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CASE REPORT
Coronary heart disease

Myocardial infarction secondary to coronary embolus in a patient with left ventricular non-compaction cardiomyopathy: a case report

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Background Coronary embolism is a rare cause of myocardial infarction (MI). We present a case report which emphasizes the importance of intracoronary imaging in these cases to identify the pathophysiological mechanism of MI.

Case summary A 53-year-old male with no past medical history presented to the hospital with typical angina. Electrocardiogram and serum troponin I level trend confirmed non-ST-elevation myocardial infarction. Coronary angiography showed no evidence of any obstructive coronary artery disease, but two small thrombi were noted in the distal first obtuse marginal branch. Optical coherence tomography imaging confirmed this finding in absence of any underlying atherosclerotic plaque rupture or erosion. Cardiac magnetic resonance imaging revealed the diagnosis of non-compaction cardiomyopathy with severely depressed left ventricular function. Transmural MI was revealed by late gadolinium enhancement in the mid-lateral wall. Based on the pathophysiology of the MI confirmed by intracoronary imaging, antiplatelet medications were discontinued, and the patient was discharged on warfarin. Medical therapy was initiated for his cardiomyopathy. The patient recovered well and was asymptomatic at 1-year follow-up visit.

Discussion Intracoronary imaging plays an important role to supplement coronary angiography to confirm the pathophysiology of MI in coronary embolism cases. This is important as it alters management in these patients.

Keywords Acute coronary syndrome • Coronary embolism • Coronary angiography • Imaging • Case report

Learning points

- Coronary embolism should be considered in the differential diagnosis of patients presenting with acute coronary syndromes.
- Intracoronary imaging in useful in the assessment of patients presenting with acute coronary syndromes and angiographically ambiguous lesions to identify the underlying pathophysiology and customize the therapeutic strategy.

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Introduction

Myocardial infarction (MI) secondary to coronary embolism (CE) occurs in about 3% of cases and is usually underdiagnosed.[1] Differentiating this condition from acute coronary syndromes (ACS) is important as it alters the management plan. We present a patient with type 2 MI secondary to CE likely attributed to left ventricular (LV) inter-trabecular thrombus in the setting of non-compaction cardiomyopathy.

Timeline

Day of presentation	Patient diagnosed with non-ST-elevation myocardial infarction and was started on therapy for presumed acute coronary syndrome.
Day 2	Coronary angiography and intravascular imaging revealed coronary embolism in absence of underlying plaque rupture or erosion. Antiplatelet medications and statin were discontinued.
Day 3	Cardiac imaging revealed left ventricular (LV) non-compaction and LV ejection fraction of 21%. Medical therapy was initiated for cardiomyopathy.
Day 4	Patient was discharged from hospital on anticoagulant therapy for coronary embolism and goal-directed medical therapy for cardiomyopathy.
3 months	Left ventricular ejection fraction remained <35% despite maximally tolerated medical therapy, and implantable cardioverter-defibrillator was recommended for primary prevention.
12 months	Patient remained asymptomatic when seen in outpatient follow-up appointment.

Case presentation

A 53-year-old male presented to our hospital with substernal, non-radiating, squeezing chest pain that started 18 hours prior to his presentation. It was associated with dyspnoea, palpitations, and diaphoresis. He was haemodynamically stable, and physical exam was unrevealing. He had no medical problems except active smoking history since he was a teenager. He had no history of premature coronary artery disease in his family. Electrocardiogram showed sinus rhythm with ventricular rate of 64 beats per minute and no evidence of ST elevations (*Figure 1*). Troponin-I peaked at 18.6 ng/mL (normal < 0.04 ng/mL). Global Registry of Acute Coronary Events score was consistent with low mortality risk. Myocardial infarction was the working diagnosis based on the clinical symptoms. Myocarditis or pulmonary embolism could be in the differential diagnoses.

Patient was started on appropriate medical management for presumed ACS, including a dual antiplatelet regimen and heparin drip. Coronary angiogram (Figure 2*A,B* and *Videos 1* and 2) was performed the next day and revealed angiographically normal coronary arteries except two small filling defects noted in the distal first obtuse marginal branch of the left circumflex artery. Optical coherence tomography (OCT) imaging (*Figure 2C* and *Video 3*; Supplementary material online, *Video S1*) confirmed the presence of red organized thrombus in the coronary lumen, with no evidence of plaque rupture. Aspiration thrombectomy was not done due to limited thrombus burden. Transthoracic echocardiogram showed severely dilated LV (LV end-diastolic dimension of 7.3 cm) with severely reduced systolic function at 15%. There were prominent trabeculations of LV suggestive of non-compaction. The right ventricle was normal in size and function. Contrast and agitated saline demonstrated no evidence of interatrial shunt. Cardiac magnetic resonance imaging (MRI) (*Figure 2D–F* and Supplementary material online, *Videos S2–S4*) was performed for further evaluation of the cardiomyopathy, which confirmed the diagnosis of LV non-compaction cardiomyopathy with a ratio of non-compacted to normal myocardium of 4.5:1 in the lateral wall. It also revealed a dilated LV (indexed LV end-diastolic volume of 191 mL/m^2) with a severely reduced systolic function (LV ejection fraction 21%). There was severe global hypokinesis with akinesis of the mid-lateral wall. Early gadolinium enhancement imaging with a long inversion time showed no intracardiac thrombus. Late gadolinium enhancement (LGE) imaging demonstrated transmural infarction of the mid-lateral wall, as well as mid-myocardial LGE of the mid to distal septum, a pattern associated with non-ischaemic cardiomyopathy (*Figure 2G,H*). The right ventricle showed no findings of non-compaction and was normal in size and shape with mildly reduced ejection fraction at 47%. Atrial fibrillation and paradoxical thrombus were considered as a source of the thrombus; however, they were very unlikely given the absence of arrhythmias on telemetry monitoring and absence of shunts on agitated saline echocardiography study.

Given the MRI findings and intracoronary imaging in the absence of any overt source of thromboembolism, it was presumed that the thrombus likely originated from the non-compacted LV myocardium resulting in CE and subsequently type 2 MI. Due to the underlying pathophysiology of MI, antiplatelet drugs were discontinued. Given the low calculated 10-year atherosclerotic cardiovascular disease risk, no statin was prescribed. The patient was discharged home on warfarin with low-molecular-weight heparin bridging. Medical therapy was initiated for the new diagnosis of non-compaction cardiomyopathy, including metoprolol, lisinopril, and spironolactone. The patient was closely followed as an outpatient after his initial presentation. Follow-up echocardiographic imaging at 3 months after initial presentation showed persistently reduced LV ejection fraction at 20–25% despite guideline directed medical therapy. Consequently, the patient was referred to electrophysiology for consideration of implantable cardioverter-defibrillator. On annual follow-up, the patient remained asymptomatic.

Discussion

Coronary embolism can happen due to thrombus dislodgment from the left heart chambers, or can arise from the venous circulation, i.e., paradoxical embolism in the presence of patent foramen ovale. Infrequently, the cause of CE can be iatrogenic, due to structural or coronary procedures.[2] Shibata *et al.* proposed a scoring system which aids in diagnosing this condition. Coronary angiography is the mainstay of diagnosis as it can show a single or multiple abrupt occlusions of distal, small calibre, epicardial coronary arteries, in the

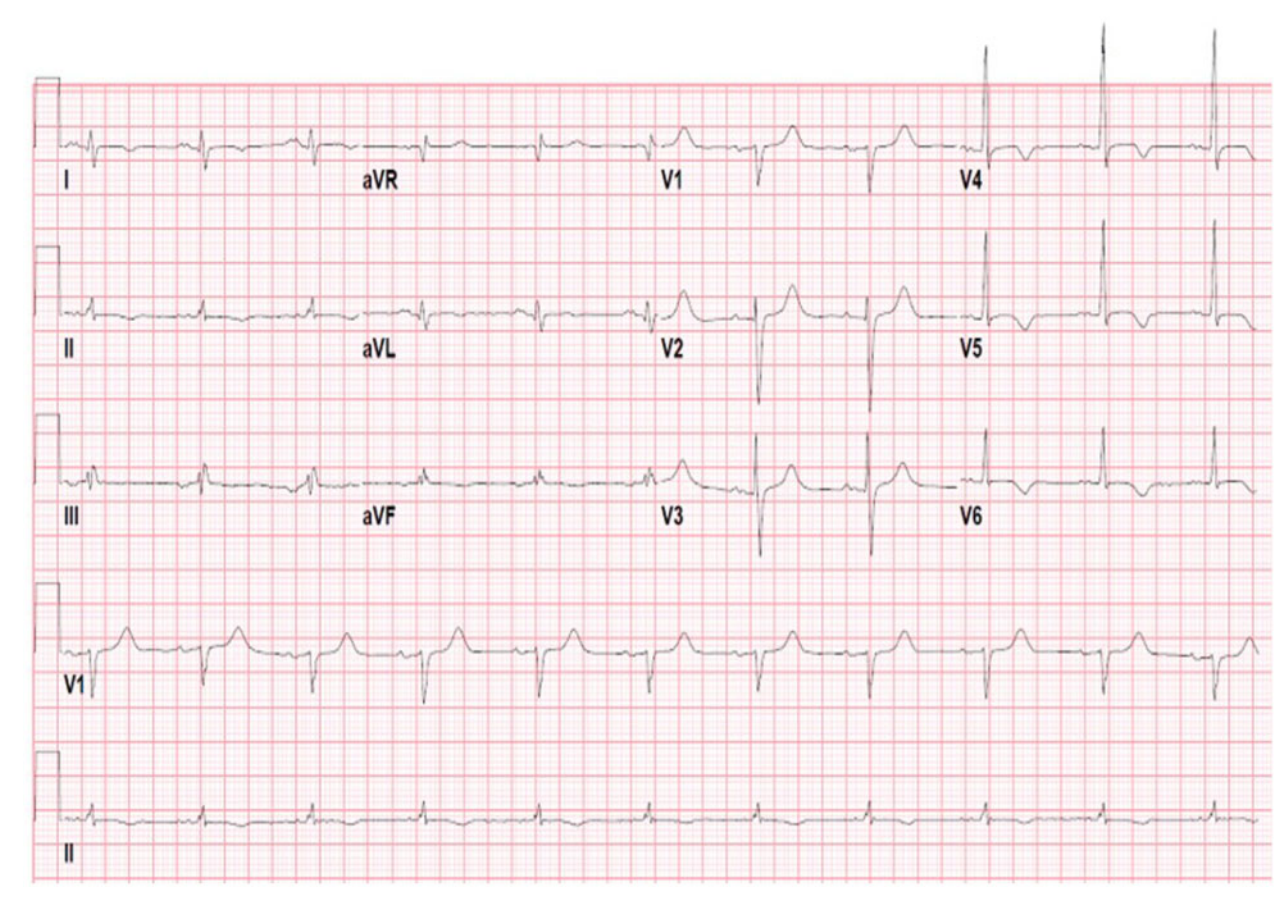


Figure 1 Electrocardiogram at presentation showing sinus rhythm with ventricular rate of 64 beats per minute, non-specific T wave changes in inferior leads, and T wave inversions in lateral leads suggestive of myocardial ischaemia.

absence of underlying atherosclerotic disease.[1] Intravascular imaging, as described in this case, can play a significant role in the diagnosis and further management of CE.[2] Intravascular ultrasound (IVUS) and OCT are the two mainstay intracoronary imaging modalities and have their own strengths and weaknesses. Detection of thrombus is better with OCT due to increased resolution; however, OCT does not have good penetration to assess the depth of the plaque when compared to IVUS.[3] In cases of MI with non-obstructive coronary arteries (MINOCA), intracoronary imaging is critical in excluding acute plaque disruption. Exploration of the underlying pathophysiology in these cases by OCT or IVUS is of paramount importance, as it can guide short- and long-term medical management.[4]

Myocardial infarction secondary to CE is associated with higher mortality, compared to patients with acute atherothrombotic disease.[1] In patients with CE, further investigations should be performed to identify the source of embolism. Contrast transthoracic echocardiography is a first-line test to rule out the presence of thrombus in cardiac chambers. Concomitant performance of an agitated saline study helps to rule out interatrial shunting. In the setting of a patent foramen ovale, studies to rule out any venous thrombosis should be considered. Transoesophageal echocardiogram can exclude any left atrial appendage thrombus. Cardiac computerized tomography and cardiac MRI also have an important role for detection of intracardiac thrombus.[1] There is no role of routine thrombophilia testing in CE patients.[2]

Initial management of patients with CE mirrors that of patients with ACS, until underlying plaque rupture or erosion can be ruled out. In the presented patient case, he was initially loaded with two antiplatelet agents, and started on intravenous unfractionated heparin. Once OCT revealed a diagnosis of type 2 MI due to CE with normal coronary arteries, the antiplatelet drugs were safely discontinued. Despite LV non-compaction being revealed by echocardiography and cardiac MRI, no intracardiac thrombus was identified. In the absence of another source of thromboembolism, it was presumed that CE occurred due to a thrombus that dislodged from the trabeculations of LV in setting of LV non-compaction. Due to the inherent risk of further events, decision was made to place the patient on lifelong oral anticoagulation.

Treatment of CE depends on the extent of embolism, and any underlying atherosclerotic disease. Aspiration thrombectomy, although controversial, is recommended if there is high thrombotic burden. When aspiration thrombectomy is performed, pathological examination of the acquired specimen should be done to aid in the diagnosis of the origin of the embolus.[2] IVUS or OCT can help detect any underlying coronary atherosclerotic plaque and plaque disruption, in addition to guiding any subsequent percutaneous coronary

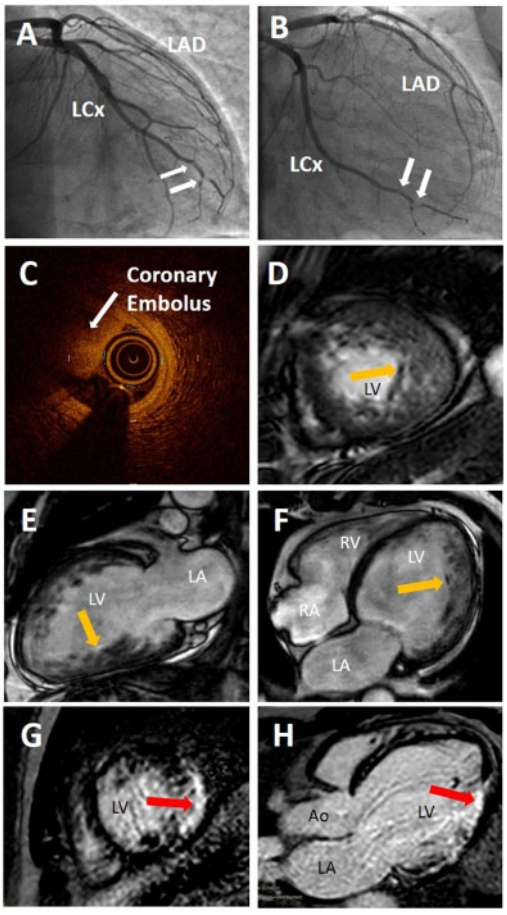
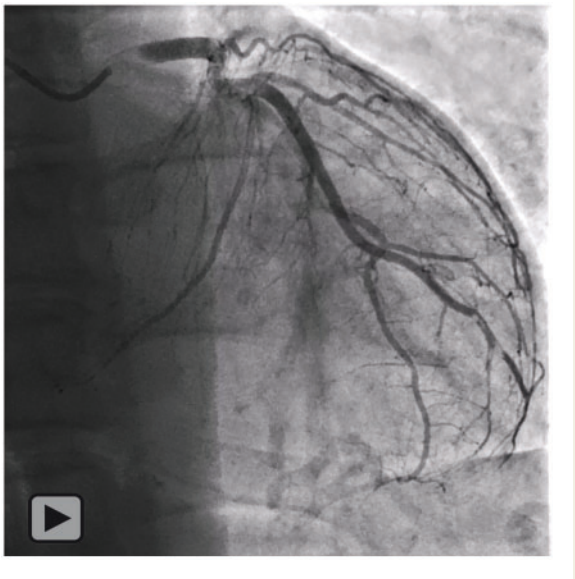
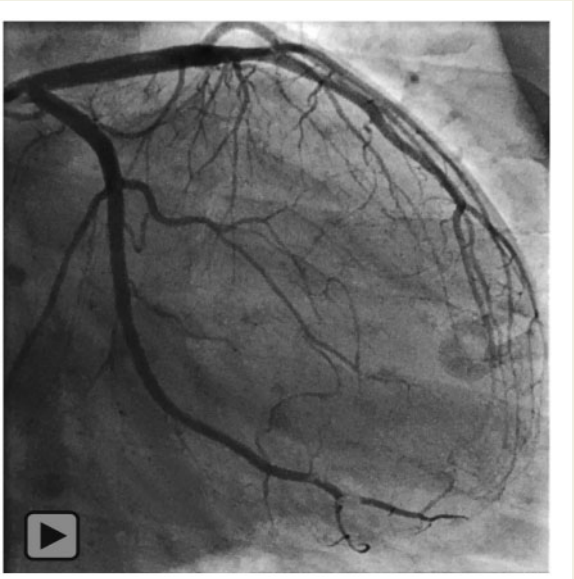


Figure 2 Anteroposterior (*A*) and right anterior oblique caudal (*B*) views of coronary angiography showing two filling defects (suggestive of thrombus, marked by white arrows) in first obtuse marginal branch of left circumflex artery. Optical coherence tomography showing coronary thrombus (*C*, white arrow). Non-compacted myocardium (marked by yellow arrows) seen on cardiac MRI in short axis (*D*), two-chamber (*E*), and four-chamber (*F*) views. Cardiac magnetic resonance imaging demonstrated transmural late gadolinium enhancement (marked by red arrows) consistent with myocardial infarction of the mid-lateral wall, shown in short axis (*G*) and left ventricular outflow tract (*H*) views. Ao, aorta; AP, anteroposterior; LA, left atrium; LAD, left anterior descending artery; LCX, left circumflex artery; LGE, late gadolinium enhancement; LV, left ventricle; MRI, magnetic resonance imaging; RA, right atrium; RAO, right anterior oblique; RV, right ventricle.

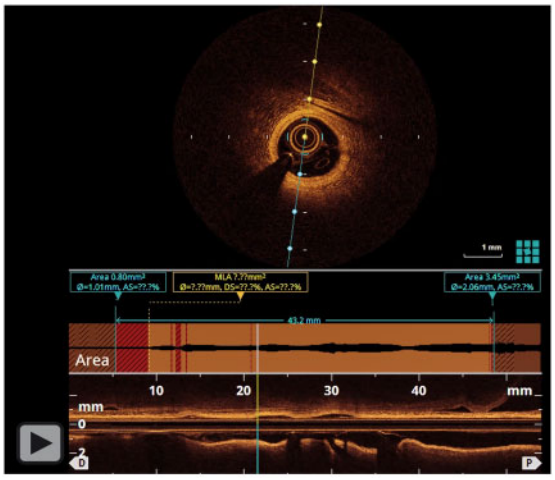


Video 1 Anteroposterior view of coronary angiogram showing angiographically normal coronaries except two filling defects in first obtuse marginal branch of left circumflex artery.



Video 2 Right anterior oblique caudal view of coronary angiogram showing angiographically normal coronaries except two filling defects in first obtuse marginal branch of left circumflex artery.

intervention if needed.[4] The mainstay medical therapy of CE is anticoagulation. The duration of anticoagulation depends on the underlying aetiology. Some patients have a reversible procoagulant state due to recent trauma, hospitalization, or use of some medications, such as oral contraceptives. Patients with vulnerable blood with propensity to thrombosis, including those with active malignancy, or antiphospholipid antibody syndrome should be identified. If there are no risk factors, then short-term anticoagulation for about 3 months should be considered. If the patient has established risk factors, lifelong anticoagulation should be considered. In patients with atrial fibrillation, anticoagulation should be continued indefinitely, irrespective of the stroke risk as calculated by the CHA_2DS_2-VASc score.[1,2] In the absence of documented atrial fibrillation in the acute setting, prolonged ambulatory electrographic monitoring can be considered. There is some literature to support that irrespective of any prior thromboembolic events, patients with LV non-compaction may benefit from anticoagulation to reduce their risk of future thromboembolic



Video 3 Optical coherence tomography pullback of first obtuse marginal branch showing coronary thrombus and no evidence of underlying plaque rupture (0:00:06 seconds).

events.[5,6] Even though no clear-cut recommendations exist, lifelong anticoagulation should be considered in patients with LV non-compaction if they have LV ejection fraction less than 40% and/or atrial fibrillation.[7]

Conclusions

Coronary embolism is an underdiagnosed cause of type 2 MI, which requires diligent history taking, physical examination, and specific investigations to identify the underlying source. Intravascular imaging plays a crucial role in differentiating CE from underlying acute atherothrombosis, coronary spasm, or dissection. Non-compaction cardiomyopathy is a rare aetiology of CE. Medical therapy and avoidance of revascularization if possible is the therapeutic strategy of choice.

Lead author biography

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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 these cases 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 images and associated text has been obtained from the patient in line with COPE guidelines.

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