Chronic Cardiac Transplant Rejection: Evaluation With Magnetic Resonance Imaging
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A 35-year-old man was admitted with pitting edema on both lower legs, dyspnea on exertion, and orthopnea for 2 months before presentation. He had undergone cardiac transplantation for idiopathic dilated cardiomyopathy 12 years previously and had a history of mild acute rejection during the immediate postoperative period. The rejection was well controlled and transplant function was restored. However, 9 years after transplantation, coronary angiography revealed total occlusion of the left anterior descending coronary artery, and echocardiography demonstrated akinesia of the interventricular septum at the apical level, which was clinically diagnosed as transplant vasculopathy. The left atrium was then progressively dilated, and the global left ventricular systolic function was shown to be normal on regular echocardiography at 6-month intervals.

Echocardiography on admission revealed significant diastolic dysfunction (E/E' = 20) with abnormal septal motion and increased volume of both atria. The patient underwent cardiac magnetic resonance imaging on admission. Four-chamber cine images showed prominent biatrial enlargement with normal-sized ventricles (Figure 1). T2-weighted black blood short-axis imaging demonstrated abnormal signal intensity in the right side of the interventricular septum (Figure 2A). The abnormal high-signal area was not due to slow flow artifact with reference to the cine image taken at the same anatomic position (Figure 2B). On delayed-enhancement magnetic resonance imaging, hyperenhancement was noted not only in the abnormal T2 signal area but also in the subendocardial portion of all left ventricular segments (Figure 2C). The pattern of delayed enhancement was not explained by a particular vascular territory, so we suggested extensive fibrosis.

Endomyocardial biopsy was performed from the right side of the interventricular septum. Histological specimen demonstrated myocyte hypertrophy, interstitial fibrosis, and replacement fibrosis, suggestive of chronic transplant rejection (Figure 3).

Disclosures

None.
Figure 2. T2-weighted black blood image at midventricular level (A) demonstrates a high-signal intensity area on the right side of the ventricular septum. The abnormal signal area was not due to slow flow artifact with reference to the cine image at the same level (B). Delayed-enhancement imaging (C) shows circular sub-endocardial hyperenhancement as well as hyperenhancement on the right side of the ventricular septum.

Figure 3. Histological section of the myocardium (hematoxylin-eosin stain, magnification ×100) revealed myocyte hypertrophy, interstitial fibrosis, and replacement fibrosis.