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BOOK REVIEW

Inflammatory and Infectious Basis of Atherosclerosis

(Mehta, J. L., ed., Birkhauser Verlag, Basel-Boston-Berlin, 2001)

The book "Inflammatory and Infectious Basis of Atherosclerosis" edited by Jay L. Mehta is focused on the mechanisms of highly specific cell and molecular responses to endogenous and exogenous anti-inflammatory and pro-atherogenic risk factors. The book consists of sixteen chapters written by specialists from different countries. It develops the idea of Rassel Ross that atherosclerosis is an inflammatory disease. The first four chapters are devoted to the theoretical and pathologic aspects of inflammation and atherosclerosis. The next six chapters analyze the contribution of free radicals, oxidized low-density lipoproteins (ox-LDL), angiotensin II, and some cytokines to inflammation and atherosclerosis development.

In the first chapter, G. Schmitz and M. Torzewski analyze in detail the mechanisms of involvement of endogenous (chemically modified lipoproteins) and exogenous (pathogens cytomegalovirus and *Chlamydia pneumoniae*) antigens in triggering chronic inflammation and the progress of atherogenesis. The authors regard the prevention of lipoprotein modification as an important direction of therapeutic strategy.

The next chapter written by R. Virmani, F. Kolodgie, A. Burke, and A. Farb considers the pathologic aspects of inflammation in coronary atherosclerosis. The site of lesion of the atherosclerotic plaque fibrous capsule is characterized by a high density of macrophages and T lymphocytes and a low content of smooth muscle cells (SMC). The cytokine interferon γ (INF-γ) secreted by T lymphocytes inhibits collage synthesis and SMC proliferation and promotes their apoptosis, leading to the plaque destabilizing. In addition, cytokines activate macrophages rich in matrix metalloproteinases (MMP), which stimulate degradation of collagen, proteoglycans, and elastins.

The next chapters written by A. C. van der Wal, O. J. de Boer, and A. E. Becker focuses on pathology of acute coronary syndromes, morphological characteristics of unstable atherosclerotic plaque, as well as on inflammatory and reparative processes occurring in the unstable plaque. One of the chapters (D. S. Zander) is devoted to the analysis of risk factors and the mechanisms of atherosclerosis rapid progressing in transplantation.

The authors of chapter "Myocardium Reperfusion: Inflammation State" (K. A. Youker, N. F. Frangogiannis, and M. L. Entman) used the model of ischemic myocardium reperfusion to analyze the cellular and molecular mechanisms of injured myocardium healing from acute inflammation to tissue reparation. Positive aspects of inflammatory response leading to infarct tissue healing and remodeling and negative effect on tissue reparation of nonspecific antiinflammatory therapy are discussed.

Chapter "LOX-1 as Potential Inflammation Mediator in Atherosclerosis" (M. Chen and T. Sawamura) discusses the data on the structural organization of the gene encoding LOX-1 (a lectin-like receptor for ox-LDL), its functions as an ox-LDL scavenging receptor expressed by endothelial cells, and positive regulation of LOX-1 expression by pro-atherogenic and antiinflammatory factors, such as TNF-α, IL-1β, IFN-γ, ox-LDL.

Chapter "Angiotensin II as Inflammation Mediator in Atherosclerosis" (M. I. Phillips, S. Kagiyama, H. Chen, and J. L. Mehta) considers in detail the functions of angiotensin II in the cell, including the activation of transcriptional factor NF-κB, which regulates the expression of cytokines (IL-6 and IL-8), growth factors, and MCP-1 (monocyte chemoattractant protein 1). The latter activates T lymphocytes and, together with the growth factors, stimulates proliferation of smooth muscles of vessels. Angiotensin II activates the AT_1R receptor, thereby stimulating LOX-1 expression. Antagonists of this receptor block the expression of LOX-1 and MCP-1 mRNA. In addition, it is shown that blockade of AT_1R with losartan significantly decreased the expression of MMP-1 but not its inhibitor (TIMP-2) in animals with hypercholesterolemia.

The next chapter (B. Schieffer and H. Drexler) continues discussing the effect of angiotensin II as inflammation mediator stimulating the synthesis and release of inflammation cytokines, including IL-6, which determines the considerable contribution of the rennin–angiotensin system in development of acute coronary syndromes.

Chapter " Heme Oxygenase 1, Inflammation, and Atherosclerosis" (A. Agarwal, N. Hill-Kapturczak, and

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H. S. Nick) provides the characteristics of heme oxygenase (HO) isoforms—inducible (HO-1), constitutive (HO-2), and homologous to the latter HO-3. The functions of heme degradation products—carbon oxide (CO), which exerts vasodilator activity mediated by cGMP and anti-aggregation activity (towards platelets), and biliverdin, which is reduced by biliverdin-reductase to bilirubin—are considered. It is noted that HO-1 expression in atherosclerosis is an important adaptive mechanism underlying cytoprotective and antioxidant effects.

The chapter "Lymphocyte Activation in Acute Coronary Syndromes" (S. de Servi and A. Mazzone) describes the acceleration of atherosclerotic process and frequent occurrence of restenoses after coronary angioplastics induced by increased concentration of cytokine IL-2, which is produced by T lymphocytes, or its receptor. In addition, cytokine IFN-γ secreted by the activated T lymphocytes enables destabilization of atherosclerotic plaques, because it inhibits collagen synthesis and SMC proliferation and stimulates their apoptosis.

The last four chapters discuss clinical aspects of inflammation and infection in atherosclerosis and give a prognostic estimation of some inflammation mediators in the development of atherothrombosis and other atherosclerotic complications.

On the whole, the book analyzes in detail the cellular and molecular processes in atherosclerosis and inflammation. Experimental and pathologic data summarized in this book confirm the hypothesis that atherosclerosis is basically an inflammatory disease displaying all peculiarities characteristic of this type of diseases.

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