Cureus
Part of SPRINGER NATURE

Open Access Case Report

# Bilateral Acute Limb Ischaemia Following Myocardial Infarction in a Patient With Left Ventricular Thrombus and Severe Peripheral Arterial Disease

**Review began** 10/17/2026
**Review ended** 10/27/2026
**Published** 10/30/2026

© **Copyright** 2026
Onyekwere et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

**DOI:** 10.7759/cureus.118827

Blaise Onyekwere [1], Adedapo Adesokan [1], Omoruyi Uwagboe [1], John Moreman [2]

1. Emergency Medicine, Sherwood Forest Hospitals NHS Foundation Trust, Sutton-in-Ashfield, GBR 2. Diagnostic Cardiology, Nottingham City Hospital, Nottingham University Hospitals NHS Trust, Nottingham, GBR

**Corresponding author:** Blaise Onyekwere, blaise.onyekwere@gmail.com

## Abstract

A 69-year-old woman presented with sudden severe bilateral lower-limb pain and paraesthesia one week after an episode of severe chest pain. Examination demonstrated bilateral pallor, cold limbs, profound sensory loss, absent distal pulses and left foot motor deficit. She was hypotensive and hypothermic, with a blood pressure of 88/55 mmHg and lactate of 3.5 mmol/L, raising concern for severe acute limb ischaemia (ALI).

Computed tomography (CT) angiography demonstrated extensive bilateral arterial occlusion with acute and chronic features, including acute-appearing thrombus at the right common femoral bifurcation and further superficial femoral and popliteal occlusions bilaterally. No acute aortic syndrome was identified. Cardiac investigations showed ST-segment elevation on her electrocardiogram (ECG), troponin greater than 7000, severe left ventricular (LV) systolic dysfunction (ejection fraction less than 35%) and an LV thrombus. The recent myocardial infarction, LV thrombus and fresh clot retrieved at embolectomy supported a cardioembolic mechanism.

The patient received unfractionated heparin and was transferred urgently to a tertiary vascular centre, where bilateral Rutherford IIb ALI was diagnosed. She underwent bilateral common femoral and right popliteal embolectomies with bilateral fasciotomies. Fresh thrombus was retrieved, although the right anterior compartment was non-viable. She subsequently recovered and was discharged on apixaban, aspirin and bisoprolol.

This case highlights the need for rapid recognition and treatment of bilateral ALI, consideration of cardiac embolism following myocardial infarction, exclusion of acute aortic pathology and urgent anticoagulation and revascularisation. It also demonstrates that acute thromboembolism can occur on a background of severe chronic peripheral arterial disease, producing a complex multifactorial presentation.

**Categories:** Cardiology, Emergency Medicine, Cardiac/Thoracic/Vascular Surgery
**Keywords:** acute limb ischemia, arterial embolism, left ventricular thrombus, myocardial infarction, peripheral arterial disease

## Introduction

Acute limb ischaemia (ALI) is a vascular emergency defined by a sudden reduction in limb perfusion, usually with a symptom duration of less than two weeks, that threatens tissue viability. Prompt recognition, assessment of limb viability and timely revascularisation are central to reducing the risk of irreversible neuromuscular injury, amputation and death [1-5]. The presentation may result from arterial embolism, in-situ thrombosis in diseased arteries, thrombosis of a previously treated vessel or less common causes such as dissection and trauma [1-3].

Bilateral ALI should raise concern for a proximal or systemic process, including aortic saddle embolism, acute aortic thrombosis, aortic dissection or systemic embolisation. However, acute occlusion may also occur in limbs with established peripheral arterial disease (PAD), making the distinction between embolism and thrombosis clinically important but not always straightforward [1-6]. Chronic atherosclerotic disease can limit baseline perfusion and collateral reserve, while superimposed acute thrombosis or embolism may precipitate abrupt and severe symptoms.

Left ventricular thrombus is a recognised complication of acute myocardial infarction, particularly when myocardial injury is extensive and LV systolic function is impaired. Thrombus within the LV can cause systemic embolisation, including peripheral arterial occlusion [7,8]. Although contemporary reperfusion and antithrombotic treatment have reduced the incidence of post-infarction LV thrombus, embolic

complications remain clinically important [7].

In the contemporary reperfusion era, left ventricular (LV) thrombus remains a recognised complication of ST-segment elevation myocardial infarction (STEMI). A systematic review and meta-analysis of patients treated with contemporary percutaneous coronary intervention reported a pooled LV thrombus incidence of 5.6% across all STEMI territories (95% confidence interval [CI], 4.3-7.0%) and 12.7% following anterior STEMI (95% CI, 9.8-15.6%) [9]. These estimates describe LV thrombus formation, not the incidence of clinical embolism. LV thrombus formation is explained by Virchow's triad: endocardial injury and inflammation associated with myocardial infarction, blood stasis due to impaired LV contraction (particularly apical akinesis or dyskinesis), and a prothrombotic state. Large infarct size, anterior STEMI, severe LV systolic dysfunction and apical wall-motion abnormalities increase the risk of thrombus formation. Once formed, an LV thrombus may embolise into the systemic circulation and cause complications including acute limb ischaemia [7-9].

The clinical assessment of ALI includes pain, pallor, pulselessness, poikilothermia, paraesthesia and paralysis. Sensory loss and motor deficit are concerning signs of a threatened limb and should prompt immediate vascular assessment [1-3,10]. We report a case of severe bilateral ALI following a probable recent myocardial infarction, with echocardiographically identified LV thrombus and extensive chronic PAD, in which fresh thrombus was retrieved from multiple arterial territories.

## Case Presentation

A 69-year-old woman presented to the Resuscitation Department of a district general hospital in the East Midlands at 20:15 with sudden-onset severe bilateral lower-limb pain and paraesthesia. She reported an episode of severe chest pain approximately one week earlier for which she had not sought medical attention. On the day of presentation, she developed bilateral leg pain from approximately 18:00, which progressed rapidly.

On initial vascular assessment, summarised in Table *1* below, only the femoral pulses were palpable. Both lower limbs were pale and insensate to the level of the thighs, and she was unable to move the left foot.

| Clinical finding | Right lower limb | Left lower limb |
|---|---|---|
| Appearance / temperature | Pale; cold peripheries | Pale; cold peripheries |
| Femoral pulses | Palpable | Palpable |
| Distal pulses | Absent | Absent |
| Sensation | Insensate to the level of the thighs | Insensate to the level of the thighs |
| Motor function | reduced power | Unable to move the foot |
| Bedside arterial ultrasound | Absent or significantly reduced arterial perfusion below the femoral arteries | Absent or significantly reduced arterial perfusion below the femoral arteries |
| Rutherford classification | Treating vascular team recorded bilateral category IIb ALI; separate limb-specific criteria not documented | Treating vascular team recorded bilateral category IIb ALI; separate limb-specific criteria not documented |

**TABLE 1: Documented clinical findings in the lower limbs**

ALI: Acute limb ischaemia

She was alert with a Glasgow Coma Scale (GCS) score of 15/15 [11], but appeared severely unwell and was in considerable pain. Her blood pressure was 88/55 mmHg, heart rate 98 beats/min, respiratory rate 18 breaths/min, oxygen saturation 98% on room air and temperature 34°C. The initial lactate concentration was 3.5 mmol/L.

Given the acute bilateral presentation and profound neurological deficit, acute aortic pathology, including aortic dissection, was considered. Clinical examination demonstrated bilateral lower limb tenderness, pallor, cold peripheries and absent distal pulses, with sensory loss extending to the thighs and left-foot motor deficit. These findings were consistent with severe ALI and an immediately threatened limb; the clinical

classification was subsequently confirmed as Rutherford category IIb [1-3,9].

Initial laboratory investigations showed marked leucocytosis and neutrophilia, elevated lactate, hyponatraemia, hypoalbuminaemia and a markedly elevated troponin T concentration, as summarised in Table *2* below.

| Investigation | Patient result | Reference range | Interpretation |
|---|---|---|---|
| White blood cell count (× $10^9$/L) | 28.56 | 3.6–10.2 | Elevated |
| Neutrophil count (× $10^9$/L) | 21.91 | 1.7–7.5 | Elevated |
| Lactate (mmol/L) | 3.5 | 0.1–1.8 | Elevated |
| Sodium (mmol/L) | 129 | 135–145 | Low |
| Potassium (mmol/L) | 4.9 | 3.5–5.3 | Within range |
| Albumin (g/L) | 27 | 35–50 | Low |
| Alkaline phosphatase (U/L) | 123 | 30–130 | Within range |
| Activated partial thromboplastin time (s) | 24.7 | 27.0–37.3 | Slightly shortened |
| Prothrombin time (s) | 12.8 | 11.6–14.6 | Within range |
| Troponin T (ng/L) | >7,000 | <14 | Markedly elevated |

**TABLE 2: Initial laboratory investigations**

The elevated lactate, in combination with hypotension and severe bilateral limb ischaemia, was concerning for significant tissue and systemic hypoperfusion. The marked leucocytosis and neutrophilia may have reflected physiological stress associated with acute severe tissue ischaemia, although alternative causes would need to be considered in the clinical context.

Bedside vascular ultrasound demonstrated absent or significantly reduced arterial perfusion below the femoral arteries bilaterally. Urgent CT angiography of the aorta and lower-limb arterial circulation was performed because of concern for acute aortic pathology. The aorta and major branches to the distal external iliac arteries opacified, and no dissection flap, penetrating ulcer, intramural haematoma or contrast extravasation was identified. Consultant radiological review concluded that there was no acute aortic syndrome.

The CT angiogram instead demonstrated extensive multilevel arterial occlusion with both acute and chronic features. On the right, there was complete occlusion of the profunda femoris artery, considered likely to be chronic, and acute-appearing thrombus at the common femoral artery bifurcation extending into the profunda femoris and superficial femoral artery origins (Figure *1*). There was also long-segment occlusion of the distal superficial femoral artery extending into the popliteal artery, with minimal opacification of the anterior tibial, peroneal and posterior tibial arteries. The absence of distal contrast opacification was considered potentially related to distal embolisation or contrast timing.

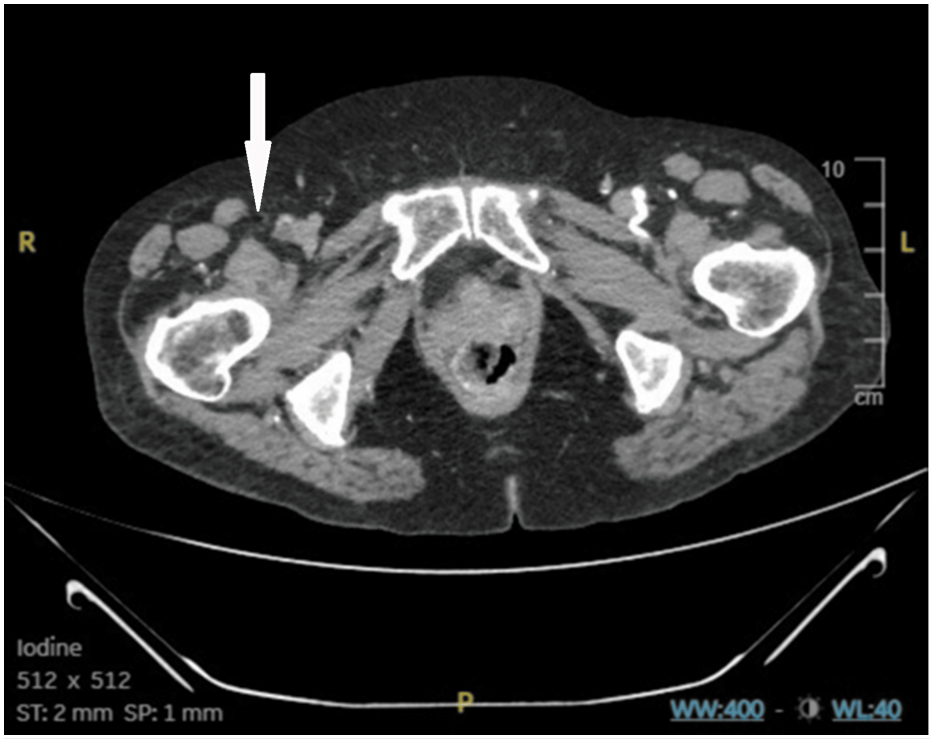



**FIGURE 1: CT image showing right common femoral occlusion**

On the left, CT angiography showed a shorter segment of mid-superficial femoral artery occlusion with distal recanalisation (Figure *2*). Further thrombus was present in the distal superficial femoral artery at the adductor canal, with weak reconstitution at the P1 segment of the popliteal artery. The popliteal artery was completely occluded, with no contrast beyond the P2 segment. There was thready opacification of the anterior tibial and peroneal arteries and no opacification of the posterior tibial artery. Overall, the imaging showed extensive bilateral multilevel occlusive disease without acute aortic pathology.

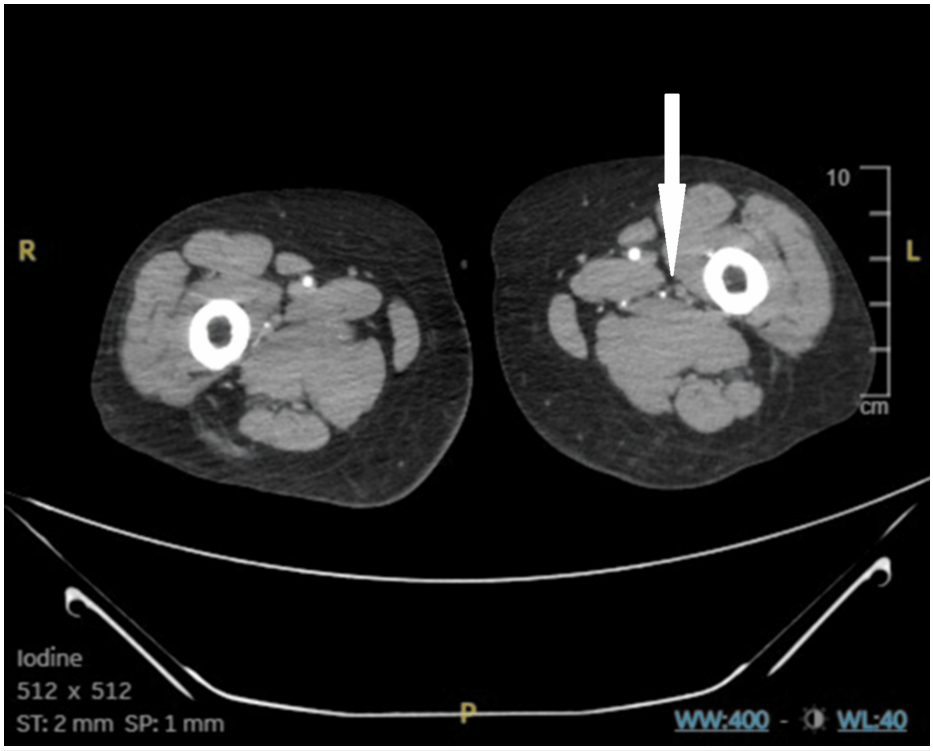



**FIGURE 2: CT image showing left superficial femoral artery occlusion**

The history of severe chest pain approximately one week earlier prompted cardiac investigation. Electrocardiography (ECG) showed ST-segment elevation in leads I, II and V4-V6, and troponin T was greater than 7,000 ng/L. Echocardiography performed demonstrated an LV thrombus (Figure 3) and severe LV systolic dysfunction, with an estimated ejection fraction below 35%. A pericardial effusion was also reported (Figure 4). The CT angiogram showed a pericardial effusion measuring approximately 13 mm in axial thickness with attenuation of approximately 40 Hounsfield units; this appearance raised the possibility of blood or proteinaceous material but was not, by itself, diagnostic of the effusion's composition.

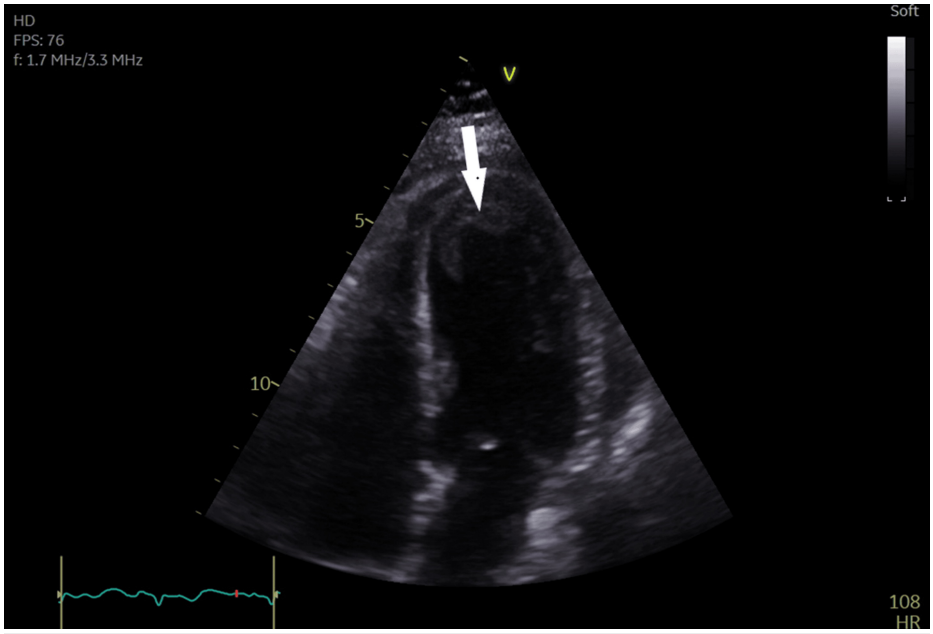



**FIGURE 3: ECHO showing LV thrombus**

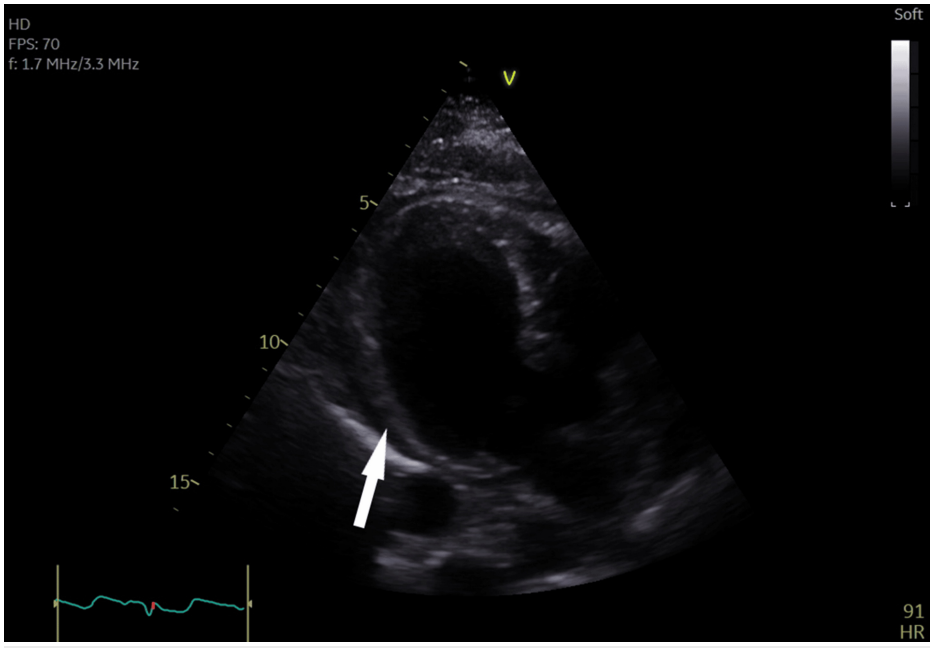



**FIGURE 4: ECHO showing pericardial effusion**

The recent chest-pain episode, ECG changes, markedly elevated troponin, severe LV systolic dysfunction and documented LV thrombus supported a plausible cardiac source of systemic embolisation [7,8]. The patient was urgently discussed with the tertiary vascular team. She received 5,000 IU of unfractionated heparin before transfer and arrived at the tertiary vascular centre at 23:01, approximately 2 hours and 46 minutes after arrival at the district general hospital.

At the tertiary vascular centre, the patient was diagnosed with bilateral Rutherford category IIb ALI [1,2,10]. She underwent bilateral common femoral artery embolectomies, right popliteal artery embolectomy and bilateral lower-limb fasciotomies. Fresh clot was extracted during embolectomy, and the right anterior compartment was found to be non-viable. She was subsequently discharged on bisoprolol 2.5 mg once daily, apixaban 5 mg twice daily and aspirin 75 mg once daily. Vascular and cardiology follow-up and regular physiotherapy were arranged.

## Discussion

This case illustrates severe bilateral ALI occurring approximately one week after a likely myocardial infarction in a patient with a documented LV thrombus and extensive pre-existing PAD. The principal diagnostic challenge was to determine whether the arterial occlusions reflected a proximal aortic event, cardioembolism, in-situ thrombosis in diseased arteries or a combination of mechanisms. The abrupt bilateral symptoms and profound neurological deficit warranted urgent consideration of acute aortic pathology. CT angiography excluded an acute aortic syndrome and delineated the multilevel peripheral arterial occlusions, allowing urgent specialist vascular management.

The cardiac findings support an acute thromboembolic contribution. LV thrombus can form after myocardial infarction through the interaction of myocardial injury, blood stasis associated with impaired ventricular contraction and a prothrombotic milieu [7]. It is a recognised source of systemic emboli, and embolic complications remain a major concern in patients with post-infarction LV thrombus [7,8]. In this patient, the recent chest-pain episode, ST-segment elevation, markedly elevated troponin, severe LV systolic dysfunction and echocardiographically identified LV thrombus form a coherent clinical picture supporting cardioembolism.

The retrieval of fresh clot from the common femoral and right popliteal arterial territories provides additional evidence of an acute occlusive process. Nevertheless, the findings do not prove that every occlusion was embolic. CT angiography also demonstrated chronic-appearing profunda femoris occlusion and extensive multilevel arterial disease. ALI can arise from embolism or thrombosis superimposed on PAD, and the two mechanisms may coexist [1-3,6]. Chronic disease may have reduced the perfusion reserve of both limbs and contributed to the severity and distribution of the presentation. The most cautious interpretation is therefore acute thromboembolism superimposed on severe chronic PAD.

The clinical severity is reflected in the neurological deficit and subsequent Rutherford category IIb classification. The Rutherford system identifies category IIb limbs as immediately threatened, with salvage dependent on prompt revascularisation [1,2,10]. Contemporary guidance emphasises emergency assessment of limb viability, rapid vascular specialist involvement and revascularisation for salvageable threatened limbs. Systemic unfractionated heparin is recommended at diagnosis unless contraindicated, to limit thrombus propagation while definitive treatment is organised [1,2,4,5]. In this case, heparin was administered before transfer and the patient reached the vascular centre less than three hours after initial hospital arrival.

Revascularisation was followed by bilateral fasciotomies. Severe ischaemia and reperfusion can cause swelling and compartment syndrome, which may further compromise tissue perfusion and function; fasciotomy may be required when the clinical circumstances indicate substantial risk [1-3]. The finding of a non-viable right anterior compartment underscores the extent of tissue injury already present. It also reinforces that even rapid escalation may not fully reverse damage once prolonged or severe ischaemia has occurred.

This case also demonstrates the value and limits of diagnostic imaging. Bedside vascular ultrasound was reported to show markedly reduced arterial perfusion, while CT angiography excluded acute aortic pathology and mapped the occlusions. Current guidance supports rapid clinical assessment and bedside Doppler evaluation; additional imaging should be selected when it is likely to change management and must not introduce an unacceptable delay to treatment [1,2]. Here, concern for an aortic cause made urgent CT angiography clinically relevant.

The patient was discharged on apixaban for the documented LV thrombus, alongside aspirin and bisoprolol. Anticoagulation decisions for LV thrombus should account for embolic risk, bleeding risk, thrombus characteristics, ventricular function and follow-up imaging. The American Heart Association scientific statement discusses oral anticoagulation for post-infarction LV thrombus and notes that direct oral anticoagulants may be a reasonable alternative to vitamin K antagonists in selected patients, although treatment duration and follow-up should be individualised [8,12]. The case report does not establish comparative efficacy of apixaban or the optimal duration of therapy; these decisions should remain under the treating cardiology team with follow-up imaging.

The main limitation of this report is that the interpretation of the embolic mechanism is based on the clinical chronology, cardiac findings, CT angiography and operative clot retrieval rather than direct pathological confirmation linking the retrieved clot to the LV thrombus.

### Learning points

Bilateral ALI should prompt consideration of proximal aortic disease and systemic embolisation, including embolism from an LV thrombus after myocardial infarction. Acute and chronic arterial disease can coexist, so a cardiac embolic source should not lead clinicians to disregard underlying PAD. Sensory loss and motor deficit are signs of a threatened limb. Rapid assessment of limb viability, early vascular consultation, anticoagulation when not contraindicated and timely revascularisation are central to limb salvage.

In a patient with recent myocardial infarction and severe LV systolic dysfunction, an LV thrombus is a plausible source of systemic arterial embolisation. The mechanism should be described as supported by the overall clinical picture unless direct evidence proves causation. The choice and duration of anticoagulation for LV thrombus require individualised specialist assessment, including consideration of bleeding risk and follow-up cardiac imaging.

## Conclusions

Bilateral ALI is an uncommon and potentially devastating vascular emergency. In this patient, recent chest pain, ECG changes, markedly elevated troponin, severe LV systolic dysfunction, a documented LV thrombus and fresh thrombus retrieved during embolectomy strongly supported an acute thromboembolic component. Extensive chronic PAD was also present, indicating a multifactorial vascular presentation. Rapid recognition of threatened limb viability, assessment for acute aortic pathology, prompt anticoagulation, urgent vascular referral and definitive revascularisation were central to management.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Blaise Onyekwere, Adedapo Adesokan, Omoruyi Uwagboe

**Acquisition, analysis, or interpretation of data:** Blaise Onyekwere, Adedapo Adesokan, John Moreman

**Drafting of the manuscript:** Blaise Onyekwere, Adedapo Adesokan

**Critical review of the manuscript for important intellectual content:** Blaise Onyekwere, Omoruyi Uwagboe, John Moreman

### Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

## References

1. Björck M, Earnshaw JJ, Acosta S, et al.: Editor's choice—European Society for Vascular Surgery (ESVS) 2020 clinical practice guidelines on the management of acute limb ischaemia. Eur J Vasc Endovasc Surg. 2020, 59:173-218. 10.1016/j.ejvs.2019.09.006
2. Gornik HL, Aronow HD, Goodney PP, et al.: 2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS guideline for the management of lower extremity peripheral artery disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2024, 149:e1313-e1410. 10.1161/CIR.0000000000001251
3. Natarajan B, Patel P, Mukherjee A: Acute lower limb ischemia - etiology, pathology, and management . Int J Angiol. 2020, 29:168-174. 10.1055/s-0040-1713769
4. McNally MM, Univers J: Acute limb ischemia. Surg Clin North Am. 2018, 98:1081-1096. 10.1016/j.suc.2018.05.002
5. Arnold J, Koyfman A, Long B: High risk and low prevalence diseases: acute limb ischemia . Am J Emerg Med. 2023, 74:152-158. 10.1016/j.ajem.2023.09.052
6. Olinic DM, Stanek A, Tătaru DA, Homorodean C, Olinic M: Acute limb ischemia: an update on diagnosis and management. J Clin Med. 2019, 8:1215. 10.3390/jcm8081215
7. Camaj A, Fuster V, Giustino G, et al.: Left ventricular thrombus following acute myocardial infarction: JACC state-of-the-art review. J Am Coll Cardiol. 2022, 79:1010-1022. 10.1016/j.jacc.2022.01.011
8. Levine GN, McEvoy JW, Fang JC, et al.: Management of patients at risk for and with left ventricular

thrombus: a scientific statement from the American Heart Association. Circulation. 2022, 146:205-223. 10.1161/CIR.0000000000001092

9. Sacoransky E, Yu Jia Ke D, Dave P, Alexander B, El Sherbini A, Abunassar J, Abuzeid W: Incidence of left ventricular thrombus following STEMI in the modern era via multimodality imaging: a systematic review and meta-analysis. Int J Cardiol Heart Vasc. 2024, 52:101396. 10.1016/j.ijcha.2024.101396
10. Rutherford RB, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, Jones DN: Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg. 1997, 26:517-538. 10.1016/S0741-5214(97)70045-4
11. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale . Lancet. 1974, 304:81-84. 10.1016/S0140-6736(74)91639-0
12. Haller PM, Kazem N, Agewall S, et al.: Oral anticoagulation in patients with left ventricular thrombus: a systematic review and meta-analysis. Eur Heart J Cardiovasc Pharmacother. 2024, 10:444-453. 10.1093/ehjcvp/pvae042