Sir, the recent report on CD4+ and CD8+ in dyslipidemia is very interesting. Pereira de Moura et al concluded that 'it was possible to show a reduction of some molecules after application of acetylsalicylic acid.' In fact, the role of CD4+ and CD8+ in dyslipidemia is still a myth. There is still no direct biological process and molecular function of CD4+ and CD8+ that is directly relating to lipid metabolism. The interesting concern is on the existence of dyslipidemia in the patients with impaired T cell function such as those with HIV infection. There has never been any detected relationship between CD4+ or CD8+ count with the level of blood lipid. In addition, the blood lipid disorder in cases with problem of HIV can also be effect by other factors such as exercise.

Dear Sir, I appreciate the comments you made on my article. In fact there is not any proof about the relationship between any type of T cell and the lipid metabolism. But there is a lot of evidence about some inflammatory molecules, like cell adhesion molecules, interleukines, and others, produced by the inflammatory cells, like T Cell and macrophages, with not only the pathogenesis of atherosclerosis, but also with lipoproteins and their pro-atherogenics changes. For example, peripheral blood mononuclear cells (PBMCs) from familial hypercholesterolemia patients spontaneously released significantly higher levels of macrophage inflammatory protein (MIP)-1α, MIP-1β and interleukin (IL)-8, and had a significantly lower oxLDL-stimulatory ratio for MIP-1α and MIP-1β than cells from healthy controls. Spontaneous release of these inflammatory molecules correlated positively with plasma concentrations of total and LDL cholesterol. In vitro studies showed that FH serum but not control serum was able to induce enhanced spontaneous release of chemokines in PBMCs from both FH patients and control subjects. In 94 consecutive admissions with stable coronary artery disease (CAD) - 39 patients with hypercholesterolaemia (HC) and 22 patients with mixed hyperlipidaemia (HL), the serum TNFalpha levels were higher in all CAD groups than in healthy subjects. IL-10 levels were higher in group HC than in controls. In all CAD patients TNF-alpha showed a negative correlation with HDL-cholesterol and a positive correlation with triglycerides. The authors concluded that immune activation (TNF-alpha, sTNFR 1, sTNFR 2, and IL-10) in CAD patients is related to serum lipids levels. In 80 hypercholesterolemic patients and 80 matched healthy subjects, the hypercholesterolemic patients had enhanced levels of sCD40L compared with healthy subjects.

With best regards.

José PEREIRA DE MOURA

REFERENCES


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