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Perspective

Symptoms of Systemic Lupus Erythematosus Disease in the Heart

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DESCRIPTION

Chronic autoimmune disease Systemic Lupus Erythematosus (SLE) is defined by the existence of antibodies that are directed against several body antigens and may cause a wide range of non-specific clinical symptoms. It has a clear link with multiple immune response genes and is more common in females. It is characterised by various autoantibodies, including those to native DNA, and by both humoral and cellular immunologic abnormalities, including the generation of autoantibodies to cell nucleus components. Most SLE patients experience cardiovascular symptoms at some point over the course of their illness, with acute fibrinous pericarditis and pericardial effusion being the most frequent [1]. Even in people with limited symptoms, echocardiography has shown an increased frequency of pericardial effusion.

A variety of cardiac symptoms, such as pericarditis, myocarditis, valvular disease, atherosclerosis, thrombosis, and arrhythmias, might be present in SLE patients. In SLE, heart disease is linked to higher morbidity and mortality rates. The most frequent cardiac abnormality in people with Systemic Lupus Erythematosus (SLE) is pericarditis; however valve lesions, myocardial lesions, and coronary channel lesions can also happen. In the past, cardiac symptoms were serious, life-threatening, and frequently fatal [2]. As a result, they were frequently discovered during post-mortem investigations. These days, cardiac symptoms are frequently non-symptomatic and modest. But echocardiography and other noninvasive exams like cardiac computed tomography or heart magnetic resonance imaging can often detect them.

Positive anti Ro/SS-A, anti La/SS-B, anti-cardiolipin (aCL), and anti-double-stranded DNA tests are associated with several cardiovascular problems (anti-dsDNA). The pathogenic pathways behind various SLE cardiac characteristics, including various valve affections, have not yet been adequately explained by the majority of these antibodies. ACL antibodies, disease activity, or illness duration were not consistently linked to endothelial dysfunction in early SLE cases without CVDs, but rather to renal disease, diastolic hypertension, and diabetes in SLE [3].

As the disease advances, atherosclerotic plaque formation in the carotid arteries is more likely to occur in SLE patients, which increases the risk of cardiovascular events.

Rarely do heart symptoms appear as the first signs of lupus, and cardiac conditions may go for a long time without showing any symptoms. However, lupus-related heart problems can be very dangerous [4]. The importance of understanding the pathophysiology of cardiovascular problems cannot be overstated.

The third main cause of death in SLE is cardiovascular and cerebrovascular events, which are disproportionately correlated with age and gender. Cardiovascular illness, however, has a substantial correlation with disability and contributes to early mortality as well.

Young adults have been known to experience angina pectoris, myocardial infarction, and/or severe coronary atherosclerosis, particularly when risk factors including hypertension and hyperlipidemia developed after extended corticosteroid medication. Very rarely, coronary arteritis may cause symptoms similar to these. The prognosis for congestive heart failure, whether it has a single or numerous etiologies, is grim [5].

The mainstay of treatment for pericarditis, myocarditis, vasculitis, and coronary arteritis continues to be corticosteroid therapy. Nonsteroidal anti-inflammatory drugs (NSAIDs), antiarrhythmic medications, antihypertensive medications, vasodilator medications, intensive endocarditis prophylaxis, and sporadically intracardiac pacing when warranted also play a significant role in the overall management of cardiovascular manifestations of SLE [6].

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