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Case Report

A case of acute heart failure due to myocardial infiltration of mycosis fungoides

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ABSTRACT

Mycosis fungoides (MF) has been reported to be the most common cutaneous lymphoma with a good prognosis and myocardial infiltration is clinically rare. We hereby report a case of rapidly progressing acute heart failure due to myocardial infiltration by MF. Perfusion cardiac magnetic resonance imaging (MRI) demonstrated a massive perfusion defect in the left ventricle (LV) where multiple nodular enhancement areas by delayed enhancement MRI could be documented in the postero-lateral wall of the LV, which resulted in a deterioration of the LV function and mitral regurgitation. Autopsy confirmed the myocardial infiltration by the MF, which corresponded with the MRI findings.
<**Learning objective:** Symptomatic heart failure patients with myocardial infiltration by mycosis fungoides (MF) have a poor prognosis because they could not undergo chemotherapy for primary disease. Therefore, early diagnosis is important for improvement of prognosis. The routine assessment of the cardiac involvement by cardiac magnetic resonance imaging as well as transthoracic echocardiography should be performed for an early recognition of myocardial infiltration even in asymptomatic MF patients.>

Introduction

Myocardial infiltration is reported to occur in 20–25% of patients with malignant lymphomas. Mycosis fungoides (MF) is the most common cutaneous T-cell lymphoma with a good prognosis. Although nodal, visceral, and blood involvement, and myocardial infiltration are frequent in the advanced stages, symptomatic cardiac involvement is rare. The cardiac involvement is usually asymptomatic and incidentally diagnosed by autopsy. We hereby describe a case of symptomatic cardiac involvement due to MF.

Case report

A 72-year-old female with a 12-year history of MF was referred to our hospital due to a cough for a few weeks. Although she underwent narrowband ultraviolet B (NB-UVB) and psoralen and ultraviolet A (PUVA) light therapy for plaque-stage MF (T1 N0 M0) over the past decade, the development of a lesion mass was documented on the right thigh the prior year. She underwent interferon-γ therapy for tumor-stage MF (T3 N0 M0). A physical examination revealed mild tachycardia (93/min), hypertension (166/110 mmHg), and desaturation (SpO2 98%, O_2 nasal 2L). The B-type natriuretic peptide (BNP), aspartate aminotransferase (AST),

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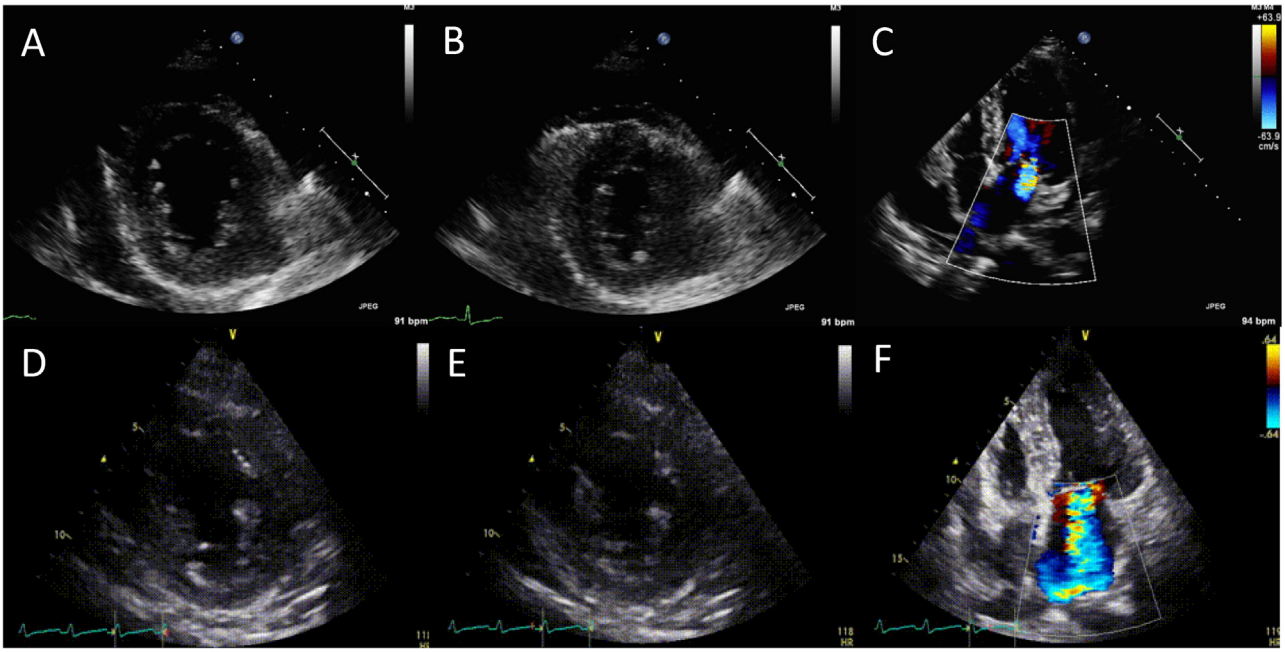

Fig. 1. (A–C) Transthoracic echocardiography on admission. A massive pericardial effusion was documented. Of note, the left ventricular wall is thickened and the wall motion is hyperkinetic. The diastolic phase in (A) and systolic phase in (B) are shown. Mitral regurgitation is considered as "moderate" in (C). (D–F) Transthoracic echocardiography 10 days after admission. The left ventricular asynergy, especially of the postero-inferior wall, is significantly obvious, which results in worsening mitral regurgitation. The diastolic phase in (D) and systolic phase in (E) are shown. Of note, the motion of the postero-inferior wall of the left ventricle is significantly reduced. The mitral regurgitation is considered as "severe" in (F).

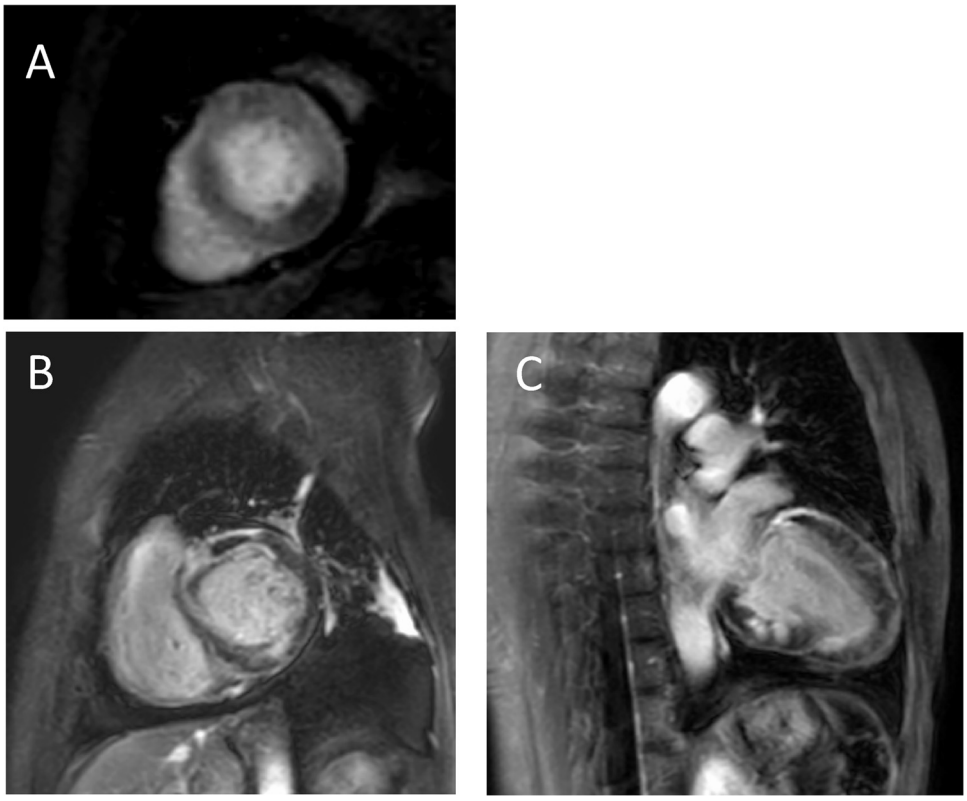

Fig. 2. (A) Perfusion cardiac magnetic resonance imaging: a patchy perfusion defect is located throughout the left ventricular wall. Of note, a severe perfusion defect is especially noted located on the postero-lateral wall. The delayed enhancement magnetic resonance imaging in the short (B) and longitudinal views (C): multiple focal nodular enhancement areas on the postero-lateral wall of the left ventricle are located in the perfusion defect area.

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