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ABSTRACTS

Lipoprotein(a) as a cardiovascular risk factor: current status

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Aims
The aims of the study were, first, to critically evaluate lipoprotein(a) [Lp(a)] as a cardiovascular risk factor and, second, to advise on screening for elevated plasma Lp(a), on desirable levels, and on therapeutic strategies.
Methods and results. The robust and specific association between elevated Lp(a) levels and increased cardiovascular disease (CVD)/coronary heart disease (CHD) risk, together with recent genetic findings, indicates that elevated Lp(a), like elevated LDL-cholesterol, is causally related to premature CVD/CHD. The association is continuous without a threshold or dependence on LDL- or non-HDL-cholesterol levels. Mechanistically, elevated Lp(a) levels may either induce a prothrombotic/anti-fibrinolytic effect as apolipoprotein(a) resembles both plasminogen and plasmin but has no fibrinolytic activity, or may accelerate atherosclerosis because, like LDL, theLp(a) particle is cholesterol-rich, or both. We advise that Lp(a) be measured once, using an isoform-insensitive assay, in subjects at intermediate or high CVD/ CHD risk with premature CVD, familial hypercholesterolaemia, a family history of premature CVD and/or elevated Lp(a), recurrent CVD despite statin treatment, ≥3% 10- year risk of fatal CVD according to European guidelines, and/ or ≥10% 10-year risk of fatal + non-fatal CHD according to US guidelines. As a secondary priority after LDL-cholesterol reduction, we recommend a desirable level for Lp(a), 80th percentile (less than 50 mg/dL). Treatment should primarily be niacin 1–3 g/day, as a meta-analysis of randomized, controlled intervention trials demonstrates reduced CVD by niacin treatment. In extreme cases, LDL-apheresis is efficacious in removing Lp(a).
Conclusion**.** We recommend screening for elevated Lp(a) in those at intermediate or high CVD/CHD risk, a desirable level, 50 mg/dL as a function of global cardiovascular risk, and use of niacin for Lp(a) and CVD/CHD risk reduction.
Keywords: Lipids • Hyperlipidemia • Prevention • Myocardial infarction • Stroke

Atorvastatin and fatty acids concentration in patients with IHD

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Abstract
The aim of the study was to examine effects of atorvastatin on blood levels of fatty acids. Subjects and methods.
18 participants with coronary heart disease and dyslipidemia, 8 men и 10 women, 47 – 74 years of age (mean age 58.2±6.6) were assigned to receive 10 mg atorvastatin daily for 12 weeks. Circulating fatty acids in a drop of blood from a fingertip were assessed by capillary gas chromatography before and after 12 weeks of treatment with atorvastatin.
Results. After treatment with atorvastatin there were no significant changes for the main fatty acids concentrations except increase in level of arachidonate by 41% (from 5,67±1,67% of total fatty acids to 8,02±2,00%, p=0,034).
Conclusions. The treatment with statins leads to increase in arachidonic acid concentration in patients with coronary heart disease.

Keywords: fatty acids, coronary heart disease, dyslipidemia, atorvastatin.

Contents of proinflammatory cytokines, chemoattractants and destructive metalloproteinases in varrious tipe of unstable atherosclerotic plaques

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Abstract
Typical and significant inflammatory, destructive biomarkers and chemoattractants of various types of unstable plaques (lipid type, inflammatory-erosive type, necrotic type) of coronary arteries were determined and studied in coronary atherosclerosis men without acute coronary syndrome. The increased inflammatory activity (increased concentrations of interleukin 1-beta, interleukin 6, interleukin 8, interleukin 18 and monocytes chemotactic protein – 1) was typical not only for unstable plaques inflammatory-erosive type, but for lipid type too in comparison with necrotic type of unstable plaques. On the other hand, increased destructive activity (increased levels of tumor necrotic factor alpha, endothelin 1 and decreased level of tissue inhibitor of metalloproteinase-1) was typical for necrotic type of unstable plaques only.
Keywords: atherosclerotic plaques of coronary arteries, lipid, inflammatory-erosive and necrotic types of unstable plaques, inflammatory cytokines, chemoattractants, destructive matrix metalloproteinases.

Visualization, composition and structure of endothelial glycocalyx

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Abstract
In normal state, a complex multicomponent system called glycocalyx is present on the surface of endothelial vascular system. The structure of the glycocalyx is determined by a group of proteoglycans, glycoproteins and glycosaminoglycans, originating from endothelial cells and blood flow. Due to its complexity and location on the border of the system of blood circulation, glycocalyx participates in a number of functions supporting the metabolism of the vascular wall. In pathological conditions undergo complete or partial loss of this structure, which leads to inconsistencies in the vascular wall and change its functions. In the first part of this review considers the history of detection and determination of endothelial glycocalyx structure, utilized methods and approaches. Described in detail the molecular composition of the glycocalyx, properties of its components and overall glycocalyx structure..
Keywords: endothelium, glycocalyx, proteoglycans, glycoproteins, glycosaminoglycans, spatial structure, in vivo microscopy.

Lipoprotein-associated phospholipase A2 – a new position in the risk stratification?

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Abstract
Authors consider possibility of using lipoprotein-associated phospholipase A2 (Lp-PLA2) for the assessment of atherosclerotic plaque's status and for its risk stratification application.

Mechanisms of Lp-PLA2 secretion regulation and Lp-PLA2 impact on the atherosclerotic plaque destabilization are described.
Keywords: Lipoprotein-associated phospholipase A2, risk stratification, unstable atherosclerotic plaque.

Atherosclerosis and coronary heart disease: some aspects of pathogenesis

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Abstract
The authors examine the current views on the pathogenesis of atherosclerosis. Fundamental research in molecular biology, genetics, immunology greatly expanded understanding of the pathogenesis of atherosclerosis. The review stresses the important role of inflammatory, hemostatic and thrombotic factors in the pathogenesis of atherosclerosis.
Keywords: Atherosclerosis, pathogenesis, inflammatory, hemostatic, thrombotic factors