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Tricuspid Chordae Tendineae Rupture Detected After Repair of a Delayed Traumatic Left Ventricular Aneurysm: A Case Report

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Conflict of interest:

None declared

Patient:

Male, 20-year-old

Final Diagnosis:

Left ventricular aneurysm rupture • tricuspid chordae tendineae rupture

Symptoms:

Loss of consciousness

Clinical Procedure:

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Specialty:

Cardiology

Objective:

Rare disease

Background:

Delayed traumatic cardiac rupture complicated by secondary tricuspid chordae tendineae rupture is an exceedingly rare clinical entity. We report this case to describe the temporal sequence of events from blunt myocardial contusion to left ventricular aneurysm formation and rupture, followed by secondary tricuspid chordae tendineae rupture. While the precise mechanism linking these events remains to be determined, we propose a potential pathophysiological connection.

Case Report:

A patient presented with rupture of a posterior left ventricular aneurysm 54 days after a fall from a height. The patient was emergently placed on venoarterial extracorporeal membrane oxygenation (VA-ECMO) for circulatory support and subsequently underwent surgical repair of the aneurysm. Postoperative echocardiography revealed new-onset tricuspid chordae tendineae rupture, prompting elective mechanical tricuspid replacement. Histopathological examination of resected valve tissues revealed chronic degenerative changes. To our knowledge, this is the first report documenting this temporal sequence of delayed left ventricular aneurysm rupture followed by tricuspid chordal rupture after surgical repair.

Conclusions:

This case highlights the importance of continued clinical and echocardiographic vigilance after severe blunt cardiac injury, particularly for detecting late-emerging structural complications such as aneurysm formation and valvular dysfunction. It also illustrates the potential utility of VA-ECMO as a critical bridging strategy to stabilize patients with traumatic cardiac rupture before surgical repair. The observed temporal association between aneurysm repair and subsequent tricuspid chordal rupture is noted, though the exact causal relationship and underlying mechanism remain uncertain.

Keywords:

Extracorporeal Membrane Oxygenation • Heart Aneurysm • Heart Injuries • Heart Rupture • Tricuspid Valve Prolapse

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Introduction

Most patients with cardiac rupture resulting from blunt chest trauma die prior to hospital admission, and the incidence of this injury among admitted survivors is < 0.5% [1]. Delayed cardiac rupture, most commonly secondary to aneurysmal rupture, is associated with a high mortality rate [2]. Traumatic valvular chordae tendineae rupture is typically an isolated injury that manifests as severe acute valvular regurgitation. It is most often caused by direct blunt chest force, with rare reported cases of iatrogenic injury during cardiac surgery [3,4].

Herein, we present a case of delayed left ventricular aneurysm rupture following a fall from a height, further complicated by tricuspid chordae tendineae rupture detected after aneurysm repair. The patient was successfully managed with venoarterial extracorporeal membrane oxygenation (VA-ECMO) support, surgical cardiac rupture repair, and tricuspid valve replacement. Delayed rupture of a left ventricular aneurysm resulting from blunt chest trauma and traumatic tricuspid chordae tendineae rupture have previously been reported as distinct complications in case series [4,5]; however, the novelty of this case lies in documenting a unique temporal association: the detection of tricuspid chordal rupture following surgical repair of a delayed traumatic left ventricular aneurysm. While the co-occurrence of these 2 injuries is extremely rare, the exact mechanism linking the aneurysm repair to the chordal rupture remains uncertain. This report primarily aims to highlight the necessity of dynamic monitoring for late complications after severe blunt cardiac injury and to discuss the rational use of VA-ECMO for managing similar cases.

Case Report

A 20-year-old man presented with a 6-hour history of sudden loss of consciousness. Fifty-four days prior, he had sustained a fall from approximately 10 m, which led to multiple fractures, right hemopneumothorax, bilateral pulmonary contusions, and hemorrhagic shock. During the initial hospital stay, his high-sensitivity troponin I level peaked at 2147.9 pg/mL and normalized on post-injury day 17. Electrocardiography showed sinus tachycardia. Transthoracic echocardiography (TTE) revealed mild tricuspid regurgitation (regurgitant jet area 3.8 cm²), without evidence of abnormalities in cardiac chamber size, valve structure, or contractility. Chest computed tomography demonstrated normal morphology of the great vessels, with no significant abnormalities in the cardiac chambers or pericardium. After comprehensive supportive care, his condition improved, and he was discharged 1 month after admission.

Six hours prior to readmission, he experienced sudden loss of consciousness during limb rehabilitation at another hospital,

accompanied by tachypnea and limb convulsions. His vital signs were as follows: blood pressure, 51/39 mm Hg; heart rate, 130 beats/min; oxygen saturation by pulse oximetry, unmeasurable. Emergency measures, including mechanical ventilation, fluid resuscitation, and vasopressor administration, were initiated, and the patient was urgently transferred to our intensive care unit (ICU). On arrival, his heart rate was 148 beats/min and blood pressure 75/50 mm Hg while receiving norepinephrine (1.0 µg/kg/min) and dopamine (12 µg/kg/min). Bedside TTE revealed a 0.7-cm discontinuity in the left ventricular posterior myocardium, accompanied by large pericardial effusion, and no significant structural valvular abnormalities were detected (**Figure 1A**).

The patient was diagnosed with cardiac rupture and tamponade. After consultation with cardiac surgeons and after written informed consent was obtained from the patient's family, VA-ECMO was initiated at an initial flow rate of 3 L/min without anticoagulation, and emergency thoracotomy was subsequently performed. A median sternotomy was performed without cardiopulmonary bypass. Exploration revealed approximately 300 ml of blood in the pericardial cavity. A saccular thin-walled outpouching measuring approximately 4 × 3 × 2 cm was observed on the posterior wall of the left ventricle, consistent with a ventricular aneurysm, which exhibited a small, localized rupture site (**Figure 1B**). During the operation, a double purse-string suture was first placed using 4-0 Prolene with pledgets along the normal myocardium at the edge of the ventricular aneurysm. After gradual tightening of the purse-string, the aneurysm was reinforced and closed using interrupted and continuous 4-0 Prolene sutures with pledgets. Postoperatively, the target mean arterial pressure was 65 to 70 mm Hg, ECMO blood flow was maintained at 2.0 to 2.5 L/min, and vasoactive drugs were gradually tapered. On postoperative day 1, laboratory tests demonstrated markedly elevated liver enzyme levels (alanine aminotransferase 2066 U/L; aspartate aminotransferase 948 U/L), and a TTE was performed, revealing severe tricuspid regurgitation (regurgitant jet area 11.4 cm²). Subsequently, transesophageal echocardiography (TEE) confirmed rupture of the chordae tendineae and prolapse of the anterior tricuspid leaflet, and revealed a dilated right atrium, with preserved right ventricular size and contractility (**Figure 2A**).

After consultation with cardiac surgeons, valve surgery was recommended pending improvement in liver function. On day 3, the patient's hemodynamic status stabilized, and he was successfully weaned off ECMO. On day 6, with improved liver function, a second thoracotomy was performed with cardiopulmonary bypass support. Direct intraoperative inspection revealed significant dilation of the tricuspid valve annulus, rupture of the anterior leaflet chordae tendineae, and severe valvular regurgitation. In general, native tricuspid valve repair may be preferable when technically feasible, as it avoids

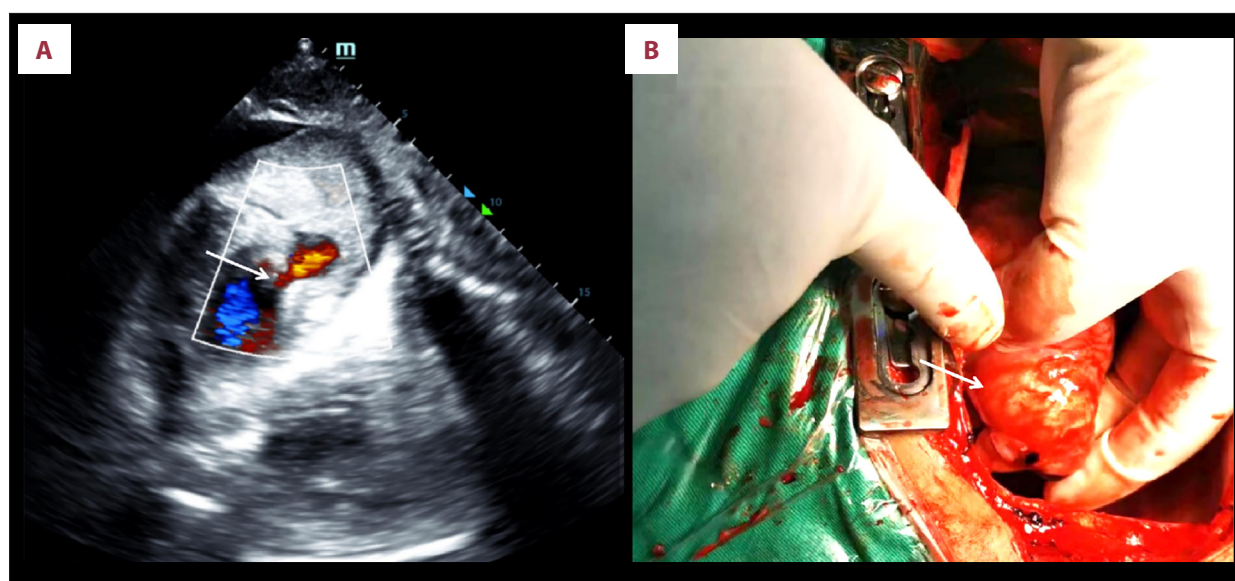


Figure 1. Echocardiography and surgical findings of cardiac injury. (A) Transthoracic echocardiography, subxiphoid view, shows a 0.7-cm discontinuity of the posterior left ventricular wall myocardium (arrow) with massive hemopericardium. (B) Emergency thoracotomy reveals a left ventricular posterior wall aneurysm (measuring 4 × 3 × 2 cm, arrow).

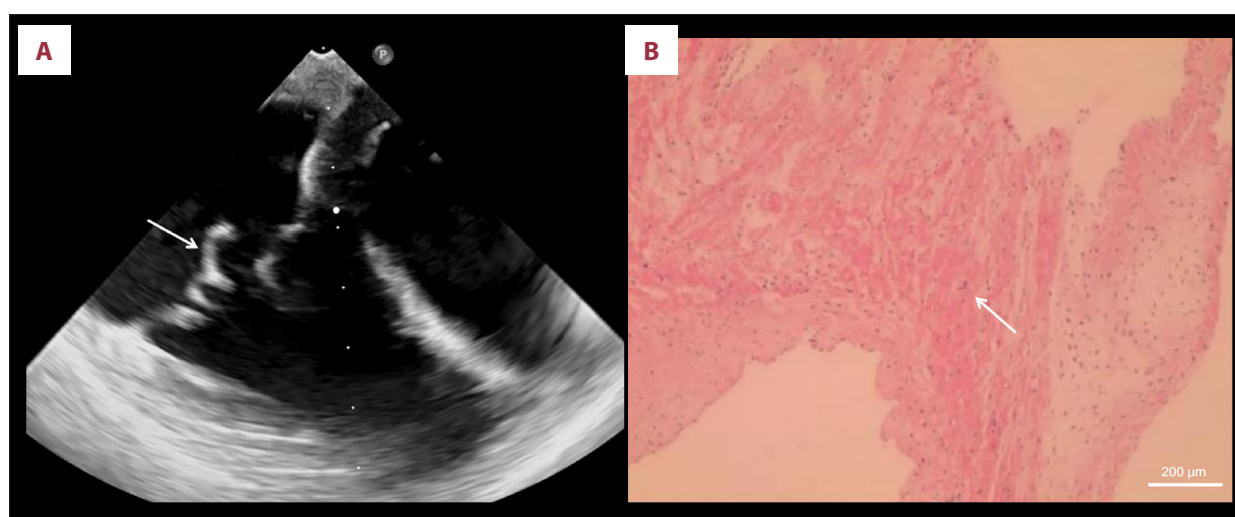


Figure 2. Transesophageal echocardiographic 4-chamber view and histopathological micrograph of the resected tricuspid leaflet (hematoxylin and eosin stain, original 40 × magnification). (A) Chordae tendineae rupture of anterior tricuspid leaflet resulting in prolapse (arrow). (B) Fibrous tissue hyperplasia of tricuspid leaflet with collagenization and hyaline degeneration (arrow; scale bar = 200 μm).

prosthesis-related considerations. In the present case, intraoperative valvuloplasty was attempted but did not achieve acceptable functional results; consequently, tricuspid valve replacement was performed. Given the patient's young age (20 years), a mechanical heart valve was preferred to a bioprosthetic valve to reduce the risk of early structural deterioration and subsequent multiple reinterventions. Histopathological examination of the resected valve revealed fibrous tissue hyperplasia with collagenization and hyaline degeneration (Figure 2B).

The patient's condition stabilized postoperatively. He was transferred from the ICU on day 14 and discharged on day 21. At the 2-year follow-up, the patient reported no limitations in daily activities and TTE showed preserved prosthetic valve function. The clinical timeline of key events throughout the patient's course is shown in Figure 3.

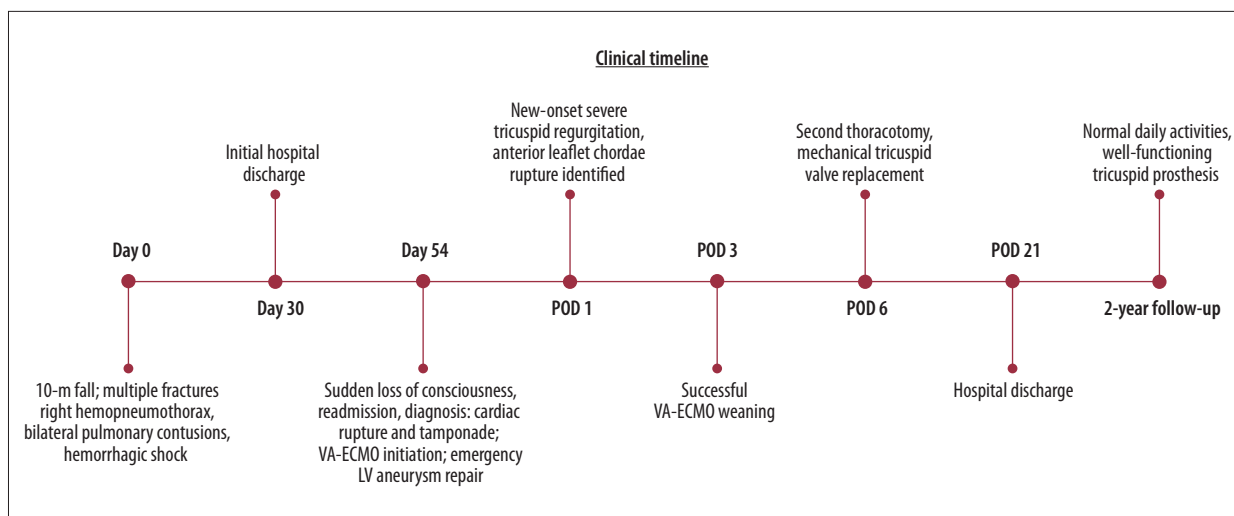


Figure 3. Schematic clinical timeline demonstrating key clinical events following blunt chest trauma. VA-ECMO, venoarterial extracorporeal membrane oxygenation; LV, left ventricular; POD, postoperative day.

Discussion

The clinical manifestations of blunt cardiac injury vary greatly depending on the mechanism and severity of the injury. Consistent with previous reports, delayed cardiac rupture remains a rare but life-threatening consequence of blunt cardiac injury [5]. However, unlike previously reported cases, our patient was subsequently found to have tricuspid chordal rupture after aneurysm repair, indicating a potentially more complex progression of traumatic cardiac injury than is typically described. This case highlights several important diagnostic considerations. First, a markedly elevated high-sensitivity troponin I level followed by a slow decline after the initial trauma may indicate severe myocardial damage. Clinicians should maintain a high index of suspicion in such patients. Even after symptom resolution, normalization of electrocardiogram findings, and the return of troponin levels to baseline, long-term echocardiographic monitoring should be considered to screen for aneurysm formation [6,7]. Second, new-onset severe valvular regurgitation after cardiac surgery should not be attributed solely to surgical trauma. In patients with a history of severe blunt cardiac injury, delayed valvular changes secondary to trauma should be considered. Transesophageal echocardiography and pathological examination are crucial for determining etiology.

From a mechanistic perspective, this case suggests a potential sequential cardiac injury model that differs from the isolated events commonly reported in the literature. Initial trauma-induced myocardial contusion led to myocyte necrosis, scar formation, and wall thinning, ultimately resulting in a true aneurysm [8]. The aneurysm ruptured under hemodynamic stress, causing acute cardiac tamponade. Preoperative TTE did not reveal significant tricuspid regurgitation or chordal rupture. However, owing to acute hemodynamic collapse, TEE could

not be performed for further confirmation prior to ventricular aneurysm repair. Furthermore, as neither the valve nor the right ventricle was manipulated during the procedure, intraoperative TEE was omitted.

Postoperative severe tricuspid regurgitation was observed, and the excised valve tissue showed fibrosis and hyaline degeneration, suggesting that occult injury may have predated ventricular aneurysm repair. Surgical repair of the ventricular aneurysm may have reduced left ventricular compliance and increased right ventricular load via ventricular interdependence [9]. This remains a hypothesis, given that direct measurement of right ventricular pressure was not performed during the perioperative period. However, despite the reduction in right heart preload afforded by VA-ECMO, postoperative TEE demonstrated persistent right atrial dilation, indirectly suggesting an increased right ventricular pressure. Therefore, we hypothesize that the rupture of the chordae tendineae following ventricular aneurysm repair was precipitated by hemodynamic alterations affecting the previously compromised chordae. This case cannot distinguish among delayed traumatic injury from chronic degenerative susceptibility, inflammation-related changes, or perioperative precipitation. This complex progression underscores the importance of dynamic monitoring after severe blunt cardiac injury.

Given the risk of bleeding, the application of ECMO in trauma patients warrants caution; its use in traumatic cardiac ruptures is rarely reported. The risk of intraoperative mortality during cardiac rupture repair is high, and direct surgical repair in the setting of severe shock may precipitate cardiac arrest. The “VA-ECMO-first bridging” strategy rapidly corrected refractory shock, restored organ perfusion, and established a critical therapeutic window for the surgical team. In adult patients

with trauma supported by VA-ECMO, the high risk of bleeding necessitates an individualized minimal anticoagulation strategy, and systemic heparinization may be withheld for 48 hours to 5 days when clinically indicated [10,11]. In this case, during ECMO support following aneurysm repair, serial laboratory monitoring of coagulation parameters and platelet counts was performed every 6 to 12 hours. The platelet count fluctuated between 63 and $74 \times 10^9/L$, and the activated partial thromboplastin time ranged from 47 to 72 s. These laboratory abnormalities prompted adoption of an anticoagulation-free strategy. Circuit monitoring consisted of daily visual inspection of the circuit and oxygenator; no visible oxygenator clotting or evidence of circuit thrombosis was identified. During ECMO support, the patient produced 150 mL of bloody drainage via the pericardial tube within the first 6 hours post-operatively and received 450 mL of plasma. No additional blood-product transfusions were administered. This experience adds to the limited evidence base supporting the feasibility of anticoagulation-free VA-ECMO in selected trauma patients.

As this is a single-case report, the proposed pathophysiological mechanism and treatment strategy require validation using additional clinical data.

Conclusions

This case underscores the critical lesson that serious structural cardiac complications can emerge after apparent clinical

recovery from severe blunt cardiac injury. Therefore, continued clinical and echocardiographic vigilance is essential. Newly detected severe valvular regurgitation after cardiac surgery in such patients warrants prompt reassessment, as its cause may not be solely iatrogenic. VA-ECMO can serve as a valuable bridging strategy in select critically ill patients with traumatic cardiac rupture. Finally, the proposed causal relationship between aneurysm repair and subsequent tricuspid chordal rupture remains uncertain and requires further investigation.

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Institution Where Work Was Done

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Patient Consent

Written informed consent 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.

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