Role of inflammation in hypertension

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Abstract
Even though the mechanisms that mediate essential hypertension (HT) are not fully understood, an immunological-inflammatory mechanism could be the common pathway for diverse pathophysiological mechanisms. We analyze in a simplified way the participation of the immune system in HT. T lymphocytes (TL) and antigen presenting cells (APCs) are components of the immune system capable of generating proinflammatory cytokines. They cause endothelial damage, vasoconstriction, and decreased urinary sodium excretion. CD4+ and CD8+ TL are effector cells, causally implicated in the development of HT, whereas type γδ TL play their pathogenic role in HT enhancing endothelial dysfunction. Additionally, a immunomodulation decrease by regulatory TL, worsens endothelial dysfunction and reduces vasodilation in experimental HT. Results of recent studies indicate that lymphocyte activation would be mediated by antigens captured by antigen APCs for subsequent presentation to "naive" TL. On the other hand, proinflammatory states such as obesity, the change of the intestinal microbiota and the increase in salt intake favors TL and APC activation, contributing to HT development.

Author keywords
Cytokines; Hypertension; Inflammation; Lymphocytes