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Sonographic evaluation of para- and perirenal fat thickness is an independent predictor of early kidney damage in obese patients

Xiaodong Sun · Fang Han · Wei Miao · Ningning Hou · Zhibin Cao · Guangdong Zhang

Abstract

Purpose   To determine whether para- and perirenal fat ultrasonographic thickness (PFUT) is related to increased urinary albumin excretion and whether PFUT is an independent indicator of early kidney damage in obese subjects.

Method   Sixty-seven nonhypertensive, nondiabetic obese patients and 34 age- and sex-matched normal healthy volunteers were involved in this study. Clinical characteristics, blood biochemistry, PFUT, and urinary albumin/creatinine ratio (ACR) of the subjects were measured. The intraoperator and interoperator coefficient of variation was 5.6 and 3.2 %, respectively.

Results   ACR and PFUT were significantly higher in obese patients than those of normal healthy volunteers. PFUT was higher in obese patients with microalbuminuria than those with normoalbuminuria. Correlation analysis showed PFUT had a positive correlation with body mass index (BMI, $r = 0.677$, $P < 0.01$),

waist circumference (WC, $r = 0.686$, $P < 0.01$), plasma free fatty acids (FFAs, $r = 0.589$, $P < 0.01$), and ACR ($r = 0.610$, $P < 0.01$). ACR had a positive correlation with BMI ($r = 0.444$, $P < 0.01$), WC ($r = 0.440$, $P < 0.01$), and plasma FFAs ($r = 0.496$, $P < 0.01$). Multivariate regression analyses showed that ACR could be predicted by PFUT.

Conclusions   PFUT may be an independent predictor of early kidney damage in nonhypertensive, nondiabetic obese patients, and PFUT could be a useful tool for the assessment of visceral fat and early kidney damage in obese patients.

Keywords   Obesity · Kidney · Ultrasonography

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Introduction

It is known that obesity is not only closely associated with microalbuminuria, an early manifestation of kidney damage, but that it also has a negative impact on ischemic cardiovascular disease [1–3]. There are several mechanisms to explain why obesity may initiate and exacerbate kidney damage, such as hyperglycemia, hyperinsulinemia, and hypertension. However, the PREVEND study showed that 75 % of microalbuminuria occurred in the nonhypertensive, nondiabetic population [4]. Further studies have shown that body fat distribution and abdominal fat accumulation are closely related to increased urinary

albumin excretion and lead to obesity-related kidney damage [5, 6]. Therefore, the convenient evaluation of body fat distribution is very important.

In many studies, body fat distribution was evaluated using a variety of methods, including waist circumstance measurement [7], hip circumference measurement, waist–hip ratio, evaluation of visceral fat by computed tomography (CT) [8, 9], and magnetic resonance imaging (MRI) [10, 11]. Although waist circumstance measurement is noninvasive and convenient, it does not accurately quantify body fat distribution because waist circumstance measurement includes all subcutaneous fat and abdominal contents and subcutaneous fat is more abundant in females than in males. Moreover, subcutaneous fat is usually reduced in advanced old age [12]. CT and MRI present another set of problems, as these imaging modalities are not considered to be desirable measurement options in many cases because CT provides considerable ionizing radiation exposure and MRI is both time-consuming and expensive.

It is worth noting that several noninvasive sonographic methods for measuring body fat distribution have also been reported. Armellini et al. [13] first demonstrated that ultrasonography could be useful in the direct evaluation of intra-abdominal fat deposits. Since then, Kawasaki et al. [14] evaluated a new method of determining visceral fat by measuring para- and perirenal fat thickness on abdominal sonography. Para- and perirenal fat ultrasonographic thickness (PFUT), as part of abdominal visceral fat, is in fact thought to possibly play a significant role in obesity-related microalbuminuria, as it surrounds the kidney directly and demonstrates a closer relationship to kidney function than other intra-abdominal fat deposits [15].

In the present study, we sought to first determine whether PFUT is related to increased urinary albumin excretion and to then ascertain whether PFUT can serve as an independent and robust indicator of early kidney damage in nonhypertensive, nondiabetic obese subjects.

Subjects and methods

Subjects

Nonhypertensive, nondiabetic obese patients (OB group) and normal healthy volunteer (NC group), who are Chinese People, from clinic in our hospital were recruited to this study. Inclusion criteria were as follows: (1) age between 18 and 50 years age; (2) OB group: body mass index ≥ 25 kg/m^2, waist circumference ≥ 90 cm in men, ≥ 80 cm in women, adopted the cutoffs as suggested for Asians [16]; NC group: body mass index 18–23 kg/m^2, waist circumference ≤ 80 cm in men, ≤ 75 cm in women; (3) fasting plasma glucose <6.1 mmol/L and 2 h glucose <7.8 mmol/L; (4) systolic blood pressure ≤ 130 mmHg, diastolic blood pressure ≤ 80 mmHg; (5) total serum cholesterol <6.0 mmol/L; (6) normal liver and renal function. None of the subjects were smokers or had potentially confounding disorders such as cardiovascular disease, autoimmune disease, chronic liver disease, secondary obesity, or long-term use of drugs which affects glucose metabolism such as diuretics, glucocorticoids, etc. We then divided the obese patients into two groups according to their levels of microalbuminuria: obese patients with normoalbuminuria group (OB-NA group) included patients who had normoalbuminuria (urinary albumin/creatinine ratio <30 mg/g), and obese patients with microalbuminuria group (OB-MA group) included patients who had overt microalbuminuria (urinary albumin/creatinine ratio ≥ 30 mg/g) [17]. This study was approved by the ethics committee of our hospital, and informed consent was obtained from all subjects.

Assessment methodology

Height, body weight, waist circumference, and blood pressure were measured using standard methods as described previously [18]. In brief, patients' height and body weight were measured without shoes after overnight fasting by using an electronic scale to the nearest 1 cm and 0.1 kg, respectively. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Plasma glucose, serum lipids, and serum creatinine were measured using a Beckman CX4 automatic biochemical analyzer. Plasma free fatty acids (FFAs) concentrations were determined using colorimetric assays. Urinary albumin/creatinine ratio (ACR) was used as an index of urinary albumin excretion. Early-morning urine samples (first void) were collected from all subjects. Urinary albumin and urinary creatinine concentration were measured by the immunoturbidimetric and Jaffe methods, respectively. eGFR was calculated with the abbreviated Modification of Diet in Renal Disease (MDRD) formula

recalibrated for Chinese [GFR = 186 × (SCr × 0.011)$^{-1.154}$ × (age)$^{-0.203}$ × (0.742 if female) × (1.233 if Chinese)].

Sonographic examination

Sonographic evaluation of PFUT was determined using a GE Logiq S6 ultrasound scanner (General Electric, New York, NY, USA) equipped with a 3.5 MHz convex probe as described previously [14]. The patient was positioned in the supine position and the probe was kept perpendicular to the skin on the lateral aspect of the abdomen. Longitudinal scanning was performed, and the probe was moved laterally to the position where the surface of the kidney was almost parallel to the skin (Fig. 1). As light a pressure as possible was exerted on the probe so that the perirenal fat layers were not compressed. PFUT was then determined from the inner side of the abdominal musculature to the surface of the kidney. The average of the ultrasound measurement values on both sides was regarded as the PFUT. PFUT was measured a total of three times by a sonographer. Interoperator repeatability was assessed as difference toward comparison with CT in 45/101 subjects. The intraoperator and interoperator coefficient of variation was 5.6 and 3.2 %, respectively.

Statistical analysis

All data are subjected to statistical analysis by using SPSS 16.0 statistical package. The data are expressed as mean ± standard deviation (SD) for normally distributed variables and median (interquartile range) for skewed variables. Statistical analysis was performed by one-way ANOVA. The number of inter-group differences was detected using Student–Newman–Keuls (SNK) method. Univariate and multivariate analyses were performed to correlate ACR with independent variables. Skewed variables or nonlinear related variables were log-transformed. P value of less than 0.05 was considered statistically significant.

Results

Clinical characteristics

A total of 67 nonhypertensive, nondiabetic obese patients and 34 age- and sex-matched normal healthy volunteers were enrolled as participants in this study. Clinical characteristics of the subjects are described in Table 1. There were no significant differences in age, gender, and blood pressure in the three groups ($P > 0.05$). Compared with normal healthy volunteers, obese patients had a higher weight, BMI, and waist circumference. However, there were no significant differences in the above indices between obese patients with microalbuminuria and obese patients with normoalbuminuria.

Biochemistry parameters

As expected, serum triglyceride and FFAs levels in obese patients were significantly higher than those observed in normal healthy volunteers (Table 1). There were no significant differences in total cholesterol and fasting plasma glucose levels in the three groups ($P > 0.05$). In the obese patients, the plasma FFAs level was higher in patients with microalbuminuria than in those with normoalbuminuria. However, no significant difference was found in patients' serum triglyceride levels in the two obese groups. Urinary albumin excretion was significantly higher in obese patients than in those of normal healthy volunteers (Table 1).

Echographic parameters

As expected, the mean PFUT was significantly higher in obese patients than in normal healthy volunteers

Fig. 1 Sonographic measurement of para- and perirenal fat thickness. Longitudinal marker shows the distance between the musculature of the abdominal wall and the surface of the kidney

Table 1 Clinical characteristics and blood biochemistry of three groups

Group	NC ($n = 34$)	OB-NA ($n = 32$)	OB-MA ($n = 35$)
Males/females	16/18	16/16	17/18
Age (years)	28 ± 4.8	29.5 ± 8.2	30.4 ± 9.15
Height (m)	1.65 ± 0.08	1.63 ± 0.09	1.65 ± 0.09
Weight (kg)	57.7 ± 8.6	83.1 ± 13.0*	81.5 ± 12.7*
BMI (kg/m^2)	21.1 ± 1.8	30.92 ± 3.38*	29.98 ± 3.54*
WC (cm)	74.8 ± 1.8	99.8 ± 11.5*	101.2 ± 9.2*
SBP (mmHg)	117 ± 7	120 ± 9	121 ± 8
DBP (mmHg)	73 ± 5	76 ± 5	76 ± 6
FBG (mmol/L)	4.48 ± 0.31	4.71 ± 0.41	4.77 ± 0.65
TC (mmol/L)	4.29 ± 0.74	4.30 ± 0.81	4.64 ± 0.56
TG(mmol/L)	0.85 ± 0.27	1.57 ± 0.77*	1.80 ± 0.61*
LDL-C (mmol/L)	2.33 ± 0.64	2.41 ± 0.58	2.73 ± 0.51*#
HDL-C (mmol/L)	1.54 ± 0.25	1.32 ± 0.28*	1.39 ± 0.36
FFAs (umol/L)	396.1 ± 82.9	647.0 ± 170.2*	812.5 ± 219.6*#
eGFR (mL/min/1.73 m^2)	124.6 ± 20.0	128.5 ± 16.8	129.1 ± 18.7
ACR (mg/g)	3.85 (1.3–20.7)	4.35 (1.20–20.41)	89.3 (30.2–160.9) *#
PFUT (mm)	7.95 ± 1.57	23.98 ± 4.87*	29.02 ± 4.92*#

NC normal control, *OB-NA* obese patients with normoalbuminuria, *OB-MA* obese patients with microalbuminuria, *BMI* body mass index, *WC* waist circumstance, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *FBG* fasting blood glucose, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *FFAs* free fatty acids, *eGFR* estimated glomerular filtration rate, *ACR* urinary albumin/creatinine ratio, *PFUT* para- and perirenal fat ultrasonographic thickness

* $P < 0.05$ compared to NC group, # $P < 0.05$ compared to OB-NA group

Table 2 Correlation analysis between PFUT, ACR, and other different indices

	PFUT	ACR		PFUT	ACR		PFUT	ACR
BMI	0.677*	0.444*	TC	0.135	0.194	Glucose	0.152	0.163
WC	0.686*	0.440*	TG	0.357*	0.364*	FFAs	0.589*	0.496*
SBP	0.183	0.094	LDL-C	0.302*	0.313*	eGFR	0.083	−0.033

BMI body mass index, *WC* waist circumstance, *SBP* systolic blood pressure, *TC* total cholesterol, *TG* triglyceride, *LDL-C* low-density lipoprotein cholesterol, *FFAs* free fatty acids, *ACR* urinary albumin/creatinine ratio, *eGFR* estimated glomerular filtration rate, *PFUT* para- and perirenal fat ultrasonographic thickness

* $P < 0.01$

(26.54 ± 5.48 vs 7.95 ± 1.57 mm, $P < 0.05$). In the obese patients, PFUT was higher in patients with microalbuminuria than in those with normoalbuminuria (29.02 ± 4.92 vs 23.98 ± 4.87 mm, $P < 0.05$). There was no significant difference between the right and left side in each group (NC right vs left 7.86 ± 1.52 vs 7.94 ± 1.63 mm; OB-NA 23.90 ± 4.94 vs 24.06 ± 4.97 mm, OB-MA 29.06 ± 4.82 vs 28.98 ± 5.12 mm, $P > 0.05$).

Correlation and regression analysis

Correlation analysis showed that PFUT had a positive correlation with BMI ($r = 0.677$, $P < 0.01$), waist circumference ($r = 0.686$, $P < 0.01$), plasma FFAs ($r = 0.589$, $P < 0.01$), and ACR ($r = 0.610$, $P < 0.01$). ACR had a positive correlation with BMI ($r = 0.444$, $P < 0.01$), waist circumference($r = 0.440$, $P < 0.01$), plasma FFAs ($r = 0.496$, $P < 0.01$),

Table 3 Unstandardized and standardized β-coefficients for associations of ACR with PFUT, FFAs, TG and LDL-C, age and SBP in multivariate analysis

ACR	Unstandardized β-coefficients	SE	Standardized β-coefficients	P
PFUT	0.023	0.005	0.445	0.000
FFAs	0.001	0.000	0.208	0.067
TG	0.001	0.096	0.001	0.988
LDL-C	0.166	0.095	0.150	0.084
Age	0.005	0.008	0.055	0.509
SBP	0.001	0.007	0.008	0.926

TG triglyceride, *LDL-C* low-density lipoprotein cholesterol, *FFAs* free fatty acids, *ACR* urinary albumin/creatinine ratio, *eGFR* estimated glomerular filtration rate, *SBP* systolic blood pressure, *PFUT* para- and perirenal fat ultrasonographic thickness ($r^2 = 0.442$, $P < 0.001$)

and serum triglyceride ($r = 0.364$, $P < 0.01$) (Table 2) Multivariate regression analyses showed that ACR could be predicted by PFUT ($r^2 = 0.442$, $P < 0.001$) (Table 3).

Discussion

In this study, we demonstrated that PFUT is related to increased urinary albumin excretion and that PFUT may be a good indicator of early kidney damage in nonhypertensive, nondiabetic obese subjects.

Since obesity-related microalbuminuria was first reported by Weisinger in 1974 [2], numerous studies have consistently confirmed the presence of proteinuria in obesity, also known as obesity-related glomerulopathy [19, 20]. Klausen et al. [21] found that there existed a strong association between microalbuminuria and obesity, as based on an observational study of 2,696 subjects. In our study, we also found that urinary albumin excretion was significantly higher in the obese patients than in the normal healthy subjects, which is consistent with our previous study [22]. However, more evidence demonstrated that it was not obesity per se, but rather body fat distribution, that is more closely related to kidney damage [23, 24]. Pinto-Sietsma et al. [24] found that lean subjects with central fat distribution (i.e., excessive visceral fat) are also at high risk of microalbuminuria.

More recently, several studies on the use of sonography for the evaluation of visceral fat volume have been published [25, 26]. Suzuki et al. [27]

showed that the abdominal fat index may be an indicator of visceral fat deposition. However, Kawasaki et al. [14] found that the abdominal fat index is not a good indicator of visceral fat due to its inherent measurement difficulty. They used PFUT as a fat-measurement method and found that PFUT correlated significantly with the visceral fat area and that a PFUT of ≥ 10 mm reflects increased visceral fat deposition [14]. In our study, we found that PFUT was significantly higher in obese patients than in normal healthy volunteers. In the obese patients, PFUT was higher in patients with microalbuminuria than in those with normoalbuminuria. Correlation analysis showed that PFUT had a positive correlation with ACR. Multivariate regression analyses showed that ACR could be predicted by PFUT. Taken together, these findings all suggested that PFUT may be a good indicator of visceral fat area and may serve as an independent and accurate predictor of microalbuminuria in nonhypertensive, nondiabetic obese subjects.

Some mechanisms may account for PFUT as an independent predictor of microalbuminuria in obese patients. Part of the perirenal fat can seep into the renal sinus, compress the renal parenchyma and renal vessels, leading to increased renal interstitial hydrostatic pressure and reduced renal blood flow and, ultimately, to kidney damage [28]. In addition, excessive FFAs released from para- and perirenal fat escape into the kidney and lead to renal lipotoxicity by increasing intracellular fatty acids metabolites. In our study, we found that even in obese patients, plasma FFAs levels were higher in patients with microalbuminuria than in those with normoalbuminuria. Correlation analysis showed that PFUT had a positive correlation with FFAs and that FFAs also had a positive correlation with ACR.

In the present study, we confirmed the association of PFUT and urinary albumin excretion in obese subjects. In fact, Lamacchia et al. [15] found that estimates of the glomerular filtration rate, renal resistance index, and uricemia could be predicted by PFUT in 151 subjects with type 2 diabetes, suggesting that PFUT is an independent predictor of kidney dysfunction in this disease. However, in that study, microalbuminuria was not predicted by PFUT likely because the patients recruited had different characteristics. First of all, they were affected by diabetes which may promote glomerular damage by means of mechanisms which are partly different from those activated

by obesity; Moreover, most of the patients were taking antihypertensive therapy which is known to contribute to the reduction in the urinary albumin excretion rate and then it may likely have interfered in the association between PFUT thickness and ACR.

Limitation of our study was that the cross-sectional design in our study helps generate hypotheses, but does not prove the cause–effect relationship between PFUT and early renal damage in obese patients. Therefore, the study design may limit the generalizability of the results but should not influence the internal validity of our findings. However, it should be noted that the study subjects consisted of characterized obese patients with normal glycemia and blood pressure. In addition, the sample size was small, for it is a little difficult to select the obese patients with normal glycemia and blood pressure. The prospective design and larger samples are needed to better clarify the role of PFUT in early kidney damage in obese patients. Admittedly, taking urinary samples for ACR measurement would be much easier and convenient. However, PFUT measurement may improve our estimates of the risk of early kidney damage in obese patients, considering body fat distribution and abdominal fat accumulation are closely related to increased urinary albumin excretion and cardiovascular disease.

In summary, we have shown that urinary albumin excretion was higher in obese patients than in normal healthy subjects. PFUT was higher in obese patients with microalbuminuria than in obese patients without microalbuminuria. Therefore, we believe that PFUT may be an independent predictor of early kidney damage in nonhypertensive, nondiabetic obese patients and that PFUT may be a useful tool for the assessment of visceral fat and early kidney damage in obese patients.

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