International Journal of Medical Toxicology & Forensic Medicine
2026, Volume 16, E52537

Case Report

Plasma Cell–Rich Viral Myocarditis Identified at Autopsy: A Case Report

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Citation Qavam S, Emaminia MS, Bakhtiyari H, Hafezi Ahmadi M, Bakhtiyari A. Plasma Cell–Rich Viral Myocarditis Identified at Autopsy: A Case Report. International Journal of Medical Toxicology and Forensic Medicine. 2026; 16:E52537.

https://doi.org/10.22037/ijmtfm.v16.52537

Article info:

Received: 20 June, 2026

Accepted: 10 July, 2026

Published: 17 August, 2026

Keywords:

Plasma cell–rich, Viral myocarditis, Autopsy

ABSTRACT

Background: Viral myocarditis is a clinically significant inflammatory disease of the myocardium and a well-established cause of sudden and unexpected death, particularly in young and middle-aged individuals. Histopathological diagnosis at autopsy may be challenging, especially when inflammatory patterns deviate from classic lymphocytic predominance.

Case Presentation: We report the case of a 37-year-old man with a known history of chronic heart failure and seizures who presented with generalized weakness and died within 24 hours of hospital admission despite supportive management. A complete medicolegal autopsy with detailed histopathological examination was performed. Microscopic evaluation revealed plasma cell–rich lymphocytic myocarditis, accompanied by pathological findings consistent with acute respiratory distress syndrome (ARDS), including diffuse interstitial edema and inflammatory infiltration of the lungs. Additional findings included mild hepatitis and acute tubular necrosis, indicating multiorgan involvement.

Conclusion: The constellation of findings supports death due to fulminant viral myocarditis associated with systemic inflammatory response and secondary ARDS. This case highlights the diagnostic value of meticulous myocardial histopathological assessment, particularly the recognition of plasma cells as supportive evidence of viral myocarditis, and underscores the potential for rapid clinical deterioration and fatal outcome in patients with underlying cardiac disease.

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Introduction

Viral myocarditis is an important inflammatory disorder of the cardiac muscle, most commonly occurring following viral infections. It encompasses a broad clinical spectrum ranging from subclinical disease to acute heart failure, life-threatening arrhythmias, and sudden cardiac death [1, 2]. The clinical relevance of viral myocarditis is particularly notable in the context of sudden death, as epidemiological studies have demonstrated a substantial contribution of myocarditis to unexpected fatalities, especially among younger individuals. An epidemiological autopsy-based study from Denmark reported that approximately 6% of sudden deaths in individuals aged 1–49 years were attributable to myocarditis [3, 4]. Consequently, careful investigation and documentation of myocarditis in autopsy cases are essential to accurately establish the cause-and-effect relationship between myocardial inflammation and death.

Case Presentation

A 37-year-old man with a medical history of chronic heart failure and seizures presented to the emergency department with generalized weakness. According to medical records, the patient had been receiving medical therapy but had no documented history of prolonged hospitalization or other systemic diseases. The duration of hospitalization during this admission was less than 24 hours. Despite initial supportive and therapeutic interventions, the patient died shortly after admission. A complete medicolegal autopsy, including histopathological examination, was performed to determine the cause of death.

Macroscopic Findings

Brain and meninges: The cerebral cortex and basal nuclei demonstrated normal thickness and architecture. No evidence of hemorrhage, necrosis, or structural disruption was identified.

Lungs: Both lungs exhibited diffuse congestion and increased density on serial sectioning, suggestive of acute inflammatory changes and pulmonary edema.

Liver, kidneys, and heart: Gross examination revealed normal size and consistency of these organs, with no remarkable macroscopic lesions. The ventricular wall thickness and cardiac chamber dimensions were within normal limits, and no gross structural abnormalities were identified.

Microscopic Findings

Brain and meninges: Histological sections showed no significant pathological abnormalities.

Lungs: Microscopy revealed findings consistent with acute respiratory distress syndrome (ARDS), including chronic inflammatory cell infiltration within the interalveolar septa, widespread interstitial edema, injury to type I pneumocytes, and hyperplasia of type II pneumocytes. These changes were compatible with viral-induced pulmonary inflammation. In addition, extensive pulmonary parenchymal injury associated with scattered chronic inflammatory cell infiltration, epithelial and endothelial cell damage, as well as marked edema and congestion, supported the interpretation that the ARDS occurred in the setting of a chronic viral inflammatory process (Figure 1).

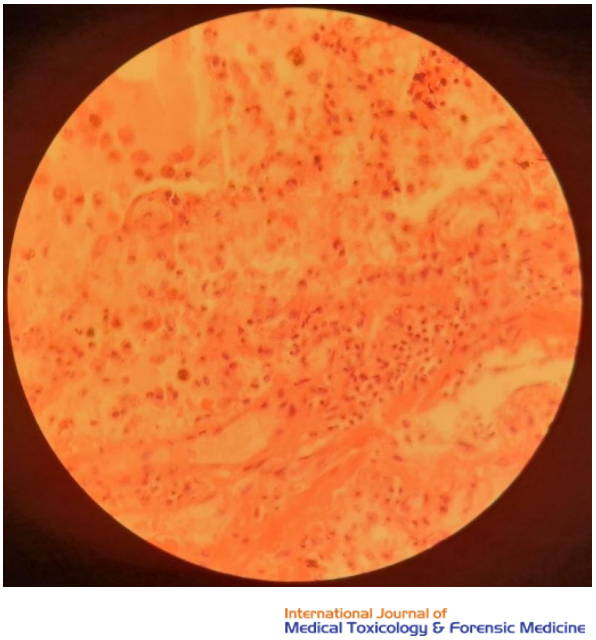

Figure 1. Histopathological section of lung tissue showing interstitial edema, thickening of alveolar septa, and chronic inflammatory cell infiltration within the pulmonary interstitium, consistent with inflammatory lung injury.

Liver: Sections demonstrated mild hepatitis characterized by focal hepatocellular necrosis accompanied by chronic inflammatory cell infiltration.

Kidneys: Histopathological examination revealed features of acute tubular necrosis (ATN), indicative of acute kidney injury.

Heart: The most significant findings were observed in the myocardium. Histological examination showed clear evidence of viral myocarditis, characterized by prominent infiltration of chronic inflammatory cells, predominantly lymphocytes, with a conspicuous

presence of plasma cells within the myocardial interstitium. Plasma cells constituted a considerable component of the inflammatory infiltrate and were consistently identified in multiple examined sections. Microscopic evaluation of the prepared sections from the myocardium and intramyocardial vessels demonstrated infiltration by chronic inflammatory cells, including a considerable number of plasma cells within the myocardial tissue, supporting the diagnosis of myocarditis (Figure 2).

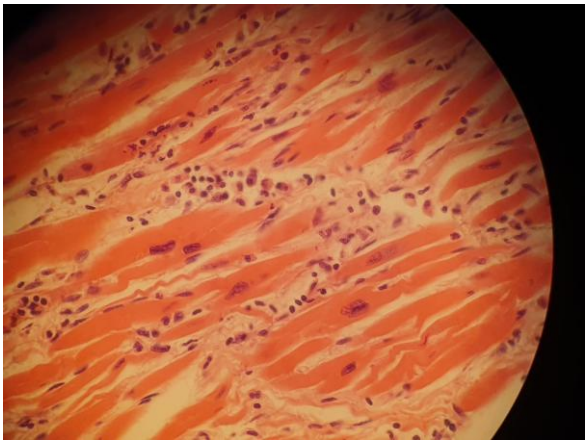

Figure 2. High-power photomicrograph of the myocardium showing a dense lymphoplasmacytic inflammatory infiltrate within the myocardial interstitium, with a considerable number of plasma cells among the inflammatory cells, supporting the diagnosis of plasma cell-rich myocarditis.

Based on the clinical history, rapid disease progression, and comprehensive macroscopic and microscopic findings, the cause of death was determined to be most consistent with viral myocarditis accompanied by ARDS secondary to systemic viral inflammation. Hepatic and renal changes were interpreted as secondary organ involvement in the course of a severe systemic inflammatory process.

Discussion

Although direct virologic confirmation was not available, the constellation of pulmonary and myocardial histopathological findings, together with the rapid clinical course and multiorgan inflammatory involvement, was considered most consistent with a viral etiology. The coexistence of ARDS and viral myocarditis indicates the activation of a widespread systemic inflammatory response. Primary lung injury in ARDS leads to extensive cytokine release and systemic inflammatory mediator dissemination, which may contribute to secondary cardiac injury. Several respiratory viruses, including influenza virus, adenovirus, and coronaviruses, are known to induce both ARDS and myocarditis [5-7]. Patients with prior heart failure who develop viral ARDS are at increased risk of cardiogenic shock and multiorgan failure.

Autopsy findings in such patients often reveal characteristic pathological lesions. Reported cases have shown myocardial interstitial edema and extensive necrosis, along with classic pulmonary features of ARDS, including diffuse alveolar damage and acute alveolar edema. Systemic inflammation further compromises cardiac output, leading to renal hypoperfusion and acute tubular necrosis, as observed in this case [8]. These observations highlight the importance of close cardiovascular monitoring in patients with ARDS, including early assessment with cardiac biomarkers, natriuretic peptides, and echocardiography, along with prompt supportive management [9, 10].

Conclusion

The pathological findings in this case—including lymphoplasmacytic myocardial infiltration, severe pulmonary edema consistent with ARDS, and acute tubular necrosis of the kidneys—reflect multiorgan injury resulting from a severe systemic inflammatory process. Previous studies have demonstrated that viral myocarditis is frequently associated with chronic lymphocytic infiltration and myocardial interstitial edema, and its coexistence with ARDS promotes widespread cardiopulmonary inflammation. This case supports the concept that the simultaneous presence of viral myocarditis and ARDS, particularly in the setting of underlying cardiac disease, initiates a vicious cycle of systemic inflammation and multiorgan dysfunction. Early recognition and comprehensive management of this interaction may be critical in preventing progression to fatal outcomes.

Acknowledgments

The authors would like to express their sincere gratitude to all those who contributed to the preparation of this manuscript

Conflicts of Interest

The authors report there are no competing interests to declare.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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