Case of a Large Dissecting Intramyocardial Hematoma Treated Conservatively

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Abstract

An intramyocardial dissecting hematoma is a rare mechanical complication after an acute myocardial infarction that carries a high mortality rate. Because intramyocardial dissecting hematomas are associated with multiple cardiac complications, cardiac imaging is an integral component to guiding therapy. We present a case of an intramyocardial dissecting hematoma treated conservatively. Here we explore the role of surgery in patients with intramyocardial dissecting hematomas as well as issues of optimal medical management including the decision to anticoagulate. In conclusion, this report offers a unique commentary on a rare case of an intramyocardial dissecting hematoma.

Résumé

Un hématome disséquant intramyocardique est une rare complication mécanique après un infarctus aigu du myocarde qui comporte un taux élevé de mortalité. Parce que les hématomes dissection intramyocardique associés à de multiples complications cardiaques, l’imagerie cardiaque est une composante intégrale de guider le traitement. Nous présentons un cas d’hématome disséquant intramyocardique traités conservativement. Ici, nous examinons le rôle de la chirurgie chez les patients souffrant d’hématomes dissection intramyocardique ainsi que des questions de gestion médicale optimale y compris la décision d’anticoagulate. En conclusion, ce rapport offre un unique commentaire sur un cas rare d’un hématome disséquant intramyocardique.

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A dissecting intramyocardial hematoma is a rare mechanical complication after an acute myocardial infarction (AMI) that carries a high mortality rate. The pathophysiology of a dissecting intramyocardial hematoma involves hemorrhagic dissection through an area of necrotic tissue between the spiral myocardial fibres of the ventricle. In intramyocardial dissections are most commonly seen as a complication of an acute AMI, but have also been described following blunt chest trauma, and rarely, from cardiac echinococcus infections. Because intramyocardial hematomas are associated with multiple cardiac complications such as ventricular rupture, biventricular dysfunction, and thrombus formation, cardiac imaging is an integral component to guiding therapy. We present a case of intramyocardial dissection treated with conservative management.
Case
A 58-year-old male presented to the emergency department with a one-day history of shortness of breath occurring at rest. Further history revealed progressive dyspnea with exertion over the prior two months and intermittent chest tightness. He denied symptoms of typical angina. His medical history was significant for hypertension, hypercholesterolemia, and peripheral vascular disease with remote aorto-bifemoral bypass surgery. He had a 20-pack year history of smoking. His only medication on presentation was low-dose aspirin.

On examination, his vital signs were as follows: temperature of 36.6°C, heart rate of 98 BPM, blood pressure of 131/105 mmHg, respiratory rate of 20, and an oxygen saturation of 99% on room air. His precordial examination was normal and there was no evidence of volume overload. Initial laboratory investigations showed a mildly elevated high-sensitivity troponin I which peaked at 39 ng/L. Chest radiography showed mild vascular redistribution but no evidence of overt heart failure. ECG showed sinus rhythm with poor R-wave progression but no ST-segment deviation or Q-waves to suggest a prior MI. The patient was subsequently admitted to hospital for further work-up of his shortness of breath.

A transthoracic echocardiogram was performed which showed a dissecting intramyocardial hematoma from the mid segment of the left ventricular septum extending to the apex of the left ventricle (Figure 1 and Supplemental Videos 1 and 2). The hematoma occupied approximately 50% of the left ventricular cavity, and the estimated left ventricular ejection fraction was 25–30%. The hematoma did not appear to receive any flow from the cavity of the left ventricle itself. There was no left ventricular thrombus identified. All other segments of the ventricle were either hypokinetic or akinetic and thinned. Surgical options were explored, but it was determined that the patient would be at excessive risk for morbidity and mortality with surgical resection of the hematoma due to the extent of left ventricular involvement. The patient was started on medical treatment for congestive heart failure. Due to the concern for myocardial rupture, no anticoagulation or anti-platelet therapies were given. Cardiac catheterization was not performed as there was no good option for revascularization.

Six days later, cardiac magnetic resonance imaging (MRI) was performed to reassess the hematoma. The MRI identified a well-defined heterogeneity within the left ventricular mid to apical cavity extending into the apex, raising concern for an intramural dissecting hemorrhage of the myocardium or intracavitary thrombus (Supplemental Video 3). There was also late enhancement of the left ventricular septal and apical segments extending into the right ventricle, indicating an extensive left anterior descending artery territory AMI (Figure 2). Although it was unclear whether the thrombus was contained within the myocardium, anticoagulation was not pursued because of the concern that it would impair healing of, or potentially worsen, the hematoma. A repeat echocardiogram was performed two months after initial presentation, which showed an ejection fraction of 20–25%, a new large apical thrombus, and complete thrombosis of the intramyocardial dissection (Figure 3 and Video 4). This was confirmed by cardiac MRI (Figure 4). The patient was subsequently started on warfarin therapy.

Figure 1: Two-dimensional echocardiogram from four-chamber view showing the somewhat mobile intramyocardial dissecting hematoma occupying the distal one third of the LV cavity (arrow).

Figure 2: Post-gadolinium enhanced cardiac MRI showing an extensive antero-septal and apical transmural MI (yellow arrow) extending into the apical part of the RV and the non-enhancing intramyocardial dissecting hematoma (red arrow).
There is limited data available on the appropriate management of dissecting intramyocardial hematomas following AMI. This is mainly due to their relative infrequency, with the literature limited to case reports and case series. Acute surgical options include application of pericardial patches or other prosthetic material (such as gore-tex or teflon felt), accompanied by excision of necrotic tissue, and coronary artery bypass grafting. A case review in 1993 examined survival rates in 16 patients with intramyocardial hematomas treated either surgically or medically. This case review observed that only 10% of patients treated conservatively survived past 30 days in contrast to all patients treated surgically. While this may suggest that surgical management offers a better prognosis, the finding may have also been due to patients undergoing surgery being at lower risk (due to anatomic factors related to the hematoma or patient comorbidities) compared to those treated conservatively. Conversely, Vargas-Barron et al., examined 15 patients with intramyocardial dissections with a 12-month follow-up period, with 9 patients presenting with an apical free-wall dissection and 6 patients with dissections extending into the septum and/or right ventricle. In the first group, all patients were treated conservatively with all patients surviving to follow up at one year, although 4 patients had worsening heart failure. In the second group, 80% of those treated surgically died, compared to 50% who underwent coronary angioplasty and 100% of those conservatively managed. This study suggests that conservative management may be a reasonable option in patients with less complicated hematomas, while those with more complicated features are at a high mortality risk irrespective of the course of treatment. While our patient likely fit into the latter group with a more extensive dissection, the primary reason for surgical exclusion was the extent of the hematoma without well-perfused residual tissue to surgically remodel the ventricle.

Patients who are conservatively managed for an intramyocardial hematoma are at high risk for further major adverse events and require close follow-up. Concomitant heart failure secondary to MI can lead to left ventricular dysfunction and significant comorbidities, and treatment with proven heart failure medications is essential. Patients with a reduced ejection fraction are also at increased risk of apical thrombus formation. In patients with a myocardial dissection, the decision to anticoagulate must carefully balance the increased risk of stroke and possibility of dissection extension, a potentially devastating consequence. Studies investigating ventricular remodelling after an AMI suggest substantial remodelling, infarct thinning, and reduction of infarct extent typically occur within the first month of healing. Thus, deferring any anticoagulation for at least 4 weeks may be prudent to allow for healing of the hematoma, as long as no clear indications (e.g., left ventricular thrombus) arise.

Discussion
This case describes a left ventricular dissecting intramyocardial hematoma which was likely the result of a late presenting AMI. Although coronary angiography was not performed to identify the culprit lesion, based on non-invasive imaging we suspect that the myocardial dissection originated in the left ventricle following an extensive antero-septal AMI. While the mild troponin elevation and lack of overt ischemic signs on electrocardiogram challenge this assertion, the severe regional hypokinesis and signs of ventricular thinning and remodelling on echocardiogram and MRI suggested infarcted tissue, likely in the LAD territory.
In our patient, a follow-up echocardiogram performed after two months revealed a large apical thrombus, and anticoagulation was initiated at that point in time.

Little is known about the long-term survival of conservatively treated dissecting intramyocardial hematomas. In a study of 8 patients with intramyocardial dissecting hematomas treated with medical management, six were alive at a mean follow up of 12 months. One case report has identified a case of a medically treated intramyocardial dissection with event free follow up extending to 40 months. Cases of prolonged survival seem to be related to a decrease in size or complete resolution of the hematoma, as was seen in our patient, underscoring the need for serial cardiac imaging both to determine prognosis and to guide therapeutic decisions. With improvement or resolution of the hematoma, the primary risks of morbidity and mortality will likely be related to heart failure as well as arrhythmias from scarring; long-term prognosis will depend on optimal heart failure management (e.g., evidence based heart failure medications and evaluating for implantable cardioverter defibrillator [ICD] and cardiac resynchronization therapy [CRT] placement). Finally, in appropriate patients, cardiac transplantation may be considered as a treatment option.

References
**Supplemental Materials**

Video 1. Two-dimensional echocardiogram from four-chamber view showing the somewhat mobile intramyocardial dissection occupying the distal one third of the left ventricular cavity. There is akinesia of the distal and apical anterior and septal myocardial segments and reduced left and right ventricular systolic function.

Video 2. Echocardiogram with Definity contrast from four-chamber view showing contrast does not enter the contained intramyocardial dissection.

Video 3. Cine cardiac MR imaging in four-chamber view showing the dissection extending from the mid interventricular septum to the mid anterolateral segment and occupying the left ventricular apex.

Video 4. Two-dimensional echocardiogram from four-chamber view showing a large wall-adherent left ventricular apical thrombus. Left ventricular systolic function is severely reduced.