A 59-year-old man was admitted with exertional dyspnea. He had a 10-year history of uncontrolled systemic hypertension and the maximum level of blood pressure was 200/140 mm Hg. Chest x-ray showed cardiomegaly with pulmonary venous congestion, and the cardiothoracic ratio was 74%. Coronary arterial disease was excluded with selective coronary angiography, and hypertensive heart disease was diagnosed. Contrast-enhanced MRI demonstrated that the left ventricle dilated with systolic dysfunction, and the hypertrophy of the left ventricular wall was identified (Figure 1A and 1B). The left ventricular end-diastolic dimension was 94 mm, and the left ventricular ejection fraction was 21%. The thickness of the interventricular septum was 16 mm, the lateral wall was 8 mm, and the anterior wall was 8.4 mm. Furthermore, diffuse late gadolinium enhancement (LGE) occurred in the left ventricular wall, mainly in the midwall of the interventricular septum and the subendocardium of the free wall (Figure 2A). In the inferior and inferolateral segments of the left ventricle, near-transmural LGE presented (Figure 2B and 2C).

One week later, the informed consent was obtained and heart transplantation was performed. The pathological findings of the explanted heart were compared with the previous in vivo MRI. In the midwall of interventricular septum with LGE, myocardial interstitial fibrosis was identified (Figure 3A and 3B). The collagen volume fraction was 16% in the histological section, which came from the region of midwall LGE in the interventricular septum. In the subendocardium of inferior and inferolateral segments, myocardial replacement fibrosis was detected (Figure 4A and 4B). In the mid and outer myocardium of the corresponding wall, however, diffuse myocardial interstitial fibrosis was identified (Figure 4C and 4D), especially in the subepicardium. The epicardial coronary arteries were normal in the patient.

Previous researches have suggested that 45% of patients with systemic hypertension had myocardial LGE. Furthermore, pathological findings have confirmed that myocardial interstitial fibrosis was common in systemic hypertension. However, the mechanism of LGE was still unclear in these patients. LGE relied on relative differences in signal intensities. Therefore, it was accepted that myocardial interstitial fibrosis was usually missed by LGE. In this study, we made a comparison between the myocardial LGE and histological findings in a patient with systemic hypertension who underwent heart transplantation. The results of this study demonstrated the histological basis of myocardial LGE in patients with systemic hypertension: localized myocardial interstitial fibrosis in the midwall (or outer myocardium) and replacement fibrosis in the subendocardium.

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**Disclosures**

None.

**References**

Figure 1. Cine-MRI in 4-chamber views (A, diastole; B, systole) showed that the left ventricle dilated associated with hypertrophic myocardium. The left ventricular end-diastolic volume was 737.3 mL, and the left ventricular end-systolic volume was 583 mL. The left ventricular mass indexed was 276.2 g/m².

Figure 2. The corresponding LGE-MRI (A, 4-chamber view; B, 2-chamber view; C, short-axis view) demonstrated that LGE occurred in the interventricular septum (midwall, arrowhead) and left ventricular free wall (subendocardium). In the inferior segment of left ventricle, near transmural LGE was identified (arrow). LGE indicates late gadolinium enhancement.

Figure 3. The histological sections from the interventricular septum (A, Masson trichrome stain) showed that myocardial interstitial fibrosis was mainly localized in the midwall (B, Masson trichrome stain).
Figure 4. The histological sections from the inferior wall of the left ventricle (A, Masson trichrome stain) demonstrated that replacement fibrosis occurred in the subendocardium (B, Masson trichrome stain), mild myocardial interstitial fibrosis presented in the midwall (C, Masson trichrome stain), and marked myocardial interstitial fibrosis in the subepicardium (D, Masson trichrome stain).
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