A 62-year-old man with a history of smoking, hypertension, and hypercholesterolemia was admitted for an inferior ST-segment elevation myocardial infarction (Figure 1). Coronary angiography performed 1 hour after the onset of chest pain revealed that the posterolateral artery was aneurysmal and obstructed. This blockage was treated by percutaneous coronary intervention with balloon angioplasty but without stent implantation. At the end of the procedure, a small area of contrast agent stagnation was found in the pericardial area. Chest x-ray revealed no evidence of pleural effusion or pulmonary edema (Figure 2).

Transesophageal echocardiography (TTE) (ViVid 7; GE Healthcare, Chalfont St. Giles, UK) showed inferior wall hypokinesis with a preserved left ventricular ejection fraction of 50%. We also found a large septal mass and a small pericardial effusion. As shown in Figure 3, this mass expanded into the right ventricle and had a high echodensity. A myocardial contrast TTE revealed complete enhancement of the mass (Figure 4), suggesting a myocardial mass instead of a thrombus.

Cardiac computed tomography (GE Healthcare) with an iodinated contrast agent ruled out the presence of a tumor. However, we discovered a large myocardial hematoma of the inferior interventricular septum, measuring 41×38×70 mm, with active bleeding (Figure 5 and online-only Data Supplement Movie I) from the posterior descending artery. These findings suggested a septal hematoma caused by perforation of the posterior descending artery during the initial percutaneous coronary intervention. Given the risks of hematoma progression or septal rupture, we performed a second coronary angiography to attempt a percutaneous hemostatic procedure. Direct injection into the right coronary artery confirmed extravasation of contrast agent from the posterior descending artery, which suggested active bleeding from this coronary artery (Figure 6A). Because the extravasation disappeared after balloon inflation of the posterior descending artery, the feeding artery was embolized using 3 steel coils. Contrast agent stagnation disappeared completely after coil embolization (Figure 6B).

A repeat-contrast TTE performed 1 day later showed no enhancement of the myocardial hematoma (Figure 7). The patient underwent magnetic resonance imaging 10 days later (clinical 3-T ACHIEVA; Philips Medical Systems, Eindhoven, the Netherlands). Four-chamber imaging showed an abnormal signal area in the right side of the interventricular septum, sparing the subendocardium (Figure 8 and online-only Data Supplement Movie II). This high-signal area was localized to the inferior interventricular septum on short-axis views. The patient was discharged from the hospital on a regimen of antiplatelet agents (aspirin and clopidogrel), a β-adrenergic blocker, an angiotensin-converting enzyme inhibitor, and a statin.

The 3-month postembolization follow-up TTE revealed an impressive regression of the intramyocardial hematoma (Figure 9) with only a slight myocardial mass on the basal interventricular septum. Cardiac magnetic resonance imaging confirmed that the hematoma had regressed as the original area of abnormal signaling had disappeared (Figure 10). Six months after the procedure, the patient remained free from cardiovascular events.

Intramyocardial hematoma is a subacute, partial rupture of the myocardium. These hematomas usually occur after myocardial infarction, chest trauma, surgery, or percutaneous coronary intervention, but they can also develop spontaneously.1 The spontaneous survival rate has been reported to be 10%.2 Standard TTE usually shows an echo-free or hypoechoic intramyocardial neocavity. Previous reports of intramyocardial hematoma describe incomplete enhancement on contrast TTE, compared with the complete enhancement seen with a malignant tumor.3 However, as our case demonstrates, complete enhancement of a myocardial mass can also suggest an intramyocardial hematoma. In such instances, cardiac magnetic resonance imaging should be used to make the correct diagnosis. Intramyocardial hematoma is usually managed surgically,4 but conservative management has also been described. For example, coronary coil embolization has been prescribed for coronary artery rupture during percutaneous coronary intervention.

To the best of our knowledge, this is the first article describing the use of coil embolization to treat intramyocardial hematoma resulting from myocardial infarction. Because our patient recovered uneventfully after the procedure, this treatment could be considered for intramyocardial hematoma.


The online-only Data Supplement is available with this article at http://circ.ahajournals.org/cgi/content/full/121/8/e220/DC1.

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Disclosures

None.

References


Figure 1. Standard 12-lead echocardiogram showing an ST-segment elevation in inferior leads.

Figure 2. Chest x-ray revealing no evidence of pleural effusion or pulmonary edema.

Figure 3. Transthoracic echocardiography in the apical 4-chamber view (A) and the parasternal short-axis view (B) revealing a large septal mass expanding into the right ventricle, characterized by myocardial echodensity (arrowheads).
Figure 4. Contrast transthoracic echocardiography showing complete enhancement of the mass.

Figure 5. Cardiac computed tomography with iodinated contrast agent revealing a large myocardial hematoma of the inferior interventricular septum with active bleeding from the posterior descending artery (A and B). Cardiac computed tomography (volume rendering) showing an active bleeding (arrowhead) from the posterior descending artery (arrow) in left lateral view (C) and inferior view (D).
Figure 6. Right coronary angiography before (A) and after (B) posterior descending artery coil embolization showing the active bleeding (arrowhead) and the steel coils (arrow).

Figure 7. Contrast transthoracic echocardiography performed 1 day after coil embolization of the posterior descending artery, showing a lack of enhancement of the myocardial hematoma.

Figure 8. Magnetic resonance steady-state free precession sequences performed in short-axis view (A) and in apical 4-chamber view (B), highlighting an abnormal signal in the right side of the inferior interventricular septum, sparing the subendocardium (arrows). Delayed enhancement sequence performed in short-axis view (C) and in apical 4-chamber view (D), revealing an area of transmural enhancement corresponding to the inferior infarcted area (arrowhead) and the interventricular septum hematoma (arrow).
Figure 9. Three-month follow-up transthoracic echocardiography showing the regression of intramyocardial hematoma on an apical 4-chamber view (A) and a parasternal short-axis view (B). A small myocardial mass on the basal interventricular septum is observed on the parasternal short-axis view (B, arrow).

Figure 10. Magnetic resonance steady-state free precession sequences showing the hematoma regression on a short-axis view (A, arrow). Delayed enhancement sequence (B) revealing an area of transmural enhancement corresponding to the inferior wall infarct (arrow) and the hematoma sequelae (arrowhead).
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