Inflammation and the pathogenesis of atrial fibrillation

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Although Atrial fibrillation (AF) is the most common cardiac arrhythmia, the development of preventative therapies for AF has been disappointing. The infiltration of immune cells and proteins that mediate the inflammatory response in cardiac tissue and circulatory processes is associated with AF. The presence of inflammation in the heart or systemic circulation can predict the onset of AF and recurrence in the general population, as well as in patients after cardiac surgery, cardioversion, and catheter ablation. Furthermore, mediators of the inflammatory response can alter atrial electrophysiology and structural substrates, leading to increased vulnerability to AF. Inflammation modulates calcium homeostasis and connexins, which are associated with triggers of AF and heterogeneous atrial conduction. Myolysis, cardiomyocyte apoptosis, and the activation of fibrotic pathways via fibroblasts, transforming growth factor-β and matrix metalloproteases are also mediated by inflammatory pathways, which can all contribute to atrial structural remodelling. A detrimental complication of AF, such as thromboembolism, is associated with inflammatory activity. Understanding the complex pathophysiological processes and dynamic changes of AF-associated inflammation helps to identify specific anti-inflammatory strategies for the prevention of AF.