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CASE REPORT
Cardiovascular surgery

Surgical removal of multiple left ventricular thrombi with video-assisted cardioscopy: a case report

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Background Left ventricular (LV) thrombus is a lethal complication of coronary artery disease that can lead to embolization and sudden death. There is no clear consensus on the optimal treatment for LV thrombi. There is a paucity of case series about surgical excision of LV thrombus in patients with coronary vessel disease. For that, there is insufficient evidence to support surgical excision of LV thrombus and recommend the optimal timing of this procedure.

Case summary We report a case of a 52-year-old man with a history of percutaneous catheter intervention for mid-right coronary artery lesion 3 years ago. He presented with two-vessel coronary artery disease with three LV thrombi. The thrombi were mobile and protuberant. We performed coronary artery bypass grafting in both vessels and LV thrombectomy with video-assisted cardioscopy.

Discussion Mobile or protuberant thrombus is the most important risk factor for embolization of LV thrombus. On the other hand, LV thrombus size rarely appears in studies as a risk factor for embolization, and when it does, it is a lesser risk factor. There are no case reports describing simultaneous formation of three LV thrombi after myocardial infarction, and it is not known if the risk of embolism is high in such cases. Our patient had very fragile thrombi, and thrombectomy was performed along with coronary artery bypass grafting due to the high risk of embolism.

Keywords Left ventricular thrombus • Multiple thrombi • Coronary artery disease • Myocardial infarction • Video-assisted cardioscopy • Coronary artery bypass grafting • Case report

ESC curriculum 3.1 Coronary artery disease • 7.5 Cardiac surgery • 3.2 Acute coronary syndrome

Learning points

- For patients who are unable to tolerate anticoagulation therapy and those who are perceived to be at a high risk of embolization, surgical removal of the left ventricular (LV) thrombus is one of the options, especially in cases with a mobile, protuberant, or multiple thrombi.
- Because of the high risk of thrombus dislodgement due to cardiac manipulations at the time of operation, thrombectomy should be performed for patients with another indication for cardiac surgery.
- Video-assisted cardioscopy is one of the options for thrombus removal using a transaortic approach facilitating faster recovery of the myocardium from the inflammatory process without sustaining the additional injury compared with ventriculotomy.
- The medical treatment for LV thrombi within 10 days after infarction could reduce the risk of surgery or even avoid surgery.

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Introduction

Left ventricular (LV) thrombus is a lethal complication of coronary artery disease that can cause embolization and sudden death. However, there is no clear consensus on the optimal treatment strategy and its timing. There is a paucity of case series about surgical excision of LV thrombus, and there is insufficient evidence to recommend surgical excision of LV thrombus and its timing.

In our case, the thrombi were mobile and protruding, increasing the risk of embolization. We planned for two-vessel coronary artery bypass grafting (CABG) and LV thrombectomy with video-assisted cardioscopy (VAC).

Summary figure

Thirteen years before admission to our hospital

Percutaneous coronary intervention (PCI) was performed in the context of acute myocardial infarction at that time period for a mid-right coronary artery lesion.

Seven days before admission to our hospital

He presented with dyspnoea, and echocardiography revealed no significant valvular disease and marked cardiac function deterioration [ejection fraction (EF): 30%]. He has started heart failure treatment.

One day before admission to our hospital

Coronary angiography (CAG) was performed to find the cause and revealed two-vessel coronary diseases. Elective PCI was planned.

On the day of admission to our hospital

After controlling heart failure, echocardiography revealed diffuse hypokinesis of the LV wall with three mobile and pedunculated thrombi. Emergency two-vessel CABG was performed along with LV thrombectomy with VAC.

Postoperative day (POD) 0

The patient was extubated.

POD 13

Echocardiography confirmed LV thrombi removal.

POD 16

The patient was discharged from the hospital.

Case presentation

A 52-year-old male patient presented with a chief complaint of dyspnoea for several days and visited a neighbouring hospital with oedema in both lower legs 7 days before the surgery. He underwent PCI for a mid-right coronary artery lesion 13 years ago in the context of acute myocardial infarction and was administered antiplatelet drugs, including aspirin at 100 mg and prasugrel at 3.75 mg, without anticoagulant agents. He also was diagnosed with diabetes, dyslipidaemia, and hypertension. Echocardiography revealed no significant valvular disease or any formation of LV thrombus and marked cardiac function deterioration (EF: 30%). Coronary angiography was performed to find the cause. The CAG showed a new stenosis of mid-left anterior descending artery (LAD) and right coronary artery without elevated troponin or ST-segment on electrocardiography (ECG) (*Figure 1A* and *B*). The laboratory results were as follows: creatinine 1.17 mg/dL (normal: 0.65–1.07 mg/dL), blood urea nitrogen (BUN) 40.6 mg/dL (normal: 8–20 mg/dL), D-dimer 2.0 μg/mL (normal: <1.0 μ/mL), protein C 117% (normal: 64–146%), and protein S 74% (normal: 67–164%). He was administered diuretics and β-blockers for acute heart failure. Elective PCI was initially planned, but on the following day of CAG which was 6 days after the hospitalization in the neighbouring hospital, transthoracic echocardiogram (TTE) showed three mobile LV thrombi with normal LV size (LV internal end-diastolic and end-systolic diameters of 52 and 39 mm, respectively), moderately to severely reduced LV systolic function (EF of 44%), severe hypokinesis–akinesis of the inferoposterior wall (base–apex), severe hypokinesis of the anteroseptal–lateral wall (mid–apex), and diffuse hypokinesis of the LV free wall. The TTE demonstrated no signs of LV aneurysm. The thrombi were sized 35 × 10, 26 × 11, and 13 × 12 mm, respectively. He was transferred to our hospital for surgical consultation. Preoperative computed tomography showed three pedunculated thrombi (*Figure 2A* and *B*). Monitoring the thrombi's progress with oral warfarin was considered; however, given their extreme mobility and protruding nature, there was a high risk of embolism. Therefore, surgery was decided as the best course of action. We planned for two-vessel CABG and LV thrombectomy with VAC (STORZ IMAGE1 H3-Z, HOPKINS Ⅱ TELESCOPE, KARL STORZ, Germany). Cardiopulmonary bypass was established through median sternotomy using standard aortic and bicaval cannulation. The left atrium was accessed via the transseptal approach. An endoscope was used to confirm the apical thrombi, but owing to the difficulty in visualization, the approach was abandoned. Subsequently, the ascending aorta was transversely incised, and three LV apical thrombi were confirmed endoscopically using a transaortic approach (*Figure 3*). The thrombi were carefully removed with forceps. The thrombi were fragile and were easily detached from the LV wall. Coronary revascularization was achieved by grafting saphenous vein grafts and the left internal mammary artery to the posterior descending artery and LAD, respectively. Postoperative pathological examination showed fresh thrombi with no signs of organization. No neoplastic changes or malignant findings were observed. Oral anticoagulant (OAC) therapy was initiated with warfarin [target international normalized ratio (INR) = 2–3]. On POD 7, we confirmed the complete removal of the LV thrombi by TTE and computed tomography (*Figure 4*), and the patient was discharged home in an ambulant condition at 16 POD. He is currently under follow-up care at his previous hospital. Ejection fraction was improved (LVEF: from 30 to 48%), and LV diastolic and systolic diameters were reduced (from 54/47 to 51/40) 3 months postoperatively, with no new intraventricular thrombosis.

Discussion

There are three broad pathogenic mechanisms of formation of LV thrombus.[1] Stasis attributable to impaired ventricular function is a major cause of thrombus formation. The second mechanism is endocardial injury caused by chest wall trauma[2] or even coronary artery spasm[3] as well as ischaemic coronary disease that induces monocytes and macrophages to produce compromised extracellular remodelling such as production of abnormal collagen I and fibrosis.[4] This impairs the integrity of the endocardium predisposing to the formation of LV thrombus. The third mechanism is hypercoagulability induced by inflammation such as cardiomyopathy and infection and by coagulation disorders such as deficiencies of proteins C and S.[5,6] Jordan *et al.*[7] reported that the occurrence of LV thrombus after an acute myocardial infarction is related to the size of the infarct (i.e. the larger the area of myocardial necrosis, the greater is the incidence of mural clot). Anterior wall infarctions typically result in a greater area of necrotic myocardium than do infarctions in other areas. In addition, the stasis caused by the dyskinetic apex, which frequently occurs secondary to anterior infarction, also increases the occurrence and propagation of LV thrombi.[8] Because our case demonstrated no evidence of acute myocardial infarction as indicated by the negative troponin test, it seemed that his LV thrombi formed secondary to ischaemic cardiomyopathy with acute decompensation.

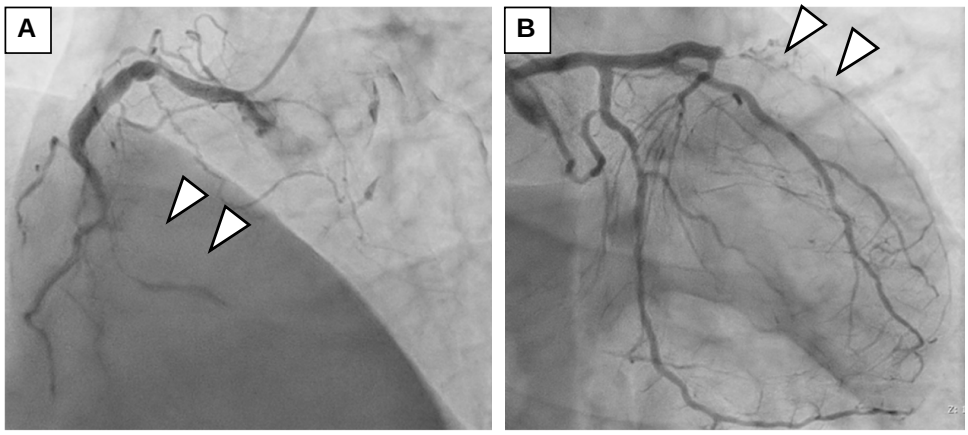


Figure 1 Coronary angiography showing a new stenosis of right coronary artery (*A*) and left anterior descending artery (*B*).

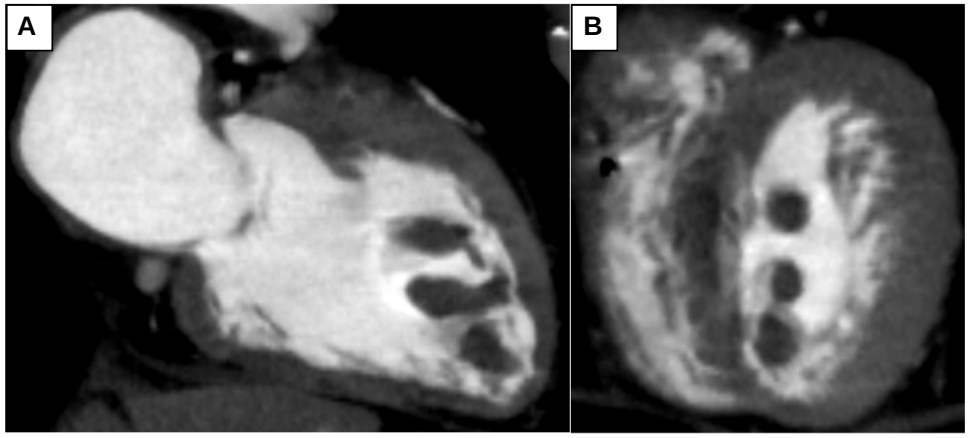


Figure 2 Computed tomography showed multi-thrombi in the left ventricle. (*A*) Long-axis view. (*B*) Short-axis view.

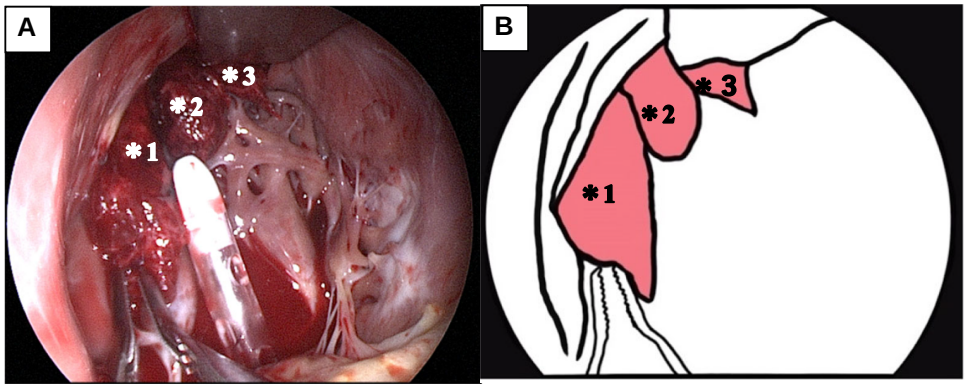


Figure 3 (*A*, *B*) Apical view with video-assisted cardioscopy from aortotomy. *Thrombi.

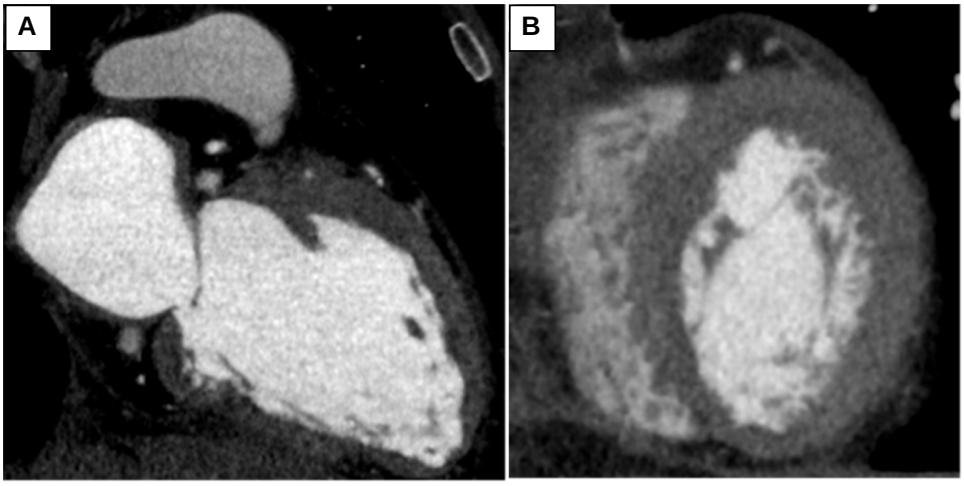


Figure 4 Postoperative computed tomography: the thrombi have been completely removed. (*A*) Long-axis view. (*B*) Short-axis view.

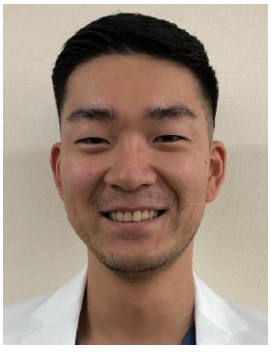
While considering the treatment methods, it is necessary to first clarify the characteristics of LV thrombi that cause embolism. In general, a mobile or protuberant thrombus is the most important risk factor for LV thrombus embolization,[1] and LV thrombus size is rarely detected as a risk factor for embolization; even when it is detected, it is considered a minor risk factor. On the other hand, the embolic risk of multiple thrombi is unknown. However, Cabin and Roberts[9] suggested a relationship between the amount of intraluminal thrombus exposed to cardiac blood flow and the subsequent embolic episodes. Similarly, Cabin and Roberts[9] reported that a protruded thrombus exposed to blood flow on three sides is more likely to dislodge and produce a systemic embolus compared with an intramural thrombus that is exposed to blood flow on only one side. These reports suggest that the multiple LV thrombi in our patient posed a high risk of embolization due to the high exposure to cardiac blood flow. Indeed, Mine *et al.*[10] reported cerebral infarction caused by multiple LV thrombi. The 2022 American Heart Association (AHA) Scientific Statement[1] recommends 3-month OAC therapy with repeat imaging in the acute setting. Therefore, considering these factors, surgical removal of the LV thrombus is one of the options for patients who are unable to tolerate anticoagulation therapy and those who are perceived to be at a high risk of embolization such as in cases with a mobile, protuberant, or multiple thrombi.

In addition, because of the high risk of thrombus dislodgement due to cardiac manipulations at the time of CABG, thrombectomy should be performed for patients requiring cardiac surgery for another indication.[11] Indeed, the LV thrombi in this case were very fragile with a seemingly high risk of embolization. Thus, surgical removal of the thrombi during CABG seemed a pragmatic approach.

There is no clear consensus on the optimal therapeutic modalities for LV thrombus. Video-assisted cardioscopy is one of the options for thrombus removal using a transaortic approach. This facilitates faster recovery of the myocardium from the inflammatory process without the additional injury caused by ventriculotomy.[12] There is no clear consensus on the timing of initiating the optimal treatment. Stratton *et al.*[13] reported the presence of LV thrombi was associated with an increased embolic risk in the first few weeks after experiencing acute myocardial infarction. In fact, all embolic events occurred after 1 month or more of experiencing myocardial infarction, and a previous study showed that systemic emboli typically occur after an interval of ≥10 days after infarction.[8] Emergency surgery may not be needed within at least 10 days of onset, and preoperative exams may be performed and surgical risks may be reduced in those 10 days. Alternatively, the clot may be dissolved and surgery may be avoided. Thus, medical treatment for LV thrombi within 10 days after infarction could reduce the risk of surgery or even avoid surgery. Regarding anticoagulant therapy for LV thrombus, AHA recommends vitamin K antagonists. They also consider direct OACs as a reasonable alternative for patients who struggle to consistently achieve a therapeutic INR range or for whom frequent INR checks are impractical.[1] In this case, we prescribed warfarin for the LV thrombi for at least 3 months and biaspirin for post-CABG management for lifetime.

This is a case report. Further research is required to draw more definitive conclusions.

Lead author biography



He graduated from Niigata University, School of Medicine Niigata, Japan. His field of interest is cardiac surgery.

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Data availability

No new data were generated or analysed in support of this research.

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