

Table 2

Variables	Control (n: 64)	1 vessel-plugged (n: 33)	2 vessel-plugged (n: 48)	3 vessel-plugged (n: 20)
TC (mg/dL)	197±23	178±40	190±40	180±44
TG (mg/dL)	119 (114-144)	104 (93-151)	137 (120-164)	113 (69-169)
LDL-C (mg/dL)	124±21	117±34	125±36	117±33
HDL-C (mg/dL)	48 (45-53)*	40 (37-45)a,‡	36 (35-42)	33 (29-36)
VLDL-C (mg/dL)	35 (30-70)	51 (47-58)	48 (40-52)	44 (30-55)
Apo B (mg/dL)	102±21	102±38	105±28	103±31
Apo AI (mg/dL)	153±29	149±26	147±21	138±24
Lp (a) (g/L)	12 (18-37)‡	13 (11-25)‡	25 (23-46)	28 (10-47)
Apo E (mg/dL)	4.5 (4.1-4.7)	4.5 (4.1-5.3)	4.3 (4.2-5.2)	3.4 (2.9-4.8)
LHDL-C (mg/dL)	13 (15-19)	14 (13-17)	12 (12-16)	11 (10-15)
IMHDL-C (mg/dL)	24 (22-25)	22 (20-24)	21 (19-22)	19 (16-20)
SHDL-C (mg/dL)	8.0 (6.9-8.8)*	3.0 (3.0-5.5)	4.0 (3.3-4.8)	4.4 (2.4-4.4)
HDL-LpPL A2 (ng/mL)	118 (75-143)*	36 (27-60)	34 (31-47)	34 (24-67)
PON1 activity (EU/L)	138 (61-191)	63 (40-177)	72 (57-161)	87 (72-141)
hs-CRP (mg/dL)	0.18 (0.21-0.38)	0.19 (0.8-0.79)	0.39 (0.21-1.63)	0.32 (0.18-1.12)

Lipidic profiles of the individuals with and without angiographic findings.

Table 3

	LHDL-C (r, p)	IMHDL-C (r, p)	SHDL-C (r, p)	HDL-Lp PL A2 (r, p)	PON1 (r, p)
LHDL-C	-	NS	NS	NS	NS
IMHDL-C	-	-	0.589, 0.006	NS	NS
SHDL-C	-	-	-	-0.596, 0.006	-0.551, 0.012
HDL-Lp PL A2	-	-	-	-	NS

Spearman rank correlations between HDL subfractions and HDL-associated with enzymes in patients with 3 vessel-plugged

OP-089

The Association Between Peri-aortic Fat and Long-term Incidence of Major Adverse Cardiovascular Events

Zeynettin Kaya¹, Seref Ulucan¹, Hüseyin Katlandur¹, Ahmet Keser¹, Abdullah Tuncez², Yusuf İzzettin Alihanoglu⁴, Duran Efe⁵, Mehmet Kayrak², Mehmet Siddik Ülgen¹

¹Department of Cardiology, Mevlana University, Konya, ²Department of Cardiology, Necmettin Erbakan University, Konya, ³Konya Numune State Hospital, Konya, ⁴Department of Cardiology, Pamukkale University, Denizli, ⁵Department of Radiology, Mevlana University, Konya

Background: Peri-aortic fat tissue is one of the visceral adipose deposits. Visceral adipose tissue is metabolically active and it is suggested that has proatherogenic effects induced by oxidative stress. Previous studies have shown that the relationship between peri-aortic adipose tissue and metabolic risk factors, coronary artery disease, and systemic inflammation. In this study, the association between peri-aortic adipose tissue and long-term incidence of major adverse cardiovascular events (MACE) was investigated.

Methods: 372 men, 61 women, a total of 433 consecutive patients between the ages 40- 75 were enrolled to the retrospective cohort study. Peri-aortic fat volumes were measured by electrocardiogram-gated 64-multi-detector computed tomography. In

terms of the long-term incidence of MACE the three-year follow-up results of patients were evaluated. Patients were divided into two groups (group 1 that MACE was detected and group 2 those followed without any problem) according to results.

Results: MACE (4 death, 22 nonfatal myocardial infarction (7 patients with STEMI and 15 non-STEMI), 4 ischemic stroke, 9 new onset atrial fibrillation, 5 newly diagnosed heart failure development) was detected in 44 (10.2%) patients during follow-up. Demographic and clinical characteristics were similar in both groups. Peri-aortic fat volumes were found statistically significantly high in group 1 (35.4±26.1 vs. 24.1±14.9, p=0.000). A multiple logistic regression analysis showed that peri-aortic fat volume (hazard ratio: 1.03 (95%CI 1.01-1.05), p=0.001), glomerular filtration rate (hazard ratio: 0.98 (95%CI 0.96-0.99), p=0.028), and male gender (hazard ratio: 4.76 (95%CI 1.08-20.90), p=0.039) were independent predictors of development of MACE. ROC analysis demonstrated that peri-aortic fat volumes above 29.6 was predict to development of MACE at sensitivity of 45.45% and at specificity of 76.55% (AUC: 0.61 (95% CI 0.567 to 0.661) p=0.015). In addition, CRP failed to predict MACE.

Conclusion: Peri-aortic fat volume can predict the development of long-term MACE independent of other clinical variables.

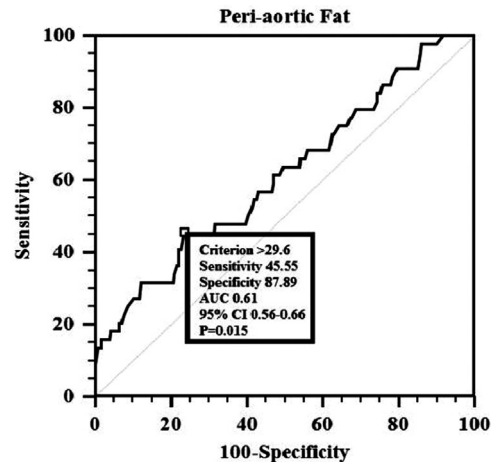


Table 1

		Hazard Ratio	95%CI for Hazard Ratio	P
Step 8	Peri-aortic fat	1.03	1.01-1.05	0.001
	Male gender	4.76	1.08-20.90	0.039
	GFR	0.98	0.96-0.99	0.028

Logistics regression analysis for independent predictors of long term major adverse cardiovascular events (GFR: Glomerular filtration rate) (R2:0.59, P=0.000)

Lipid

OP-090

High-density Lipoprotein Subfractions and Influence of Endothelial Lipase in Healthy Turkish Population: A Study in a Land of Low High-density Lipoprotein Cholesterol

Harun Kılıç¹, Enver Atalar², İncilay Lay², Nuray Yazihan³, Uğur Saygısunar⁴, Fatih Büyükkam⁴, Ramazan Akdemir¹
¹Sakarya University, Sakarya, ²Hacettepe University, Ankara, ³Ankara University, Ankara, ⁴Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara

Purpose: Low high-density lipoprotein (HDL) is prevalent in Turkey. HDL levels in Turkish population are 10-15 mg/dL lower than those of adults in the United States and Western Europe. Endothelial lipase (EL) regulates lipoprotein metabolism, mainly HDL metabolism. Decrease in the lipid content of HDL is thought to increase its capacity to remove cellular cholesterol; small, lipid-poor HDL particles thus represent more-efficient cholesterol acceptors than their large, lipid-rich counterparts. Aim of this study is to investigate HDL subfractions and effect of EL on HDL levels in healthy Turkish population.

Methods: A hundred two healthy subjects included to the study (Mean age 29,1+22 years, 42 female). Subjects who have secondary factors that can affect HDL metabolism excluded. HDL subfractions were assayed by combining a single precipitation method by heparin/Mn/Ds with a direct HDL assay. EL concentrations measured by competitive enzyme immunoassay (EIA) technique.

Results: Mean HDL levels were 56,2+14,4 mg/dL in women, 42,5+11,7mg/dL in men. Small HDL concentrations did not differ statistically between <40 mg/dL, >40 and <60 mg/dL, and >60 mg/dL total HDL groups (Table 1). High HDL levels were