Clinical Case Report

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Diagnostic value of cardiac magnetic resonance and fluorodeoxyglucose-positron emission tomography for cardiac sarcoidosis with previous myocardial infarction

A case report

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Abstract

Rationale: Diagnostic difficulty due to overlapped clinical findings exists in cardiac sarcoidosis (CS) patients who also have coronary artery disease. Since cardiac magnetic resonance (CMR) and fluorodeoxyglucose-positron emission tomography (FDG-PET) evaluate different pathological processes, that is, fibrosis and inflammation respectively, the combination may be useful in such a case.

Patient concerns: A 77-year-old man was admitted due to heart failure and advanced atrioventricular block who was previously diagnosed with cutaneous sarcoidosis and old myocardial infarction (MI) with angiographical evidence.

Diagnosis: He was finally diagnosed with CS using CMR and FDG-PET by specifying the myocardial lesion of sarcoidosis.

Interventions: He was treated with corticosteroids based on the diagnosis.

Outcomes: The focal high uptake on FDG-PET was improved and he had a better clinical course without further cardiac events.

Lessons: Our case suggests that CMR and FDG-PET are complimentary, and the combination is useful for diagnosis of CS, particularly in cases that have previous MI.

Abbreviations: AV-block = atrioventricular block, CAD = coronary artery disease, CMR = cardiac magnetic resonance, CS = cardiac sarcoidosis, DE = delayed enhancement, FDG-PET = fluorodeoxyglucose positron emission tomography, JCS = Japanese Circulation Society, MI = myocardial infarction.

Keywords: cardiac magnetic resonance, fluorodeoxyglucose-positron emission tomography, myocardial infarction, sarcoidosis

1. Introduction

To our knowledge, little has been reported about cardiac sarcoidosis (CS) that overlaps with myocardial infarction (MI); its incidence is not clear. The exclusion of coronary artery disease (CAD) is required for diagnosis of CS. Both CS and MI may cause abnormal ventricular wall motion, heart failure, and arrhythmia including advanced atrioventricular block (AV-block) and ventricular tachycardia, and they may complicate the diagnosis in patients with CS who have CAD. Furthermore, positive delayed enhancement (DE) on cardiac magnetic resonance (CMR) is detected in both CS and MI. Indeed, most of the diagnostic criteria of the Japanese Circulation Society (JCS) overlap with that of MI.[1]

We report a case of CS in the setting of previous MI that was successfully diagnosed using CMR and fluorodeoxyglucose-positron emission tomography (FDG-PET) to specify the sarcoidosis lesion. CMR and FDG-PET are useful for diagnosis of CS, even in cases that have previous MI.

2. Case report

A 77-year-old man diagnosed with cutaneous sarcoidosis was admitted to our hospital resulting from heart failure with AV-block (Fig. 1A and B). His cardiovascular history included several previous percutaneous coronary interventions for ST-elevated MI and old MI to the left anterior descending and left circumflex arteries, respectively. Echocardiography showed hypokinesis of the posterolateral and antero-septal walls and no basal thinning of the ventricular septum (Fig. 1C). No significant coronary artery stenosis was detected during the emergency coronary angiography. CMR showed hypokinesis in antero-septal, antero-apical, and posterolateral walls with subendocardial DE corresponding with the coronary tree of previous MI (arrow-

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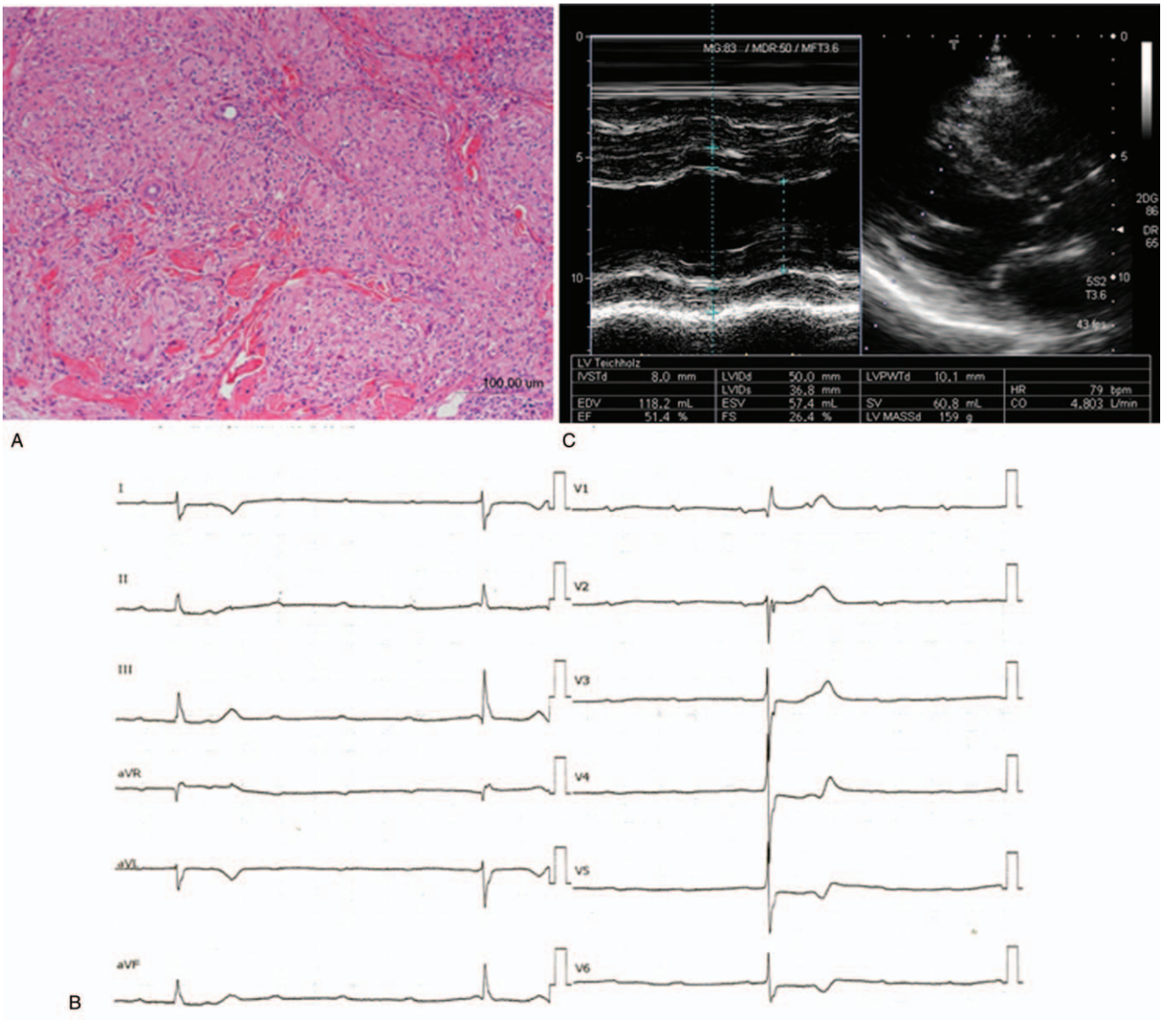

Figure 1. (A) Cutaneous sarcoidosis was previously diagnosed with histopathological evidence of noncaseating epithelioid cell granulomas. (B) Electrocardiography showing complete atrioventricular block. (C) Hypokinesis of antero-septal, antero-apical, and posterolateral walls were detected by echocardiography. These findings were consistent with the previous history of coronary artery disease. Basal thinning of the ventricular septum was not detected.

heads, Fig. 2A and B). Additionally, mid-wall DE was confirmed in the basal-anterior wall (arrows, Fig. 2A and B) with high intensity on a black-blood T2 weighted image (Fig. 2C). Subsequent FDG-PET imaging demonstrated high uptake in the basal-anterior wall (Fig. 2D and E). The patient was successfully diagnosed with CS using combined imaging modalities despite overlapping previous MI. The patient was treated with corticosteroids; the focal high uptake on FDG-PET was improved and had a better clinical course without further cardiac events.

Informed consent was obtained from the patient. Ethical approval was waived for our institutional review board.

3. Discussion

CMR has been able to detect myocardial inflammation and fibrosis and has prominent diagnostic value in CS, reported as having 100% sensitivity and 78% specificity.[2] Despite the excellent sensitivity of CMR, DE findings are observed both in CS and MI as a result of inflammation and subsequent fibrosis. Only positive DE has been included in the diagnostic criteria of the recent guidelines by the JCS,[1] though the detailed features, such as DE distribution, involvement of myocardial layer and the relationship with T2 images, has not been referenced. The DE of the myocardial layer in MI shows a subendocardial or transmural DE pattern[3,4]; in contrast, mid-wall or epicardial DE is preponderantly observed in non-ischaemic cardiomyopathy including CS.[5,6] However, CS sometimes shows subendocardial and transmural DE patterns, and DE findings could not distinguish the CS from MI in such a setting. Notably, most of the DE distribution does not correspond to the coronary perfusion area in CS and is often involved in the basal-septal wall.[7,8] In addition, while DE is detected in both differential conditions of inflammation and fibrosis,[9,10] the presence of a

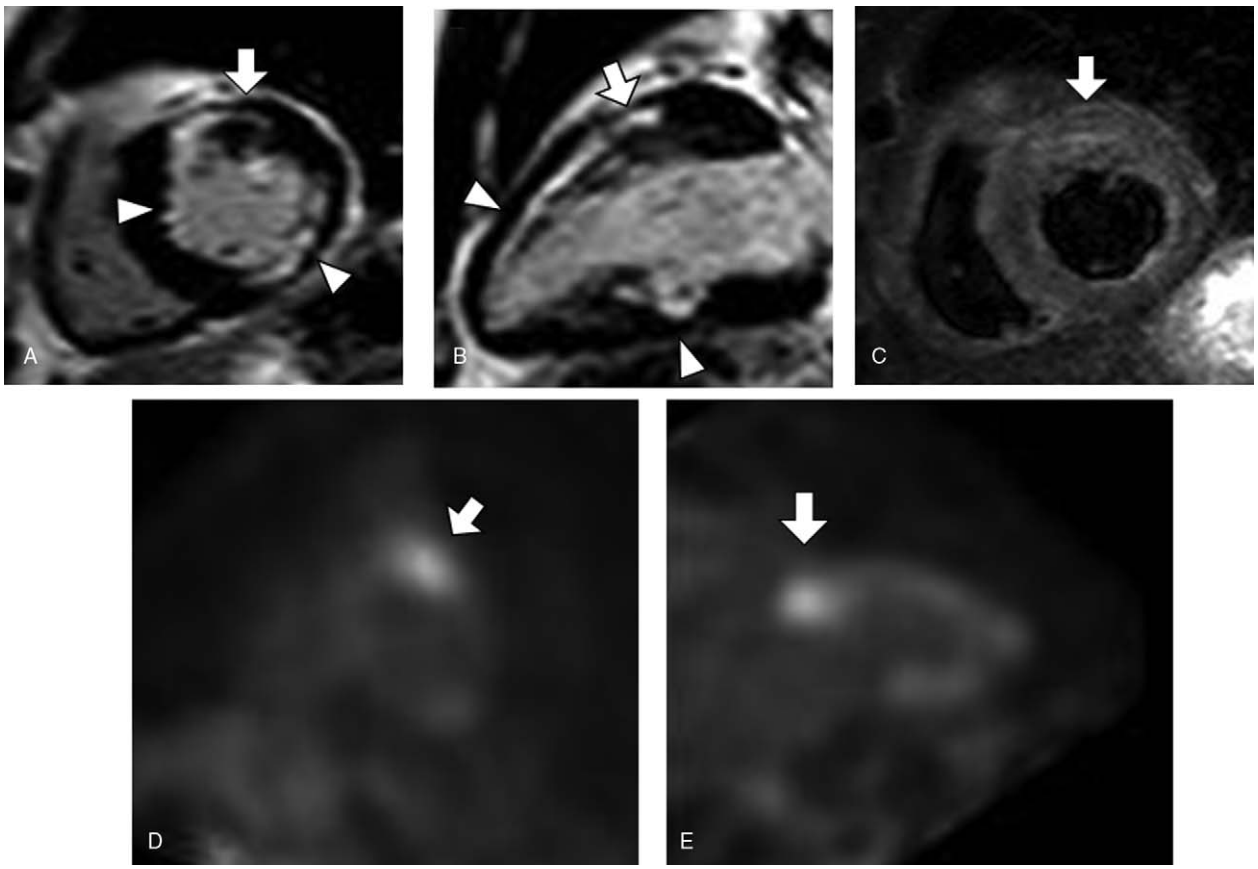

Figure 2. (A, B) Mid-wall delayed enhancement (DE) was confirmed in the basal-anterior wall (arrows), whereas subendocardial DE was confirmed in the antero-septal, antero-apical and posterolateral walls corresponding with previous myocardial infarction (arrowheads) on DE images of cardiac magnetic resonance. (C) Black-blood T2 weighted image showed high intensity of the basal-anterior wall (arrow). (D, E) Fluorodeoxyglucose-positron emission tomography (FDG-PET) imaging showed high uptake in the basal-anterior wall (arrows). (D) Short axis view. (E) Long axis view. DE = delayed enhancement, FDG-PET = fluorodeoxyglucose-positron emission tomography

hyper-intense T2 signal concomitant with DE indicated that oedema consistent with active inflammation is helpful for the diagnosis of CS, while previous MI shows a contrasting finding, that is, the absence of a high-intensity of T2 signal.[9] Detection of active inflammation on FDG-PET that represents the characteristics of CS is useful for diagnosis with 87% sensitivity and 38% specificity.[10] Furthermore, as is the case for this patient, FDG-PET may help with therapeutic management by confirming the inflammatory condition that is associated with ventricular dysfunction progression.[11]

Concerning the recent JCS criteria, this patient with old MI was previously diagnosed with cutaneous sarcoidosis according to histopathological evidence and fulfilled four of the five major criteria for cardiac involvement of sarcoidosis: high-grade atrioventricular block, left ventricular contractile dysfunction with a 47% ejection fraction, focal up-take in the basal-anterior wall on FDG-PET, and multiple positive DE on CMR. Importantly, the 4 major criteria except the FDG-PET are also observed in patients with previous MI; thus, the detailed analysis of each finding is required for clarifying the underlying pathophysiology in the two diseases. Based on the previous MI history, the hypokinesis of left ventricular antero-septal, antero-apical, and posterolateral walls might be caused by previous MI. For DE on CMR, subendocardial DE of antero-septal, antero-apical and posterolateral walls were consistent with the coronary perfusion area in support of previous MI. Additionally, the basal-anterior DE existed focally in the mid-wall of the myocardial layer, with high intensity on the black-blood T2 image, indicating that the basal-anterior lesion was caused by active sarcoidosis. Additionally, FDG-PET demonstrated focal high uptake in the basal-anterior wall corresponding with the mid-wall DE lesion. Furthermore, sudden onset of a high-grade AV-block without significant coronary artery stenosis on emergency angiography, unlikely to be caused by non-acute MI,[12] suggests that it was induced by active inflammation of cardiac sarcoidosis that was confirmed by CMR and FDG-PET findings.

4. Conclusion

The patient with previous MI was successfully diagnosed with CS using both CMR and FDG-PET and was treated with corticosteroids. CMR and FDG-PET evaluate different pathological processes, that is, fibrosis and inflammation respectively, and the true diagnostic accuracy of these modalities as compared with each other remains unclear. However, this case suggests that they are complimentary, and the combination is useful particularly in such a case overlapped with previous MI. Prospective studies are necessary to evaluate the utility of these modalities in the diagnosis and management of patients with CS.

Author contributions

Data curation: Takayuki Kawamura, Takashi Nakamura.
Validation: Yoshitaka Iwanaga.
Writing – original draft: Masakazu Yasuda.
Writing – review & editing: Yoshitaka Iwanaga, Salvatore De Rosa, Ciro Indolfi, Shunichi Miyazaki.

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