Editorial

Inflammation in Atherosclerosis: Current Therapeutic Approaches

During the last years, it has been well established the crucial role of inflammatory process in the initiation and progression of atherosclerosis. Large scale studies have reported that the majority of the patients with coronary artery disease have one or more traditional/novel risk factors predisposing to atherosclerosis and strongly associated to inflammatory process [1, 2]. Moreover, several studies have reported that most atherosclerotic manifestations could be prevented if the risk factors were eliminated [3]. Inflammatory mediators have been found to participate in crucial steps of atherosclerosis. More mechanistically, endothelial dysfunction is characterized by enhanced expression of adhesion molecules which mediate the attachment of circulating monocytes and lymphocytes. Not only leukocytes, but also endothelial cells are capable to secrete cytokines and growth factors promoting migration and proliferation of smooth muscle cells. Moreover, chemokines including monocyte chemoattractant protein-1 produced by vascular wall cells in response to modified lipoproteins, regulate the transendothelial migration of adherent monocytes [4, 5]. Monocytes in turn interact with endothelial cells and increase matrix metalloproteinases (MMPs) production such as MMP-9. Within the intima, monocytes mature into macrophages which proliferate and amplify the inflammatory response by the secretion of growth factors and cytokines [6]. Cytokines and growth factors provoke smooth muscle cells' mobilization, proliferation and expression of metalloproteinases that degrade elastin and collagen, allowing their penetration into the expanding lesion [6, 7]. Finally, foam cell accumulation leads to metalloproteinase overexpression, which subsequently triggers extracellular matrix destabilization. Apparently, the inflammatory response is involved in many processes related to plaque development.

Thus it has become evident that monitoring inflammatory process is crucial. In addition, identification of risk factors strongly associated to inflammation as well as potential therapeutic approaches to modify inflammatory process is of great importance. In the present issue of the journal we have included the most important pathophysiological aspects of inflammation in atherosclerosis focusing on major risk factors and the potential anti-inflammatory therapeutic approaches.

The first two papers by Tousoulis *et al* [8] and Charakida *et al* [9] discuss thoroughly the role of inflammation in the pathophysiology of atherosclerosis. In addition, they provide the current available data on the main drug categories that have been proved to ameliorate the inflammatory state in atherosclerosis.

The following three articles by Androulakis *et al* [10], Siasos *et al* [11] and Kampoli *et al* [12] focus on major risk factors of atherosclerosis that are strongly associated with increased inflammatory process. Hypertension, hyperlipidemia and diabetes mellitus are major risk factors known to promote inflammation within the atherosclerotic plaque. These articles report the associated mechanisms as well as classic and novel therapeutic approaches used in the prevention or the modification of inflammatory process in these specific group of patients

Moreover, the articles by Dumitriu *et al* [13], Della Bona *et al* [14] and Toutouzas *et al* [15] focus on a different aspect of the inflammatory response in atheroslcerosis. They provide insights into the role of the immune system and especially the activity of B and T cells. In addition, classic and novel inflammatory markers for monitoring of atherosclerosis as well as novel therapeutic approaches are presented. It is also worth mentioning the contribution of inflammatory process to plaque formation, erosion and rupture which is mainly discussed in the article by Toutouzas *et al* [15].

Finally, the article by Van-Assche *et al* [16] discusses the role of the genotypic background in the initiation and progression of atherosclerosis. In addition, it expands on the current attempts to modify atherosclerosis using inflammation-associated gene therapy.

Conclusively, I strongly believe that the present issue provides a comprehensive insight into the role of inflammation in atherosclerosis and the respsective therapeutic approaches.

Finally, I would like to extend my thanks and appreciation to all the authors and acknowledge the high-quality reviews submitted to the current issue of therapeutic approaches targeting inflammatory process in atherosclerosis, with the expectation to trigger the interest of the scientific community towards this direction.

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