

Cardiovascular Consequences of Autoimmune Disease Progression in Lupus and Rheumatoid Arthritis

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ABOUT THE STUDY

Autoimmune diseases such as lupus erythematosus and Rheumatoid Arthritis (RA) are known to affect multiple organ systems beyond their primary sites of inflammation. Among the most significant systemic consequences are Cardiovascular (CV) complications, which are increasingly recognized as critical aspects of these diseases. Both Systemic Lupus Erythematosus (SLE) and RA are associated with a heightened risk of cardiovascular morbidity and mortality.

Cardiovascular pathophysiology in lupus and rheumatoid arthritis

The underlying pathophysiological mechanisms linking autoimmune diseases with cardiovascular issues are multifaceted, involving inflammatory processes, autoantibody production, and alterations in lipid metabolism.

Inflammation and endothelial dysfunction in both lupus and RA, chronic systemic inflammation plays a pivotal role in the development of cardiovascular complications. Elevated levels of pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF- α), Interleukin-6 (IL-6), and Interleukin-1 beta (IL-1 β) contribute to endothelial cell dysfunction and promote atherosclerosis. In SLE, the presence of Antinuclear Antibodies (ANAs) and other autoantibodies can directly affect the vascular endothelium, leading to impaired nitric oxide production and increased oxidative stress.

Autoantibodies and direct vascular impact specific autoantibodies associated with lupus, such as anti-Phospholipid antibodies (aPLs), are known to be particularly damaging. These antibodies can increase the risk of atherosclerotic plaque formation and contribute to thrombotic events by promoting a hypercoagulable state. RA is also associated with autoantibodies like Rheumatoid Factor (RF) and Anti-Citrullinated Protein Antibodies (ACPAs), which can exacerbate systemic inflammation and contribute to CV complications through similar mechanisms.

Lipid dysregulation dyslipidemia is commonly seen in both conditions and is a critical risk factor for Cardiovascular Disease (CVD). Patients with SLE and RA often exhibit an altered lipid profile characterized by increased levels of triglycerides and Low-Density Lipoprotein (LDL) cholesterol, alongside reduced levels of High-Density Lipoprotein (HDL) cholesterol. These lipid abnormalities contribute to the development of plaque within the arteries and accelerate atherosclerosis.

Manifestations of cardiovascular complications

The cardiovascular complications that can arise from lupus and RA are diverse, ranging from subclinical changes to life-threatening events.

Atherosclerosis and Coronary Artery Disease (CAD) atherosclerosis is one of the most common cardiovascular manifestations in patients with autoimmune diseases. SLE and RA patients often present with premature CAD, which can lead to angina, myocardial infarction, and other serious cardiac events. Studies have shown that individuals with SLE are more likely to develop CAD at an earlier age compared to the general population, with mortality rates related to CAD significantly higher in SLE patients.

Pericarditis and myocarditis pericarditis, an inflammation of the pericardial sac surrounding the heart, is commonly seen in lupus and, to a lesser extent, in RA. In lupus, it can occur due to both autoimmune activity and the deposition of immune complexes. Myocarditis, which refers to inflammation of the heart muscle itself, can lead to reduced cardiac function and may contribute to heart failure if left untreated.

Heart failure and cardiomyopathy chronic inflammation in SLE and RA can lead to the development of cardiomyopathy, which may eventually progress to heart failure. This is often due to a combination of direct myocardial inflammation and secondary effects, such as damage to cardiac vessels or valvular disease. SLE patients, especially those with a history of nephritis or coexisting hypertension, are at a higher risk for developing heart failure.

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Stroke and Peripheral Artery Disease (PAD) autoimmune diseases also elevate the risk of cerebrovascular events. The presence of aPLs in lupus patients is strongly associated with an increased risk of ischemic stroke. RA patients are more prone to PAD due to chronic inflammation, which contributes to arterial stiffness and reduced blood flow, potentially leading to claudication or critical limb ischemia.

Risk factors and predisposing conditions

Several factors exacerbate cardiovascular risks in lupus and RA patients. Genetic predisposition plays a significant role, with certain Human Leukocyte Antigen (HLA) types linked to both autoimmune disease development and increased cardiovascular risk. Additionally, common comorbidities such as hypertension, diabetes, and obesity compound the risk.

Monitoring and management strategies

Given the significant cardiovascular risk in lupus and RA patients, early detection and comprehensive management are paramount. Routine screening for CV risk factors, including

blood pressure, lipid profiles, and markers of inflammation, should be incorporated into standard care for these populations.

Lifestyle modifications patients should be encouraged to adopt lifestyle changes that include a heart-healthy diet, regular physical activity, and smoking cessation. Diets rich in omega-3 fatty acids, fruits, and vegetables can help counteract inflammatory processes and improve lipid profiles.

Pharmacologic interventions treatment of underlying inflammation is need for reducing cardiovascular risk. The use of Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), Disease-Modifying Antirheumatic Drugs (DMARDs), and biologics can control systemic inflammation and lower the risk of CV events. Statin therapy is recommended to manage dyslipidemia, and anticoagulant therapy may be considered for patients with high aPL titers to mitigate thrombotic risks.

The cardiovascular consequences of autoimmune diseases like lupus and RA are profound, contributing to increased morbidity and mortality. Understanding the complex interplay of inflammation, autoimmunity, and metabolic abnormalities is key to improving patient outcomes.