PERI-AORTIC FAT TISSUE THICKNESS IN PERITONEAL DIALYSIS PATIENTS

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◆ **Background:** Thoracic peri-aortic fat tissue (PFT) and epicardial adipose tissue (EAT) are metabolically active visceral fat deposits surrounding the thoracic aorta and the heart, respectively. Various studies have demonstrated a positive predictive value of both PFT and EAT for coronary artery disease in the general population. In the present study, we aimed to investigate PFT thickness and the independent predictors of PFT in peritoneal dialysis (PD) patients.

◆ **Methods:** Our cross-sectional study enrolled 35 PD patients (10 women, 25 men) and 30 age-and-sex-matched healthy subjects (15 women, 15 men). We measured PFT, thoracic artery calcification (TAC), EAT, and coronary artery calcification (CAC) by electrocardiogram-gated 64–multi-detector computed tomography.

◆ **Results:** The measured PFT, EAT, CAC, and TAC were significantly higher in the PD group than in the healthy subjects ($p < 0.05$ each). In the PD group, PFT and TAC were significantly correlated ($r = 0.33$, $p = 0.007$). Also, PFT measurements were positively correlated with EAT and total CAC in the PD and the control group alike ($r = 0.58$, $p = 0.001$ and $r = 0.54$, $p = 0.01$ respectively). A stepwise linear regression analysis revealed that age, duration of hypertension, and being a PD patient were independent predictors of PFT.

◆ **Conclusions:** Measured PFT was higher in PD patients than in healthy subjects and, in the PD population, was also shown to be related to calcification scores and EAT.

(PD) and hemodialysis (1). Atherosclerosis, endothelial dysfunction, coronary artery calcification (CAC), left ventricular hypertrophy, and visceral abdominal obesity are the most commonly encountered risk factors in the pathogenesis of cardiovascular diseases in ESRD patients (2–4). Visceral adipose tissue has been observed to contribute to systemic inflammation and demonstrated to be even more relevant to unfavorable clinical outcomes (5). Although clinically convenient, central obesity may not precisely reflect the extent of visceral adipose tissue (6). Epicardial fat tissue (EAT) is the true visceral fat deposit of the heart, accounting for approximately 20% of total heart weight and covering 80% of the cardiac surface, mostly in the grooved segments along the paths of coronary arteries (7–9).

Recently, multi-detector computed tomography (MDCT) in healthy subjects and patients at high risk of coronary artery disease (CAD) showed a close relationship between CAD, thoracic aortic calcification (TAC), and EAT (10–14). Like EAT, thoracic peri-aortic fat tissue (PFT) is considered a metabolically active organ that has a role in the pathogenesis of atherosclerosis and other vascular diseases (15–17). In two recent studies, PFT was found to be associated with peripheral artery disease, abdominal aorta calcification, and CAC (18,19). As a noninvasive imaging method, MDCT can provide reliable data about PFT, EAT, and visceral adipose tissue (20–22). In patients who have a high risk of cardiovascular disease, MDCT can therefore visualize PFT, EAT, TAC, and CAC, all of which are predictors of CAD. However, most of the large randomized controlled MDCT studies have excluded ESRD patients. The exact relationship between PFT and other parameters is therefore unknown in the ESRD population.

Recently, we showed that EAT is significantly increased in ESRD patients, and in that study, we found that EAT was significantly associated with malnutrition–inflammation–atherosclerosis–calcification syndrome (23). However, the literature contains no data about PFT in ESRD patients. We therefore aimed to evaluate

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Cardiovascular diseases are the most common cause of morbidity and mortality in patients with end-stage renal disease (ESRD) receiving peritoneal dialysis

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PFT thickness and independent predictors of PFT in PD patients.

METHODS

The study protocol was approved by the Medical Ethics Committee of Selcuk University (Meram School of Medicine, Konya, Turkey). Written informed consent was obtained from all participants.

Between February and June 2009, this cross-sectional study enrolled 35 ESRD patients (10 women, 25 men; mean age: 54 ± 12 years) who had been receiving PD for 6 months or more at the Dialysis Unit of Selcuk University and 30 healthy control subjects (15 women, 15 men; mean age: 50 ± 11 years).

We screened patients 18–70 years of age who willing to participate in the assessment of PFT, TAC, CAC, and EAT by MDCT. A review of medical records (including information on age, sex, weight, duration of renal replacement therapy, medications, and primary disease leading to ESRD) was used to assess patients for study enrollment. Exclusion criteria were angina pectoris or documented CAD; congestive heart failure; active infection; autoimmune disease; severe secondary hyperparathyroidism (intact parathyroid hormone above 500 pg/mL); and nephrotic-range proteinuria. Of 60 patients screened, 25 were excluded from the study: 5 with documented CAD, 7 with congestive heart failure (New York Heart Association class III–IV), 5 with active infection, 5 with severe secondary hyperparathyroidism; and 3 with an autoimmune disease. The remaining 35 ESRD patients were enrolled in the study. No patient included in the study had nephrotic-range proteinuria or arrhythmia based on electrocardiography. Healthy age- and sex-matched individuals referred from outpatient clinics of the Internal Medicine Department of Selcuk University ($n = 30$) were enrolled as control subjects. They had to meet the same inclusion and exclusion criteria as the patients.

An Erka sphygmomanometer (PMS Instruments Limited, Berkshire, UK) with an appropriate cuff size was used to measure the systolic and diastolic blood pressure (BP) of the patients and healthy subjects. Measurements were taken with the individuals in the upright sitting position after 5 minutes or more of rest. Two readings were recorded for each individual. The mean value of the two readings was defined as the BP. Patients with systolic and diastolic BP readings of 140 mmHg and 90 mmHg or higher and those who were already on antihypertensive treatment were considered hypertensive.

All patients used the same conventional PD solutions (1.36%, 2.27%, and 3.86% glucose; lactate buffer; 1.25%

calcium) from Baxter Healthcare (Deerfield, IL, USA). None of the patients used amino-acid- or icodextrin-containing PD solutions.

We searched our patient database and reviewed the results of the standardized peritoneal equilibration test that had been performed in the same period that the coronary MDCT evaluation was done. Daily glucose loads were calculated from measurements of glucose absorption at the end of 4 hours of the standardized peritoneal equilibration test (24). Monthly glucose loads were calculated by multiplying the daily glucose loads by the total number of days in the associated month.

Of the 35 PD patients, 9 were taking antihypertensive drugs: 5 were taking angiotensin converting-enzyme inhibitors; 3, an angiotensin II receptor blocker; and 1, both a calcium channel blocker and an angiotensin converting-enzyme inhibitor. A total of 20 patients (57%) were taking calcium-containing phosphate binders, and 22 (63%) were taking active vitamin D preparations. No patient was using a vitamin K antagonist.

BIOCHEMICAL ANALYSES

Venous blood samples for biochemical analyses were drawn after an overnight fast and (in the PD patients) before the first exchange. All biochemical analyses—including total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and plasma triglycerides—used an oxidase-based technique on the Roche/Hitachi Modular System (Mannheim, Germany) in the Central Biochemistry Laboratory of the Meram School of Medicine.

EVALUATION OF CAC AND EAT

Unenhanced coronary computed tomography (CT) was quantified on electrocardiography-gated cardiac CT machine retrospectively using a 64-slice MDCT (Somatom Sensation 64: Siemens Medical Solutions, Erlangen, Germany). The coronary CT protocol used a slice collimation of 64×0.6 mm; a gantry rotation time of 0.33 s; a pitch of 0.2 degrees; a tube voltage of 120 kV; and a tube current of 600 mAs. If the patient's heart rate was greater than 65 beats per minute, heart-rate control was achieved using a beta-blocker. Multiplanar data reconstructions were obtained in standardized ventricular short-axis planes at the basal, mid-cavity, apical, and horizontal long axis plane with a 3-mm slice thickness and a 2-mm slice interval (25). To quantify EAT volume, all reconstructions were transferred to a computer-based workstation. A CT attenuation threshold between –200 and –20 Hounsfield units (HU) was used

Figure 1 — (A,B) Epicardial adipose tissue. (C,D) Coronary artery calcification.

to isolate epicardial fat [Figure 1(A,B)]. To quantify CAC, all reconstructions were transferred to a computer-based workstation (Syngo Ca Scoring Wizard: Siemens Medical Solutions). The definition of CAC was more than 2 contiguous pixels measuring more than 130 HU as designated by Agatston *et al.* (26). Values for the left anterior descending coronary artery [Figure 1(C,D)], circumflex coronary artery, and right coronary artery were totaled to calculate the CAC.

EVALUATION OF PFT AND TAC

All image analyses were performed on a dedicated workstation (Leonardo Workstation: Siemens Medical Solutions) as previously described (21). Adipose tissue quantification was performed by a semi-automated method that required manual definition of borders. To identify fat pixels for the calculation of adipose volumes, CT attenuation thresholds were used (window width: -195 HU to -45 HU; window center: -120 HU). Thoracic PFT was defined as the area immediately surrounding the thoracic aorta anteriorly by a horizontal line through the esophagus, connected to the left

costovertebral joint; and posteriorly by the anterior edge of the vertebral body and the right lateral border of the vertebral body (21). From the carina, a 6.75-cm column around the thoracic aorta was quantified [Figure 2(A,B)].

To quantify TAC, all reconstructions were transferred to a computer-based workstation [Syngo Ca Scoring Wizard, Figure 2(C,D)]. A calcified lesion was defined by the presence of at least 3 connected pixels with attenuation exceeding 130 HU. In addition, an Agatston score was calculated by multiplying the lesion area by the attenuation score in Hounsfield units (26).

Measurements of PFT, TAC, EAT, and CAC were evaluated by two radiologists. An intra-class correlation coefficient was used to evaluate inter- and intra-observer variability for the PFT and EAT measurements. For intra-observer variability, one observer reanalyzed 30 randomly selected records after 1 month. For inter-observer variability, the other observer similarly reanalyzed the same patient records at the same time. Intra-observer variability was found to be 0.1%, and inter-observer variability, 0.2% for the measurements of both PFT and EAT.

Figure 2 — (A,B) Peri-aortic fat tissue. (C,D) Thoracic artery calcification.

STATISTICAL ANALYSES

Statistical analyses were performed using the Statistical Package for Social Sciences (Windows version 15.0: SPSS, Chicago, IL, USA). Parametric data are reported as means $\pm$ standard deviation, and nonparametric data, as medians and interquartile range. The normal distribution of all variables was tested using the Kolmogorov–Smirnov test. Dichotomous variables were compared using the chi-square test. Between-group statistical differences for parametric data were analyzed using the Student t-test. For nonparametric data, the Mann–Whitney U-test was used. Linear associations between continuous variables were assessed using Spearman correlation.

Multivariate linear regression analyses were undertaken to identify independent associations between PFT and other variables. Age, body mass index, serum uric acid, total CAC, EAT, TAC, diabetes, duration of hypertension, and group were entered into the regression model as independent variables; PFT was entered as the dependent variable. The backward elimination method was preferred in the stepwise regression analysis, and

$p > 0.1$ used as a criterion for elimination in the model. At the end of the fourth step, 3 variables remained statistically significant in the model, $p < 0.05$ being considered significant for all tests.

A power analysis was performed using a Minitab 16 packet program. Sample volume was calculated as $n = 30$ for each group to determine the difference for 5 cm³ PFT with 80% power. However, the study sample was enlarged to 35 patients in the PD group because of a probability of inadequate record quality secondary to increased vascular calcification.

RESULTS

BASELINE CHARACTERISTICS OF PATIENTS

Table 1 shows the baseline characteristics of the 35 PD patients and 30 healthy subjects. The causes of ESRD in the patient group were diabetic nephropathy ($n = 12$), chronic glomerulonephritis ($n = 6$), hypertensive nephropathy ($n = 9$), polycystic kidney disease ($n = 5$), chronic pyelonephritis ($n = 1$), and unknown ($n = 2$). The groups showed no differences with respect to age, sex,

body mass index, and systolic and diastolic BP (Table 1) or to serum levels of low-density and high-density lipoprotein cholesterol, triglycerides, albumin, alanine aminotransferase, and hemoglobin (Table 2). Values for PFT, EAT, CAC, and TAC were significantly higher in the PD group than in the healthy control subjects (Table 2, Figure 3).

The mean daily and monthly glucose loads in the patients were 70.8 ± 26.5 g and 2118 ± 794 g respectively. We observed no relationship between PFT and the daily and monthly glucose loads (both $r = 0.07$, $p = 0.7$).

The mean daily and monthly elemental calcium loads in the patients were 917 ± 390 mg and $27\,536 \pm 11\,714$ mg respectively. These elemental calcium loads were found to be positively correlated with vitamin D use (both $r = 0.45$, $p = 0.007$) and with TAC (both $r = 0.43$, $p = 0.01$).

Measurements of PFT were not significantly higher in diabetic PD patients than in nondiabetic PD patients (18.8 ± 11.7 cm³ vs 18.9 ± 11.0 cm³, $p = 0.96$).

RELATIONS BETWEEN PFT, TAC, CAC, AND INFLAMMATION

We observed a statistically significant correlation between PFT and TAC in the PD group ($r = 0.33$, $p = 0.007$). Also, PFT measurements were positively correlated with total CAC in PD patients ($r = 0.54$, $p = 0.01$) and healthy control subjects ($r = 0.36$, $p = 0.05$). Only 3 healthy

TABLE 1
Demographic and Clinical Features of the Study Participants

Parameter	Group		<i>p</i> Value
	Healthy subjects	PD patients	
Participants (<i>n</i>)	30	35	
Age (years)	50 ± 11	54 ± 12	0.23
Sex (<i>n</i> men/women)	15/15	25/10	0.15
Mean BMI (kg/m ²)	26 ± 5	28 ± 5	0.13
Mean dialysis vintage (months)	—	56 ± 26	—
Mean blood pressure (mmHg)			
Systolic	132 ± 8	135 ± 26	0.60
Diastolic	82 ± 7	84 ± 16	0.55
Cause of CKD [<i>n</i> (%)]			
Diabetes	—	12 (34)	—
Glomerulonephritis	—	6 (17)	—
Hypertension	—	9 (26)	—
Polycystic kidney disease	—	5 (14)	—
Chronic pyelonephritis	—	1 (3)	—
Unknown	—	2 (6)	—

PD = peritoneal dialysis; BMI = body mass index; CKD = chronic kidney disease.

TABLE 2
Laboratory Features, Vascular Calcification, and Visceral Fat Tissue in the Study Groups

Parameter	Group		<i>p</i> Value
	Healthy subjects (<i>n</i> =30)	PD patients (<i>n</i> =35)	
Hemoglobin (mg/dL)	12.4 ± 2.2	11.5 ± 1.9	0.11
Albumin (g/dL)	3.9 ± 0.9	3.6 ± 0.4	0.23
Uric acid (mg/dL)	—	5.4 ± 1.0	—
ALT (IU/L)	16 ± 6	17 ± 6	0.68
Cholesterol (mg/dL)			
LDL	94 ± 6.0	120 ± 31	0.17
HDL	38 ± 12	37 ± 13	0.83
Triglycerides (mg/dL)	128 ± 88	170 ± 104	0.12
Calcium (mg/dL)	—	9.0 ± 0.7	—
Phosphorus (mg/dL)	—	4.1 ± 0.9	—
Median intact PTH (pg/mL)	—	240 (221)	—
Glucose load (g)			
Daily	—	70.8 ± 26.5	—
Monthly	—	2118 ± 794	—
Calcium load (mg)			
Daily	—	917 ± 390	—
Monthly	—	$27\,536 \pm 11\,714$	—
A1C (%)	—	7.8 ± 1.5	—
Median total CAC	0 (2)	18.8 (116)	0.02
EAT (cm ³)	120 ± 43	170 ± 79	0.003
PFT (cm ³)	13 ± 7	19 ± 11	0.02
Median TAC	0 (0)	0 (147)	0.04

PD = peritoneal dialysis; ALT = alanine aminotransferase; LDL = low-density lipoprotein; HDL = high-density lipoprotein; PTH = parathyroid hormone; CAC = coronary artery calcification; EAT = epicardial adipose tissue; PFT = peri-aortic fat tissue; TAC = thoracic aortic calcification.

Figure 3 — Correlation between peri-aortic fat tissue and epicardial adipose tissue in healthy control subjects.

control subjects had detectable TAC measurements, and so correlation analyses involving TAC were not performed in that group.

Correlation analysis for PFT and high-sensitivity C-reactive protein showed no relation between those parameters ($r = 0.17$, $p = 0.2$). Correlations between CAC, TAC, and high-sensitivity C-reactive protein were also not statistically significant ($r = 0.24$, $p = 0.06$ and $r = 0.08$, $p = 0.56$ respectively).

RELATIONS BETWEEN EAT, TAC, AND CAC

In PD patients, we observed a statistically significant relation between EAT and CAC ($r = 0.34$, $p = 0.007$) and also a strong correlation between total CAC and TAC ($r = 0.66$ and $p = 0.001$).

RELATIONS BETWEEN PFT AND EAT

Measurements of PFT were significantly correlated with EAT in PD patients ($r = 0.58$, $p = 0.001$) and healthy control subjects ($r = 0.37$, $p = 0.05$). The linear association between PFT and EAT was stronger in the PD patients.

PREDICTORS OF PFT

In the bivariate correlation analysis, PFT correlated positively with age, body mass index, duration of hypertension, serum uric acid levels, TAC, CAC, and EAT (Table 3). The stepwise linear regression analysis revealed that age, status as a PD patient, and duration of hypertension were all independent predictors of PFT. In univariate linear regression analysis, diabetes mellitus was not found to be a predictor of PFT ($\beta = -0.154$, $t = -0.03$, $p = 0.96$).

Table 4 shows the regression results.

DISCUSSION

The present study is the first to evaluate PFT in ESRD patients. These are our three main findings:

- PFT is significantly higher in PD patients than in healthy control subjects.
- Measurements of PFT are positively correlated with both EAT and total CAC in PD patients and control subjects. Also, PFT is correlated with TAC in PD patients. In the PD patients, the linear association between PFT and CAC was stronger than that between EAT and CAC.
- Age, status as a PD patient, and duration of hypertension are independent predictors of increased PFT.

To our knowledge, this is the first study to demonstrate that PFT thickness is increased in PD patients. Growing evidence suggests that there is an important link between specific adipose tissues and cardiovascular diseases, including atherosclerosis, aortic calcification, and peripheral arterial disease (17–19). The association between TAC and CAD has been demonstrated in previous studies, and traditional cardiovascular risk factors have been shown to share a role in atherosclerosis of the aorta and the coronary arteries (27,28).

Peri-aortic fat tissue is a novel fat deposit surrounding the thoracic aorta. In recent studies, PFT has been found to be associated with peripheral arterial disease, abdominal aorta calcification, and CAC (18,19). In the present study, we showed that PFT and TAC were significantly higher in PD patients than in healthy control subjects. The relation between PFT and TAC in PD patients was found to be statistically significant.

Epicardial adipose tissue, a metabolically active visceral fat deposit surrounding the heart, is considered to be a good indicator of the presence and extension of CAD (29). Sarin *et al.* (30) showed that higher EAT volumes are associated with increased CAC, CAD, and

TABLE 3
Linear Relationship Between Peri-aortic Fat Tissue and Other Parameters in Peritoneal Dialysis Patients

Parameter	<i>r</i>	<i>p</i> Value
Age (years)	0.57	0.001
Body mass index (kg/m ²)	0.46	0.001
Duration of HTN	0.62	0.01
Uric acid (mg/dL)	0.40	0.01
Total CAC score	0.54	0.01
Epicardial adipose tissue	0.58	0.001
Thoracic aortic calcification	0.33	0.007

HTN = hypertension; CAC = coronary artery calcification.

TABLE 4
Independent Predictors of Peri-aortic Fat Tissue in Linear Regression Analysis^a

Parameter	β	95% CI	<i>p</i> Value
Age	0.50	0.27 to 0.59	0.001
Duration of HTN	0.48	0.21 to 0.76	0.005
Group (being a PD patient)	3.2	1.10 to 6.80	0.05

CI = confidence interval; HTN = hypertension; PD = peritoneal dialysis.

^a $r^2 = 0.53$; adjusted $r^2 = 0.51$; model statistics: $p = 0.001$.

metabolic syndrome. In a recent study, Yorgun *et al.* (14) demonstrated that EAT thickness is associated with aortic atherosclerosis only in patients with critical coronary atherosclerosis. In that study, advanced age, EAT thickness, and critical coronary stenosis were found to be independent predictors of plaque score in descending thoracic aorta atherosclerosis (14). In the present study, EAT and total CAC were significantly higher in PD patients than in healthy control subjects, and the relation between EAT and CAC was found to be statistically significant in PD patients.

The foregoing risk factors are similar to those acting in the pathogenesis of atherosclerosis. Recent clinical and epidemiologic studies have shown that uric acid may have a contributory causal role in the pathogenesis of hypertension and kidney diseases (31–33). Therefore, when taken together, these results can explain the relationship between visceral fat (including both PFT and EAT), extended atherosclerosis, and vascular calcification.

The results of our study suggest that the linear association between PFT and CAC is stronger than the relation between EAT and CAC in PD patients. We can therefore conclude that, compared with measurement of EAT, measurement of PFT may be more valuable for predicting CAD risk.

In healthy control subjects, PFT also correlated with CAC. However, the linear relation between PFT and CAC was found to be much stronger in PD patients than in healthy control subjects.

Measurements of PFT correlated significantly with EAT in PD patients and healthy control subjects alike. Nonetheless, the linear association between PFT and EAT was stronger in PD patients than in healthy control subjects.

Glucose metabolism is altered in patients with chronic kidney disease even in its earlier stages. The main explanations for this pathology in the chronic kidney disease population are increased insulin resistance and gluconeogenesis, and abnormal glucose uptake. Those factors can be attributed to the high glucose load absorbed from peritoneal dialysate, which may cause metabolic disturbances, including hypertriglyceridemia, diabetes, and obesity (34). In addition, continuous exposure to extremely high dialysate glucose concentrations leads to the formation of advanced glycosylation end-products (35,36). In a retrospective 5-year cohort study, Wu *et al.* (37) showed that the combination of diabetes, obesity, and low Kt/V is significantly correlated with high dialysate glucose concentration during the first 3 years of therapy. In the present study, the mean daily and monthly glucose loads in the patients were

70.8 ± 26.5 g and 2118 ± 794 g respectively. In the linear correlation analysis, we did not observe a correlation between the daily or monthly glucose load and measurements of PFT or EAT. Our study sample was too small to define a relationship between glucose load and visceral fat tissue; however, the additional glucose exposure in PD patients could be one of the potential explanations for differences in PFT between the PD patients and the control subjects.

Measurements of PFT were not significantly higher in diabetic than in nondiabetic PD patients (18.8 ± 11.7 cm³ vs 18.9 ± 11.0 cm³, $p = 0.96$). In the univariate regression analysis, diabetes was not found to be an independent predictor of PFT. That result can be attributed to two main factors: First, the mean HbA1C level in the diabetic patients was $7.8\% \pm 1.5\%$, which means that their glycemic control was not poor. Second, the study sample was relatively small for identifying a relation between diabetes and PFT.

Calcium-based phosphate binders have been found to be associated with progressive CAC and aortic calcification in hemodialysis patients when mineral metabolism is poorly controlled (38). London *et al.* (39) showed that ESRD patients with low bone activity are susceptible to worsening of vascular calcification and stiffening when exposed to a high calcium load. Demirci and coworkers (40) also demonstrated that patients receiving PD fluid with a high calcium concentration (1.75 mmol/L) showed an increase in arterial stiffening compared with patients using PD fluid with a low calcium concentration (who showed no change). Additionally, administration of large doses of vitamin D analogs may also contribute to vascular calcification (41,42). Findings in the present study are consistent with those in previous studies, and we observed a positive correlation between daily and monthly calcium loads and TAC and vitamin D use.

Our study has two main limitations. First, this analysis of PD patients focusing on PFT was cross-sectional in nature. Second, as mentioned earlier, the sample size was relatively small. Being that this was not a prospective controlled study, we cannot draw cause-and-effect conclusions from our findings.

CONCLUSIONS

Our study showed that measurements of PFT were higher in PD patients than in healthy control subjects. Measured PFT was also shown to be related to calcification scores and EAT in PD patients. Further well-designed randomized controlled trials are needed to confirm our results.

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DISCLOSURES

No author has a financial conflict of interest to disclose.

REFERENCES

1. Kremen J, Dolinkova M, Krajickova J, Blaha J, Anderlova K, Lacinova Z, *et al.* Increased subcutaneous and epicardial adipose tissue production of proinflammatory cytokines in cardiac surgery patients: possible role in postoperative insulin resistance. *J Clin Endocrinol Metab* 2006; 91:4620–7.
2. Parfrey PS, Foley RN, Harnett JD, Kent GM, Murray D, Barré PE. Outcome and risk factors of ischemic heart disease in chronic uremia. *Kidney Int* 1996; 49:1428–34.
3. Collins AJ. Cardiovascular mortality in end-stage renal disease. *Am J Med Sci* 2003; 325:163–7.
4. Parfrey PS, Foley RN, Harnett JD, Kent GM, Murray DC, Barré PE. Outcome and risk factors for left ventricular disorders in chronic uraemia. *Nephrol Dial Transplant* 1996; 11:1277–85.
5. Després JP, Ferland M, Moorjani S, Nadeau A, Tremblay A, Lupien PJ, *et al.* Role of hepatic-triglyceride lipase activity in the association between intra-abdominal fat and plasma HDL cholesterol in obese women. *Arteriosclerosis* 1989; 9:485–92.
6. van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Visser M. Abdominal diameters as indicators of visceral fat: comparison between magnetic resonance imaging and anthropometry. *Br J Nutr* 1993; 70:47–58.
7. Iacobellis G, Corradi D, Sharma AM. Epicardial adipose tissue: anatomic, biomolecular and clinical relationships with the heart. *Nat Clin Pract Cardiovasc Med* 2005; 2:536–43.
8. Corradi D, Maestri R, Callegari S, Pastori P, Goldoni M, Luong TV, *et al.* The ventricular epicardial fat is related to the myocardial mass in normal, ischemic and hypertrophic hearts. *Cardiovasc Pathol* 2004; 13:313–16.
9. Shirani J, Berezowski K, Roberts WC. Quantitative measurement of normal and excessive (cor adiposum) sub-epicardial adipose tissue, its clinical significance, and its effect on electrocardiographic QRS voltage. *Am J Cardiol* 1995; 76:414–18.
10. Djaberi R, Schuijff JD, van Werkhoven JM, Nucifora G, Jukema JW, Bax JJ. Relation of epicardial adipose tissue to coronary atherosclerosis. *Am J Cardiol* 2008; 102:1602–7.
11. Mazurek T, Zhang L, Zalewski A, Mannion JD, Diehl JT, Ara-fat H, *et al.* Human epicardial adipose tissue is a source of inflammatory mediators. *Circulation* 2003; 108:2460–6.
12. Park MJ, Jung JI, Oh YS, Youn HJ. Assessment of epicardial fat volume with threshold-based 3-dimensional segmentation in CT: comparison with the 2-dimensional short axis-based method. *Korean Circ J* 2010; 40:328–33.
13. Eroglu S, Sade LE, Yildirim A, Bal U, Ozbicer S, Ozgul AS, *et al.* Epicardial adipose tissue thickness by echocardiography is a marker for the presence and severity of coronary artery disease. *Nutr Metab Cardiovasc Dis* 2009; 19:211–17.
14. Yorgun H, Canpolat U, Hazirolan T, Sunman H, Ateş AH, Gürses KM, *et al.* Epicardial adipose tissue thickness predicts descending thoracic aorta atherosclerosis shown by multidetector computed tomography. *Int J Cardiovasc Imaging* 2011; :[Epub ahead of print].
15. Chatterjee TK, Stoll LL, Denning GM, Harrelson A, Blomkalns AL, Idelman G, *et al.* Proinflammatory phenotype of perivascular adipocytes: influence of high-fat feeding. *Circ Res* 2009; 104:541–9.
16. Yudkin JS, Eringa E, Stehouwer CD. “Vasocrine” signalling from perivascular fat: a mechanism linking insulin resistance to vascular disease. *Lancet* 2005; 365:1817–20.
17. Fantuzzi G, Mazzone T. Adipose tissue and atherosclerosis: exploring the connection. *Arterioscler Thromb Vasc Biol* 2007; 27:996–1003.
18. Fox CS, Massaro JM, Schlett CL, Lehman SJ, Meigs JB, O'Donnell CJ, *et al.* Periaortic fat deposition is associated with peripheral arterial disease: the Framingham heart study. *Circ Cardiovasc Imaging* 2010; 3:515–19.
19. Lehman SJ, Massaro JM, Schlett CL, O'Donnell CJ, Hoffmann U, Fox CS. Peri-aortic fat, cardiovascular disease risk factors, and aortic calcification: the Framingham Heart Study. *Atherosclerosis* 2010; 210:656–61.
20. Mahabadi AA, Massaro JM, Rosito GA, Levy D, Murabito JM, Wolf PA, *et al.* Association of pericardial fat, intrathoracic fat, and visceral abdominal fat with cardiovascular disease burden: the Framingham Heart Study. *Eur Heart J* 2009; 30:850–6.
21. Schlett CL, Massaro JM, Lehman SJ, Bamberg F, O'Donnell CJ, Fox CS, *et al.* Novel measurements of periaortic adipose tissue in comparison to anthropometric measures of obesity, and abdominal adipose tissue. *Int J Obes (Lond)* 2009; 33:226–32.
22. 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; 117:605–13.
23. Turkmen K, Kayikcioglu H, Ozbek O, Solak Y, Kayrak M, Samur C, *et al.* The relationship between epicardial adipose tissue and malnutrition, inflammation, atherosclerosis/calcification syndrome in ESRD patients. *Clin J Am Soc Nephrol* 2011; 6:1920–5.
24. Twardowski ZJ, Nolph KD, Khanna R, Prowant BF, Ryan LP, Moore HL, *et al.* Peritoneal equilibration test. *Perit Dial Bull* 1987; 7:138–47.

25. Abbara S, Desai JC, Cury RC, Butler J, Nieman K, Reddy V. Mapping epicardial fat with multi-detector computed tomography to facilitate percutaneous transepical arrhythmia ablation. *Eur J Radiol* 2006; 57:417–22.
26. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol* 1990; 15:827–32.
27. Taniguchi H, Momiyama Y, Fayad ZA, Ohmori R, Ashida K, Kihara T, *et al.* *In vivo* magnetic resonance evaluation of associations between aortic atherosclerosis and both risk factors and coronary artery disease in patients referred for coronary angiography. *Am Heart J* 2004; 148:137–43.
28. Khoury Z, Gottlieb S, Stern S, Keren A. Frequency and distribution of atherosclerotic plaques in the thoracic aorta as determined by transesophageal echocardiography in patients with coronary artery disease. *Am J Cardiol* 1997; 79:23–7.
29. Ahn SG, Lim HS, Joe DY, Kang SJ, Choi BJ, Choi SY, *et al.* Relationship of epicardial adipose tissue by echocardiography to coronary artery disease. *Heart* 2008; 94:e7.
30. Sarin S, Wenger C, Marwaha A, Qureshi A, Go BD, Woomert CA, *et al.* Clinical significance of epicardial fat measured using cardiac multislice computed tomography. *Am J Cardiol* 2008; 102:767–71.
31. Kanbay M, Ozkara A, Selcoki Y, Isik B, Turgut F, Bavbek N, *et al.* Effect of treatment of hyperuricemia with allopurinol on blood pressure, creatinine clearance, and proteinuria in patients with normal renal functions. *Int Urol Nephrol* 2007; 39:1227–33.
32. Goicoechea M, de Vinuesa SG, Verdalles U, Ruiz-Caro C, Ampuero J, Rincón A, *et al.* Effect of allopurinol in chronic kidney disease progression and cardiovascular risk. *Clin J Am Soc Nephrol* 2010; 5:1388–93.
33. Menè P, Punzo G. Uric acid: bystander or culprit in hypertension and progressive renal disease? *J Hypertens* 2008; 26:2085–92.
34. Fortes PC, de Moraes TP, Mendes JG, Stinghen AE, Ribeiro SC, Pecoits-Filho R. Insulin resistance and glucose homeostasis in peritoneal dialysis. *Perit Dial Int* 2009; 29(Suppl 2):S145–8.
35. Nakayama M, Kawaguchi Y, Yamada K, Hasegawa T, Takazoe K, Katoh N, *et al.* Immunohistochemical detection of advanced glycosylation end-products in the peritoneum and its possible pathophysiological role in CAPD. *Kidney Int* 1997; 51:182–6.
36. Ateshkadi A, Johnson CA, Founds HW, Zimmerman SW. Serum advanced glycosylation end-products in patients on hemodialysis and CAPD. *Perit Dial Int* 1995; 15:129–33.
37. Wu HY, Hung KY, Hu FC, Chen YM, Chu TS, Huang JW, *et al.* Risk factors for high dialysate glucose use in PD patients—a retrospective 5-year cohort study. *Perit Dial Int* 2010; 30:448–55.
38. Chertow GM, Raggi P, Chasan-Taber S, Bommer J, Holzer H, Burke SK. Determinants of progressive vascular calcification in haemodialysis patients. *Nephrol Dial Transplant* 2004; 19:1489–96.
39. London GM, Marchais SJ, Guérin AP, Boutouyrie P, Métivier F, de Vernejoul MC. Association of bone activity, calcium load, aortic stiffness, and calcifications in ESRD. *J Am Soc Nephrol* 2008; 19:1827–35.
40. Demirci MS, Ozkahya M, Asci G, Sevinc E, Yilmaz M, Demirci C, *et al.* The influence of dialysate calcium on progression of arterial stiffness in peritoneal dialysis patients. *Perit Dial Int* 2009; 29(Suppl 2):S15–17.
41. Mizobuchi M, Finch JL, Martin DR, Slatopolsky E. Differential effects of vitamin D receptor activators on vascular calcification in uremic rats. *Kidney Int* 2007; 72:709–15.
42. Wang AY. Vascular and valvular calcification in chronic peritoneal dialysis patients. *Int J Nephrol* 2011; 2011:198045.