Cureus
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Open Access Case Report

Coronary Embolism Followed by Concurrent Superior Mesenteric Artery and Infrarenal Aortic Embolism in Atrial Fibrillation Despite Full Anticoagulation and Dual Antiplatelet Therapy: A Fatal Case of Acute Mesenteric Ischemia

Review began 11/05/2026
Review ended 11/12/2026
Published 11/15/2026

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DOI: 10.7759/cureus.119699

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Abstract

We describe a patient with atrial fibrillation (AF) in whom embolic events involved the coronary, mesenteric, renal, and aortic territories within four days, the last of them during full-dose anticoagulation and dual antiplatelet therapy.

A 52-year-old woman presented in AF with ST-segment elevation myocardial infarction caused by thrombi in the proximal and distal apical segments of the left anterior descending artery, without atherosclerotic disease; revascularization was planned for a later stage. Echocardiography showed an apical left ventricular (LV) thrombus, and therapeutic low-molecular-weight heparin was started. On the fourth day after admission, despite full-dose anticoagulation and dual antiplatelet therapy, she developed abdominal pain and distension with hemodynamic instability. Computed tomography angiography showed an extensive filling defect in the superior mesenteric artery from its origin, a concurrent filling defect in the infrarenal abdominal aorta, left renal infarction, pneumatosis intestinalis, and portal venous gas. Emergency laparotomy revealed necrosis of the entire small bowel and colon with microperforations. Given the non-salvageable extent of necrosis, the abdomen was closed, and comfort care was provided. The patient died 12 hours later.

In this patient, a second embolic event occurred within days of an embolic myocardial infarction despite therapeutic anticoagulation, and the CT findings at diagnosis already indicated non-salvageable bowel. The case illustrates why new abdominal symptoms in an anticoagulated patient with a recent cardioembolic event warrant immediate imaging and surgical assessment.

Categories: Radiology, General Surgery, Cardiac/Thoracic/Vascular Surgery
Keywords: acute mesenteric ischemia, atrial fibrillation, coronary embolism, left ventricular thrombus, low-molecular-weight heparin, pneumatosis intestinalis, portal venous gas, superior mesenteric artery embolism

Introduction

Acute mesenteric ischemia (AMI) is a sudden interruption of intestinal blood flow that progresses from mucosal injury to transmural necrosis, perforation, and death if not treated promptly. It results from four mechanisms: arterial embolism, arterial thrombosis, non-occlusive mesenteric ischemia, and mesenteric venous thrombosis [1,2]. AMI accounts for a small fraction of acute abdominal admissions, yet its in-hospital mortality remains between 50% and 80% in most series [1-3]. Arterial embolism to the superior mesenteric artery (SMA) is the most frequent mechanism, and the heart is the source in the large majority of cases [2,4].

The clinical picture changes as ischemia deepens. In the early phase, ischemia is limited to the mucosa, and the hallmark is severe abdominal pain out of proportion to the physical examination: the abdomen may be soft, with mild tenderness and no peritoneal signs [1,2]. Nausea, vomiting, and sudden bowel evacuation are common in embolic cases, and the triad of abdominal pain, gut emptying, and heart disease such as atrial fibrillation should raise immediate suspicion [2,4]. As necrosis becomes transmural, the examination shows abdominal distension, absent bowel sounds, guarding, rebound tenderness, and hemodynamic instability, sometimes with gastrointestinal bleeding [1,2]. Laboratory findings are nonspecific: leukocytosis, metabolic acidosis, and elevated lactate are common but appear late, and a normal lactate does not exclude the diagnosis [1]. Because early signs are subtle, diagnosis is often delayed, and CT angiography should be obtained as soon as AMI is suspected [1].

Atrial fibrillation (AF) is the classic embolic substrate, and its emboli can reach any arterial territory.

How to cite this article
Rizon M, Da Silva Eli G, Damasceno T K, et al. (November 15, 2026) Coronary Embolism Followed by Concurrent Superior Mesenteric Artery and Infrarenal Aortic Embolism in Atrial Fibrillation Despite Full Anticoagulation and Dual Antiplatelet Therapy: A Fatal Case of Acute Mesenteric Ischemia. Cureus 18(11): e119699. DOI 10.7759/cureus.119699

Coronary embolism accounts for about 3% of acute myocardial infarctions, and AF is its most frequent cause [5]. An anterior infarction can in turn produce left ventricular (LV) thrombus, a second potential embolic source [6,7]. Current recommendations support therapeutic anticoagulation in this setting [7].

Anticoagulation lowers but does not abolish embolic risk [8,9]. Reports of sequential coronary and mesenteric embolism, and of mesenteric embolism under documented therapeutic anticoagulation, are scarce [10-14], and simultaneous embolism to the SMA and infrarenal aorta is uncommon. We describe a fatal case combining these features and discuss its imaging findings and the surgical decision-making it required.

Case Presentation

A 52-year-old woman with previously diagnosed atrial fibrillation was admitted to Hospital do Rocio, a tertiary referral Hospital in Campo Largo, Brazil, through the chest pain protocol with chest pain. She was in atrial fibrillation (AF), and the admission electrocardiogram demonstrated AF with a ventricular rate of approximately 118 bpm and ST-segment elevation in leads V1-V5, consistent with an acute anterior ST-segment elevation myocardial infarction (STEMI) (Figure *1*).

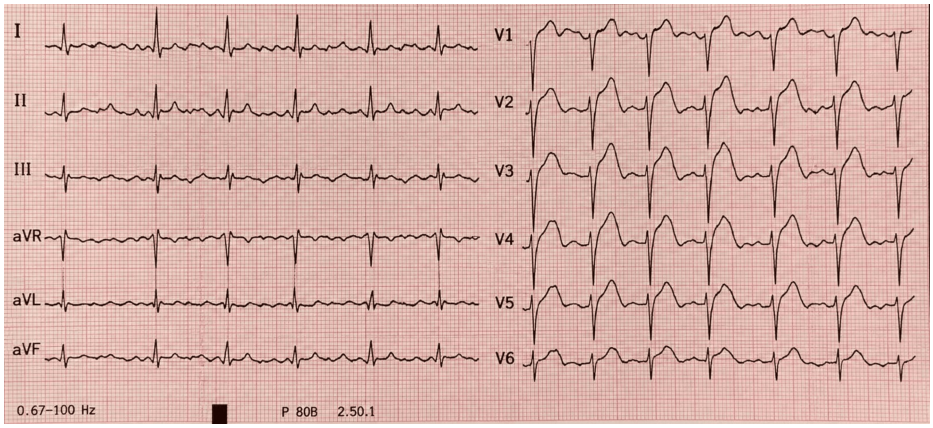


FIGURE 1: Admission electrocardiogram

Eletrocardiogram showing atrial fibrillation with a ventricular rate of approximately 118 bpm and ST-segment elevation in leads V1-V5, consistent with acute anterior myocardial infarction.

High-sensitivity troponin I was 7517.7 pg/mL, and creatine kinase-MB was 25.5 ng/mL. Coronary angiography through right radial access showed a right-dominant circulation. A thrombus was adhered to the wall of the proximal third of the left anterior descending artery (LAD), with preserved thrombolysis in myocardial infarction (TIMI) grade 3 distal flow, and a second thrombus was present in its distal apical segment (Figure *2*). There were no atherosclerotic lesions in the left main, circumflex, or right coronary arteries. No coronary intervention was performed; she was admitted to the intensive care unit for anticoagulation, with revascularization planned for a later stage. The embolic mechanism was attributed to AF.

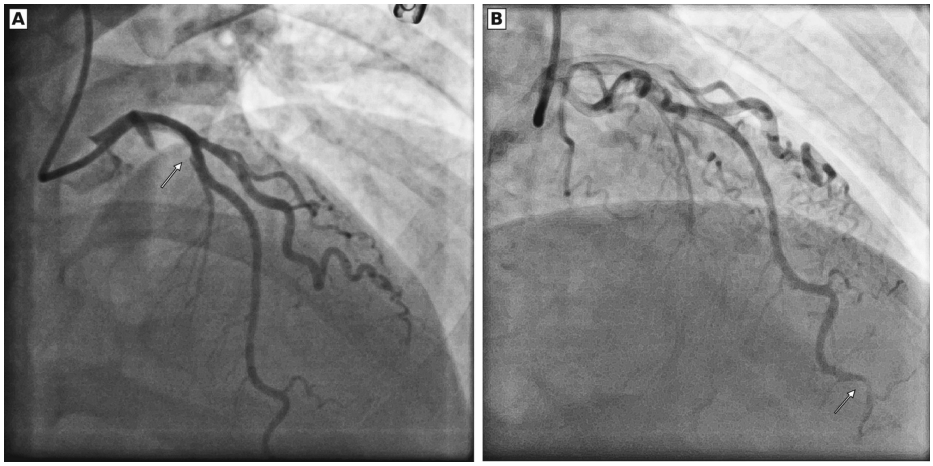


FIGURE 2: Coronary angiography

Left coronary angiography in two projections. (A) The arrow indicates the thrombus adhered to the wall of the proximal third of the left anterior descending artery, with preserved distal flow. (B) The arrow indicates thrombus in the distal apical segment of the left anterior descending artery. The left main and circumflex arteries show no atherosclerotic lesions.

Transthoracic echocardiography, performed shortly after admission, showed AF, a moderately enlarged left atrium, and a mildly dilated LV with an ejection fraction of 57% by Simpson's method. There was severe hypokinesia of the apical segments with akinesia of the apex, and an echogenic mass attached to the apical endocardium, consistent with a mural thrombus measuring approximately 18 × 12 mm. Right ventricular size and function and the cardiac valves were normal, and there was no pericardial effusion. AF persisted throughout the hospitalization. Given AF with a recent embolic event and the LV thrombus, she was started on enoxaparin 80 mg subcutaneously every 12 hours, a full therapeutic dose adjusted to body weight, together with dual antiplatelet therapy with aspirin 100 mg and clopidogrel 75 mg once daily. This regimen was maintained in the intensive care unit throughout the hospital stay. Her body weight was 80 kg, so this dose corresponded to 1 mg/kg. Estimated creatinine clearance (Cockcroft-Gault) was 64 mL/min on admission and 104 mL/min on day three. No doses were missed, and the last scheduled dose before the abdominal event was also given. Anti-Xa activity was not measured because the assay was not available at our institution.

On the fourth day after admission, at 16:00, while on optimized anticoagulation, she developed diffuse abdominal pain and distension. Physical examination confirmed marked abdominal distension with diffuse guarding and poor peripheral perfusion with cyanosis of the extremities. She was in shock that did not respond to crystalloid resuscitation and required vasoactive drugs, with escalating doses before surgery. This presentation corresponded to the late, transmural phase of AMI rather than the early pattern of pain out of proportion to examination findings. Laboratory results from admission to the postoperative period are shown in Table *1*.

Parameter	Day 0 (admission)	Day 2	Day 3	Day 5 (7 hours after surgery)	Reference range
Hemoglobin (g/dL)	11.6	–	–	11.4	12.0-16.0
Mean corpuscular volume (fL)	68.9	–	–	68.4	80.0-100.0
Mean corpuscular hemoglobin (pg)	22.1	–	–	21.9	26.0-34.0
Leukocytes (/mm³)	14,800	–	–	10,400	4000-10,000
Band neutrophils (%)	6	–	–	52	≤10
Platelets (/mm³)	633,000	–	–	687,000	140,000-400,000
Prothrombin time (INR)	0.97	0.95	–	1.14	0.8-1.2
Activated partial thromboplastin time (s)	29.5	30.3	–	28.5	24.9-37.0
Creatinine (mg/dL)	1.29	0.92	0.8	3.4	0.66-1.09
C-reactive protein (mg/L)	149.5	82.7	224	504.2	<5.0
High-sensitivity troponin I (pg/mL)	7517.70	–	1900.50	2503.50	<11.6
Creatine kinase-MB mass (ng/mL)	25.5	–	–	–	0.6-6.3
Arterial pH	7.33	–	–	7.14	7.35-7.45
Arterial bicarbonate (mEq/L)	19	–	–	8.4	22.0-26.0
Base excess (mEq/L)	-6.3	–	–	-12.6	-0.2 to 3.0
Arterial lactate (mmol/L)	1.4	–	–	14.1	<1.8

TABLE 1: Laboratory results from admission to the postoperative period

Day 0 values are from the admission samples; C-reactive protein was measured in a second sample on the same day. Postoperative samples were collected seven hours after laparotomy.

INR - international normalized ratio

Contrast-enhanced computed tomography angiography of the abdomen, performed two and a half hours after the onset of abdominal pain (pain onset at 16:00; image acquisition at 18:30), showed an extensive filling defect in the SMA beginning at its origin (Figure 3).



FIGURE 3: Superior mesenteric artery embolism

Axial contrast-enhanced CT angiography, arterial phase, at the level of the renal hila. The arrow indicates the non-opacified superior mesenteric artery arising from the opacified aorta, consistent with embolic occlusion beginning at its origin.

A second filling defect was present in the infrarenal abdominal aorta, with distended small bowel loops, pneumatosis intestinalis, and a wedge-shaped perfusion defect in the lateral portion of the left kidney, consistent with renal infarction (Figure *4*).

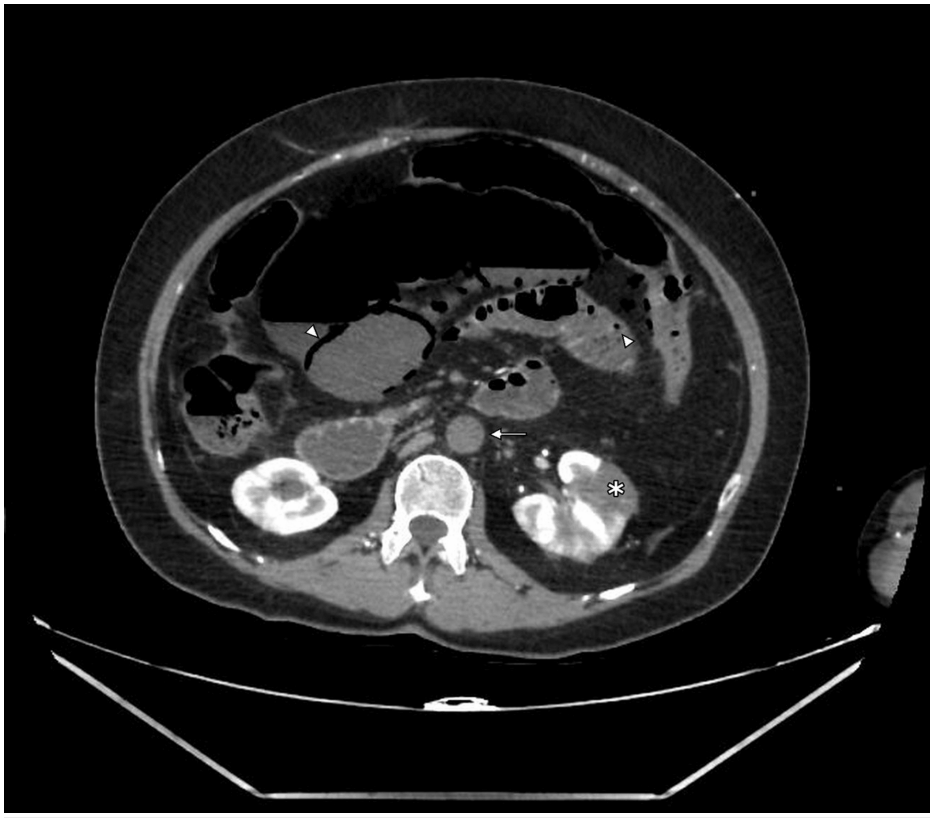


FIGURE 4: Infrarenal aortic embolism, renal infarction, and pneumatosis intestinalis

Axial contrast-enhanced CT angiography, arterial phase, below the renal arteries. The arrow indicates the non-opacified lumen of the infrarenal abdominal aorta, consistent with an occlusive embolus. Arrowheads indicate intramural gas in the wall of distended small bowel loops, consistent with pneumatosis intestinalis. The asterisk marks the non-enhancing lateral portion of the left kidney, consistent with renal infarction.

Gastric distension and branching gas in the periphery of the liver, consistent with portal venous gas, were also identified (Figure *5*).



FIGURE 5: Portal venous gas and gastric distension

Axial contrast-enhanced CT of the upper abdomen. Arrows indicate branching gas foci extending to the periphery of the hepatic parenchyma, consistent with portal venous gas. Marked gastric distension with an air-fluid level is also seen.

The patient was taken to the operating room for emergency laparotomy at 19:00. The entire small bowel and colon were necrotic, with multiple microperforations (Figure *6*). Resection was considered futile: the extent of necrosis was incompatible with survival, and the patient was hemodynamically unstable. The abdomen was closed, and after discussion with the family she was transferred to the intensive care unit for comfort care. She died 12 hours after surgery, before the planned coronary revascularization could be performed. The sequence of events is summarized in Table *2*.



FIGURE 6: Intraoperative findings

Intraoperative photograph at emergency laparotomy. Necrosis of the entire small bowel, with violaceous and gray-green segments, and of the colon, with hemorrhagic and edematous mesentery. No viable bowel segment was identified.

Time	Event
Day 0	Admission via chest pain protocol; AF and ST-segment elevation myocardial infarction
Day 0	Coronary angiography: thrombi in proximal and distal apical LAD, TIMI 3 flow, no atherosclerosis; revascularization planned for a later stage
Day 0	Echocardiography: AF, apical akinesia, apical LV thrombus (18 × 12 mm); ICU admission; enoxaparin 80 mg subcutaneously every 12 hours, aspirin, and clopidogrel
Day 4	16:00: abdominal pain, marked distension, guarding; shock refractory to crystalloids, escalating vasoactive drugs
Day 4	18:30: CT angiography, 2.5 hours after pain onset: SMA and infrarenal aortic emboli, left renal infarction, pneumatosis, portal venous gas
Day 4	19:00: emergency laparotomy; necrosis of entire small bowel and colon; abdomen closed
Day 4–5	Comfort care in ICU; death 12 hours after surgery

TABLE 2: Timeline of clinical events from admission to death

AF - atrial fibrillation; LAD - left anterior descending artery; TIMI - thrombolysis in myocardial infarction; LV - left ventricular; CT - computed tomography; SMA - superior mesenteric artery; ICU - intensive care unit

Written informed consent for publication of this case report and the accompanying images was obtained from the patient's next of kin.

Discussion

This case illustrates recurrent cardioembolism from AF across four arterial territories - coronary, mesenteric, renal, and aortic - within four days and despite therapeutic anticoagulation combined with dual antiplatelet therapy. The coronary event fulfilled the criteria for definite coronary embolism proposed by Shibata et al., with one major criterion, angiographic coronary thrombus without atherosclerotic components, and two minor criteria, the absence of significant stenosis in the remaining coronary arteries and AF as an embolic risk factor [5]. The apical LV thrombus, already present with apical akinesia on the echocardiogram obtained at admission, was a second potential source, and its timing relative to the infarction could not be established. Imaging cannot distinguish a left atrial from a ventricular origin of the mesenteric embolus, but both share the same pathophysiology and management implications.

The persistence of embolic risk under anticoagulation is explained by thrombus biology. Recently formed cardiac thrombi, in the left atrial appendage or in the LV apex after anterior infarction, are friable and most prone to embolize in the first days [6,7]. Anticoagulation prevents propagation and promotes resolution, but it does not immediately dissolve existing thrombus, so embolic events remain possible early in treatment [6,7]. Patients with coronary embolism also have higher mortality and a substantial rate of recurrent thromboembolism compared with other infarction patients [5]. Our patient embolized again within four days of the index coronary event, which supports treating an embolic infarction as a marker of high recurrent risk.

Few previously reported cases resemble ours. Hwang et al. described a patient with anterior myocardial infarction in whom a large pedunculated LV thrombus formed despite continuous heparin and warfarin; SMA embolism followed left ventriculography and was treated with embolectomy and LV aneurysmectomy [10]. Azari et al. reported mesenteric ischemia after a large mobile apical LV thrombus complicating myocardial infarction, and the thrombus was removed surgically [11]. In AF, Cheng and Wu described SMA occlusion in the days after an inferior myocardial infarction, in a patient whose heparin had been stopped because of occult gastrointestinal bleeding and a falling platelet count [12]. Pay et al. reported synchronous coronary and mesenteric thromboembolism with angiographic confirmation in both territories [13]. Jing et al. described SMA embolism in a patient taking reduced-dose rivaroxaban and attributed it to inadequate anticoagulation during catheter ablation [14]. Marschall et al. reported recurrent cerebral and coronary embolism in a patient with AF and mechanical valves despite an international normalized ratio at or above target, caused by a persistent left atrial appendage thrombus [15].

Our case differs from these reports in two respects. The mesenteric event occurred during uninterrupted, weight-adjusted, full-dose low-molecular-weight heparin (LMWH) combined with dual antiplatelet therapy, and the second event involved the SMA origin, the left kidney, and the infrarenal aorta at the same time. Larger series show that embolism under anticoagulation is not exceptional. In a pooled analysis of 5413 patients with AF and recent cerebral ischemia, about one in five were already taking oral anticoagulants, and prior anticoagulation was associated with a higher risk of recurrent stroke [8]. In patients with LV thrombus, stroke and systemic embolism occur with both vitamin K antagonists and direct oral anticoagulants [9]. Together, these data indicate that patients with an embolic infarction and a cardiac thrombus remain at risk of systemic embolism even when anticoagulation is considered adequate.

Other explanations should be considered before attributing such an event to anticoagulation failure. LMWH is usually given without laboratory monitoring [16], but drug exposure may be inadequate in patients with obesity or renal impairment. Our patient received 1 mg/kg every 12 hours with an estimated creatinine clearance of 64 to 104 mL/min, but anti-Xa activity could not be measured. Adequate anticoagulant exposure therefore cannot be confirmed, and we describe this event as embolism despite appropriately dosed therapeutic LMWH rather than as proven anticoagulation failure. Heparin-induced thrombocytopenia (HIT) can also cause arterial thrombosis during heparin therapy. The platelet count usually begins to fall four or more days after heparin is started, or within hours in patients exposed to heparin in the previous 100 days [17]. Because our patient had received heparin since the day of coronary angiography, the timing was compatible with HIT. Her platelet count did not fall; it was 633,000/mm^3 on admission and 687,000/mm^3 after surgery (Table *1*), which made HIT very unlikely. Thrombocytosis with microcytic, hypochromic anemia was present from admission, a pattern suggestive of a reactive process, but a myeloproliferative neoplasm was not investigated.

The pattern of this second embolic event explains its severity. Most SMA emboli lodge 3 to 10 cm beyond the origin, near the middle colic artery, sparing the proximal jejunum and transverse colon [2,4]. In our patient, the filling defect began at the SMA origin and coexisted with an infrarenal aortic defect and left renal infarction, suggesting a single large embolus that fragmented or a shower of fragments. Ostial occlusion removes this proximal sparing, which accounts for necrosis of the entire small bowel and of the right and transverse colon. Necrosis extending to the left colon suggests that inferior mesenteric flow was also compromised. Associated aortic, renal, or peripheral emboli should therefore be actively sought on CT, because they change both prognosis and revascularization strategy.

CT also showed how advanced the ischemia already was at diagnosis. Biphasic CT angiography is the diagnostic test of choice for suspected AMI and should be performed without delay, even in patients with

impaired renal function [1]. Pneumatosis intestinalis and portal venous gas indicate advanced ischemia; together, they correlate with transmural infarction and high mortality, although neither is pathognomonic in isolation [18,19]. Portal venous gas extends to the liver periphery, which distinguishes it from the central distribution of pneumobilia [20] (Figure *5*). In our patient, these signs were already present on CT obtained two and a half hours after the onset of abdominal pain. This suggests that ischemia began before symptoms were recognized, possibly because the patient was recovering from a recent myocardial infarction under close cardiac care, and that a short interval between symptom onset and imaging does not guarantee early-stage disease. C-reactive protein, which had fallen from 149.5 mg/L on admission to 82.7 mg/L on day two, rose again to 224.0 mg/L on day three, the day before abdominal symptoms (Table *1*). This second rise is nonspecific, but it is consistent with ischemia beginning before symptoms were recognized.

These findings framed the surgical decision. Current guidelines recommend immediate laparotomy in patients with peritonitis or signs of necrosis, with revascularization before resection when bowel is potentially viable and damage-control surgery with a planned second look [1,3]. Endovascular and hybrid approaches suit stable patients without signs of transmural necrosis [1,3]. In our patient, pan-intestinal necrosis with perforation in an unstable patient made revascularization and resection futile. Total enterocolectomy would have left a patient with a recent infarction dependent on lifelong parenteral nutrition, with a minimal chance of surviving the operation. Exploration with abdominal closure and comfort care, agreed upon with the family, is an accepted option in this setting [1,3].

This report has limitations. It describes a single patient, so the association between embolic infarction, LV thrombus, and early recurrent embolism under anticoagulation cannot be generalized. The source of the mesenteric embolus, left atrial or ventricular, could not be determined. Because no bowel resection was performed, there was no surgical specimen for histological confirmation of the embolic material. Anti-Xa activity was not available, so inadequate drug exposure cannot be excluded with certainty, and a myeloproliferative neoplasm was not investigated as a contributor to thrombosis.

Conclusions

This case shows that AF can cause recurrent embolism to the coronary, mesenteric, renal, and aortic territories within days of an embolic myocardial infarction associated with LV thrombus, despite full-dose anticoagulation and dual antiplatelet therapy. In such patients, anticoagulation should not lower clinical suspicion: new abdominal pain, distension, or unexplained hemodynamic deterioration warrants immediate CT angiography. In this patient, ostial SMA occlusion, concurrent aortic embolism, pneumatosis intestinalis, and portal venous gas indicated non-salvageable bowel and supported a timely discussion of comfort care with the family.

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.

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Drafting of the manuscript: Marcelo Rizon, Gabriel Da Silva Eli, Tauana K. Damasceno, Monica Celezinsky

Critical review of the manuscript for important intellectual content: Marcelo Rizon, Monica Celezinsky

Supervision: Marcelo Rizon, Gabriel Da Silva Eli, Tauana K. Damasceno, Monica Celezinsky

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors have declared that no AI tools were used during the work or in the drafting of this article. The authors take full responsibility for the work. **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.

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