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Case Report Article

Beyond the Lungs: Viral Pneumonia Triggering ARDS and Myocarditis Rescued by ECMO

Kapil Khanna[1,2], Rupanshi Madan[3], Aayush Goyal[4], Rinku Garg[5], Rohit Saroha[5]

[1]Physiology, Santosh Deemed to be University, Ghaziabad, Delhi NCR, IND
[2]Cardiology, Yashoda Superspeciality Hospital, Kaushambi, Delhi NCR, IND
[3]Pulmonary Medicine, Yashoda Superspeciality Hospital, Kaushambi, Ghaziabad, Delhi NCR, IND
[4]Cardiothoracic Surgery, Yashoda Superspeciality Hospital, Kaushambi, Ghaziabad, Delhi NCR, IND
[5]Physiology, Santosh deemed to be university, Ghaziabad, IND

Corresponding Author:

Kapil Khanna
Physiology, Santosh Deemed to be University, Ghaziabad, Delhi NCR, IND

Email:
kapil76khanna@gmail.com

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ABSTRACT

Viral pneumonias can extend beyond pulmonary involvement, triggering multisystem complications such as acute respiratory distress syndrome (ARDS) and fulminant myocarditis. ARDS is a recognized complication of severe viral pneumonia, whereas concomitant acute viral myocarditis with cardiogenic shock remains uncommon. These combined presentations are often refractory to conventional critical care measures, posing significant therapeutic challenges and contributing to high mortality rates. Extracorporeal membrane oxygenation (ECMO) has become an advanced life-support strategy for such critically ill patients, providing temporary cardiopulmonary assistance, especially when maximal conservative therapies fail.

Extracorporeal membrane oxygenation (ECMO), particularly veno-venous (VV) ECMO, is established for refractory ARDS, although its role in combined pulmonary-cardiac viral injury is less frequently documented.

Keywords: *Pneumonia, ARDS, Myocarditis, ECMO.*

INTRODUCTION

Viral pneumonias are increasingly recognized as systemic illnesses that extend beyond isolated pulmonary involvement, precipitating complications such as acute respiratory distress syndrome (ARDS) and fulminant myocarditis [1,2]. ARDS is a well-established consequence of severe viral pneumonia; however, concomitant acute viral myocarditis with hemodynamic compromise or cardiogenic shock remains relatively uncommon and is associated with substantial morbidity and mortality [3]. These combined pulmonary-cardiac presentations are frequently refractory to conventional intensive care measures, including lung-protective mechanical ventilation and standard hemodynamic support, thereby necessitating escalation to advanced organ support modalities [4].

Extracorporeal membrane oxygenation (ECMO) has emerged as a pivotal rescue therapy for selected patients with severe, potentially reversible respiratory or cardiopulmonary failure when maximal conservative therapies fail [5,6]. In particular, veno-venous (VV) ECMO is an established modality for refractory ARDS, yet its role in severe viral pneumonia with concurrent myocardial involvement is less frequently reported, especially in young patients without prior comorbidities [7]. In this context, we present a rare case of influenza B-associated viral pneumonia complicated by severe ARDS and evolving acute myocarditis in a previously healthy young adult, successfully managed with timely VV ECMO support [8].

CASE PRESENTATION

We report a rare case of a previously healthy 28-year-old female diagnosed with influenza B-associated viral pneumonia leading rapidly to severe ARDS, multi-organ dysfunction, and evolving acute myocarditis. She presented with high-grade fever, productive cough, progressive dyspnea, and hypoxemia refractory to conventional oxygen therapy. Chest imaging revealed extensive bilateral ground-glass opacities and consolidations consistent with diffuse alveolar damage due to viral pneumonia [8,9]. Laboratory findings showed elevated inflammatory markers, including C-reactive protein (CRP),

procalcitonin (PCT), and ferritin, alongside rising cardiac troponins suggestive of myocardial injury. High-resolution computed tomography (HRCT) of the chest demonstrated multifocal bilateral ground-glass opacities and consolidations affecting all lobes, consistent with viral pneumonia and evolving ARDS [8,9].

Echocardiographic assessment revealed reduced left ventricular ejection fraction (LVEF 35-40%) with global hypokinesia, consistent with acute myocarditis but without overt cardiogenic shock at presentation [8,9]. She was managed conservatively with broad-spectrum intravenous antibiotics, antivirals, corticosteroids, and oxygen support through high-flow nasal cannula (HFNC), non-invasive ventilation (NIV), and subsequent invasive lung-protective mechanical ventilation using low tidal volumes and high positive end expiratory pressure (PEEP) along with prone positioning [4]. Despite these measures, the patient developed severe ARDS with a worsening oxygenation index (PaO_2/FiO_2 ratio < 80 mm Hg), hemodynamic compromise, and hypercapnia [8,9]. Given the refractory hypoxemia and anticipated reversibility of myocarditis, the multidisciplinary team initiated veno-venous ECMO. This was instituted to provide extracorporeal oxygenation and carbon dioxide removal, facilitating ultra-protective mechanical ventilation. Cannulation was performed using a two-site approach with a drainage cannula inserted into the femoral vein and a reinfusion cannula placed in the right internal jugular vein [8] permitting adequate blood flow and gas exchange without the need for a dual lumen catheter. Systemic anticoagulation with unfractionated heparin was commenced per ECMO protocol to prevent circuit thrombosis [2] and maintain adequate ECMO flow.

Activated clotting time (ACT) was monitored serially targeting it between 180 and 220 seconds to balance the antithrombotic efficacy and bleeding risk, a critical determinant of ECMO success in cardiovascular patients.

Post VV ECMO initiation, there was rapid improvement in arterial oxygenation, with the PaO_2/FiO_2 ratio rising above 200 within the first 12 hours. This improvement allowed ultra-protective ventilation with reduced tidal volumes (<4 mL/kg) and low respiratory rates to facilitate lung rest and minimize barotrauma [4]. Concurrently, continuous cardiac monitoring and serial echocardiograms demonstrated incremental recovery of myocardial function, supported by downward trending cardiac enzymes, aligning with resolution of myocarditis [4]. Throughout the 6-day ECMO course, monitoring continued for complications including bleeding, thrombosis, and infection. Regular imaging and laboratory investigations confirmed progressive resolution of pulmonary infiltrates (Figure *1* and Figure *2*) and normalization of systemic inflammatory and metabolic parameters. The patient was successfully weaned off VV ECMO following restoration of adequate lung function and gas exchange, evidenced by radiological improvement and PaO_2/FiO_2 ratio > 300 on minimal ventilatory support. After decannulation, the patient was extubated and continued rehabilitation. At discharge, full functional recovery was observed with no residual cardiopulmonary deficits.

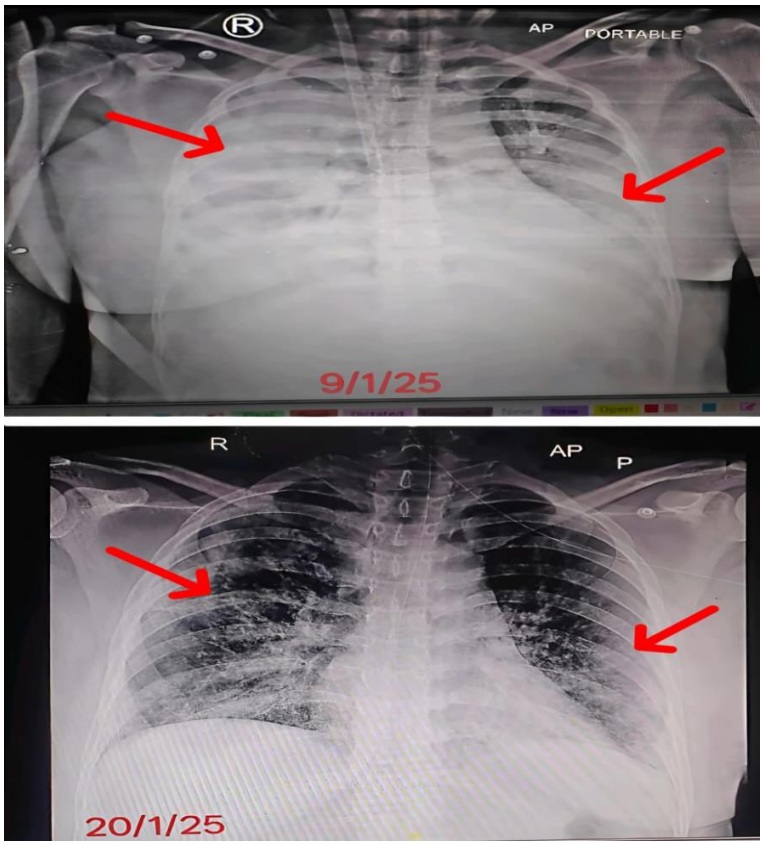

FIGURE 1: Chest X-Ray (9/1/25-20/1/25)

09.01.2025-Chest X ray- Haziness in right lung field and left lower zone with obscured both Costo-Phrenic angle.

20.01.25 Chest X-Ray - Inhomogeneous opacities in right middle and lower zone and left Lower zone**.**

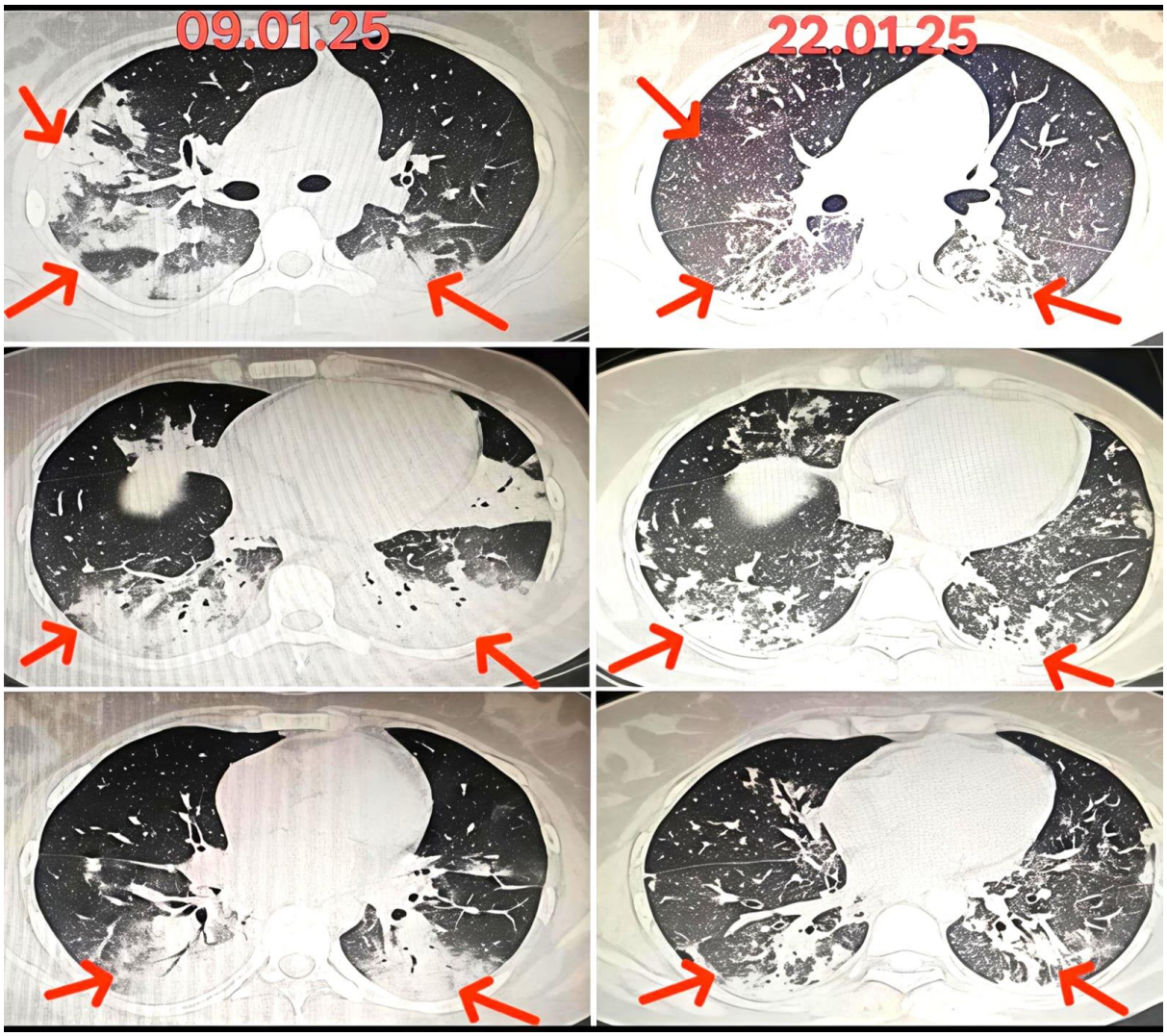

FIGURE 2: HRCT Chest (09.01.25 and 22.01.25)

09.01.25 - HRCT Chest – Bilateral lung fields show extensive multi-focal airspace consolidation showing air bronchograms with surrounding ground glass haze – s/o extensive multi-focal pneumonitis. 22.01.2025 HRCT-Chest - Partial resolution of the previous lesions.

DISCUSSION

This case highlights the complex and rapid systemic impact of severe viral respiratory infections, which may extend beyond the lungs to involve the myocardium, resulting in a combined syndrome of ARDS and acute viral myocarditis. Such dual organ involvement complicates clinical management, increases mortality risk, and challenges conventional ICU supportive care strategies. Viral pneumonia-related myocarditis, though relatively uncommon, is increasingly recognized in severe respiratory viral infections, including influenza and SARS-CoV-2 [7,8]. The myocarditis contributes to cardiac dysfunction, worsens hemodynamics, and potentiates hypoxemia by impairing pulmonary circulation and right heart function. Early recognition through clinical, biochemical, and echocardiographic evaluation is critical to guide therapeutic decisions [9].

Extracorporeal membrane oxygenation (ECMO) has evolved as a pivotal rescue therapy for patients with refractory hypoxemia due to ARDS and those with myocardial impairment unresponsive to standard care. Veno-venous (VV) ECMO provides effective extracorporeal gas exchange support, allowing lung-protective, ultra-low volume ventilation strategies that mitigate ventilator-induced lung injury and promote alveolar healing [5]. In this mode, oxygenated blood is returned to the venous circulation, reducing pulmonary vascular resistance and right ventricular afterload, which may indirectly benefit myocardial function [9]. The success of ECMO in viral pneumonia-associated ARDS was notably demonstrated during the 2009 H1N1 influenza pandemic, where survival rates improved significantly in patients receiving ECMO compared to conventional ventilation alone [5,7]. More recent experiences during the COVID-19 pandemic further support the selective use of VV ECMO in severe ARDS, with survival influenced strongly by careful patient selection, timing of initiation, and the experience of the ECMO center [9]. Myocardial recovery in viral myocarditis supported by ECMO is facilitated by reduced cardiac workload and normalization of oxygen delivery, which decreases myocardial ischemia and inflammation. While venoarterial (VA) ECMO provides direct cardiac support, VV ECMO can still contribute significantly

by improving oxygenation, decreasing right ventricular afterload, and enabling myocardial rest when hemodynamics is relatively preserved. In this patient, the stable hemodynamics and absence of overt cardiogenic shock made VV ECMO an appropriate modality. A multidisciplinary approach is essential to optimize ECMO outcomes. Collaboration among intensivists, cardiologists, pulmonologists, infectious disease specialists, perfusionists, and specialized nursing teams ensures comprehensive patient management. This includes strict anticoagulation management, prevention and early detection of ECMO-related complications, nutritional and rehabilitation support, and vigilant monitoring for organ recovery [9].

Despite its benefits, ECMO is associated with potential complications including bleeding, thrombosis, infection, and technical issues such as cannula malposition. These risks underscore the necessity for rigorous patient selection, timely initiation before irreversible multiorgan failure ensues, and care in experienced centers to minimize adverse events [9].In summary, this case illustrates that timely VV ECMO can be life-saving in severe viral pneumonia complicated by ARDS and myocarditis, by enabling lung and myocardial recovery through extracorporeal gas exchange and cardio-respiratory supportive measures [10]. Continued research into ECMO indications, timing, and multidisciplinary care protocols is vital to enhance survival and functional recovery in this critically ill population. Extracorporeal membrane oxygenation has increasingly been used in severe cardiopulmonary failure to provide temporary respiratory and circulatory support while allowing recovery of the underlying pathology [11].

In the present case, veno-venous extracorporeal membrane oxygenation (VV-ECMO) was selected instead of veno-arterial ECMO because the patient primarily exhibited refractory hypoxemic respiratory failure with relatively preserved hemodynamics despite evidence of myocardial involvement. VV-ECMO provided adequate extracorporeal gas exchange and allowed implementation of ultra-protective ventilation strategies while facilitating myocardial recovery through improved oxygen delivery and reduced cardiopulmonary stress.

This case highlights the importance of early recognition of cardiac involvement in severe viral pneumonia and demonstrates the life-saving potential of timely VV-ECMO support in carefully selected patients with refractory hypoxemia.

CONCLUSION

In severe viral pneumonia complicated by refractory ARDS and myocarditis, veno-venous ECMO serves as a critical, life-saving intervention by providing extracorporeal respiratory support without the need for direct cardiac circulatory assistance. VV ECMO facilitates profound improvements in oxygenation and carbon dioxide removal, enabling lung-protective ventilation strategies that reduce ventilator-induced lung injury and promote pulmonary recovery. Moreover, by improving systemic oxygen delivery and reducing right.

ventricular afterload, VV ECMO indirectly supports myocardial function, which is particularly valuable in viral myocarditis cases without overt cardiogenic shock. This case highlights the importance of early recognition and timely initiation of VV ECMO in selected patients, reinforcing its role as a vital component of advanced supportive care in complex viral cardiorespiratory failure. The expanding use and evidence base for VV ECMO underscore the need for multidisciplinary expertise and careful patient selection to optimize outcomes in this high-risk population.

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