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Commentary

Cardiovascular Disease Risk in Women with Chronic Rheumatic Diseases

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DESCRIPTION

The immune system created to identify foreign antigens and damage-related molecular patterns in to protect the body against invasive pathogens. The heart frequently interacts with immune cells that might be directed during chronic inflammatory conditions like autoimmune disorders to its strong vascular circulation and lymphatic network. Cardiovascular Disease (CVD) risk is inversely correlated with the severity of chronic rheumatologic illnesses and is increased. This connection between autoimmune illness and CVD may be influenced by a number of factors. Initially, the adaptive immune cells cause damage to the heart microvasculature by directing them to self-antigens on vascular endothelial and smooth muscle cells. Autoantibodies can potentially stimulate the clotting system, resulting in thrombosis in the coronary arteries and ensuing ischemia. Inflammatory cytokines set up the innate and adaptive immune systems' cells to target cardiac cells with their cytotoxic actions, leading to vascular, myocardial, and valvular dysfunction. The overexpression of certain cells in some autoimmune illnesses also causes persistent pericardial, endocardial, and myocardial inflammation.

Immune function is impacted by sex hormone changes brought on by menopause, pregnancy, and estrous cycle variations. While sex hormones probably play a part, women are also more vulnerable to social and psychological stressors, which have a negative impact on immune function and inflammation. As a result, there is a complex interaction between an individual's stress susceptibility, hormonal microenvironment and hereditary and predisposing variables that influence autoimmune disease severity and impact on their cardiovascular system in a specific female. Various processes at work that could interact with conventional CVD risk factors most cardiac structures including the conduction system, valves, myocardial, coronary vasculature, pericardium, endocardium, and periadventitial tissues, may be impacted by chronic autoimmune rheumatologic diseases. Rheumatologic disease patients had accelerated atherosclerosis, which is not entirely explained by conventional risk factors. The specific mechanism for this early and aggressive coronary and other major artery atherosclerosis is unknown although it is thought to be connected to the persistent systemic inflammation that occurs with these illnesses. Additionally, many women lack obstructive CAD, but those with autoimmune diseases may develop angina owing to heart ischemia due to processes such as abnormal micro vascular vasoreactivity, impaired vasodilation, capillary rarefaction, and/or heightened prothrombic activity.

Circulating immune complexes and autoantibodies in Systemic Lupus Erythematosus (SLE) can harm the myocardial, endocardium, micro vascular and epicardial coronary arteries, cardiac valves, and pericardium. SLE prefers female patients. Women with SLE suffer chest pain caused in addition to obstructive atherosclerosis CAD but also by micro vascular dysfunction/disease, with stress imaging investigations revealing reduced vasodilator reserve. Patients with SLE have frequently been reported to have valvular heart disease. Up to 25% of SLE patients have been reported to have endocardial lesions such as widespread valvular thickening, valvular nodules, marantic vegetations, and concomitant valvular stenosis or regurgitation. These patients are more likely to have anticardiolipin antibodies. Valve thickness was more common than valve malfunction, but both were associated with increased SLE activity and advanced age. Patient with SLE, especially those with renal dysfunction are more likely to develop hypertension. Moreover, they are more likely to experience heart failure than the general population, with almost similar chances of experiencing Heart Failure with preserved Ejection Fraction (HFpEF) and Heart Failure with reduced Ejection Fraction (HFrEF). C-Reactive Protein (CRP) levels show a clear correlation between the inflammatory burden and the risk of developing heart failure.

SLE patients were found to have an enhanced risk of heart failure mortality as well as an increased risk of other adverse cardiac events such as atrial fibrillation, stroke, and venous thromboembolism when compared to control subjects. Autoantibodies that predominantly target joint synovium are the hallmark of RA, a chronic inflammatory condition that can also, to a lesser extent, damage other non-articular organs through a similar collection of immune mediators. The cumulative effects of RA and flares both raise the risk of CVD. Independent of other risk variables, women with RA had a 45% higher chance of

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dying from CVD than women without RA. Inflammation in RA has been demonstrated to increase arterial stiffness, promote endothelial dysfunction, impact lipid metabolism, and destabilize atherosclerotic plaque exposing patients to plaque rupture/erosion and MI. Even after accounting for CVD risk factors, numerous investigations have revealed that individuals with RA had an increased risk of heart failure compared to those without the disease. Psoriasis is a chronic inflammatory disorder that frequently results in different skin lesions due to an increase in epidermal replacement. It is regulated by a complex interplay of cytokines, chemokines, T cells, dendritic cells, and perhaps skin bacteria. Psoriasis is caused by a chronic immune-mediated inflammation that can potentially have an impact on other organ systems. Research have indicated that those with severe psoriasis and psoriatic arthritis had a higher chance of dying from CVDs such as MI and stroke. Chronic systemic autoimmune diseases are significant risk factors that greatly affect women in addition to conventional CVD risk factors. Atherosclerosis, heart failure, systemic endothelial and micro vascular dysfunction, and thrombosis are all predisposed by underlying autoimmune dysfunction and inflammation. Comorbid disorders, the length of an autoimmune condition, the severity of the disease, and the suppression of underlying inflammation are all factors that affect the risk of CVD. The incidence of CVD in people with underlying autoimmune diseases may be reduced by early detection and screening for CVD risk factors in this population. The advancement of care for women will benefit from multidisciplinary, team-based care, drug testing, and productive team investigations concentrating on systemic autoimmune disorders.