International Journal of Anesthesia and Clinical Medicine
2024, Vol. 12, No. 1, pp. 11-14
https://doi.org/10.11648/j.ijacm.20241201.13

Case Report

Thrombosis of Prosthetic Tricuspid Valve During Veno-Arterial Extracorporeal Membrane Oxygenation Support: A Case Report

Yunyi Zhang[1], Shuhua Luo[2, *]

[1]Department of Anesthesia and Operation Center, West China Hospital of Sichuan University, Chengdu, China
[2]Department of Cardiovascular Surgery, West China Hospital of Sichuan University, Chengdu, China

Abstract

Extracorporeal membrane oxygenation (ECMO) is frequently used for severe postcardiotomy cardiogenic shock in patients with bioprosthetic valves. Acute prosthetic valve thrombosis (PVT) is a rare complication after valve replacement surgery and significantly increases morbidity and mortality. Patients who develop PVT on ECMO could significantly influence the long-term durability of the bioprosthetic valves. However, previous studies only analyzed risk factor and treatment of the mitral valve thrombosis during ECMO support. The mechanism of thrombosis on the tricuspid valve was still unknown. Here we describe the symptoms and treatment of a valve replacement patient who developed bioprosthetic tricuspid valve thrombosis during veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Meanwhile, the patient's mechanical prosthetic mitral valve functioned normally. An emergency re-do tricuspid prosthesis replacement was performed, and the patient finally developed the successful decannulation. At 6 months follow-up, the patient showed asymptomatic and had a reasonable quality of life. The pathophysiology of tricuspid valve thrombosis may be different from the left heart. Our case highlights that the risk of thrombosis associated with a prosthesis in the tricuspid position can be even higher in the setting of VA-ECMO support. In such patients, promoting forward blood flow across the prosthesis and improving levels of anticoagulation may be particularly important.

Keywords

Prosthetic Valve Thrombosis, Extracorporeal Membrane Oxygenation, Bioprosthetic Tricuspid Valve, Case Report

1. Introduction

Acute prosthetic valve thrombosis (PVT) is a rare complication of valve replacement surgery that significantly increases risk of morbidity and mortality. Approximately 1.7% of patients developed severe postcardiotomy cardiogenic shock that requires veno-arterial extracorporeal membrane oxygenation (VA-ECMO) [1, 2]. Patients who develop PVT on ECMO could significantly influence the long-term durability of the bioprosthetic valves [3]. Pathophysiology and risk factors of mitral as well as aortic PVT has been reported in previous studies during VA-ECMO support [4-10]. However, the thrombosis of tricuspid prosthesis has not been previously described in such settings. Since the blood flow velocity and pressure of right heart was quite different from those in the left heart. We believe that the pathophysiology of tricuspid valve thrombosis was distinct. In

*Corresponding author: drshuhualuo@gmail.com (Shuhua Luo)

Received: 17 January 2024; **Accepted:** 31 January 2024; **Published:** 20 February 2024

this case report, we describe a patient who developed bioprosthetic tricuspid valve thrombosis during postoperative VA-ECMO support and underwent successful rescue surgery. Meanwhile, the patient's mechanical prosthetic mitral valve functioned normally. We discuss the possible causes and clinical course of PVT in patients on VA-ECMO support, as well as potential strategies to manage them.

2. Case Presentation

A 46-year-old woman was admitted to our hospital because of worsening, persistent right heart failure with dyspnea, fatigue, and edema of the lower limbs. At the age of 26, she had undergone mechanical prosthetic valve replacement (27 mm, tilting disc, GK Company, Xi'an, China) for mitral rheumatic disease at another medical facility. After that procedure, the dose of warfarin was adjusted to maintain an international normalized ratio at 2.5-3, and no thromboembolic or bleeding events were observed after mitral valve replacement.

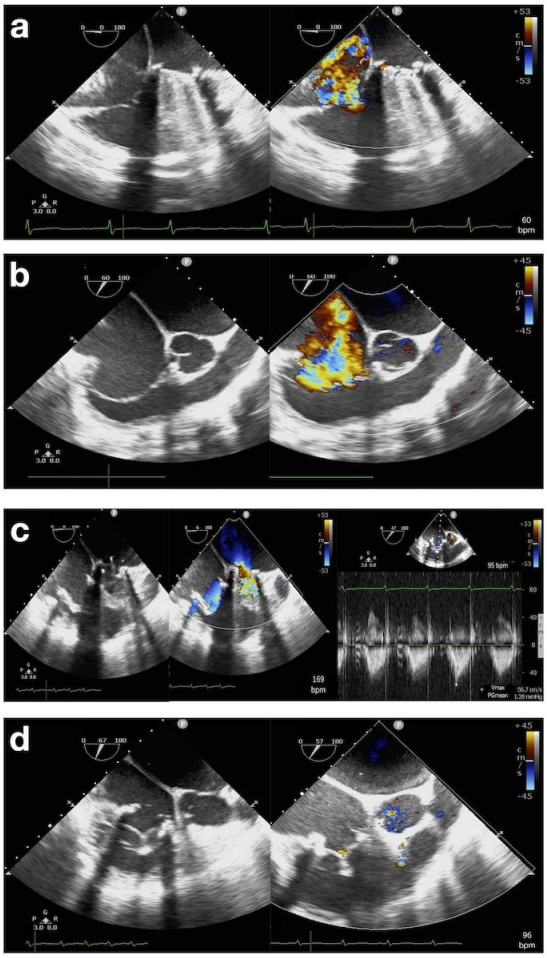

Figure 1. *(a-b) Preoperative transesophageal echocardiography revealed severe tricuspid regurgitation and a normally functioning mitral prosthesis: (a) mid-esophageal four-chamber view, (b) right ventricle inflow-outflow view. (c-d) Postoperative transesophageal echocardiography showed good bioprosthetic tricuspid valve function in (c) diastole and (d) systole (bpm = beats/min).*

After initiating ECMO, activated clotting time was titrated to 180-220 seconds [11] using intravenous heparin infusions, as per institutional protocols. Other coagulation parameters were routinely monitored, such as activated partial thromboplastin time, antithrombin III, fibrin, and platelets (Figure 2a). Despite full-flow support, arterial lactate increased to 11.4 mmol/L during the first 7 hours on ECMO, so blood flow was increased to 6 L/min (approximately 100 ml/kg/min), which facilitated lactate clearance and end-organ perfusion. By 36 hours on ECMO, ventricular function had recovered and flow was reduced to 3 L/min (approximately 55 ml/kg/min) (Figure 2b).

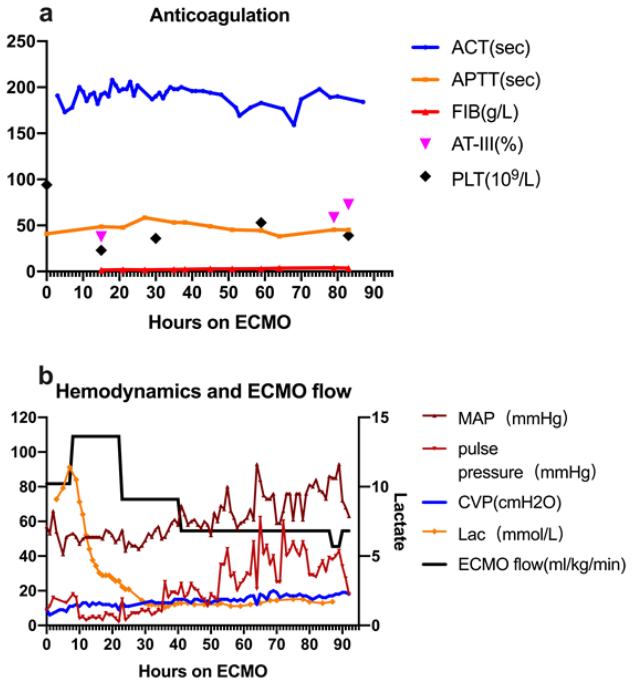

Figure 2. *(a) Anticoagulation parameters during extracorporeal membrane oxygenation (ECMO) support: activated clotting time (ACT), activated partial thromboplastin time (APTT), antithrombin III (AT-III), fibrin (FIB), and platelet count (PLT). (b) Hemodynamic parameters, including mean arterial pressure (MAP), pulse pressure, central venous pressure (CVP), lactate level (Lac), and ECMO flow.*

Transthoracic echocardiography was performed daily to assess the function of the ventricle and the prosthesis. No stasis or distention was observed for left or right ventricular blood. After 5 days of ECMO, we attempted to decannulate the patient but were unsuccessful because central venous pressure (25 mmHg) rose substantially immediately after flow was reduced to 1.5 L/min (approximately 30 ml/kg/min).

Bedside transthoracic echocardiography revealed severe tricuspid prosthesis stenosis with restricted leaflet motion, as well as mean pressure gradients as high as 13.5 mmHg (Figure 3). The patient was returned immediately to the operating room. Direct visualization of the bioprosthetic tricuspid valve revealed thrombosis on the ventricle side, which significantly

restricted leaflet motion and obstructed the orifice. No evidence of thrombosis was found in the atria, ventricles, or mitral prosthesis. The thrombosed tricuspid prosthesis was replaced and the patient was successfully decannulated one day after surgery. Postoperative recovery was uneventful, and echocardiography before discharge showed no thrombosis as well as good function of mitral and tricuspid prostheses. Follow-up after 6 months showed that the patient remained asymptomatic and was able to perform daily activities without assistance.

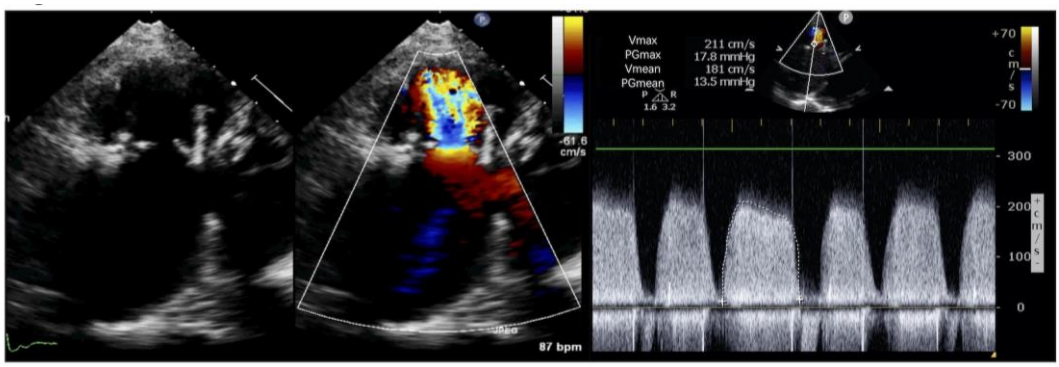

Figure 3. *Emergency bedside transthoracic echocardiography prior to decannulation revealed severe stenosis of the tricuspid prosthesis, leading to flow acceleration. The apical four-chamber view is shown.*

3. Discussion

For patients with prosthetic valves, VA-ECMO is a recognized risk factor of thrombosis [3]. Acute postoperative PVT can occur, primarily in the mitral position [4-10]. To the best of our knowledge, this is the first report of prosthetic tricuspid valve thrombosis during VA-ECMO support. The presence of mitral and tricuspid prosthetic valves in the same patient provided a unique opportunity to analyze and compare the mechanism of thrombosis occurring on right- or left-sided prostheses in the setting of VA-ECMO support.

Maintaining adequate systemic anticoagulation is critical to preventing PVT. The Extracorporeal Life Support Organization (ECLS) registry has published recommendations on optimal anticoagulation management for patients on ECMO, including titration to achieve an activated clotting time of 180-220 seconds [11]. The present case suggests that while this recommendation may be adequate for patients with a mitral prosthesis, those with a tricuspid prosthesis may require stronger anticoagulation. The risk of PVT needs to be carefully balanced against the risk of bleeding in patients undergoing ECMO, which can be challenging to accomplish soon after surgery [12]. Monitoring other coagulation parameters, such as serum levels of anti-Factor Xa antibody, may be useful for ensuring adequate anticoagulation [13]. Bivalirudin may also be useful for strengthening anticoagulation [13].

In addition to systemic anticoagulation, the hemodynamic pathophysiology of prosthetic mitral valve thrombosis in patients undergoing VA-ECMO support has been clearly described [8]. Left ventricular distension, inadequate left ventricular ejection and consequent blood stasis within the left ventricle can occur during support. This can prevent the opening of the mitral prosthetic valve, resulting in PVT [14]. Tricuspid PVT may occur by a different mechanism. Since ECMO drains blood directly from the systemic venous system, blood stasis is less likely to occur in the right ventricle. In the present case, we suspect that the aggressive venous drainage of ECMO during high-flow support substantially reduced, or even eliminated, transvalvular flow across the tricuspid prosthesis. As a result, the prosthetic tricuspid valve could not open, leading to thrombosis and possibly commissural fusion. Although we did not directly monitor pulmonary artery pressure, our hypothesis is supported by daily transthoracic echocardiography, which showed no right ventricle distension or blood stasis during VA-ECMO. While loss of transvalvular flow need not induce thrombosis in a native tricuspid valve, it may strongly increase risk of PVT in a prosthetic valve, which can exert strongly procoagulant effects.

Echocardiography is powerful for diagnosing bioprosthetic thrombosis, but it may be less reliable in a setting of VA-ECMO support. Various echocardiographic criteria have been suggested for detecting thrombosis [15], including increased gradient across the prosthetic valve, thickened valve cusps or abnormal cusp motion, but all of these can be masked by reduced transvalvular flow in patients undergoing ECMO.

Managing patients with prosthetic valves who receive VA-ECMO support is a challenge. While the literature has focused on thrombosis in left-sided prosthetic valves, the present case suggests that risk of thrombosis can be even higher in patients with a prosthetic tricuspid valve who undergo VA-ECMO support. Furthermore, tricuspid PVT may occur through different mechanisms than mitral or aortic PVT. The present case suggests that patients with tricuspid prosthetic valves may require special ECMO strategies to encourage forward blood flow across the prosthesis, frequent echocardiography monitoring and stronger anticoagulation.

4. Conclusion

Acute PVT on VA-ECMO is a rare complication of valve replacement surgery that significantly increases risk of morbidity and mortality. Our case highlights that the risk of thrombosis associated with a prosthesis in the tricuspid position can be even higher in the setting of VA-ECMO support. In such patients, promoting forward blood flow across the prosthesis, frequent echocardiography monitoring and stronger anticoagulation may be particularly important.

Abbreviations

ECMO: Extracorporeal Membrane Oxygenation
PVT: Prosthetic Valve Thrombosis
VA-ECMO: Veno-arterial Extracorporeal Membrane Oxygenation
Bpm: Beats/min
ACT: Activated Clotting Time
APTT: Activated Partial Thromboplastin Time
AT-III: Antithrombin III
FIB: Fibrin
PLT: Platelet Count
MAP: Mean Arterial Pressure
CVP: Central Venous Pressure
Lac: Lactate
ECLS: Extracorporeal Life Support Organization

Acknowledgments

The authors declare no conflicts of interest.

Conflicts of Interest

The authors declare that they have no competing interests.

References

[1] Wang JG, Han J, Jia YX, Zeng W, Hou XT, Meng X. Outcome of veno-arterial extracorporeal membrane oxygenation for patients undergoing valvular surgery. *PLoS One*. 2013; 8(5): e63924. Published 2013 May 23. https://doi.org/10.1371/journal.pone.0063924

[2] Alhijab FA, Tantawy TM, Ismail HH, et al. Venoarterial extracorporeal membrane oxygenation for postcardiotomy cardiogenic shock: The impact of cannulation strategy on survival. *Perfusion*. 2023; 38(7): 1444-1452. https://doi.org/10.1177/02676591221114954

[3] Arafat AA, AlBarrak M, Kiddo M, et al. Extracorporeal membrane oxygenation for postcardiotomy cardiogenic shock after valve replacement. *Perfusion*. Published online January 16, 2023. https://doi.org/10.1177/02676591231152723

[4] Groenewoud R, Gunning D, Fava C, Sharpe R, Valchanov K, Shayan H. Successful thrombolysis of early bioprosthetic mitral valve thrombosis following extracorporeal membrane oxygenation: Case report. *Perfusion*. Published online February 16, 2023. https://doi.org/10.1177/02676591231157200

[5] Callisen H, Sen A, Lanza L, et al. Management of Acute Mechanical Mitral Valve Thrombosis With Venoarterial Extracorporeal Membrane Oxygenation (ECMO). *J Cardiothorac Vasc Anesth*. 2017; 31(1): 240-242. https://doi.org/10.1053/j.jvca.2016.05.009

[6] Tsiouris A, Nemeh H, Borgi J. Early acute thrombosis of bioprosthetic mitral valve presenting with cardiogenic shock. *Gen Thorac Cardiovasc Surg*. 2013; 61(3): 152-154. https://doi.org/10.1007/s11748-012-0121-4

[7] Challa A, Latona J, Fraser J, et al. Mitral valve bio-prosthesis and annuloplasty thrombosis during extracorporeal membrane oxygenation: case series. *Eur Heart J Case Rep*. 2020; 4(3): 1-6. Published 2020 Apr 24. https://doi.org/10.1093/ehjcr/ytaa085

[8] Dahl AB, Gregory SH, Ursprung E, Kawabori M, Couper GS, Hueneke R. Acute Presentation of Bioprosthetic Mitral Valve Thrombosis in a Patient on Venoarterial Extracorporeal Membranous Oxygenation. *J Cardiothorac Vasc Anesth*. 2019; 33(3): 844-849. https://doi.org/10.1053/j.jvca.2018.05.007

[9] Cevasco M, Saha A, Garan A R, et al. Incidence and Outcomes of Prosthetic Valve Thrombosis during Extracorporeal Membrane Oxygenation Support for Postcardiotomy Shock. *The Journal of Heart and Lung Transplantation*, 2019, 38(4): S423. https://doi.org/10.1016/j.healun.2019.01.1078

[10] Kagiyama N, Okura H, Nezuo S, et al. Two cases of acute bioprosthetic mitral valve thrombosis immediately after mitral valve replacement. *Circulation*. 2014; 129(6): e328-e330. https://doi.org/10.1161/CIRCULATIONAHA.113.005583

[11] Thiagarajan RR, Barbaro RP, Rycus PT, et al. Extracorporeal Life Support Organization Registry International Report 2016. *ASAIO J*. 2017; 63(1): 60-67. https://doi.org/10.1097/MAT.0000000000000475

[12] Labarrere CA, Dabiri AE, Kassab GS. Thrombogenic and Inflammatory Reactions to Biomaterials in Medical Devices. *Front Bioeng Biotechnol*. 2020; 8: 123. Published 2020 Mar 12. https://doi.org/10.3389/fbioe.2020.00123

[13] Murphy DA, Hockings LE, Andrews RK, et al. Extracorporeal membrane oxygenation-hemostatic complications. *Transfus Med Rev*. 2015; 29(2): 90-101. https://doi.org/10.1016/j.tmrv.2014.12.001

[14] Noly PE, Tang PC. Commentary: Venoarterial extracorporeal membrane oxygenation, ventricular dysfunction, and a mitral valve prosthesis: A recipe for valve thrombosis? *JTCVS Tech*. 2020; 3: 209-210. Published 2020 May 5. https://doi.org/10.1016/j.xjtc.2020.04.022

[15] Egbe A, Pislaru SV, Ali MA, et al. Early Prosthetic Valve Dysfunction Due to Bioprosthetic Valve Thrombosis: The Role of Echocardiography. *JACC Cardiovasc Imaging*. 2018; 11(7): 951-958. https://doi.org/10.1016/j.jcmg.2017.06.022