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Dyslipidaemia as a Poteintial Risk Factor for Venous Thromboembolism: A 12 Year Population Based Cohort of Cardiovascular Risk Free Subjects

Arie Steinvil [1], Shlomo Berliner [1], Varda Shalev [2], Dan Justo [1], Itzhak Shapira [1], Ori Rogowski [1]

[1] *Departments of Internal Medicine "D" and "E, Tel-Aviv Sourasky Medical Center,*

[2] *Department of Medical Informatics, Maccabi Healthcare Services, Tel-Aviv, Israel*

Background: Venous thromboembolism (VTE) and cardiovascular disease (CVD) have been implicated as having shared common risk factors. In the past years dyslipidemia has emerged as a potential risk factor for VTE. In order to identify the potential risk of an unfavorable lipid profile on the risk of future VTE (pulmonary embolism and/or deep vein thrombosis), we have analyzed the medical data base of the a large health care maintenance organization. We investigated the ability of the first ever documented lipid profile to predict a VTE or a CVD event among subjects with no history of CVD risk factors or prothrombotic states and compared their risk to a group of matched controls.

Methods: Using the Maccabi database we have analyzed individuals in the age range of 18-55 for men and 18-65 for women, who performed their first ever fasting lipid profile tests, while being free from any prior CVD or VTE events or potential risk factors. We than retrieved data on three reported event groups: the CVD event group, the VTE group and the healthy control group. Multiple exclusions were used in order to isolate the risk attributed to the lipid profile components for a VTE or a CVD event. Using a case-control study design, we compared the mean level of each lipid concentration between the patients and controls. For each VTE and CVD patient we matched 5 randomly chosen controls based on gender, age and number of years of follow-up and compared the mean lipid level for each group using blocked ANOVA.

Results: Included in the present analysis were 337,776 individuals with no prior history of CVD risk or a prothrombotic state for whom a first ever lipid profile was available. Of this cohort 332,036, were defined as healthy controls, 3,421 entered the CVD event group, and 2,319 entered the VTE event group. The respective female ratio was of 66, 30 and 73. The respective mean age for males was 40, 47 and 43, and 41, 50 and 43 for females. We found statistically significant differences in the levels of all lipids evaluated for women, and significant differences in the levels of HDL-C and triglycerides for men, with unfavorable levels in the VTE group. The absolute differences were minor for LDL and total cholesterol, and more significant for HDL, with a very siginificat difference regarding the triglyceride levels.

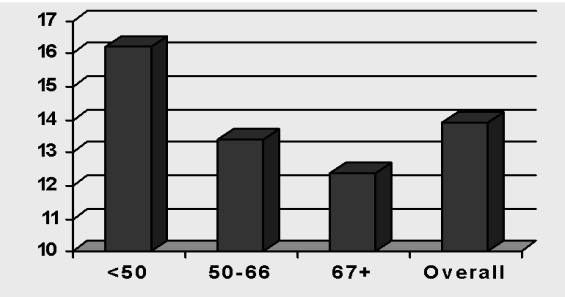
Conclusion: Higher levels of LDL cholesterol, total cholesterol and triglycerides and lower levels of HDL cholesterol were found in a group of individuals that developed VTE event with no prior VTE or CVD event as compared to matched healthy controls.

Predictive Value of Pulse Pressure in Patients with Acute Coronary Syndrome

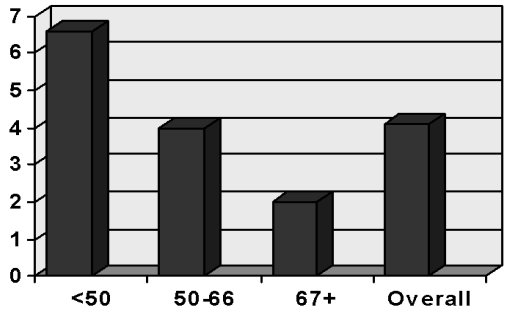
Donna Zwas [1], Michal Benderly [2], Yair Elizur [1], Radostina Ilieva [1], Israel Gotsman [1], Aharon Alcalai [1], Andrei Keren [1], Chaim Lotan [1]

[1] Cardiology Department, Heart Institute, Hadassah-Hebrew University Medical Center, Jerusalem, [2] Cardiology Department, Neufeld Cardiac Research Institute, Chaim Sheba Medical Center, Ramat gan, Israel

Background: High pulse pressure (PP) has been shown to be an independent predictor of cardiac events in patients with and without coronary artery disease. We sought to explore the predictive value of PP in patients in the setting of acute coronary syndrome (ACS).
Methods: Data was collected from the 2008 ACSIS (Acute Coronary Syndrome Israeli Survey). Systolic and diastolic blood pressures were recorded at the time of presentation to medical personnel. Patients who were in cardiogenic shock (CS) were excluded. PP was divided into tertiles for analysis (low =<50mmHg, mid 50-67mmHg, high ≥68mmHg).
Results: PP was available for 1760 of 1766 patients enrolled. 40 patients were excluded due to CS. Mean blood pressure was 143/83 with pulse pressure of 60mmHg. Mortality and MACE were highest in the low pulse pressure group (5.2% and 14.9% respectively) compared with 3.6% and 13.2% in the midPP group and 1.9% and 12.3% in the high PP group. (p = .01) Patients in the mid and higher PP group were older and had a higher incidence of hypertension, diabetes, and hyperlipidemia, and a higher incidence of cardiac, neurological and peripheral vascular disease. Patients in the lower PP group had a higher incidence of STEMI and were more likely to have moderately or severely decreased ejection fraction. On multivariate analysis including age, gender, smoking, and diabetes, high PP remained a strong predictor of survival (OR 0.6 95% CI 0.4-0.84, p<.004). In multivariate analysis of patients with STEMI, PP remained a predictor of survival, even after inclusion of systolic blood pressure as a variable (OR 0.3, 95% CI 0.1-0.7, p=.01)
Conclusions: Higher PP in the setting of ACS is a predictor of decreased MACE and increased survival. In the acute setting, high PP may be a marker of increased cardiac output and increased cardiovascular reserve.



30 Day MACE



30 day mortality

Prognostic Value of Serum Uric Acid for Risk Stratification of Patients with Coronary Artery Disease

Yafim Brodov, Pierre Chouraqui, Ilan Goldenberg, Valentina Boyko, Lori Mandelzweig, Shlomo Behar

Heart Institute and Neufeld Cardiac Research Institute, Sheba Medical Center, Ramat Gan

Background: The relationship between serum uric acid (SUA) levels and coronary artery disease (CAD) are conflicting. We hypothesized that a combined assessment of SUA levels and renal function could provide incremental prognostic information in patients with CAD.
Objectives: We assessed the relationship between elevated SUA, renal function and prognosis among 3107 CAD patients enrolled in the Bezafibrate Infarction Prevention (BIP) study.
Methods: The risk of all-cause mortality and the primary end point (PEP), including fatal or nonfatal myocardial infarction or sudden death, was assessed by SUA quintiles, and combined SUA and estimated glomerular filtration rate (eGFR) categories.
Results: All-cause mortality and PEP rates rose significantly with increasing SUA quintiles (8.5-12.9%, $p<0.002$; 5.3-7.9%, $p<0.05$; and 12.6-18.6%, $p<0.007$) measured from lowest to highest quintile, respectively. After multivariate analysis, patients in the highest SUA quintile ($\geq$6.73 mg/dl) exhibited increased risk for PEP (HR: 1.38 [95% CI: 1.00-1.90]), but not for all-cause mortality (HR: 1.27 [95% CI: 0.87-1.88]). Patients in the highest SUA quintile and renal dysfunction (eGFR < 60 ml/min/1.73 m^2) exhibited the highest risk of all-cause mortality and PEP [HR:1.6 (95% CI: 1.10-2.33) and HR: 1.53 (95% CI :1.09-2.16), respectively].
Conclusion: In patients with CAD, elevated SUA levels are associated with an increased risk of PEP. The combined assessment of SUA and renal function provides incremental prognostic data and identifies a high-risk subgroup for all-cause mortality beyond assessment using a single marker.

Statin Therapy and Coronary Plaque Characteristics on 64 Slice Cardiac CT in Asymptomatic Type 2 Diabetics

David Halon [1], Tamar Gaspar [2], Idit Dobrecky-Mery [1,2], Mali Azencot [1], Nisan Yaniv [3], Nathan Peled [2], Basil Lewis [1]

[1] Cardiovascular Medicine, Lady Davis Carmel Medical Center, [2] Radiology Department, Lady Davis Carmel Medical Center, [3] Cardiology Department, Clalit Community Health Services, Haifa, Israel

Background: Statins decrease coronary heart disease related adverse events but their effect on coronary plaque characteristics is unclear. Calcification may stabilize coronary plaques and be associated with fewer adverse events and better clinical outcome. We prospectively examined 64 slice coronary CT angiograms (CTA) in an asymptomatic patient cohort with type 2 diabetes mellitus to examine differences in coronary plaque characteristics between pts receiving or not receiving statin treatment.

Patients and Methods: Type 2 diabetics, 55-74 yrs, with no history of clinical coronary disease underwent CTA in the confines of a prospective, ongoing, outcomes study. Coronary plaque was assessed visually as absent, calcified (≥50% calcium), non-calcified (no calcium) or mixed (<50% calcium) using a 17 segment coronary arterial model and total length of each plaque type was measured.

Results: In 120 pts (age 63.7 yr, 44 (36.7%) men) coronary plaque was present in 40 (90.9%) men and 65 (85.5%) women (ns) but was more extensive in men (total plaque length 56±56 vs 30±31 mm, p=0.02). At study entry 86 (72%) pts were taking statins. Total plaque length was similar for pts with and without statins (40±44 vs 38±33 mm (ns) and 40% of plaques were calcified. In pts taking statins the total length of calcific plaque was greater and the total length of non-calcific/mixed plaque was decreased (Table).

Plaque characteristics in relation to statin therapy

	No Statin	Statin	p-value
Calcified plaque (mm) (% of total plaque length)	8.9±17.1 (23.5)	20.9±31.0 (51.7)	0.01
Non-calcified/mixed plaque (mm) (% of total plaque)	28.9±38.3 (76.5)	19.5±23.4 (48.3)	
Total plaque (mm) (m±1SD)	37.9±43.3	40.4±44.1	0.77

Conclusions: In asymptomatic type 2 diabetics undergoing 64 slice cardiac CTA: 1. Overall extent of coronary plaque was similar in pts taking or not taking statins. 2. In patients receiving statins the proportion of calcified coronary plaque was greater and there was a lesser extent of non-calcific/mixed plaque 3. Differences in plaque composition may relate to the beneficial effect of statins in coronary patients.

Visceral Fat Predicts Coronary Atherosclerosis Independently from the Metabolic Syndrome in Asymptomatic Diabetic Patients – a 64 Slice CT Study

Alla Khashper [1], Tamar Gaspar [1], Idit Dobrecky-Mery [2], Mali Azencot [2], Nathan Peled [1], David A Halon [2]

[1] *Radiology,* [2] *Cardiovascular Medicine, Carmel Medical Center, Haifa, Israel*

Background: Non-alcoholic fatty liver disease (NAFLD) may accompany the metabolic syndrome, is common in diabetics and may be a marker of pre-clinical atherosclerosis. Recent studies suggest that visceral adiposity (accumulation of fat in the intra-abdominal cavity) may predict adverse coronary events. We examined relation of abdominal fat distribution and NAFLD to presence of coronary atheroma on 64 slice coronary CT angiography (CTA) in asymptomatic subjects with diabetes mellitus (DM) enrolled, in an ongoing prospective outcomes study.

Methods: Non-enhanced chest CT was performed in 318 pts to determine abdominal fat distribution and cardiac CTA (Philips, Brilliance 64 scanner) to determine coronary atheroma. Fatty liver was diagnosed when liver density was 10 HU or more below spleen density. Increased internal abdominal diameter was taken to represent visceral abdominal fat accumulation. Metabolic syndrome was diagnosed from clinical characteristics according to NCEP III criteria. Patients with alcohol abuse were excluded.

Results: Extent of pre-hepatic fat pad and internal abdominal diameter were increased in pts with multi-vessel coronary plaque (MVCP) (fat pad 15.8±6.7 vs 13.5±5.6 mm, $p=0.005$; abdominal diameter 158.4±33.6 vs 135.8±27.7 mm, $p<0.001$). Fatty liver was common (29.2% pts) and its presence correlated with prevalence of several adverse risk factors [BMI ($p<0.001$), waist circumference ($p<0.001$), lower HDL cholesterol ($p=0.005$) and higher triglycerides ($p=0.001$)], but did not correlate with presence of coronary plaque or MVCP. Metabolic syndrome was present in 266 (83.6%) pts and predicted more MVCP [162 (61.1%) pts with vs 21 (40.4%) pts without metabolic syndrome, $p=0.006$]. Prehepatic fat ($p=0.008$) and internal abdominal diameter ($p<0.001$) predicted MVCP independently from the presence of metabolic syndrome.

Conclusions: In asymptomatic subjects with DM and no history of CAD: 1) Visceral fat as evidenced by pre-hepatic fat pad and internal abdominal diameter predicted presence of multi-vessel coronary plaque independently from the presence of the metabolic syndrome. 2. Fatty liver did not predict presence of coronary atheroma. 3. These data support recent suggestions that visceral fat may play a role in the pathogenesis of coronary heart disease.

Mycoplasmal Infections in Patients with Coronary Heart Disease

Leonid Barski [1], Roman Nevzorov [1], Jacob Horowitz [2], Shulamith Horowitz [3]

[1] Medicine F, Soroka University Medical Center, [2] Medicine A, Soroka University Medical Center, [3] Mycoplasma laboratory, Faculty of Health Sciences, Ben-Gurion University, Beer-Sheva, Israel

Background: Clinical and epidemiological features of coronary heart disease (CHD) may not be explained only by established risk factors. The role of infectious pathogen in the development and rupture of atherosclerotic plug remains elusive but some associations among Chlamydia Pneumoniae , Mycoplasma pneumoniae and CHD have been reported. We investigate the relationship of Mycoplasmal infections and CHD.

Methods: We performed prospective cohort analysis of 150 consecutive patients with CHD and 98 healthy blood donors. Three groups were compared: a group of patients with acute coronary syndrome (included 85 patients), a group of patients with known CHD who admitted due to unrelated to CHD reasons (65 patients) and a group of healthy blood donors (98 patients). We performed analysis of the antibodies titers for M.pneumoniae, M.fermentans , M.hominis and Ureaplasma urealyicum measured by the agglutination test and specific enzyme-linked immunosorbent assay (ELISA) in all three groups.

Results: Analysis of the antibodies titers for these infectious agents did not reveal any significant difference in the presence of the Mycoplasma antibodies among the patients with acute coronary syndrome, patients with known CHD hospitalized due to unrelated to CHD reasons and healthy blood donors.

Conclusions: In our limited series we did not found any significant association among different types of Mycoplasmal infections and CHD.

Predictors of Plaque Destabilization in 'Vulnerable Patients'

Diego Medvedofsky [1], Shmuel Schwartzenberg [2], Gad Keren [2], Jacob George [2]

[1] *Internal Medicine, B, Meir General Hospital, Kfar Saba, Israel,* [2] *Cardiology, Cardiac Catheterization, Tel Aviv Sourasky Medical Center, Tel Aviv, Israel*

Background: It is not yet understood why some patients with prexisting coronary atherosclerosis develop recurrent cardiac events ('vulnerable patients') whereas others, with a similar atheromatous burden remain asymptomatic.

Objective: We sought to determine if any laboratory tests might be related to a negative history of myocardial infarction in patients with angiographically significant coronary artery disease.

Methods and results: We studied a total of 109 patients aged 41-88 years without renal failure and severe heart failure who were catheterized in the last 2 years in our institution and were found to have 2 or 3 vessel coronary artery disease. Out of this population, 37 patients with a history of more than one event of myocardial infarction were matched with 37 patients without evidence of myocardial infarction with regard to the number of coronary arteries involved, age, sex and major cardiovascular risk factors. After the matching process, of the various parameters recorded, we found there were statistically significant lower hemoglobin levels in the non-ischemic versus the ischemic patients (13.0 gm/dl versus 13.8 gm/dl, p=0.042). Other tested parameters including platelets, white blood cells and its differential, were not predictive of coronary events.

Conclusion. Hemoglobin levels were signficatly elevated in 'vulnerable patients' as compared with asymptomatic patients with similar coronary atherosclerotic burden. Higher hemoglobin levels superimposed on a preexisting coronary artery disease could have contribued to the coronary plaque destabilization.

Familial Correlation in Electrocardiographic Criteria for LVH in Israeli Families from The Kibbutz Settlements Family Study

David Planer [1], Nir Sharon [2], Atila Bulut [1], Chaim Lotan [1], Yechiel Friedlander [2]

[1] *Heart Institute,* [2] *School of Public Health, Hadassah – Hebrew University Medical Center, Jerusalem, Israel*

Background: Electrocardiographic voltage criteria of Left ventricular hypertrophy (LVH) are known risk factor for adverse cardiac events. The genetic factors determining electrocardiographic criteria of LVH were studied in an unselected population sample of 80 families residing in kibbutz settlements in Israel.

Methods: A total of 465 healthy individuals aged 15-97 from kibbutz settlements in the north of Israel were requited to the study in 1992-1993. Anthropometric measurements, detailed medical questionnaire, 12 leads ECG and blood samples were taken from all participants. Measurements of QRS complex parameters were completed manually in all ECGs. Sokolow-Lyon index, Cornell voltage criteria and HLVM (electrocardiographic estimation of LV mass) were calculated. The degree of resemblance among family members was expressed by adjusted and unadjusted inter- and intraclass correlation coefficients for LVH criteria.

Results: A total of 394 ECGs were analyzed. The Mean Sokolow-Lyon index was 18.1+/-6mm, Cornell voltage criteria 13.5+/-5mm and HLVM 116.4+/-15gr. There was a significant familial aggregation of HLVM as indicated by unadjusted and adjusted inter- and intraclass correlation coefficients significantly different from zero (table1) – correlation were significant only between siblings and parent-offspring pairs, while no correlation was found in spouse-spouse.

Correlation in Sokollow-Lyon index was found only among siblings; correlation in adjusted Cornell voltage criteria was significant only in parent-offspring pairs.

Conclusions: Our preliminary results provide evidence for significant family aggregation at least for HLVM. Future complex segregation analysis will determine the pattern of genetic inheritance of electrocardiographic LVH parameters and might enable mapping of the responsible genes for LVH.

Table 1: Familial correlations for electrocardiographic LVH parameters among family members in kibbutz settlements

		Parent-offspring			**Sibling-sibling**			**Spouse-spouse**		
		n	**R (SE)**	**p**	**n**	**R (SE)**	**p**	**n**	**R (SE)**	**p**
HLVM	**1**	294	0.19(0.08)	**0.02**	123	0.41(0.1)	**0.0001**	105	0.168(0.1)	0.1
	2	284	0.34(0.07)	**<0.0001**	123	0.39(0.1)	**0.0006**	103	0.08(0.1)	0.47
Sokolow-Lyon	**1**	295	0.02(0.07)	0.8	123	0.21(0.1)	**0.04**	106	-0.02(0.1)	0.9
	2	284	0.04(0.07)	0.57	123	0.27(0.11)	**0.01**	103	-0.13(0.1)	0.2
Cornell Voltage Criteria	**1**	295	0.12(0.06)	0.07	123	0.1(0.1)	0.29	106	-0.05(0.1)	0.6
	2	284	0.14(0.06)	**0.02**	123	0.13(0.1)	0.21	103	-0.07(0.1)	0.5

1- Unadjusted familial correlation

2- Adjusted familial correlation to sex, age, BMI, BMI^2, blood pressure