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Viral Myocarditis Presenting as ARDS and Cardiogenic Pulmonary Edema: Diagnostic Challenges and Therapeutic Implications: A Case Report

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Abstract **Case Report**

Viral myocarditis is a major cause of acute heart failure, with a heterogeneous clinical presentation ranging from asymptomatic forms to fulminant, life-threatening cases. Its presentation as an unusual combination of acute respiratory distress syndrome (ARDS) and cardiogenic acute pulmonary edema (APE) poses a major diagnostic challenge, particularly in the intensive care setting. This situation carries a risk of delayed diagnosis and treatment. This article analyzes the pathophysiological mechanisms, diagnostic challenges, and multidisciplinary management strategies for this severe form of viral myocarditis.

Keywords: Viral myocarditis, ARDS, cardiogenic acute pulmonary edema, cardiogenic shock, intensive care.

INTRODUCTION

Viral myocarditis is an inflammation of the myocardium secondary to infection by viruses with cardiac tropism (enteroviruses, parvovirus B19, HHV6, adenoviruses, myxoviruses, influenza viruses, SARS-CoV-2). It is a major cause of acute heart failure in young adults and individuals with no known cardiovascular history. It can cause direct myocardial damage or damage mediated by an exacerbated immuno-inflammatory response [1, 2]

It can progress to a fulminant form characterized by acute heart failure, cardiogenic shock, and sometimes ARDS, resulting in polymorphic clinical presentations [3]

The coexistence of ARDS and cardiogenic pulmonary edema is rare and can lead to diagnostic delay, particularly when the initial presentation is dominated by respiratory distress. This situation poses diagnostic and therapeutic challenges in the emergency setting[4, 5]

We report a case illustrating this complex diagnostic scenario in the intensive care unit.

PATIENT AND METHOD

The patient was a 24-year-old woman, married and the mother of one child, with a history of appendectomy 4 years prior. The patient presented to the emergency department with exertional dyspnea; her symptoms had begun one month prior with a productive cough accompanied by two episodes of minor hemoptysis, and her condition had progressively worsened with the onset of exertional dyspnea classified as NYHA Class 3.

The clinical examination in the emergency department revealed that the patient was alert and afebrile, blood pressure of 95/50 mmHg, heart rate of 108 beats per minute, respiratory rate of 30 breaths per minute with oxygen saturation of 86% on room air; the pulmonary examination revealed basal dullness and bilateral crackles.

The chest CT scan showed pulmonary parenchymal abnormalities consisting of ground-glass opacities, both central and peripheral, with nodular consolidations in places, creating a “crazy paving” appearance, suggestive of alveolar hemorrhage; bilateral pleural effusions, moderate on the right and mild on the left, of fluid density, with passive consolidation opposite (**Figure 1**).

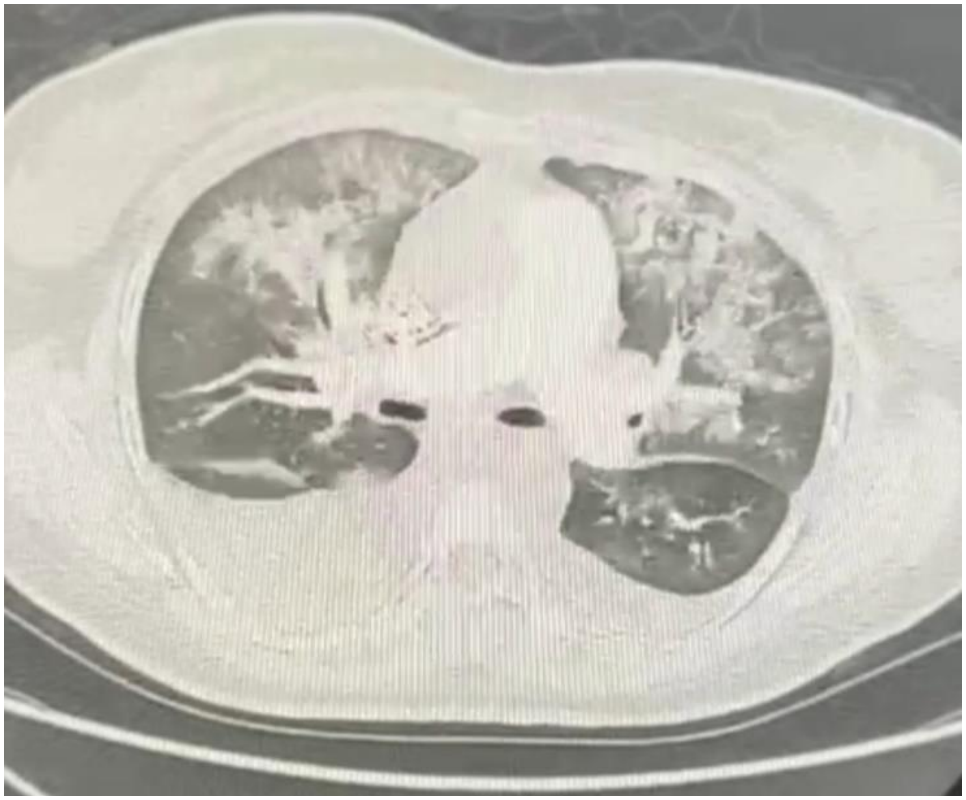

Figure 1: Chest CT scan showing pulmonary parenchymal abnormalities consisting of central and peripheral ground-glass opacities, creating a "crazy paving" appearance with bilateral pleural effusions of fluid density

On cardiac evaluation, the ECG revealed a regular sinus rhythm, HR 101, left-axial heart, circumferentially inverted T waves, and echocardiography showed a dilated LV with impaired systolic function, LVEF 28%, normal filling pressures, non-dilated right atrium, minimal myocardial infarction, no pulmonary hypertension, non-dilated right chambers, compliant inferior vena cava, and pericardial effusion (**Figure 2 and Figure 3)**

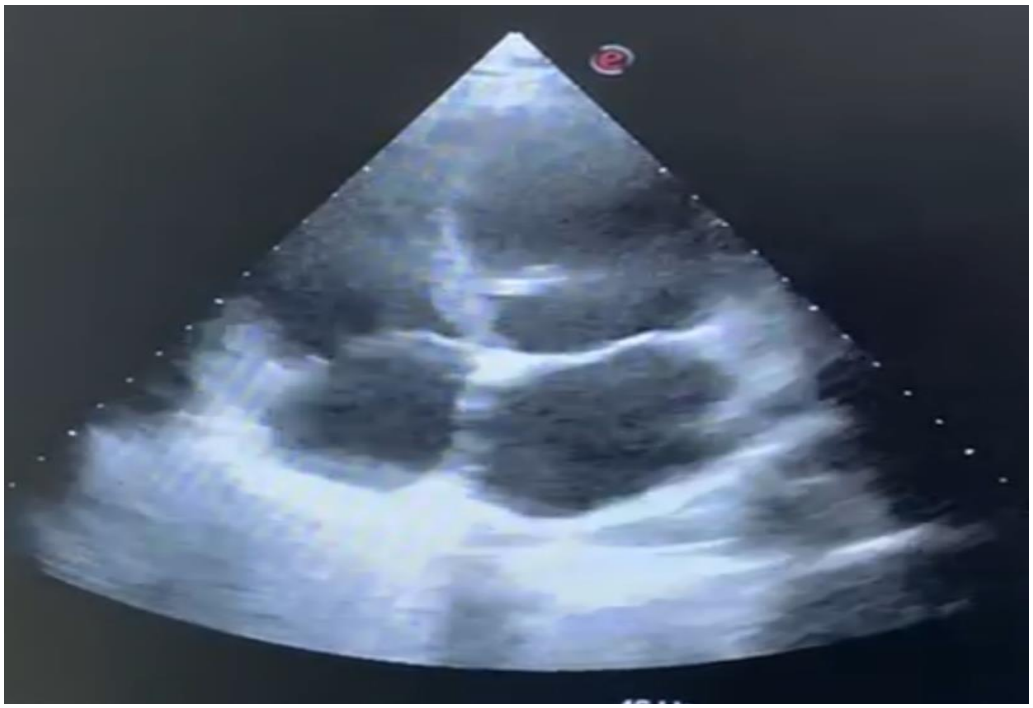

Figure 2: Four-chamber echocardiogram showing a dilated LV

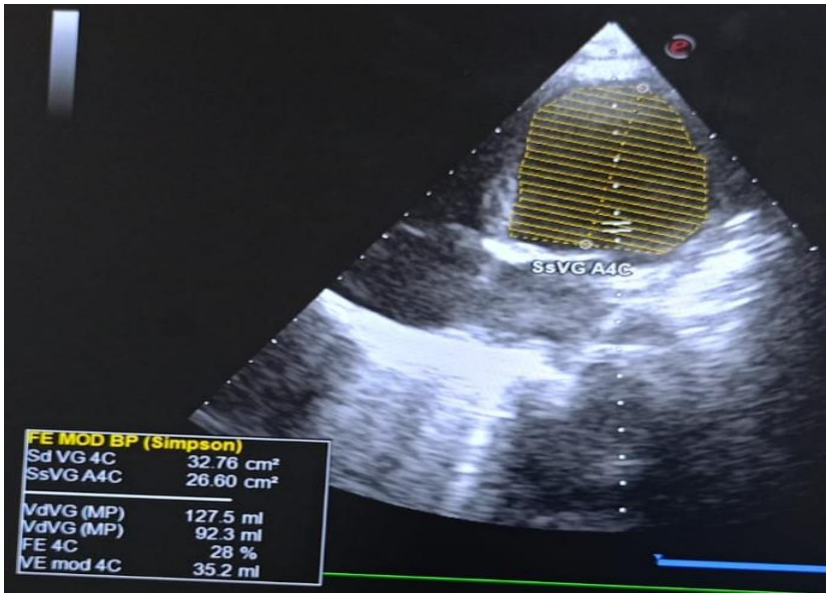

Figure 3: Echocardiogram showing a left ventricle with impaired systolic function and an LVEF of 28%

Laboratory results showed a white blood cell count of 16,000, CRP of 27, procalcitonin of 0.25, troponin of 537, and BNP of 4,456; immunological testing for autoimmune antibodies was negative, allowing us to rule out systemic disease. Microbiological analysis performed on samples from both sites (respiratory secretions and pericardial effusion), as well as multiplex PCR testing, indicated that two samples were positive for rhinovirus and enterovirus, confirming the diagnosis of a viral etiology.

The patient was admitted to the intensive care unit, placed in a prone position, and received respiratory support via non-invasive ventilation (NIV), hemodynamic support with a vasopressor (norepinephrine) and a positive inotropic agent (dobutamine), and adjunctive treatment with corticosteroids and diuretics.

After 2 days of treatment, the patient's condition improved clinically, with a reduction in dyspnea and an increase in LVEF from 28% to 40%. The gradual weaning off vasoactive drugs allowed for the initiation of treatment for heart failure with lasilix combined with a beta-blocker

The patient's condition improved favorably after 7 days of treatment, with clinical improvement and complete resolution of dyspnea, allowing for her transfer to the cardiology ward, where she received care for 1 month and was discharged with a systolic cardiac function of 60%.

DISCUSSION

The term "myocarditis" refers to inflammation of the myocardium, which has various causes, including both infectious and autoimmune etiologies. It was with advances in medicine and particularly in the field of microbiology, through the development of PCR in the 1990 that infectious myocarditis was definitively distinguished from autoimmune myocarditis[6, 7]

Viral myocarditis accounts for 70 to 90% of infectious myocarditis, while bacterial myocarditis accounts for less than 5% and is more common in resource-limited countries, primarily caused by Corynebacterium diphtheriae, Staphylococcus aureus, Streptococcus, and Borrelia burgdorferi (Lyme disease). Fungal myocarditis is very rare (<1%), caused by Candida and Aspergillus, and occurs almost exclusively in immunocompromised patients. Finally, parasitic myocarditis has a high prevalence in certain endemic regions, with the main causative agents being Trypanosoma cruzi (Chagas disease), Toxoplasma gondii, and Trichinella spiralis[3, 8]

The annual incidence of myocarditis is estimated at 10 to 22 cases per 100,000 people in the general population and is believed to account for 5 to 12% of unexplained sudden deaths in young adults. It primarily affects young people, with peak incidence between the ages of 20 and 40, and is more common in men, likely due to hormonal and immunological factors. The actual prevalence is probably underestimated because many forms are asymptomatic or present with few symptoms[9, 10]

From a pathophysiological perspective, viral myocarditis progresses in three phases: a viral invasion phase that infects cardiomyocytes, leading to direct cellular necrosis; an immune phase marked by an inflammatory response, sometimes disproportionate, causing diffuse myocardial damage responsible for acute systolic dysfunction; and finally, a remodeling phase that may lead to chronic dilated cardiomyopathy[3, 11]

The fulminant form often occurs after a recent viral episode (fever, myalgia, flu-like syndrome) and is

associated with cardiovascular clinical signs such as cardiogenic pulmonary edema, hypotension, cardiogenic shock, ventricular or supraventricular arrhythmias, as well as respiratory manifestations including severe hypoxemia, bilateral pulmonary infiltrates, ARDS, etc.[12, 13]

Severe forms requiring admission to the ICU are rare but serious: these are primarily fulminant myocarditis, which accounts for approximately 10% of acute myocarditis cases. Among patients admitted to the ICU for cardiogenic shock, acute myocarditis is implicated in 2 to 4% of cases [14]

The association between viral myocarditis and acute respiratory distress syndrome (ARDS) is exceptional and rarely described. The coexistence of ARDS and cardiogenic pulmonary edema is a major diagnostic pitfall; viral ARDS can mimic cardiogenic pulmonary edema, and vice versa [15]

In the presence of severe respiratory failure combined with acute heart failure, a diagnosis of fulminant acute myocarditis is strongly suspected.

Echocardiography is the key test; it reveals acute left ventricular or biventricular dysfunction. Chest X-rays and CT scans may show similar findings: diffuse opacities, ground-glass opacities, and consolidation. The ECG shows nonspecific abnormalities: repolarization abnormalities, arrhythmias. Laboratory findings: Elevated troponins, increased BNP, inflammatory markers, immunological profiles, viral serology, and viral PCR (including influenza and SARS-CoV-2) point toward an etiology[7, 16, 17]

To confirm the diagnosis, after partial stabilization, cardiac MRI is the gold standard non-invasive method, showing: diffuse myocardial edema, non-systematic late subepicardial enhancement, in accordance with the modified Lake Louise criteria, consistent with acute myocarditis. Endomyocardial biopsy remains the definitive test, based on histological analysis according to the Dallas classification, and is indicated primarily in fulminant or atypical forms, allowing for histological and virological confirmation[18, 19]

For our patient, the radiological and echocardiographic findings allowed us to assess the severity and cardiorespiratory management of respiratory distress, and the results of multiplex PCR on respiratory and pericardial samples pointed toward a viral etiology through the isolation of rhinovirus and enterovirus

Management includes hemodynamic support aimed at stabilizing the patient using inotropes (dobutamine) for low cardiac output and vasopressors (norepinephrine), with or without respiratory support via protective mechanical ventilation with appropriate PEEP, loop diuretics at cautious doses, and strict fluid restriction. No specific immunosuppressive therapy is initiated in the absence of histological evidence. In refractory fulminant forms, circulatory support is essential for survival; etiological treatment is combined with symptomatic management using antivirals which are rare and have limited indications and treatment for acute heart failure, in accordance with the recommendations[7, 14]

Fulminant forms may recover completely by the 10 th day of treatment through a gradual improvement in oxygenation and hemodynamic recovery, particularly thanks to early circulatory support. However, dilated cardiomyopathy may persist long-term; acute mortality ranges from 5% to 20%, reaching up to 40% in fulminant forms without circulatory support, and complete recovery of cardiac function is observed in 50% to 70% of surviving cases [14, 20]

CONCLUSION

Viral myocarditis presenting as ARDS and cardiogenic pulmonary edema poses a significant diagnostic and therapeutic challenge. The overlap of respiratory and cardiac symptoms requires a multimodal approach that integrates clinical findings, imaging, biomarkers, and, in some cases, biopsy. Management relies on optimal hemodynamic and respiratory support, with early use of circulatory support in fulminant cases. A better understanding of viral and immune mechanisms will improve early diagnosis and future therapeutic strategies.

Conflicts of Interest: The authors declare no conflicts of interest

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