Inflammation, immunity, and hypertension / Arisya Agita, M Thaha Alsagaff

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Abstrak

ABSTRACT
The immune system, inflammation and hypertension are related to each other. Innate and adaptive immunity system triggers an inflammatory process, in which blood pressure may increase, stimulating organ damage. Cells in innate immune system produce ROS, such as superoxide and hydrogen peroxide, which aimed at killing pathogens. Long-term inflammation process increases ROS production, causing oxidative stress which leads to endothelial dysfunction. Endothelial function is to regulate blood vessel tone and structure. When inflammation lasts, NO bioavailability decreases, disrupting its main function as vasodilator, so that blood vessels relaxation and vasodilatation are absent. Effector T cells and regulatory lymphocytes, part of the adaptive immune system, plays role in blood vessels constriction in hypertension. Signals from central nervous system and APC activates effector T lymphocyte differentiation and accelerate through Th-1 and Th-17 phenotypes. Th-1 and Th-17 effectors participate in inflammation which leads to increased blood pressure. One part of CD4+ is the regulatory T cells (Tregs) that suppress immune response activation as they produce immunosuppressive cytokines, such as TGF-I and IL-10. Adoptive transfer of Tregs cells can reduce oxidative stress in blood vessels, endothelial dysfunction, infiltration of aortic macrophages and T cells as well as proinflammatory cytokine levels in plasma circulation.