Multimodality Imaging to Diagnose Isolated Cardiac Sarcoidosis and Determine Regional Inflammatory Activity Levels

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INTRODUCTION

Cardiac sarcoidosis is one of the uncommon causes of cardiac failure and is associated with poor prognosis if untreated.[1,2] Early diagnosis of cardiac sarcoidosis is of great clinical importance, as corticosteroid therapy can provide symptomatic relief and improve the long-term prognosis for patients with cardiac sarcoidosis.[3] There is increasing recognition of isolated cardiac sarcoidosis in recent years, although its diagnosis remains challenging.[1] Here we report the case of a 71-year-old woman with isolated cardiac sarcoidosis using a noninvasive diagnostic approach and discuss the role of current imaging modalities in the diagnostic algorithm for cardiac sarcoidosis and its therapeutic approaches.

CASE PRESENTATION

A 71-year-old woman presented with a 2-day history of nonradiating dull chest pain on a background of exertional dyspnea (New York Heart Association class II) and bilateral edema of the lower extremities over the past few months. She had a history of factor V Leiden with a pulmonary embolus and deep venous thrombosis, as well as asthma, hypertension, and hypothyroidism, but no history of cardiac symptoms. On examination, the patient was hypertensive with moderate dependent edema. Electrocardiography demonstrated sinus rhythm with left atrial overload, premature ventricular complexes, left ventricular hypertrophy, and T-wave inversion in leads III and aVF (Figure 1). Sequential serum high-sensitivity troponin levels were stable at 17 ng/L (normal range, <14 ng/L). Serum N-terminal pro–brain natriuretic peptide was elevated at 4,417 ng/L (normal range, <900 ng/L) and serum angiotensin-converting enzyme level was normal (37 U/L; normal range, 20–70 U/L). Computed tomography showed no pulmonary embolism and no significant axillary, mediastinal, or hilar lymphadenopathy, with only mild atherosclerotic coronary plaque. There was no evidence of significant coronary artery disease demonstrated on computed tomographic coronary angiography at presentation and previous invasive coronary angiography.

Transthoracic echocardiography demonstrated a mildly dilated left ventricle with severe regional systolic dysfunction (ejection fraction 29%; Figure 2). Wall motion abnormalities involved the septal and inferior walls, crossing typical coronary distribution territories (Figure 3F, Videos 1A–1C). There was septal hypokinesia (Figure 3D, Video 1A). There was moderately severe mitral regurgitation with mild pulmonary hypertension (right ventricular systolic pressure 46 mm Hg). There was evidence of significant diastolic dysfunction with borderline shortened mitral deceleration time (166 msec), reduced septal and lateral e′-wave values (0.04 m/sec), and markedly elevated filling pressures (E/e′ ratio 22). Transpulmonary gradient was normal, as indicated by an echocardiographic pulmonary–to–left atrial ratio (tricuspid regurgitation maximum velocity divided by the E/e′ ratio) of 0.14 m/sec, suggesting postcapillary pulmonary hypertension.[4]

Cardiac magnetic resonance imaging (MRI), positron emission tomography (PET) using ^{18}F-fluorodeoxyglucose (FDG), and resting myocardial perfusion imaging using ^{99}Tc tetrofosmin were subsequently performed to characterize the tissue abnormalities in the ventricle. Cardiac MRI demonstrated patchy, nodular areas of left ventricular enhancement, predominantly in the basal left ventricle inferior and lateral wall (Figure 3E, Videos 2 and 3), corresponding to the akinetic areas on echocardiography (Figure 3F). There was subepicardial late gadolinium uptake in the distal septal wall (Figure 3C).

Nuclear medicine imaging with ^{99}Tc tetrofosmin and FDG PET revealed a significant focal perfusion defect in the inferior wall of the left ventricle (Figure 3A) with concordant significant reduced FDG uptake (Figure 3B). There was a significant focal perfusion defect in the anterior septal wall (Figure 3A) with mismatched preserved FDG uptake (Figure 3B). These combined techniques suggested combination of patchy active and chronic cardiac sarcoidosis, with no extracardiac sarcoidosis identified. Specifically, there was no FDG uptake in the mediastinum to suggest hilar lymphadenopathy involvement (Figure 3G). Initial conservative management without immunosuppression was chosen by the patient. Repeat echocardiography in the 3-month interval showed stable ventricular function.

DISCUSSION

Sarcoidosis is a systemic disease characterized by noncaseating granulomatous disorder of unknown etiology. Sarcoidosis most commonly affects the respiratory system, but the clinical presentation is highly variable.[5] Approximately 5% of patients have cardiac symptoms, but cardiac involvement is likely to be underdiagnosed, with 27% of patients with sarcoidosis identified to have pathologic

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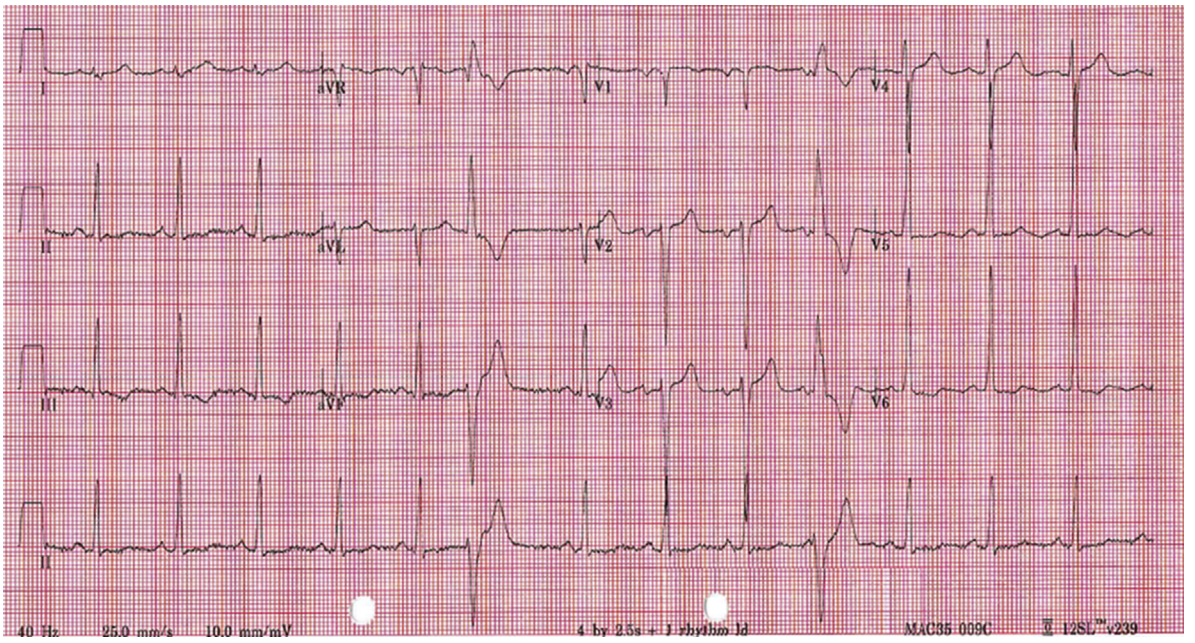

Figure 1 Twelve-lead electrocardiogram showing sinus rhythm with a vertical axis and normal PR and QTC intervals. The QRS duration is normal, and there is inferolateral T-wave inversion. Left atrial overload and left ventricular hypertrophy are also demonstrated.

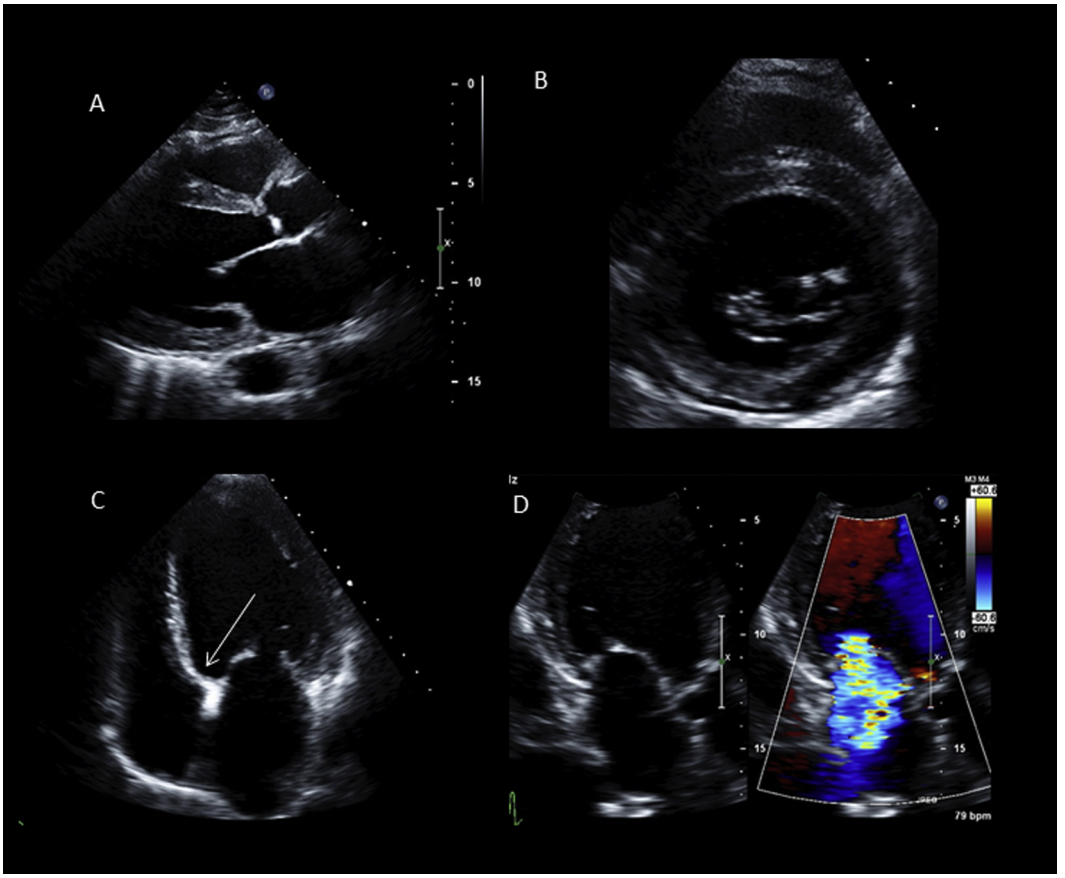

Figure 2 Transthoracic echocardiogram demonstrating dilated left ventricle on parasternal images **(A,B)**, with septal thinning (*white arrow*) on the apical four-chamber view **(C)**. There was moderately severe functional mitral regurgitation **(D)**.

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