CASE REPORT

A case of left main coronary artery embolus further embolising to the left anterior descending artery

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SUMMARY

Coronary embolism is an uncommon cause of myocardial infarction. The usual source of a coronary embolus is an intracardiac thrombus or vegetation. Embolisation to the left main coronary artery is an extremely rare event and is usually fatal. We present a case of a 38-year-old woman with embolisation to the left main coronary artery which further embolised distally to the left anterior descending artery leading to a non-ST elevation myocardial infarction. The non-occlusive nature of the left main coronary artery embolus might have led to a favourable prognosis in our patient.

BACKGROUND

Coronary embolism is an uncommon cause of acute myocardial infarction (MI). An autopsy study showed prevalence of coronary embolism in about 13% of infarct cases.¹ Most coronary emboli tend to involve the left coronary system due to flow characteristics and aortic valve morphology.²

Acute left main coronary artery (LMCA) obstruction is a rare condition with most affected patients dying due to sudden death or cardiogenic shock.³ Thrombus formation due to acute plaque rupture is the most common cause of LMCA obstruction.³ However, embolic occlusion of LMCA has also been reported with causes including vegetations from infective endocarditis, intracardiac thrombus, complication of valve repair, papillary fibroelastoma and idiopathic.^{4–9}

In this report, we present a case of left main coronary embolism without significant occlusion and a distal embolism to the left anterior descending (LAD) artery causing an ST elevation MI (STEMI). The source of embolism in our case was unclear, but probably resulted from an intramural thrombus due to pre-existing cardiomyopathy.

CASE PRESENTATION

A 38-year-old African-American woman with a medical history of hypertension, diabetes mellitus, HIV/AIDS and hyperlipidaemia presented with a 1-day history of worsening central chest pain that started while having dinner the night before and radiated to the left arm and shoulder. She also complained of nausea and a single episode of emesis since the same duration. The patient had a history of positive nuclear stress test noted a few months earlier with a fixed perfusion defect and akinesis of the apical part consistent with a prior MI and mildly enlarged ventricle with a fixed ejection fraction of 35%. Further questioning revealed that she had smoked about 5 cigars per day for the

past 20 years. On examination, her blood pressure was 160/90 mm Hg and pulse rate was 90/min. Rest of the examination was within normal limits.

INVESTIGATIONS

Laboratory studies revealed an elevated troponin of 0.3 ng/mL; blood urea nitrogen (BUN) and creatinine were 22 and 1.5 mg/dL, respectively. ECG showed a sinus rhythm, poor R wave progression in V3–V5 and Q waves in V3 and V4. With an impression of non-STEMI, the patient was started on a heparin drip and β -blockers; aspirin and statin were continued. The patient was admitted under cardiology service with strict haemodynamic monitoring and catheterisation was deferred awaiting normalisation of renal function.

TREATMENT

On the second day of admission, the patient's renal function normalised. Meanwhile serum troponin continued to rise from 0.3 to 17 ng/mL. The patient was immediately taken to the catheterisation laboratory. An area of eccentric appearing plaque with no angiographic stenosis and no evidence of plaque rupture was visualised in the LMCA. In addition, a large non-occlusive thrombus was visualised in the LMCA (figure 1). Additional evaluation of the thrombus with intravascular ultrasound revealed presence of a non-mobile thrombus attached to the wall of the LMCA (figure 2).

Figure 1 Coronary angiography showing partial occlusion of the left main coronary artery likely due to a thrombus.

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Figure 2 Intravascular ultrasound of the left main coronary artery revealing the presence of a non-occlusive thrombus attached to the arterial wall.

Multiple aspiration thrombectomies were then performed. There was reduction in the amount of thrombus on subsequent angiography.

Meanwhile, the LAD artery revealed proximal luminal irregularities and distal 100% thrombotic occlusion which was treated with aspiration thrombectomy and ballooning and drug-eluting stent placement. Intravascular ultrasound revealed left main luminal area of 13.5 mm². An emergent surgical consult for possible coronary bypass was performed. It was later concluded that this was likely a thromboembolic event arising from the left ventricle due to her lowered ejection fraction. Hence, bypass surgery was deferred and heparin drip and eptifibatide drip were continued. Meanwhile, an intra-aortic balloon pump (IABP) was placed to optimise diastolic dysfunction. Our plan was to repeat the cardiac catheterisation in a few days to ensure the resolution of the thrombus.

OUTCOME AND FOLLOW-UP

The patient's hospitalisation was complicated by worsening kidney injury postcatheterisation. BUN/creatinine elevated from normal range to 22 and 2.7 mg/dL, respectively. Fractional excretion of sodium (FENA) was <1% suggestive of contrast-induced nephropathy. Meanwhile her haematocrit dropped from 29 to 19.9 possibly due to IABP-induced haemolysis and she required 1 unit of blood transfusion. The patient was taken to the catheterisation laboratory for removal of IABP and a quick assessment of LMCA. The LMCA revealed a stable-appearing eccentric plaque with no visualisation of a mobile thrombus. Distally, LAD artery stent was found to be patent. The patient made a good recovery subsequently and was sent home on long-term anticoagulation with warfarin.

On her follow-up visit at 3 months posthospitalisation, patient was doing well and devoid of any cardiac symptoms. We chose not to do a repeat cardiac catheterisation and intravascular ultrasound as she was doing very well and continue to follow her up closely.

DISCUSSION

Coronary embolism is an uncommon cause of MI. The rarity of coronary embolism has been attributed to the right-angled

take-off of the coronary arteries from the root of aorta, marked difference in calibre between aorta and coronary arteries and filling of coronary artery during diastole.²

Thrombus in the LMCA often occurs due to plaque destabilisation.³ Embolus to the LMCA is rare with very few cases reported. Among the reported cases, sources of emboli include intracardiac thrombus secondary to cardiomyopathy, valvular heart disease or prosthetic mitral valve, peripheral venous thrombi (paradoxical emboli), calcified particles during aortic valve replacement, infective endocarditis and papillary fibroelastoma.^{4–11}

In our patient, the LMCA thrombus was non-occlusive; however, there was an additional thrombus fragment that caused complete occlusion of the LAD. Although, an eccentric plaque was visualised in the LMCA, it appeared stable without any plaque changes. No thrombus was visualised in the cardiac chambers. It is possible that the thrombus originated in the left ventricle given the patient's history of cardiomyopathy with lowered ejection fraction. This argument was further strengthened when an intracardiac thrombus was visualised on the patient's follow-up echocardiography 2 months later. Maddoux *et al*⁶ reported a similar case of LMCA embolus in which the aetiology was unclear but was likely related to cardiomyopathy with mural thrombus.

Management of LMCA occlusion is controversial.¹² Reported treatment options include bypass surgery, percutaneous coronary intervention (PCI), anticoagulation heparin or glycoprotein IIb/IIIa inhibitors and thrombolysis. Current evidence suggests that PCI could be as good as coronary artery bypass graft in the management of LMCA occlusion.¹³ In our patient, the distal LAD artery occlusion was successfully managed with balloon angioplasty and stenting. However, there was some confusion in the management of the LMCA thrombosis, particularly because it was thromboembolic in origin, non-occlusive and the patient was haemodynamically stable. A limited aspiration thrombectomy was attempted and anticoagulation was continued with heparin drip and integrilin. Follow-up angiography performed 48 h later confirmed resolution of the thrombus. Evidence regarding the efficacy of anticoagulants in the treatment of LMCA thrombus is limited to a few case reports.^{12–15}

In conclusion, we report a unique case of thromboembolic MI with complete occlusion of the LAD artery and partial occlusion of the LMCA possibly arising from a mural thrombus. We also document successful resolution of the LMCA thrombus with anticoagulation therapy with heparin and integrilin drip.

Learning points

- ▶ Embolic occlusion of the coronary arteries is a rare cause of myocardial infarction, most often due to embolisation of an intracardiac thrombus or vegetation.
- ▶ Embolisation to the left main coronary artery is an extremely rare event with very few cases reported and can be rapidly fatal.
- ▶ Management of a non-occlusive left main coronary artery embolism is unclear; anticoagulation with heparin and eptifibatide may lead to successful resolution, as in our case.

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Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 Prizel KR, Hutchins GM, Bulkley BH. Coronary artery embolism and myocardial infarction. *Ann Intern Med* 1978;88:155–61.
- 2 Cheng TO. Coronary embolism. *Int J Cardiol* 2009;136:1–3.
- 3 Hori T, Kurosawa T, Yoshida M, *et al.* Factors predicting mortality in patients after myocardial infarction caused by left main coronary artery occlusion: significance of ST segment elevation in both aVR and aVL leads. *Jpn Heart J* 2000;41:571–81.
- 4 Jaffe R, Shiran A, Rubinshtein R. Left main coronary artery occlusion due to thrombus embolization from a prosthetic mitral valve. *JACC Cardiovasc Interv* 2013;6:43–4.
- 5 Leontyev S, Borger MA, Battellini R, *et al.* Embolic occlusion of the left main coronary artery following an isolated aortic valve replacement. *J Card Surg* 2011;26:168–70.
- 6 Maddoux GL, Goss JE, Ramo BW, *et al.* Left main coronary artery embolism: a case report. *Cathet Cardiovasc Diagn* 1987;13:394–7.
- 7 Pawlaczyk R, Lango R, Klapkowski A, *et al.* Low cardiac output due to left main coronary embolism during mitral valve replacement. *J Card Surg* 2010;25:658–60.
- 8 Raju V, Srinivasan M, Padmanaban C, *et al.* Left main coronary artery embolus: unusual presentation of papillary fibroelastoma of the aortic valve. *Tex Heart Inst J* 2010;37:365–7.
- 9 Smith WH, Wolff MR, Kohmoto T. Images in cardiology. Percutaneous removal of embolised vegetation from left main coronary artery. *Heart* 2006;92:84.
- 10 Prizel KR, Hutchins GM, Bulkley BH. Coronary artery embolism and myocardial infarction: a clinicopathologic study of 55 patients. *Ann Intern Med* 1978;88:155–61.
- 11 Waller BF, Dixon DS, Kim RW, *et al.* Embolus to the left main coronary artery. *Am J Cardiol* 1982;50:658–60.
- 12 Gupta R, Rahman M, Uretsky B, *et al.* Left main coronary artery thrombus: a case series with different outcomes. *J Thromb Thrombolysis* 2005;19:125–31.
- 13 Alam M, Huang HD, Shahzad SA, *et al.* Percutaneous coronary intervention vs. coronary artery bypass graft surgery for unprotected left main coronary artery disease in the drug-eluting stents era—an aggregate data meta-analysis of 11,148 patients. *Circ J* 2013;77:372–82.
- 14 Michaels AD, Whisenant B, MacGregor JS. Multivessel coronary thrombosis treated with abciximab (ReoPro) in a patient with essential thrombocythemia. *Clin Cardiol* 1998;21:134–8.
- 15 Rechavia E, Wurzel M. Platelet glycoprotein IIb/IIIa receptor blockade in acute myocardial infarction associated with thrombotic occlusion of the left main coronary artery. *Circulation* 1998;98:1249–50.

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