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**2.P.174 Apolipoproteins and lipoprotein particles level in Moroccan patients with previous myocardial infarction**

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To investigate the role of lipoprotein particles in the relationship with the progression of coronary atherosclerosis, we studied a Moroccan patients with previous myocardial infarction as the primary manifestation of the coronary artery disease presentation. These patients suffered a verified myocardial infarction before the age of 50 years and none of them have suffered re-infarction within the succeeding 6 months. Plasma lipid variables including total cholesterol (total-cho), triglycerides, high density lipoprotein cholesterol (HDL-cho), low density lipoprotein cholesterol (LDL-cho), apolipoprotein AI (apo AI), apo B, apo AI-containing particles without apoA II (Lp AI) and with apo AII (Lp AI:AI), and Lp (a) were examined. Plasma lipids were analysed by the usual enzymatic methods. Apolipoproteins and lipoprotein particles were measured by ELISA and EIA. The results were compared to healthy control group. The mean plasma total-cho, triglycerides, and Lp AI:AI levels of patients did not differ significantly from that of control subjects. Patients had lower plasma of HDL-cho ( $P < 0.05$ ), apo AI ( $P < 0.05$ ), and Lp AI ( $P < 0.001$ ) than control subjects. However, patients had higher plasma of LDL-cho ( $P < 0.001$ ) and Lp (a) ( $P < 0.001$ ). Among patients, smokers had a higher risk of developing coronary artery disease, followed by those with high levels of Lp (a) and those with low levels of Lp AI. In all patients the best predictor of cardiovascular risk was the Lp AI plasma level and the independent risk factor Lp (a) plasma level, suggesting that the cholesterol reverse transport system is altered in patients with previous myocardial infarction.

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**2.P.175 Carotid atherosclerosis and vascular risk factors in patients attending an Italian Lipid Clinic**

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Intima media thickening (IMT) of carotid artery is a measurable marker of atherosclerotic risk factors and it may be considered as a cardiovascular risk factor itself. Here we present the results of an observational study performed in 963 patients attending the Lipid Clinic of the Enrica Grossi Paoletti Center in Milan. The aim of the study was to assess the prevalence of carotid atherosclerotic lesion in patients with and without major cardiovascular events and to evaluate its association with the presence and numerosity of principal atherosclerotic risk factors. Maximal- and Mean-Maximal IMT were assessed using B-Mode ultrasonography and measured with an electronic caliper. IMTs were significantly higher in men vs women and were directly related to age, SBP, TC, LDL-C, TG, blood glucose and uric acid and inversely with HDL-C. Among risk factors, IMT was the best discriminant between patients with and without coronary heart disease, peripheral artery disease, but not cerebrovascular disease. The IMT was also linearly related to the number of vascular risk factor, either in the whole group, or after stratification into 3 different age classes. These observations establish the highly predictive validity of the B-Mode imaging of carotid atherosclerosis in the screening of individuals at high risk of vascular disease.

**2.P.176 Management of low-density lipoprotein cholesterol for secondary prevention of cardiac event after myocardial infarction**

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Prospective studies suggest that hypercholestelema is an very important risk factor for ischemic heart disease. To clarify the management of low-density lipoprotein cholesterol (LDL) levels for secondary prevention of cardiac event (re-infarction) after myocardial infarction, we measured LDL cholesterol levels of 254 patients after myocardial infarction with thrombolytic therapy. Patients were divided into three groups following LDL levels ( $<100$  mg/ml,

100–140 mg/ml, 140 mg/ml $<$ ). Each group were compared as cardiac event rate (Breslow-Gehan-Wilcoxon test) for 10 years.

LDL levels	$<100$ mg/ml,	100–140 mg/ml,	140 mg/ml $<$ ,
After 1 y	0%	0%	0%
After 3 ys	2%	2%	11%
After 5 ys	5%	5%	14%
After 8 ys	5%	15%	20%
After 10 ys	5%*	22%	20%

\*:  $p = 0.03$

This result showed that LDL levels should be keeping under 100 mg/ml to prevent for cardiac event. We concluded that lower control of LDL cholesterol levels was needed for secondary prevention after myocardial infarction.

**2.P.177 High level homocysteine, LDL modification and CHD**

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Patients with high levels of circulating homocysteine are at high risk of developing premature atherosclerosis.

Many reports have suggested that even a moderate increase of homocysteine concentration is associated with an increased risk of vascular disease.

The mechanism involved is unclear; in vitro homocysteine exhibits oxidate activity; the hyperhomocysteinemic animals (pigs) exhibit in vivo lipid peroxidation.

The reported patient, with premature C.H.D. treated by percutaneous transluminal coronary angioplasty exhibit high homocysteine levels with peroxidative modification of LDL as reported in Table 1.

T. Ch.	(mg/dl)	215
LDL. Ch.	(mg/dl)	124
HDL. Ch.	(mg/dl)	31
Triacylglic.	(mg/dl)	297
Homocys. v.n.: $\leq 7.5$	( $\mu$ M)	16.5
MDA v.n. $\leq 7.5$	( $\mu$ M)	10.7
Dienes v.n. $\leq 80 \mu$ M	( $\mu$ M)	156
Lp(a) v.n. $\leq 30$ mg/dl	(mg/dl)	103

High homocysteine levels can modifie LDL and HDL by oxidative mechanism causing

Foam cells formation, endothelial dysfunction and in succession arteriosclerosis plaque, in the patient premature CHD high Lp(a) concentration can be also considered

**2.P.178 Smoking and transvascular leakage of albumin: Preliminary findings**

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Cigarette smoking is a major risk factor for the development of atherosclerosis. Smoking might act through endothelial damage. To evaluate microvascular dysfunction, the Transcapillary Escape Rate of Albumin (TERalb), a measure of the unidirectional flux of plasma proteins out of the vascular bed, has been measured in eighteen non-diabetic healthy subjects (14 males, 4 females). No subject had cardiovascular or renal disease, all of them were normotensive ( $<140/90$  mmHg by ambulatory blood pressure monitoring) and normoalbuminuric (AER range:  $2.1\text{--}4 \mu\text{g/min}$ ). Seven subjects were current smokers, three were ex-smokers and eight had never smoked. The three groups were similar for age, sBP and dBP, BMI, serum creatinine, total-, LDL- and HDL-cholesterol, Apo-A1, Apo-B and fibrinogen levels. TERalb, calculated from the slope of the linear regression of the log-transformed radioactivity decay of the bolus injected  $^{125}\text{I}$  human albumin, resulted higher in both current smokers ( $7.2 \pm 1.4\%/h$ ,  $M \pm SD$ ) and ex-smokers ( $7.14 \pm 1.5\%/h$ ), than in subjects who had never smoked ( $5.1 \pm 1.1\%/h$ , log-transformed TERalb,  $p = 0.01$ ). Since, serum albumin, haematocrit, plasma volume were similar in all groups, outflux of albumin, calculated as TERalb  $\times$  IVMA (intravascular mass of albumin) was consistently higher in current and ex-smokers than in non-smokers ( $9.6 \pm 2.9$  and  $8.9 \pm 3.7$  vs  $5.1 \pm 1.2 \text{ g/h} \times 1.73 \text{ m}^2$ ,  $p = 0.02$ ). Our preliminary results suggest that cigarette smoking is associated with abnormal microvascular function in otherwise asympomatic subjects. Microvascular bed of subjects who have given up smoking, keep memory of the smoke-induced damage.