Isolated Septal Branch Myocardial Infarction Due to Coronary Spasm Mimicking Non-Ischemic Late Gadolinium Enhancement Pattern on Cardiac Magnetic Resonance Imaging

Naotsugu Iwakami, MD; Teruo Noguchi, MD; Yoshihiko Ikeda, MD; Emi Tateishi, MD; Yoshiaki Morita, MD; Hideaki Kanzaki, MD; Mitsuhiro Takewa, MD; Toshihisa Anzai, MD; Hisao Ogawa, MD; Satoshi Yasuda, MD

Figure 1. Late gadolinium enhancement (LGE) and ergonovine provocation testing. Cardiac magnetic resonance imaging showed (A, B) a patchy mid-septal wall LGE lesion, and (C, D) an endocardial LGE lesion. (E–J) Coronary angiography in (E, F, H, I) the right anterior oblique and (G, J) left anterior oblique views. (E–G) Ergonovine provocation testing produced significant spasm at the proximal site of the septal and diagonal branches, which was (H–J) relieved by nitroglycerin treatment.
Patchy mid-septal wall late gadolinium enhancement (LGE) patterns on cardiac magnetic resonance imaging (CMR) are usually suggestive of non-ischemic pathophysiology, such as idiopathic dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy, or cardiac sarcoidosis. Even with coronary artery disease and a subendocardial LGE pattern in other areas, mid-wall LGE lesions are attributed to non-ischemic cardiomyopathies. In this report, the detailed coronary anatomy of this region was found to explain the mechanism for the misleading LGE pattern of the basal septum, and therefore should be included in the differential diagnosis in order to discriminate coronary artery disease from non-ischemic cardiomyopathies.

A 47-year-old woman with a past medical history significant for hypertension was transferred to the emergency department complaining of severe nocturnal chest pain that lasted for 30 min at midnight for 3 consecutive days. She presented with ongoing chest pain, cardiac enzyme elevation (creatine kinase [CK], 423 IU/L; CK-MB, 57 IU/L), ST-T depression in the lateral leads, and lateral wall asynergy. Acute coronary syndrome was suspected, but urgent coronary angiography showed no organic stenosis. The symptoms spontaneously resolved before nitroglycerine treatment and did not recur during the hospital stay. Given that the cardiac enzyme levels soon normalized, and the electro- and echocardiographic changes were subtle, further examination was not done and she remained free of medication at that time. Lateral ventricular wall asynergy persisted on echocardiography for months, however, therefore CMR was done 3 months later to evaluate the etiology of myocardial damage using a standardized clinical protocol on a 1.5-T system (Magnetom Sonata, Siemens, Erlangen, Germany) with a 4-channel surface coil. LGE was identified using a segmented inversion-recovery (IR) prepared true-fast imaging with steady-state precession (FISP) sequence with electrocardiogram triggering 10 min after the administration of 0.15 mmol/kg body weight gadolinium diethylenetriamine penta-acetic acid. LGE data were obtained during the mid-diastolic phase with an inversion time of 300 ms. Seven to 9 contiguous short-axis slices at 10-mm intervals and 3 standard long-axis slices were obtained in a single breath hold. Other imaging variables were as follows: 65 segments; echo time, 1.73 ms; flip angle, 60°; field of view, 340×255 mm; matrix, 256×129; and voxel size, 1.3×2.0×8.0 mm³. We also acquired cine imaging using a true-FISP sequence (echo time, 1.3 ms; repetition time, 2.6 ms; flip angle, 60°; slice thickness, 8 mm; gaps, 2 mm; in-plane resolution, 4.17×2.73 mm) over multiple breath holds in contiguous short-axis slices encompassing the entire left ventricle (LV) and 3 standard long-axis slices. CMR showed 2 LGE lesions in the endocardium of the lateral wall of the LV and in the mid-layer of the basal septum, suggestive of non-ischemic cardiomyopathy such as DCM or cardiac sarcoidosis (Figures 1A–D). Septal wall asynergy was not detected on echocardiography at the first presentation or on cine CMR 3 months later. Thorough assessment consisting of laboratory testing, endomyocardial biopsy (Figure S1), and gallium scintigraphy showed no specific findings of cardiac sarcoidosis. Finally, the patient underwent ergonovine provocation testing to evaluate nocturnal chest pain. The symptoms and ST-T changes were completely reproduced with focal spasms in the first septal and first diagonal branches that supplied both LGE lesions, which were relieved after nitroglycerin (Figures 1E–J; Movie S1). She was diagnosed with non-ST-segment elevation myocardial infarction due to isolated coronary spasm of the first septal and first diagonal branches.

The present patient had a mid-septal wall LGE pattern due to septal branch myocardial infarction. In particular, in this case, the simple knowledge that septal branch infarction can produce this pattern may have enabled the diagnosis to be reached more quickly. This misleading LGE pattern in the basal septum can be explained by prior detailed anatomical studies. The right ventricular side of the interventricular septum is known to be perfused partly from the right ventricular lumen, and the LV endocardium can be spared owing to the interventricular complementary networks of the septal branches. Indeed, some patients with hypertrophic cardiomyopathy following transmural septal myocardial ablation reportedly had LGE patterns similar to that observed in the present case. Soriano et al reported some cases of mid-wall LGE with coexisting subendocardial LGE in their 71 CMR cases, which they attributed to either non-ischemic or ischemic origin.

In conclusion, even when a patchy LGE pattern in the midlayer of the septum is observed, coronary artery disease should be considered as part of the differential diagnosis, in addition to non-ischemic cardiomyopathies.

References


6. Song JK. Role of noninvasive imaging modalities to better understand the mechanism of left ventricular outflow tract obstruction and tailored lesion-specific treatment options. *Circ J* 2014; 78: 1808 – 1815.


a few inflammatory cells with moderate interstitial and perivascular fibrosis, and no specific features of primary or secondary cardiomyopathy or myocarditis.

Supplementary File 2

Movie S1. Ergonovine provocation testing indicated significant coronary spasm at the proximal site of the septal and diagonal branches, which was relieved by nitroglycerin treatment.

Please find supplementary file(s):

Supplementary Files

Supplementary File 1

Figure S1. Histology of endomyocardial biopsy specimens, showing