LETTER TO THE EDITOR

Subclinical cardiac involvement: A subtle foe in the setting of systemic sarcoidosis

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To the editor,

In clinical practice, sarcoidosis has been mostly encountered in the form of multisystem disease (1-6). In their recently published article (1), Uslu Biner I, et al. have reported an interesting case of systemic sarcoidosis in a young male patient presenting with characteristic muscular findings on nuclear imaging (namely 'tiger man' sign) (1). Based on this interesting case (1), we would like to underscore potential implications of subclinical cardiac sarcoidosis (CS) in similar cases with multisystem involvement: First, CS is more likely to emerge as part of systemic sarcoidosis rather than presenting as an isolated entity (6). Clinically, CS may be further categorized into two clinical patterns: clinically manifest and subclinical CS. Importantly, clinically manifest CS generally presents with overt findings including malignant arrhythmogenesis, conduction blocks and left ventricular dysfunction (myocardial wall motion abnormalities with or without heart failure) (6-8). However, this form of CS emerges in only 5% of patients with sarcoidosis (6). On the other hand, subclinical CS is a more common phenomenon and may present with one of the following clinical scenarios: clinically silent CS, subclinical myocardial dysfunction or diastolic dysfunction (6,9,10). Epidemiologically, around 1 in every 4 patients with systemic sarcoidosis harbors clinically silent CS (6). These patients have classical patchy myocardial involvement in the absence of myocardial wall motion abnormalities, and hence, are generally asymptomatic in terms of cardiovascular symptoms (6). Accordingly, routine imaging modalities including echocardiogram is usually normal in the setting of clinically silent CS potentially suggesting the need for advanced diagnostic modalities including cardiac magnetic resonance imaging (with late gadolinium enhancement (LGE) and fluorodeoxyglucose

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positron emission tomography (FDG-PET) imaging (6). On the other hand, other subclinical CS forms including subclinical myocardial dysfunction (as might be demonstrated with the evaluation of advanced echocardiographic parameters including global longitudinal strain) and diastolic dysfunction (9,10) may be associated with non-specific cardiovascular symptoms including exertional dyspnea. Pathogenetically, these subclinical CS forms, besides being associated with early myocardial granulomas, may also arise as a consequence of oxidative and inflammatory mileu of systemic sarcoidosis (9,10). Of note, potential cardiac involvement might have contributed to the existing dyspnea in the patient (1). However, he apparently had no active CS (ongoing myocardial inflammation as demonstrated with FDG-PET (11,12). However, he might have harbored inactive CS (clinically manifest or subclinical) as might be demonstrated with cardiac MRI (1). We wonder about the findings on cardiac imaging (echocardiogram and MRI) (if any) and electrocardiogram in the patient (1). Second, subclinical CS might also have prognostic implications (8,10,11). Accordingly, subclinical myocardial dysfunction in patients with sarcoidosis was found to be associated with adverse cardiac events including new-onset arrhythmias, etc. and/or future evolution of overt CS (10). In patients with any form of subclinical CS, an existing LGE on cardiac MRI may potentially serve as an arrhythmogenic trigger and warrants further diagnostic strategies including ventricular arrhythmia stimulation for the decision-making for cardiac device therapy (8,11). As a last remark, we hold the opinion that patients with systemic sarcoidosis presenting with extensive muscular involvement (for ins; those presenting with 'tiger man' sign) may have a particular proclivity for myocardial involvement. This notion might, in part, be based on the similar histopathological characteristics of the myocardium and striated muscle providing an analogous mileu for granulomatous infiltration in this context. However, this speculation needs to be established. Therefore, we recommend close follow-up of the patient (1) with regard to future evolution of CS even if his heart is proved to be intact on advanced cardiac imaging. In conclusion, subclinical CS, unlike its clinically manifest counterpart, is a subtle and diverse phenomenon that might not be so easy to diagnose. However, as analogous to the clinically manifest CS, this phenomenon has also prognostic implications. Therefore, patients with systemic sarcoidosis should also be evaluated in a regular and detailed manner for subtle symptoms and signs of subclinical cardiac involvement. As a speculation, extensive muscular involvement may also suggest a higher likelihood of existing or future CS. Finally, combined use of advanced echocardiogram, cardiac MRI and nuclear imaging should be the routine strategy for the timely diagnosis and management of subclinical CS.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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