

Atherosclerosis and Inflammation

1.11 Prevalence of Metabolic Alterations In Hypertensive Patients with Stable vs Unstable Angina

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Introduction: Stable and unstable angina represent extremely different conditions, both by clinical and pathophysiological point of view.

Aim. To investigate the prevalence of lipidic and metabolic alterations in a population of hypertensive patients with stable vs unstable angina.

Methods. We prospectively enrolled 63 hypertensive patients with ischemic heart disease, aged from 30 to 70 years. The study population was divided in 18 patients with stable angina (SA) and 45 with unstable angina (UA). Patients with co-morbidities (i.e. diabetes, COPD etc.), previous myocardial infarction and left ventricular dysfunction (EF < 50%) were excluded. Therapy with statins or allopurinol represented exclusion criteria. In all patients we assessed blood glucose, total cholesterol (TC), LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), triglycerides (TG), apolipoprotein A1 (apoA1), apolipoprotein B (apoB), lipoprotein(a) [Lp(a)], uric acid, hs-PCR and serum creatinine. All patients underwent standard blood pressure measurement and transthoracic echocardiography (TTE). Left ventricular mass (LVM) was indexed to height^{2,7} (LVM/h^{2,7}) and left ventricular hypertrophy (LVH) was defined as > 51 g/m^{2,7} in men and > 47 g/m^{2,7} in women. Inappropriate left ventricular mass (ILVM) was also calculated. All patients underwent coronary angiography.

Results. Patients with UA presented higher levels of apoB, apoB/apoA1 ratio, Lp(a), uric acid, hsPCR and fasting glucose (p<0,05). ApoA1 levels were significantly lower in patients with UA compared to SA (p<0,05). However, both groups did not differ significantly for TC, LDL-C, HDL-C, TG (p=ns). SA and UA patients significantly differed for LVM/h^{2,7}, with higher prevalence of LVH and ILVM in patients with UA (p<0,05). After a multivariate analysis, adjusted for age, gender, BMI, SBP, DBP, RWT, the presence of UA was an independent predictor of higher apoB, apoB/apoA1 ratio, uric acid and hs-PCR levels (p<0,05); conversely, UA resulted associated to lower apoA1 levels (p<0,05). Both groups did not differ for the prevalence of coronary disease, as assessed by angiography (p=ns).

Conclusions. In a population of hypertensive patients, the presence of UA compared to SA was associated to higher levels of pro-atherogenic (apoB) and lower levels of anti-atherogenic apolipoproteins (apoA1), independently from cholesterol values. Our results suggest a superiority of apolipoproteins compared to non-HDL-C to identify patients with more advanced atherosclerotic disease and inflammation, with higher risk toward plaque instability.