Plasma lipoproteins and atherosclerosis in man: an immunohistochemical study

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Abstract

The localization pattern of apoLDL, the major protein constituent of the plasma low density lipoproteins, was determined in normal and atheromotic areas of human arteries from various vascular beds. Goat antisera were prepared against apoLDL, conjugated with a fluorescein isothiocyanate label, and purified by affinity chromatography on a solid immunoadsorbent of LDL Sepharose. By fluorescence microscopy, the following distribution patterns of apoLDL were found: in fibrous plaques spread diffusely throughout the lipid core usually together with acid mucopolysaccharides, along bands of collagen in fibrotic areas of intimal plaques and aneurysms, and often, but not always, accompanied by lipid deposition, along fragmented fibers of elastaica and collagen bundles, and in smooth muscle cells and macrophages of fatty streaks and fibrous plaques of subjects with type II hyperlipoproteinemia. No LDL protein was detected in arterial segments without atherosclerotic involvement. This information may help to elucidate those tissue components which are responsible for the retention of LDL, thereby leading to its accumulation and potential contribution to the pathogenesis of atherosclerosis.
In another study, normolipidemic men with CAD and subjects with dysbetalipoproteinemia had elevated levels of IDL when compared to controls; furthermore, increased levels of IDL were not detected with conventional lipid screening. B. Thus, IDL may be major determinant of atherogenic potential of LDL. Triglyceride rich lipoproteins and apo C-III A. Elevations in serum TG are associated with an increased risk for atherosclerosis and some experts consider them to be an independent risk factor. In addition, elevated levels of apo C-III are correlated with and may be causative for hypertriglyceridemia. In future studies, in addition to continuously exploring the complex mechanisms of lipid/lipoprotein-related atherosclerosis in animal models, it will be important to apply convincing findings from animal models into human clinical trials. View chapter Purchase book. Read full chapter. A target-lesion approach alone, however, will not eliminate the threat posed by all of the vulnerable plaques to come, and their overall risk determines the long-term prognosis. To develop drugs that can protect vulnerable plaques against rupture and thrombosis, it is imperative to develop new, preferably small-animal, models in which the biology and dynamics of vulnerable plaques can be studied. The penetration of low-density lipoproteins through the endothelium, the contact of these particles with the potential foam cells, the effect of various cellular migratory and growth factors, the prostaglandin system and the hormonal status are important factors in the mechanisms leading to the formation of the atherosclerostic plaque. Disturbances in lipid metabolism are rarely recognized by signs such as formation of xanthomata. Even the measurement of plasma lipids does not always allow conclusions to be drawn with regard to the complicated relationship between lipoprotein concentrations an