-Sunyer FX.** The role of very-low-calorie diets in obesity. *Am J Clin Nutr* 1992;56(1 suppl):240S–3S.
27. **Fox CS, Massaro JM, Hoffmann U, et al.** Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* 2007;116:39–48.
28. **Iacobellis G, Malavazos AE, Corsi MM.** Epicardial fat: from the bio-molecular aspects to the clinical practice. *Int J Biochem Cell Biol* 2011; 43:1651–4.
29. **Sicari R, Sironi AM, Petz R, et al.** Pericardial rather than epicardial fat is a cardiometabolic risk marker: an MRI vs echo study. *J Am Soc Echocardiogr* 2011;24:1156–62.
30. **Kawasaki S, Aoki K, Hasegawa O, et al.** Sonographic evaluation of visceral fat by measuring para- and perirenal fat. *J Clin Ultrasound* 2008;36:129–33.
31. **Guzzaloni G, Minocci A, Marzullo P, et al.** Sagittal abdominal diameter is more predictive of cardiovascular risk than abdominal fat compartments in severe obesity. *Int J Obes (Lond)* 2009;33:233–8.
32. **Onat A, Hergenç G, Yüksel H, et al.** Neck circumference as a measure of central obesity: associations with metabolic syndrome and obstructive sleep apnea syndrome beyond waist circumference. *Clin Nutr* 2009; 28:46–51.
33. **Ribeiro-Filho FF, Faria AN, Kohlmann O Jr, et al.** Ultrasonography for the evaluation of visceral fat and cardiovascular risk. *Hypertension* 2001;38:713–7.
34. **Stolk RP, Meijer R, Mali WP, et al.** Ultrasound measurements of intraabdominal fat estimate the metabolic syndrome better than do measurements of waist circumference. *Am J Clin Nutr* 2003;77:857–60.
35. **Gradmark AM, Rydh A, Renström F, et al.** Computed tomography-based validation of abdominal adiposity measurements from ultrasonography, dual-energy X-ray absorptiometry and anthropometry. *Br J Nutr* 2010;104:582–8.
36. **Neri S, Signorelli SS, Mondati E, et al.** Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. *J Intern Med* 2005;257: 346–51.
37. **Liu KH, Chan YL, Chan WB, et al.** Sonographic measurement of mesenteric fat thickness is a good correlate with cardiovascular risk factors: comparison with subcutaneous and preperitoneal fat thickness, magnetic resonance imaging and anthropometric indexes. *Int J Obes Relat Metab Disord* 2003;27:1267–73.