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Mechanisms in Hypertension and Target Organ Damage

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Editorial

The heart, blood arteries, brain, kidney, retina, and other target organs are all affected by hypertension, which can be worsened by damage and metabolic abnormalities. It is a complex disease in which several immune cells and components have been implicated. Because the incidence and mortality rates of heart and cerebrovascular illness have increased, the ultimate goal of antihypertensive medication is to reduce the incidence and mortality rates of heart and cerebrovascular disease in patients with hypertension.

As a vital organ in T lymphocyte ontogenesis, the thymus has been demonstrated to be critical in optimising immune system function throughout life; thus, the pathological processes of high blood pressure are thought to be strongly linked to the thymus. According to studies, the thymus undergoes continual shrinkage or hypofunction as it ages. When Fukuda investigated age-related changes in haematological values, serum biochemical constituents, and weights of various organs in both genders of SHR/Izm, Stroke-prone SHR, and WKY/Izm rat strains, they found that the values of thymus weights were lower in Spontaneously hypertensive rats (SHR) than in Wistar Kyoto (WKY) rats. A previous study by Svendsen found that the salt-dependent phase of deoxycorticosterone acetate salt hypertension did not develop and the decreased perivascular in ltration of immune cells following renal infarction was not present in athymic 'nude' mice. However, if the thymus gland was transplanted into these athymic mice, then the capacity for developing salt-driven hypertension was restored.

The role of the thymus and the inammatory procedure T cell formation and maturation are known to be dependent on the thymus. T cells are created in the thymus, and T cells are subjected to positive and negative selection, resulting in a large functional MHC-restricted naive T cell receptor repertoire. T cells travel throughout diverse thymus microenvironments during development, where they interact with stromal cells to produce signals important for thymocyte survival, proliferation, differentiation, and selection. Helper T cells (Th), regulatory T cells (Tregs), and cytotoxic T cells can all be differentiated from naive T cells. Within the thymus, specific and complex processes are involved in the production and development of the specific T cell lineage, and various signalling pathways are engaged in these activities.

If thymocytes respond to these antigens spontaneously, they are subjected to negative selection, either through death or into Treg lineages. Tregs are now known to be created in vivo via two distinct routes. The thymus produces the bulk of functionally mature Treg cells, with certain clones deviating into the thymus-derived forkhead box (Fox)p3+ Treg cell lineage after recognising self-antigen. Interleukin (IL)-4, IL-17, and interferon (IFN) - are all produced by Th cells. Tregs can also produce the cytokine IL-10. The growth of activated B-cells and mast cells is regulated by IL-4. In the absence of vascular tissue, IL4 promotes the conversion of activated macrophages to M2 cells while inhibiting the activation of traditional activated macrophage M1 cells. If thymocytes respond to these antigens spontaneously, they are subjected to negative selection, either through death or into Treg lineages. Tregs are now known to be created in vivo via two distinct routes. The thymus produces the bulk of functionally mature Treg cells, with certain clones deviating into the thymus-derived forkhead box (Fox)p3+ Treg cell lineage after recognising self-antigen. Interleukin (IL)-4, IL-17, and interferon (IFN)- are all produced by Th cells. Tregs can also produce the cytokine IL-10. The growth of activated B-cells and mast cells is regulated by IL-4. In the absence of vascular tissue, IL4 promotes the conversion of activated macrophages to M2 cells while inhibiting the activation of traditional activated macrophage M1 cells.

Increased macrophage repair (M2), in combination with IL-10 and transforming growth factor (TGF)-secretion, reduces pathological inammation. The proinammatory response is induced and regulated by IL17. Other cytokines [IL-6, TGF-, tumour necrosis factor (TNF)-, granulocyte colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, granulocyte- The inammatory procedure involves all of these cytokines, chemokines, and immune cells. These factors and cytokines, on the other hand, aid in the inflamatory response. As a result, thymus function can have an impact on the inammatory response.

Low-grade inflammation has been shown to have a role in the pathophysiology of hypertension and is implicated in a number of mechanisms that increase blood pressure development. Endothelial damage and activation of the renin system can occur as a result of inflammatory stimuli, and studies have shown that activation of the intrarenal renin-angiotensin system (RAS) and endothelial dysfunction are significant in the development of hypertension. Endothelial dysfunction may be caused by nitric oxide (NO) and superoxide in hypertension, and the balance between the two may be more essential than the absolute amounts of either. Other cross-sectional studies have found a link between essential hypertension and C-reactive protein (CRP), TNF, and IL-6 [1-5].

Conflict of Interest

None

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