Thrombus detachment causing lower limb embolism in acute myocardial infarction

Introduction

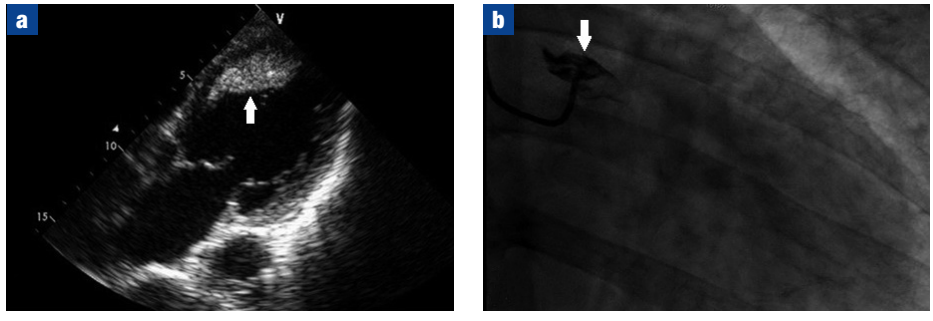
Left ventricular thrombus is a serious complication of acute myocardial infarction. Thrombus detachment can cause arterial embolism. The incidence of lower limb arterial embolism is rare. This article presents a case of a patient with acute anterior wall myocardial infarction who developed a left lower limb arterial embolism as a result of detachment of left ventricular thrombus. The patient received repeated emergency left femoral artery embolectomy, finally in combination with catheter-directed thrombolysis with good prognosis. This case highlights that early revascularization should be performed in patients who have had an acute myocardial infarction to prevent left ventricular thrombus, and that emergency embolectomy combined with thrombolysis should be considered when recurrent thromboembolism occurs.

Discussion

Most left ventricular thrombus occurs within 1 week of acute anterior wall myocardial infarction, usually in the apex of the heart (Solheim et al, 2010). Cardiac chamber endothelial injury and ventricular aneurysm formation are common causes of ventricular thrombosis (Zielinska et al, 2008). The incidence of left ventricular thrombus before reperfusion treatment is as high as 60% (Stokman et al, 2001). Indeed this patient was admitted for acute anterior wall myocardial infarction for 6 days without percutaneous coronary intervention and echocardiography revealed apical wall motion abnormality and thrombosis.

With the development of revascularization, the mortality and complications of acute myocardial infarction have been significantly reduced (Bates, 2014; McLeod et al, 2017). Primary percutaneous coronary intervention

Figure 1. **a.** Transthoracic echocardiography shows an apical thrombus formation in the left ventricle. **b.** Coronary angiography shows left main coronary artery was completely occluded.



CASE REPORT

A 60-year-old man was admitted with exertional chest pain for 4 months which had been aggravated with dyspnoea for 6 days. His risk factors included long-term smoking. His blood pressure was 82/62 mmHg and heart rate was 98 beats/min. Electrocardiogram indicated that he had had an acute anterior wall myocardial infarction. Troponin T and brain natriuretic peptide levels were markedly elevated. The main diagnosis was acute anterior wall myocardial infarction complicated with cardiogenic shock.

Echocardiography showed apical wall motion abnormalities and a left ventricular thrombus at the apex (*Figure 1a*). The patient received dual antiplatelet agents, statins and enoxaparin with dopamine to maintain blood pressure. Coronary angiography on the 7th day after admission suggested a left main coronary artery occlusion (*Figure 1b*); percutaneous coronary intervention was recommended but the patient refused.

On the 8th day, the patient reported pain in the left lower limb. Physical examination showed a pale left lower limb with reduced skin temperature and lack of pulse. Flow imaging indicated acute left femoral artery embolism (*Figure 2a*), and echocardiography revealed a missing left ventricular thrombus. The thrombus was immediately removed via an emergency left femoral artery incision, and angiography confirmed unblocked left arterial circulation (*Figure 2b*). It also revealed an occluded posterior tibial artery, which was unblocked by embolectomy using an catheter and balloon dilatation. On the 9th day, the patient suffered recurrent left lower limb thromboembolism confirmed by flow imaging and an emergency embolectomy was conducted again. Urokinase was continuously administered as the catheter was placed into the left femoral artery and enoxaparin was also applied. Repeat echocardiography did not reveal left ventricular thrombus and thromboembolism did not recur.

The patient underwent coronary angiography and stent implantation on the 21st day and was discharged on the 23rd day. He was followed up for 6 months and was in good health.

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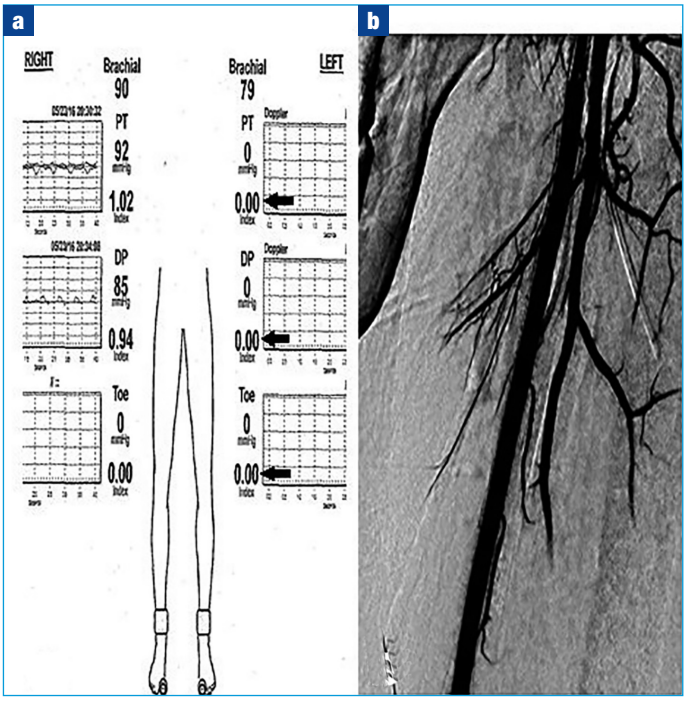
Figure 2. **a.** Flow imaging of the lower limbs show ankle brachial index of left posterior tibial artery, dorsalis pedis and toe artery was 0. **b.** Lower limb angiography shows the left femoral artery was unblocked after thrombectomy.



may decrease left ventricular thrombus by directly unblocking the infarct-related arteries as soon as possible, thus saving more myocardium, reducing the infarct scope, preventing formation of ventricular aneurysm and reducing mural thrombosis.

Transthoracic echocardiography is the most simple and rapid method of locating left ventricular thrombus, with a sensitivity of 95% and a specificity of 86% (Stratton et al, 1982). The occurrence of thromboembolism mainly depends on the morphology of thrombus. Left ventricular thrombus which is protruding, singular, fluffy or irregular in boundary, or has good mobility is prone to detachment, thus causing thromboembolism (van Dantzig et al, 1996). On transthoracic echocardiography the left ventricular thrombus in this case was found to have these morphological characteristics.

Systemic anticoagulation can reduce the risk of left ventricular thromboembolism (Butman, 1991). The administration of heparin or low molecular heparin is recommended as class I by the American College of Cardiology/American Heart Association for patients with ST elevation myocardial infarction who are at high risk for systemic embolism (Antman et al, 2004). This patient was treated with enoxaparin immediately after the discovery of left ventricular thrombus. European Society of Cardiology guidelines recommend that patients with acute ST segment elevation myocardial infarction accompanied with left ventricular mural thrombus should undergo dual antiplatelet treatment combined with oral anticoagulant therapy up to 6 months (Ibanez et al, 2018). However, neither warfarin nor new oral anticoagulants were used after the patient's discharge because the left ventricular thrombus had disappeared by the time he was discharged as a result of continuous anticoagulation and thrombolysis.

Embolectomy and thrombolytic drugs were used for the treatment of acute left lower ischaemia. The first thromboembolism was unquestionably caused by left ventricular thrombus. The patient's acute ischaemic symptoms of the left lower limb were serious and his whole status was very poor. This might have caused lower limb necrosis or even death if emergency embolectomy had not been performed. The patient experienced thromboembolism again after embolectomy despite anticoagulation being given. The thrombus might have resulted from tiny left ventricular thrombus or local thrombosis as a result of surgical trauma on the vascular intima of lower limb artery. The patient thus received repeat emergency embolectomy, which was combined with thrombolysis because of the risk of recurrent thromboembolism. **BJHM**

LEARNING POINTS

- Early revascularization should be performed in patients with acute myocardial infarction to prevent left ventricular thrombus.
- Primary percutaneous coronary intervention potentially decreases left ventricular thrombus by directly unblocking the infarct-related arteries, thus saving more myocardium, reducing the infarct scope and preventing ventricular aneurysm formation.
- Emergency embolectomy combined with thrombolysis should be considered when recurrent thromboembolism occurred on the basis of anticoagulation.

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