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Topic 04 – Valvular heart disease and general cardiology

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High levels of circulating lipoprotein-associated phospholipase A2, but not oxidized LDL, are associated with severe aortic stenosis; comparison with coronary artery disease

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Purpose: Aortic stenosis (AS) is a disease process akin to atherosclerosis and has been linked to several risk factors for coronary artery disease (CAD). Lipoprotein-associated phospholipase A2 (Lp-PLA2) is an inflammatory biomarker secreted in the atherosclerotic plaque. We hypothesized a relationship between serum Lp-PLA2, plasma oxidized LDL (oxLDL) and AS, in contrast with CAD.

Methods: Between December 2009 and June 2010, from 494 subjects scheduled for diagnostic coronary angiography, we selected 234 patients (71.2% men) included into 2 groups: Group "CAD": patients with only CAD and Group "AS": patients with AS and normal coronary arteries. Lp-PLA2 mass was assessed in serum with a Plac® – test turbidimetric immunoassay. Control Lp-PLA2 values were specifically obtained in 61 subjects aged 44.5±17.6y without cardiovascular risk factors or cardiac treatment.

Results: In healthy controls, Lp-PLA2 was 163±43 µg/L (men: 166±45 µg/L; women: 159±39 µg/L, non significant). Lp-PLA2 were significantly higher in patients than in controls (219.2±49.1 vs 163.0±43.0; p=0.01). Lp-PLA2 increased in AS-patients, and were not significantly different than CAD-patients (217.3±44.5 vs. 220.4±51.9, respectively; NS). Moreover, Lp-PLA2 levels were similar in patients with single-vessel atherosclerosis (215.2±52.0) but significantly lower than in patients with three-vessel atherosclerosis (251.9±5.7 µg/L; p=0.03).

OxLDL levels were 42±13 U/L, which was within normal values. oxLDL values were 46±12 and 38±12 U/L for AS-patients and CAD-patients, respectively, (p<0.0001). Circulating oxLDL levels were not affected by gender, diabetes or hypertension but were significantly correlated with serum apoB, LDL-cholesterol, triglycerides and ApoA-I. We found no correlation between Lp-PLA2 and oxLDL. Patients on statin treatment had significantly lower plasma oxLDL levels than patients without any hypolipidemic treatment.

Conclusion: We report for the first time that high serum levels of Lp-PLA2 were associated with severe AS. This could have practical implications because statins and renin-angiotensin-aldosteron system inhibitors seemed to be a promising treatments of AS.

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Do you need to check systematically the INR during the RAMADAN's fasting period?

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Introduction: During Ramadan, Muslims fast during the daylight hours for a month. A pharmacokinetic variability of several medications can occurred in this period. However, the effect of Ramadan fasting on INR (International normalized ratio) is unknown.

Aim of this study: was to evaluate the fasting Ramadan effect on the variability of the INR in patients treated at long term with oral anticoagulant.

Methods: 67 fasting patients (aged 60±11.4 years) treated at long term by acenocoumarol in the evening who fasted during Ramadan participated in this open, prospective and single center study. Venous blood samples were taken 1 week before Ramadan (INR1), on the 15st (INR2) and 29th days (INR3) of Ramadan. Exclusion criteria was an INR1<1.7 or INR1>4.5 before Ramadan and patient taking twice daily dose.

Results: Compared before Ramadan, the mean INR2 was significantly increased during the 15th days of Ramadan, 4.1±1.7 vs 2.9±0.8, respectively (p<0.001). A Vitamin K antagonist overdose (INR2>5) was obtained in 19 patients (28.4%). The mean variation of INR2 during this first period was +46.5% (–35%, +256.4%). We excluded for the third measure all patients with an INR2>5. The mean INR3 (n=42 pts) was significantly higher than the INR2, 3.4±0.88 vs 2.9±0.6 (p<0.0001). Compared to INR2, the mean variation of INR3 during this second period was +16.1% (–29.6%, +114.4%). Finally during Ramadan a VKA overdose was noted in 31.3% of cases. By multivariate analysis we found two independent predictors factors linked to a high INR during Ramadan: an initial INR>3 before Ramadan (OR=3.57; p=0.037) and a dose of acenocoumarol ≤4 mg (OR=3.5; p=0.041).

Conclusions: At our knowledge this is the first study demonstrate that intermittent fasting led to increase significantly the level of anticoagulation during this holy period. After this pilot study, we recommend checking systematically the INR before and during this period in order to reduce the INR around 2.

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Impact of chronic obstructive pulmonary disease on long-term prognosis in coronary artery disease

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Purpose: We aimed to assess the long term prognosis associated with COPD in a contemporary large cohort of pts with established CAD.

Methods: 783 consecutive male pts hospitalized in 2001-2004 for coronary artery disease were considered. The median follow-up was 7.17 years. Total mortality was predicted with a Cox proportional hazard model.

Results: Mean age (SD) was 60.2 (8.1), 144 pts (18.4%) were diabetic, mean glycaemia was 5.9 mmol/l (2.1), 155 pts (19.8%) were smokers, mean blood pressure was 139 (20)/84 (11) mmHg and median heart rate was 61 bpm [Interquatile range (IQR)] [57-70]. Mean HDL cholesterol was 43 mg/dl (11), mean LDL cholesterol 124 mg/dl (39) and median triglycerides were 147 mg/dl IQR [109-197]. Mean Cockcroft-Gault creatinine clearance was 87 ml/min and 11 pts (1.4%) had a severe chronic renal failure (lower than 30 ml/min). Mean left ventricular ejection fraction was 0.53 (0.13). 88.5% were on antiplatelet therapy, 75.2% on beta-blocker, 66% on statin therapy and 54.8% on ACE inhibitors or ARB.

A previous history of COPD was present in 3.5% of pts; 37% of them had a beta-blocker therapy. The cumulative seven-year total mortality rate was 17.9% in the whole sample (51.8% in pts with COPD and 16.7% in those without, p<0.001). Among COPD pts with beta-blocker therapy, the mortality rate was 30% while mortality reached 64.7% in COPD pts without beta-blocker (p=0.08).

After multivariate adjustment for age, diabetes, tobacco consumption (none, ≤40 pack-years, >40 pack-years), heart rate, left ventricular ejection fraction (>0.5; ≤ 0.5 and >0.35; ≤0.35), duration of CAD, ankle-brachial index (>0.9; ≤0.9 and >0.6; ≤0.6), history of stroke, statin therapy and coronary revascularization, hazard ratio for all-cause death was 2.22 (95% CI [1.15; 4.26] p=0.016) in pts with COPD compared to those without.

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