Getting over a Broken Heart: Intramyocardial Dissecting Hematoma as Late Presentation of Myocardial Infarction

Sumeet Gandhi, MD, FRCPC, Douglas Wright, MD, FRCPC, FASE, and Omid Salehian, MD, MSc, FRCPC, FACC, FAHA, *Hamilton, Ontario, Canada*

INTRODUCTION

Well-described mechanical complications of myocardial infarction (MI) include ventricular septal rupture, papillary muscle rupture with secondary severe mitral regurgitation, and ventricular free wall rupture with either rapid progression of hemodynamic instability and death or containment with pseudoaneurysm formation. Intramyocardial dissecting hematoma is a rare mechanical complication of MI; increased familiarity with echocardiography and multimodality cardiac imaging is essential for a rapid diagnosis and subsequent management.

CASE PRESENTATION

A 42-year-old man presented to the emergency department with a 2-week history of shortness of breath on exertion and new-onset cough. Before this presentation, he sought medical attention and received a presumptive diagnosis of pneumonia and was treated with oral antibiotics, with no alleviation of his symptoms. His symptoms progressed further, with development of orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema in the preceding 3 days. He denied any history of chest pain, palpitations, presyncope, syncope, or claudication. His medical history was unremarkable, with no coronary artery disease, valvular heart disease, or congestive heart failure. He had no known cardiac risk factors and was not taking any regular medications. Before the recent illness, he was physically active and enjoyed recreational biking and running. He worked as a welder, infrequently smoked marijuana, and had a remote history of cocaine use 7 years prior. There was no family history of cardiovascular disease or sudden death.

Physical examination revealed a comfortable patient in no acute distress. Initial evaluation of vital signs revealed a regular heart rate of 99 beats/min and blood pressure of 122/74 mm Hg that was equal bilaterally. He was afebrile, and his oxygen saturation was 93% on room air. Jugular venous pressure was elevated at 6 cm above the sternal angle with a normal waveform and a positive hepatojugular reflux. Carotid pulse was of normal volume and contour, with no audible bruits. Precordial palpation revealed an enlarged apical impulse, which was laterally and inferiorly shifted by one interspace. There were no heaves or thrills palpable. Cardiac auscultation revealed normal S_1 and S_2, with an audible S_3 at the apex. There were no other additional sounds or murmurs. Respiratory examination revealed clear and equal breath sounds bilaterally, with the presence of bibasilar crackles at both lung bases. Peripheral pulses were palpable, and there was minimal bilateral pitting edema at the ankles. Abdominal examination was unremarkable, with no audible abdominal or femoral bruits. Initial blood work revealed a normal complete blood count and normal renal and coagulation profiles. Serum cardiac troponin I level was mildly elevated at 0.075 μg/L (upper limit of normal <0.045 μg/L). Chest radiography showed cardiomegaly with mild interstitial edema. Twelve-lead electrocardiography (Figure 1) showed normal sinus rhythm, with q waves in leads V_1 to V_6 with 1 mm of ST-segment elevation and biphasic T waves. Because of suspicion of a late presentation extensive anterior MI, the patient was transferred to the coronary care unit for ongoing management.

Urgent echocardiography (Figure 2, Videos 1 and 2) was performed and showed a severely dilated left ventricle with severely depressed systolic function and an ejection fraction that was <20%, with multiple regional wall motion abnormalities, including akinesis of the entire apex, anterior wall, inferior wall, and interventricular septum. No hemodynamically significant valvular dysfunction was present. The right ventricle was of normal size, with mild systolic dysfunction. There was a well-delineated echogenic layer separating the mid–left ventricular (LV) cavity from the apex. Between the apex and this echogenic structure, there was a mixture of echolucent and echogenic components, without evidence of communication with the mid-LV cavity by both Doppler color flow and microbubble contrast (Definity; Lantheus Medical Imaging, North Billerica, MA). No pericardial effusion or ventricular septal defects were present (Figure 3, Video 3). The suspected diagnosis was late-presentation extensive anterior MI with the rare mechanical complication of an intramyocardial dissecting hematoma involving the entire apex, with early thrombus formation within the dissection plane.

Coronary angiography (Figure 4, Video 4) revealed single-vessel disease with subtotal occlusion of the proximal left anterior descending coronary artery with visible intraluminal thrombus and mild disease in the left circumflex artery and the right coronary artery. Both computed tomographic (CT) angiography and cardiac magnetic resonance imaging (MRI) were performed for further characterization of the mechanical complication. The initial CT angiogram, obtained on the second day of hospitalization, revealed a severely dilated left ventricle with a crescentic low-attenuation filling defect in the apical portion of the LV cavity. There was a thin rim of myocardium overlying the apex, with no definite full-thickness rupture identified. On the luminal side of the filling defect, there was a rim of myocardium evident to suggest that the filling defect was intramyocardial. Given the superior tissue characterization, cardiac MRI with gadolinium

From the Division of Cardiology, Department of Medicine, McMaster University, Hamilton, Ontario, Canada.

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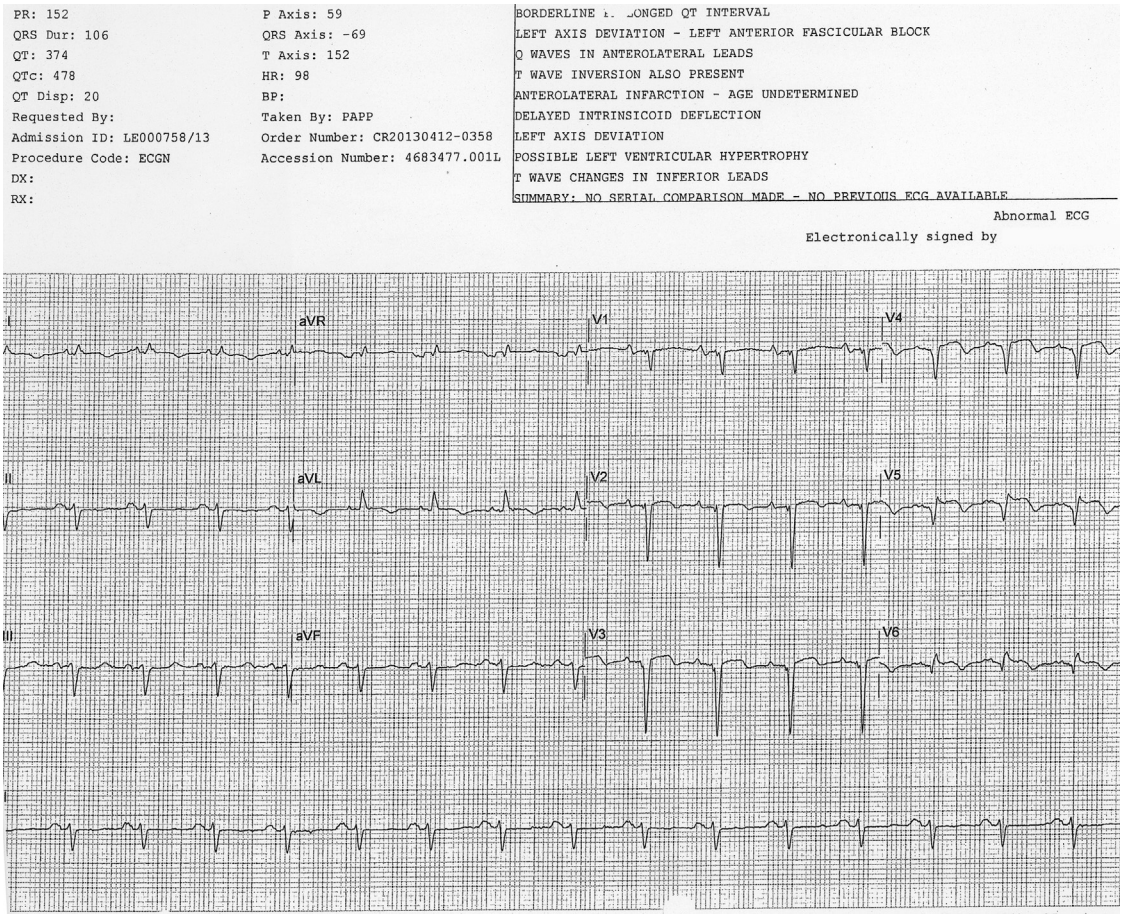

Figure 1 Twelve-lead electrocardiogram demonstrating sinus rhythm, left axis deviation, and Q waves across the precordial leads. Evolving T waves and ST-segment deviation are seen in leads V_3 to V_6.

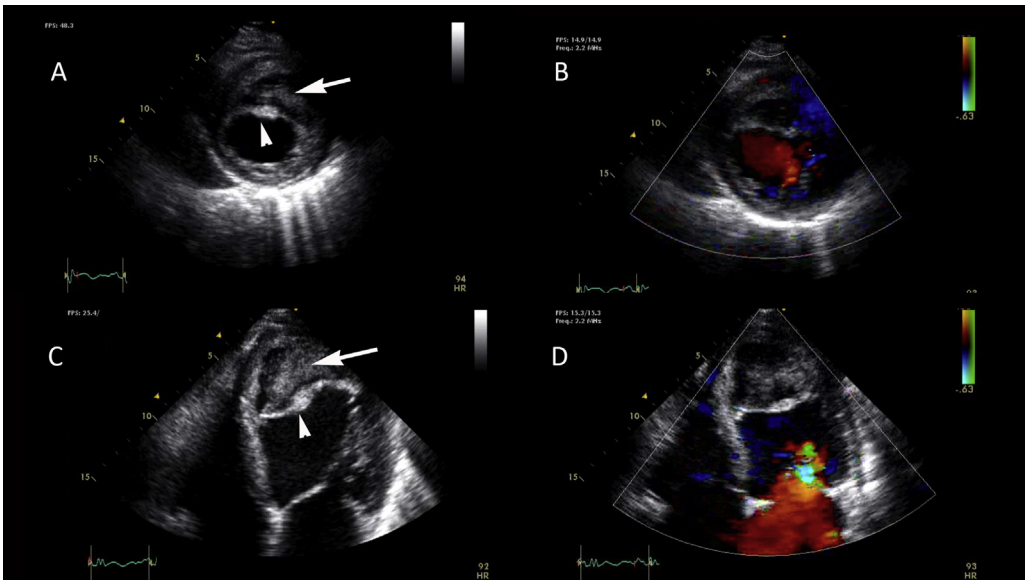

Figure 2 Transthoracic echocardiogram from parasternal **(A,B)** and apical **(C,D)** windows. A layer of myocardium is seen (*arrowhead*), starting in the distal third of the myocardium. Distal to this layer, there is mobile echodensity (*arrow*), consistent with thrombus. Color Doppler images **(B,D)** show no obvious flow within the distal cavity at the Nyquist limit of 63 cm/sec.

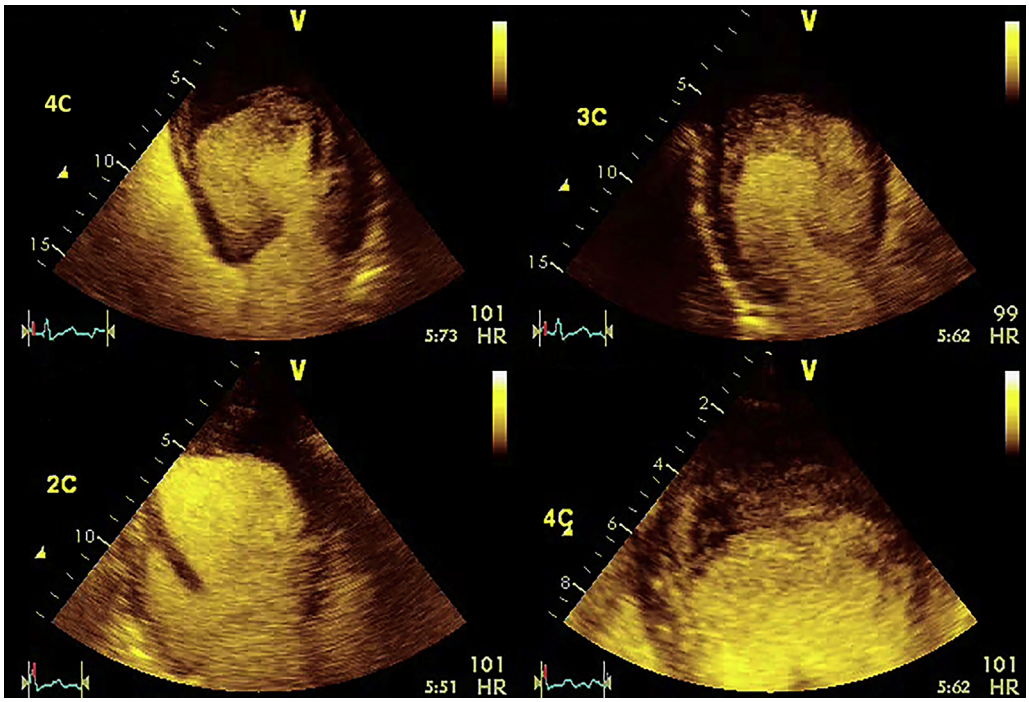

Figure 3 Transthoracic echocardiogram with microbubble injection shown in a quad screen. Apical four-chamber (4C), apical long-axis (3C), and apical two-chamber (2C) images are shown. The *lower right* image is a close-up view of the apical 4C image. A filling defect is seen in the apex of the left ventricle.

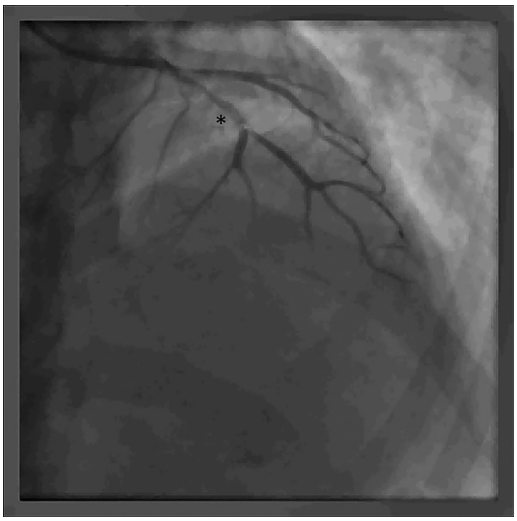

Figure 4 Coronary angiogram with a left injection (right anterior oblique view with cranial angulation) showing a subtotal occlusion of the proximal left anterior descending coronary artery (*asterisk*).

enhancement was obtained on the fifth day of hospitalization (Figure 5, Videos 5 and 6). This showed a multilayered appearance to the thrombus intermediate to a high-intensity layer with the same consistency as blood, indicating a thrombus. Postgadolinium sequences also revealed a large signal void in the apical aspect of the LV lumen, in keeping with an apical intracavitary thrombus. There was no definitive evidence of complete rupture or pericardial effusion. Postgadolinium sequences showed extensive endocardial enhancement, in keeping with a subacute infarction in the area supplied by the left anterior descending coronary artery.

After heart team evaluation, the consensus opinion was that surgical repair would offer a benefit only in the event of progression of the dissection and complete rupture of the myocardium; repair of the infarcted tissue would not improve LV function, and because of the lack of viable myocardium, surgical revascularization would provide little to no benefit. Furthermore, the extent of surgical repair might have an adverse impact on the subvalvular mitral apparatus, leading to impaired mitral valve function. The decision regarding anticoagulation was made alongside our thrombosis experts. The patient was continued on low-dose aspirin at 81 mg/d and started on intravenous unfractionated heparin for anticoagulation to reduce the risk for embolization of the LV intracavitary apical thrombus, as identified on cardiac MRI. Serial echocardiography at 10 and 14 days after admission showed thrombosis of the intramyocardial dissecting hematoma with a more homogeneous appearance and less surface mobility; there was no progression to full-thickness rupture. His hospital stay was complicated by sustained monomorphic ventricular tachycardia that was acutely treated with cardioversion and intravenous amiodarone infusion with subsequent dual-chamber implantable cardioverter-defibrillator insertion for secondary prevention of sudden cardiac death. He was discharged home in stable condition on medical therapy with aspirin, warfarin, and optimal dosing of an angiotensin-converting enzyme inhibitor, a β-blocker, amiodarone, and a statin. Six months after discharge, the patient was seen in the outpatient cardiology clinic and was asymptomatic (New York Heart Association class I–II heart failure symptoms), and he remained physically active, biking and swimming several miles at a time. Echocardiography at the time showed persistence of severe LV dysfunction, absence of LV cavity apical thrombus, and resolution of the ventricular dissection (Figure 6, Video 7). Although arrangements were made for follow-up cardiac MRI, the patient did not attend the appointment.

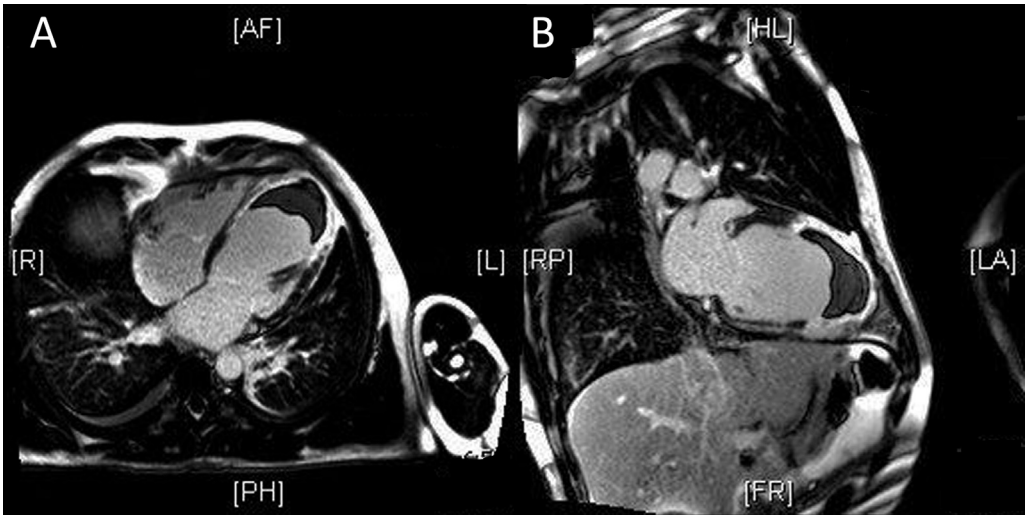

Figure 5 Cardiac MRI from four-chamber **(A)** and two-chamber **(B)** views after gadolinium injection. These show evidence of late gadolinium enhancement at the LV apex. Dense material is seen at the LV apex, representing thrombus within this region. *AF*, Anterior; *FR*, inferior; *HL*, high lateral; *L*, left; *LA*, left anterior; *PH*, posterior; *R*, right; *RP*, right posterior.

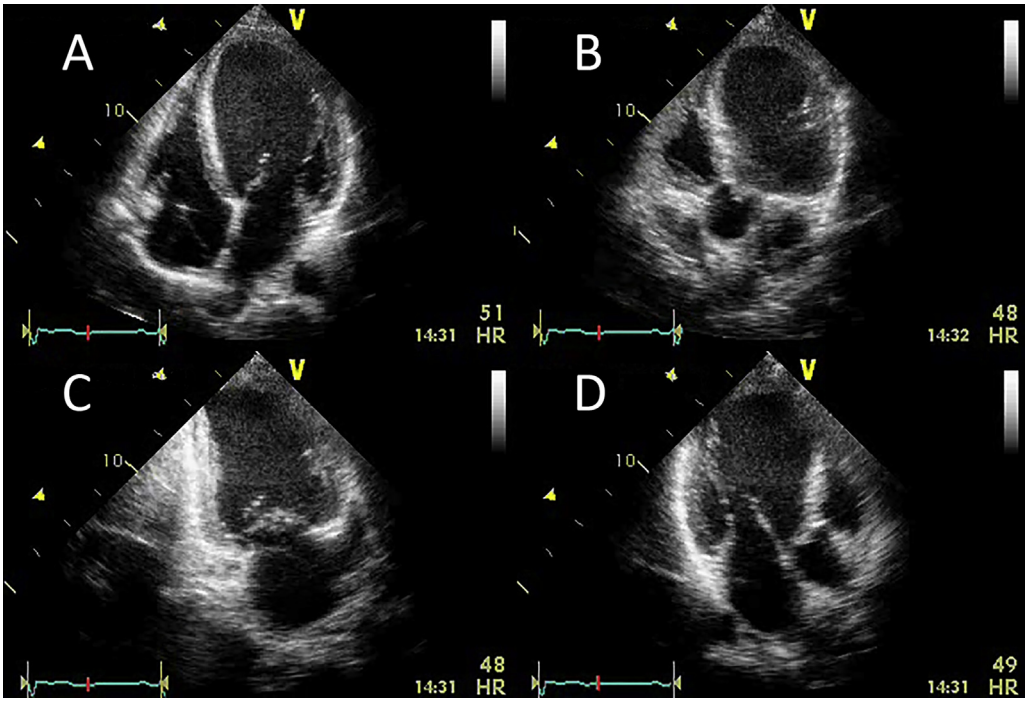

Figure 6 Final transthoracic echocardiogram performed 6 months after initial admission. Images are acquired from the apical windows and represent apical four-chamber **(A)**, apical five-chamber **(B)**, apical two-chamber **(C)**, and apical long-axis **(D)** views. These show further remodeling of the left ventricle, with thinning and scarring of the apical segments. No obvious LV apical thrombus is seen.

DISCUSSION

Complications of a late-presentation MI can be mechanical, arrhythmic, embolic, or inflammatory in nature. Mechanical complications may include ventricular septal rupture, acute or subacute mitral regurgitation due to papillary muscle rupture, and ventricular free wall rupture, which presents as rapid progression of hemodynamic instability, pericardial effusion, and death or pseudoaneurysm formation in the setting of a contained rupture.[1,2] The finding of an intramyocardial dissecting hematoma is quite rare and was unexpected. The underlying pathophysiology includes perforation of the spiral myocardial fibers at the site of the infarction separated by hemorrhagic channels with thrombosis, creating a neocavitation in the myocardium without full-thickness rupture, and preservation of the endocardial and epicardial borders.[3,4] The approach to suspected diagnosis involves multimodality cardiac imaging. Transthoracic echocardiography is the initial noninvasive investigative modality used when there is suspicion of a mechanical complication of MI. Focused assessment of the area of interest includes interrogation in multiple echocardiographic planes as well as assessment of adjacent

cardiac structures, and competency of the interventricular septum and cardiac valves. Echocardiographic findings of intramyocardial dissecting hematoma include an extensive area of infarction of the involved coronary artery, with possible superimposed thrombus and preservation of the endocardial and epicardial border. Color Doppler may show low-velocity flow in the area of dissection. However, full-thickness tear or rupture of the myocardium resulting in pseudoaneurysm formation is a surgical emergency and must be ruled out. Echocardiographic features to identify a pseudoaneurysm include a new pericardial effusion, an outpouching of the LV segment with a narrow neck, and systolic expansion. Color Doppler imaging often identifies somewhat turbulent flow into the neck of the false aneurysm. Microbubble contrast agents can aid in the diagnosis, with contrast visualization in the pericardial space suggesting a communication between the LV cavity and the pericardial space. In cases with presence of superficial thrombus close to the area of interest, a diagnosis of intramyocardial dissecting hematoma may be difficult. Cardiac MRI offers increased spatial resolution and can visualize the extent of thrombosis and hemorrhage into the myocardium and delineate the endocardium. T2-weighted images can identify and differentiate thrombus from blood, as well as subendocardial late gadolinium enhancement and clear delineation of the intact endocardial and epicardial borders of the myocardium, with lack of intense enhancement of the pericardium, suggesting the absence of extension beyond the myocardium into the pericardial space. As in our case, cardiac CT imaging may be also useful to delineate a rim of myocardium on the luminal side of the defect, with lack of intense pericardial enhancement again suggesting an absence of extension beyond the myocardium into the pericardial space. Furthermore, at institutions at which cardiac MRI is not readily available, CT imaging can provide supportive information in suspected cases of intramyocardial dissecting hematoma.

The management of intramyocardial dissecting hematoma is challenging, with only anecdotal evidence to guide clinicians. The principles of management include the decision regarding surgical intervention, anticoagulation, serial imaging to assess for progression and possible myocardial rupture, as well as clinical management of heart failure, ongoing ischemia, and shock. The benefit of systemic anticoagulation includes reducing the risk for systemic embolization from the LV cavity and clot propagation, which must be balanced with the catastrophic risk for full rupture while the patient is on full-dose anticoagulation. Several considerations need to be made regarding surgical intervention. The friable myocardium has poor tensile strength with risk for tearing of the sutures in the myocardium at the site of surgical repair. Intraventricular repair with pericardial patch, as well as Teflon patch and glue, has been used with success.[5] Postoperative LV geometry and mitral valve function must be evaluated at the time of surgical consideration. Hence, multimodality imaging can provide important information to assist surgical decision making.

In our case, serial echocardiography and cardiac MRI within 1 week of hospitalization already began to show subsequent thrombosis of the intramyocardial dissecting hematoma with resolution of intracavitary thrombus on systemic anticoagulation. This leads us to believe that in the absence of a full-thickness tear of the myocardium, conservative management could be recommended because of the high surgical risk and potential healing of the myocardium over time. Patients with intramyocardial dissecting hematoma may have high risk for ongoing morbidity and mortality. These patients are at risk for complications commonly seen in those with ischemic cardiomyopathy, such as ventricular arrhythmias from scarred myocardium, recurrent episodes of congestive heart failure, low cardiac output, LV remodeling and development of functional mitral regurgitation, and sudden cardiac death.

CONCLUSION

Intramyocardial dissecting hematoma is a rare mechanical complication of MI. Echocardiography is essential for rapid assessment in patients with late-presentation MI with suspicion for mechanical complications; intramyocardial dissecting hematoma is a diagnosis of exclusion. Often further multimodality cardiac imaging is indicated to confirm the absence of free wall rupture and to characterize both intracavitary thrombus and hematoma formation and propagation within the dissecting plane. The management of intramyocardial dissecting hematoma is anecdotal and limited to case series observations. Surgical repair may be reserved for those with rapid progression or complete rupture and those who require surgical revascularization. Anticoagulation could be considered to reduce the risk for clot embolization from true LV cavity, but its use should be balanced by the high risk for progression of dissecting plane and hemorrhage into the pericardial space. Patients should be managed by a multidisciplinary team with input from cardiologists with expertise in echocardiography, radiologists and/or cardiologists with cardiac MRI expertise, thrombosis specialists, and cardiac surgeons.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.07.008.

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