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Oxidized low density lipoproteins: The bridge between atherosclerosis and autoimmunity. Possible implications in accelerated atherosclerosis and for immune intervention in autoimmune rheumatic disorders.

Suciu CF[1], Prete M[2], Ruscitti P[3], Favoino E[2], Giacomelli R[3], Perosa F[4].

Author information

1 Department of General Medicine M3, University of Medicine and Pharmacy of Targu Mures, Romania.
2 Department of Biomedical Sciences and Human Oncology (DIMO), Systemic Rheumatic and Autoimmune Diseases Unit, University of Bari Medical School, Bari, Italy.
3 Department of Biotechnological and Applied Clinical Science, Division of Rheumatology, University of L'Aquila, L'Aquila, Italy.
4 Department of Biomedical Sciences and Human Oncology (DIMO), Systemic Rheumatic and Autoimmune Diseases Unit, University of Bari Medical School, Bari, Italy. Electronic address: federico.perosa@uniba.it.

Abstract

Atherosclerotic vasculopathy is a multifactorial process causing vessels damage and cardiovascular diseases, the leading causes of death worldwide. Atherosclerotic plaque is the asymptomatic primary, elementary, lesion of atherosclerotic vasculopathy. Accumulation of the oxidized low-density lipoprotein (oxLDL) at sub endothelial sites is now recognized as one of the major trigger events in plaque formation. The concomitant presence at the plaque site of cells belonging to either natural or adaptive immunity, the detection of autoantibodies to oxLDL, the cross-reactivity of oxLDL with anti-phospholipid antibodies, in addition to the clinical evidence of increased rates of cardiovascular events in several rheumatic diseases, has stimulated intensive research to define interconnections between the immune system and traditional risk factors at the molecular levels in order to explain accelerated atherosclerosis. Here, we critically review the results of previous and recent studies, which have disclosed molecules of both innate or adaptive immunity involved in atherosclerosis, focusing primarily on B cells and autoantibodies, where data are more consolidated. Particular attention has also been paid to molecules that may be predictive markers of atherosclerosis progression and can be potential targets for immune intervention to delay the atherosclerotic process. The latter include CD20 antigen, molecules involved in the BAFF-BAFF receptor axis, inflammatory molecules and modified LDL. The successful results of a recent randomized controlled clinical trial targeting inflammasome with anti-IL1β monoclonal antibody in non-autoimmune conditions, prove that specific immunotherapy can be a promising and effective strategy to control atherosclerosis in rheumatic diseases as well.

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