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INTERPLAY HDL PARTICLES AND INFLAMMATION MARKERS IN CORONARY STENOSIS

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Abstract

Introduction: Inflammation provides important links between risk factors and the mechanisms of atherosclerosis. The clinical value and the interrelationship of HDL was followed with acute phase proteins hsCRP, fibrinogen and SAA, with apo-lipoproteins, A-I and B and serum levels of cytokines in 198 patients with cardiovascular disease.

Methods: On exclusion criteria (MI, heart failure, CHD >2 years, anticoagulant therapy, 198 patients were recruited and were subdivided with stenosis <50% and >50% in accordance with CASS. Lipids were measured on OLYMPUS AU640. LDL-ox was determined by immunosorbent assay and SAA by immunonephelometry. Plasma MDA was assayed using a high performance liquid chromatography method based on the classic thiobarbituric acid (TBA) reaction. Serum levels of cytokines and hsCRP were analyzed by solid-phase chemiluminescent immunometric assay on DPC Immulite 1.000.

Results: Highest ox-LDL was associated with highest percent of stenosis and HDL is highly inversely related to the degree of stenosis. The HDL data were confirmed with a similar significant change of apo A(I) concentration from 134mg% with normal vessels and 123,0 mg with >50% stenosis. HDL-C and apo A(I) are directly inversely related to the degree of stenosis and directly to the acute phase proteins. TNFα ($p<0.1$) and IL6 are related to the degree of stenosis.

Conclusions: HDL-c is highly inversely related to the degree of stenosis, directly related to the APP and inversely to pro-inflammatory cytokines. SAA is responsible for the reassembly and dysfunction of HDL. Cytokines are mainly related to the dysfunction of HDL.

Keywords: cytokines, acute phase proteins, cardiovascular events, high density lipoproteins, and apoproteins.

Introduction

Atherosclerosis is an inflammatory process that can be initiated by infectious or endothelium damaging agents and aggravated by accepted and recognized risk factors such as LDL-C and ox-LDL (1,2). Many of the mechanisms involved in the process of atherogenesis are very similar to those seen in other chronic inflammatory diseases. Plasma markers of inflammation are increased in patients with atherosclerosis and varied with the site and extent of the disease, but do not provide diagnostic power above established risk factors (3). HDL is a truly independent predictor of risk and evidence now shows that increasing the precursor, apo A(I), is nearly always protective (4). There are many plausible and proven mechanisms by which HDL can inhibit atherosclerosis, including removal of cholesterol, anti-oxidation, anti-inflammation and very importantly, anti-monocyte adherence actions. Thus, an understanding of HDL metabolism and the structural changes as the remodeling are critical to explaining why increased HDL is protective (5).

Chronic infection creates a proinflammatory state which is characterized by long term elevation of cytokines and acute phase proteins and the conditions which are associated with the clinical outcome and manifestations of atherosclerotic disease (6). The most prominent cells that invade in evolving lesions are monocyte-derived macrophages and T-lymphocytes producing a wide array of soluble inflammatory mediators important in the initiation and perpetuation of the disease (7). The production of pro-inflammatory cytokines in chronic inflammation is the result of the T cell monocytes interactions and that mechanism seems to be blocked by the negative acute phase protein apolipoprotein A-I. Prospective epidemiologic studies have consistently demonstrated that markers of inflammation are independent predictors of cardiovascular events (8,9). The gap in preventive strategy prompted the search for new biomarkers in risk stratification of cardiovascular