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CASE REPORT

Right Ventricular Failure in a Patient Supported with VV-ECMO: Implications and Therapeutic Strategies

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Abstract

A 46-year-old woman was admitted to the intensive care unit with severe acute respiratory distress syndrome (ARDS) due to Legionella pneumonia. Refractory hypoxemia developed despite conventional lung-protective mechanical ventilation and the patient was managed with veno-venous extracorporeal membrane oxygenation. Subsequent right ventricular (RV) failure developed and was successfully treated with a percutaneously inserted right ventricular assist device attached to an oxygenator. This report discusses the pathophysiology, diagnosis, and treatment of RV failure as a complication of ARDS in the context of the current literature and provides a rationale for the use of mechanical circulatory support devices in this setting.

Introduction

RV dysfunction complicates 22-50% of acute respiratory distress syndrome (ARDS) cases and contributes to increased mortality [1]. Treatment with veno-venous extracorporeal membrane oxygenation (VV-ECMO) has been shown to improve pulmonary hemodynamics [2,3] and a large randomized controlled study of ARDS patients demonstrated that duration of vasopressor use was shorter in the VV-ECMO group [4]. As VV-ECMO therapy alone does

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not directly unload the right ventricle (RV), a right ventricular support device that is coupled to an oxygenator and can bypass the RV may be beneficial in cases where severe RV dysfunction complicates ARDS. In this report, a case is presented and used to discuss the pathophysiology, diagnosis, and treatment of RV failure as a complication of ARDS in the context of the current literature and presents a clinical algorithm to guide the use of mechanical circulatory support devices in this setting.

Case Report

A 46-year-old woman with a past medical history significant for scleroderma-related interstitial lung disease was transferred to an ECMO-capable intensive care unit (ICU) with a diagnosis of Legionella pneumonia. She was not known for prior cardiovascular disease and her most recent estimated systolic pulmonary artery pressure (PAP) by transthoracic echocardiography, a year prior, was 34 mmHg.

On the day of her transfer, she was intubated due to worsening hypoxemia. Despite optimization of conventional mechanical ventilation her oxygen saturation (SpO_2) remained below 80%. Upon arrival, the PaO_2/FiO_2 ratio was 86 mmHg while paralyzed and ventilated with a pressure-controlled setting targeting a tidal volume of 6 ml/kg predicted body weight with a positive end-expiratory pressure (PEEP) of 10 cmH_2O and a fraction of inspired oxygen of 1. Due to refractory hypoxemia from Legionella related ARDS and the rapidity of deterioration, VV-ECMO support was implemented. A 31 Fr double-lumen Avalon catheter (Getinge, Gothenburg, Sweden) was inserted via the right internal jugular vein. The patient suffered a cardiac arrest necessitating extracorporeal cardiopulmonary resuscitation (eCPR). A 19 Fr arterial return cannula was advanced into the left femoral artery for veno-arterial (VA-ECMO) support and a 21 Fr venous cannula was placed in the right femoral vein for additional drainage. Return of spontaneous circulation was achieved within 10 minutes after her oxygenation improved with extracorporeal support.

Periprocedural echocardiography after resuscitation demonstrated normal biventricular systolic function. Gradually, VA-ECMO was weaned along with vasopressors resulting in conversion to VV-ECMO 32 hours later. Due to the extensive pneumonia and severe intrapulmonary shunting, oxygenation was persistently poor, requiring VV-ECMO flows up to 5.4 L/min to maintain a PaO_2 above 70 mmHg. A target PCO_2 of < 40 mmHg was maintained with a sweep gas flow of 8 L/min.

After 150 hours of VV-ECMO support, her mean arterial pressure decreased < 60 mmHg that was treated with a norepinephrine infusion. Concomitantly, the central venous pressure (CVP) rose to 20 mmHg and metabolic acidosis with high lactate developed. Transesophageal echocardiography revealed a dilated, severely hypokinetic RV whereas the LV was small with a normal systolic function. A decision was made to replace the existing Avalon double lumen cannula with a Protek Duo cannula (TandemLife, Pittsburgh, PA, USA) in series with an oxygenator. Increased intravascular volume was treated with diuretics while milrinone, inhaled nitric oxide (iNO) and sildenafil were utilized to address increased pulmonary vascular resistance (PVR). Gradually the blood pressure and lactate improved, and vasoactive agents were discontinued. Oxygenation and ventilation also improved leading to cessation of iNO after 4 days and milrinone after 9 days. Blood flow and sweep gas were slowly waned and ECMO support was stopped 16 days after the catheter change. The patient was transferred to her community hospital for further rehabilitation one month later. A summary of the patient's ventilatory, hemodynamic, and ECMO parameters over the course of hospitalization is presented in **Table 1**.

Table 1 - Details of hemodynamics and respiratory measurements 1/2

	ECMO				Respiratory Support				Hemodynamics					Arterial Blood Gas		
	Mode	ECBF	SGF	FdO_2	Mode	PIP	PEEP	FiO_2 Vent	CVP	SBP	DBP	MAP	HR	SpO_2	PaO_2	$PaCO_2$
Day 0	VA	4.16	5	1	PC	26	10	0.7	15	113	65	81	80	85%	60.6	45
Day 1		5.35	7	0.65		21	15	0.4	17	114	70	85	81	97%	69.9	35

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Table 1 - Details of hemodynamics and respiratory measurements 2/2

	ECMO				Respiratory Support				Hemodynamics					Arterial Blood Gas		
	Mode	ECBF	SGF	FdO_2	Mode	PIP	PEEP	FiO_2 Vent	CVP	SBP	DBP	MAP	HR	SpO_2	PaO_2	$PaCOO_2$
Day 2	VV	5.3	5	0.8	PC	22	15	0.4	19	106	67	80	91	99%	135	37
Day 3		5.4	5	0.7	HFNC	22	15	0.4	6	121	69	86	104	100%	146	33
Day 4		3.3	3.75	0.65		22	15	0.4	10	126	76	93	96	95%	70	38
Day 5		3.7	4	0.8		22	15	0.7	10	143	81	102	91	95%	75	45
Day 6		5.48	8	100		22	15	0.7	3	108	63	78	116	93%	75.9	41
Day 7	ProTek Duo	5.42	8	100		22	15	0.5	9	112	61	78	96	91%	61.2	35.6
Day 8		4.39	4	0.8	CPAP	15	15	0.3	14	108	68	81	68	100%	184	33.1
Day 9		4.64	5	0.8		14	15	0.3	15	108	66	80	115	99.4	134	36.7
Day 10		4.48	6.5	0.8		20	20	0.3	12	103	56	72	83	99.9	146	38.6
Day 11		4	2.5	0.6		24	20	0.3	6	107	58	74	69	93.9	71.5	61.4
Day 12		4.5	3.5	0.8	PSV	31	15	0.6	12	81	51	61	58	100	180	45
Day 13		4.57	4.5	0.6		32	20	0.5	12	117	58	78	88	99.1	98.5	48.7
Day 14		4.43	3	0.6		30	12	0.5	6	98	58	71	119	92	58	64
Day 15		4.38	3.5	0.5		30	12	0.6	8	127	64	85	74	97.9	79	49
Day 16		3.8	3.5	0.6		30	12	0.5	8	148	66	93	93	96	65	46
Day 17		3.75	5	0.7		31	12	0.5	NA	112	53	73	97	97.9	93.5	36.2
Day 18		3.63	5	0.7		30	12	0.5	NA	114	54	74	96	99	86.7	35.7
Day 19		3.16	5	0.7		30	12	0.5	NA	103	57	72	100	94.7	59.9	37.9
Day 20		2.99	5	0.7		29	12	0.5	NA	114	55	75	102	98.8	91.5	36.5
Day 21		2.74	3	0.4		29	12	0.5	NA	115	59	78	109	99.7	128	36.9
Day 22		2.78	5	0.3		30	12	0.5	NA	138	70	93	101	94	63	26
Day 23	Decannulation	2.63	0	0.21		22	10	0.5	13	113	57	76	116	98.3	90	47

Abbreviations:

ECMO = extracorporeal membrane oxygenation, ECBF = extracorporeal membrane oxygenation blood flow, SGF = sweep gas flow, FdO_2 = fraction of delivered oxygen (through ECMO circuit), PIP = peak inspiratory pressure, PEEP = positive end-expiratory pressure, FiO_2 = fraction of inspired oxygen, CVP = central venous pressure, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, HR = heart rate, VA = veno-arterial ECMO, VV = veno-venous ECMO, PC = pressure control, HFNC = high flow nasal cannula, CPAP = continuous positive airway pressure, PSV = pressure support ventilation.

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Comment

In patients with severe ARDS, hemodynamic instability is frequently attributed to septic shock. However, many patients develop hemodynamic collapse secondary to right ventricular (RV) dysfunction and/or limitation secondary to increased intravascular volume and RV afterload [5]. Based on her medical history of interstitial lung disease, this patient had risk factors for pulmonary hypertension and RV dysfunction and the development of severe ARDS further contributed to hemodynamic deterioration [1].

Patients with ARDS are at risk for the development of RV dysfunction due to a variety of mechanisms primarily altering PVR and resulting in increased RV afterload. At the level of the pulmonary arterioles, hypoxemia and hypercapnia contribute to vasoconstriction. In addition, inflammation, microvascular thrombosis and activation of endothelial cells result in intravascular occlusion, further increasing PVR [6]. Finally, extrinsic compression of the pulmonary vasculature may occur due to the application of increased airway and transpulmonary pressure, further afterloading the RV. When alveolar pressure exceeds pulmonary venous pressure, West zone 1 and 2 (non-zone 3) conditions are favored [7]. In non-zone 3 lung units, the down-stream pressure to RV ejection is no longer left atrial pressure, but rather alveolar pressure [7-9]. The impact of mechanical ventilation on right ventricular afterload is increased in the setting of reduced lung compliance, when transpulmonary pressures are higher (as in ARDS) or when left atrial pressure is low, as can occur during RV limitation or hypovolemia [10, 11].

RV dysfunction is the representation of an impaired end systolic pressure volume relationship and is caused by a reduction in contractility or increased in afterload [5]. Compared to the LV, the RV operates at lower pressures and is more sensitive to changes in afterload. In response to an increase in afterload, the RV will increase end-diastolic volume and dilate to maintain stroke volume [11]. The RV end-diastolic pressure volume relationship has an abrupt increase when preload is maximal. When preload is maximal, RV limitation has occurred. The clinical implication is that volume loading at that point will only increase CVP without any rise in cardiac output and any increase in afterload will result in a direct decrease in stroke volume. A combination of RV dysfunction and limitation can lead to RV failure which can be diagnosed when there are clinical signs of systemic congestion and hypoperfusion. Demonstration of an elevated CVP greater than pulmonary artery occlusion pressure with low cardiac index also supports the diagnosis of RV failure. Echocardiography is frequently utilized to evaluate RV function. Echocardiographic signs that suggest RV dysfunction/limitation include low global systolic function, paradoxical motion of the interventricular septum, and an increase in the RV end diastolic dimension to LV end diastolic dimension ratio [12].

High driving pressure, high $PaCO_2$ and low PaO_2/FiO_2 ratio are independent risk factors for right ventricular dysfunction in the setting of ARDS [13]. When these risk factors are present, serial monitoring of surrogates of RV function such as systolic PAP, CVP and echocardiographic parameters can help in early identification and management. A thorough review of this topic is provided elsewhere [5]. Medical management includes optimizing RV preload by carefully limiting fluid loading and judicious diuresis, increasing RV contractility with inotropic agents such as milrinone or dobutamine, and reducing afterload with pulmonary vasodilators and ventilation strategies that limit plateau and driving pressure [14], while aiming for normalization of blood gases [1, 11, 15, 16]. VV-ECMO may reduce the mean PAP and PVR by lowering the PCO_2 and improving oxygenation [3], but it does not directly unload the RV [2] (**Figure 1**). Conversion to a venous-arterial-venous (VAV) circuit allows for bypass of both the LV and RV but adds more risk and complexity.

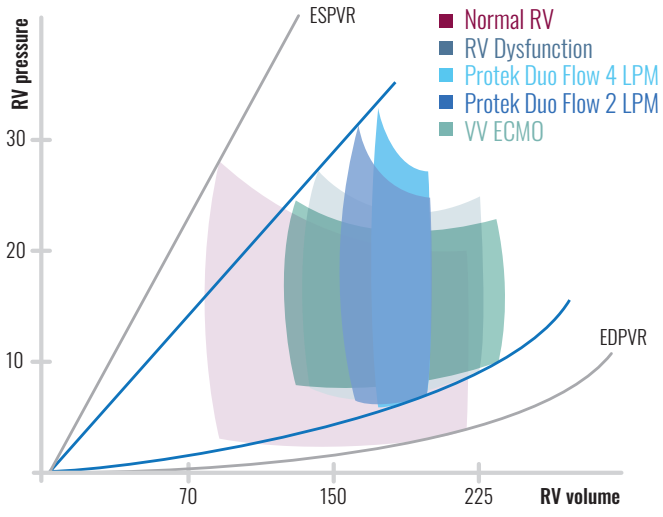

Figure 1. Pressure-volume loops of the right ventricle. The normal right ventricle is compared to right ventricular dysfunction with and without VV-ECMO and the Protek Duo device at different flows. ESPVR = end systolic pressure volume relationship; EDPVR = end diastolic pressure volume relationship.

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The TandemLife Protek Duo is a dual lumen 29 Fr catheter that can be percutaneously inserted via the right internal jugular vein (**Figure 2**).

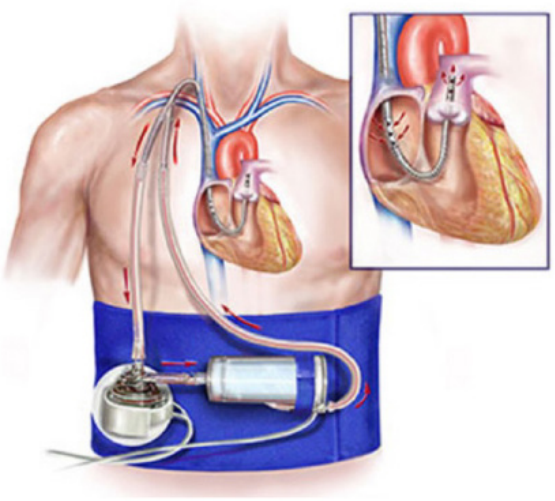

Figure 2. TandemLife Protek Duo device illustrating insertion via the right internal jugular vein into the right ventricle demonstrating bypass of blood flow from the right ventricle into the main pulmonary artery.

The proximal lumen functions as an inflow cannula and is positioned in the right atrium. The distal lumen has a multifenestrated tip and is positioned in the main pulmonary artery before the bifurcation. Blood is drained from the right atrium into the extracorporeal pump allowing flows up to 5 L/min and is returned to the main pulmonary artery via a distal lumen bypassing the RV. An oxygenator can be connected to the circuit for ECMO support to provide oxygenation and CO_2 removal, decreasing PVR and unloading the RV. The device decreases RV preload and stroke volume but also increases afterload by increasing PAP. The increase in afterload is balanced by the increase in oxygen content of the mixed venous blood and CO_2 removal provided by the oxygenator, potentially improving RV hemodynamics overall. The main disadvantage is that the cannula needs to be inserted under fluoroscopy and the single lumen configuration may limit the total flow delivered. Considering the TandemLife ProTek Duo in a situation where there is isolated RV failure will be more beneficial to the RV and less invasive than converting to a VAV circuit. A decision algorithm incorporating this device into the care of such patients is presented in **Figure 3**.

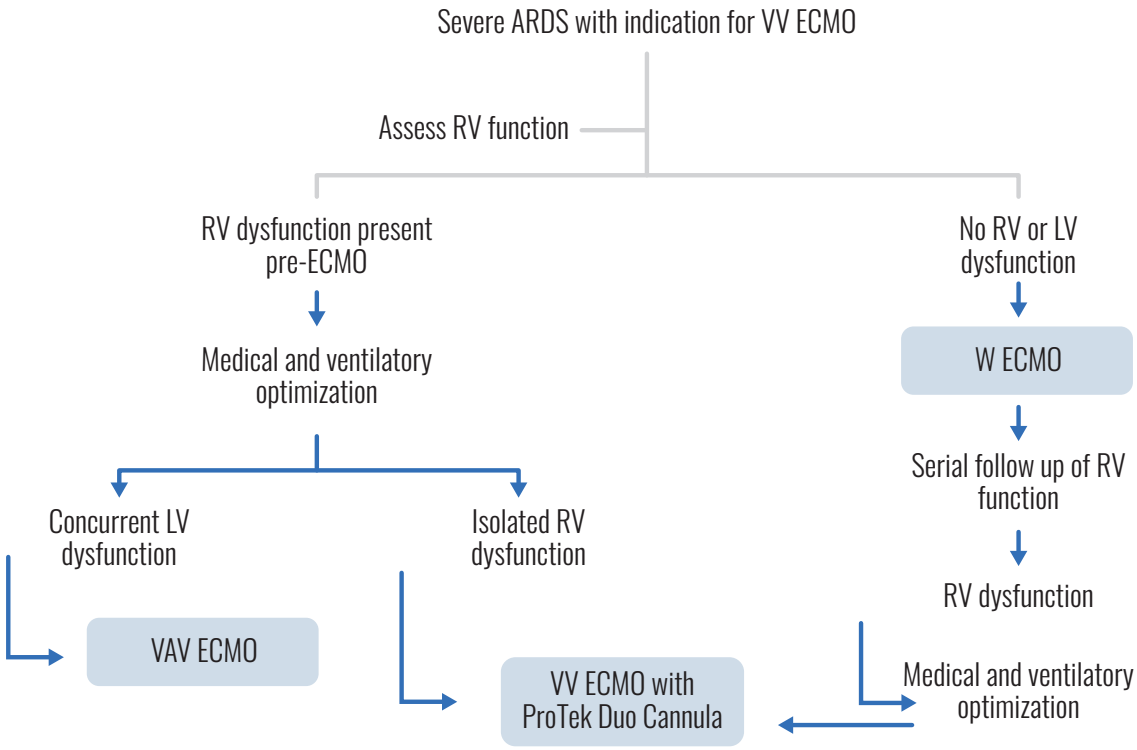

Figure 3. Decision algorithm for mechanical support of RV dysfunction in ARDS. ARDS = acute respiratory distress syndrome, VV-ECMO = veno-venous extracorporeal membrane oxygenation, RV = right ventricle, LV = left ventricle, VAV-ECMO= Veno-arterial-venous extracorporeal membrane oxygenation

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References

1. Zochios V, Parhar K, Tunnicliffe W, Roscoe A, Gao F. The Right Ventricle in ARDS. Chest 2017; 152(1):181-193.
2. Reis Miranda D, van Thiel R, Brodie D, Bakker J. Right ventricular unloading after initiation of venovenous extracorporeal membrane oxygenation. Am J Respir Crit Care Med 2015; 191(3):346-348.
3. Spinelli E, Slobod D, Giani M, Di Pierro M, Zhao Z, Lissoni A, Tubiolo D, Tagliabue P, Grasselli G, Foti G et al: ESICM LIVES 2022: part 1. Abstract: Physiologic impact of increasing ECMO blood fow rate in severe ARDS patients. Intensive Care MedicineExperimental 2022; 10(2):39.
4. Combes A, Hajage D, Capellier G, Demoule A, Lavoué S, Guervilly C, Da Silva D, Zafrani L, Tirot P, Veber B et al. Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome. N Engl J Med 2018; 378(21):1965-1975.
5. Magder S, Slobod D, Assanangkornchai N. Right Ventricular Limitation: A Tale of Two Elastances. Am J Respir Crit Care Med 2022.
6. Price LC, McAuley DF, Marino PS, Finney SJ, Griffiths MJ, Wort SJ. Pathophysiology of pulmonary hypertension in acute lung injury. Am J Physiol Lung Cell Mol Physiol 2012; 302(9): L803-815.
7. Permutt S, Bromberger-Barnea B, Bane HN. Alveolar pressure, pulmonary venous pressure, and the vascular waterfall. Med Thorac 1962; 19:239-260.
8. Brower R, Wise RA, Hassapoyannes C, Bromberger-Barnea B, Permutt S. Effect of lung inflation on lung blood volume and pulmonary venous flow. J Appl Physiol 1985; 58(3):954-963.
9. Howell JB, Permutt S, Proctor DF, Riley RL. Effect of inflation of the lung on different parts of pulmonary vascular bed. J Appl Physiol. 1961; 16:71-76.
10. Repessé X, Vieillard-Baron A, Geri G: Value of measuring esophageal pressure to evaluate heart-lung interactions applications for invasive hemodynamic monitoring. Ann Transl Med 2018; 6(18):351.
11. Repessé X, Vieillard-Baron A: Right heart function during acute respiratory distress syndrome. Ann Transl Med 2017; 5(14):295.
12. Harjola VP, Mebazaa A, Čelutkienė J, Bettex D, Bueno H, Chioncel O, Crespo-Leiro MG, Falk V, Filippatos G, Gibbs S et al. Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology. Eur J Heart Fail 2016; 18(3):226-241.
13. Mekontso Dessap A, Boissier F, Charron C, Bégot E, Repessé X, Legras A, Brun-Buisson C, Vignon P, Vieillard-Baron A. Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact. Intensive Care Med 2016; 42(5):862-870.
14. Slobod D, Assanangkornchai N, Alhazza M, Mettasittigorn P, Magder S. Right Ventricular Loading by Lung Inflation During Controlled Mechanical Ventilation. Am J Respir Crit. Care Med 2022.
15. Petit M, Jullien E, Vieillard-Baron A. Right Ventricular Function in Acute Respiratory Distress Syndrome: Impact on Outcome, Respiratory Strategy and Use of Veno-Venous Extracorporeal Membrane Oxygenation. Frontiers in Physiology 2022; 12.
16. Jardin F, Vieillard-Baron A. Is there a safe plateau pressure in ARDS? The right heart only knows. Intensive Care Med 2007; 33(3):444-447.