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Risk of Cardiovascular Illness in Women with Chronic Rheumatic Conditions

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Introduction

The immune system was designed to recognize foreign antigens and damage-associated molecular patterns in order to defend the body from pathogens. The heart frequently comes into contact with immune cells that can be misdirected in chronic inflammatory states like autoimmune diseases because of its robust vascular circulation and lymphatic network. The risk of cardiovascular disease (CVD) is inversely proportional to the severity of chronic rheumatologic disorders. This connection between autoimmune disease and CVD may be caused by a number of different factors. First, the adaptive immune cells are directed to harm the cardiac microvasculature by antibodies to self-antigens on vessel endothelial and smooth muscle cells. Autoantibodies can likewise prompt enactment of the coagulation framework, finishing in apoplexy inside coronary vessels with resulting ischemia. Fiery cytokines make preparations of the inborn and versatile insusceptible frameworks to coordinate their cytotoxic exercises towards cardiovascular cells, prompting vascular, myocardial, and valvular brokenness. In conclusion, a few immune system sicknesses are described by the overactivity of explicit cells prompting constant pericardial, endocardial, and myocardial irritation [1].

The majority of autoimmune diseases are more common in women than in men and significantly impacted. Immune function is affected by changes in sex hormones caused by menopause, pregnancy, and estrous cycles. Women are also more likely to be exposed to psychological and social stressors, which can have a negative impact on immune function and inflammation. As a result, the severity and impact of an autoimmune disease on a female's cardiovascular system are determined by a complex interaction between the individual's susceptibility to stress, hormonal milieu, environment, and genetic and predisposing factors. With multiple mechanisms operating, some of which may interact with conventional CVD risk factors, the reconnaissance and treatment of patients with rheumatologic immune system conditions merit exceptional consideration [2].

Description

Most cardiac structures, including the conduction system, valves, myocardium, coronary vasculature, pericardium, endocardium, and periadventitial tissues, can be affected by chronic autoimmune rheumatologic disorders. Traditional risk factors cannot fully account for the accelerated atherosclerosis seen in rheumatologic conditions. Although the exact cause of this early and aggressive coronary and other major vessel atherosclerosis is unknown, it is thought to be connected to the chronic systemic inflammation that goes along with these conditions. In addition, many women do not have accelerated coronary artery disease (CAD), but those with autoimmune conditions may experience angina as a result of cardiac ischemia caused by mechanisms like abnormal microvascular vasoreactivity, impaired vasodilation, capillary rarefaction, and/or heightened prothrombic activity [3].

Customary gamble evaluations, for example, the Framingham risk score don't completely represent these fiery problems. In fact, in patients with rheumatoid arthritis (RA), the Framingham risk score underestimates cardiovascular risk by 65% in men and 103% in women. The AHA/ACC rules on CVD counteraction consider rheumatologic messes as a "risk enhancer." Despite this, only 41% of RA patients with known coronary calcifications were identified by this risk assessment tool as being at high risk for coronary atherosclerosis. Patients with rheumatologic disorders frequently exhibit arrhythmias and abnormalities in the conduction system. These conditions can be the initial sign of cardiac disease and, in some cases, may be the cause of sudden cardiac death. Myocardial and pericardial contribution might be available in up to 50 % of patients with rheumatologic messes. For patients with rheumatologic disorders to receive adequate screening and prompt detection of cardiac complications, it is essential to have a greater awareness of these cardiac pathologies [4].

The myocardium, endocardium, microvascular and epicardial coronary arteries, cardiac valves, and the pericardium can be affected by circulating immune complexes and autoantibodies in SLE. With a female-to-male ratio of 9:1, SLE prefers women. Black, Asian, Native American, and Hispanic women are two to three times more likely than White women to develop SLE. There is also a racial and ethnic predisposition. Myocardial infarction (MI), which is the leading cause of death in SLE, has been linked to higher rates of SLE. SLE-positive premenopausal women (those between the ages of 35 and 44) were found to have a 50-fold higher risk of MI than age-matched controls. Studies demonstrating impaired vasodilator reserve on stress imaging indicate that women with SLE frequently report experiencing chest pain not only as a result of obstructive atherosclerosis CAD but also as a result of microvascular dysfunction or disease [5].

Patients with SLE have frequently been described as having valvular heart disease. Diffuse valvular thickening, valvular nodules, marantic vegetations, and associated valvular stenosis or regurgitation are examples of these endocardial lesions, which have been reported in up to 25% of SLE patients, particularly those with anticardiolipin antibodies. Valve dysfunction was less common than valve thickening; However, both were associated with increased SLE activity and older age. Up to 10% of SLE patients may have arrhythmias like sinus tachycardia, atrial fibrillation, and atrial ectopy. Myocardial fibrosis, inflammation, autoantibody infiltration into the myocardium and conduction tissue, and myocardial ischemia are underlying mechanisms that cause arrhythmias and conduction disorders. Cardiometabolic illnesses like corpulence and prediabetes are common in numerous immune system conditions. In light of the increased risk of cardiovascular disease (CVD), it is essential to identify and treat CVD risk factors like hypertension and hyperlipidemia, promote healthy weight and dietary choices, and offer advice on quitting smoking. [2].

Although pain may prevent patients from engaging in physical activity, it is essential to encourage as much regular physical activity as is tolerated, such as swimming, resistance training, walking, and low-impact exercises. The coronary calcium score can be used to discuss primary prevention strategies like statins and is a powerful risk marker and predictor of future CVD risk. Patients with RA had a higher prevalence of coronary artery calcification in a

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large registry compared to non-RA patients (n=45,815) who were evaluated for suspected CAD with coronary CT angiography. The burden of coronary artery calcification is also higher in SLE patients than in non-SLE patients, even at younger ages. While stable coronary calcification inside the media is a marker of hazard, it is the unsteady, incendiary plaques with lipid-rich center and miniature calcifications that are inclined to burst and prompt occasions. Non-invasive imaging of plaque activity and advanced imaging techniques like optical coherence tomography may help women with autoimmune conditions be better stratified for risk when investigating the characterization of plaques and the type of calcification they contain [3].

For instance, if intra-plaque calcifications at higher risk are identified, treatment strategies may be implemented more aggressively. Testing such as echocardiography and the coronary artery calcium score may be considered appropriate in the early detection and treatment of CVD risk factors in women with autoimmune conditions. Refreshed proposals for essential counteraction of CVD in ladies have been as of late distributed to direct clinical consideration. Multiple CVD trials have looked into targeting inflammation, with varying results. The Canakinumab Calming Apoplexy Result Study (CANTOS) showed that Canakinumab (150 mg at regular intervals), an immunizer focusing on IL-1β, decreased the paces of repetitive CVD occasions among patients with history myocardial dead tissue and raised provocative markers. Colchicine, a relatively inexpensive and widely used medication for the treatment of gout and pericarditis, has also been studied. In patients who had recently suffered a myocardial infarction, the Colchicine Cardiovascular Outcomes Trial (COLCOT) demonstrated that this medication was effective in preventing major adverse cardiac events [3].

Colchicine was effective in reducing cardiovascular events in patients with stable CAD. While observational examinations have shown that methotrexate is related with decreased hazard of CVD, the Cardiovascular Irritation Decrease Preliminary (CIRT) showed no advantage of low portion methotrexate in lessening occasions in patients with earlier myocardial dead tissue. Immune system issues fundamentally restricted to the joints, like RA, are regularly all around endured during pregnancy without critical ramifications for mother or child. But conditions like SLE and APLS, which are marked by widespread inflammation and autoantibodies, can harm the mother and fetus. Miscarriage, gestational diabetes (especially if systemic steroids are needed), preterm birth, fetal growth restriction, and hypertensive disorders of pregnancy can all occur during pregnancy in SLE. Women with lupus nephritis, anti-Ro/SSA or anti-La/SSB antibodies, or APLS positivity are more likely to experience pregnancy complications [4].

Complication risk is correlated with disease activity in the year prior to conception. Underlying cardiovascular risk factors like diabetes, hypertension, or obesity further exacerbate pregnancy risk. In addition to increasing the mother's risk of hypertensive disorders and cardiovascular disease (CVD) postpartum, hypertension during pregnancy is linked to cardiac structural changes, adverse cardiometabolic risk, and increased CVD risk in the offspring. For instance, the Nord-Trndelag Health (HUNT) Study demonstrated that, in comparison to children of normotensive pregnancies, those of hypertensive pregnancies had higher blood pressures in young adulthoo. Women with autoimmune disorders complicated by pulmonary arterial hypertension, advanced heart failure, severe restrictive lung disease, or chronic kidney disease (Cr >2.8 mg/dL) are considered unfit to become pregnant. During pregnancy, low molecular weight heparin and low dose aspirin are typically used to treat APLS patients to improve pregnancy outcomes [5].

Conclusion

Chronic systemic autoimmune disorders are significant risk enhancers that significantly affect women, in addition to conventional CVD risk factors. Systemic endothelial and microvascular dysfunction, thrombosis, and premature and accelerated atherosclerosis are all predisposed by underlying autoimmune dysfunction and inflammation. CVD risk is influenced by the presence of comorbid conditions, the duration of an autoimmune condition, the severity of the disease, and suppressive treatment of underlying inflammation. In people with autoimmune conditions, early detection and screening for CVD risk factors may reduce the risk of cardiovascular disease in this group. Women's care will benefit from multidisciplinary, team-based care, clinical trials, and collaborative team-science studies on systemic autoimmune conditions.

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