Arch Cardiovasc Imaging. 2015 May; 3(2): e28299. DOI: 10.5812/acvi.28299

Published online 2015 May 27. **Letter**

Latent Ventricular Dysfunction in Systemic Lupus Erythematosus

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Received: April 16, 2015; **Revised:** May 16, 2015; **Accepted:** May 23, 2015

Keywords: Stress Echocardiography; Right Ventricular Systolic; Diastolic Dysfunction; Lupus Erythematosus

Dear Editor,

Cardiovascular involvement increases the morbidity and mortality rates in patients with systemic lupus erythematosus (SLE). Subtle myocardial dysfunction is far more common than overt clinical involvement. Recently, for an earlier detection of ventricular dysfunction in SLE patients, newer echocardiographic techniques such as two-dimensional and three-dimensional speckle tracking and stress echocardiography have been suggested by some investigators such as Poorzand et al. that can uncover stress-induced left and right ventricular dysfunction and pulmonary hypertension (1).

Of course, stress-induced reduction in the left ventricular (LV) ejection fraction is generally used to detect and evaluate coronary artery disease (2). However, this report of an SLE patient not only proves that significant right ventricular (RV) dysfunction may also happen, but also studies its implications in terms of specific conditions.

This report describes a 45-year-old woman with a confirmed diagnosis of SLE who was referred to our heart center for evaluation with exercise stress echocardiography due to atypical chest pain, fatigue, and dyspnea (functional class II). The patient had no known history of coronary artery disease, arrhythmia, any type of cardiomyopathy, or central nervous system disease. On examination, she had blood pressure of 110/60 mmHg in the supine position, oxygen saturation of 96%, and heart rate of 78 beats/minutes and regular. Electrocardiography showed normal findings. Transthoracic echocardiography (TTE) demonstrated normal size of the four chambers, normal function of both ventricles, mild regurgitation of the mitral and tricuspid valves, and pulmonary artery pressure (PAP) of 30 mmHg at rest.

The peak of the early (E) and late (A) diastolic velocities and also tissue Doppler velocities were measured for both LV and RV. In addition, myocardial systolic and early diastolic peak velocities (Em) and the mitral and tricuspid E/Em ratio were calculated. After TTE, the patient underwent exercise stress echocardiography and achieved 80% of the maximal predicted heart rate. The test was terminated due to severe fatigue and dyspnea. TTE views, obtained immediately after the exercise test, showed a normal LV size and function without any regional wall motion abnormality but with moderate RV systolic dysfunction, RV enlargement (3.9 cm), and severe RV diastolic dysfunction (E/Em = 9, E/A = 2.4, and E deceleration time = 110 ms), interestingly, in addition to a drastic rise in PAP (PAP = 65 mmHg).

This patient underwent left and right heart catheterization 24 hours later; the results revealed normal coronary arteries, RV, right atrium, and PAP at rest (PAP = 30 mmHg).

Pulmonary artery hypertension (PAH) is a severe, theoretically serious, complication of connective tissue disorders, such as SLE, systemic sclerosis, and to a lesser degree, rheumatoid arthritis and polymyositis. The pathophysiological mechanisms resulting in PAH, followed by RV dysfunction, are very similar in these disorders. Remarkably, Alkotob et al. showed that stress-induced PAH was very prevalent in their patients with scleroderma insofar as 46% were distinguished by an abnormal increase in their PAP (PAP > 40 mmHg) during exercise (3). Accordingly, our report interestingly suggests that the isolated systolic and diastolic dysfunction of the right heart can be a sign of stress-induced PAH and that Doppler tissue imaging seems to be a promising new technique to rule in/out the suspicion of stress-induced significant PAH with concomitant ventricular failure in patients with connective tissue disorders when the noninvasively estimated PAP and ventricular function at rest are normal (4).

Elevated pulmonary and ventricular pressure is not the only reason for RV dysfunction in patients with connective tissue disorders, and the disturbance and disruption

of the myocardial microcirculation and also interstitial fibrosis as the hallmarks of the primary myocardial involvement can also impair the function of the subendocardial fibers. Nevertheless, in our SLE patient, a rise in PAP was the main reason for the RV enlargement with significant systolic and diastolic dysfunction (5).

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