CARDIOVASCULAR DISEASE AND INFLAMMATION

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Abstract: The study reviewed research on the function of endothelium local factors, including the build-up of smooth muscle cells, T and B lymphocytes, macrophages, matrix metalloproteinase (MMP), and high-sensitivity C-reactive protein (CRP). In coronary artery disease (CAD), oxidized low-density lipoprotein (OLDL) and CRP are directly linked to inflammatory arterial damage. One of the leading causes of death and disability in developed nations is coronary heart disease (CHD). Although numerous risk factors for the onset and progression of CHD have been investigated, the primary cause and trigger for acute coronary problems remain unclear. Atherosclerosis and thrombosis are the primary processes that cause acute coronary syndrome (ACS) [1]. The so-called susceptible atherosclerotic plaques are typically the target of inflammation. Endothelial cells, SMC, monocytes, neutrophils, platelets, macrophages, T-lymphocytes, and B-lymphocytes are the primary active constituents of loose connective tissue.

Early on in the development of arterial disease, as well as throughout the phase of AB instability and destruction, there are indications of a local nonspecific inflammatory process in atherosclerosis. The involvement of inflammation in the process of AB destabilization has been examined to a larger extent and it has been demonstrated that lipids are not engaged in the mechanism of AB degradation [1]. The inflammatory response in atherosclerotically altered arteries includes circulating leukocytes in addition to the equivalent arterial wall cells. Both macrophages and lymphocytes are essential to this process [3].

The innate (non-specific) and adaptive immune systems work together to maintain the delicate balance between the inflammatory and anti-inflammatory states when the immune system is active. The capacity to alter receptor expression for novel autosomal or foreign substances is a hallmark of the adaptive immune system. Its role is to control the immune response and to preserve humoral and cellular immunity. T-helpers (CD4+), cytotoxic T-killers (CD8+), and B-lymphocytes are among the cells that make up the adaptive immune system. CD4+ T cells can develop into at least three categories following antigen activation: 1) Th1, which produces proinflammatory cytokines like γ-interferon (IFNγ) and is important in cellular immunity; 2) interleukin-2 (IL-2), lymphotoxin, and Th2, which preserve humoral resistance and release anti-inflammatory cytokines like IL-4 and IL-10;Macrophages may congregate in a central core in the typical atherosclerotic plaque where they can undergo apoptosis producing the "necrotic core" of the atherosclerotic lesion or release MMPs which degrade the extracellular matrix promoting plaque rupture. Coronary spasm due to smooth muscle hyperreactivity is the predominant cause of myocardial infarction in patients with a history of vasospastic angina, although this event is rare. However, coronary vasoconstriction and thrombosis are deeply interrelated. On the one hand occlusive coronary spasm and distal blood stagnation are known to cause a transient several fold increase of fibrinopeptide A in systemic blood. On the other serotonin, a substance released by activated platelets, is known to produce occlusive spasm in patients with variant angina and ischemia due to distal vessel constriction in patients with chronic stable angina. The cytokines secreted by activated inflammatory cells have the potential to activate the endothelium transforming its antiadhesive and anticoagulant properties into adhesive and procoagulant properties. Indeed, endothelial cells stimulated by IL-1, TNF or endotoxin express adhesion molecules such as intercellular adhesion molecule-1 (ICAM-1) and E-selectin on their surface and secrete soluble chemoattractants such as monocyte chemoattractant protein-1 (MCP-1), monocyte colony stimulating factor (M-CSF) and IL-8. Of

note, in activated endothelial cells different adhesion molecules and chemoattractants are expressed almost simultaneously, thus suggesting a concerted activation of different genes probably, related, at least partially, to the activation of the nuclear factor κB (NF-κB)[6]. The latter was initially described in lymphocytes where it controls the activation of genes which encode for the κ chains of immunoglobulins.