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Autologous Pericardial Blood Reinfusion as a Bridge to Surgical Repair of Ventricular Perforation During Transcatheter Aortic Valve Implantation: A Case Report of Rescue Measures

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Abstract

Cardiac tamponade due to ventricular perforation is an uncommon but potentially fatal complication of transcatheter aortic valve implantation (TAVI). An 80-year-old man undergoing elective transfemoral TAVI under monitored anesthesia care developed acute hemodynamic collapse immediately after valve deployment, with the guidewire still positioned in the left ventricle. Transthoracic echocardiography demonstrated a moderate-to-large pericardial effusion. Emergency management included tracheal intubation, fluids and vasopressor support, pericardiocentesis, and direct reinfusion of aspirated pericardial blood through femoral venous access. Repeated reinfusion was associated with transient hemodynamic stabilization before emergency surgical repair of a lateral ventricular wall perforation. Rotational thromboelastometry (ROTEM sigma; Tem Innovations GmbH, Munich, Germany) obtained at the start of surgical repair of the ventricular perforation, after reinfusion, showed largely preserved clot formation. The patient was extubated approximately eight hours after surgery but subsequently developed severe respiratory failure requiring further ventilatory support; invasive life support was eventually withdrawn after multidisciplinary assessment. This case illustrates the potential role of direct pericardial blood reinfusion as a bridge to definitive hemostasis. Because reinfusion was performed alongside pericardial drainage, fluid administration, and vasopressor support, its specific contribution to the patient's hemodynamic stabilization cannot be determined.

Categories: Cardiology, Cardiac/Thoracic/Vascular Surgery, Anesthesiology
Keywords: autologous blood reinfusion, cardiac tamponade, case report, rotational thromboelastometry, transcatheter aortic valve implantation, ventricular perforation

Introduction

Transcatheter aortic valve implantation (TAVI) is an established treatment for severe symptomatic aortic stenosis, but rare procedural complications remain life-threatening. In a single-center registry of 1,247 TAVI procedures, cardiac tamponade occurred in 1.7%, left ventricular (LV) perforation caused nearly half of these cases, and in-hospital mortality among patients with tamponade was 43% [1]. Guidewire-related LV perforation can cause rapidly progressive hemopericardium and circulatory collapse and has been associated with guidewire manipulation, delivery system movement, and unfavorable cardiovascular anatomy [2-5]. When percutaneous treatment is not feasible, emergent cardiac surgery (ECS) is required; in a large European registry, this occurred in 0.76% of transfemoral TAVI procedures, with an in-hospital mortality of 46.0% among these patients [6].

While definitive hemostasis is arranged, maintaining systemic perfusion despite ongoing pericardial bleeding is a major challenge. Direct reinfusion of aspirated pericardial blood provides immediately available autologous volume and has been described in iatrogenic hemorrhagic tamponade during percutaneous cardiac procedures [7,8], but its hemodynamic and hemostatic effects remain incompletely characterized. Similar reinfusion of unprocessed autologous blood has also been described in other settings, such as traumatic hemothorax and ruptured ectopic pregnancy [9-11].

We report a case requiring pericardial drainage, repeated autologous blood reinfusion, and emergency surgical repair, focusing on the role and limitations of reinfusion as temporary circulatory support and its potential effect on coagulation.

Case Presentation

An 80-year-old man with severe symptomatic aortic stenosis was scheduled for elective transfemoral TAVI following Heart Team assessment. He had a medical history of hypertension, dyslipidemia, heart failure (New York Heart Association class II) [12], and dysphagia under investigation. He was classified as American Society of Anesthesiologists physical status IV [13]. Preprocedural transthoracic echocardiography and CT

angiography indicated suitability for the planned intervention (Figure 1).

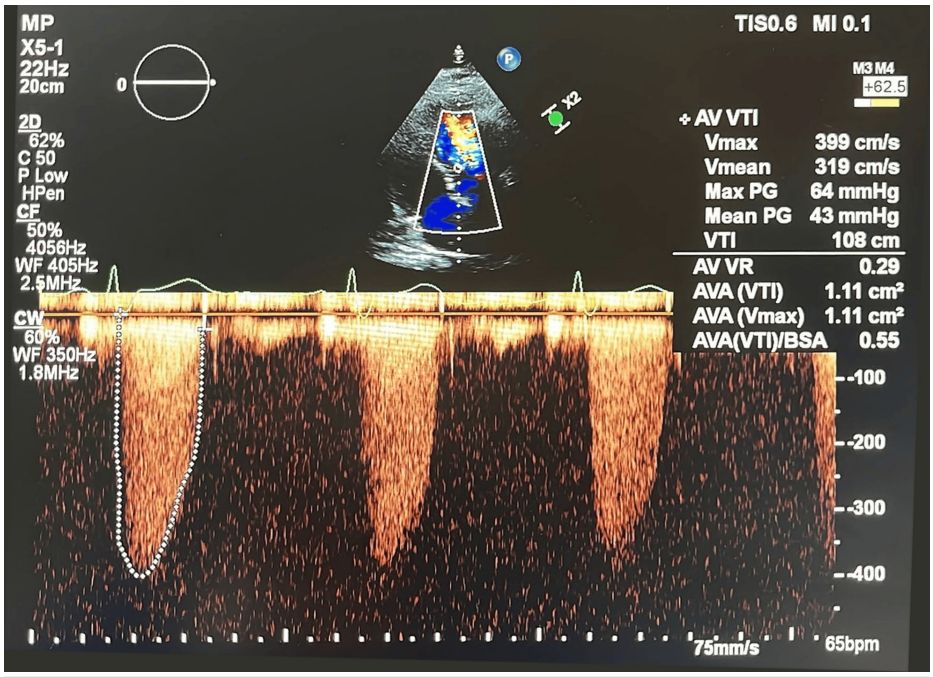


FIGURE 1: Preprocedural continuous-wave Doppler of the aortic valve.

Peak velocity 3.99 m/s, mean gradient 43 mmHg, aortic valve area 1.11 cm² (indexed 0.55 cm²/m²), and Doppler velocity index 0.29, consistent with severe aortic stenosis.

The procedure was performed in the cardiac catheterization laboratory under monitored anesthesia care, with initial doses of fentanyl 50 µg and midazolam 1 mg, followed by additional doses titrated as required throughout the procedure. Antibiotic prophylaxis with cefazolin 2 g and gentamicin 5 mg/kg and unfractionated heparin 100 IU/kg were administered. Femoral arterial access was established for the intervention and aortic root angiography, and femoral venous access was available for temporary ventricular pacing. A self-expanding transcatheter aortic valve was implanted via the transfemoral route.

Immediately after deployment of the aortic prosthesis, with the flexible LV guidewire still positioned within the ventricular cavity (Figure 2), the patient developed sudden hemodynamic instability with agitation and confusion. Transthoracic echocardiography demonstrated a moderate-to-large pericardial effusion (Figure 3), supporting the diagnosis of acute cardiac tamponade.

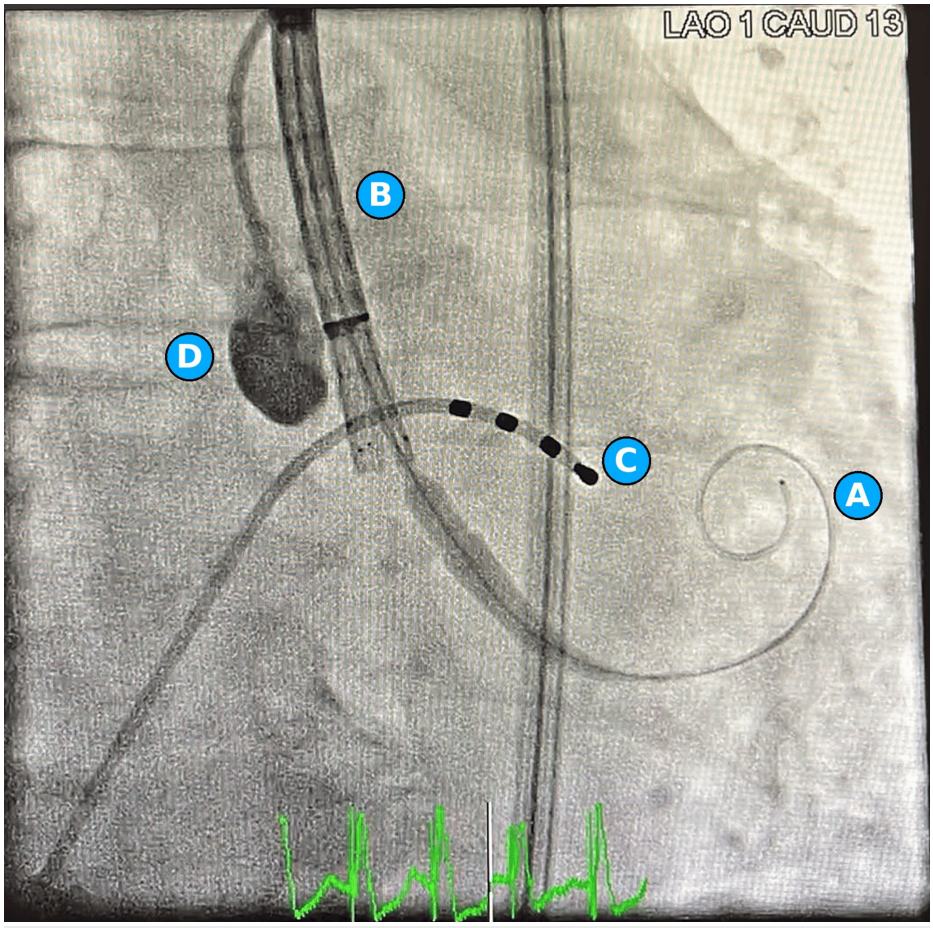


FIGURE 2: Fluoroscopy during TAVI.

Left anterior oblique 1°, caudal 13° projection before valve release. (A) Preshaped guidewire looped within the LV cavity. (B) Delivery system with the undeployed self-expanding aortic valve prosthesis. (C) Temporary pacing catheter. (D) Contrast.

LV: left ventricular; TAVI: transcatheter aortic valve implantation

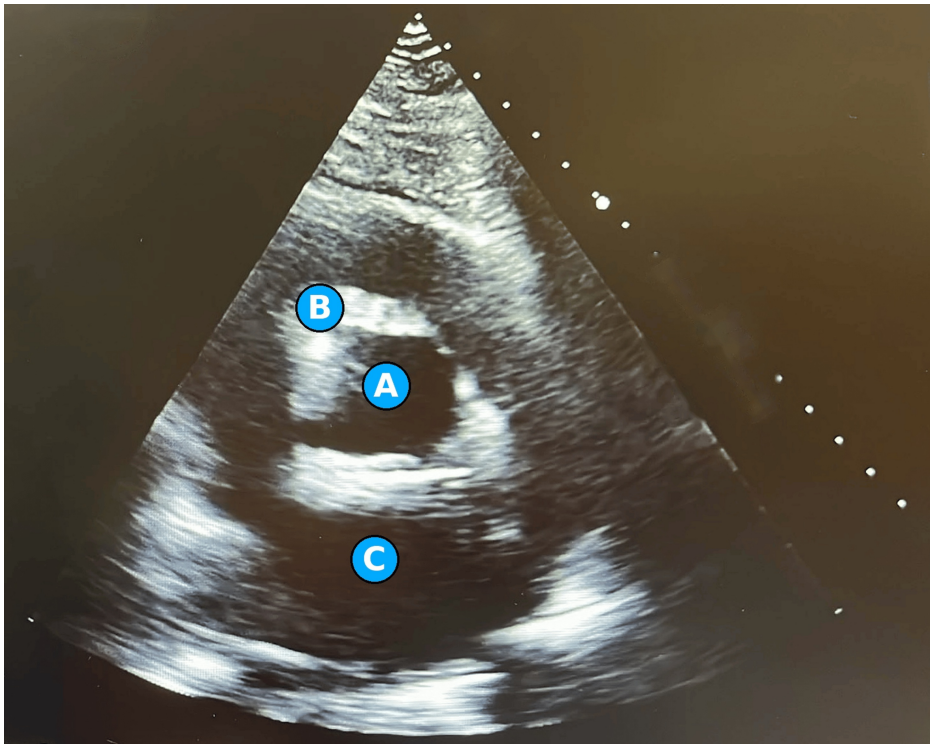


FIGURE 3: Transthoracic echocardiography during hemodynamic collapse.

Short-axis view showing (A) the LV cavity, (B) the LV myocardium, and (C) a moderate-to-large free pericardial effusion.

LV: left ventricular

The patient underwent tracheal intubation after induction with fentanyl 2 µg/kg, propofol 0.7 mg/kg, and rocuronium 1.2 mg/kg, using a 7.5 mm endotracheal tube, followed by invasive mechanical ventilation with a lung-protective strategy in volume-controlled mode (tidal volume 8 mL/kg predicted body weight, positive end-expiratory pressure 5 cmH_2O, fraction of inspired oxygen 0.5, respiratory rate 14 breaths/min). Circulatory support included crystalloid resuscitation (approximately 15-20 mL/kg) and a noradrenaline infusion titrated up to a maximum of 1.1 µg/kg/min. Emergency pericardiocentesis with drain placement yielded approximately 1500 mL of blood on initial drainage. Under aseptic conditions, the aspirated pericardial blood was used for direct reinfusion: it was manually drawn into syringes and reinjected through femoral venous access without filtration, in aliquots of approximately 50 mL over three minutes. After the initial pericardial aspiration and reinfusion, heparin was reversed with protamine, and tranexamic acid 1 g was administered.

Contrast-enhanced CT was performed to localize the bleeding source and assess the feasibility of percutaneous repair. Imaging demonstrated a defect in the lateral LV wall, considered likely to be guidewire-related (Figure *4*). Following multidisciplinary review of the imaging findings and clinical course by the anesthesiology, interventional cardiology, cardiac imaging, and cardiac surgery teams, emergency surgical repair was decided. Throughout transport to CT, image acquisition, multidisciplinary decision-making, and transfer to the operating room, recurrent hemodynamic instability was managed with repeated reinfusion of pericardial blood; each 50-100 mL reinfusion was associated with a transient increase in mean arterial pressure of approximately 15-20 mmHg. In total, including the initial reinfusion, approximately 550 mL of pericardial blood was reinfused before surgery.

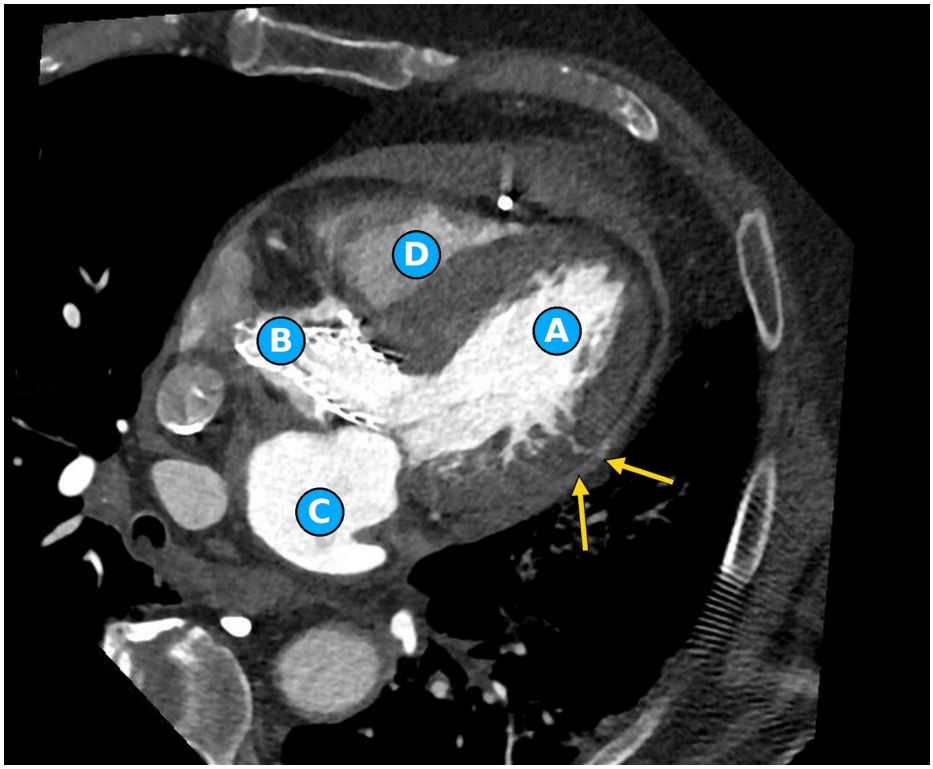


FIGURE 4: Contrast-enhanced CT, arterial phase.

Axial (transverse) arterial-phase image showing (A) the LV, (B) the transcatheter aortic valve prosthesis, (C) the left atrium, and (D) the RV. Yellow arrows indicate the site of the lateral LV wall perforation.

LV: left ventricular/ventricle; RV: right ventricle

Rotational thromboelastometry (ROTEM; ROTEM sigma, Tem Innovations GmbH, Munich, Germany) performed in the operating room at the start of surgical ventricular repair, approximately one hour after the onset of the complication and following reinfusion of approximately 550 mL of autologous pericardial blood, showed largely preserved clot formation (Figure *5*). No fibrinogen concentrate, plasma, or platelets had been administered before sampling. Extrinsically activated thromboelastometry (EXTEM; tissue factor-activated) clotting time and maximum clot firmness (MCF) were 61 seconds and 53 mm, respectively, and fibrin-based thromboelastometry (FIBTEM; extrinsically activated with platelet inhibition by cytochalasin D, reflecting the fibrinogen contribution to clot firmness) MCF was 20 mm, all within reference ranges. Intrinsically activated thromboelastometry (INTEM; contact-activated) and heparinase-modified INTEM (HEPTEM; which neutralizes the heparin effect) clotting times were 220 seconds and 228 seconds, respectively, both within reference ranges and without a meaningful difference between assays, consistent with reversal of procedural heparin. INTEM and HEPTEM MCF were marginally below the lower reference limit (48 mm and 47 mm, respectively; reference 50-72 mm).

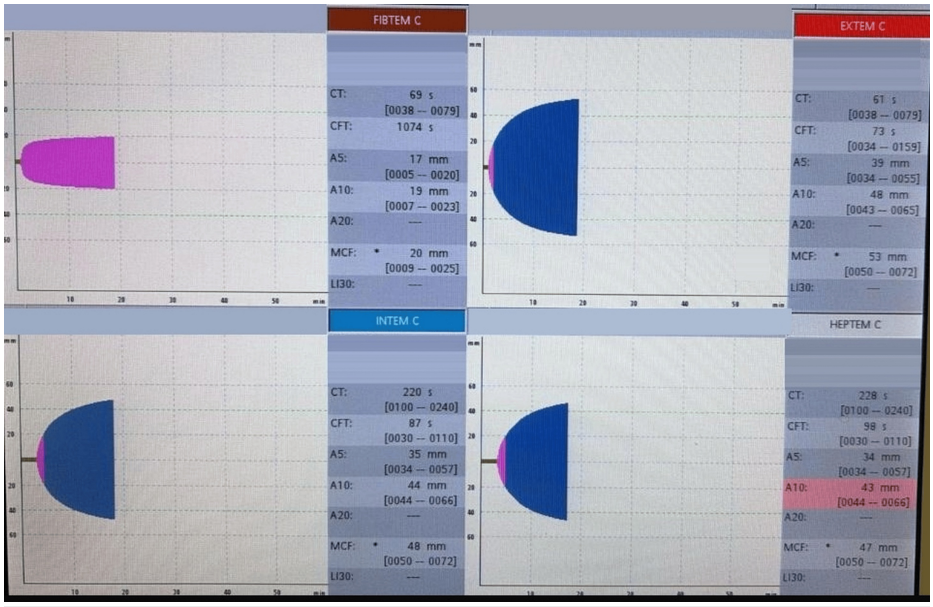


FIGURE 5: ROTEM at the start of surgical ventricular repair.

Obtained in the operating room approximately one hour after the onset of the complication and following reinfusion of approximately 550 mL of autologous pericardial blood. FIBTEM (upper left): MCF 20 mm; EXTEM (upper right): CT 61 seconds, MCF 53 mm; INTEM (lower left): CT 220 seconds, MCF 48 mm; HEPTEM (lower right): CT 228 seconds, MCF 47 mm. Reference ranges are shown in brackets beside each value.

A5, A10, A20: clot amplitude at five, 10, and 20 minutes after CT; CFT: clot formation time; CT: clotting time; EXTEM: extrinsically activated thromboelastometry; FIBTEM: fibrin-based thromboelastometry (extrinsic activation with platelet inhibition); HEPTEM: heparinase-modified INTEM; INTEM: intrinsically activated thromboelastometry; LI30: lysis index at 30 minutes after CT; MCF: maximum clot firmness; ROTEM: rotational thromboelastometry

Median sternotomy revealed hemopericardium with substantial intrapericardial clot burden. A bleeding site on the lateral LV wall was identified and closed with a hemostatic suture. The repair was performed without cardiopulmonary bypass. Intraoperative transesophageal echocardiography showed a normally functioning aortic prosthesis, and cell salvage recovered 300 mL of blood. Complete atrioventricular block required temporary epicardial pacing, and a permanent pacemaker was implanted on postoperative day 2. The patient was transferred to the intensive care unit intubated and sedated and was extubated approximately eight hours after surgery without immediate complications.

Over the following days, he developed severe respiratory failure associated with probable aspiration pneumonia in the context of dysphagia, ventilatory muscle fatigue, and inadequate pain control; after failure of a noninvasive ventilation trial, he was reintubated on postoperative day 3. On the same day, he had a pulseless ventricular tachycardia cardiac arrest, with return of spontaneous circulation after one cycle of advanced life support, and subsequently developed septic shock with multiorgan dysfunction. Following repeated weaning failure and multidisciplinary assessment of the expected benefit of ongoing treatment, invasive organ support was withdrawn on postoperative day 12, and care was focused on comfort. He died on postoperative day 14. The clinical course is summarized in Figure *6*.

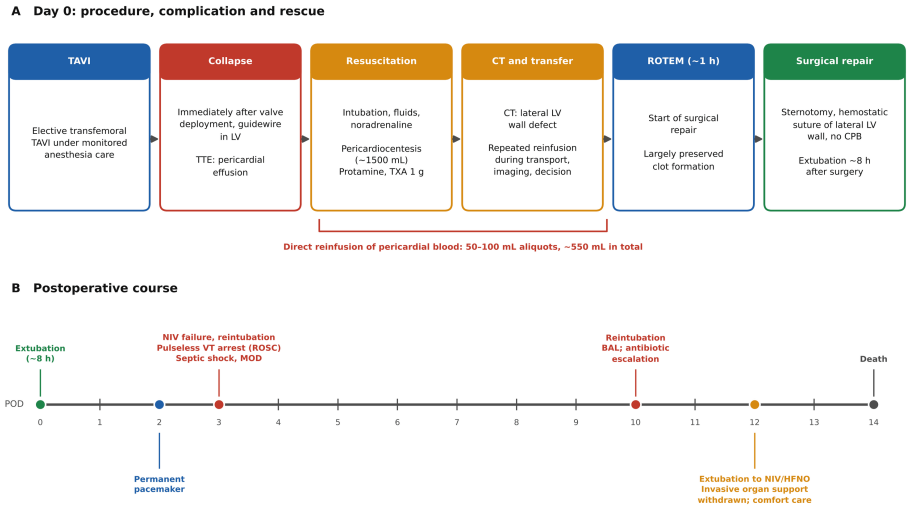


FIGURE 6: Timeline of clinical events.

(A) Sequence of events on the day of the procedure, from TAVI to surgical repair; the red bracket indicates the period of direct pericardial blood reinfusion. (B) Postoperative course.

BAL: bronchoalveolar lavage; CPB: cardiopulmonary bypass; HFNO: high-flow nasal oxygen; LV: left ventricle; MOD: multiorgan dysfunction; NIV: noninvasive ventilation; POD: postoperative day; ROSC: return of spontaneous circulation; ROTEM: rotational thromboelastometry; TAVI: transcatheter aortic valve implantation; TTE: transthoracic echocardiography; TXA: tranexamic acid; VT: ventricular tachycardia

Discussion

This case illustrates the challenge of maintaining systemic perfusion during ongoing pericardial hemorrhage while definitive surgical hemostasis is arranged. The onset of deterioration immediately after valve deployment, with the guidewire still in the LV, and the operative finding of a lateral LV wall perforation support a guidewire-related mechanism. However, the precise mechanism should be distinguished from the anatomically confirmed injury. Published cases describe contributions from guidewire configuration, ventricular geometry, and movement of the delivery system along the aortic access route [2-5]. These observations support careful review of procedural imaging without establishing that a particular anatomical risk factor was present in this patient.

The site of cardiac injury may influence prognosis. A single-center registry identified LV guidewire- or catheter-related injury as the most frequent mechanism among its post-TAVI tamponade cases [1]. In a separate heterogeneous series of iatrogenic cardiac and vascular perforations, in-hospital mortality was 75.0% among eight patients with LV laceration and 11.5% among 26 with right ventricular (RV) injury [14]. The small subgroups and differing injury mechanisms limit direct comparison and prevent these figures from being interpreted as an individualized risk estimate.

Outcomes after ECS also depend on the underlying complication and case mix. In a single-center cohort of 1,594 TAVI procedures performed over 13 years, 16 patients (1.0%) required ECS, mainly for valve embolization (n = 9), annular rupture (n = 3), and LV or RV perforation by a guidewire or pacemaker lead (n = 3); 30-day and one-year mortality were 18.8% and 31.3%, respectively [15]. In the larger European Registry on Emergent Cardiac Surgery during TAVI, an investigator-initiated multicenter observational registry, ECS was required in 0.76% of 27,760 procedures, with LV guidewire perforation and annular rupture accounting for approximately half of the indications; in-hospital mortality among patients requiring ECS was 46.0% overall and 50.8% after LV guidewire perforation, and death usually occurred early (median 0 days after ECS), with 52.0% of deaths occurring on the day of surgery [6]. Causes of death were not reported separately, and respiratory complications were not specifically described. These population-level findings therefore contextualize the severity of surgical rescue but do not explain the late respiratory deterioration in this individual patient.

Direct reinfusion of aspirated pericardial blood offers immediately available autologous volume during ongoing hemorrhage. Fiocca et al. described its use in 30 patients with iatrogenic hemorrhagic tamponade, including as a bridge to surgery, and reported no major adverse reactions directly attributable to reinfusion [7]. In the present case, repeated reinfusion accompanied transient hemodynamic stabilization before definitive repair. Nevertheless, concurrent pericardial decompression, fluid administration, and vasopressor therapy preclude attribution of the response to reinfusion alone. The technique should therefore be

described as an adjunct to resuscitation rather than as an independently proven survival intervention.

Direct reinfusion must also be distinguished from processed intraoperative cell salvage. Potential risks of unprocessed blood include mechanical hemolysis, coagulation disturbances, thromboembolism, contamination, and exposure to activated coagulation components or inflammatory mediators [8]. Available observational data are reassuring but cannot exclude uncommon complications, particularly during large-volume reinfusion. Direct reinfusion of unprocessed pericardial blood may also trigger a systemic inflammatory response, potentially mediated by activated inflammatory mediators and damage-associated molecular patterns, and acute respiratory distress syndrome with disseminated intravascular coagulation has been reported after its use [16]. In the present patient, postoperative respiratory failure had other plausible contributors, including probable aspiration pneumonia in the context of dysphagia, but a contribution of reinfusion-related inflammation cannot be excluded. Findings from electrophysiology procedures may not fully apply to ventricular hemorrhage during TAVI [8]. Temporary hemodynamic improvement should not delay definitive hemorrhage control.

Reinfusion of unprocessed autologous blood has also been described outside cardiac procedures. In trauma, blood shed into the pleural cavity was shown to have markedly reduced fibrinogen (<50 vs. 288 mg/dL) and platelet counts compared with venous blood, indicating that it is not equivalent to fresh whole blood [9]. Nevertheless, in a propensity-matched multicenter analysis of 272 trauma patients, reinfusion of unwashed hemothorax blood reduced allogeneic red blood cell and platelet requirements without increasing complications, mortality, or coagulopathy [10]. Similarly, in ruptured ectopic pregnancy, gauze-filtered but unwashed peritoneal blood has been reinfused during laparotomy without embolic, coagulopathic, or infectious complications in a prospective series of 42 patients [11]. Although derived from different clinical settings and largely observational, these data are consistent with the present observation and support the potential role of unprocessed autologous blood as a temporizing measure when allogeneic blood is not immediately available, while highlighting its depleted hemostatic content.

Viscoelastic testing, including thromboelastography and ROTEM, allows rapid point-of-care assessment of whole-blood hemostasis and is used to guide targeted blood component therapy; in adults undergoing cardiac surgery, viscoelastic-guided strategies have been associated with reduced blood loss and transfusion requirements [17]. It may therefore be particularly useful after autologous blood reinfusion, cell salvage, or massive resuscitation. In the present case, ROTEM obtained at the start of surgical ventricular repair, after reinfusion of approximately 550 mL of autologous pericardial blood, showed preserved fibrinogen-dependent clot firmness (FIBTEM MCF 20 mm) and normal EXTEM clotting time and clot firmness, with only marginal reductions in INTEM and HEPTEM MCF. These findings do not suggest a clinically significant reinfusion-associated coagulopathy at that time point, which is relevant given the concern that unprocessed pericardial blood, exposed to the fibrinolytic activity of the pericardium, might impair hemostasis after reinfusion [8]. However, tranexamic acid administered before sampling may have attenuated any fibrinolytic contribution from the reinfused blood, partially confounding this interpretation. Conversely, because no fibrinogen concentrate, plasma, or platelets had been administered before sampling, preserved fibrinogen-dependent clot firmness cannot be attributed to hemostatic replacement. More broadly, this observation must be interpreted with caution: ROTEM was obtained at a single time point, and no preprocedural viscoelastic assessment was available for comparison. The hemostatic effect of reinfusion is likely to depend on several factors, including the volume reinfused, preexisting coagulation abnormalities, concurrent fluid administration, antifibrinolytic therapy, and anticoagulation and its reversal. ROTEM also does not assess platelet function and cannot exclude other reinfusion-related complications, such as hemolysis. As a single-case observation, these findings cannot be generalized, and further studies with serial coagulation and viscoelastic monitoring are needed to define the hemostatic impact of direct pericardial blood reinfusion.

This case also highlights the challenges of managing a life-threatening complication in a non-operating room anesthesia setting. During an initially planned procedure under monitored anesthesia care, abrupt cardiovascular collapse may necessitate immediate airway control, invasive ventilation, and simultaneous hemodynamic resuscitation. In the cardiac catheterization laboratory, imaging equipment, procedural drapes, and room configuration may restrict access to the patient and complicate the deployment of additional personnel and equipment. Preparedness therefore requires readily accessible airway and resuscitation devices, clearly defined team responsibilities, and established pathways for urgent surgical escalation. In this case, the need for CT assessment introduced an additional challenge: maintaining circulatory support during transport and imaging while determining the feasibility of percutaneous repair. The experience underscores the importance of anticipating conversion to general anesthesia and coordinating diagnostic assessment with ongoing resuscitation and timely access to definitive hemorrhage control.

The postoperative course further illustrates that correction of the structural injury does not ensure overall recovery. Respiratory deterioration should be interpreted in the context of the documented pulmonary complications, ventilatory course, pain control, and baseline vulnerability. This single case cannot establish a survival benefit from reinfusion or determine whether it contributed to subsequent organ dysfunction. Its educational value lies in describing the sequence of rescue measures and the clinical decisions connecting temporary stabilization with definitive repair.

Conclusions

This case illustrates the need for prompt echocardiographic assessment and coordinated multidisciplinary management of acute tamponade during TAVI. Direct autologous reinfusion of pericardial blood may provide temporary circulatory support while definitive surgical hemostasis is arranged, but its independent contribution to outcome cannot be established from a single case. Successful repair should be distinguished from overall clinical recovery, particularly in older patients with a complicated postoperative course.

Sudden hemodynamic deterioration during TAVI warrants prompt assessment for cardiac tamponade. Transient stabilization with reinfusion does not replace definitive hemorrhage control, and successful repair does not guarantee postoperative recovery. In this patient, viscoelastic testing after reinfusion did not suggest a clinically significant coagulopathy, but a single observation cannot establish the hemostatic safety of the technique, which warrants further study.

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: Mariana Flor de Lima

Acquisition, analysis, or interpretation of data: Mariana Flor de Lima, Ana Fonte Boa

Drafting of the manuscript: Mariana Flor de Lima, Ana Fonte Boa

Critical review of the manuscript for important intellectual content: Mariana Flor de Lima, Ana Fonte Boa

Supervision: Ana Fonte Boa

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.

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