JA Clinical Reports

CASE REPORT

Open Access

Unusual severe hypoxemia due to unilateral pulmonary edema after conventional cardiopulmonary bypass salvaged by veno-venous extracorporeal membrane oxygenation: a case report

Masataka Fukuda[1], Hiroaki Sakai[2], Keito Koh[3], Sonoko Sakuraba[3], Nozomi Ando[1], Masakazu Hayashida[1] and Izumi Kawagoe[1*]

Abstract

Background We report a case in which veno-venous extracorporeal membrane oxygenation (V-V ECMO) saved the life of a patient who developed severe hypoxemia due to unusual unilateral pulmonary edema (UPE) after cardiopulmonary bypass (CPB).

Case presentation A 69-year-old man underwent aortic valve replacement and coronary artery bypass grafting. Following uneventful weaning off CPB, he developed severe hypoxemia. The ratio of arterial oxygen tension to inspired oxygen fraction (PaO_2/FiO_2) decreased from 301 mmHg 5 min after CPB to 42 mmHg 90 min after CPB. A chest X-ray revealed right-sided UPE. Immediately established V-V ECMO increased PaO_2/FiO_2 to 170 mmHg. Re-expansion pulmonary edema (REPE) was likely, as the right lung remained collapsed during CPB following the accidental opening of the right chest cavity during graft harvesting.

Conclusions V-V ECMO was effective in improving oxygenation and saving the life of a patient who had fallen into unilateral REPE unusually developing after conventional CPB.

Keywords Cardiopulmonary bypass, Re-expansion pulmonary edema, Unilateral pulmonary edema, Veno-venous extracorporeal membrane oxygenation

Background

Acute pulmonary edema after cardiac surgery develops due to cardiogenic and non-cardiogenic causes and occasionally leads to severe hypoxemia that cannot be managed with mechanical ventilation alone [1, 2]. Unilateral pulmonary edema (UPE) after on-pump minimally invasive cardiac surgeries (MICS) has been increasingly reported [3]. However, reports on UPE after conventional on-pump cardiac surgeries remain very rare [4]. Herein, we report a case in which veno-venous extracorporeal membrane oxygenation (V-V ECMO) saved the life of a patient with severe hypoxemia due to UPE that

*Correspondence:
Izumi Kawagoe
ikawago@juntendo.ac.jp
[1] Department of Anesthesiology and Pain Medicine, Juntendo University Hospital, Tokyo 113-8431, Japan
[2] Department of Anesthesia, Fujieda Municipal General Hospital, Shizuoka, Japan
[3] Department of Anesthesiology and Pain Medicine, Juntendo University Shizuoka Hospital, Shizuoka, Japan

developed acutely after weaning off conventional cardiopulmonary bypass (CPB).

Case presentation

A 69-year-old man (162 cm, 48.5 kg), with a history of coronary artery disease (CAD) previously treated with percutaneous transluminal coronary angioplasty, hypertension as well as diabetes mellitus under medication, and end-stage diabetic nephropathy on hemodialysis, was scheduled for aortic valve replacement (AVR) and coronary artery bypass grafting (CABG) for newly diagnosed severe aortic stenosis (AS) and recurrent CAD. Preoperative transthoracic echocardiography showed a global left ventricular ejection fraction (in the presence of regional hypokinesis) of 53%, left ventricular diastolic/systolic dimensions of 37/27 mm, and AS due to calcified valve leaflets, with an aortic valve area (AVA) of 0.5 cm^2 and indexed AVA of 0.19 cm^2/m^2. Coronary angiography showed 90% stenotic lesions in the right coronary artery (RCA) and left circumflex artery (LCX).

After the establishment of an arterial line, general anesthesia was induced with midazolam 4 mg and fentanyl 400 μg. Vecuronium bromide 8 mg was given to facilitate tracheal intubation. A central venous catheter and pulmonary artery catheter (PAC) were placed via the right internal jugular vein. Transesophageal echocardiography (TEE) was also used. General anesthesia was maintained with propofol 2 mg/kg/h and remifentanil 0.3 μg/kg/min. Following a median sternotomy, the right internal thoracic artery (RITA) graft and saphenous vein graft (SVG) were harvested. During RITA harvesting, the right chest cavity was accidentally opened but was subsequently left opened until immediately before chest closure. After graft harvesting, CPB was started with the arterial cannula placed in the ascending aorta and venous cannulas placed in the superior and inferior vena cava (IVC). First, on-pump beating CABG was performed, including a RITA-LCX anastomosis and two sequential SVG-RCA anastomoses. Then, the ascending aorta was cross-clamped, and AVR was performed with a Trifecta biological valve. During CPB, the patient received transfusions of 12 units of red cell concentrates and 4 units of irradiated fresh-frozen plasma (FFP).

After completion of the surgical repair, adequate de-airing of the heart was facilitated with a manual lung inflation maneuver and volume loading of the heart. Prior to weaning from CPB, mechanical ventilation was resumed with an inspired oxygen fraction (FiO_2) of 1. The patient was successfully weaned off CPB with some inotropic support. His pulmonary artery pressure (PAP) remained normal, and cardiac index (CI) remained at approximately 2.5 L/min/m^2. TEE revealed the well-functioning artificial valve, well-preserved left ventricular wall motion, trivial mitral regurgitation (MR), and no enlargement of the left atrium (LA). A significant volume of retained blood was evacuated from the right thoracic cavity. The patient was uneventfully weaned off CPB. The durations of CPB and aortic cross-clamping were 251 min and 127 min, respectively.

After administration of protamine, 4 units of irradiated FFP and 20 units of platelet concentrates were transfused. Shortly after the start of the transfusion, however, he developed progressive hypoxemia. The ratio of arterial oxygen tension to FiO_2 (PaO_2/FiO_2) decreased from 301 mmHg 5 min after CPB to 140 mmHg 45 min after CPB and, further, to 42 mmHg 90 min after CPB (Fig. 1) . As soon as the surgical wound was closed 475 min after the start of surgery, anesthesiologists inserted a return cannula into the right atrium via the left internal jugular vein, while surgeons inserted a drainage cannula into the IVC via the right femoral vein to establish V-V ECMO. A chest X-ray showed diffuse unilateral pulmonary consolidation in the right lung with no increase in a cardiothoracic ratio, indicative of non-cardiogenic UPE (Fig. 2). V-V ECMO with a flow rate of 2 L/min successfully increased PaO_2/FiO_2 to 170 mmHg (Figs. 2 and 3).

The patient was transferred to the intensive care unit. Continuous hemodiafiltration was started via a catheter placed in the left femoral vein. The improvement in oxygenation was maintained with V-V ECMO and pressure-controlled ventilation employing a lung-protective strategy (Fig. 1). The patient was weaned off V-V ECMO on postoperative day (POD) 3 (Fig. 1) and was extubated on POD 7. The patient was, however, re-intubated due to aspiration on POD 7 and re-extubated on POD 13. He returned to the general ward on POD 17 and was subsequently discharged in an ambulatory condition on POD 78. Neither antihuman leukocyte antigen (HLA) antibody nor anti-granulocyte antibody was detected in samples of the transfused blood products.

Discussion

Acute cardiogenic or non-cardiogenic pulmonary edema sometimes develops in patients undergoing major cardiovascular surgery, although most cases present with bilateral pulmonary edema (BPE) [5]. Reportedly, UPE, which accounts for 2.1% of cardiogenic pulmonary edema (CPE) cases, usually occurs in the right lung [6]. Although there is no specifically established treatment for UPE that differs from BPE, UPE is associated with more severe conditions requiring mechanical ventilation and more intensive inotropic support [6]. The major cause of cardiogenic UPE is MR due to its propensity to flow anatomically into the right superior pulmonary vein [7]. Another underlying mechanism of right-sided UPE is a difference in interstitial flow in the lungs due to the right-sided lymphatic vessels having a smaller aperture and a longer distance to the thoracic duct than left-sided lymphatic vessels [8, 9].

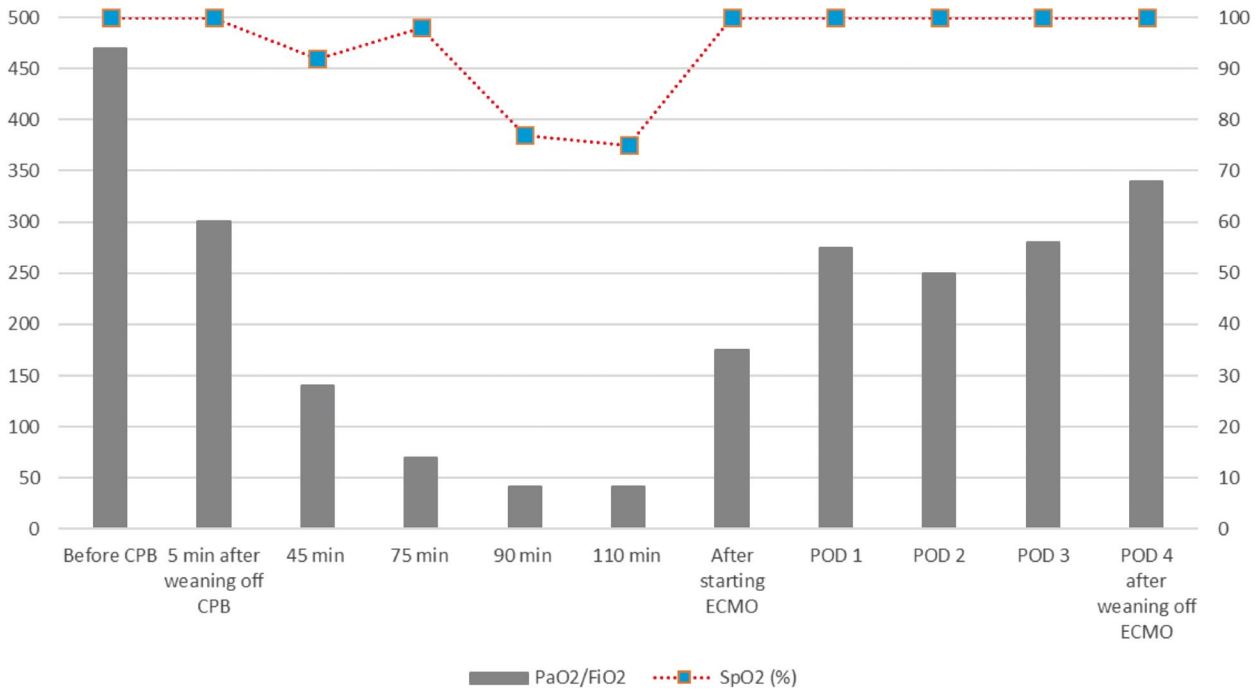

Fig. 1 PaO_2/FiO_2 ratio and SpO_2 during and after surgery. PaO_2/FiO_2, ratio of arterial oxygen tension to inspired oxygen fraction; SpO_2, percutaneous arterial oxygen saturation; CPB, cardiopulmonary bypass; ECMO, extracorporeal membrane oxygenation; POD, postoperative day

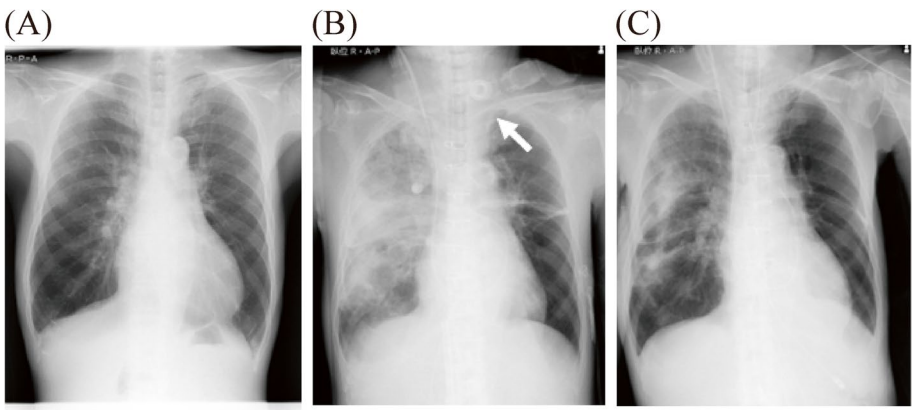

Fig. 2 Chest X-rays **A** before surgery, **B** after chest closure, and **C** on postoperative day 5. The chest X-ray taken immediately after chest closure (**B**) showed diffuse unilateral pulmonary consolidation in the right lung, with no increase in cardiothoracic ratio, consistent with non-cardiogenic, unilateral pulmonary edema. That taken on POD 5 (**C**) showed improvement in unilateral pulmonary consolidation. The white arrow depicted in **B** indicates a return cannula placed via the left internal jugular vein

In our patient, UPE occurred in the right lung. However, CPE was unlikely, since TEE did not reveal significant MR or LA enlargement, and instead revealed well-preserved left ventricular wall motion, while the PAC revealed normal PAP and CI, and chest X-ray revealed neither the increased cardiothoracic ratio nor "butterfly pattern" typically seen in CPE.

UPE can develop also due to other pathological conditions, such as acute pulmonary emboli, re-expansion pulmonary edema (REPE), and external compression of the pulmonary vasculatures by a tumor or hematoma, resulting in asymmetrical inflow to the pulmonary arteries or outflow to the pulmonary veins [10]. In our case, the right lung was left collapsed during CPB after discontinuation of mechanical ventilation, since the right chest cavity was opened during RITA graft harvesting. Furthermore, blood retention in the cavity might have aggravated the collapse. Therefore, REPE due to rapid re-expansion of the right lung following its collapse likely caused right-sided

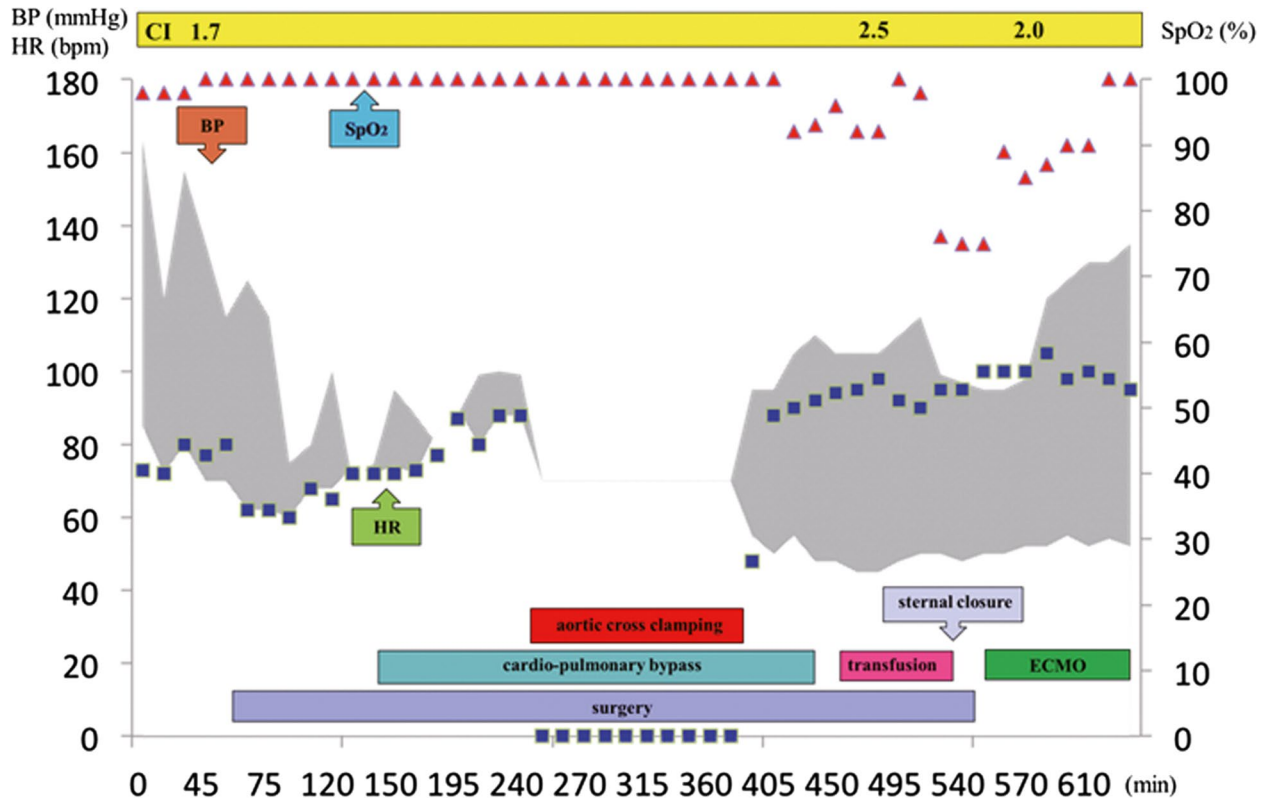

Fig. 3 Hemodynamic changes before, during, and after surgery. Anesthesia starts from 0 on the horizontal axis. BP, blood pressure; HR, heart rate; CI, cardiac index; SpO , percutaneous arterial oxygen saturation; ECMO, extracorporeal membrane oxygenation

UPE. Typically, REPE develops only after the lung collapsed for 72 h or longer is rapidly reinflated [11]. However, since on-pump MICS via right mini-thoracotomies using one-lung ventilation are increasingly being performed, it has become evident that right-sided unilateral REPE can develop much earlier after CPB [12, 13]. Lung collapse is associated with the sequestration of leucocytes, and inflammatory activity is triggered when oxygen is supplied during re-expansion and concomitant reperfusion of the lung [14, 15]. This inflammatory response might be enhanced by multiple factors during cardiac surgery [12]. The inflammatory response to re-expansion and reperfusion can be aggravated by the generalized inflammatory response generated by CPB [12], as suggested by the association between prolonged CPB time and the development of UPE in MICS [13, 14]. The FiO_2 set at 1.0 upon resuming ventilation might have worsened the inflammatory reaction during re-expansion, though we reduced the FiO_2 to 0.5 immediately after measuring blood gas 5 min after CPB. Furthermore, the restricted bronchial artery blood flow during CPB might worsen lung ischemia [16]. Therefore, the combination of right lung collapse and prolonged CPB time might have resulted in the unilateral REPE in our case, although unilateral REPE has rarely been reported in patients undergoing conventional on-pump cardiac surgery [4], unlike those undergoing on-pump MICS [12, 13].

Since UPE occurred after the start of blood transfusion in our patient, we also considered the possibility of transfusion-related acute lung injury (TRALI) [17–19]. However, anti-HLA antibody or anti-granulocyte antibody, which reportedly triggers lung microvascular endothelial damage [19], was not detected in samples of transfused blood products. Additionally, TRALI usually presents with BPE [18]. Therefore, TRALI less likely caused UPE than REPE in our case.

The use of ECMO is indicated when a poor therapeutic response to conventional mechanical ventilation and a high mortality rate are predicted in patients with reversible lung injury [20, 21]. When considering the indication for ECMO, it is recommended to estimate the Murray score, in which a score of 2.5 or more indicates "severe" lung injury [21, 22]. Our patient was indicated for ECMO, since he was highly likely to die from severe hypoxemia without using ECMO, given his Murray score estimated to be 2.75 and PaO_2/FiO_2 as low as 42 mmHg.

In conclusion, we reported a rare case of UPE with severe hypoxemia that developed after conventional on-pump cardiac surgery. REPE seemed the likely cause of the UPE. V-V ECMO was quite effective in improving oxygenation and saving the patient's life.

Abbreviations

AS	Aortic stenosis
AVA	Aortic valve area
AVR	Aortic valve replacement
BPE	Bilateral pulmonary edema
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
CI	Cardiac index
CPB	Cardiopulmonary bypass
CPE	Cardiogenic pulmonary edema
FFP	Fresh-frozen plasma
FiO_2	Inspired oxygen fraction
IVC	Inferior vena cava
LA	Left atrium
LCX	Left circumflex artery
MICS	Minimal invasive cardiac surgery
MR	Mitral regurgitation
PAC	Pulmonary artery catheter
PaO2/FiO2	Ratio of arterial oxygen tension to fraction of inspired oxygen
PAP	Pulmonary artery pressure
POD	Postoperative day
RCA	Right coronal artery
REPE	Re-expansion pulmonary edema
RITA	Right internal thoracic artery
SVG	Saphenous vein graft
TRALI	Transfusion-related acute lung injury
TEE	Transesophageal echocardiography
UPE	Unilateral pulmonary edema
V-V ECMO	Veno-venous extracorporeal membrane oxygenation

Acknowledgements
The authors thank and respect the patient and the Juntendo Hospital operating room staff.

Authors' contributions
MF, HS, and SS performed the anesthesia and postoperative care. MF, HS, and MH wrote the manuscript. KK and NA collected the data. MH and IK edited the manuscript critically. The authors have read and approved the final manuscript.

Funding
None.

Availability of data and materials
The datasets related to this report are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The institutional review board of our hospital approved this case presentation.

Consent for publication
The patient gave written informed consent for publication of this case report and any accompanying images.

Competing interests
The authors declare that they have no competing interests.

Received: 4 September 2023 Revised: 25 September 2023 Accepted: 26 September 2023
Published online: 07 October 2023

References

1. Bignami E, Frati E, Meroni R, Verzini A, Pozzoli A, Benussi S, Alfieri O. Extracorporeal venovenous membrane oxygenation in the treatment of respiratory insufficiency following cardiac surgery. J Card Surg. 2014;29(2):270–3.
2. Vardas PN, Matthews C, Rosati CM, Beckman DJ. Severe re-expansion pulmonary edema after conventional cardiac surgery: identification and management. J Card Surg. 2019;34(6):525–7.
3. Moss E, Halkos ME, Binongo JN, Murphy DA. Prevention of unilateral pulmonary edema complicating robotic mitral valve operations. Ann Thorac Surg. 2017;103(1):98–104.
4. Toyama M, Nakayama M, Fujita Y, Shimazu S, Sawasaki T. Unilateral pulmonary edema during aortic valve replacement through median sternotomy. Kyobu Geka. 2020;73(6):417–22.
5. Ware LB, Matthay MA. Clinical practice. Acute pulmonary edema. N Engl J Med. 2005;353(26):2788–96.
6. Attias D, Mansencal N, Auvert B, Vieillard-Baron A, Delos A, Lacombe P, N'Guetta R, Jardin F, Dubourg O. Prevalence, characteristics, and outcomes of patients presenting with cardiogenic unilateral pulmonary edema. Circulation. 2010;122(11):1109–15.
7. Bekiaridou A, Kartas A, Moysidis DV, Papazoglou AS, Patsiou V, Baroutidou A, Kamperidis V, Giannakoulas G. Severe mitral regurgitation causing unilateral pulmonary edema: a case report. J Cardiol Cases. 2022;26(2):130–3.
8. Alarcón JJ, Guembe P, de Miguel E, Gordillo I, Abellás A. Localized right upper lobe edema. Chest. 1995;107(1):274–6.
9. Lesieur O, Lorillard R, Thi HH, Dudeffant P, Ledain L. Unilateral pulmonary oedema complicating mitral regurgitation: diagnosis and demonstration by transoesophageal echocardiography. Intensive Care Med. 2000;26(4):466–70.
10. Hirata K, Ishimine T, Nakayama I, Yagi N, Wake M, Takahashi T, Taniguchi N, Tengan T. Unilateral left pulmonary edema caused by contained rupture of the ascending aortic dissection. Intern Med. 2021;60(5):751–3.
11. Kasmani R, Irani F, Okoli K, Mahajan V. Re-expansion pulmonary edema following thoracentesis. CMAJ. 2010;182(18):2000–2.
12. Keyl C, Siepe M. Unilateral lung injury after minimally invasive cardiac surgery: more questions than answers. Eur J Cardiothorac Surg. 2016;49(2):505–6.
13. Irisawa Y, Hiraoka A, Totsugawa T, Chikazawa G, Nakajima K, Tamura K, Yoshitaka H, Sakaguchi T. Re-expansion pulmonary oedema after minimally invasive cardiac surgery with right mini-thoracotomy. Eur J Cardiothorac Surg. 2016;49(2):500–5.
14. Sue RD, Matthay MA, Ware LB. Hydrostatic mechanisms may contribute to the pathogenesis of human re-expansion pulmonary edema. Intensive Care Med. 2004;30(10):1921–6.
15. Boyle EM Jr, Pohlman TH, Cornejo CJ, Verrier ED. Endothelial cell injury in cardiovascular surgery: ischemia-reperfusion. Ann Thorac Surg. 1996;62(6):1868–75.
16. Huffmyer JL, Groves DS. Pulmonary complications of cardiopulmonary bypass. Best Pract Res Clin Anaesthesiol. 2015;29(2):163–75.
17. Jaworski K, Maślanka K, Kosior DA. Transfusion-related acute lung injury: a dangerous and underdiagnosed noncardiogenic pulmonary edema. Cardiol J. 2013;20(4):337–44.
18. Silliman CC, Ambruso DR, Boshkov LK. Transfusion-related acute lung injury. Blood. 2005;105(6):2266–73.
19. Peters AL, Van Stein D, Vlaar AP. Antibody-mediated transfusion-related acute lung injury; from discovery to prevention. Br J Haematol. 2015;170(5):597–614.
20. Tonna JE, Abrams D, Brodie D, Greenwood JC, Rubio Mateo-Sidron JA, Usman A, Fan E. Management of adult patients supported with venovenous extracorporeal membrane oxygenation (VV ECMO): guideline from the extracorporeal life support organization (ELSO). ASAIO J. 2021;67(6):601–10.
21. Peek GJ, Mugford M, Tiruvoipati R, Wilson A, Allen E, Thalanany MM, Hibbert CL, Truesdale A, Clemens F, Cooper N, Firmin RK, Elbourne D, CESAR trial collaboration. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. Lancet. 2009;374(9698):1351–63.
22. Combes A, Schmidt M, Hodgson CL, Fan E, Ferguson ND, Fraser JF, Jaber S, Pesenti A, Ranieri M, Rowan K, Shekar K, Slutsky AS, Brodie D. Extracorporeal life support for adults with acute respiratory distress syndrome. Intensive Care Med. 2020;46(12):2464–76.

Publisher's Note