Inflammation, immunity, and hypertension

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Abstrak

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.
.....Sistem imun, proses inflamasi serta hipertensi saling berkaitan. Sistem imun alamiah dan adaptif memicu proses inflamasi, mengakibatkan peningkatan tekanan darah yang merangsang kerusakan organ. Sel pada sistem imun alamiah memproduksi ROS seperti superoksida dan hidrogen peroksida yang bertujuan membunuh patogen. Proses inflamasi jangka-panjang meningkatkan produksi ROS, menyebabkan stress oksidatif, mengakibatkan disfungsi endotel. Endotel berfungsi mengatur tonus dan struktur pembuluh darah. Saat inflamasi, bioavaibilitas NO menurun, mengganggu fungsi utamanya, sehingga mencegah relaksasi dan vasodilatasi pembuluh darah. Sel T efektor dan limfosit regulatori, bagian dari sistem imun adaptif, berperan pada konstriksi pembuluh darah pada hipertensi. Sinyal dari sistem saraf pusat dan APC mengaktivasi diferensiasi limfosit T efektor, dan mempercepat melalui Th-1 dan fenotip Th-17. Efektor Th-1 dan Th-17 berpartisipasi dalam inflamasi, mengakibatkan peningkatan tekanan darah. Salah satu bagian CD4+ adalah regulatory T cells (Tregs) yang menekan aktivasi respons imun dengan memproduksi sitokin imunosupresif seperti TGF- dan IL-10. Transfer adoptif dari sel Tregs menurunkan stres oksidatif pada pembuluh darah, disfungsi endotel, infiltrasi dari makrofag aorta dan sel T serta kadar sitokin proinflamasi dalam sirkulasi plasma