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Left Ventricular Free-Wall Injury During Emergency Pericardial Drainage After Percutaneous Coronary Intervention-Related Coronary Perforation

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 63-year-old

Final Diagnosis: Cardiac tamponade

Symptoms: Cold extremities, poor responsiveness, and progressive hemodynamic collapse

Clinical Procedure: —

Specialty: Cardiology

Objective: Unusual clinical course

Background: Coronary perforation during percutaneous coronary intervention (PCI) can cause pericardial bleeding and shock. Emergency pericardiocentesis may be lifesaving, but persistent bloody drainage after apparently successful coronary sealing creates a diagnostic trap: clinicians may assume ongoing coronary leakage and miss catheter-related injury. We report a safety lesson from this setting.

Case Report: A 63-year-old woman weighing 37 kg underwent PCI for a severely calcified right coronary artery lesion. Coronary perforation after stenting was sealed with a covered stent. After transfer to the ward, she developed shock and increased apical pericardial effusion. Our treating team performed real-time echocardiography-guided apical pericardiocentesis, draining 200 mL of bloody fluid with transient improvement. Routine catheter-position checks, including agitated saline injection, contrast fluoroscopy through the catheter, pressure-waveform recording, and blood gas or oxygen saturation testing, were not performed or not documented. Drainage remained bloody, with approximately 1000 mL additional output, recurrent hypotension, and a hemoglobin decrease from 102 to 67 g/L. Repeat coronary angiography showed no recurrent extravasation. Fluoroscopy/digital subtraction angiography showed an abnormal catheter trajectory, with the distal tip projected over the aortic region. Computed tomography was not performed because the patient proceeded directly to emergency surgery. Surgical exploration confirmed left ventricular free-wall penetration. The catheter was removed only after pledgeted repair was secured. The patient recovered well.

Conclusions: This case emphasizes a safety message rather than a proven intravascular route. After PCI-related perforation, persistent bloody drainage with shock should prompt immediate drain-position reassessment and controlled removal if intracardiac or intravascular malposition is possible.

Keywords: Iatrogenic Disease • Pericardiocentesis

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Introduction

Coronary artery perforation is an uncommon but serious complication of percutaneous coronary intervention (PCI). In practical terms, it represents disruption of the coronary vessel wall during guidewire, balloon, atherectomy, or stent manipulation. The Ellis classification remains widely used to describe angiographic severity, and large-vessel perforation may rapidly cause pericardial bleeding, hemodynamic collapse, emergency surgery, or death [1,2]. Heavily calcified lesions, aggressive lesion preparation, and high-pressure balloon dilation are recognized clinical contexts in which coronary injury may occur [1-4].

Emergency management usually begins with hemodynamic support, balloon tamponade, anticoagulation reversal when appropriate, and lesion-specific sealing strategies such as covered-stent implantation [3-5]. When pericardial blood accumulates and shock develops, pericardiocentesis may be life-saving. Pericardiocentesis itself, however, can cause serious complications, including cardiac chamber perforation, coronary or vascular injury, pneumothorax, arrhythmia, and catheter malposition [6-11].

This case report has a focused aim: to highlight a safety problem that can occur after coronary perforation appears to have been sealed. Persistent bloody drainage and recurrent shock should not be interpreted automatically as ongoing coronary leakage. Instead, clinicians should promptly reassess the drainage catheter position, use available confirmation methods, and avoid blind removal when intracardiac or intravascular placement is possible.

Case Report

A 63-year-old woman was admitted with exertional chest tightness for more than 1 year, worsening over 2 weeks. Her body weight was 37 kg. She had hypertension but no diabetes, dyslipidemia, smoking or alcohol use, renal dysfunction, bleeding history, previous PCI, or coronary artery bypass grafting. Admission electrocardiography, cardiac troponin, and N-terminal pro-B-type natriuretic peptide were not significantly abnormal. Baseline renal function and coagulation testing were within normal limits.

Coronary angiography showed a normal left main coronary artery, 80% tubular stenosis in the mid-left anterior descending artery, no significant stenosis in the left circumflex artery, and diffuse 90% calcified stenosis extending from the ostium to the mid segment of the right coronary artery. Optical coherence tomography of the proximal-to-mid right coronary artery showed fibrocalcific plaque. PCI of the right coronary artery was undertaken, while staged treatment of the left anterior descending lesion was deferred.

The right coronary artery intervention was performed with guide-extension catheter support. After lesion preparation with a 2.5 × 10 mm Wolverine cutting balloon, 2 Resolute Integrity drug-eluting stents measuring 3.0 × 30 mm and 3.5 × 30 mm were implanted sequentially from the ostium to the mid-right coronary artery. Each stent was deployed at 12 atm. The vascular access route, guiding catheter and guidewire types, heparin dose, protamine use, and activated clotting time were not retrievable from the available records. Repeat angiography showed contrast extravasation from the mid-right coronary artery, consistent with angiographically significant coronary perforation. A 3.0 × 15 mm covered stent was implanted at the perforation site and deployed at 14 atm. Post-dilation was performed with 3.0 × 15 mm and 3.5 × 15 mm non-compliant balloons at 18 atm. Final angiography showed disappearance of the leak, an angiographically satisfactory stent result, and thrombolysis in myocardial infarction (TIMI) grade 3 distal flow (**Figure 1**).

Two bedside echocardiographic examinations after the procedure showed only a small apical pericardial effusion of approximately 3-4 mm. The patient was returned to the ward on aspirin and clopidogrel. Shortly afterward, she developed cold extremities, poor responsiveness, and progressive hemodynamic collapse. Blood pressure and oxygen saturation were difficult to measure, and heart rate increased to 120 beats/min. Our treating team initiated rapid fluid resuscitation and vasopressor support with norepinephrine plus metaraminol. Emergency bedside echocardiography showed increased pericardial effusion, with maximal apical effusion thickness of 13.9 mm. Saved images did not document right-sided chamber collapse, inferior vena cava plethora, or Doppler respiratory variation. Pre- and post-drainage echocardiographic images suitable for publication were not available. The event was therefore regarded as clinically significant pericardial effusion with shock rather than definite echocardiographic tamponade.

Emergency real-time echocardiography-guided bedside pericardiocentesis was performed through an apical approach by the treating cardiology team. The documentation did not state why a subxiphoid approach was not selected. The apical pocket was the site where the effusion was measured, but saved images comparing access windows were unavailable. Approximately 200 mL of bloody fluid was initially drained, and the patient's consciousness and blood pressure transiently improved. The drainage catheter was inserted to a depth of 10 cm. The misplaced device was the pericardial drainage catheter used for the apical pericardiocentesis; its commercial name, manufacturer, and French size could not be retrieved from the record.

Routine safety checks after needle entry and before or immediately after catheter advancement were not performed or not documented. These included agitated saline injection, contrast

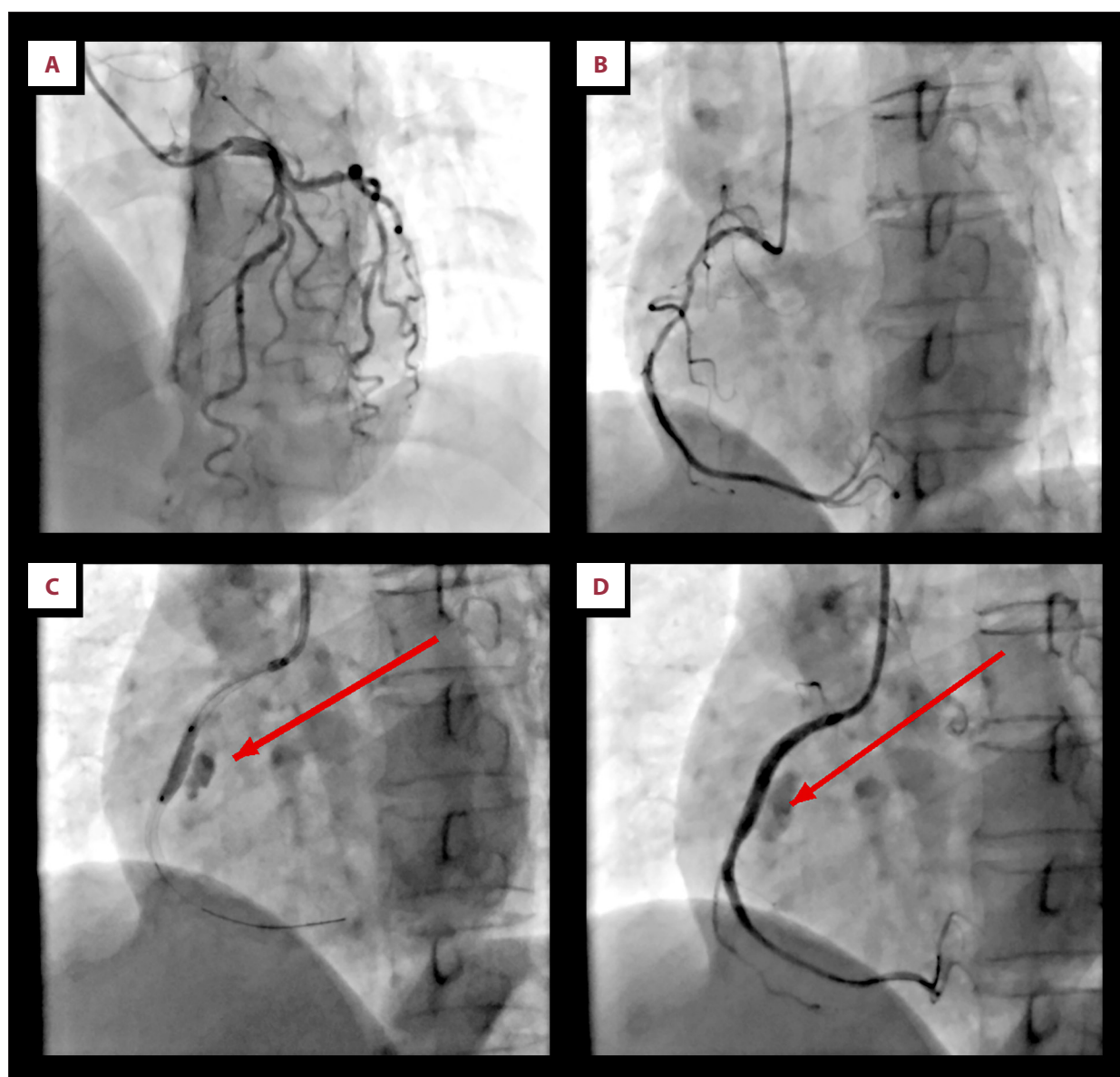


Figure 1. Coronary angiographic findings during percutaneous coronary intervention. (A) Baseline left coronary angiography. (B) Baseline right coronary angiography showing diffuse severe calcified stenosis from the ostium to the mid segment of the right coronary artery. (C) Contrast extravasation from the mid-right coronary artery after stent implantation (arrow). (D) Final right coronary angiography after covered-stent implantation showing no residual contrast leakage and TIMI grade 3 flow (arrow indicates the treated segment). TIMI, thrombolysis in myocardial infarction.

injection through the catheter under fluoroscopy, pressure-waveform recording to exclude entry into a cardiac chamber or great artery, oxygen saturation or blood gas analysis of aspirated blood, and formal documentation of whether the aspirated blood clotted in the basin.

The drainage subsequently remained bloody, with an additional output of approximately 1000 mL. Recurrent hypotension followed, and hemoglobin decreased from 102 g/L before surgical rescue to 67 g/L after pericardial drainage. Persistent

or recurrent coronary bleeding was initially suspected, and the patient was transferred emergently to the catheterization laboratory.

Repeat coronary angiography showed patent coronary flow and no obvious recurrent extravasation from the right coronary artery or other coronary arteries. Fluoroscopy and digital subtraction angiography demonstrated an abnormal catheter trajectory, with the distal tip projected over the aortic region, raising concern for intracardiac or intravascular malposition.

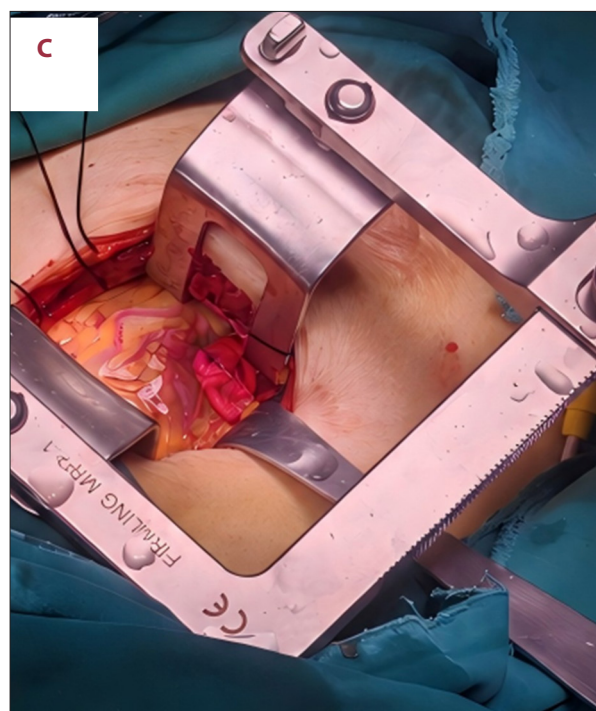
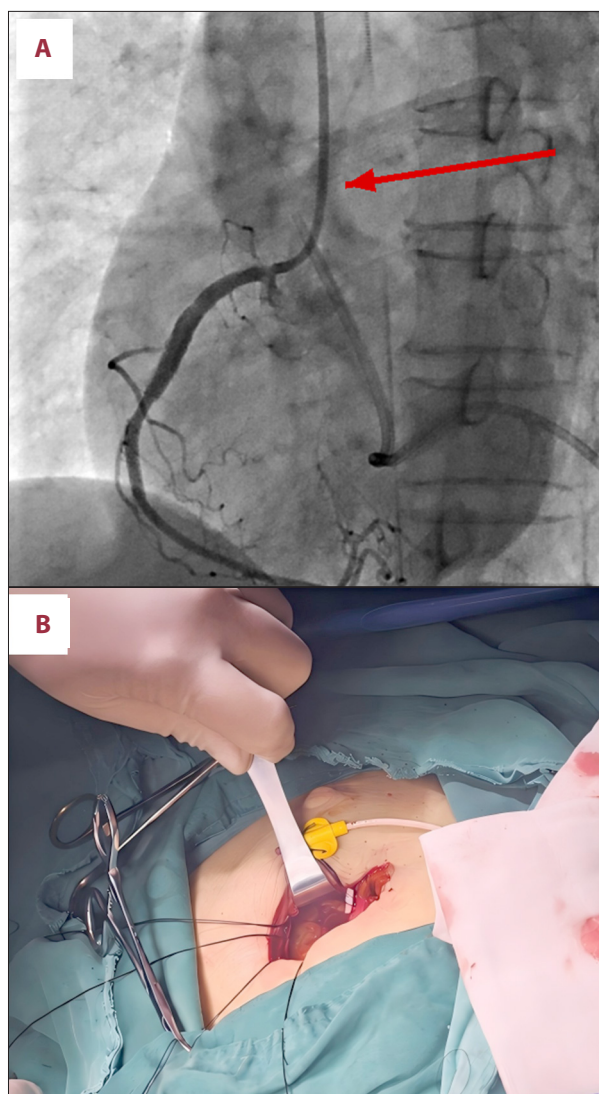


Figure 2. Fluoroscopic suspicion of drainage catheter malposition and surgical repair. **(A)** Fluoroscopy/digital subtraction angiography showing an abnormal catheter trajectory; the arrow marks the distal catheter tip projected over the aortic region. The device shown is the pericardial drainage catheter used for apical pericardiocentesis, although the commercial name and size were not retrievable. The image is interpreted together with the operative finding because catheter contrast injection, pressure-waveform recording, CT, and blood gas testing were not performed. **(B, C)** Surgical exposure and pledgeted repair of the left ventricular puncture site before catheter removal; cardiopulmonary bypass was not required.

No contrast injection through the drainage catheter, arterial pressure tracing, or blood gas analysis from the catheter was available. Computed tomography (CT) was not performed because the patient proceeded directly to emergency surgical exploration. The catheter was left in place and was not removed in the catheterization laboratory.

Emergency surgical exploration was performed through a 5-cm transverse intercostal incision 1 interspace below the puncture site. The catheter was found to penetrate the left ventricular free wall and pass near the free edge of the left lung. There was no large residual pericardial collection at the time of surgery. The pericardium was opened and suspended. A pledgeted 3-0 Prolene mattress suture was placed around the ventricular puncture site before catheter removal. The catheter was then withdrawn under direct vision, and the suture was tightened to achieve hemostasis (**Figure 2**). Cardiopulmonary bypass was not required.

During resuscitation, the patient received 400 mL of red blood cell suspension and 600 mL of plasma. After surgery, no recurrent active bleeding was observed. Because 2 drug-eluting stents and 1 covered stent had just been implanted, aspirin plus clopidogrel was continued after multidisciplinary discussion. She stayed in the intensive care unit for 3 days and was discharged after a total hospital stay of 12 days. PredischARGE transthoracic echocardiography showed a left ventricular ejection fraction of 65%, no pericardial effusion, and preserved valvular function. Written informed consent for publication of the case details and clinical images was obtained from the patient.

A concise timeline of the clinical course is provided in **Table 1**.

Table 1. Timeline of the clinical course.

Time point	Clinical event
Admission	A 63-year-old woman, 37 kg, was admitted for exertional chest tightness worsening over 2 weeks; history included hypertension but no diabetes, dyslipidemia, smoking, alcohol use, renal dysfunction, bleeding history, PCI, or CABG
Initial tests	Admission electrocardiography, troponin, and NT-proBNP were not significantly abnormal; renal function and coagulation tests were within normal limits
Diagnostic angiography	Mid-LAD 80% stenosis; ostial-to-mid RCA diffuse 90% calcified stenosis
RCA PCI	Cutting-balloon preparation followed by 2 drug-eluting stents from the RCA ostium to mid-RCA; vascular access route, guiding catheter and guidewire types, heparin dose, protamine use, and ACT were not retrievable from the records
Coronary perforation	Mid-RCA contrast extravasation after stenting; 3.0 × 15 mm covered stent implanted; final angiography showed no leak and TIMI grade 3 flow
Ward deterioration	Shock with cold extremities and altered responsiveness; blood pressure was unmeasurable and heart rate was 120 beats/min; norepinephrine and metaraminol were used by the treating team
Emergency echocardiography	Increased apical pericardial effusion to 13.9 mm; right-sided chamber collapse, IVC plethora, Doppler respiratory variation, and publishable pre/post-drainage echocardiographic images were not available
Pericardiocentesis	Real-time echocardiography-guided apical puncture drained 200 mL of bloody fluid with transient improvement; catheter depth 10 cm; catheter commercial name and size were not retrievable; agitated saline, contrast injection, pressure waveform, blood gas/oxygen saturation testing, and clotting assessment were not performed or not documented
Recurrent collapse	Additional bloody drainage, approximately 1000 mL; hemoglobin decreased from 102 to 67 g/L
Reassessment	Repeat coronary angiography showed no recurrent coronary leak; fluoroscopy/DSA showed an abnormal catheter trajectory, with the distal tip projected over the aortic region, raising concern for intracardiac or intravascular malposition; CT was not performed before surgery
Surgery	Small intercostal incision; catheter penetration of the left ventricular free wall was confirmed; pledgeted Prolene repair was secured before catheter removal; no cardiopulmonary bypass
Recovery	Red blood cells 400 mL and plasma 600 mL were transfused; ICU stay was 3 days; total hospitalization was 12 days; predischarge echocardiography showed LVEF 65%, no pericardial effusion, and preserved valvular function

ACT, activated clotting time; CABG, coronary artery bypass grafting; CT, computed tomography; DSA, digital subtraction angiography; ICU, intensive care unit; IVC, inferior vena cava; LAD, left anterior descending artery; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PCI, percutaneous coronary intervention; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction.

Discussion

This case should be read primarily as a safety lesson. The initial event, PCI-related right coronary artery perforation, was evident angiographically and was treated with a covered stent. The later combination of recurrent shock, high-volume bloody drainage, and a rapid hemoglobin fall despite the absence of recurrent coronary extravasation indicated that the original explanation was no longer sufficient. In that setting, persistent coronary leakage and a second catheter-related injury needed to be considered side by side.

The mechanism should be stated cautiously. Operative exploration confirmed left ventricular free-wall penetration. Fluoroscopy and digital subtraction angiography raised concern for an abnormal intracardiac or intravascular course because the distal catheter tip projected over the aortic region. However, the exact route was not definitively proven by catheter contrast injection, arterial pressure waveform, CT, blood gas analysis, or oxygen saturation testing. The term suspected intravascular malposition is therefore used as a mechanistic interpretation rather than as a fully proven anatomical diagnosis.

Published reports show that pericardiocentesis-related injury can involve coronary vessels, cardiac chambers, or major

vessels [12-16]. Accordingly, catheter malposition itself is not the sole novelty of this report. The more useful clinical point is diagnostic anchoring: once a coronary perforation has occurred, subsequent bloody drainage may be automatically attributed to persistent coronary bleeding. This assumption can delay recognition of a new injury introduced during emergency drainage.

The indication for pericardiocentesis was based on the clinical emergency rather than on complete saved echocardiographic criteria for tamponade. The patient had shock and increasing pericardial effusion after a documented coronary perforation, and the initial 200 mL of drainage was followed by transient improvement. However, because preserved echocardiographic images did not show classic tamponade physiology, alternative or concurrent contributors to shock - including ongoing bleeding, acute anemia, relative hypovolemia, and vasodilatory effects during resuscitation - cannot be excluded. No CT images were available to supplement this assessment.

Several routine checks can reduce the risk of unrecognized intracardiac or intravascular catheter placement. These include visualization of the needle or guidewire when possible, agitated saline injection under echocardiography, contrast injection through the catheter under fluoroscopy, use of more than 1 fluoroscopic projection, pressure-waveform recording, and assessment of aspirated blood by clotting behavior, oxygen saturation, or blood gas analysis [6-11]. In our case, these checks were not performed or were not documented. This omission is part of the lesson of the report; it is not merely a limitation.

The route of pericardiocentesis also deserves comment. Subxiphoid, apical, and parasternal approaches may be used, and the safest route is generally the shortest path to the largest fluid pocket while avoiding lung, liver, internal mammary vessels, and myocardium [6-8]. The apical route can be appropriate when the largest pocket is apical or lateral, but in a low-body-weight patient with a limited bloody effusion, the margin for error may be narrow. In this case, the reason for not using a subxiphoid approach and the access-window comparison were not retrievable from the record.

The response after suspected malposition is important. PCI complication guidance emphasizes preparation, early recognition, bailout techniques, team communication, and multidisciplinary collaboration in emergency settings [5]. Reports of misplaced pericardiocentesis catheters also show that controlled image-guided or surgical management may be required when a catheter communicates with a vascular structure [12-16]. In the present case, the decision to leave the catheter in place until surgical exposure was achieved was critical, because a catheter traversing the ventricular wall may act as a temporary plug. Blind withdrawal could have converted a partially contained injury into uncontrolled hemorrhage.

A practical response sequence can be proposed. Persistent high-volume bloody drainage, recurrent hypotension, and a rapid fall in hemoglobin, without any recurrent coronary extravasation, should trigger immediate catheter-position reassessment. Continued large-volume aspiration should not proceed reflexively without checking catheter position by available methods, such as echocardiography with agitated saline, fluoroscopy in more than 1 projection, pressure tracing, and blood gas or oxygen saturation analysis. If intracardiac or intravascular placement remains possible, the catheter should be left in situ while controlled surgical or interventional removal is prepared.

This report is limited by incomplete diagnostic documentation at the time of catheter malposition. Operative exploration confirmed left ventricular free-wall penetration, but the full catheter trajectory was not established by contrast injection through the catheter, pressure-waveform recording, blood gas analysis, oxygen saturation testing, CT, or preserved echocardiographic images documenting tamponade physiology and access-window comparison. The commercial name and size of the pericardial drainage catheter were also not retrievable. We therefore describe the distal tip as projected over the aortic region and describe intravascular malposition as suspected rather than definitively proven. These gaps limit reconstruction of the exact sequence and reduce generalizability, although they do not alter the central safety lesson.

Conclusions

After angiographic sealing of PCI-related coronary perforation, recurrent shock and massive bloody drainage following pericardiocentesis should prompt reassessment rather than automatic attribution to persistent coronary leakage. This case supports a practical safety message: confirm drain position when bleeding persists, stop reflexive large-volume aspiration if malposition is possible, and avoid blind removal until surgical or interventional control is available. The report should be interpreted as a cautionary clinical lesson, not as definitive proof of a specific intravascular catheter route.

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Patient Permission/Consent Declaration

Written informed consent for publication of the case details and accompanying clinical images was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Ellis SG, Ajluni S, Arnold AZ, et al. Increased coronary perforation in the new device era. Incidence, classification, management, and outcome. *Circulation*. 1994;90:2725-30
2. Al-Lamee R, Ielasi A, Latib A, et al. Incidence, predictors, management, immediate and long-term outcomes following grade III coronary perforation. *JACC Cardiovasc Interv*. 2011;4:87-95
3. Briguori C, Nishida T, Anzuini A, et al. Emergency polytetrafluoroethylene-covered stent implantation to treat coronary ruptures. *Circulation*. 2000;102:3028-31
4. Ford TJ, Adamson C, McCartney P, et al. Coronary artery perforations: Glasgow Natural History Study of Covered Stent Coronary Interventions (GNOCCI) study. *J Am Heart Assoc*. 2022;11:e024492
5. Ogawa T, Sakakura K, Sumitsuji S, et al. Clinical expert consensus document on bailout algorithms for complications in percutaneous coronary intervention from the Japanese Association of Cardiovascular Intervention and Therapeutics. *Cardiovasc Interv Ther*. 2025;40:1-32
6. Sinnaeve PR, Adriaenssens T. A contemporary look at pericardiocentesis. *Trends Cardiovasc Med*. 2019;29:375-83
7. Tsang TSM, Enriquez-Sarano M, Freeman WK, et al. Consecutive 1127 therapeutic echocardiographically guided pericardiocenteses: Clinical profile, practice patterns, and outcomes spanning 21 years. *Mayo Clin Proc*. 2002;77:429-36
8. Alerhand S, Adrian RJ, Long B, Avila J. Pericardial tamponade: A comprehensive emergency medicine and echocardiography review. *Am J Emerg Med*. 2022;58:159-74
9. Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases. *Eur Heart J*. 2015;36:2921-64
10. Kumar R, Sinha A, Lin MJ, et al. Complications of pericardiocentesis: A clinical synopsis. *Int J Crit Illn Inj Sci*. 2015;5:206-12
11. Ainsworth CD, Salehian O. Echo-guided pericardiocentesis: Let the bubbles show the way. *Circulation*. 2011;123:e210-e11
12. Kanda D, Takumi T, Sonoda T, et al. Coronary artery perforation secondary to lifesaving pericardiocentesis for cardiac tamponade: A case report. *BMC Cardiovasc Disord*. 2021;21:55
13. Patel B, Alampoondi Venkataramanan SV, Bojar R, et al. Acute marginal artery laceration as a complication of pericardiocentesis. *JACC Case Rep*. 2022;4:501-4
14. Tokic T, Burcar I, Gasparovic H. A patient in their 70s with bright red blood in the pericardiocentesis catheter. *JAMA Cardiol*. 2023;8:888
15. Ono Y, Shinohara K, Ishida T, et al. Migration of a pericardial drainage catheter into the right main pulmonary artery. *Acute Med Surg*. 2024;11:e968
16. Derbel H, Krichen M, Zaarour Y, et al. Percutaneous image-guided management of a misplaced pericardiocentesis catheter into the inferior vena cava. *Ther Adv Cardiovasc Dis*. 2024;18:17539447241234655