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Short Communication

A Brief Note on Hypertension in Relation to Immune System

Jenny Svedenkrans*

Department of Medicine, University of Freiburg, Breisgau, Germany

ABOUT THE STUDY

High blood pressure affects more than one billion persons globally and is the most major modifiable risk factor for cardiovascular disease death. While several factors contribute to the pathogenesis of hypertension, the immune system's function has been thoroughly established by a vast number of studies from different laboratories around the world. Immunosuppressive medicines and individual cytokine suppression prevent or relieve experimental hypertension, and investigations in genetically engineered mouse strains have shown that lymphocytes play a role in the development of hypertension and hypertensive organ harm [1]. Additionally, in autoimmune illnesses, immunological reactivity may be the driving factor behind hypertension.

Activation of the innate and adaptive immune systems causes immune cell infiltration, oxidative stress, and stimulation of the intrarenal angiotensin system. High blood pressure is caused by a combination of inflammation-induced disruption in the pressure-natriuresis link, defective vascular relaxation, and sympathetic nervous system overactivity. The intensity of inflammation is largely determined by imbalances between proinflammatory effector responses and anti-inflammatory responses of regulatory T cells [2]. Autoantigens of clinical relevance have been discovered in animal and human investigations. Ongoing research into immunological reactivity in hypertension may lead to the development of new treatment techniques for the disease [3].

Hypertension is defined as blood pressure of 140/90 mmHg or higher, and it affects 25-43% of the world's population over the age of 18, making it the biggest modifiable risk factor for death from cardiovascular disease. High blood pressure can be caused by a variety of factors, but in the great majority of individuals, there is no known reason [4]. These patients are classified as having main or essential hypertension, which is uncontrolled in 8-12%of patients despite improved therapy alternatives.

Essential hypertension is caused by the interplay of environmental, genetic, anatomical, neurological, endocrine, humoral, and hemodynamic variables. These variables have been consolidated in the Page Mosaic theory, regarding which it has been noted that "its flaw is that it cannot be proved false." Immunology and autoimmunity do not appear in the octagonal Page Mosaic; nonetheless, Harrison has observed that inflammation exists upstream or downstream of each interrelated component [5].

The first studies on the involvement of immune cells in hypertension were conducted over 50 years ago, but research on immunity in the aetiology of hypertension remained scarce until 16-18 years ago, when a reawakened interest spurred an exponential increase in the number of publications in this field. This study discusses the research that has proven the immune system's crucial role in the pathophysiology of essential hypertension [6].

Three decades ago, studies found that the absolute number of CD4+ T cells was lowered after Angiotensin Converting Enzyme (ACE) inhibitor administration, implying that Renin-Angiotensin System (RAS) inhibition had an effect on cellular immunity. These discoveries were never pursued further. Recently, a connection between immune suppression and hypertension relief has been discovered. Untreated HIV-positive patients with chronically low numbers of CD4+ T cells have a lower frequency of systolic hypertension than treated HIV patients and uninfected control participants, according to data from the Multicenter AIDS Cohort research. Increased levels of expression of Intercellular Adhesion Molecule 1 (ICAM-1) and were linked to activated macrophages. These results revealed a link between CD8+T cell activation and impaired arterial relaxation, as well as a link between monocyte activation and immune cell vascular infiltration [7]. The importance of the immune system in human hypertension is reinforced by a study in which eight individuals with essential hypertension were given Mofetil Mycophenolate (MMF) for concomitant rheumatoid arthritis or psoriasis without changing their antihypertensive medication or diet. MMF therapy reduced blood pressure while also decreasing urine RANTES and Tumor Necrosis Factor (TNF-). When MMF was stopped, blood pressure reverted to pre-treatment levels, implying that the improvement in hypertension was due to immune suppression.

CONCLUSION

High blood pressure is the hemodynamic result of numerous causes, and there is now conclusive evidence that immunity plays a role in the pathophysiology of essential hypertension. The onset

Correspondence to: Jenny Svedenkrans, Department of Medicine, University of Freiburg, Breisgau, Germany, E-mail: Svedenkransjena@edu.gr.com

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and progression of hypertension may be viewed as a sequential process of immune system activation. This process is initiated by stimuli that activate innate immunity in tandem with an adaptive immune response to neoantigens. Chronic renal impairment and arteriosclerosis exacerbate hypertension and its implications.

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