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Case Report

Late-gadolinium enhancement in a subject with normal left ventricular function [☆]

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ARTICLE INFO

Article history:

Received 6 August 2020

Revised 11 September 2020

Accepted 12 September 2020

Keywords:

Mid-layer enhancement in late-gadolinium enhancement
Normal left ventricular function

ABSTRACT

A 27-year-old man visited our hospital after experiencing palpitations. His 12-lead electrocardiogram and chest radiograph were unremarkable. Blood test results showed normal plasma brain natriuretic peptide level (<5.8 pg/mL). Transthoracic echocardiography revealed normal left ventricular structure and function by demonstrating left ventricular wall thickness of 10 mm, end-diastolic dimension of 46 mm, end-systolic dimension of 31 mm, and ejection fraction of 64%. Pulsed-wave Doppler echocardiography demonstrated normal E/e' ratio of 7.5. Cardiac magnetic resonance imaging showed normal coronary artery. However, there was massive late-gadolinium enhancement at the mid-layer wall, suggesting massive left ventricular fibrosis. This case reveals that left ventricular function may be normal even in massive late-gadolinium enhancement. Pathophysiology other than fibrosis might have contributed to this specific finding in late-gadolinium enhancement.

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Introduction

Cardiac fibrosis is characterized by fibroblast accumulation and excess deposition of extracellular matrix proteins [1]. Left ventricular (LV) reactive fibrosis takes place primarily as an adaptive response to pressure or volume overload. Then, reactive fibrosis will progress to cell necrosis and replacement fibrosis. Cardiac fibrosis increases stiffness of left ventricle,

and impedes contraction and relaxation of heart [2]. Therefore, detection of myocardial fibrosis is necessary for risk stratification and treatment of cardiac diseases. A myocardial biopsy may have some risk and unreliability, because patterns of myocardial fibrosis are considerably diverse. Cardiac magnetic resonance imaging (C-MRI) provides a simple means of detecting myocardial fibrosis in a variety of pathologies [3]. We experienced a rare case of diffuse mid-layer fibrosis represented by C-MRI with normal cardiac function.

[☆] Declaration of competing interest: None.

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<https://doi.org/10.1016/j.radcr.2020.09.015>

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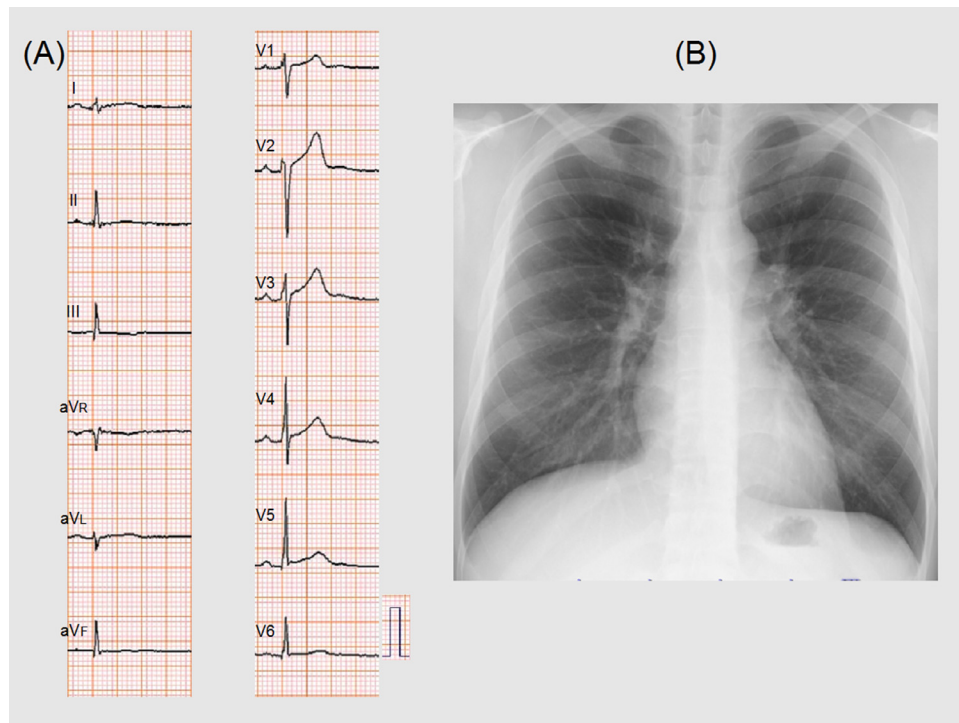


Fig. 1 – Twelve-lead electrocardiogram is unremarkable (A) and chest radiograph is normal, showing a cardiothoracic ratio of 46% (B).

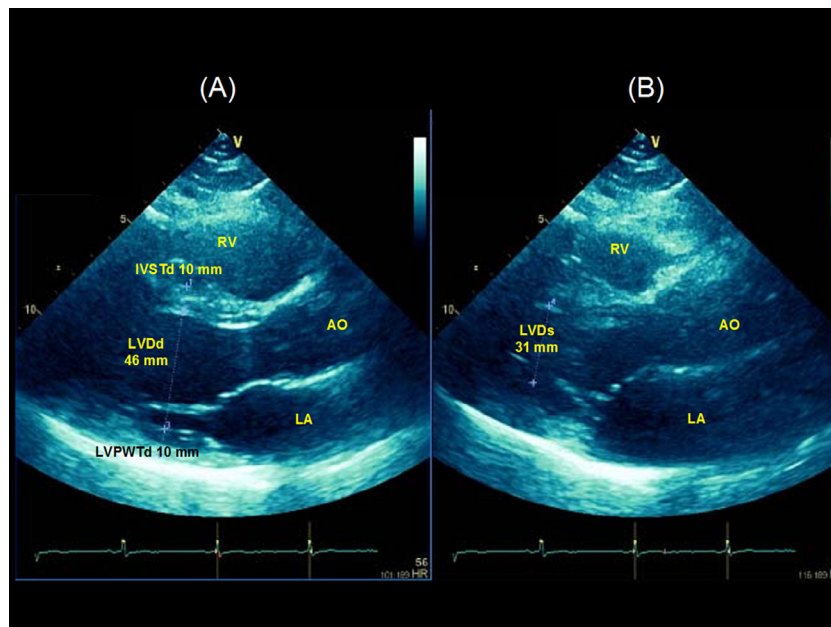


Fig. 2 – Parasternal long-axis view of transthoracic echocardiography during end-diastole (A) and end-systole (B). Thickness of interventricular septum and left ventricular (LV) posterior wall are 10 mm each (A). LV end-diastolic dimension and end-systolic dimension are 46 mm and 31 mm, respectively. There is a hyperechoic nodule at the interventricular septum (arrow) and double layer image at the posterior wall (arrowheads).

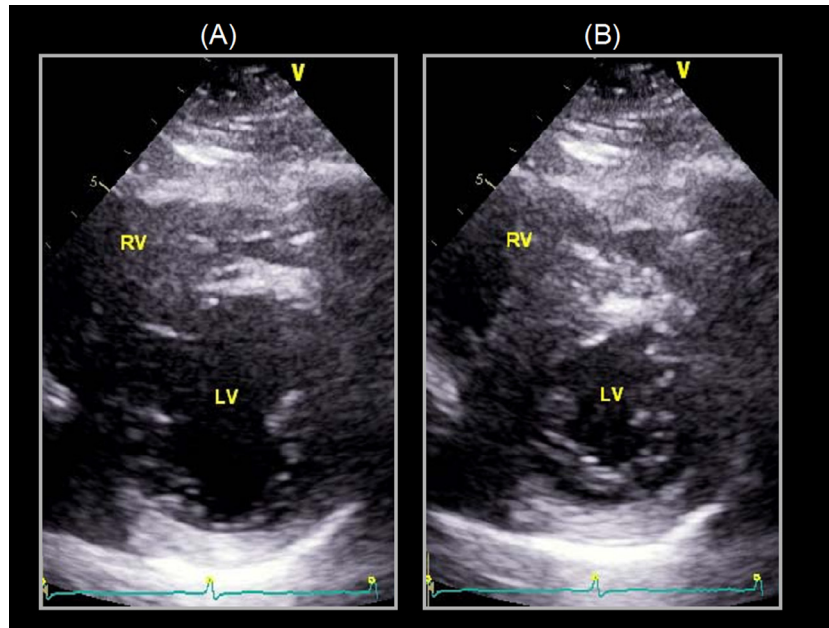


Fig. 3 – Parasternal short-axis view of transthoracic echocardiography during end-diastole (A) and end-systole (B). As in [Fig. 2](#), there is a hyperechoic nodule at the interventricular septum (arrow) and double layer image at the posterior wall (arrowhead).

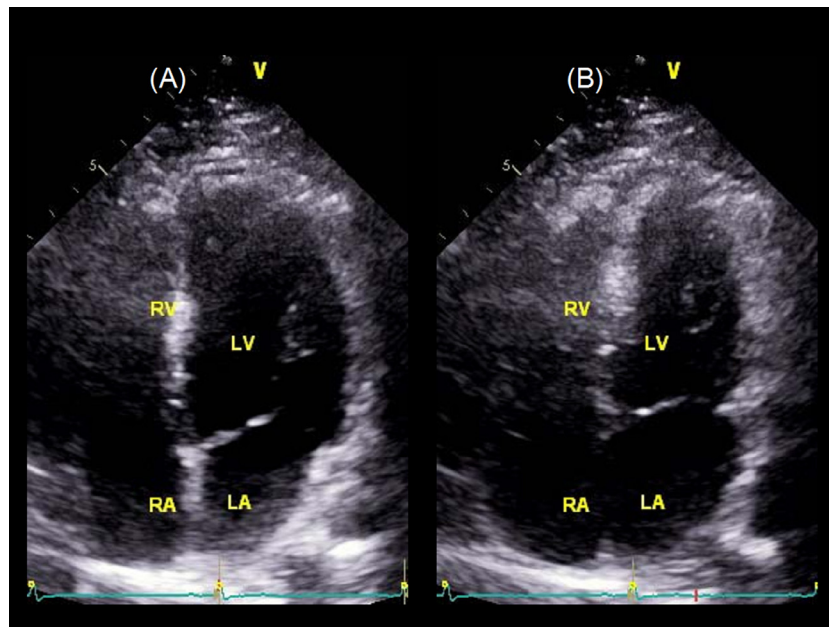


Fig. 4 – Apical echocardiographic image during end-diastole (A) and end-systole (B) shows normal LV wall motion.

Case report

A 27-year-old man visited our hospital after experiencing palpitations. On physical examination, his blood pressure was 122/96 mm Hg and pulse rate was 71 beats per minute. Blood test results showed mildly abnormal liver function. Plasma brain natriuretic peptide level was normal (<5.8 pg/mL).

Serum calcium level, plasma angiotensin-converting enzyme level, and plasma renin activity were normal. Plasma aldosterone level was slightly elevated (226 pg/mL). The antinuclear antibody test result was negative. Ophthalmoscopy findings were normal. A 12-lead electrocardiogram ([Fig. 1A](#)) and a chest radiograph with cardiothoracic ratio of 46% ([Fig. 1B](#)), showed no remarkable findings. Holter electrocardiogram revealed single premature ventricular contractions.

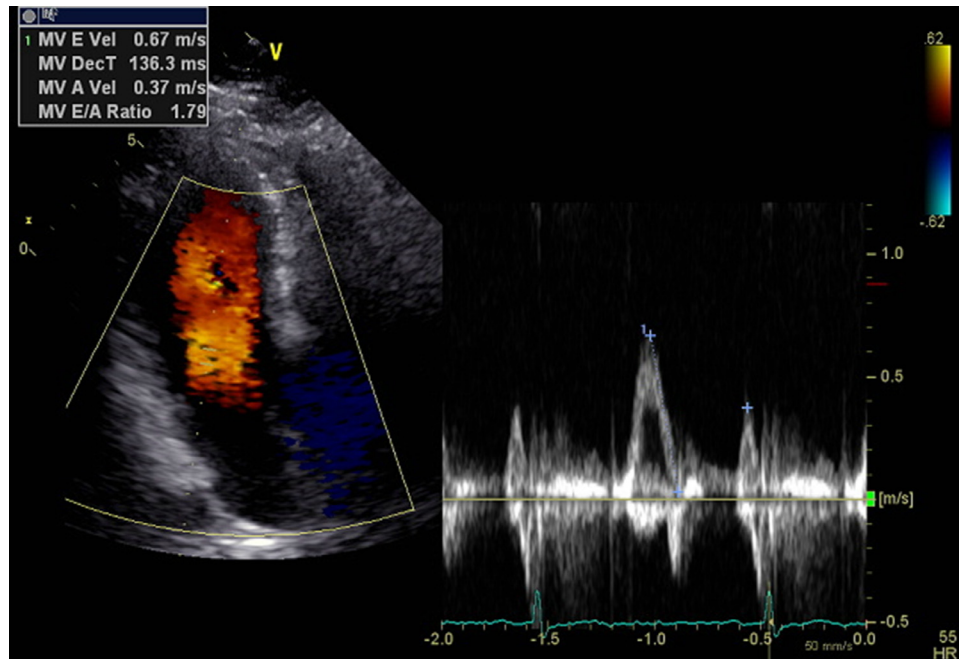


Fig. 5 – Pulsed-wave Doppler echocardiography of the mitral inflow showed E wave of 67 cm/s, A wave of 37 cm/s, and deceleration time of E wave of 136 ms.

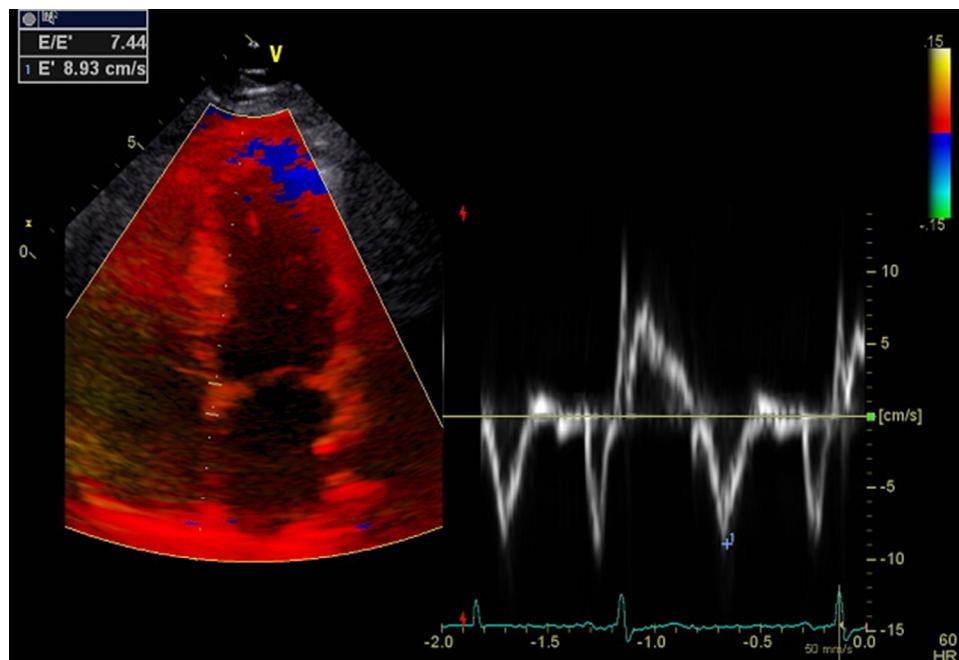


Fig. 6 – A pulsed-wave tissue Doppler echocardiography of the mitral septal annulus showed e' wave of 8.9 cm/s and E/e' ratio of 7.5.

Parasternal long axis view of transthoracic echocardiography revealed that both the interventricular septum and left ventricular (LV) posterior wall were 10 mm thick (Figs. 2 and 3). LV end-diastolic dimension and end-systolic dimension were 46 mm and 31 mm, respectively. Apical echocardiographic image showed normal LV wall motion with ejection fraction of

64% (Fig. 4). A color-flow Doppler echocardiography presented no data suggesting significant valvular heart disease. Pulsed-wave Doppler echocardiography at the mitral inflow showed an E wave of 67 cm/s, an A wave of 37 cm/s, and deceleration time of the E wave of 136 ms (Fig. 5). A pulsed-wave tissue Doppler echocardiography at the mitral septal annulus

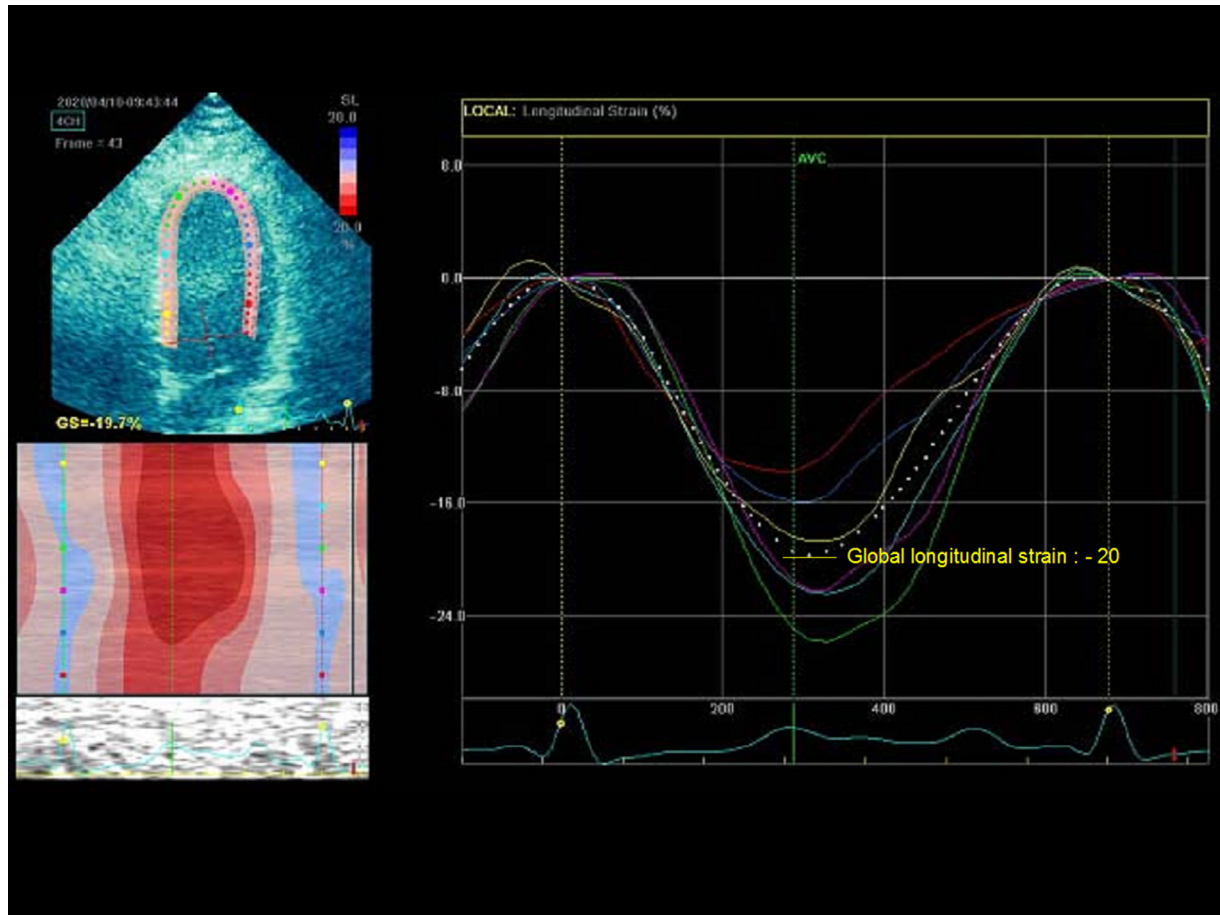


Fig. 7 – Global longitudinal strain obtained from speckle tracking of an apical 4-chamber image showed peak contraction of -20 .

showed e' wave of 8.9 cm/s and E/e' ratio of 7.5 (Fig. 6), indicating normal diastolic function. Global longitudinal strain obtained from speckle tracking of an apical 4-chamber image showed normal peak contraction of -20 (Fig. 7) [4].

C-MRI revealed no significant coronary artery stenosis (Fig. 8). Late-gadolinium enhancement (LGE) revealed mid-wall striae involving the left ventricle (Fig. 9, arrows), representing LV diffuse myocardial fibrosis [3]. The patient complained chest discomfort after treatment with angiotensin-converting enzyme inhibitor. His symptom improved after the drug was changed to spironolactone. He did not come to our hospital thereafter because he moved to another city.

Discussion

In the present case, a transthoracic echocardiography showed that LV thickness, size, and ejection fraction were all within normal limits. Pulsed-wave Doppler echocardiography showed normal E/e' ratio and value of global longitudinal strain rate was normal. Thus, LV systolic and diastolic function was normal even with massive myocardial fibrosis

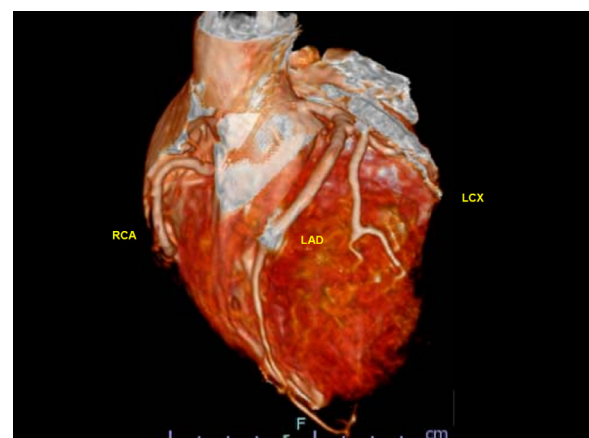


Fig. 8 – Cardiac magnetic resonance imaging (C-MRI) shows no significant coronary artery stenosis.

represented by broad midwall striae by LGE. However, it is unlikely that the intact inner and outer layers of the myocardium extinguished the negative effect of myocardial fibrosis.

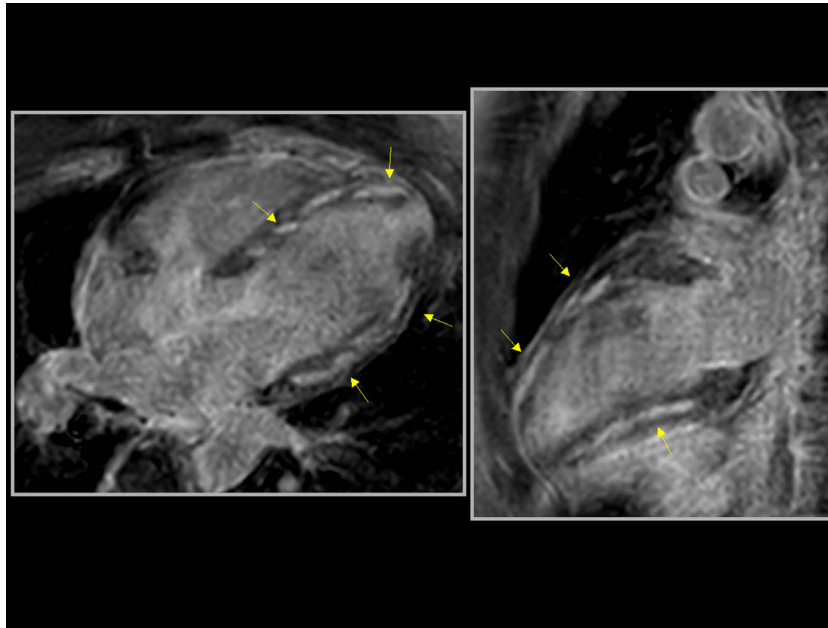


Fig. 9 – Late-gadolinium enhancement reveals diffuse mid-wall striae of the left ventricle (arrows).

Gadolinium chelates are known to be interstitial contrast agents that rapidly distribute from the intravascular space and equilibrate with the interstitial space. They are immediately excluded from the intracellular space, and fibrotic lesions are presented as bright spots compared to normal cardiac tissues in LGE. However, this technique simply measures increased extracellular spaces, and does not necessarily demonstrate increased myocardial fibrosis or collagenous deposition. In the present case, other cardiac disease such as edematous or infiltrative interstitial diseases might have contributed to mid-wall striae with normal LV systolic and diastolic function [3].

The patient had no obvious history of cardiac disease, and the blood test findings and echocardiography data did not suggest sarcoidosis, collagen disease, or primary aldosteronism. Previous reports showed that the patterns of enhancement in LGE were disease-specific: Subendocardial fibrosis occurs in ischemic cardiomyopathy and endomyocardial fibrosis, whereas patchy and focal patterns are reported in hypertrophic cardiomyopathy and sarcoidosis, respectively [5]. Midlayer striae are observed in nonischemic dilated cardiomyopathy with an incidence of approximately 30% [6].

This is a rare case of diffuse midwall striae involving the left ventricle in LGE without LV systolic or diastolic dysfunction. This finding suggests that midventricular striae may occur in apparently healthy young subjects and may not directly reveal cardiac fibrosis. Previous reports showed that the presence of midventricular fibrosis is a predictor of a combined endpoint of mortality and hospitalization [7]. Careful echocardiographic follow-up is planned for early detection of LV dysfunction.

Conclusion

This is a rare case of diffuse LV midlayer striae in LGE without LV systolic or diastolic dysfunction. Careful echocardiographic follow-up is needed for early detection of possible impairment of cardiac function.

Informed consent

Written consent to anonymous publication of this case report was given.

Acknowledgments

I would like to thank Dr Masahiro Obata for their wonderful collaboration. You supported me greatly and were always willing to help me. I would like to thank Mr. Yoshiharu Saito and Yuji Ogata for taking echocardiographic images.

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