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Perspective

Evaluating Proximal Aortic Stiffness in Children and Adolescents with Active Systemic Lupus Erythematosus

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ABOUT THE STUDY

Individuals with Systemic Lupus Erythematous (SLE) are predisposed to early atherosclerosis, and the risk of atherosclerosis-related myocardial infarction is up to 50 times higher in young SLE patients compared to age-matched controls. A high prevalence of conventional risk factors, long-term corticosteroid use, the presence of antiphospholipid antibodies, and proatherogenic pathophysiologic phenomena inherent to SLE, such as dyslipidemia, immune system activation, endothelial cell apoptosis, oxidative stress, and chronic immune complex formation, all of which result in a chronic low grade inflammatory state, endothelial dysfunction

SLE patients should thus be considered a group at risk for the development of coronary artery disease, comparable to individuals with diabetes, in which risk factors should be identified and treated promptly.

Increased arterial stiffness, as demonstrated by increased Pulse Wave Velocity (PWV) in the proximal aorta, is one of the recently approved ultrasound-derived indicators of early, asymptomatic atherosclerosis. PWV can be thought of as an indirect assessment of the aorta's biophysical qualities. The larger the proportion of stroke volume that can be absorbed in systole and released into the peripheral circuit in diastole, the more compliant the central arteries are. The stiffer the artery, the less it absorbs, and more of the stroke volume travels down the vascular system at a faster PWV.

The current article aimed to measure proximal aortic stiffness in adolescents with SLE, its relationship to disease activity, and whether it can be used as an indicator of subclinical atherosclerosis in this patient population.

Accelerated atherosclerosis is a significant source of morbidity and death in SLE patients, emphasizing the significance of identifying and treating modifiable risk factors. The proximal aorta's Pulsed Wave Velocity (PWV) is a relatively recent method for assessing arterial stiffness. Researchers discovered that patients with active SLE had considerably greater PWV than controls. PWV was similarly raised in this research of older SLE patients, and variables related with increased PWV were illness severity in premenopausal women and age in postmenopausal women.

Although both studies link atherosclerosis to higher PWV, the specific source of this increased arterial stiffness in SLE is unknown. Although arterial stiffness may be an early event in the development of atherosclerosis, other processes, particularly immunological pathways, may be implicated in the pathophysiology of arterial stiffening in SLE patients.

Extended durations of immune complex-mediated symptoms, such as vasculitis and glomerulonephritis, combined with hypocomplementemia predispose SLE patients to arterial stiffness. Prospective longitudinal studies in SLE patients during activity and quiescence would help clarify whether higher PWV during activity is a measure of disease activity or may be utilized as a very early marker of atherosclerosis in this patient population.

This is similar to the argument made for traditional laboratory risk indicators such as C-Reactive Protein (CRP), which similarly fail to detect premature atherosclerosis in SLE patients since they may be increased owing to the illness or infective consequences.

Increased PWV cannot thus be utilized as a certain early predictor of atherosclerosis in this young patient population; instead, it most likely represents arterial inflammation, which can lead to atherosclerosis. The small sample size and lack of lipid profile tests in our patients limit the scope of this investigation.

CONCLUSION

SLE disease activity in children and adolescents is related with increased PWV and proximal aortic arterial stiffness. This suggests that illness-induced inflammation, rather than early atherosclerosis, is the source of increased arterial stiffness in children and adolescents with SLE disease activity. Further longitudinal research is needed to evaluate whether increased PWV and arterial stiffness in SLE are reversible with disease activity reduction, and whether/when elevated PWV may be employed as a marker of early atherosclerosis in this patient population.

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