

Catastrophic visceral arterial embolization after intra-aortic balloon pump withdrawal: a case report of mechanical thrombus stripping

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<https://doi.org/10.26599/1671-5411.2026.06.008>

Intra-aortic balloon pump (IABP) counterpulsation remains one of the most accessible forms of temporary mechanical circulatory support for cardiogenic shock and high-risk coronary intervention. Its physiologic benefits, augmentation of diastolic coronary perfusion and reduction of afterload, make it a pragmatic bridge during periods of profound instability. Nonetheless, IABP therapy carries clinically meaningful complications, including access-site bleeding, limb ischemia, and less commonly visceral ischemia. Reported vascular complication rates vary widely across contemporary series, ranging from 0.94% to 31.1%,^[1] reflecting differences in patient risk profiles, device size, anticoagulation practices, and monitoring strategies. In routine practice, clinicians often navigate a narrow therapeutic margin: preventing thromboembolism while minimizing bleeding, particularly in elderly patients with shock physiology and diffuse atherosclerosis.

To our knowledge, this case represents a rare documented instance of catastrophic, multi-territory visceral arterial embolization occurring within minutes of IABP withdrawal, with imaging findings suggestive of mechanical thrombus stripping along the catheter path. Previous studies have largely attributed IABP-related visceral ischemia to balloon malposition or length mismatch, causing mechanical compromise of the major abdominal branches.^[2–4] In contrast, removal-triggered embolic showering is rarely reported, diagnostically misleading, and may rapidly progress beyond salvage. By coupling a classic diagnostic challenge (early non-specific abdominal computed tomography findings) with decisive computed tomography angiography (CTA) evidence and an anatomic “track sign” of the thrombus, this report provides a practical warning rele-

vant to the full IABP lifecycle, including weaning and removal.

A 76-year-old man with a history of hypertension and dyslipidemia presented with acute chest pain and profound hypotension (75/50 mmHg). Electrocardiography revealed diffuse ST-segment depression with ST-segment elevation in lead aVR, consistent with severe left main or proximal multivessel ischemia. Emergent coronary angiography confirmed critical left main coronary stenosis (> 95%) involving the left anterior descending artery/left circumflex with impaired flow (Thrombolysis in TIMI flow grade 1) and severe right coronary artery stenosis (95%). Cardiogenic shock developed before revascularization (cardiac index: 1.8 L/min per m²; lactate: 4.2 mmol/L). Given the hemodynamic collapse and high-risk anatomy, an IABP (40 cc, 7.5 Fr sheath) was inserted via the right common femoral artery under fluoroscopy. With 1:1 counterpulsation, perfusion parameters improved (cardiac index: 2.3 L/min per m²; mean arterial pressure: 70 mmHg). Primary percutaneous coronary intervention was performed, with the implantation of a drug-eluting stent in the left main coronary artery and restoration of TIMI flow grade 3. The patient was transferred to the cardiac intensive care unit for stabilization and consideration of a staged right coronary intervention.

Initial laboratory evaluation showed hemoglobin of 132 g/L, platelet count of 198×10^9 /L, creatinine of 98 μmol/L, and D-dimer levels of 2.41 mg/L. Unfractionated heparin was initiated with a target activated partial thromboplastin time (aPTT) of 60–80 s. On hospital Day 2, gross hematuria with clots developed, accompanied by a rapid hemoglobin decline to 89 g/L within approximately 12 h. Cystoscopy revealed diffuse bladder mucosal bleeding (aPTT:

82 s). Unfractionated heparin was continued; however, due to active bleeding, the intensity of anticoagulation was intentionally reduced to achieve a lower target aPTT range (35–45 s), with dose adjustments guided by continuous infusion and serial aPTT monitoring. This approach prioritized control of hematuria while maintaining a minimal antithrombotic effect. Transthoracic echocardiography on Day 3 revealed left ventricular dilation (left ventricular end-diastolic dimension: 62 mm), reduced systolic function (left ventricular ejection fraction: 35%), and dense spontaneous echo contrast (SEC) (grade 3–4) throughout the left ventricular cavity, without a definite intracardiac thrombus (Figure 1).

On Day 7, following hemodynamic stabilization, the IABP was weaned (1:1 → 1:2 → 1:3) and placed on complete standby for 30 min prior to removal (aPTT: 38 s). The device was uneventfully withdrawn. Within 5 min, the patient developed abrupt, severe, diffuse abdominal pain (9/10), abdominal distension, and mild hypotension. Early abdominal computed tomography revealed gallbladder distension and wall edema, raising concerns about acalculous cholecystitis. However, the clinical course rapidly deteriorated with rising lactate (6.4 mmol/L), marked D-dimer elevation (12.79 mg/L), substantial transaminase increase, and acute kidney injury. Given the initial computed tomography interpretation suggesting acalculous cholecystitis and concerns about contrast-induced nephropathy in the setting of progressive acute kidney injury, vascular imaging was initially deferred in favor of supportive measures and close observation of the patient.

However, as the patient's biochemical parameters continued to deteriorate markedly over the subsequent hours, CTA was ultimately performed 18 h after IABP removal and demonstrated catastrophic visceral arterial embolization: multiple filling defects in the superior mesenteric artery branches, near-complete occlusion of the proper hepatic artery, splenic infarction, bilateral renal cortical infarctions, and segmental small-bowel ischemia with pneumatosis (Figure 2). Notably, an irregular mural thrombus was present along the right iliac arterial catheter tract, supporting a withdrawal-associated thrombus-stripping mechanism. Emergency surgery was recommended by the multidisciplinary team but was declined by the family. Despite maximal vasopressor therapy and intensive care support, the patient progressed to refractory shock with multiorgan failure and died on Day 9 of hospitalization.

This case highlights an uncommon but devastating com-

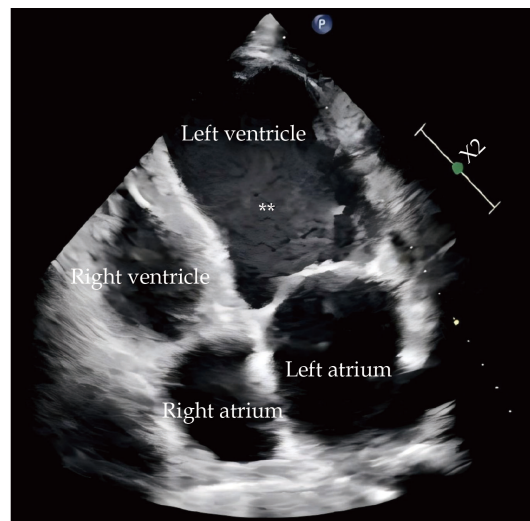


Figure 1 Transthoracic echocardiography demonstrating severe spontaneous echocontrast. Apical four-chamber transthoracic echocardiographic view obtained on hospital Day 3 showing dense spontaneous echocontrast (grade 3–4) within the left ventricular cavity (asterisks), appearing as swirling “smoke-like” echogenicity with fine particulate (“sludge-like”) echoes, consistent with marked blood stasis and a prothrombotic milieu during IABP support. No definite intracardiac thrombus was visualized on this view. IABP: intra-aortic balloon pump.

plication occurring during the “routine” phase of IABP therapy: device withdrawal. While IABP adverse events are commonly associated with bleeding and limb ischemia, the present case was characterized by a minute-scale onset of severe abdominal pain immediately after removal, followed by multi-territory visceral arterial occlusions on CTA. The temporal proximity to withdrawal, together with the arterial distribution (mesenteric, hepatic, splenic, and renal) and the iliac catheter-track thrombus, forms a coherent mechanistic narrative of mechanical thrombus stripping and embolic showering. This pattern differs from the more frequently discussed paradigm of visceral ischemia due to balloon malposition or anatomic mismatch, which can directly compromise the celiac axis or the superior mesenteric artery while the device remains *in situ*.^[2–4] In contrast, in our patient, the trigger was withdrawal itself, emphasizing that the vulnerable window extends beyond insertion and maintenance to weaning and removal.

Several lines of evidence support the thrombus-stripping mechanism in this case. Firstly, the immediate temporal relationship between device withdrawal and symptom onset (≤ 5 min) argues against progressive *in situ* thrombosis. Secondly, the multi-territory distribution pattern is consistent with proximal embolic showering rather than a localized mechanical obstruction. Thirdly, the presence of a

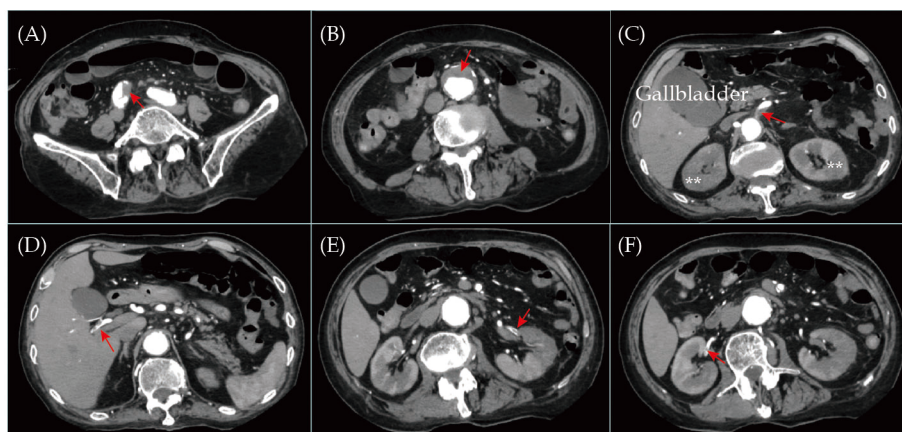


Figure 2 Computed tomography angiography demonstrating catheter-track thrombus and catastrophic visceral embolization after IABP withdrawal. Representative axial CTA images obtained 18 h after IABP removal. (A) Pelvic level showing an irregular thrombus within the right iliac artery along the presumed catheter pathway (red arrow); (B) mural thrombus within the abdominal aorta along the catheter track (red arrow); (C) filling defect in the superior mesenteric artery trunk/branches consistent with acute embolization (red arrow), accompanied by a distended gallbladder and renal cortical perfusion defects (asterisks); (D) near-complete occlusion of the proper hepatic artery with markedly reduced hepatic arterial perfusion (red arrow); and (E & F) bilateral renal artery thromboembolic signs (intraluminal filling defects, red arrows) with downstream renal cortical infarction (asterisks). Additional ischemic findings, including splenic infarction and segmental bowel ischemia with pneumatosis, were observed on the full CTA series (not shown). CTA: computed tomography angiography; IABP: intra-aortic balloon pump.

residual mural thrombus along the catheter track on CTA provides direct anatomical evidence of thrombus formation at the device-vessel interface. Last but not least, the prothrombotic milieu in the patient, evidenced by severe SEC, reduced anticoagulation intensity, and low cardiac output, created favorable conditions for catheter-associated thrombus formation.

The broader literature underscores why this complication can easily be missed. The rates of vascular complications associated with IABP therapy vary widely, reflecting heterogeneous populations and practices.^[1] Contemporary cohort data show that clinically relevant vascular events remain present even with the use of modern devices and protocols.^[5] The mechanisms of visceral ischemia include positional obstruction, embolization arising from device manipulation within atherosclerotic vessels, and amplification by systemic low-flow states.^[2-4] Diagnostic ambiguity is a central challenge; early abdominal symptoms may mimic non-ischemic entities, and computed tomography interpretation can be confounded by device-related artifacts, potentially delaying definitive vascular imaging.^[6] Anticoagulation strategies further contribute to the complexity of real-world studies. Bleeding is among the most frequent complications during IABP support, and de-escalation may be unavoidable in cases of hemorrhage. However, evidence comparing anticoagulated and non-anticoagulated IABP patients suggests that major bleeding and thrombotic endpoints

may not differ substantially across approaches in some settings.^[7,8] In our case, the clinical dilemma was explicit: severe bleeding mandated anticoagulation reduction, yet the patient remained in a prothrombotic state.

A key imaging clue in this patient was severe left ventricular SEC (grade 3–4), which serves as a visible surrogate for stasis and low-shear blood flow, conditions that promote erythrocyte aggregation and thrombin generation. Importantly, although no definite intracardiac thrombus was visualized on transthoracic echocardiography, the absence of a discrete left ventricular thrombus does not exclude thromboembolic risk. Grade 3–4 SEC represents a continuum of prothrombotic transformation, and clinical evidence demonstrates that severe left ventricular SEC is independently associated with increased thromboembolic events, even without frank thrombus formation.^[9] In cardiogenic shock, systemic hypoperfusion and inflammatory activation can further amplify coagulation, creating a thrombogenic environment that extends beyond the cardiac chambers. In our patient, the combination of SEC (grade 3–4), reduced left ventricular ejection fraction (35%), and subtherapeutic anticoagulation (aPTT: 38 s) likely extended the prothrombotic milieu to include the entire catheter-blood interface along the aortoiliac pathway. When coupled with reduced anticoagulation intensity and prolonged device duration (7 days), catheter-track thrombus formation is biologically plausible. The detection of mural thrombus along the iliac path-



way on CTA provides direct anatomic evidence that aligns with the clinical event of withdrawal-associated mechanical stripping with subsequent embolic dissemination. In retrospect, the presence of severe SEC suggests that reduced-intensity anticoagulation may have been insufficient to mitigate thrombotic risk in this high-risk setting; however, escalation was constrained by active bleeding. In this context, alternative anticoagulation strategies were considered; however, unfractionated heparin was maintained due to its short half-life and reversibility with protamine, which was deemed critical in the setting of active bleeding. Escalation or substitution with longer-acting agents was therefore not pursued.

The clinical and imaging constellation in this case, severe SEC, subtherapeutic anticoagulation, catheter-track thrombus, and hyperacute post-withdrawal timing, supports a mechanistically distinct scenario of withdrawal-triggered thrombus stripping in a prothrombotic milieu. This observation both extends and contrasts with the recent case series by Wohlfahrt, *et al.*^[10] who reported six cases of mesenteric ischemia occurring during active IABP support (1–19 days post-insertion). Their cases were primarily attributed to anatomic-to-device length mismatch, with the distal balloon extending beyond the renal arteries in four of six patients and atheroembolism from disrupted plaque. In contrast, our patient developed catastrophic multi-territory embolization within 5 min of device withdrawal despite appropriate balloon positioning during support, with CTA directly demonstrating a residual catheter-track mural thrombus, findings that implicate the removal process itself as the proximate trigger rather than *in situ* mechanical obstruction. Although alternative mechanisms, such as atheroembolism from catheter manipulation or dislodgement of pre-existing aortic plaque, cannot be definitively excluded, the temporal relationship to device withdrawal together with the catheter-track mural thrombus is most consistent with a withdrawal-associated mechanical process. Nevertheless, both experiences underscore a common clinical imperative: abdominal pain in IABP-treated patients, whether during support or immediately post-removal, warrants urgent CTA and heightened suspicion for visceral ischemia, given the high mortality (66% in the Wohlfahrt series, fatal in our case) associated with this complication.

Taken together, the present evidence supports a transition-phase hazard during standby, weaning, and removal, emphasizing that the vulnerable window extends beyond device insertion and maintenance into the weaning phase and removal. This case supports a workflow-level rule: abrupt

abdominal pain, rising lactate levels, or rapidly evolving organ dysfunction after IABP withdrawal should prompt urgent CTA to exclude catastrophic visceral ischemia and enable time-critical multidisciplinary intervention.

Catastrophic visceral arterial embolization can occur immediately after IABP withdrawal and may be driven by withdrawal-associated mechanical thrombus stripping along the catheter pathway in patients with a prothrombotic low-flow milieu. The early presentation is clinically deceptive and may mimic non-ischemic abdominal pathology, risking diagnostic delay. Clinicians should recognize weaning and removal as high-risk periods and intensify post-removal monitoring. Based on this experience and emerging case reports, we propose that abrupt abdominal pain accompanied by biochemical deterioration (rising lactate, D-dimer elevation, transaminase increase, or acute kidney injury) within hours after IABP withdrawal should prompt urgent visceral ischemia consideration and expedited CTA evaluation, particularly in patients with identified risk factors including low cardiac output, severe SEC, reduced anticoagulation intensity, and prolonged device duration. Early CTA and rapid multidisciplinary decision-making may preserve potentially salvageable time windows in this time-critical complication.

ACKNOWLEDGMENTS

This research did not receive any specific grant from funding agencies. All authors had no conflicts of interest to disclose.

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Please cite this article as: DAI BL, WANG SP, LU HC, CHEN N, YIN WQ. Catastrophic visceral arterial embolization after intra-aortic balloon pump withdrawal: a case report of mechanical thrombus stripping. *J Geriatr Cardiol* 2026; 23(6): 368–372. DOI: 10.26599/1671-5411.2026.06.008

