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Left Ventricular Free-Wall Rupture After Acute Myocardial Infarction Imaged by Cardiovascular Magnetic Resonance

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ABSTRACT

This manuscript describes a rare situation where after acute myocardial infarction a patient underwent cardiovascular magnetic resonance (CMR), which identified a previously undiagnosed rupture of the free wall of the LV, leading to successful surgical management.

A 54-year-old man was admitted to the emergency department with intermittent angina at rest that had commenced 12 hours prior to admission. A 12-lead ECG showed ST-segment depression in V3-V4 precordial leads and Q waves in lateral leads. Biochemical cardiac markers showed CK-MB of 350 ng/mL. An admission echocardiogram showed no pericardial effusion. The patient was diagnosed as having non-ST-segment elevation myocardial infarction, and standard medical therapy was initiated prior to early invasive angiography. This showed an 80% stenosis of the left anterior descending artery, an occluded left circumflex, and 80% stenosis of the right coronary artery. The patient became asymptomatic in the acute coronary unit and was booked for surgical revascularization.

Cardiovascular magnetic resonance (CMR) was requested to assess myocardial viability, anatomy, and function on the 4th day after myocardial infarction. This was performed on a 1.5 Tesla GE Signa CV/I scanner (Wakeusha, Wisconsin). Left ventricular short-axis and long-axis steady-state free precession cine images were acquired, and corresponding planes inversion-recovery gradient-echo imaging was used to assess late gadolinium enhancement 10 to 20 minutes after 0.2 mmol/kg intravenous gadolinium contrast agent.

The CMR images showed a significant pericardial effusion, lateral transmural myocardial infarction with large region of microvascular obstruction and localized wall thinning at the border between normal and infarcted myocardium (Fig. 1 and Movie), strongly suggesting the diagnosis of sub-acute ventricular free wall rupture, knowing there had been recent myocardial infarction. Four chamber cine images showed compression of the right atrial wall suggesting imminent cardiac tamponade (Movie). There was also evidence of a linear increase of signal in the lateral wall, enlarging during systole, which may reflect tissue changes due to infarction and microvascular obstruction or possible myocardial splitting. Left ventricular ejection fraction by CMR was estimated at 35%. Segmental function showed akinesia of inferolateral and anterolateral walls, from base to apex and inferior and anterior hypokinesia, with normal thickening of the inter-ventricular septum.

The patient underwent surgical repair with bovine pericardial patch and biological glue (N-butyl-2-cyanoacrylate) (Fig. 2) and surgical revascularization of LAD and RCA territories. The left circumflex artery did not receive a graft due to the lack of myocardial viability demonstrated by CMR. The patient recovered well and was discharged 7 days after surgery.

Data from The National Registry of Myocardial Infarction that reviewed over 300 thousand patients showed that cardiac rupture was responsible for 7.3% of all deaths (1). Risk factors for myocardial rupture have been identified: thrombolytic therapy (2), no previous history of angina or MI, ST-segment elevation or Q wave development on the initial ECG and peak MB-creatine kinase >150 IU/L, anterior MI, age >70, and female gender(3, 4).

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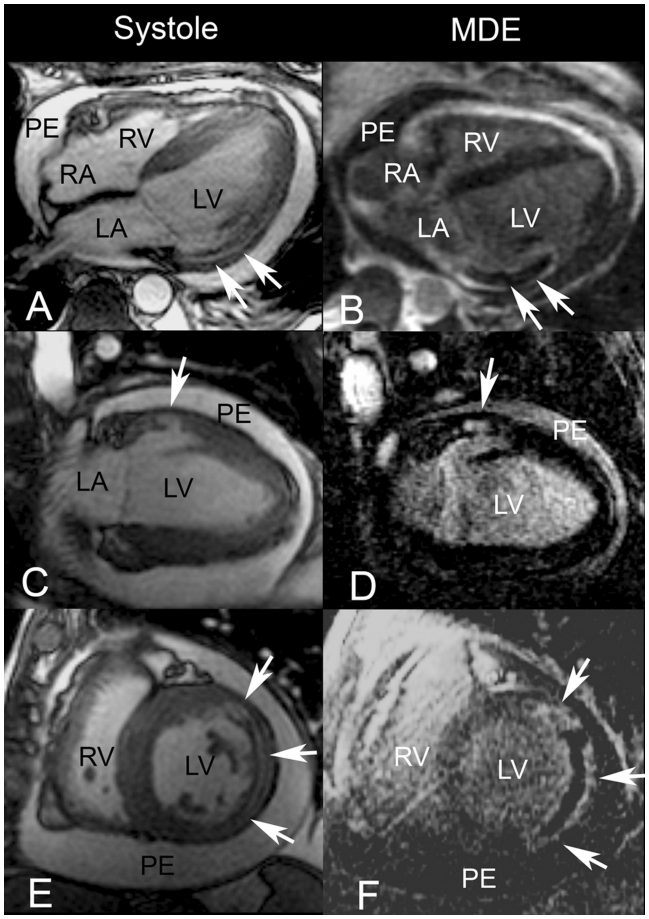

Figure 1. Cardiovascular magnetic resonance in a patient with left ventricular free wall rupture. On the left are the systolic cine images, and on the right column, the myocardial late gadolinium enhancement images of four-chamber (Panels A and B), two-chamber (Panels C and D) and short-axis views (Panels E and F). Arrows indicate a line of increased signal intensity in the cine images (A and E). In panels C and D, the arrows show a severe and localized thinning of anterolateral wall, which suggested myocardial rupture to explain the pericardial effusion. The pericardial effusion (PE) can be seen in all images.

Myocardial rupture occurs within the first 5 days after MI in about one-half of these cases (5–7). A slit-like tear in the infarcted myocardium is usually found after early rupture, commonly affecting the LV anterior or lateral walls near the junction of the infarcted and normal myocardium.

Survival depends primarily upon the rapid recognition of myocardial rupture and provision of immediate therapy. Patients displaying suggestive symptoms, signs, and ECG changes require a bedside echocardiogram. Immediate surgery is indicated if the pericardiocentesis identifies the fluid as blood. Medical therapy aimed at hemodynamic stabilization should also be instituted (8–10).

The use of CMR in a case of cardiac rupture has been reported (11). In our case, the diagnosis was based on CMR and confirmed at surgery; the relatively early diagnosis probably contributing to the successful outcome.

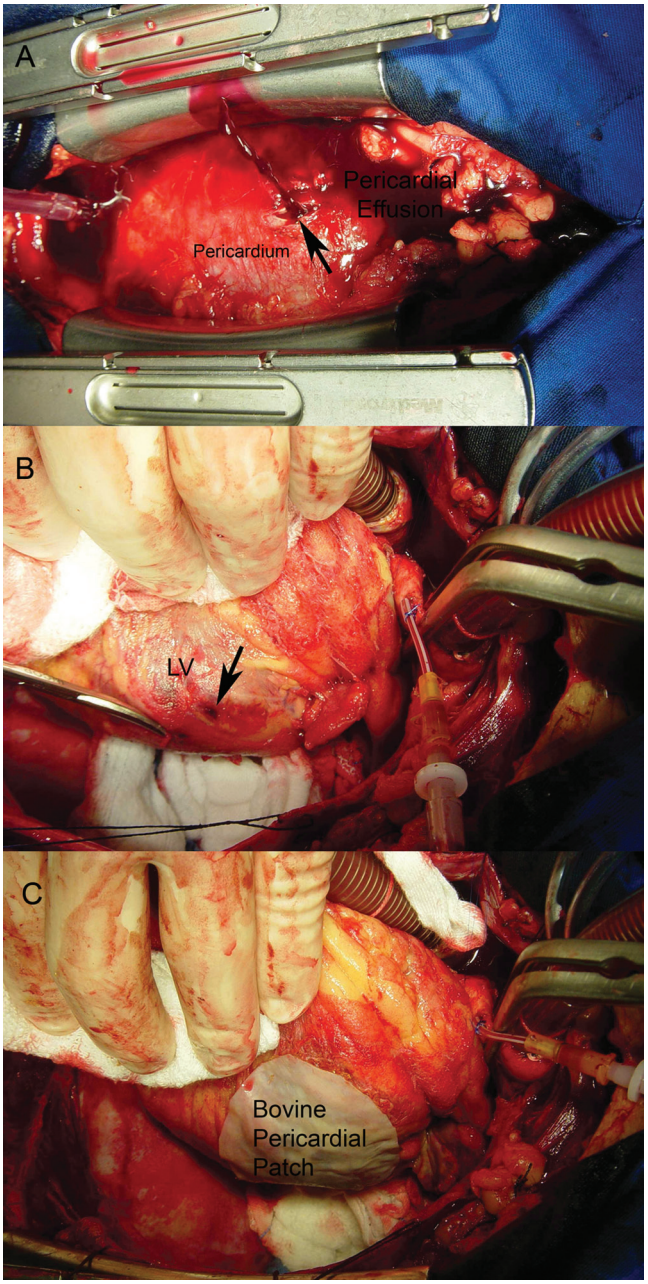

Figure 2. Surgical Views. **A,** A blood jet (arrow) escaping after incision of the tense pericardium. **B,** The location of the ventricular wall rupture. **C,** Surgical repair with a bovine pericardial patch.

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