

Intramyocardial dissecting haematoma mimicking left ventricular clot, a rare complication of myocardial infarction: a case report

Tjen Jhung Lee *, Aslannif Roslan, Khai Chih Teh, and Azmee Ghazi

Department of Cardiology, Institute Jantung Negara (National Heart Institute), 145, Jalan Tun Razak, 50400 Wilayah Persekutuan Kuala Lumpur, Malaysia

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Background

Intramyocardial dissecting haematoma is a rare complication of myocardial infarction (MI) associated with high mortality rates. Studies and research of this occurrence are limited largely to isolated case reports or case series.

Case summary

We report a case of late presenting MI, where on initial echocardiogram had what was thought to be an intraventricular clot. However, upon further evaluation, the patient actually had an intramyocardial haematoma, with the supporting echocardiographic features to distinguish it from typical left ventricular (LV) clot. While this prevented the patient from receiving otherwise unnecessary anticoagulation, this diagnosis also put him at a much higher risk of mortality. Despite exhaustive medical and supportive management, death as consequence of pump failure occurred after 2 weeks.

Discussion

This report highlights the features seen on echocardiography which support the diagnosis of an intramyocardial haematoma rather than an LV clot, notably the various acoustic densities, a well visualized myocardial dissecting tear leading into a neocavity filled with blood, and an independent endocardial layer seen above the haematoma. Based on this report, we wish to highlight the importance of differentiating intramyocardial haematomas from intraventricular clots in patients with recent MI.

Keywords

Intramyocardial haematoma • Dissecting haematoma • Complication of myocardial infarction • Case report

Learning points

- Intramyocardial haematoma is a rare complication of myocardial infarction.
- Delayed presentation of intramyocardial haematoma can mimic left ventricular clot on transthoracic echocardiogram.
- Serial echocardiogram is useful in evaluating and differentiating between the two pathologies.
- Acute heart failure from intramyocardial haematoma carries a significant risk of mortality.

Introduction

Intramyocardial dissecting haematoma (IMH) is a rare occurrence, manifesting as a sequela of myocardial infarction (MI). The pathophysiology behind this has been postulated to be either rupture of intramyocardial blood vessels into the myocardium as a result of acute myocardial ischaemia, increased tissue friability with decreased tensile strength in the infarcted area, and acute increase in coronary perfusion pressure.^{1,2}

* Corresponding author. Tel: +60168880464, Email: tjenjhung@gmail.com

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This phenomenon can occur in the left ventricular (LV) free wall, LV septal wall, or even the right ventricular free wall.³ We describe a case of LV free wall intramyocardial haematoma in a gentleman who presented with a delayed presentation of anterior MI. The commonest modality for diagnosis is transthoracic echocardiography (TTE). However, in delayed presentations, intramyocardial haematoma may mimic an LV intraventricular clot, which as a complication of MI occurs much more frequently.

Timeline

Time	Event
1959	Born, no significant childhood/adolescent illnesses
Past 20 years	Active smoker
Past 10 years	Background history of hypertension, diabetes mellitus dyslipidaemia, medicated, and followed-up at his local health practice
Early October	Experienced mild numbing chest pain, breathlessness, and dizziness at home. Did not seek medical attention
23 October 2018	Presented to Emergency Department breathless, in overt state of acute pulmonary oedema (APO) Urgent echocardiogram: presence of severe hypokinesia over anterolateral walls, and possible left ventricular thrombus Non-invasive ventilation Coronary care unit care instituted On bilevel positive airway pressure ventilation, intravenous infusion Frusemide infusion and intravenous inotropic support
24 October 2018	Dependant on non-invasive ventilation (NIV) and two inotropes for haemodynamic support Repeat echocardiogram: dissecting haematoma seen over the anterior-lateral wall Renal and liver impairment
25 October 2018	Dependant on NIV and one inotrope for haemodynamic support Renal and liver impairment improving Good diuresis with Frusemide infusion Multidisciplinary team meeting with Cardiac Surgeon and Cardiologist—conservative approach
29 October 2018	Another episode of pulmonary oedema, worsening breathlessness and oxygenation Repeat echocardiogram: dissecting haematoma seen over the anterior-lateral wall. Deteriorating renal function with uraemia
30 October 2018	Intubated and ventilated for type one respiratory failure due to pulmonary oedema Haemodialysis (continuous veno-venous haemodialysis (CVVHD)) instituted
4 November 2018	Clinical condition deteriorating despite escalation of inotropes and CVVHD. On high ventilator setting Patient succumbed due to pump failure

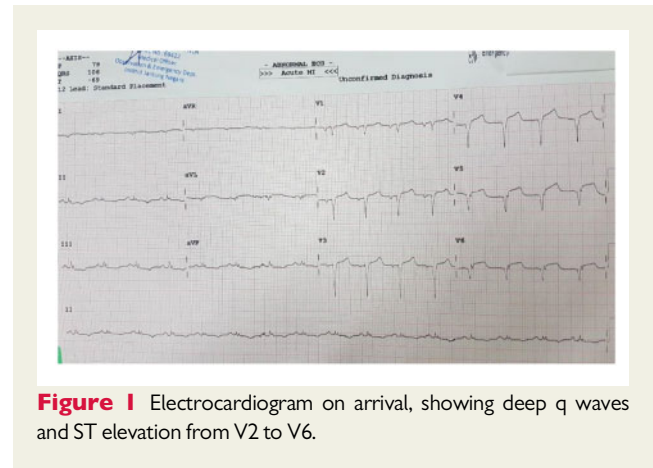


Figure 1 Electrocardiogram on arrival, showing deep q waves and ST elevation from V2 to V6.

Case presentation

A 59-year-old gentleman with a background history of hypertension, diabetes mellitus, and dyslipidaemia presented to our hospital on 23 October 2018, having been referred to our centre for further management of a late presenting anterior MI. He had chest pain for the preceding week but delayed seeking medical attention due to its mild nature. On presentation he reported central chest discomfort, and on examination, was evidently breathless. His blood pressure was 88/70 mmHg, with a heart rate 110 b.p.m., a respiratory rate of 30 breaths per minute, and oxygen saturations of 89% on room air. Fine crackles were audible up to the middle zone of both lungs, the jugular venous pressure was raised and he exhibited pedal oedema in both lower limbs. Auscultated heart sounds were normal. The electrocardiogram showed extensive Q-waves throughout the anterior to the lateral leads (*Figure 1*). Hence a diagnosis of Late Presentation Extensive Myocardial Infarction complicated by Cardiogenic Shock was made.

His care level was escalated rapidly to the coronary care unit and an urgent TTE revealed generalized hypokinesia, a depressed ejection fraction (EF) of 20% and the presence of a large echogenic mass abutting the apical to mid-segment of the anterolateral wall of the LV. The first operator had made a provisional diagnosis of an LV clot. Prior to starting anticoagulation, a repeat TTE was ordered, clearly demonstrating a dissection in the basal anterolateral LV wall, extending into the aforementioned mass. Thus a diagnosis of LV IMH was made. The myocardium seen covering the intramyocardial haematoma was akinetic. He did not have pericardial effusion, and the interventricular septum was intact. There were no valvular abnormalities. Notable biochemical abnormalities at presentation included raised Troponin T at a level of 772 ng/L (normal < 14 ng/L), in keeping with the extensive degree of myocardial damage. His renal and hepatic function were also impaired, indicating organ damage from the cardiogenic shock.

A joint decision for conservative management was made by the cardiologist and cardiothoracic surgeon, because of the markedly reduced cardiac function and multiorgan impairment. In view of these factors, he was deemed to be of prohibitively high risk of surgery, and he did not consent to it.

The treating team embarked on an uphill task of treating his acute heart failure with medical management. Inotropic support together

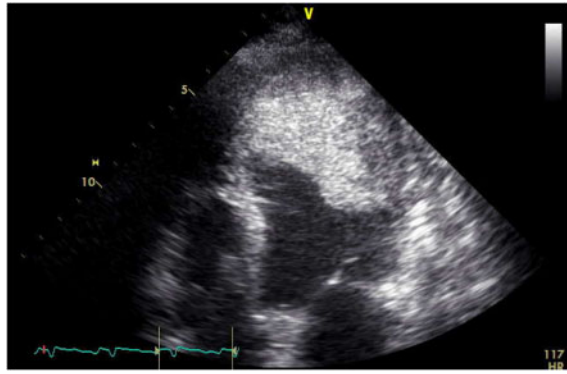


Figure 2 Initial echocardiogram at presentation with mass abutting the anterolateral apical to mid-walls of the left ventricular in the apical four-chamber view. The mass was initially thought to be a left ventricular clot.

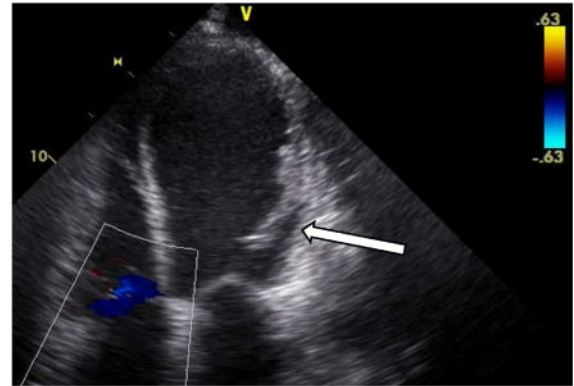


Figure 4 Dissection tear over basal anterolateral left ventricular wall leading into the intramyocardial haematoma.

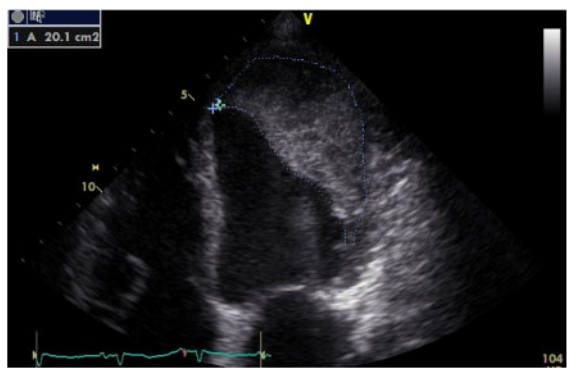


Figure 3 Border of intramyocardial haematoma with dissection edge seen close to basal anterolateral segment of left ventricular in apical four-chamber view. This is the repeat echocardiogram after 3 days of observation in our centre.

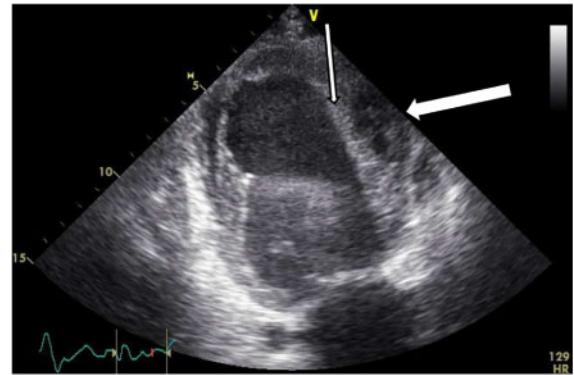


Figure 5 Echo-lucent neocavitation containing haematoma (broad arrow) behind thin and mobile layer of endomyocardium (narrow arrow).

with judicious infusions of Furosemide were the cornerstone of our treatment. He was given single antiplatelet therapy with oral Clopidogrel, and anticoagulated with subcutaneous Enoxaparin. His haemodynamics were closely monitored with real-time measurement of arterial blood pressure, calculated cardiac output, cardiac index, and peripheral vascular resistance by means of the Edwards EV1000 clinical platform.

Early initiatives were taken to address the various organs which were impaired due to the acute heart failure. He received continuous veno-venous haemodialysis for worsening renal function, was electively intubated for progressive respiratory failure, and received judiciously selected antibiotics when he exhibited clinical signs of infection and raised inflammatory markers.

Despite exhaustive medical and supportive management, his condition progressively deteriorated. Death as a consequence of LV failure occurred 13 days after admission.

Discussion

The incidence of intramyocardial haematoma has not been well documented, and thus far isolated case reports or case series have been published. In a case series by Leitman *et al.* published in 2018, 42 cases of IMH have been diagnosed and published thus far. This series also concluded that low EF, age more than 60 years, and late diagnosis, all are predictors of in-hospital mortality.⁴ While the majority of IMH cases have been reported in the context of acute MI, this has also been seen in blunt cardiac trauma, or with the application of a stabilizer device for off-pump coronary revascularization.

Ventricular wall rupture is a well-known complication of MI. Survival rates are poor, with the majority of patients succumbing soon after the diagnosis is made. The friable myocardial tissue resulting from an infarction leads to a tear from the endomyocardium through the myocardium and epicardial tissues and into the pericardial space, leading to rapid LV failure, pericardial tamponade, and imminent death.

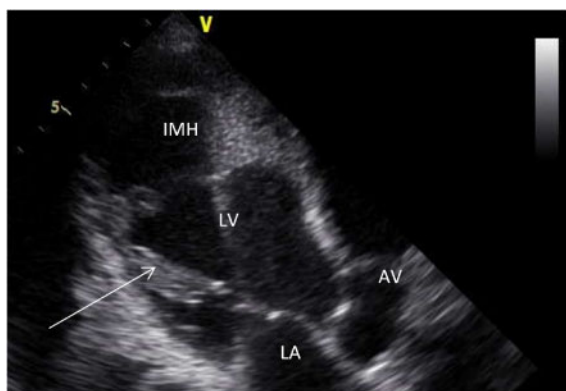


Figure 6 Three-chamber view of left ventricle, left atrium, and aortic root demonstrating the left ventricular basal inferolateral wall dissection tear (arrow) leading into the intramyocardial haematoma.

When the tear through the myocardium is limited within the myocardial tissues, a haematoma can form, leading to an intramyocardial haematoma. In pathological studies of IMH, 79% of cases had a direct tear through the endocardium and myocardium leading into the neocavity filled with haematoma. The remaining 9% of cases showed infiltration of blood through the ventricular wall, without a direct tear or dissection.⁵

The underlying mechanism is thought to be due to haemorrhagic dissection among the spiral myocardial fibres, creating a neocavitation within the left ventricle enclosed by myocardium.⁶ The haematoma formation is most likely due to (i) rupture of intramyocardial vessels into the media, (ii) decreased tensile strength of the infarcted area, and (iii) acute increase of coronary capillary perfusion pressure.

The diagnosis of IMH is usually first made with TTE, but a multimodality approach with computed tomography or magnetic resonance imaging has been encouraged in patients fit for these tests. Unfortunately, as demonstrated in the case above, most patients are simply too haemodynamically unstable to be mobilized for such exams.

Thus Vargas-Barrón et al. in his paper 'Dissecting intramyocardial haematoma: clinical presentation, pathophysiology, outcomes and delineation by echocardiography' had outlined the echocardiographic features that support a diagnosis of intramyocardial haematoma: (i) the formation of one or more neocavitations within the tissue with an echo-lucent centre, (ii) a thinned and mobile endomyocardial border surrounding the cavitory defect, (iii) ventricular myocardium identified in the regions outside of the cystic areas, (iv) changes in the echogenicity of the neocavitation suggesting blood content, (v) partial or complete absorption of the cystic structure, (vi) continuity between the dissecting haematoma and one of the ventricular cavities, (vii) communication between the two ventricular chambers through the myocardial dissection, and (viii) Doppler recording of flow within the dissected myocardium.⁷

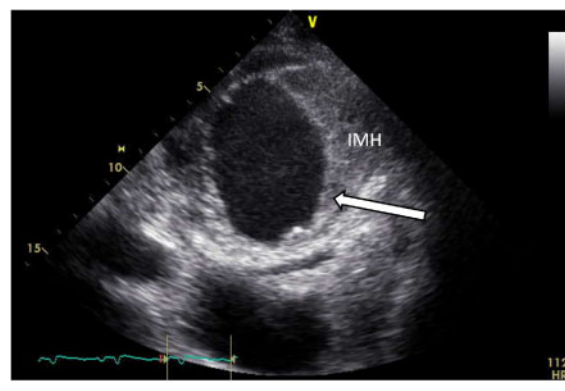


Figure 7 PSAX view delineating crescent-shaped area of intramyocardial haematoma with thin layer of endomyocardium (arrow) separating the haematoma from the left ventricular cavity.

The management of IMH depends largely on the size of the haematoma and the patient's haemodynamic stability. As the majority of patients with IMH have some degree of haemodynamic instability, the most common approach to this condition has been conservative. There have been case reports of surgical management which involved cardiac bypass, median sternotomy, and clot evacuation with subsequent ventricular wall repair. However, such procedures were done in patients who were not in cardiogenic shock.⁸

Several reports have shown good prognosis and resolution in patients with fewer comorbidities, and stable haemodynamic parameters.^{7,9}

Conclusion

The appearance of an echogenic mass within the LV cavity post-MI carries a limited differential diagnosis. While instances such as LV clot may occur more frequently, cardiologists and emergency physicians ought to be aware of the differential of an intramyocardial haematoma. This diagnosis not only carries a poorer outcome, but the management is also different.

Lead author biography



Dr Tjen Jhung Lee, MD, MRCP, is a cardiologist fellow in training at the top cardiac centre in Malaysia, the renowned Institut Jantung Negara (National Heart Institute). He has a keen interest in the field of cardiac imaging and heart failure. He is currently the principal investigator for a clinical study investigating the role of intravenous iron in patients admitted for heart failure. Dr Lee trained at the

Sechenov First Moscow State Medical University in Moscow, where he graduated with first class honours in 2011. He then completed his

residency at various regional general hospitals in Malaysia. In 2016, Dr Lee became a Member of the Royal College of Physicians of the UK. He went on to complete his training and was gazetted as an Internist with the National Specialist Register of Malaysia in 2018.

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

Consent: The author/s 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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