CASE REPORT

Cardiac Rupture in Acute Myocardial Infarction: Post-mortem MR Imaging

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We assessed acute myocardial infarction (AMI) with cardiac rupture in an autopsy using magnetic resonance (MR) imaging in vitro and histological staining. Cardiac MR imaging in vitro generated high resolution images of myocardial thinning, rupture, and epicardial hemorrhage. High signal intensity (SI) on T1 weighted images (WI) and low SI on T2WI of the rupture site corresponded with acute myocardial necrosis, edema, and hemorrhage. A rupture site rich in erythrocytes after AMI might affect SI on T1 and T2WI in cardiac MR imaging.

Keywords: autopsy, hemorrhage, rupture, signal intensity

Introduction

Noninvasive imaging plays a central role in diagnosing heart disease, assessing prognosis, and monitoring the effects of therapy. Improvements in speed, imaging quality, reliability, and range of applications of cardiac magnetic resonance (MR) imaging permit its comprehensive assessment of patients with heart disease and have made it an important means of assessing myocardial anatomy, regional and global function, and viability.¹ Cardiac rupture is a serious complication of acute myocardial infarction (AMI), and detailed MR features of this event remain unknown despite a few case reports of its visualization using cardiac MR imaging.²⁻⁴ We herein characterize the MR features of cardiac rupture using MR imaging in vitro and histological assessment of AMI in an autopsied case.

Case Report

A 79-year-old woman with hypertension and a history of smoking consulted a primary care physician for morning onset of sudden chest pain. Electrocardiography showed ST elevation in leads I, aVL, V1 to V6, and she was transferred to our hospital with a diagnosis of AMI. Cardiac ultrasonography revealed left ventricular hypertrophy, thinning of the apex to the anteroseptal wall with decreased wall motion, and pericardial effusions. She died of cardiac shock 4 hours after the onset of chest pain. We received written informed consent from a family member to perform an autopsy. Gross observation revealed a small fissure at the anterior wall with hemopericardium and epicardial hemorrhage, indicating cardiac rupture. The heart was then fixed in 10% formaldehyde for 24 hours at room temperature, rinsed with water, and submerged in water in a plastic container. Cardiac MR imaging in vitro proceeded using a 1.5-tesla superconducting system combined with an 8-channel knee-phased array coil (Signa Excite HDxt, GE Medical Systems, Waukesha, WI, USA). We acquired 3-dimensional (3D) fast spin echo (FSE) cube sequence T2-weighted images (T2WI) with fat suppression, 1,500-ms repetition time (TR), and 90-ms echo time (TE). Other parameters included: number of excitations (NEX), 6; echo train length (ETL), 110; receiver bandwidth (BW), 62.5 kHz; and acceleration factor (Acc), 2.8. We acquired 3D fast spoiled gradient-recalled acquisition in the steady state (GRASS) sequence (SPGR) T1-weighted images (T1WI) with fat suppression and parameters: TR, 14.8 ms; TE, 2 ms; and flip angle (FA), 15°; NEX, 2; and BW, 62.5 kHz. Further MR imaging parameters included: field of view

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(FOV), 180×180 mm; matrix, 224×224; slice thickness, 1.2 mm. The table and figures show cardiac MR imaging and corresponding macro- and microscopic findings. MR imaging revealed left ventricular concentric hypertrophy and a thin anteroseptal wall with cardiac rupture and hemorrhage, findings identical to macroscopic findings of the axial section of the heart (Fig. 1A). The anterior wall at the rupture site (asterisks) showed high signal intensity (SI) on T1WI (Fig. 1B) and iso SI on T2WI (Fig. 1C). The septal wall at the rupture site (cross symbols) showed high to iso SI on T1WI (Fig. 1B) and low SI on T2WI (Fig. 1C). The outer side (arrow heads) showed iso SI on T1WI (Fig. 1B) and high SI on T2WI (Fig. 1C), whereas the epicardial hemorrhage showed high SI on T1WI and low SI on T2WI (Fig. 1B and C, short arrows).

Myocardial hemorrhage, thin and wavy cardiomyocytes, and edema were histological features of the ruptured anteroseptal wall (Fig. 2C) that were compatible with the features of the early phase of AMI with hemorrhage. The area with high SI on T1WI showed edema without necrotic change (Fig. 2D) compared with the unaffected myocardium (Fig. 2B). The myocardial hemorrhage in the area with iso SI on T2WI (Fig. 2E) was widely immunopositive for glycophorin A, (DAKO, Glostrup, Denmark), a marker of erythrocytes, compared with the area of high SI on T2WI (Fig. 2F). Staining for fibrosis (Mallory-Azan) and hemisiderin deposition (Berlin blue) were negative in the infarcted area. Microscopically, hypertrophic cardiomyocytes in the area without infarction without myocyte disarray or obvious fibrosis suggested systemic hypertension. Histological cross-section of the left anterior descending artery revealed plaque erosion of an advanced atherosclerotic lesion with occlusive thrombus formation, and the thrombus was considered the cause of the AMI with cardiac rupture at the anteroseptal wall.

Discussion

Cardiac MR imaging in vitro generated high resolution images of myocardial thinning, rupture, and epicardial hemorrhage in a patient with AMI. High SI on T1WI and low SI on T2WI at the rupture site corresponded to acute myocardial necrosis, edema, and hemorrhage.

MR findings of the infarcted lesion were heterogeneous with high to iso SI on T1WI and low to iso SI on T2WI at the rupture site or iso SI on T1WI and high SI on T2WI in the outer side (Table). Published case reports of cardiac rupture describe hypoenhancement of the affected ventricular wall
caused by arterial obstruction or late gadolinium enhancement in cardiac MR imaging,2–4 but detailed MR findings of cardiac rupture have not been reported. T2-weighted images can reveal areas of high SI caused by edema in patients with AMI,5,6 and the high SI on T2WI and edema at the outer side of the lesion of our patient were compatible with these findings. The different findings in the ruptured myocardium might be caused by the presence of myocardial hemorrhage because the MR findings of epicardial hemorrhage were similar. Others have demonstrated that the proportions of oxy-, deoxy- and methemoglobin generated by erythrocyte degradation affect relaxation rates and MR images and that changes in the proportions of hemoglobin contribute to age-related changes in MR signals in hematoma.7,8 Therefore, the heterogeneous MR findings of the ruptured myocardium might be mainly affected by deoxy- and methemoglobin in extravasated erythrocytes. Furthermore, the absence of hemosiderin deposition in the lesion was compatible with the early phase of hemorrhage.

Cardiac ultrasonography cannot easily distin-
guish acute myocardial thinning in AMI from the thin fibrous wall that results from old myocardial infarction (OMI). The necrotic myocardium at the focus of a large transmural myocardial infarction undergoes thinning and ultimate replacement by fibrous tissue. Late gadolinium-enhanced MR imaging is widely used to assess myocardial viability in patients with myocardial infarction,9,10 but AMI and OMI both demonstrate late enhancement. T2-weighted imaging is considered useful to distinguish them; as described, high signal intensity on T2WI can detect tissue edema in patients with AMI, and areas of low to high Si were combined on T2WI of the infarcted lesion described herein. Myocardial thinning with an area of low to iso Si on T2WI might have reflected myocardial hemorrhage (Table).

Although this study was in vitro, MR imaging revealed left ventricular hypertrophy and thinning of the anteroseptal wall with cardiac rupture and hemorrhage, findings identical to the macroscopic features. Two of 3 reports of cardiac rupture visualized by cardiac MR imaging describe either myocardial thinning or fissuring.2,3 Improved speed and image quality and technical advances in software have enabled the use of cardiac MR imaging for diagnosing cardiac rupture.

In summary, cardiac MR imaging in vitro generated high resolution images of myocardial thinning, rupture, and epicardial hemorrhage in a patient with acute myocardial infarction. Myocardial hemorrhage after AMI might affect Si on T1WI and T2WI in cardiac MR imaging.

References