A 47-year-old woman with hypertrophic obstructive cardiomyopathy presented with worsening exertional dyspnea, paroxysmal nocturnal dyspnea, and chest pain, despite maximal medical therapy. Echocardiography showed severe ventricular hypertrophy, left ventricular outflow tract (LVOT) narrowing, a 110 mm Hg LVOT gradient, and systolic anterior motion of the mitral valve. She was referred for nonsurgical septal reduction (NSSR). Coronary angiography revealed 3 proximal septal branches of the left anterior descending artery; each was injected with ethanol through an occlusive balloon catheter. Final angiography showed low flow and incomplete contrast penetration into the treated septal branches. The patient’s LVOT gradient declined to 65 mm Hg, and she improved symptomatically. Contrast-enhanced MRI was performed at 3 time points (Figure).

MRI has been used to characterize myocardial perfusion after acute myocardial infarction (MI) due to coronary artery disease (CAD). An area of subendocardial hypoenhancement or perfusion defect is often seen on first-pass imaging, and this usually corresponds to microvascular obstruction. On delayed imaging, this subendocardial region becomes hyperenhanced along with the surrounding infarcted territory. NSSR with ethanol creates a localized infarct. Ethanol causes significant microvascular obstruction such that contrast penetration is extremely slow. Hence, the delayed image obtained shortly after the procedure shows patchy hyperenhancement. With time, the extent of microvascular obstruction declines, as seen after acute MI due to CAD. Thus, the images taken 3 weeks after the procedure show a less severe first-pass perfusion defect and greater hyperenhancement on delayed imaging. In the setting of acute MI due to CAD, microvascular obstruction affects left ventricular remodeling. How this influences the long-term outcome of hypertrophic obstructive cardiomyopathy patients who have had NSSR remains unknown.
Microvascular Obstruction After Nonsurgical Septal Reduction for the Treatment of Hypertrophic Cardiomyopathy
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