Endothelium Dysfunction, Inflammation and Cardiovascular Disorder

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Abstract

Vascular endothelium maintains tone and free flow of blood in vessels. Several studies indicate that the impairment in the maintenance of vascular tone results in vascular endothelial dysfunction (VED) results from reduced activation of endothelial nitric oxide synthase (eNOS). Various inflammatory mediators are also upregulated during VED. Inflammation is a trait of several diseases including rheumatoid arthritis, Alzheimer’s disease, asthma, and various cardiovascular disorders. Interestingly, few recent studies demonstrated the role of inflammatory mediators in the progression of VED and vascular disease associated with this. Hence, the present review has been designed to delineate the role of various inflammatory mediators in the pathogenesis of inflammation-induced VED.

Introduction

Vascular endothelium maintains tone and free flow of blood in vessels. Several studies indicate that the impairment in the maintenance of vascular tone results in vascular endothelial dysfunction (VED) results from reduced activation of endothelial nitric oxide synthase (eNOS). Various inflammatory mediators are also upregulated during VED. Inflammation is a trait of several diseases including rheumatoid arthritis, Alzheimer’s disease, asthma, and various cardiovascular disorders. Interestingly, few recent studies demonstrated the role of inflammatory mediators in the progression of VED and vascular disease associated with this. Hence, the present review has been designed to delineate the role of various inflammatory mediators in the pathogenesis of inflammation-induced VED.

Inflammation and Cardiovascular Disorders

Recent studies indicate that inflammatory mediators are implicated in the pathogenesis of various cardiovascular and inflammatory disorders that occur due to VED and their role has burgeoned. It has been reported that in the United States, Atherosclerosis, a major inflammatory cardiovascular disorder, is one of the leading causes of mortality and disability [30-31]. Atherosclerosis is a multifactorial multistep disease that involves chronic inflammation and plaque rupture [32]. In atherogenesis, the normal functions of the endothelium are distorted, resulting in aggregating an inflammatory response [33]. These lipoprotein particles can undergo oxidative modification like that of LDL and activate inflammatory functions of vascular endothelial cells [34]. Further, cytokines, peroxides, and other substances released in response to injury may damage endothelial cells to express P-selectin, ICAM-1 and E-selectin which in turn persuade process of leucocyte adhesion and subsequently their migration leading to formation of fatty streak formation [35]. Further, Urotensin II (U-II) basically a cyclic undecapeptide is found in high concentration in atheromatous lesions [36,37]. U-II accelerates foam cell formation and proliferation of VSMC suggesting development of atherosclerotic plaque [38-40]. Beside this, inflammation was also implicated in the pathogenesis of hypertension [41-42] and various cardiovascular disorder by increasing the expression of C-RP [43-44] and activating Rennin Angiotensin Aldosterone System (RAAS) and elevates the blood pressure [45-46]. Plasma CRP concentrations also predicts the risk of myocardial infarction (MI) and ischemic stroke [47]. Angiotensin II is the main culprit responsible for triggering vascular inflammation by inducing oxidative stress resulting in up-regulation of pro-inflammatory transcription factors such as NF-kB [27,48-50]. These in turn regulate the production of various inflammatory mediators that lead to endothelial dysfunction and vascular injury [41,46-47]. Elevated plasma levels of proinflammatory cytokines and chemokines such as interleukin (IL)-1, IL-6, fractalkine, and monocyte chemoattractant protein-1 (MCP-1) currently known as CC chemokine ligand 2 (CCL2) has been elicited in the pathogenesis of pulmonary hypertension [26]. Further, various studies elicit the importance of IL-6 in both acute and chronic inflammation as it act as the main inducer of acute phase reactants such as C-reactive protein fibrinogen and serum amyloid A protein [51]. Additionally, there is inhibition of caveolin that causes proliferation of VSMC [52]. Neopterin is...
secreted by macrophages following stimulation by the cytokine interferon-g and is a susceptible marker for the activation of the cell-mediated immune system [53-54]. The serum level of neopetrin is found to be elevated in patients with unstable angina and acute MI compared [55] Fig 2 shows the pathogenesis of inflammation-induced cardiovascular disorders.

Conclusion

Inflammation induce-VED has been revealed to be involved in pathogenesis of various vascular disorders by inducing C-RP urotensin and increasing the expression of various inflammatory mediators. Rho-kinase was also found to be upregulated and actively involved in Inflammation and vascular pathogenesis.

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Illustrations

Illustration 1

Fig 1. Various signalling pathways involved in the pathogenesis of inflammation-induced VED
Illustration 2

Fig 2. Pathogenesis of Inflammation induced cardiovascular disorders
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