IJEBM International Journal of Evidence-Based Medicine

International Journal of Evidence-Based Medicine

Australasia Publishing Group

Case Report

Cerebral Infarction Recognized After Transaortic Left Ventricular Thrombectomy and Coronary Artery Bypass Grafting: A Case Report of Possible Perioperative Embolization

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Received: Aug 29, 2026
Accepted: Nov 14, 2026

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Citation: Ansari ZR, Hamdulay ZA, Khan A, Jain S, Hamdulay M. Cerebral Infarction Recognized After Transaortic Left Ventricular Thrombectomy and Coronary Artery Bypass Grafting: A Case Report of Possible Perioperative Embolization. International Journal of Evidence-Based Medicine. 2026;1(4):jebm019. https://doi.org/10.63946/jebm/19982

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ABSTRACT

Left ventricular thrombus (LVT) after acute myocardial infarction may cause systemic embolization despite antithrombotic therapy. Surgical thrombectomy may be considered in selected patients with large, mobile thrombi requiring concomitant cardiac surgery.

A 57-year-old man presented with acute inferior myocardial infarction and severe left main and multivessel coronary artery disease. Initial transthoracic echocardiography showed severe left ventricular dysfunction (LVEF 25–30%) without visible LVT. Approximately 10 days after the infarction, he developed acute left upper-limb ischemia and mild left-sided weakness. Brain computed tomography showed a small right corona radiata infarct, while repeat echocardiography demonstrated a 33 × 24-mm mobile apical LVT. Upper-limb computed tomography angiography showed multilevel arterial thromboembolic occlusion, and emergency brachial embolectomy was performed. The patient subsequently underwent coronary artery bypass grafting and transaortic LVT removal. The excised thrombus was heterogeneous, with relatively organized and fresh-appearing components. Intraoperative transesophageal echocardiography showed no visible residual intracavitary thrombus. A new neurological deficit was recognized approximately 12–14 hours after separation from cardiopulmonary bypass. Computed tomography demonstrated a large right middle cerebral artery-territory infarction with an additional right cerebellar infarction.

The temporal relationship between surgery and neurological deterioration raises concern for a possible perioperative ischemic event; however, the exact time and mechanism of cerebral arterial occlusion could not be established. Absence of visible residual LVT on intraoperative echocardiography does not establish elimination of perioperative embolic risk.

Keywords: Left Ventricular Thrombus; Acute Myocardial Infarction; Coronary Artery Bypass Grafting; Cerebral Infarction; Systemic Embolization; Transaortic Thrombectomy; Perioperative Stroke

Introduction

Left ventricular thrombus (LVT) is an important complication of acute myocardial infarction, particularly in patients with extensive myocardial injury, apical akinesia or dyskinesia, and severe left ventricular systolic dysfunction. LVT may result in systemic arterial embolization, including cerebral and peripheral arterial events. Current management requires individualized assessment of thromboembolic and bleeding risks, with anticoagulation forming the principal medical treatment in patients with established LVT [1,2].

The development of LVT is associated with ventricular stasis, endocardial injury, and hypercoagulability, and patients with substantial regional wall-motion abnormalities and reduced left ventricular function are at increased risk [3]. Although transthoracic echocardiography is widely used for detection, cardiac magnetic resonance imaging may provide greater sensitivity in selected patients when echocardiographic findings are inconclusive or clinical suspicion remains high [1,4].

The morphology and clinical behavior of LVT may also influence management. Mobile or protruding thrombi may have greater embolic potential, whereas more organized or laminated thrombi may behave differently over time [1,4]. Anticoagulation remains the principal treatment strategy, although the optimal agent and duration require individualization, particularly when anticoagulation must be combined with antiplatelet therapy [1].

Surgical removal of LVT is uncommon and is generally considered in selected patients with large, mobile, or embolizing thrombi, particularly when concomitant cardiac surgery is required. Transaortic surgical thrombectomy has been described as a means of removing apical thrombus while avoiding a left atriotomy or ventricular incision [5–7]. Other approaches, including transmitral endoscopic thrombectomy, have also been reported [8].

Postoperative stroke after coronary artery bypass grafting (CABG) is a recognized but multifactorial complication. Potential mechanisms include embolization from the heart or aorta, cerebral hypoperfusion, pre-existing cerebrovascular disease, postoperative atrial fibrillation, and other perioperative factors [9–11]. The occurrence of a major cerebral infarction after cardiac surgery therefore does not by itself establish a single embolic source.

The present case is notable because a large apical LVT developed after an initially negative echocardiographic examination, subsequently caused systemic arterial embolization, and was surgically removed during CABG. Although no visible residual intracavitary LVT was identified by intraoperative transesophageal echocardiography (TEE), a major cerebral infarction was subsequently recognized during the early postoperative period. The case therefore highlights the distinction between absence of visible residual thrombus on imaging and inability to establish elimination of perioperative embolic risk.

Case Presentation

Initial Presentation and Cardiac Evaluation

A 57-year-old man presented with a recent acute inferior myocardial infarction. Coronary angiographic assessment demonstrated severe left main and multivessel coronary artery disease. Transthoracic echocardiography (TTE) performed during the initial evaluation demonstrated severe left ventricular systolic dysfunction, with an estimated left ventricular ejection fraction (LVEF) of approximately 25–30%.

The left ventricular internal diameter in diastole (LVIDd) was 43 mm and the left ventricular internal diameter in systole (LVIDs) was 37 mm. Left ventricular end-diastolic and end-systolic volumes were not available in the clinical records reviewed. The apex, apico-anterior wall, apico-septal region, and distal anteroseptal segments were akinetic, while the remaining left ventricular myocardium was generally hypokinetic. Importantly, no visible LVT was identified on the initial TTE. Thus, the subsequently documented thrombus was not known to be present at the initial echocardiographic assessment. The patient was initially managed with antithrombotic therapy following the myocardial infarction, including dual antiplatelet therapy (DAPT), consisting of aspirin 75 mg once daily and clopidogrel 75 mg once daily, together with low-molecular-weight heparin (LMWH).

Peripheral Embolic Event and Detection of Left Ventricular Thrombus

Approximately 10 days after the index myocardial infarction, the patient developed acute ischemic symptoms involving the left upper limb, accompanied by mild left-sided weakness. Noncontrast brain computed tomography (CT) demonstrated a small infarction in the right corona radiata. The available clinical documentation did not establish the precise onset time of the neurological lesion. Repeat echocardiography subsequently demonstrated a large, mobile apical LVT measuring approximately 33 × 24 mm. The thrombus was associated with the previously described region of severe apical dysfunction [Figure 1].

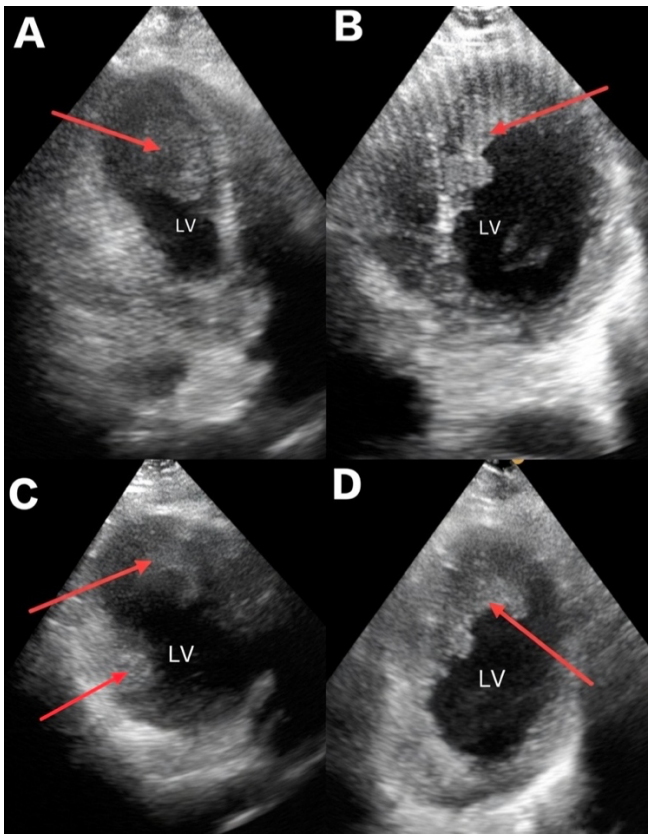


Figure 1. Two-dimensional transthoracic echocardiographic images (A–D) demonstrating a large, protruding left ventricular apical thrombus (arrows) attached to the akinetic apical myocardium and extending into the ventricular cavity. LV, left ventricle.

CT angiography of the upper limb demonstrated multilevel thromboembolic occlusion involving the left subclavian, axillary, brachial, and radial arterial circulation. Emergency brachial embolectomy was therefore performed [Figure 2].

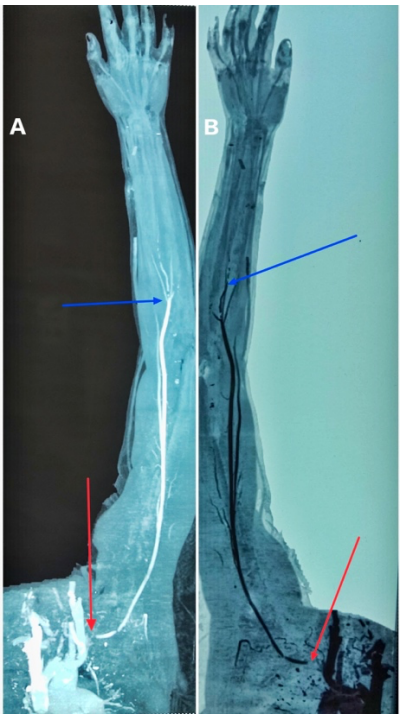


Figure 2. Computed tomography angiography showing left upper limb arterial thromboembolism (A, B) Volume-rendered CTA images demonstrate thrombosis of the left subclavian artery distal to the vertebral artery origin (red arrows) and focal brachial artery narrowing (blue arrows), with severely impaired distal runoff and absent opacification of the hand vessels.

Carotid Doppler examination showed only mild non-obstructive carotid disease without significant carotid stenosis. The combination of a large mobile LVT and simultaneous peripheral arterial embolization raised substantial concern for ongoing systemic embolic risk.

Preoperative TEE and Left Atrial Appendage Findings

Preoperative TEE demonstrated a large apical LVT with spontaneous echo contrast (SEC) within the left atrial appendage (LAA). The LVT was associated with the akinetic apical region and appeared sufficiently mobile to represent a potential source of systemic embolization.

The patient was in sinus rhythm, and no atrial fibrillation (AF) had been documented. CT imaging demonstrated a 1.9 × 1.0-cm filling defect within the LAA. This finding was considered indeterminate and was not regarded as definitive evidence of an LAA thrombus. The differential included thrombus and imaging-related filling artifact. Because spontaneous echo contrast (SEC) was present and the patient had already experienced systemic thromboembolism, the LAA was surgically ligated as an additional risk-reduction measure during the cardiac operation. No postoperative imaging was available to independently confirm complete LAA exclusion.

Antithrombotic Management

Following the acute myocardial infarction, the patient received DAPT consisting of aspirin 75 mg once daily and clopidogrel 75 mg once daily, together with LMWH. After the subsequent peripheral arterial embolic event, LMWH was discontinued and therapeutic unfractionated heparin (UFH) was initiated, with an intended aPTT target of approximately 60–80 seconds. UFH was discontinued approximately 12 hours before surgery because of the anticipated operative bleeding risk. Aspirin 75 mg daily was continued until the day before surgery.

For the surgical procedure, UFH 10,000 IU was administered as an initial bolus followed by an infusion of approximately 600 IU/hour. The ACT before initiation of cardiopulmonary bypass (CPB) was approximately 290 seconds. During CPB, anticoagulation was maintained with ACT values of approximately 450 seconds and increased to approximately 550 seconds at CPB separation. At completion of the procedure, 50 mg of protamine was administered, after which the ACT decreased to approximately 180 seconds at closure. Postoperatively, UFH was restarted approximately 4 hours after surgery with a 5,000-IU bolus followed by an infusion of approximately 600 IU/hour. The first documented postoperative aPTT was approximately 52 seconds and increased to approximately 65 seconds 6 hours later.

Following recognition of the large cerebral infarction, systemic anticoagulation was reassessed and UFH was subsequently discontinued. Aspirin 75 mg was restarted the following morning for coronary secondary prevention. At discharge, anticoagulation was transitioned to apixaban 5 mg twice daily because of the patient's thromboembolic history, while aspirin 75 mg was continued for coronary secondary prevention.

This chronology is relevant because anticoagulation was not simply omitted after surgery. Rather, anticoagulation was temporarily interrupted for the operative procedure and subsequently resumed, although the precise timing of the cerebral arterial occlusion could not be established from the available clinical data.

Surgical Procedure

The patient underwent median sternotomy for CABG with concomitant surgical removal of the LVT under general anesthesia. The operation was initially started off-pump, with the left internal mammary artery (LIMA) anastomosed to the left anterior descending artery using an Octopus stabilizer. Given the need for direct intracardiac thrombus removal and completion of the remaining coronary anastomoses, CPB was subsequently instituted.

Following systemic heparinization, the patient underwent aortic cross-clamping and CPB. An aortotomy was performed, allowing direct transaortic visualization and removal of the apical thrombus. The thrombus was attached to the left ventricular apex/apicoseptal region and was partially mobile. Macroscopically, it had a heterogeneous appearance, with relatively organized areas together with components that appeared more recent or fresh. Histopathological examination was not performed; therefore, the exact histological age and composition of the thrombus could not be determined.

The thrombus was removed and the left ventricular cavity was irrigated. Saphenous vein grafts were then constructed to the obtuse marginal and posterior descending artery territories while the patient remained on CPB. The total CPB duration was approximately 128 minutes, and the aortic cross-clamp time was approximately 73 minutes. The LAA was surgically ligated during the procedure because of the preoperative SEC and indeterminate LAA filling defect in the setting of previous systemic embolization.

Intraoperative TEE After Thrombectomy

After removal of the LVT, intraoperative TEE was repeated. No visible residual intracavitary LVT was identified on the post-thrombectomy examination.

The left ventricular apex, however, remained akinetic and severely dysfunctional, reflecting the underlying myocardial injury [Figure 3]. Thus, the post-thrombectomy TEE documented no visible residual intracavitary thrombus, but it could not establish microscopic absence of thrombotic material or eliminate the possibility that embolization had already occurred or could subsequently occur.

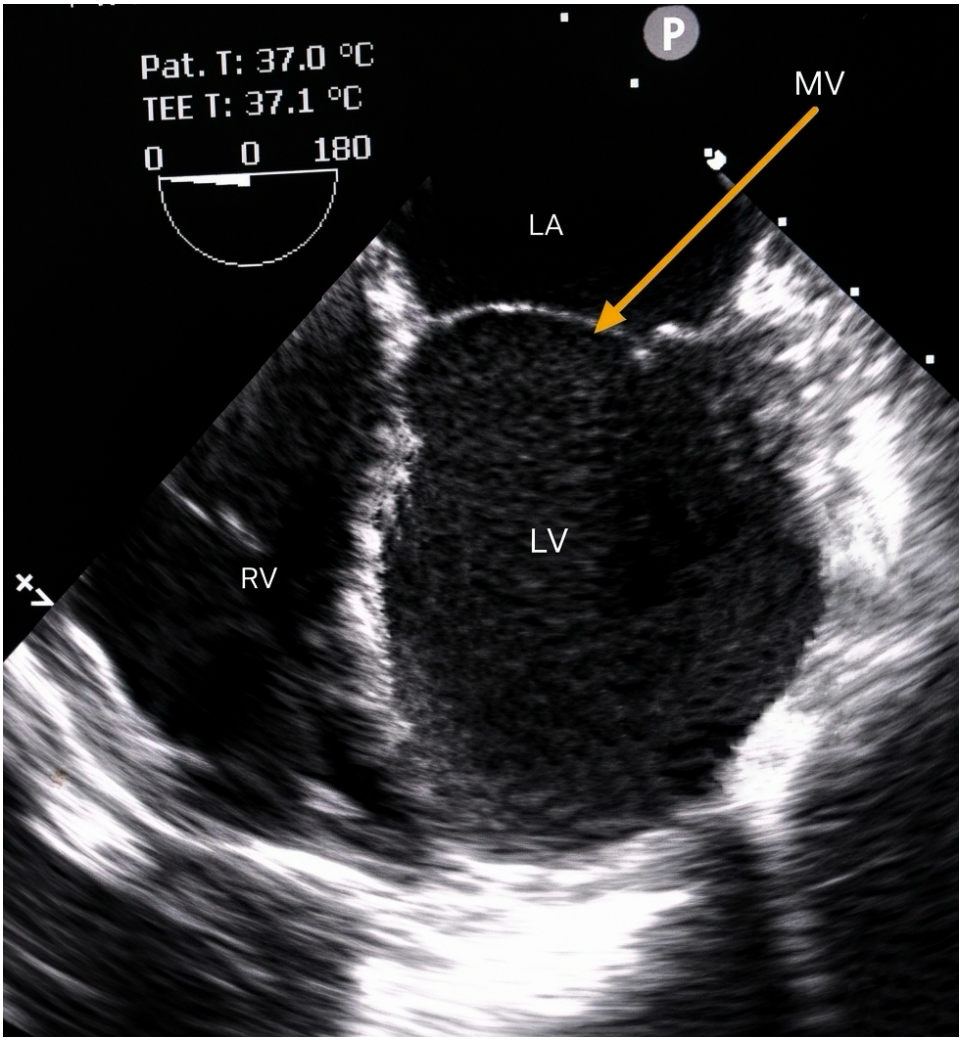


Figure 3. Intraoperative transesophageal echocardiogram following left ventricular thrombectomy. Mid-esophageal view (0°) confirming the absence of residual thrombus within the left ventricle. The arrow indicates the mitral valve. LA - left atrium; LV - left ventricle; MV - mitral valve; RV - right ventricle.

The available operative and anesthesia records did not document atrial fibrillation or sustained clinically significant arrhythmia during the intraoperative period. The patient remained in sinus rhythm during the documented perioperative period. Continuous rhythm data were not available for independent retrospective review. At CPB separation, the ACT was approximately 550 seconds. Protamine 50 mg was subsequently administered, with a post-protamine ACT of approximately 180 seconds at closure.

Early Postoperative Neurological Course

Following surgery, the patient remained intubated and mechanically ventilated. He was not receiving a continuous sedative infusion, although residual neuromuscular blockade was present during the early postoperative period. The pupils were equal and reactive.

At approximately 8 hours after surgery, the patient was arousable but incompletely alert, with a documented Glasgow Coma Scale (GCS) of E3VtM4. Approximately 12–14 hours after separation from CPB, a new left-sided motor deficit was recognized. The neurological examination at that point was limited by the patient's postoperative condition and mechanical ventilation. A formal comprehensive pre- and post-event cranial nerve, sensory, visual-field, and speech examination was not available in the clinical record. The National Institutes of Health Stroke Scale (NIHSS) was subsequently documented as 23, indicating a severe neurological deficit. The important temporal distinction is that a new neurological deficit was recognized approximately 12–14 hours after CPB

separation; this interval represents the time of clinical recognition and does not establish the actual time of cerebral arterial occlusion.

Postoperative Neuroimaging and Stroke Management

Emergency noncontrast CT of the brain demonstrated a large right middle cerebral artery (MCA)-territory infarction involving the frontal, parietal, and temporal lobes, as well as the lentiform nucleus, corona radiata, and caudate region. Associated cerebral edema was present, with approximately 3 mm of midline shift [Figure 4]. An additional infarction was identified in the right cerebellar hemisphere.

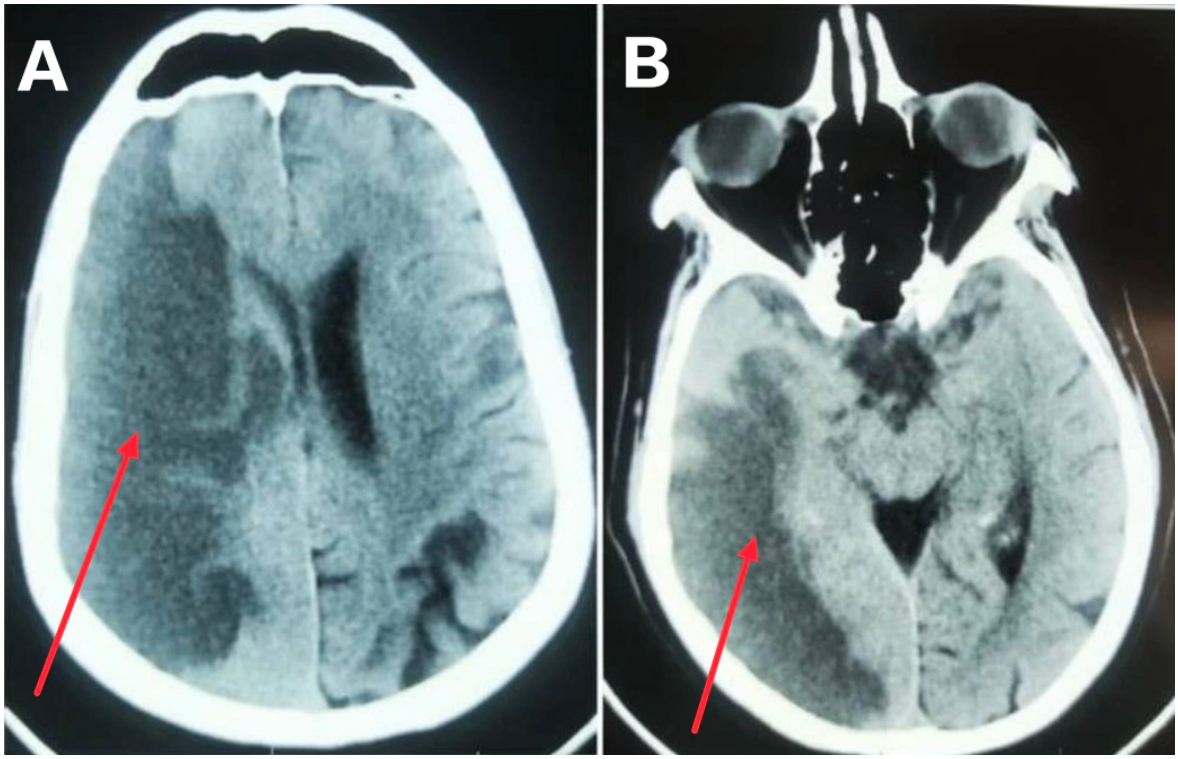


Figure 4. Axial non-contrast CT brain images (A, B) demonstrating a large right MCA territory infarction (arrows) with extensive cerebral edema, significant compression of the right lateral ventricle, and minimal leftward midline shift.CT - Computed tomography.

The postoperative infarction was substantially more extensive than the previously documented small right corona radiata infarction and was accompanied by a new right cerebellar infarction, supporting a new major ischemic event, although extension of the pre-existing lesion cannot be completely excluded. The clinical timing and established radiographic extent of infarction were considered when assessing eligibility for acute reperfusion therapy. Mechanical thrombectomy was not pursued because the onset of the neurological event could not be reliably established, the initial CT already demonstrated a large established infarction with edema, and the patient was critically ill in the immediate postoperative period. Additional intracranial vascular imaging was not obtained because of the concurrent acute kidney injury and the clinical limitations of further contrast-based evaluation at that time. MRI was also not feasible during the relevant postoperative period.

Serial noncontrast CT examinations demonstrated evolution of the infarction without hemorrhagic transformation. Management consisted of neurocritical care, neurological monitoring, and serial neuroimaging. No definitive conclusion regarding the precise intracranial arterial occlusion site or time of vessel occlusion could therefore be made.

Postoperative Renal Dysfunction and Anticoagulation

The patient's baseline serum creatinine was approximately 1.4 mg/dL. Following surgery, creatinine increased to approximately 2.5 mg/dL on postoperative day (POD) 1 and 3.6 mg/dL on POD 3, accompanied by oliguria. This was consistent with KDIGO stage 2 acute kidney injury by creatinine criteria [14]. Renal function subsequently improved, and renal replacement therapy was not required. A complete serial urea dataset was not available in the records reviewed. The renal dysfunction was clinically relevant to the assessment of further contrast-enhanced cerebral vascular imaging and contributed to the decision to avoid additional contrast exposure when the expected diagnostic and therapeutic benefit was uncertain.

The large cerebral infarction also required reassessment of the balance between thromboembolic risk and the risk of intracranial hemorrhagic transformation. UFH was therefore subsequently discontinued following recognition of the infarction. Serial CT examinations did not demonstrate hemorrhagic transformation. The available records do not provide sufficient detail to determine whether specific osmotherapy was administered. Cerebral edema was managed with supportive neurocritical care, neurological observation, and serial noncontrast CT assessment; no decompressive neurosurgical procedure was performed.

Subsequent Neurological and Clinical Course

The patient required prolonged ventilatory support and underwent tracheostomy on POD 7. He subsequently received neurological rehabilitation. Neurological recovery was incomplete. Persistent left-sided weakness remained, while sensorium and right-sided motor function gradually improved.

At discharge, the patient was receiving apixaban 5 mg twice daily together with aspirin 75 mg daily. No postoperative episode of atrial fibrillation was documented in the available clinical records. There was therefore no documented postoperative AF to provide an established alternative cardioembolic mechanism, although continuous telemetry data were not available for independent retrospective analysis.

A chronological summary of the patient's clinical course, including recurrent embolic events, diagnostic findings, surgical management, postoperative complications, and outcome, is presented in [Table 1].

Table 1. Chronological overview of the patient's clinical course, including preoperative systemic embolization, surgical intervention, timing of postoperative neurological deterioration, diagnostic findings, and outcome.

Time from index myocardial infarction	Clinical event
Day 0	Acute inferior myocardial infarction; severe left main and multivessel coronary artery disease identified.
Early after MI	TTE showed LVEF 25–30%, apical/apico-septal akinesia, and no visible LVT. LVIDd 43 mm; LVIDs 37 mm.
Approximately Day 10	Acute left upper-limb ischemia with mild left-sided weakness.
Approximately Day 10	Brain CT demonstrated a small right corona radiata infarct.
Approximately Day 10	Repeat TTE demonstrated a 33 × 24-mm mobile apical LVT.
Approximately Day 10	CT angiography demonstrated multilevel left upper-limb arterial thromboembolic occlusion; emergency brachial embolectomy performed.
Preoperative period	TEE demonstrated large apical LVT and SEC in the LAA. CT showed an indeterminate 1.9 × 1.0-cm LAA filling defect. No documented AF.
Preoperative period	LMWH discontinued; therapeutic UFH initiated. UFH stopped approximately 12 h before surgery. Aspirin 75 mg continued until the day before surgery.
Day of cardiac surgery	CABG with transaortic LVT removal and surgical LAA ligation performed.
Intraoperative	ACT approximately 290 s before CPB; approximately 450 s during CPB and approximately 550 s at CPB separation. Post-protamine ACT approximately 180 s after 50 mg protamine.
End of surgery	TEE showed no visible residual intracavitary LVT; apex remained akinetic/severely dysfunctional.
Approximately 4 h after surgery	UFH restarted with 5,000-IU bolus followed by approximately 600 IU/h infusion.
Approximately 8 h after surgery	Patient arousable but incompletely alert; GCS E3VtM4.
Approximately 12–14 h after CPB separation	New left-sided motor deficit recognized; NIHSS 23.
Early postoperative period	CT demonstrated large right MCA-territory infarction with edema and approximately 3-mm midline shift, together with a right cerebellar infarction.
Early postoperative period	Mechanical thrombectomy not pursued because onset was uncertain and CT showed a large established infarction; additional vascular imaging was not obtained.
POD 1–3	Creatinine increased from 1.4 mg/dL to 2.5 mg/dL on POD1 and 3.6 mg/dL on POD3; KDIGO stage 2 AKI; no renal replacement therapy.

Time from index myocardial infarction	Clinical event
Following infarction	UFH subsequently discontinued after reassessment of bleeding and thromboembolic risks. Serial CT showed no hemorrhagic transformation.
POD 7	Tracheostomy performed because of prolonged ventilatory requirement.
Later postoperative course	Rehabilitation initiated; persistent left-sided weakness with gradual neurological improvement.
Discharge	Apixaban 5 mg twice daily and aspirin 75 mg daily prescribed.

Discussion

This case illustrates the clinical complexity of LVT developing after acute myocardial infarction in a patient with severe ventricular dysfunction and extensive coronary disease. The initial TTE did not demonstrate an intracavitary thrombus, whereas approximately 10 days later a large, mobile apical LVT measuring 33 × 24 mm was identified in association with systemic arterial embolization. The sequence emphasizes that the absence of visible thrombus at an earlier echocardiographic examination does not exclude subsequent thrombus formation, particularly in the presence of substantial regional wall-motion abnormalities and severe LV dysfunction [1–4].

The patient had evidence of systemic thromboembolism before cardiac surgery, including acute multilevel left upper-limb arterial occlusion and a small right corona radiata infarction. The large mobile LVT provided a plausible embolic source. The subsequent surgical decision was based on the combination of a large mobile thrombus, documented systemic embolization, severe ventricular dysfunction, and the need for concomitant CABG. Surgical LVT removal remains an individualized strategy rather than a routine approach, and published reports describe transaortic and other surgical techniques in selected patients [5–8].

The macroscopic thrombus was heterogeneous, with relatively organized and fresh-appearing components. This appearance suggested thrombotic material at different stages of organization; however, histopathological examination was not performed, and therefore the age and precise composition of the thrombus cannot be established [4].

A central issue in this case is the interpretation of the post-thrombectomy TEE. No visible residual intracavitary LVT was identified after surgical removal. However, this finding should not be interpreted as proof that all thrombotic material had been eliminated or that the risk of perioperative embolization had become zero. Echocardiographic assessment can document the absence of visible residual thrombus but cannot establish whether embolization has already occurred or exclude undetectable thrombotic material [1,2].

The postoperative neurological event is therefore best interpreted cautiously. A new neurological deficit was recognized approximately 12–14 hours after CPB separation, and CT demonstrated a large right MCA-territory infarction with an additional right cerebellar infarction. The postoperative infarction was substantially more extensive than the previously documented small right corona radiata infarction and was accompanied by a new right cerebellar infarction, supporting a new major ischemic event, although extension of the pre-existing lesion cannot be completely excluded.

Importantly, the timing of neurological recognition does not establish the time of arterial occlusion. The patient had residual neuromuscular blockade and was mechanically ventilated after surgery, and the early neurological assessment was therefore constrained. Consequently, the infarction could have developed at an uncertain point during the perioperative period rather than necessarily occurring immediately after documented thrombus removal.

Several mechanisms remain possible. Perioperative embolization from residual or previously detached thrombotic material is one possibility. Other potential contributors include embolization related to cardiac manipulation, the aorta or grafting procedure, perioperative cerebral hypoperfusion, or thromboembolic disease unrelated to the removed ventricular thrombus. Post-CABG stroke is recognized as a multifactorial complication, and both embolic and hemodynamic mechanisms have been described [9–11]. The absence of documented postoperative AF makes AF less readily identifiable as an established alternative mechanism, although continuous rhythm data were not available for independent retrospective analysis.

The antithrombotic chronology is also important. Following myocardial infarction, the patient received dual antiplatelet therapy (DAPT) consisting of aspirin 75 mg once daily and clopidogrel 75 mg once daily, together with LMWH. After the peripheral arterial embolic event, LMWH was discontinued and

therapeutic UFH was initiated. UFH was temporarily interrupted approximately 12 hours before surgery because of operative bleeding concerns and restarted approximately 4 hours postoperatively, with subsequent aPTT monitoring. Thus, the postoperative cerebral infarction did not occur in the context of a simple failure to resume anticoagulation. Instead, the case illustrates the difficult balance between maintaining protection against recurrent systemic embolization and limiting bleeding risk after major cardiac surgery. The combination of anticoagulant and antiplatelet therapy requires individualized consideration because of the competing risks of recurrent embolization and bleeding [1,12,13].

The decision not to pursue mechanical thrombectomy was influenced by the uncertain onset of the neurological deficit and the CT evidence of a large established infarction with edema. Because the actual time of arterial occlusion could not be established and additional intracranial vascular imaging was not obtained, the case cannot determine whether an occlusion amenable to mechanical reperfusion was present at the time of clinical recognition.

The postoperative acute kidney injury added another layer of complexity. Creatinine increased from 1.4 mg/dL before surgery to 3.6 mg/dL by POD 3, meeting KDIGO stage 2 criteria by creatinine change [14]. The renal dysfunction contributed to the clinical reluctance to expose the patient to additional contrast imaging when the anticipated diagnostic and therapeutic benefit was uncertain.

The LAA findings should likewise be interpreted cautiously. SEC was observed, and CT demonstrated a 1.9 × 1.0-cm LAA filling defect; however, the latter was considered indeterminate rather than diagnostic of thrombus. The patient had no documented AF. LAA ligation was therefore performed as an additional risk-reduction measure in the context of previous systemic embolization and abnormal LAA findings, but postoperative imaging confirming complete exclusion was unavailable.

The principal novelty of this case is deliberately narrow: a large LVT was surgically removed, no visible residual intracavitary thrombus was identified on immediate postoperative TEE, and a major cerebral ischemic event was subsequently recognized, while the exact timing and mechanism of cerebral arterial occlusion remained uncertain. The case does not establish that the cerebral infarction was caused by residual ventricular thrombus or that the infarction necessarily occurred after TEE confirmation of thrombus removal.

Accordingly, the findings should be interpreted as hypothesis-generating rather than as evidence of a specific causal mechanism. The case emphasizes the importance of integrating thrombus morphology, serial imaging, neurological assessment, perioperative anticoagulation, and the multifactorial nature of neurological complications after cardiac surgery [1, 9–13].

Strengths and Limitations

A strength of this report is the availability of serial cardiac and neurological information spanning the initial myocardial infarction, subsequent LVT formation, systemic embolization, surgical thrombectomy, intraoperative TEE, and postoperative cerebral infarction. The report also provides detailed perioperative anticoagulation data, including heparin dosing, ACT measurements, protamine administration, and postoperative aPTT values.

Several limitations should be acknowledged. The exact onset and mechanism of the postoperative cerebral ischemic event could not be established, as the documented 12–14-hour interval reflects recognition of the neurological deficit rather than confirmed arterial occlusion. Intracranial vascular imaging was not obtained, and MRI was not feasible, precluding confirmation of the responsible vessel or a potentially treatable large-vessel occlusion. Neurological assessment was limited by mechanical ventilation and residual neuromuscular blockade, with no complete standardized pre- and post-event examination available. Histopathological examination of the excised left ventricular thrombus was not performed, limiting assessment of its age and composition. Initial echocardiography provided LV dimensions but not formal end-diastolic or end-systolic volumes. The LAA filling defect on CT remained indeterminate, postoperative confirmation of complete LAA exclusion was unavailable, and continuous telemetry data could not be independently reviewed despite no documented atrial fibrillation. Although the second antiplatelet agent was subsequently identified as clopidogrel 75 mg once daily upon retrospective review of the treatment chart and clarification with the treating team, contemporaneous documentation was limited. Complete serial urea measurements were also unavailable. Finally, as a single case report, this observation cannot establish causality between surgical thrombectomy and subsequent cerebral infarction and should therefore be interpreted as hypothesis-generating rather than evidence of a specific embolic mechanism.

Conclusion

A large mobile LVT may develop after acute myocardial infarction despite an initially negative echocardiographic examination and may be associated with systemic arterial embolization. In selected patients requiring CABG, surgical thrombectomy may provide an approach to reducing the burden of a large emboligenic thrombus. In this patient, no visible residual intracavitary LVT was identified by intraoperative TEE after transaortic thrombectomy, yet a major cerebral infarction was subsequently recognized during the early postoperative period. However, the exact timing and mechanism of cerebral arterial occlusion could not be established. The case therefore does not prove postoperative embolization from residual LVT. Rather, it emphasizes that absence of visible residual LVT on intraoperative imaging does not establish elimination of perioperative embolic risk, particularly in patients with recent systemic embolization, severe ventricular dysfunction, and complex perioperative thrombotic and bleeding risks.

List of Abbreviations

AF — Atrial fibrillation
AKI — Acute kidney injury
aPTT — Activated partial thromboplastin time
CABG — Coronary artery bypass grafting
CAD — Coronary artery disease
CMR — Cardiac magnetic resonance
CPB — Cardiopulmonary bypass
CT — Computed tomography
DAPT — Dual antiplatelet therapy
ECG — Electrocardiogram
GCS — Glasgow Coma Scale
IV — Intravenous
LAA — Left atrial appendage
LMWH — Low-molecular-weight heparin
LVEF — Left ventricular ejection fraction
LVIDd — Left ventricular internal diameter in diastole
LVIDs — Left ventricular internal diameter in systole
LVT — Left ventricular thrombus
MCA — Middle cerebral artery
MI — Myocardial infarction
MRI — Magnetic resonance imaging
NIHSS — National Institutes of Health Stroke Scale
POD — Postoperative day
RRT — Renal replacement therapy
SEC — Spontaneous echo contrast
TEE — Transesophageal echocardiography
TTE — Transthoracic echocardiography
UFH — Unfractionated heparin

Acknowledgements

Ethics Approval: The case was managed in accordance with the institutional policies of Masina Heart Institute, Mumbai. Institutional approval or waiver applicable to publication of an individual case report was handled according to the institution's requirements.

Consent for Publication: Written informed consent was obtained from the patient's legally authorized representative for publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon reasonable request.

Funding: The authors received no financial support for the research, authorship, and/or publication of this article.

Disclosure of AI Use: Grammarly was used solely for grammatical correction, language editing, and improvement of the clarity and readability of this case report. No AI tool was used to generate or modify the clinical data, scientific content, interpretation, references, or conclusions. The authors reviewed and approved the final manuscript and remain responsible for its accuracy and integrity.

Ethics approval and consent to participate: Institutional Review Board approval was waived for this case report, as it describes a single patient managed as part of routine clinical care, in accordance with institutional and national ethical guidelines.

Availability of data and materials: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Authors' contributions: ZRA conceived the study and drafted the manuscript. ZAH contributed to clinical management and manuscript revision. AK and SJ assisted with surgical data acquisition and interpretation. MH supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

Acknowledgements: The authors thank Dr. Asad Shaikh for his critical care support, and the patient and his family for their cooperation and consent for publication.

References

1. Levine GN, McEvoy JW, Fang JC, Ibeh C, McCarthy CP, Misra A, et al. Management of patients at risk for and with left ventricular thrombus: a scientific statement from the American Heart Association. Circulation. 2022;146(15):e205–23. doi:10.1161/CIR.0000000000001092
2. Camaj A, Fuster V, Giustino G, Bienstock SW, Sternheim D, Mehran R, et al. Left ventricular thrombus following acute myocardial infarction: JACC state-of-the-art review. J Am Coll Cardiol. 2022;79(10):1010–22. doi:10.1016/j.jacc.2022.01.011
3. Fardman A, Massalha E, Natanzon SS, Brodov Y, Goitein O, Chernomordik F, et al. Clinical predictors of left ventricular thrombus after myocardial infarction as detected by magnetic resonance imaging. Front Cardiovasc Med. 2023;10:1275390. doi:10.3389/fcvm.2023.1275390
4. Cruz Rodriguez JB, Okajima K, Greenberg BH. Management of left ventricular thrombus: a narrative review. Ann Transl Med. 2021;9(6):520. doi:10.21037/atm-20-7839
5. Pasli S, Kamler M, Malik R, Easo J. Complicated massive left ventricular thrombus and surgical treatment. Am J Case Rep. 2022;23:e937341. doi:10.12659/AJCR.937341
6. Eyekpegha J, Williams PD, Krishnan SV, Ruiz Segria C, Owens WA. Transaortic extraction of left ventricular thrombus. JACC Case Rep. 2025;30(22):104567. doi:10.1016/j.jaccas.2025.104567
7. Akpınar MB, Uymaz B. Transaortic cardioscopic left ventricular thrombectomy. Cardiovasc Surg Int. 2023;10(1):75–8. doi:10.5606/e-cvsi.2023.1398
8. Tanaka T, Kajimoto K, Oishi A, Nishida H, Oku R, Oyama T, et al. Endoscopic transmitral thrombectomy for left ventricular thrombus. Am J Cardiol. 2025;254:101–7. doi:10.1016/j.amjcard.2025.08.007
9. Gaudino M, Angiolillo DJ, Di Franco A, Capodanno D, Bakaeen F, Farkouh ME, et al. Stroke after coronary artery bypass grafting and percutaneous coronary intervention: incidence, pathogenesis, and outcomes. J Am Heart Assoc. 2019;8(13):e013032. doi:10.1161/JAHA.119.013032
10. Jannati M. Risk factors for stroke post coronary artery bypass graft surgery: a review of literature. Med Clin Práct. 2024;7(1):100405. doi:10.1016/j.mcpsp.2023.100405
11. Hijazi E, Al-Zoubi N, Aljarrah Q. Postoperative stroke in CABG: a ten-year study of stroke incidence and contributing risk factors in Jordan. J Med Life. 2025;18(7):621–5. doi:10.25122/jml-2024-0326
12. Haller PM, Kazem N, Agewall S, Borghi C, Ceconi C, Dobrev D, et al. Oral anticoagulation in patients with left ventricular thrombus: a systematic review and meta-analysis. Eur Heart J Cardiovasc Pharmacother. 2024;10(5):444–53. doi:10.1093/ehjcvp/pvae042
13. Gogos C, Anastasiou V, Papazoglou AS, Daios S, Didagelos M, Kamperidis N, et al. Direct oral anticoagulants versus vitamin K antagonists for the management of left ventricular thrombus after myocardial infarction: a meta-analysis. Am J Cardiol. 2024;232:18–25. doi:10.1016/j.amjcard.2024.09.008
14. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl. 2012;2(1):1–138. Available from: https://www.sciencedirect.com/journal/kidney-international-supplements/vol/2/issue/1
15. Agha RA, Franchi T, Sohrabi C, Mathew G, Kerwan A, O'Neill N, et al. The SCARE 2020 guideline: updating consensus Surgical CAse REport (SCARE) guidelines. Int J Surg. 2020;84:226–30. doi:10.1016/j.ijsu.2020.10.034