

# Two rare complications of myocardial infarction: a case report

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## Background

Mechanical complications following myocardial infarction (MI) have decreased in incidence due to the widespread use of early percutaneous coronary revascularization methods. We describe two rare complications as well as their natural history and uncertainties of the ideal management approach.

## Case summary

Sixty-two-year-old female with multiple cardiovascular disease risk factors who had a delayed presentation of ST-segment elevation myocardial infarction and went on to develop dissecting intramyocardial haematoma progressing to ventricular septal rupture and coronary ventricular fistula.

## Discussion

Intramyocardial haematoma is a rare complication of MI. It is considered to be part of the continuum of myocardial rupture which our patient eventually developed in the form of ventricular septal defect. The second rare entity in the same patient was development of a coronary ventricular fistula of the infarct-related stented, artery. The best way of managing dissecting intramyocardial haematoma is unclear with conflicting data between conservative and invasive strategies. Our patient failed the conservative approach as she progressed to frank myocardial rupture.

## Keywords

Case report • Mechanical complications of myocardial infarction • Dissecting intramyocardial haematoma • Coronary ventricular fistula • Ventricular septal rupture

## Learning points

- Increase awareness of rare but life-threatening complications of myocardial infarction namely dissecting intramyocardial haematoma and coronary ventricular fistula.
- Familiarize providers with the appearance of dissecting haematomas on various imaging modalities and their natural history.
- The appropriate strategy for managing dissecting haematomas is still unclear.

## Introduction

Intramyocardial haematoma is a well-established, albeit rare complication of myocardial infarction (MI).<sup>1–4</sup> A recent review reported only 40 cases published in the literature.<sup>5</sup> The assumed pathogenesis of this uncommon condition is either dissection of intramural blood through weakened infarcted myocardial tissue, intramural bleeding due to ruptured intramyocardial vessels, or an acute increase of coronary capillary pressure.<sup>6</sup> Intramyocardial haematoma formation has also been reported following coronary intervention.<sup>7,8</sup> In all the reported cases, this complication was acutely recognized during the procedure.

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Coronary ventricular fistula, mostly seen as a congenital condition, is another uncommon sequel of MI. There have been a few reports with percutaneous coronary intervention (PCI)<sup>9,10</sup> and is also almost always recognized acutely during the procedure.

We present a case where both intramyocardial haematoma and a coronary ventricular fistula were present in the same patient after acute transmural myocardial infarct.

## Timeline

Day 1	Onset of chest pain.
Day 2	Patient sought medical care and found to have anterior ST elevation on electrocardiogram. The mid-left anterior descending artery (LAD) was 100% occluded and percutaneous coronary intervention with drug-eluting stents was performed. Systemic anticoagulation initiated for intramural apical thrombus.
Day 6	Discharged home in stable condition.
Day 11	Patient presented with dyspnoea and was found to have haemorrhagic pericardial effusion necessitating drainage. Cardiac magnetic resonance showed intraseptal dissecting intramyocardial haematoma. Systemic anticoagulation stopped.
3 months	Worsening shortness of breath. Echocardiography showed distal ventricular septal defect (VSD), large left to right shunt and large apical left ventricular aneurysm. Coronary angiography showed distal LAD to left ventricular cavity fistulization. Patient underwent urgent patch VSD repair, ligation of coronary ventricular fistula, and left ventricular aneurysmectomy.

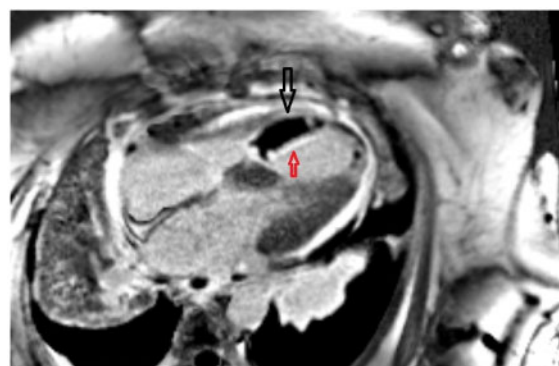
## Case presentation

Sixty-two-year-old with past medical history of hypertension, type 2 diabetes mellitus, and hyperlipidaemia who presented to the hospital with chest pain that started more than 24 h prior. Vital signs were normal except for sinus tachycardia, and cardiac examination revealed friction rub. Electrocardiogram showed ST segment elevation and QS complexes in the anterolateral precordial leads. She underwent emergency coronary angiography (Figure 1) which showed 100% mid-left anterior descending artery (LAD) occlusion. This was successfully treated with PCI and two drug-eluting stents were placed resulting in thrombolysis in myocardial infarction 2 flow (Supplementary material online, Video S1). Echocardiogram showed left ventricular ejection fraction of 25% and possible apical left ventricular thrombus for which systemic anticoagulation was initiated. Patient was discharged in stable condition 6 days later.

Patient presented again 5 days after discharge, 11 days post-MI, with symptoms of nausea, vomiting, drowsiness, and shortness of breath. An urgent echo showed large pericardial effusion with signs of early tamponade as well as a blood clot in the pericardial space. Given a concern for a contained free wall rupture, she was taken



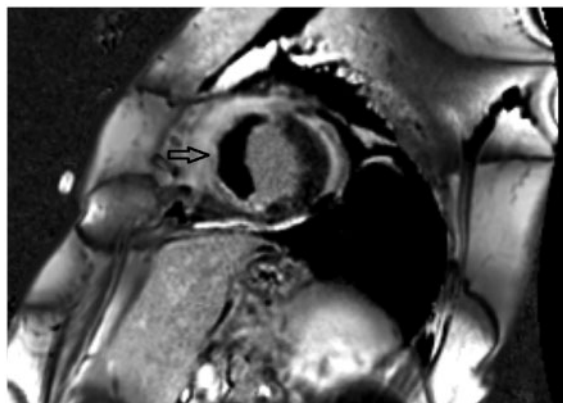
**Figure 1** Left coronary angiography showing 100% mid-left anterior descending artery occlusion.



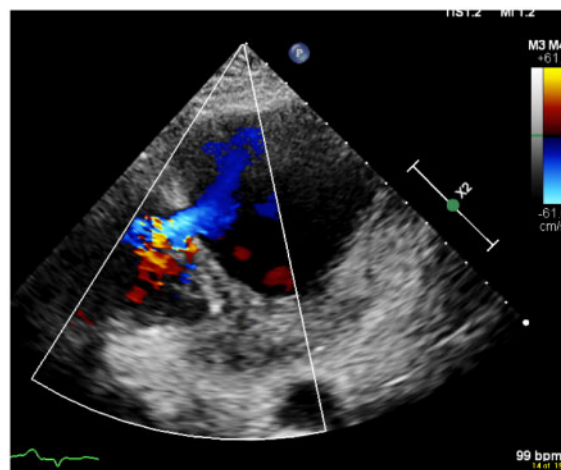
**Figure 2** Cardiac magnetic resonance delayed enhancement PSIR sequence long-axis view showing intramyocardial haematoma involving the interventricular septum. PSIR, phase sensitive inversion recovery.

emergently to the operating room and underwent subxiphoid pericardial window. Four hundred millilitre of bloody pericardial fluid was removed, and there was no evidence of free wall perforation. The aetiology for pericardial effusion was thought to be post-MI pericarditis. Subsequent imaging using cardiac magnetic resonance imaging revealed an intraseptal haematoma (Figures 2 and 3, Supplementary material online, Videos S2a, S2b, S3, and S4). Given the risk of rupture, decision was made to stop systemic anticoagulation.

Patient did well with close outpatient observation until almost 3 months post-MI, when she requested an office visit due to 2 weeks of worsening dyspnoea. An urgent echo was done showing distal ventricular septal defect (VSD) with large left to right shunt (Figure 4, Supplementary material online, Video S5) as well as large-sized apical



**Figure 3** Cardiac magnetic resonance delayed enhancement PSIR sequence short-axis view showing intramyocardial haematoma involving the interventricular septum. PSIR, phase sensitive inversion recovery.



**Figure 4** Transoesophageal coloured Doppler echocardiogram transgastric short-axis view showing a large ventricular septal defect with left to right shunt.

aneurysm. Right heart catheterization showed Qp/Qs of 2.07. Coronary angiography showed distal LAD coronary-ventricular fistula ([Supplementary material online, Video S6](#)). Patient then underwent surgical ligation of coronary ventricular fistula, patch repair of a 2 cm sized VSD with left ventricular aneurysmectomy. Patient was eventually discharged in a stable condition.

## Discussion

Mechanical complications of acute MI carry a high mortality rate and the timely recognition and treatment are imperative to prevent poor outcomes. Among the rare complications is dissecting intramyocardial haematoma which is considered part of the continuum of myocardial rupture.<sup>11</sup> Another rare complication is coronary-ventricular fistula.<sup>10</sup>

Our patient had late presentation ST-elevation myocardial infarction complicated by the development of dissecting intramyocardial haematoma within the infarcted myocardium, ventricular septal rupture, and eventually infarct-related, post-PCI, coronary-ventricular fistula.

Another potential mechanism that seems less likely in our patient is PCI-related dissecting haematoma formation and eventual myocardial rupture leading to a fistulous coronary ventricular connection. This seems unlikely given 3 months interval between coronary intervention, the diagnosis of ventricular septal rupture, and the fistula. As previously stated, post-PCI dissecting haematoma is usually acutely recognized.

To our knowledge, the combination of intramyocardial dissecting haematoma and coronary ventricular fistula has never been reported in the literature. And whether the two have a causative association remains largely unclear.

The best management strategy of post-MI intramyocardial haematomas remains controversial due to lack of significant experience with this uncommon condition. One retrospective analysis showed almost 90% mortality with medical management.<sup>3</sup> Newer analyses,

however, are showing equivalent results between surgical and medical interventions except in the case of right ventricular involvement where the surgical option is superior.<sup>12–14</sup>

In our patient, initial conservative management failed to prevent myocardial rupture with VSD ensuing 3 months after the acute infarct. Despite these complications and subsequent surgical interventions, she is still alive 2 years since her presentation.

## Lead author biography



Dr Osama Mahmoud is a 2nd year cardiovascular disease fellow at Geisinger Medical Center Danville, PA. He obtained his MBBCH degree from Suez Canal University faculty of medicine in 2010. After which he joined the family medicine residency program at Suez Canal University hospitals. In 2014, he joined the internal medicine residency program at Saint Agnes/Ascension health hospital Baltimore, MD and subsequently a Cardiology

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## Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

**Consent:** The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

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