Table 1. Anthropometric and biochemical characteristics of obese and non-obese children

Variables	Obese group (n=70)	Non- obese group (n=40)	P Value*
Age (years)	10.6 ± 2.0	11.3 ± 2.2	0.873
Gender, male	40 (57.2)	23 (57.5)	0.871
Weight (kg)	67 (42 – 91)	36 (22 – 47)	<0.001
Height, cm	135.5 (110 - 170)	135 (115 - 140)	0.966
BMI (kg/m2)	35 (31 – 38.8)	23 (15 – 24.9)	<0.001
BMI - Z score	2.2 (1.75 – 2.7)	0.5 (0.4 – 0.7)	<0.001
Waist circumference (cm)	85.5 (65 – 99)	64 (51 – 81)	<0.001
Systolic BP (mmHg)	120 (90 - 140)	105(90 – 120)	<0.001
Diastolic BP (mmHg)	80 (60 – 90)	70 (50 – 85)	<0.001
Glucose (mg/dL)	95 (65 – 110)	93 (68 – 96)	0.635
HOMA index	3.3 (0.4 – 8.1)	0.8 (0.2 – 4.5)	<0.001
Insulin (mIU/L)	16.5(1.75 – 35)	3.0 (1.9 – 16.7)	<0.001
Total cholesterol (mg/dL)	165 (130 – 240)	162 (110 – 214)	0.362
Triglyceride (mg/dL)	95 (75 – 250)	77 (57 – 250)	0.001
LDL-cholesterol (mg/dL)	103(75 – 190)	90 (55 – 153)	0.001
HDL-cholesterol (mg/dL)	43 (35 – 75)	48 (27 – 65)	0.003
CRP	1.5 (0.3 - 6)	1.6 (0.3 - 5.3)	0.735

Data are presented as mean ± standard deviation, median (minimum – maximum) or n(%). Abbreviations: BMI, body mass index; BP, blood pressure; CRP, C-reactive protein; HDL, high-density lipoprotein; HOMA (Homeostatic model assessment) index = (fasting glucose x fasting insulin concentration x0.0555)/22.5; IU, international unit; LDL, low-density lipoprotein; * by independent sample t-test or Mann-Whitney U-test, as appropriate.

Table 2. Carotid intima-media thickness and flow-mediated dilation of obese and non-obese children

Variables	Obese group (n=70)	Non-obese group (n=40)	p value*
Flow-mediated dilatation (%)	5.75 (5.1 – 19.8)	9.7 (7.8 – 26.7)	<0.001
Right carotid intima-media thickness (mm)	0.59(0.41 – 0.68)	0.45 (0.32 – 0.58)	<0.001
Left carotid intima-media thickness (mm)	0.59 (0.44 – 0.70)	0.44 (0.32 – 0.60)	<0.001
Mean carotid intima-media thickness (mm)	0.60 (0.43 – 0.68)	0.43 (0.33 – 0.59)	<0.001

Data are presented as mean ± standard deviation, median (minimum – maximum) or n (%). * by Mann-Whitney U test

Table 3. Spearman rank correlation analysis of flow mediated dilation (FMD) and carotid intima-media thickness (cIMT), with anthropometric and biochemical variables

	FMD	CIMT
Variable	r p	r p
Age	- 0.273 0.022	0.340 0.004
Obesity duration	- 0.105 0.389	0.076 0.534
Waist circumference	- 0.404 0.001	0.343 0.004
BMI Z-score	- 0.275 0.021	0.241 0.044
Insulin	- 0.400 0.001	0.521 <0.001
HOMA index	- 0.403 0.001	0.515 <0.001
Systolic BP	- 0.353 0.003	0.309 0.009
Diastolic BP	- 0.106 0.420	0.223 0.063
Triglyceride	- 0.370 0.002	0.331 0.005
HDL-Cholesterol	0.371 0.002	-0.304 0.011

Coronary Heart Diseases

Monday, October 28, 2013, 08:30 AM–09:45 AM
Hall: BISHKEK

Abstract nos: 88-93

OP-088

Pleiotropic Effects of HDL Subfraction and Associated Enzymes on Protection Against Coronary Artery Disease

Mehmet Bostan[1], Hüseyin Avni Uydu[2], Zakir Karadağ[3], Mehtap Atak[2], Yavuz Uğurlu[3], Elif Ergül[3], Ömer Şatıroğlu[1], Adnan Yılmaz[2]
[1]Recep Tayyip Erdoğan University, Medical School, Department of Cardiology, [2]Rize University, Faculty of Art and Science, Department of Chemistry, [3]Recep Tayyip Erdoğan University, Educational and Research Hospital, Department of Cardiology

Background: High-density lipoprotein cholesterol (HDL-c) levels are inversely related to risk for developing coronary artery disease (CAD). HDL particles are heterogeneous in size and composition. They may be differentially associated with other cardiovascular risk factors. The aim of the present study was to evaluate independent relationship in HDL subfraction distribution and HDL-associated enzyme activities between CAD severity and normal subjects.
Materials-Methods: Total of 101 patients with stable coronary artery disease, and 64 healthy subjects were enrolled in the study. Serum levels of lipoprotein-associated-phospholipase A2 (Lp-PLA2), Paraoxonase 1 (PON1), HDL subfraction distribution were measured to compare patient and control subjects.
Results: While marked decrease in plasma levels of HDL-C and SHDL subfraction was determined in subjects with at least 1 vessel-plugged compared to control participant ($p<0.001$) total HDL-C level for more severe type of coronary artery disease was lower than those of less severe type ($p<0.05$). Also significant increase in Lp (a) level was observed in patients with 2 and 3 vessel-plugged compared to those of the other groups ($p<0.05$) Plasma HDL-Lp PLA2 enzyme level was higher in the control group compared to each vessel disease category ($p<0.001$). However, no statically significant change was found in PON1 enzyme activity of patients with CAD though had the same trend as HDL-Lp PLA2 enzyme level. Also, statically insignificant chances among plasma hs CRP levels of study subgroups were an intriguing finding. Significant correlations between SHDL fraction and both HDL-associated enzymes were found only in 3 vessel-plugged patients. Plasma level of SHDL subfraction was negatively correlated with both PON1 activity (r:-0.551, $p<0.012$) and HDL-Lp PLA2 enzyme level (r: -0.596, $p<0.065$) in patients with 3 vessel-plugged. Plasma SHDL, HDL-Lp PLA2 enzyme and Lp (a), significantly differed between subjects with CAD and control participant.
Conclusion: We reported significant correlation total and SHDL subfraction between patients and control groups. But negative correlation was found between SHDL subfraction level and PON1 and HDL-Lp PLA2 enzyme level in patients with 3 vessel-plugged.

Table 1

Variables	Control (n: 64)	1 vessel-plugged (n: 33)	2 vessel-plugged (n: 48)	3 vessel-plugged (n: 20)
Age (years), X±SD	54±9*	60±8	63±9	59±7
BMI (kg/m2), X±SD	31 (30-33)	29 (28-31)	28 (27-29)	29 (29-21)
Waist Circumference (cm), X±SD	105±14	102±11	101±12	102±12
Gender, male, % (n)	41 (26)*	52 (17)*	72 (36)	85 (17)
Hypertension, % (n)	25 (16)‡	58 (19)	52 (26)	44 (17)
Dyslipidemia, % (n)	61 (39)	55 (18)	58 (29)	50 (10)
Diabetes, % (n)	18 (12)	16 (5)	26 (13)	35 (7)
Smoking, % (n)	8 (5)‡	15 (5)	23 (11)	35 (7)

Distribution of demographic features in the individuals with and without angiographic findings.

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 Kaya[1], Seref Ulucan[1], Hüseyin Katlandur[1], Ahmet Keser[1], Abdullah Tuncez[3], Yusuf İzzettin Alihanoğlu[4], Duran Efe[5], Mehmet Kayrak[2], Mehmet Sıddık Ülgen[1]

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

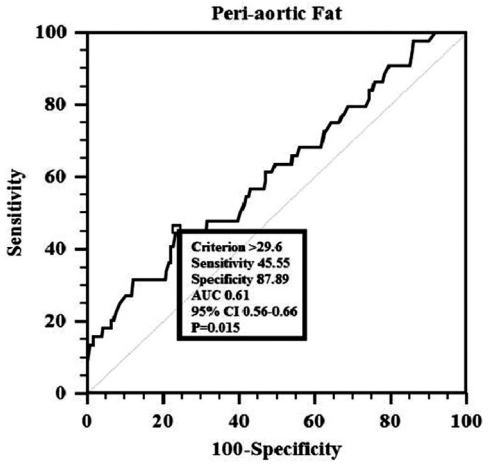

Table 1

		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 Kılıç[1], Enver Atalar[2], İncilay Lay[2], Nuray Yazihan[3], Uğur Saygısunar[4], Fatih Büyükçam[4], Ramazan Akdemir[1]

[1]Sakarya University, Sakarya, [2]Hacettepe University, Ankara, [3]Ankara University, Ankara, [4]Dışkapı 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 Europa. 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