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Deregulations in CD4+ T lymphocytes subsets promote inflammation in atrial fibrillation

Ingrid E Dumitriu
St George's University of London, UK

The precise role of inflammation in the development and perpetuation of atrial fibrillation (AF) is yet to be fully uncovered. T and B lymphocytes, the main cellular effectors of adaptive immunity, have pivotal roles in orchestrating inflammation. Different subsets of lymphocytes either promote or prevent inflammation. We are investigating a unique subset of lymphocytes, the $CD4^+CD28$(null) T cells that expand in patients with chronic inflammation. These cells secrete high levels of pro-inflammatory cytokines tumour necrosis factor-α (TNF-α) and interferon-γ (IFN-γ). The response of $CD4^+CD28$(null) T cells is normally maintained under control by regulatory T cells (Treg), a specialized subset of T lymphocytes with suppressive function that maintain immune homeostasis and prevent pathogenic immune responses. The role of $CD4^+CD28$(null) and Treg cells has not been investigated in AF. We found that $CD4^+CD28$(null) T lymphocytes were significantly increased in the circulation of AF patients compared to controls ($p<0.0001$). In addition, AF patients had a marked reduction ($p=0.0001$) in Treg cells. The ratio of $CD4^+CD28$(null) T lymphocytes to Tregs was significantly increased. In contrast, no alterations were identified in circulating B cell subsets. Levels of hsCRP, TNF-α and IFN-γ did not correlate with $CD4^+CD28$null T cell and Treg frequency. Instead, we demonstrate that the expansion of $CD4^+CD28$(null) T cells is caused by defects in apoptosis pathways and increased activation and proliferation in response to homeostatic cytokines. These novel results suggest an imbalance in the mechanisms that maintain homeostasis in the immune response, which may promote inflammation in patients with AF.

Biography

Ingrid E Dumitriu pursued her MD and a PhD Degree in Immunology from San Raffaele DIBIT Scientific Institute, Milan, Italy. She is a Reader (Associate Professor) in Cardiovascular Immunology at St George's, University of London, London UK. She leads the Cardiovascular Immunology Research Group. Her research focuses on the role of inflammation and immune cells in atherosclerosis and other cardiovascular diseases. She is a Nucleus Member of the European Society of Cardiology (ESC) Working Group on Atherosclerosis and Vascular Biology. She is also a Member of the ESC, European Atherosclerosis Society, British Society for Immunology, and ESC Working Group on Peripheral Circulation.

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