Letter to the Editor

High density lipoprotein changes in patients with microvascular angina

Moses S. Elisaf\textsuperscript{a,∗}, Sonia Athina P. Karabina\textsuperscript{b}, Alexandros D. Tselepis\textsuperscript{b}

\textsuperscript{a}Department of Internal Medicine, School of Medicine, University of Ioannina, GR 451 10 Ioannina, Greece
\textsuperscript{b}Laboratory of Biochemistry, Department of Chemistry, University of Ioannina, GR 451 10 Ioannina, Greece

Received 23 June 1998; accepted 6 July 1998

Keywords: Atherosclerosis; Cholesterol; Lipid metabolism; Lipoproteins

We have read with great interest the recently published paper by Miwa et al. on HDL abnormalities observed in patients with variant angina [1]. The authors noticed that high HDL cholesterol/ApoA1 levels associated with low HDL cholesterol and ApoA1 levels were characteristic in patients with variant angina, in whose HDL particles were large, cholesterol-rich and possibly malfunctioning. Since similar to variant angina altered endothelial-mediated vasomotion of coronary arteries is also observed in the microvasculature of patients with microvascular angina [2,3], such HDL abnormalities could have also played a role in its pathogenesis. Recently we have studied the lipid profile of patients with microvascular angina in comparison with patients with coronary artery disease (CAD) and normal subjects [4]. By contrast to the findings observed in patients with variant angina, HDL cholesterol and ApoA1 levels in microvascular angina patients did not differ from those observed in control subjects, although the levels of these parameters in both groups were significantly higher than those in the CAD patients. However, the HDL cholesterol/ApoA1 molar ratio was significantly higher in both groups of patients compared to the control population [21.9±1.2 in microvascular angina patients and 22.1±1.3 in CAD patients vs 20.7±1.8 in the control group, p<0.05 for both comparisons], while the slope of the regression line comparing HDL cholesterol and ApoA1 was greater in patients with microvascular angina than in the control subjects (p<0.05), implying an increase in larger HDL particles. Interestingly, the HDL particles appeared to be larger in patients with microvascular angina as compared to controls both in the smoking and in the non smoking subpopulations.

Thus, despite the absence of lower HDL cholesterol levels, patients with microvascular angina, as well as patients with CAD, exhibit larger HDL particles, which, along with the other lipid derangements observed in this population, could contribute to the existing functional abnormalities.

References


∗Corresponding author. Tel.: 30 651 97509; Fax: 30 651 45944.