

HEART A'FLUTTER: CARDIAC INVOLVEMENT IN LÖFGREN'S SYNDROME WITHOUT CARDIAC SARCOIDOSIS

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Abstract:

Löfgren's Syndrome is the acute presentation of pulmonary sarcoidosis, defined by the clinical triad of migratory polyarthralgia, erythema nodosum, and hilar adenopathy. It is estimated that clinically apparent cardiac involvement occurs in 5% of patients with sarcoidosis, although a higher proportion (20-25%) may have clinically silent disease.

A 56-year-old woman with a history of hypertension, hypothyroidism, obesity, and fibromyalgia presented to her primary care physician with tender, erythematous, raised nodules on her elbows and ankles, bilateral ankle swelling, fever, a dry cough, and dyspnea with minimal exertion. A biopsy of her nodular rash was consistent with erythema nodosum and chest CT demonstrated bilateral hilar lymphadenopathy, confirming a clinical diagnosis of Löfgren's Syndrome. EKG revealed normal sinus rhythm with one atrial premature contraction. Symptoms were not relieved with supportive care and NSAIDs, so prednisone was prescribed.

The patient subsequently developed worsening dyspnea, diaphoresis, and palpitations. EKG upon presentation to the Emergency Department revealed typical atrial flutter with 2:1 atrioventricular conduction. Transthoracic echocardiogram was notable for hyperdynamic left ventricular function without wall motion abnormalities, normal right ventricular size and function, and normal left and right atrial size. She was monitored on telemetry and did not have any ventricular arrhythmias or bradyarrhythmias. She received oral and IV diltiazem and metoprolol for rate control and apixaban for thromboembolic risk reduction. The patient subsequently underwent successful transesophageal echocardiogram-guided cardioversion and cavotricuspid isthmus ablation for typical atrial flutter. Due to concern for possible cardiac sarcoidosis, she underwent hybrid imaging with single-photon emission computerized tomography (SPECT) and ¹⁸F-fluorodeoxyglucose (¹⁸F-FDG) positron emission tomography (PET). SPECT imaging revealed normal myocardial perfusion with no evidence of scar. PET imaging showed normal blood pool uptake of FDG consistent with the absence of active inflammatory myocardial sarcoidosis.

While conduction abnormalities, arrhythmias, and heart failure are common manifestations of cardiac sarcoidosis (CS),⁴ little has been documented about cardiac involvement during the acute inflammatory phase of *extracardiac* sarcoidosis. Our patient had atrial flutter with no evidence of CS on hybrid SPECT/FGD-PET cardiac imaging, an imaging modality with high diagnostic accuracy for CS.⁵ Her atrial flutter may have been due to pulmonary involvement, corticosteroid-induced arrhythmia,⁶ or possibly, an early sign of CS.

Early recognition of CS in patients with systemic sarcoidosis is crucial. In general, patients with sarcoidosis carry a higher risk of adverse cardiac outcomes compared with the background population.⁷ While there remains controversy and uncertainty regarding screening for CS in patients with extracardiac sarcoidosis, it is critically important that patients such as ours be vigilantly followed for signs of progressive cardiac involvement, especially given that sudden cardiac death was found to be the presenting manifestation in as many as 14% of new cases of CS.⁸