Fibrosis, inflammation, and their relation to arterial hypertension.

Belge Hoch*

Department of Cardiology, Centre of Postgraduate Medical Education, Warsaw, Poland

Introduction

Arterial hypertension, commonly known as high blood pressure, is a prevalent condition that affects a significant portion of the population worldwide. It is a leading risk factor for cardiovascular diseases such as heart attacks, stroke, and heart failure. Although several factors contribute to the development of hypertension, two critical processes are fibrosis and inflammation. Fibrosis is a pathological process characterized by the excessive deposition of extracellular matrix components, mainly collagen, leading to tissue scarring and stiffening. In hypertension, fibrosis affects the arterial wall, leading to reduced arterial compliance and increased peripheral vascular resistance. This results in an increase in blood pressure. The fibrosis process occurs due to the activation of fibroblasts and their differentiation into myofibroblasts [1].

These cells secrete extracellular matrix components that accumulate in the arterial wall. In hypertension, the renin-angiotensin-aldosterone system (RAAS) plays a critical role in activating fibroblasts and promoting fibrosis. Inflammation is a complex process involving various immune cells and mediators. In hypertension, inflammation occurs in the arterial wall and is characterized by the infiltration of immune cells, mainly macrophages and T cells. These cells secrete cytokines and chemokines that promote inflammation and further recruit immune cells. Inflammation in hypertension is mainly driven by oxidative stress, which occurs due to the production of reactive oxygen species (ROS) by various cells, including endothelial cells, smooth muscle cells, and immune cells [2].

The ROS activate several pro-inflammatory pathways and induce cellular damage that further promotes inflammation. The relationship between fibrosis, inflammation, and hypertension is bidirectional. Fibrosis leads to the release of several pro-inflammatory mediators that promote inflammation and immune cell infiltration. Inflammation, in turn, promotes fibrosis by inducing the activation and differentiation of fibroblasts into myofibroblasts. Additionally, inflammation can also directly affect vascular smooth muscle cells and endothelial cells, leading to their dysfunction and further promoting fibrosis. Several factors can contribute to the development of fibrosis and inflammation in hypertension. These include genetic factors, lifestyle factors such as diet and exercise, and comorbidities such as diabetes and obesity. Additionally, hypertension itself can induce both processes, leading to a self-perpetuating cycle that further worsens blood pressure control [3].

Another potential target is the NLRP3 inflammasome, a complex protein complex that regulates the immune response and is involved in the development of hypertension and its complications. Inhibition of the NLRP3 inflammasome has been shown to reduce inflammation and fibrosis in various organs, including the heart and kidneys, and could be a promising approach to reduce fibrosis and inflammation in hypertension. Finally, targeting epigenetic modifications, such as DNA methylation and histone modifications, could be a promising approach to modulate fibrosis and inflammation in hypertension. Epigenetic modifications can regulate gene expression and are influenced by environmental factors, including diet and exercise. Therefore, interventions targeting epigenetic modifications could be a promising approach to reduce fibrosis and inflammation in hypertension [4].

One promising target for the modulation of fibrosis and inflammation in hypertension is the RAAS system. This system plays a crucial role in regulating blood pressure, but its activation can also promote fibrosis and inflammation. Therefore, several drugs targeting different components of the RAAS system have been developed and are currently used in the treatment of hypertension. These include ACE inhibitors, angiotensin II receptor blockers (ARBs), and aldosterone antagonists. These drugs can not only lower blood pressure but also reduce fibrosis and inflammation in the arterial wall, leading to improved vascular function and reduced cardiovascular risk. Another potential target for the modulation of fibrosis and inflammation in hypertension is the gut microbiome. The gut microbiome is a complex community of microorganisms that can influence host metabolism and immune function. Several studies have shown that dysbiosis or an imbalance in the gut microbiome, is associated with hypertension and its complications. Dysbiosis can lead to the production of metabolites that promote inflammation and oxidative stress, leading to vascular dysfunction and fibrosis [5].

Conclusion

Fibrosis and inflammation are two critical processes that contribute to the development and progression of arterial hypertension. They are interrelated and can promote each other, leading to a vicious cycle that further worsens blood pressure control. Understanding the mechanisms behind these processes and identifying targets for their modulation could

***Correspondence to:** Belge Hoch. Department of Cardiology, Centre of Postgraduate Medical Education, Warsaw, Poland, E-mail: Belge@cmkp.edu.pl

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lead to the development of novel therapies for hypertension and its associated complications. Therefore, interventions targeting the gut microbiome, such as probiotics or prebiotics, could be a promising approach to modulate fibrosis and inflammation in hypertension. Finally, lifestyle interventions, such as diet and exercise, can also modulate fibrosis and inflammation in hypertension.

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