BALNEO RESEARCH Balneo and PRM Research Journal English Edition
e ISSN 2734 - 8458 p ISSN 2734 - 844X ROMANIAN ASSOCIATION OF BALNEOLOGY

PATHOPHYSIOLOGY OF DYSLIPIDEMIAS IN INFLAMMATORY RHEUMATIC DISESASES AND CARDIOVASCULAR RISK

CHICU Mihaiela[1], ANTON Carmen[2]

Editor: Constantin MUNTEANU, E-mail: office@bioclima.ro

Balneo and PRM Research Journal DOI: http://dx.doi.org/10.12680/balneo.2021.452 Vol.12, No.3 September 2021 p: L95

Corresponding author: **CHICU Mihaiela,** E-mail: mihaela.chicu@umfiasi.ro

1. "Gr. T. Popa" University Iasi, Discipline of Pathophysiology
2. "Gr. T. Popa" University Iasi, Discpline of Medical Semilology-Gastroenterology

Abstract

Introduction. Cardiovascular diseases, cancer, chronic respiratory diseases and diabetes are the leading causes of death worldwide. Of these, cardiovascular diseases represent approx. 46.2% of all deaths. In the pathogenesis of the main causes of mortality can be found common molecular mechanisms, such as systemic inflammation and oxidative stress.

Discussion. Inflammation play a key role not only in atherogenesis but also in the acute coronary syndromes, stroke or acute peripheral ischemia. It has been shown that the association between subclinical atherosclerosis and active systemic inflammation may increase the risk of atherothrombotic complications and may increase the accuracy of cardiovascular risk determination. Atherosclerosis is considered a chronic inflammatory disease of the arterial wall in which, although under drug treatment the aggressive decrease of LDL-col to values below 50 mg / dl, reduce the inflammatory parameters are obtained, the residual cardiovascular risk is maintained.

Hypercholesterolemia is one of the main factors in proinflammatory transformation of the vascular endothelium.

Conclusions. Both statin and anti-PCSK-9 monoclonal antibodies studies confirm that they are reducing the plasmatic LDL levels, they reduce the inflammatory parameters (ESR, CRP) to a lesser extent - suggesting that VLDL and IDL residues are primarily responsible for inflammatory reaction in atherogenesis. Phenofibrates reduce the lipoproteins rich in TG by modulating PPAR-α factor when administered concomitantly with statins. Statins not only reduce the serum level of LDL-col, but also reduce the adhesion to the endothelium of monocytes and T lymphocytes by increasing the expression of "Krupple-like factor-2". Reducing cell inflammation in the atheroma plaque could stabilize atherosclerotic plaque.

Key words: *Inflammation, ATS, LDL, PPAR-a, Krupple-like factor-2*