Mediators of Inflammation

Hindawi

Special Issue on

Oxidative Stress and Inflammation in Arterial and Venous Diseases: Pitfalls and Challenges from Bench Side to Bedside

CALL FOR PAPERS

It is well-documented that chronic inflammation stands at the cornerstone of atherosclerosis. Immune cells, like macrophages or lymphocytes, and immune mediators, such as chemokines, are intimately involved in the progression of atherosclerosis from the very early stage of fatty streaks to the mature atherosclerotic plaques that constitute culprit lesions in myocardial infarction. Stimulation of toll-like receptors in the vascular system elicits proinflammatory cytokine release and recruitment of immune cells. During atherogenesis, the activation of the complement system, through sublytic C5b-9 assembly on smooth muscle and endothelial cells, fuels the production of chemotactic, pro-adhesion, and procoagulant cytokines. Although translational research offers a myriad of signaling pathways pertinent to atherogenesis that could potentially become novel therapeutic targets in atherosclerosis, the transition to clinical trials proves to be extremely challenging. While a substantial number of approaches were not successful (phospholipase A2 inhibitors, CC Chemokine Receptor 2 (CCR2) antagonists or pexelizumab, a humanized monoclonal antibody that binds to the C5 component of complement), others (such as IL-1ß blockage with canakinumab or the inhibition of triggering receptor expressed on myeloid (TREM)-1)) were rewarding. But beyond the results of these in vitro experiments and clinical trials, the relevance of such quests is gaining a better understanding on effective targeting of plaque inflammation in human atherosclerosis.

Venous pathology, in one form or another, affects as much as a third of the world's population. Proinflammatory cytokines, matrix metalloproteinases, and adhesion molecules are all involved in the etiology of chronic venous disease. The inflammation mediators are active players in both the formation and the resolution of vein thrombosis. The strong inflammatory response during the initial acute phase of vein thrombosis implies increased levels of interleukins, TNF- or C-reactive protein. Noteworthy, oxidative stress is a relevant event in the pathogenesis of chronic venous disease. Polymorphisms of the genes that encode the proinflammatory cytokines, proteinases, are bound to produce different effects upon the incidence or the severity of venous diseases. Also, polymorphisms that influence the metabolism of anti-inflammatory drugs, used in venous diseases, can modify their effectiveness.

We wish to invite authors to submit original research articles (in vitro, experimental and/or clinical studies), as well as review articles, that address the special issue of Inflammation in arterial and venous diseases.

Potential topics include but are not limited to the following:

- Novel inflammatory biomarkers in atherosclerosis
- Emerging anti-inflammatory therapeutic approach in atherosclerosis
- Epigenetic mechanisms in the immune-inflammatory response during atherogenesis
- Markers of oxidative stress and inflammation in the development and progression of venous diseases
- Influence of pharmacogenetics on the effectiveness of anti-inflammatory drugs in venous diseases

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/mi/osia/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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