# 全身性紅斑狼瘡的心血管合併症

# Cardiovascular complications in SLE

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Earlier-onset cardiovascular disease (CVD) is a substantial contributor to morbidity and mortality in patients with systemic lupus erythematosus (SLE). The traditional Framingham cardiac risk factors, such as lipid dysregulation, aging and smoking, do not fully explain this increased risk of CVD, strongly suggesting that autoimmunity contributes to accelerated atherosclerosis.

SLE-specific risk factors for accelerated atherosclerosis exist, but are poorly understood. Patients with SLE have high levels of circulating apoptotic ECs—indicative of increased vascular damage—and lower levels of circulating endothelial progenitor cells (EPCs) that are involved in the repair of damaged arterial tissues. Immune system dysfunction in SLE—including increased IFN-α production and NETosis—could potentiate vascular damage and prevent repair processes. Little agreement has been reached on whether the presence of antiphospholipid antibodies correlates with accelerated atherosclerosis. The presence of anti-oxidized LDL antibodies increases the risk of atherosclerosis in humans with SLE. Anti-apolipoprotein A-I antibodies have a role in atherosclerosis development in nonautoimmune patients. The presence of autoantibodies targeting apoA-I and HDL correlated with increased SLE disease activity. Associations between SLE disease manifestations (disease activity, duration, and damage) and atherosclerosis are not clear at present. HDL function is proinflammatory (piHDL) in many women with SLE. Oxidative stress associates with accelerated atherosclerosis in the general population, and increased oxidative stress has also been identified in patients with SLE, and is often elevated independent of disease activity. Some studies have shown that elevated homocysteine levels in patients with SLE correlated with cross-sectional and longitudinal progression of subclinical atherosclerosis. Leptin signaling contributes to atherosclerotic progression, and elevated leptin levels have been observed in adult and pediatric patients with SLE. Adiponectin levels are reduced in patients with CVD, but data linking adiponectin concentrations and SLE-associated atherosclerosis are limited and contradictory. In addition, multiple SLE therapeutics seem to affect the development and progression of atherosclerosis both positively and negatively.

Identification of SLE-specific mechanisms of, and biomarkers, for accelerated atherosclerosis should lead to the development of novel screening protocols for early detection of CVD and discovery of new therapeutic targets.