Pathogenesis of arteriosclerosis and its risk factors.

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Atherosclerosis is no longer considered a disorder due to abnormal lipid metabolism. In fact, the onset event of atherosclerosis can be an inflammatory infection that occurs decades before the disease becomes clinically apparent. The rapid development of knowledge about the etiology of atherosclerosis, combined with new target-specific therapies, is revolutionizing the treatment of atherosclerosis. As a result, various therapies are currently being evaluated for their ability to improve inflammatory pathways that may initiate and expand the process of atherosclerosis. Once initiated, atherosclerosis progresses as a result of a series of well-studied changes in the composition of the cell walls of blood vessels. Specific cytokine-mediated events in this cycle are required for lesion growth. Interventions such as percutaneous coronary interventions can treat isolated disease areas because the clinical manifestations of atherosclerosis appear very late in this process [1].

Marchan introduced the term "atherosclerosis" to describe the link between fatty degeneration and arteriosclerosis. This process affects medium and large arteries and is characterized by sub intimal, patchy intramural thickening that invades the arterial lumen. Any vascular bed can be affected by this process. The etiology, treatment, and clinical significance of atherosclerosis vary from vascular bed to bed. The earliest visible lesion of atherosclerosis is the fatty streak, which results from the accumulation of lipid-containing foam cells in the intima layer of the artery. Over time, fatty streak develops into fibrotic plaques that are characteristic of established atherosclerosis. Ultimately, lesions can develop to contain large amounts of lipids. When it becomes unstable, exposure of the upper endothelium or rupture of plaque can lead to thrombotic obstruction of the upper artery [2]. Atherosclerosis lesions are composed of three main components. The first is the cellular component, which is mainly composed of smooth muscle cells and macrophages. The second component is the connective tissue matrix and extracellular lipids. The third component is the intracellular lipid, which accumulates in macrophages and thereby converts them into foam cells. Atherosclerotic injuries create as a result of incendiary boosts, consequent discharge of different cytokines, expansion of smooth muscle cells, blend of connective tissue lattice, and collection of macrophages and lipids. In its early stages, the process of atherosclerosis is characterized by impaired endothelial function.

Atherosclerosis can be initiated when endothelial cells overexpress adhesion molecules in response to turbulence in an environment of unfavorable serum lipid profiles. Animals fed an atherosclerotic diet rapidly overexpress vascular cell adhesion molecule 1 (VCAM1). Li3 showed that expression of VCAM1 on the surface of the endothelium was an early necessary step in the pathogenesis of atherosclerosis. Increased cell adhesion and associated endothelial dysfunction then "stage" inflammatory cell recruitment, cytokine release, and lipid recruitment to atherosclerotic plaques. It is now generally accepted that the early stages of onset of atherothrombosis are primarily mediated by the inflammatory cascade. Expression of 4VCAM1 increases the recruitment of monocytes and T cells to the site of endothelial injury. Subsequent release of monocytic chemotactic protein 1 (MCP1) by leukocytes amplifies the inflammatory cascade by mobilizing additional leukocytes, activating inner leukocytes, and causing smooth muscle cell recruitment and proliferation [3].

In response to signals generated within the initial plaque, monocytes attach to the endothelium and then travel through the endothelium and basement membrane to degrade the connective tissue matrix of locally activated matrix metalloproteinases (Develop enzymes including MMP). The recruited macrophages release additional cytokines and begin to migrate through the endothelial surface to the tunica media of blood vessels. This process is further enhanced by the local release of monocyte colony stimulating factor (MCSF), which causes monocyte proliferation. Local activation of monocytes causes both cytokine-mediated progression of atherosclerosis and oxidation of low-density lipoprotein [4].

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