ISSN: 2591-7951

# Clinical characteristics of atherosclerosis.

**Harry Pacific Friedman***

Department of Cardiology, University of Maryland, Maryland, United States

*Accepted on September 28, 2021*

## Description

Atherosclerosis is a multifocal, immune inflammatory ailment of medium-sized and large arteries fuelled by lipids. Endothelial cells, leukocytes, and intimal smooth muscle cells are the key players in the progress of this disease. The maximum demoralizing significances of atherosclerosis, such as heart attack and stroke, are caused by superimposed thrombosis. After, years of indolent growth, unexpectedly becomes complicated by luminal thrombosis. If thrombosis-disposed to plaques could be identified and thrombosis prevented, atherosclerosis would be a much more benign disease. Atherosclerosis, previously deliberated a mild lipid storage ailment, actually involves a continuing inflammatory response. Current developments in basic science have established a major role for inflammation in mediating all phases of this disease from initiation through progression and, ultimately, the thrombotic complications of atherosclerosis. Plaque burst is a more common cause of coronary thrombosis in men than in women. Ruptured plaques are characterized by a huge lipid-rich core, a tinny fibrous cap that comprises few smooth muscle cells and various macrophages, angiogenesis, adventitial inflammation, and external remodeling. Ruptured plaques and, by implication, rupture-disposed to plaques have characteristic pathoanatomical structures that might be useful for their exposure *in vivo* by imaging. Lesions of atherosclerosis comprise macrophages, T cells and additional cells of the immune response, together with cholesterol that permeates from the blood. Targeted deletion of genes encoding costimulatory factors and proinflammatory cytokines results in less disease in mouse models, whereas interloping with regulatory immunity accelerates it. Distinctive as well as adaptive immune responses have been recognised in atherosclerosis, with components of cholesterol-carrying low-density lipoprotein triggering inflammation, T cell stimulation and antibody production during the course of disease. The lesions result from an unnecessary, inflammatory-fibroproliferative response to various forms of insult to the endothelium and smooth muscle of the artery wall. A huge amount of growth factors, cytokines and vasoregulatory molecules engage in this process. Our ability to control the appearance of genes encoding these molecules and to target specific cell types provides opportunities to develop new diagnostic and therapeutic agents to induce the regression of the lesions and, possibly, to prevent their development. The response-to-injury assumption of atherosclerosis proceeds into account interactions among all of the cells originate in the lesions of human atherosclerosis, the cytokines and growth factors that can be moulded by both of these cells (which are in fact formed *in vitro* and which have been demonstrated *in vivo*). It should therefore be potential to develop new diagnostic tools that will help us to make early diagnoses for patients who are at threat and define the state of lesion development in these patients. The presence of T lymphocytes cells, and some initial preliminary data, representing that antigens unique to the lesions of atherosclerosis are existing in the lesions, suggest that several of the lesions of atherosclerosis signify immune or autoimmune responses. The nature of the antigens, the response to these antigens, and the role of antigen-antibody complexes in the progression of atherogenesis are significant to understanding of this complex ailment process.

## *Correspondence to

Harry Pacific Friedman

Department of Cardiology

University of Maryland

Maryland

United States

Email: harry.pacfri@gamil.com

***Citation:*** *Friedman HP. Clinical characteristics of atherosclerosis. Arch Gen Intern Med 2021;5(8):2*