

Thrombus Migration Resulting From Cessation of Cardiopulmonary Bypass Flow in a Patient With Left Ventricular Thrombus



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INTRODUCTION

Left ventricular (LV) thrombus formation is a known complication associated with acute myocardial infarction and other conditions that impair LV function, carrying a risk for systemic embolization.^{1,2} In this report, we describe a case in which an emergent aortic cross-clamp was applied in an attempt to prevent a thrombus, which had dislodged from the LV apex into the LV outflow tract (LVOT), from migrating into the systemic circulation. However, it was later discovered that the thrombus had already migrated into the systemic circulation as an embolus before the aortic cross-clamp. It is believed that stopping cardiopulmonary bypass (CPB) while the heart was still beating, followed by cross-clamping the aorta, contributed to the thrombus migration. Our findings suggest that for a free-floating LV thrombus, the aortic cross-clamp should be applied with adjustments to CPB flow to keep the aortic valve closed or by optimizing venous drainage to empty the cardiac chambers and prevent ejection of the thrombus.

CASE PRESENTATION

A 53-year-old male patient presented with continuous chest pain for approximately 3 hours before visiting a nearby clinic. The patient was then transported by an ambulance to our hospital, arriving 4 hours after symptom onset. The patient was diagnosed with an acute ST-segment elevation myocardial infarction (STEMI), with severe antero-septal hypokinesis and an LV ejection fraction of approximately 40%. The patient immediately underwent percutaneous coronary intervention for total occlusion of the mid left anterior descending coronary artery, successfully achieving recanalization with a drug-eluting stent. On the 13th day after onset, because of an elevated D-dimer level of 3.5 $\mu\text{g/mL}$, transthoracic echocardiography (TTE) and contrast-enhanced computed tomography (CT) were performed, revealing an intraventricular thrombus at the apex of the left ventricle (Figures 1 and 2, Video 1). Continuous heparin infusion was

immediately initiated, and warfarin therapy was started the following day. On the 15th day after the onset of STEMI, the patient reported left-sided hemiparesis, prompting an emergency magnetic resonance imaging scan. The scan revealed high signal intensity on diffusion-weighted imaging in the right middle cerebral artery territory; however, magnetic resonance angiography confirmed that there was no occlusion in the major cerebral arteries. The left-sided hemiparesis symptoms completely resolved within 2 hours of stroke onset. A consultation with a neurologist confirmed the diagnosis of a cardioembolic stroke caused by an LV thrombus. Consequently, surgical LV thrombectomy was scheduled for the following day.

After the induction of general anesthesia, transesophageal echocardiography confirmed the presence of the thrombus located in the LV apex, as previously identified (Figure 3, Video 2). After administering 24,000 U heparin, the activated clotting time (ACT) was extended to only 375 seconds. Given that the preoperative antithrombin III activity was 56% on the basis of the blood test results obtained upon operating room admission, 1,000 U antithrombin III was administered. An additional 8,000 U heparin was then given, and an ACT of 431 seconds was confirmed. With full awareness of the LV thrombus, the cardiac surgeon carefully placed the arterial and venous cannulas. Simultaneously, an additional 3,000 U heparin was administered, and CPB was initiated once an ACT of >450 seconds was confirmed. After the initiation of CPB, epicardial echocardiography revealed that the thrombus had dislodged into the LVOT (Figure 4, Video 3). Because of the urgent need to prevent systemic migration of the thrombus, CPB flow was stopped before the aortic cross-clamping and was immediately restarted once the clamp was in place. However, neither transesophageal nor epicardial echocardiography could identify the presence of the thrombus. No thrombus was found upon direct inspection of the left atrium, left ventricle, and aortic root using a thoracoscope inserted via the left atrial incision. Therefore, it was concluded that the LV thrombus had entered the systemic circulation. Immediately after the completion of cardiac surgery, upon removal of the drapes, pallor of the left lower limb and absence of a palpable pulse in the left dorsalis pedis artery were noted. Notably, no elevation in lactate levels was observed in blood gas analyses during the cardiac surgery. The patient remained intubated, and emergency contrast-enhanced CT of the entire body was performed. The scan revealed no thrombotic involvement of vital organs, with the thrombus localized to the left lower limb. Approximately 50 minutes after the completion of cardiac surgery, vascular surgeons began an emergency endovascular thrombectomy in a hybrid operating room, successfully restoring blood flow to the left lower limb by removing thrombi from the anterior tibial, posterior tibial, and fibular arteries (Figure 5). The patient was admitted to the intensive care unit after the surgery and extubated 12 hours later. The patient was fully conscious, showed no neurologic abnormalities, and exhibited no

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VIDEO HIGHLIGHTS

Video 1: Two-dimensional TTE (day 13), parasternal short-axis view at the apical level, demonstrates an intraventricular thrombus attached to the LV wall.

Video 2: Two-dimensional transesophageal echocardiography (day 16, before CPB), midesophageal, LV-focused modified two-chamber (72°) view, demonstrates an intraventricular thrombus attached to the LV apical wall.

Video 3: Two-dimensional epicardial echocardiography (day 16, after CPB initiation), LV long-axis view, demonstrates a thrombus dislodged into the LVOT. The anterior echo-free space represents the placement of a fluid-filled bag between the echocardiographic probe and the heart to allow optimal imaging.

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other symptoms or signs suggestive of systemic infarct. On postoperative day 4, the patient was transferred from the intensive care unit to a general ward and was discharged without any neurologic sequelae on postoperative day 21 (Figure 6).

DISCUSSION

We report a case in which emergent aortic clamping was performed to prevent a free-floating thrombus in the LVOT from migrating into the systemic circulation. However, because of the cessation of CPB flow

while the heart was still beating, the thrombus was expelled from the heart before the aortic clamp could be applied.

Although the incidence of LV thrombus has decreased with the increased use of early reperfusion therapies, it remains a significant clinical issue. In the prereperfusion era, the incidence of LV thrombi was reported to be as high as 40% in patients with anterior infarction,^{3,4} while in the reperfusion era, it has been reported to be 4% to 8%.⁵⁻⁸ Additionally, the presence of LV thrombus is associated with increased risk for systemic embolism, stroke and mortality,⁹ underscoring the necessity of anticoagulation therapy or surgical thrombectomy.

There have been previous reports of cases in which LV thrombi dislodged during CPB, causing embolism.¹⁰ However, a strategy to prevent a dislodged LV thrombus from migrating into the systemic circulation during surgical thrombectomy has yet to be established. Given the embolism caused by the LV thrombus in the present case, we propose the following approaches as potential strategies for managing similar situations.

In situations in which an emergent cross-clamp is required and a thrombus has already dislodged within the left ventricle, the following measures may help prevent its migration into the systemic circulation. Generally, CPB pump flow is reduced transiently when cross-clamping the aorta. However, we believe that halting CPB flow led to the thrombus's being expelled from the heart because of intrinsic cardiac activity. Therefore, by maintaining aortic pressure through adjustments in CPB flow and monitoring the aortic valve via echocardiography to ensure it remains closed, the risk that it will open can be minimized. Another strategy might involve optimizing venous drainage to ensure that the heart remains completely empty, eliminating any volume in the left ventricle available for ejection, which may lower the risk for thrombus migration. By performing a cross-clamp under these conditions, it may be possible to prevent thrombus migration. Although further research is necessary, this case suggests

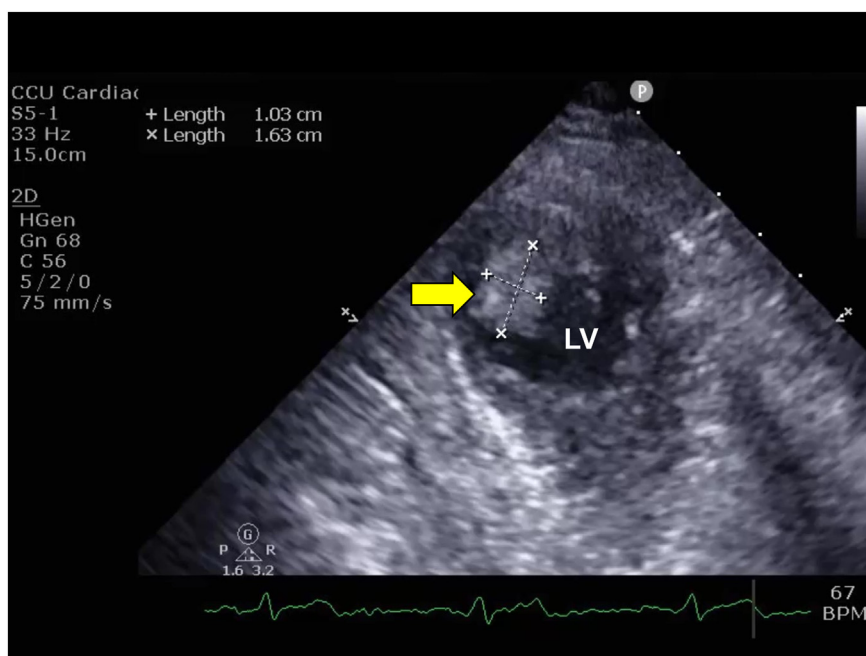


Figure 1 Two-dimensional TTE (day 13), parasternal short-axis view at the apical level, demonstrates an intraventricular thrombus attached to the left ventricular wall (arrow). LV, Left ventricle.

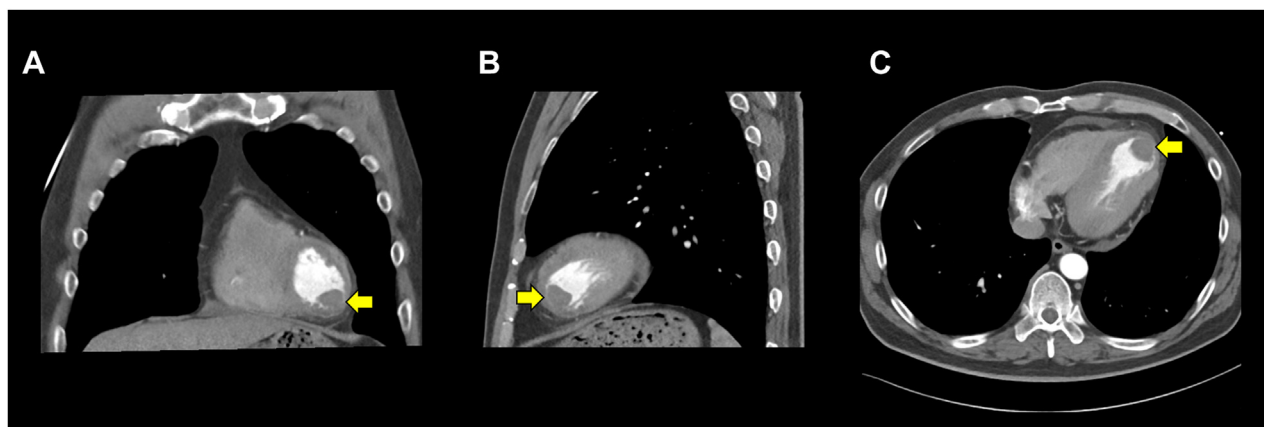


Figure 2 Contrast-enhanced computed tomographic images (day 13), multiplanar reformat in (A) coronal, (B) sagittal, and (C) axial displays, demonstrate a LV apical thrombus (arrows).

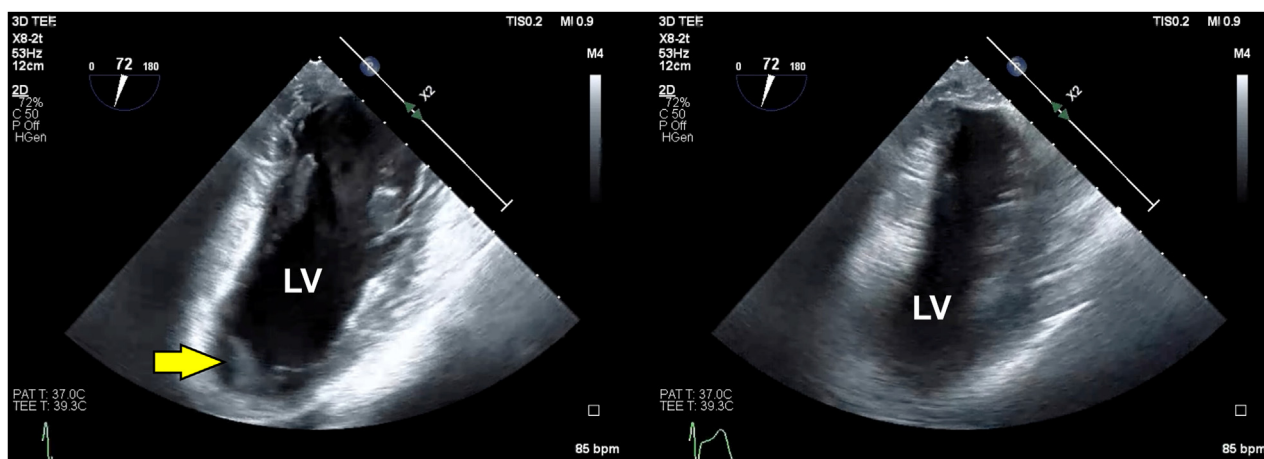


Figure 3 Two-dimensional transesophageal echocardiography (day 16, before CPB), midesophageal, left ventricular-focused modified two-chamber (72°) diastolic (left) and systolic (right) displays, demonstrates an intraventricular thrombus attached to the left ventricular apical wall (arrow). LV, Left ventricle.

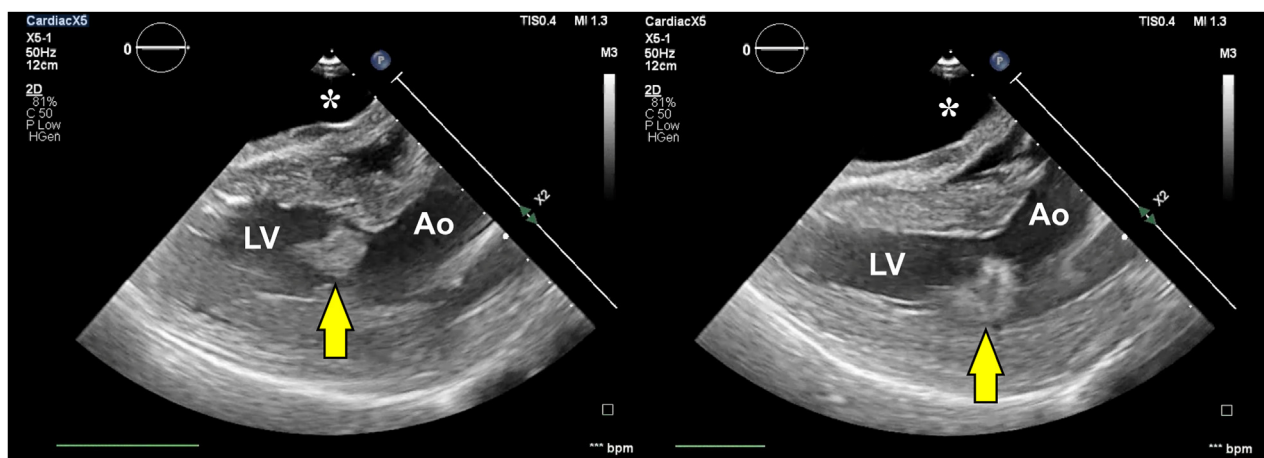


Figure 4 Two-dimensional epicardial echocardiography (day 16, after CPB initiation), left ventricular long-axis diastolic (left) and systolic (right) displays, demonstrates a thrombus (arrows) dislodged into the LVOT. The anterior echo-free space represents the placement of a fluid-filled bag (asterisk) between the echocardiographic probe and the heart to allow optimal imaging. Ao, Aorta; LV, left ventricle.



Figure 5 Angiographic images during emergency endovascular thrombectomy demonstrate (A) total occlusion in the anterior tibial, posterior tibial, and fibular arteries from the embolized thrombus with restoration of arterial flow (B) after endovascular thrombectomy.

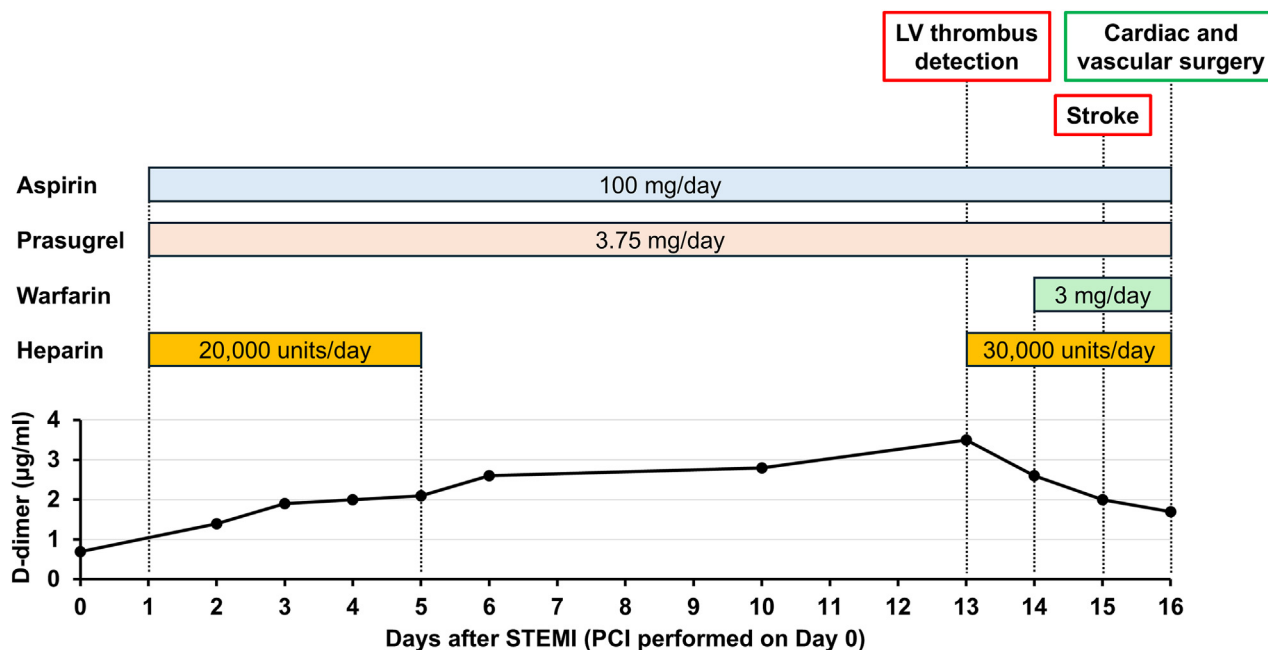


Figure 6 Timeline demonstrates the clinical presentation, management, laboratory results, detection of LV thrombus, stroke event, and surgical intervention during the prolonged hospital course. *PCI*, Percutaneous coronary intervention.

that the first approach, by increasing LV afterload, or the second, by decreasing preload, may be effective in preventing systemic embolization by reducing cardiac output.

With this patient, TTE was performed regularly until day 13 after STEMI, but no LV thrombus was detected before day 13. It has been reported that late gadolinium enhancement cardiovascular magnetic resonance has greater sensitivity than TTE, with similarly high specificity, for detecting LV thrombus in patients with ischemic cardio-

myopathy.¹¹⁻¹³ Therefore, if late gadolinium enhancement cardiovascular magnetic resonance had been performed in this case, earlier treatment might have been possible.

Suspicion of a lower limb thrombus arose immediately after the cardiac surgery, prompting us to perform emergency contrast-enhanced CT of the entire body. The findings confirmed that there were no thrombi outside the left lower limb. However, we recognize that some facilities may not have immediate access to full-body

contrast CT. In such cases, to avoid prolonged ischemia, it may be appropriate for a vascular surgeon to intervene in the same operating room using a portable C-arm or to consider immediate transfer to a hybrid operating room capable of cerebral or aortic angiography. Decisions should be made on the basis of the medical resources available at each facility.

CONCLUSION

Embolization due to dislodgement of a LV thrombus can lead to severe complications. Maintaining CPB flow to ensure that the aortic valve remains closed during aortic cross-clamping may be a potential strategy for preventing thrombus migration. Another strategy could be optimizing venous drainage so that the heart is completely empty, leaving no volume in the left ventricle to eject. However, further research is needed to validate these approaches and improve outcomes in the management of LV thrombi during cardiac surgery.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

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DISCLOSURE STATEMENT

The authors report no conflict of interest.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2024.12.007>.

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