Association Between Vascular Access Type and Visceral and Peripheral Body Fat, Nutritional and Inflammatory Parameters in Incident Hemodialysis Patients

Hemodiyaliz Hastalarında Damar Ulaşım Yolu Tipi ile Visseral ve Periferal Yağ Dokusu, Nütrisyonel ve İnflamatuvar Parametrelerin İlişkisi

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ABSTRACT

OBJECTIVE: Central venous catheters (CVCs) are preferred when a patent arteriovenous fistula (AVF) cannot be created. CVCs have been shown to be associated with increased inflammation and mortality. In the present study, we aimed to investigate a probable association between the vascular access type and BMI, total body fat, truncal fat, muscle mass, visceral (periaortic) fat, and the malnutrition inflammation atherosclerosis and calcification score (MIAC) in hemodialysis patients.

MATERIAL and METHODS: A total of 119 patients were involved. Ninety patients had patent AVF and 29 patients were undergoing hemodialysis via permanent jugular catheter. Two-dimensional echocardiography was performed to detect valvular calcification. Computed tomography was performed in all patients to detect the amount of thoracic periaortic fat tissue (T-PAFT). Biochemical analyses were performed using c8000 Architect. MIAC scores were calculated using valvular calcification, albumin, and CRP. Total body fat and truncal fat were detected using the bio impedance analysis method. Aortic calcification score (AoCS) was calculated using routine chest x-rays.

RESULTS: MIAC and AoCS was higher in patients with CVC (p=0.02 and 0.032). T-PAFT was higher in patients with AVF (1631.5±645 vs 1112.2±606.8; p=0.035). CRP was higher in patients with CVC (p=0.04). Hemodialysis vintage, calcium and albumin were lower in patients with CVC (p=0.01). Truncal fat (%), cholesterol and 25-OH vitamin D levels were lower in patients with CVC (p=0.04, p=0.02, p=0.03). T-PAFT was a significant predictor of vascular access type in favour of AVF (t=-2.17; p=0.04).

CONCLUSION: The present study revealed that HD patients with CVC had increased inflammation and decreased nutrition, visceral and truncal fat. Further prospective studies are needed to illuminate the relationship between vascular access type, nutritional parameters and body composition in HD patients.

KEY WORDS: Hemodialysis, Central venous catheter, Nutrition, Inflammation, Periaortic fat

ÖZ

AMAÇ: Santral venöz kateterler (SVK) başarılı bir arteriovenöz fistül (AVF) oluşturulamadığı durumlarda tercih edilirler. SVK'ler artmış inflamasyon ve mortaliteyle birliktedir. Bu çalışmada, hemodiyaliz hastalarında damar ulaşım yolu tipi ile vücut kitle indeksi (VKİ), toplam vücut yağı, gövdesel yağ, kas kitlesi, visseral (periaortik) yağ, malnutrisyon, inflamasyon ateroskleroz, kalsifikasyon skoru (MIAC) arasındaki muhtemel ilişki araştırılmıştır.

GEREÇ ve YÖNTEMLER: 119 hasta çalışmaya alındı. 90 hastada AVF, 29 hasta kalıcı juguler kateter mevcuttu. Valvüler kalsifikasyon iki boyutlu ekokardiyografi ile torasik periaortik yağ dokusu (T-PAFT) bilgisayarlı tomografi ile saptandı. Biyokimyasal analizler c8000 Architect cihazıyla çalışıldı. MIAC skoru, valvüler kalsifikasyon skoru, albumin ve CRP değerleri ile hesaplandı. Toplam vücut yağı ve trunkal yağ biyoimpedans analiz yöntemi ile çalışıldı. Aortik kalsifikasyon skoru (AoCS) rutin akciğer grafisinden hesaplandı.

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BULGULAR: MIAC ve AoCS, SVK olan hastalarda daha yüksekti (p=0,02 ve 0,032). T-PAFT, AVF grubunda daha yüksekti (1631,5±645'e karşı 1112,2±606,8; p=0,035). CRP, SVK olan hastalarda daha yüksekti (p=0,04). Hemodiyaliz süresi, kalsiyum ve albumin SVK olan grupta daha düşüktü (p=0,01). Gövde yağ oranı (%), kolesterol ve 25-OH vitamin D düzeyi SVK olan grupta daha düşüktü (p=0.04, p=0.02, p=0.03). T-PAFT miktarı AVF lehine damar ulaşım yolunun anlamlı bir belirleyicisi idi (t=-2,17; p=0,04).

SONUÇ: Çalışmada SVK'i olan hemodiyaliz hastalarının artmış inflamasyon, azalmış nütrisyon, visseral ve gövdesel yağ oranına sahip olduğu gözlenmiştir.

ANAHTAR SÖZCÜKLER: Hemodiyaliz, Santral venöz kateter, Nütrisyon, İnflamasyon, Periaortik yağ

INTRODUCTION

Arteriovenous fistula (AVF) is the most preferred vascular access type and associated with the lowest incidence of morbidity and mortality (1-5). CVC is associated with the greatest morbidity and mortality among the vascular accesses but could be preferred in case of failure to achieve a patent AVF and arterio-venous graft (AVG) or secondary failure of AVF/ AVG as a last choice (6-16)

Although the pathogenesis of AVF and AVG failure is not completely understood, inflammatory mediators and leukocyte migration were shown to be associated with the level of neointimal hyperplasia (NIH) (16,17). On the other hand, it has been suggested that CVC may induce inflammation, atherosclerosis and calcification and may be associated with the failure of future vascular operations.

Vascular calcification is also a component of malnutrition, inflammation, atherosclerosis/ calcification complex (MIAC) which is a well known feature of patients undergoing maintenance hemodialysis (HD) (18). Inflammation also contributes to the vascular calcification process (19). It has been demonstrated that presence of CVC and AVG was associated with 62% and 30% increase in CRP levels respectively compared to AVF (19). Higher CRP levels were shown to be associated with increased risk of CVC failure compared to lower CRP levels. Increased level of inflammatory mediators was shown in patients with CVC and AVG compared to AVF in HD patients in another study (20).

Obesity in opposite direction to malnutrition could also be a risk for failure of AVF. Obese patients had a significantly lower intraoperative blood flow (IOBF), and both obesity and low IOBF contribute to the primary maturation failure of AVF. Obesity-associated inflammation and atherosclerosis might play a role in this association (21). A larger study on incident hemodialysis patients demonstrated that poorer AVF maturity in the highest body-mass index (BMI) quartile (22). Furthermore, it has been shown that truncal fat mass may contribute to inflammation in end-stage renal disease (ESRD) (22, 23). The results of those studies suggested that obesity and truncal fat mass exhibit a distinct effect on chronic inflammation in HD patients.

Existing data are not enough to demonstrate an association between body fat composition and vascular access choice or the success of AVF. Therefore, in the present study we aimed to investigate a probable association between the vascular access type and BMI, bio-impedance analysis variables (total body fat, truncal fat, muscle mass), the malnutrition inflammation atherosclerosis and calcification score (MIAC) and vitamin D levels in maintenance hemodialysis (MHD) patients. We also studied the amount of thoracic (periaortic) fat tissue as an indicator of visceral fat, and its possible relationship with inflammatory markers, MIAC score and vascular access type.

PATIENTS and METHOD

The patients involved in the study were selected among 302 HD patients (182 M, 120 F) who were on hemodialysis (HD) for at least six months. All patients were on regular 4 hours thrice weekly regular HD at Baskent University Alanya Hospital Hemodialysis Unit via arteriovenous fistula or permanent cuffed-tunnelled jugular venous catheter using the same method of cannulation and biocompatible polysulphone high-flux dialysers (Allmed Polypure 1.8-2 m^2, Germany). Dialysate flow rates were 500-800 ml/min and blood flow rates were 250-400 ml/min. The study was approved by the local ethics committee of Baskent University Faculty of Medicine and performed according to the criteria of Declaration of Helsinki (24).

Exclusion Criteria

Patients having active inflammatory and malignant disease or using anti-inflammatory medications were excluded. Patients experiencing problems with fluid-electrolyte balance or who had severe metabolic derangement and uncontrolled blood pressure were also excluded.

Clinical and Demographic Data Collection

Patient demographic data were recorded from hemodialysis data sheets. Body-mass index and dry body weight were determined from the measurements of last three hemodialysis sessions and shown in Table I.

Evaluation of Co-Morbid Conditions

The Davies score was calculated for each patient involved in the study. The Davies score assigns 1 point for each of the following conditions: ischemic heart disease (defined as prior myocardial

infarction, angina, or ischemic changes on EKG), left ventricular dysfunction (defined as clinical evidence of pulmonary edema not due to errors in fluid balance, or history of congestive heart failure), peripheral vascular disease (includes distal aortic, lower extremity, and cerebrovascular disease), malignancy, diabetes, collagen vascular disease, and other significant pathology (*e.g.*, chronic obstructive pulmonary disease) (25). This Score is designed for peritoneal dialysis patients and commonly used in Europe for adjusting co-morbidity. The theoretical range is 0 to 7. Although the Charlson Comorbidity Index (CCI) is the most preferred co-morbidity index in HD patients, it has been shown that the Davies Score (DS) was as good as CCI to predict morbidity and mortality in HD patients (26). In one study, DS was used as co-morbidity index in pre-dialysis and hemodialysis patients (27). DS was used as a co-morbidity index to compare mortality in patients with AVF and CVC in a subdivision of the NECOSAD study (28).

Biochemical Analyses

In all patients, blood samples were drawn at a midweek HD session from the antecubital vein with the standard method. 25-OH (hydroxy) vitamin D levels were studied with the chemiluminescent micro particle immunometric assay (Architect I 10/ Abbott®). Intact PTH was measured by chemiluminescence immunometric assays using Siemens Immulite 2000® (Siemens Healthcare Diagnostics, Deerfield, Illinois, USA). Calcium, phosphorus, albumin and CRP levels were measured by standard laboratory methods using c8000 Architect (Abbott® Laboratories, Park, Illinois, USA). Total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and plasma triglyceride (TG) concentrations were determined using an oxidase-based technique with the Roche/ Hitachi® modular system (Mannheim®/Germany) (40).

Bio Impedance Analysis

Basal metabolic rate, Muscle mass, total fat mass and percentage of total fat (%), truncal fat mass, percentage of fat at upper and lower extremities separately measured by bio impedance analysis method (Tanita® Body Composition Analyser; BC-418/USA) (40).

Table I: Baseline characteristics of study population.

	Minimum	Maximum	Mean	Std. Deviation
Age (years)	19	85	62.1	14.0
BMI (kg/m^2)	23	37	27.3	4.5
Body Fat %	6	47	26.4	10.49
Truncal Fat %	3	44	24.55	11.23
Calcium (mg/dl)	5.7	10.6	8.86	0.79
Phosphorus (mg/dl)	1.4	10.3	4.75	1.62
PTH (pg/ml)	3.0	1338.0	233.99	256.35
Ferritin (mg/dl)	13.6	2000.0	764.23	466.94
Triglyceride (mg/dl)	45.0	475.0	178.77	94.57
Cholesterol (mg/dl)	92.0	353.0	168.28	43.77
Ejection Fraction %	25.00	74.00	59.61	10.3
Pulmonary artery pressure (mm-Hg)	15.00	83.00	26.61	11.18
Hemodialysis Vintage (months)	26	360	50.35	49.48
Albumin (mg/dl)	2.40	4.01	3.94	3.39
CRP (mg/dl)	0.50	64.41	13.94	13.49
Kt/ V	1.3	2.16	1.44	0.22
No of MIAC components	0	3.00	1.55	1.05
T-PAFT (mm^2)	271	3233	1532.53	66.2
25 OH Vitamin D (µg/L)	4.1	37.0	13.62	5.7
Hemoglobin (gr/dl)	8.36	14.9	11.49	1.2

BMI: Body mass index, **PTH:** Parathyroid hormone, **T-PAFT:** Thoracic periaortic fat, **MIAC:** Malnutrition, inflammation, calcification score.

Multislice Computerized Tomography (CT)

Unenhanced computed tomography (Siemens Sensation IV, Enlangen, Germany) was performed in all patients to detect the amount of periaortic fat tissue (PAFT) (18). Adipose tissue quantification was performed using a semi-automated method that required manual definition of borders. The amount of periaortic fat tissue was calculated in each patient at the level of the aortic arch with the method described previously by Horber et al (29). Thoracic PAFT (T-PAFT) was defined as the area immediately surrounding the thoracic aorta anteriorly by a horizontal line through the oesophagus, connected to the left costovertebral joint, posteriorly by the anterior edge of the vertebral body and the right lateral border of the vertebral body (30). T-PAFT showed a positive correlation with inflammation, and MIAC score in HD patients. T-PAFT was a significant predictor of valvular calcification (18). All measurements were repeated by another experienced radiologist to minimize random errors.

Echocardiography

All patients underwent two-dimensional echocardiography (Sonos 4500, HP, Willowick, OH, USA) with a 2.5 MHz multiphase array probe to assess valvular calcification after dialysis at their dry body weight while in the left decubitus position. All echocardiographs were obtained according to the recommendations of the American Society of Echocardiography and were analyzed by a single experienced cardiologist who was blinded to all clinical details (31).

Valvular calcification was defined as bright echoes of >1mm on one or more cusps of the aortic valve, mitral valve, or mitral annulus. Degree of valvular calcification was defined as 0, 1, and 2 according to the number of calcified regions. In all patients, systolic pulmonary artery pressure (sPAP) was determined, and sPAP>35mm-Hg was defined as pulmonary hypertension (PH).

MIAC Components

In the present study, the serum level of albumin and CRP, as well as CVC (score 0: absence of calcification, score 1: calcification in one valve region or annulus, score 2: calcification in two or more cusps and annulus), was used to assess the MIAC score, respectively, just as in studies evaluating cardiovascular mortality and morbidity in ESRD patients (32).

Patients having calcification in at least one valve region (valvular calcification score ≥ 1) were defined as having atherosclerosis/calcification and given 1 point for MIAC score. Patients who had serum albumin level below 3.5 g/dL were defined as having malnutrition and given 1 point for MIAC score. Serum CRP level > 10 mg/L (normal range: 0-5 mg/L) was defined as inflammation and the patient given 1 point for MIAC score. The sum of those three scores was recorded as the MIAC score. The patients therefore had MIAC scores between 0 and 3.

Indices of Dialysis Adequacy

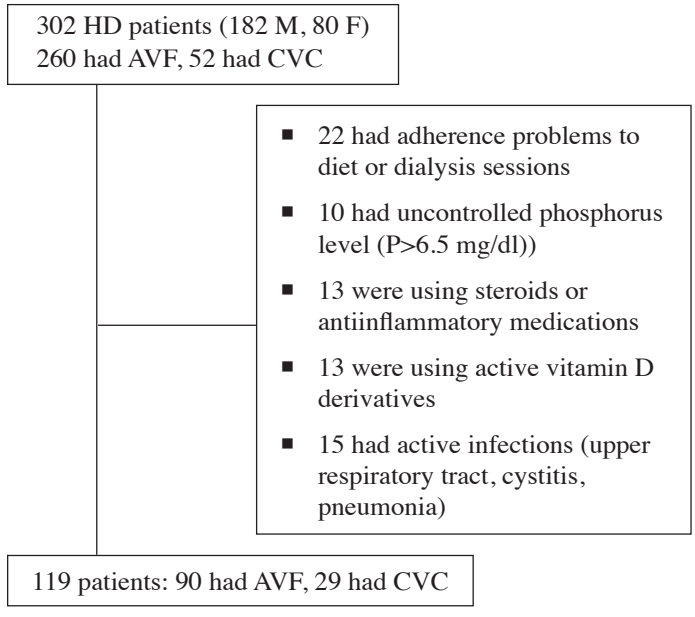
A Dialysis Outcome Quality Initiative-approved method for Kt/V calculation, Daugirdas's formula (1996), was used to evaluate dialysis adequacy. Accordingly, single pool Kt/V (spKt/V) was calculated with the formula "spKt/V= –ln(R–0.008×t)+(4–3.5×R) 0.55×UF/V," in which R was predialysis urea/postdialysis urea, t was dialysis time in hours, –ln was the negative natural logarithm, UF was weight loss in kilograms, and V was the anthropometric urea distribution volume in litres, which may be calculated with Watson's equation or simply estimated as 0.55× postdialysis weight (33,34).

Statistical Analyses

Statistical analyses were performed by using the SPSS 11.0.1 software (April 2002; IBM Corp.;NY;USA). Assumption of normal (Gaussian) distribution was tested by the One Sample Kolmogorov-Simirnov test. Simple correlations were performed Pearson's or Spearman's correlation analyses as appropriate. Comparison of variables between groups created according to vascular access type were performed by Student's t test or Mann-Whitney U tests in accordance with the distribution pattern of the variable. Binary logistic regression analysis was used to determine the independent effects of variables on the vascular access type.

RESULTS

Of the 302 patients 86 patients (28.5%) had diabetes and 216 patients (71.5 %) had no diabetes at the time of the study. Primary kidney diseases were diabetic nephropathy in 80 (26.8%), hypertension in 82 (27.3%), chronic glomerulonephritis in 49 (16.2%), nephrolithiasis in 15 (5.1%), polycystic kidney disease in 15 (5%), unknown in 32 (10.6%), obstructive nephropathy in 20 (6.7%), and amyloidosis in 7 (2.3%).



After the exclusion of inappropriate patients, 119 patients were eligible for the study. Ninety patients (75.6%) had patent arteriovenous fistula (AVF) and 29 patients (24.4%) were undergoing hemodialysis via cuffed-tunnelled permanent jugular catheter. Of the selected patients, 34 had diabetes mellitus (28.6%) while 85 (71.4%) had no diabetes. Patients who underwent HD via CVC had lower dialysis vintage of 36.1±39.8 months compared to patients with AVF (54.7±51.5 months). Six out of 29 patients with CVC had one unsuccessful attempt at AVF creation and after this attempt they refused to undergo further attempts. The rest of the patients (n=23) had at least 2 attempts at AVF creation and all failed due to different causes (narrowing of central veins in 9, inadequate venous diameter in 3, recurrent thromboses in 3 and inadequate AVF maturation with unknown cause in 8 patients). Mean time of CVC in those patients was 22.5 ± 14.6 months.

Baseline demographic characteristics and baseline laboratory results of the study group were summarized in Table I.

Simple Correlations

In all study group (n=119)

The MIAC score was positively correlated with age (r=0.33; p=0.001) and CRP (r=0.639; p=0.0001) and negatively correlated with albumin (r= -0.62; p=0.0001) and 25-OH vitamin D (r= -0.24; p=0.021). 25-OH vitamin D was negatively correlated with CRP (r=-0.22; p=0.015). CRP was negatively correlated with albumin (r=-0.46; p=0.0001). The triglyceride level was positively correlated with the body-mass index (BMI) (r=0.37; p=0.0001) and body fat percentage (%) (r=0.34; p=0.001). Vintage of hemodialysis was positively correlated with calcium (r=0.42;p=0.0001) and ferritin levels (r=0.48; p=0.0001).

In Patients with AVF (n=90)

Age was positively correlated with MIAC (r=0.29;p=0.03). The MIAC score was positively correlated with thoracic PAFT (r=0.62; p=0.001) and negatively correlated with ejection fraction (r=-0.28; p=0.04). The number of calcified cardiac valves was positively correlated with calcium (r=0.26; p=0.04). The body mass index was positively correlated with thoracic PAFT (r=0.48;p=0.001), triglyceride (r=0.308; p=0.02) and CRP (r=0.34; p=0.01). BMI was positively correlated with triglyceride (r=0.498; p=0.02) and phosphate (r=0.466; p=0.03)

In Patients with CVC (n=29)

Age was positively correlated with thoracic PAFT (r=0.89;p=0.02). The MIAC score was positively correlated with ferritin (r=0.49; p=0.013) and negatively correlated with PTH (r=-0.569; p=0.003) and 25-hydroxy vitamin D levels (r=-0.54; p=0.013).

Comparison of Patients with AVF and CVC

The MIAC and Aortic calcification scores were significantly higher in patients with CVC compared to those with AVF (p=0.02 and 0.032) (Figure 1). Thoracic PAFT was higher in patients with AVF compared to CVC (p=0.035) (Figure 1). The CRP level was higher in patients with CVC compared to those with AVF (p=0.04) (Figure 2). Hemodialysis vintage, serum calcium and albumin levels were significantly lower in patients with CVC compared to those with AVF (p=0.01) (Figure 2). Truncal fat (%), serum cholesterol and 25-OH vitamin D levels were lower in patients with CVC compared to AVF (Figure 1,2) (p=0.04; 0.02, 0.03). Detailed comparison of all metabolic parameters and body composition is shown in Table II.

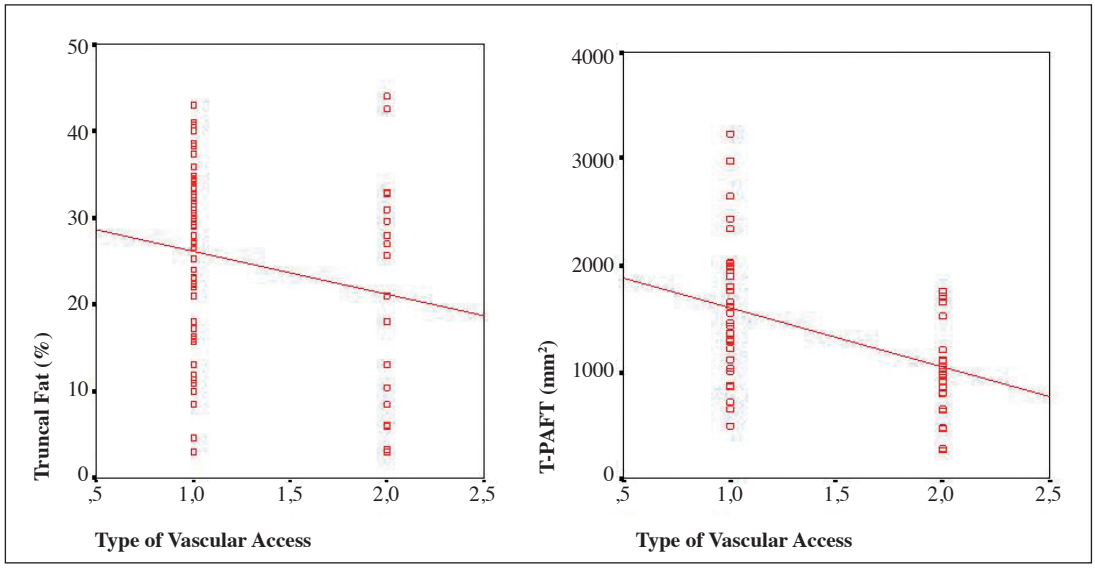


Figure 1: *Comparison of Truncal fat (%) and T-PAFT between vascular access types AVF and CVC.*
Type of the Vascular Access: 1: AVF, 2: CVC
MIAC: *Malnutrition inflammation atherosclerosis calcification score,* ***T-PAFT:*** *Amount of toracic periaortic fat tissue*
AVF: *Arteriovenous fistula,* ***CVC:*** *Central venous catheter.*

Linear regression analysis assessing vascular access type (AVF or CVC) as the dependent variable and the MIAC score and T-PAFT and CRP as independent variables revealed that only T-PAFT was a significant predictor of vascular access type in favour of AVF (t=-2.17; p=0.04). This meant that increased amount of T-PAFT is associated with an increased incidence of having AVF.

Binary logistic regression analysis assessing the vascular access type (AVF or CVC) as the dependent variable and the MIAC score and T-PAFT as the independent variables showed that increased T-PAFT favoured AVF (OR:0.9 ;p=0.03). The aim of the logistic regression analysis was to determine the most important variable in determining the type of vascular access from the independent variables that were different between the AVF and CVC groups (Table III).

Comparison of demographic, metabolic and anthropometric variables between diabetic and non-diabetic patients revealed that the diabetic patients had lower albumin levels, increased MIAC score and thoracic periaortic fat tissue and lower dialysis vintage. Types of vascular access were not different between diabetic and non-diabetic patients (Table IV).

DISCUSSION

The present study revealed that MIAC and Aortic calcification score and CRP levels were higher in hemodialysis (HD) patients with central venous catheter (CVC) compared to patients with arteriovenous fistula (AVF). That indicates inflammation and calcification processes are more prominent in patients with CVC. Previous studies showed that inflammation contributes to the vascular calcification process in patients with CKD (19). Calcification is also an important component of MIAC syndrome and a well-known feature in ESRD. Although

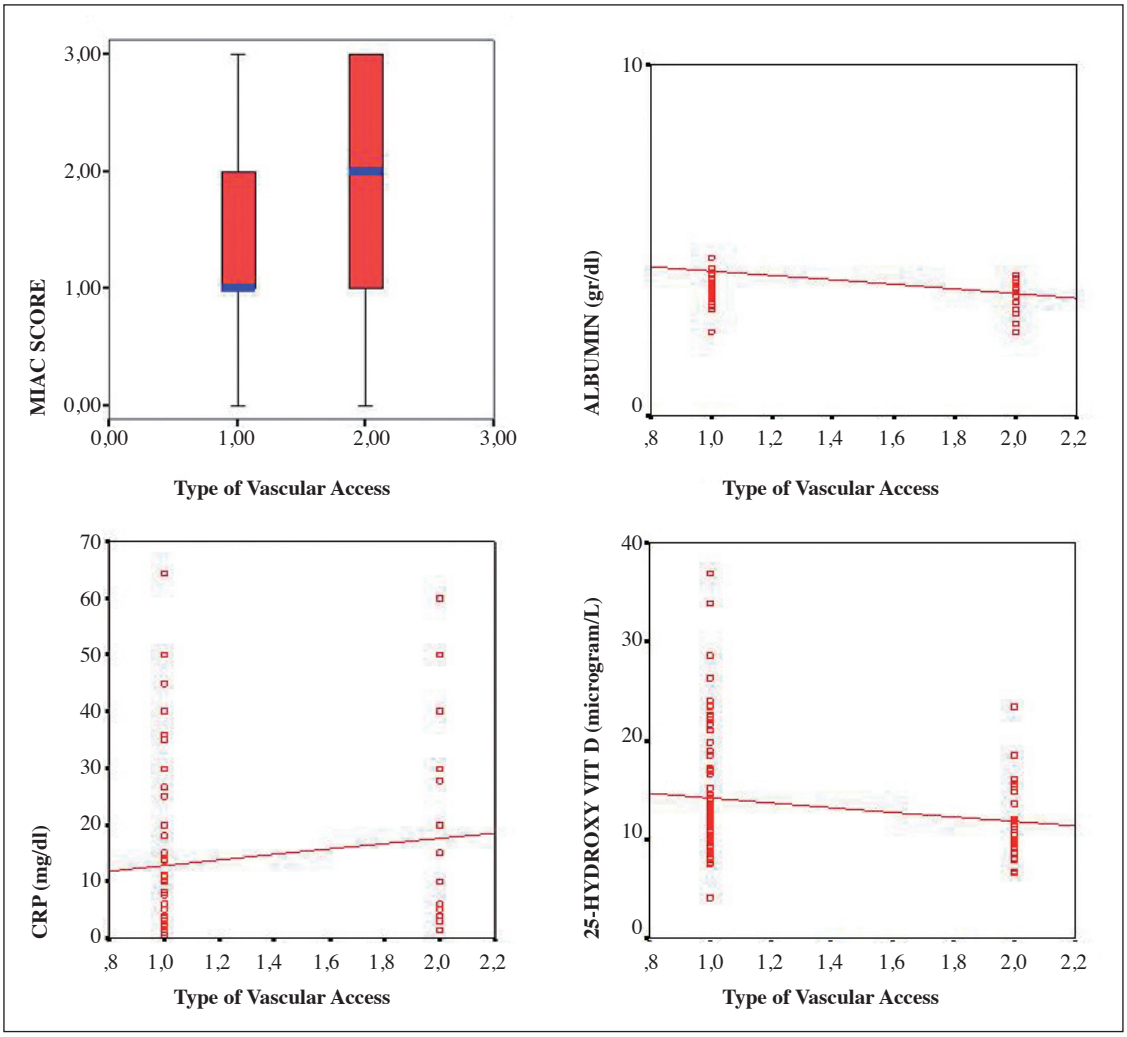


Figure 2: *Relationship between the type of the vascular access and MIAC, CRP, albumin and 25-hydroxy vitamin D levels.*
Type of the Vascular Access: 1: AVF, 2: CVC
MIAC: *Malnutrition inflammation atherosclerosis calcification score,* ***CRP:*** *C-reactive protein.*

an association between inflammation, calcification and vascular access type has previously been demonstrated, an association between vascular access type and bio-impedance analysis (BIA) parameters was first detected in our present study. In the present study, truncal fat (%), serum cholesterol and 25-OH vitamin D levels, hemodialysis vintage, serum calcium, albumin and T-PAFT were lower in patients with CVC compared to AVF. We suggested that having CVC may be associated with increased inflammation, vascular calcification, malnutrition and decreased fat storage. In addition to metabolic and inflammatory determinants, the current study showed that body fat percentage and visceral fat may also be associated with vascular access type in hemodialysis patients. In the present study, patients with CVC had lower lower truncal fat (%) and thoracic periaortic (visceral) fat compared to patients with AVF. As the present study has a cross-sectional design, we cannot be sure whether amount of truncal fat changes is dependent on the duration of CVC usage or not.

Although it was not preferred, CVC is used as the initial vascular access in up to 80% of incident HD patients in some centers (35). The results of the present study revealed that patients undergoing HD via CVC had increased MIAC score, aortic calcification and CRP levels. It has been demonstrated that CVC was associated with increased levels of inflammatory mediators, increased morbidity and mortality in HD patients (36). CRP participates in the complement-mediated removal of damaged vascular cells and may directly be involved in the pathogenesis of neointimal hyperplasia (37). Therefore, having CVC may decrease the chance of creating a functional AVF in the future.

Higher MIAC and aortic calcification scores and lower albumin levels of the patients undergoing HD via CVC may be results of the role of CRP and interleukins in vascular cell damage. We therefore suggest that rather than CVC being a result of inflammation, inflammation could be a result of CVC.

Table II: Comparison of demographic, biochemical parameters, bioimpedance analysis and T-PAFT between patients with AVF and CVC.

	Patients with AVF n= 90	**Patients with CVC n= 29**	**p**
Age (years)	61.7±13.8	63.1±14.9	0.64
Davies Score	1.2±0.8	0.96±0.82	0.12
Aortic Calcification Score	0.8±0.91	2.2±4.05	**0.023**
Number of Calcified Valves on Echocardiography	0.87±0.85	0.96±0.93	0.706
MIAC score	1.4±0.9	1.95±1.1	**0.024**
T-PAFT (mm^2)	1631.5±64	1112.2±60.8	**0.04**
Truncal Fat %	26.4±9.9	20.3±13.1	**0.023**
Body-Mass Index (kg/m^2)	27.5±4.5	26.9±4.6	0.66
25-OH Vitamin D (ng/ml)	14.2±6.1	11.8±3.9	**0.04**
Calcium (mg/dl)	8.9±0.69	8.4±0.9	**0.003**
Phosphorus (mg/dl)	4.7±1.5	4.8±1.9	0.9
Albumin (gr/dl)	4.1±3.9	3.5±0.4	**0.014**
CRP (mg/dl)	12.7±12.7	17.6±15	**0.04**
Hemoglobin (gr/dl)	11.4±1.2	11.5±1.3	0.81
Kt/V	1.42±0.22	1.5±0.21	0.66
Total Cholesterol (mg/dl)	184.9±54	162.7±38	**0.03**
Triglyceride (mg/dl)	182.1±98	168.9±81.9	0.53
Parathyroid Hormone (pg/ml)	222.8±234.3	267.1±315.2	0.822
Dialysis Vintage (months)	54.7±51.5	36.1±39.8	**0.017**
Ejection Fraction (%)	59.9±9.8	58.6±11.6	0.78
Pulmonary Artery Pressure (mm Hg)	26.1±10.3	28.3±13	0.55

Table III: A: Linear regression analysis. B: Logistic regression analysis. revealing the association of T-PAFT with vascular access type. Negative OR: -2.73 means that an increased amount of T-PAFT is associated with 2.73 times increased prevalence of having AVF.

A: Coefficients							
		Unstandardized Coefficients		**Std. Coefficients**	**t**	**Sig.**	
Model		**B**	**Std. Error**	**Beta**			
1	(Constant)	1.463	0.22		6.535	0.00	
	MIAC score	0.190	0.10	0.367	1.811	0.08	
	T-PAFT (mm^2)	-3.983E-04	0.00	-0.507	-2.733	0.01	
	CRP (mg/dl)	3.851E-03	0.01	0.102	0.503	0.61	
a-Dependent Variable: Vascular Access type.							
B: Variables in the Equation							
		B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp (B)**
Step 1	MIAC Score	1.088	.583	3.488	1	.062	2.969
	T-PAFT (mm^2)	-.003	.001	4.886	1	.027	0.997
	Constant	.711	1.408	.255	1	.614	2.035
a-Variable(s) entered in step 1: MIAC score. T-PAFT.							

T-PAFT: Thoracic peri-aortic fat tissue, **MIAC Score:** Malnutrition, inflammation, atherosclerosis score, **CRP:** C-reactive protein.

Table IV: Baseline demographic, metabolic and antrophometric parameters in diabetic and non-diabetic patients.

	Diabetic (n=34)	**Non-Diabetic (n=85)**	**p**
Age	63.5±10.4	61.5±15.6	0.68
CVC/AVF	9/25	20/65	0.37**
BMI (kg/m2)	27.8±4.5	27.1±4.6	0.8
Body Fat (%)	27.3±9.4	26.9±10.5	0.54
Calcium (mg/dl)	8.9±0.7	8.8±0.7	0.87
Phosphorus (mg/dl)	4.4±1.5	4.7±1.6	0.62
PTH (pg/ml)	185.4±183.4	231.3±249.5	0.76
MIAC score	2.0±0.8	1.4±1.1	**0.007***
T-PAFT(mm2)	1860±590	1387±889	**0.04***
CRP (mg/L)	16.1±11	13.4±14.9	0.75
Albumin (gr/dl)	3.5±0.3	4.02±3.8	**0.04***
25-OH vitamin D (μg/L)	12.5±6.5	14.2±5.6	0.56
Kt/V	1.41±0.24	1.46±0.23	0.87
Dialysis Vintage (months)	36.2±29	55.0±55.2	**0.02***
Ejection Fraction (%)	60.1±9.4	59.5±10.9	0.63
Ferritin (ng/ml)	682.3±582	786.1±444	0.56
Triglyceride (mg/dl)	181.6±97.7	178.3±91	0.86
Cholesterol (mg/dl)	169.5±35.2	165.9±46.3	0.76

T-PAFT: Thoracic periaortic fat, **PTH:** Parathyroid hormone, **CVC:** Patients with central venous catheter, **AVF:** Patients with arteriovenous fistula
:** Parameters that have statistical significant difference. *:** p value of chi square test.

Periodic measurements of nutritional parameters, visceral and peripheral fat content, inflammation, MIAC and other calcification scores along with CVC usage are needed to make more specific suggestions.

Primary or secondary failure of AVF compels the use of CVC in some instances. Many factors have been proposed to explain AVF primary failure in HD patients including obesity, DM, female sex, etc. Obesity has been shown to be a risk factor for lower prevalence of functioning AVF (38). However, another study by Chan MR et al. speculated that obesity was not associated with increased AVF revision rates or failure and only poorer AVF maturity at the highest BMI quartile (22). In the present study, although BMI was similar between patients with AVF and CVC, visceral and peripheral fat stores were diminished in patients with CVC. We suggest that this is a result of MIAC syndrome becoming more pronounced by CVC implantation.

We could not detect any relationship between the amount of abdominal or visceral fat and AVF revision rates or failure. Although we could not demonstrate a significant difference in BMI between the AVF and CVC groups, the AVF group had higher percent of body fat and periaortic visceral fat compared to the CVC group. Some studies have revealed that truncal fat mass may contribute to inflammation in end-stage renal disease (ESRD) (23). The results of those studies suggested that truncal fat mass exhibits a distinct effect on chronic inflammation in HD patients. In addition, a positive correlation between T-PAFT and MIAC complex is also identified in ESRD patients (39). In the current study we observed lower CRP levels in patients with AVF compared to CVC despite larger T-PAFT and body fat percentages. It may be speculated that T-PAFT "as a source of inflammation" may lose its volume as a result of excessive function in patients with CVC. Another possibility is a loss of volume due to decrease in body fat mass. Serial measurements of T-PAFT, body fat mass and CRP before and after CVC implantation in time may reveal the exact relationship between T-PAFT, inflammation and malnutrition.

In conclusion, the present data revealed that CVC implantation triggers the components of MIAC in HD patients. Body fat stores may therefore diminish with the use of CVC. Further prospective studies including serial measurements of BIA parameters, T-PAFT or other visceral fat regions of the body may further highlight the exact relationship between vascular access type and changes in the body composition over time.

Conflict of Interest: No conflict of interest is declared by the authors.

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