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(CASE REPORT)

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Coxsackievirus B3 myocarditis with severe complications: Case report and a brief review of the literature

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Abstract

Coxsackievirus B3 (CVB3) commonly induces fulminant viral myocarditis (VM), a significant diagnostic dilemma. The onset of fulminant VM is characterized by nonspecific flu-like symptoms that can be misinterpreted as other diseases, such as acute coronary syndrome (ACS) or cardiomyopathy. Fulminant VM is typically described as rapid progression to acute biventricular failure and cardiogenic shock. A low threshold for suspicion, along with a multidisciplinary approach to this condition, is essential for early recognition and aggressive treatment.

We present the case of a previously healthy 23-year-old female who presented with fulminant CVB3 myocarditis. The disease progressed to acute, severe dilated cardiomyopathy. Despite maximal therapy, the patient experienced a catastrophic natural history of life-threatening ventricular arrhythmias and complete heart block. Furthermore, she had rapid development of critical thromboembolic complications, such as acute ischemic stroke due to LV thrombosis and acute bilateral pulmonary embolism. Serology was positive for CVB3 (IgM) with a fourfold increase in IgG titers. The diagnosis was supported by an endomyocardial biopsy showing lymphocytic myocarditis and the positivity of myocardium for CVB3 viral RNA.

This case highlights the importance of maintaining a high degree of suspicion, which leads to timely diagnosis and individualized immunomodulatory therapy based on tissue diagnosis, along with rapid neurologic intervention to minimize thromboembolic and neurological sequelae. The serious course of the illness became evident when refractory cardiogenic shock occurred on hospital day 28, despite maximal supportive therapy.

Keywords: Myocarditis; Viral; Immune; Coxsackievirus B3; Fulminate; Endomyocardial biopsy; Ischemic stroke; Left ventricular thrombosis

1. Introduction

Myocarditis is an inflammation of the heart muscle (myocardium) that can weaken the heart and impair its ability to pump blood efficiently. It can be caused by viral or other infections, autoimmune conditions, exposure to toxins, or certain medications. Symptoms typically include chest pain, shortness of breath, fatigue, and irregular heartbeats; while it is often mild, severe cases can lead to heart failure. [1] [2]

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In developed countries, viral infections are the leading cause of myocarditis. Coxsackievirus B is a non-enveloped, single-stranded RNA enterovirus that remains among the implicated pathogens in myocarditis. [3] [4] The pathology can be explained by two mechanisms: first, the virus itself damages cardiomyocytes; then, the immune response targets the heart muscle. The process of immune-driven inflammation continues, with progressive cardiac damage leaving the ventricles struggling to function. [4] [5]

While most patients recover spontaneously, a subset of patients progress to severe complications with rapid, severe heart failure, cardiogenic shock, and life-threatening arrhythmias. Its presentation often masquerades as ACS, making early diagnosis a real challenge. [6] Recent strides and the use of cardiac magnetic resonance imaging (CMR) and endomyocardial biopsy with polymerase chain reaction (PCR) detection of the virus have sharpened accuracy. Combining CMR with serology or PCR testing for pathogen identification improved early diagnostic accuracy. [7]

Differentiating CVB3 myocarditis from ACS is important, as both share many characteristics, such as elevated troponin levels and diffuse ST-segment abnormalities. CMR remains the cornerstone of noninvasive evaluation, showing diffuse myocardial edema and nonischemic late gadolinium enhancement in mid-wall or subepicardial patterns. [7] Endomyocardial biopsy (EMB), considered the diagnostic gold standard, typically shows lymphocytic infiltration, myocyte necrosis, and detection of viral RNA by PCR. However, EMB has variable sensitivity and is used in conjunction with other tools, such as CMR and detailed histology/immunohistochemistry (IHC) studies. [8] [9]

Despite improvements in recognition and supportive care, outcomes in fulminant CVB myocarditis remain poor. Despite aggressive intervention, in-hospital mortality can exceed 50%. [10] Treatments include intravenous immunoglobulin, inotropes, mechanical circulatory support with Veno venous (VV), and Extracorporeal Membrane Oxygenation (ECMO). [11] The survivors of the acute phase usually recover near-normal systolic function, but up to 20% develop chronic dilated cardiomyopathy or repeated arrhythmias due to persisting autoimmune injury. [6]

Here we present a case of Coxsackievirus B3 myocarditis (CVB3 myocarditis) in a previously healthy young woman who, despite aggressive management, rapidly fell into severe biventricular dysfunction, refractory arrhythmias, thromboembolic complications, and multiorgan failure. The case underscores the virulence of Coxsackievirus B myocarditis and the importance of early diagnosis and management. This case epitomizes the devastating natural history of fulminant CVB myocarditis and calls for early recognition, personalized immunomodulatory intervention, and strategies to reduce thromboembolic and neurological complications in the management of viral myocarditis.

2. Case Presentation

2.1. Clinical picture and initial examination

A 23-year-old female was brought to the emergency department with progressive dyspnea, orthopnea, and bilateral lower extremity edema. She reported that she had fever, generalized myalgia, and fatigue in the past three weeks. The week before, she experienced shortness of breath, chest pain, and fatigue that affected her normal daily performance. She did not have a history of cardiac disease and reported no cardiomyopathy or other cardiac disease in her family. The patient denied recent travel and sick contacts, and she did not smoke or drink alcohol. The patient had a known medical history of mild asthma, effectively treated with inhaled corticosteroids, and a history of autoimmune thyroiditis under levothyroxine treatment.

2.2. Physical examination and primary lab tests

On physical examination, her heart rate was 118 bpm, tachypnea, RR 26/min, BP 72/62 mmHg, temperature 38.1 °C, and jugular venous distention at 12 cm H2O. The cardiac examination revealed a shifted apical impulse, S3 gallop, and a soft-pitched systolic murmur at the apex. Mid-thigh pitting edema with cool extremities was present, and pulmonary auscultation revealed bibasilar crackles suggestive of pulmonary congestion.

Lab results revealed mild leukocytosis (12,500/mL) with lymphocytosis, high Troponin I (8.2 ng/mL), and B-type natriuretic peptide (BNP) 4,850 PG/ML. The inflammatory markers showed that CRP was elevated (85 mg/L) and ESR was elevated (62 mm/HR). Renal function showed a slight increase in creatinine (1.4 mg/dL), and liver transaminases were slightly elevated.

2.3. Diagnostic investigations, differential diagnosis, and final diagnosis

An electrocardiography showed the presence of sinus tachycardia with diffuse ST-segment elevation and frequent premature ventricular contractions. The chest radiography demonstrated cardiomegaly, bilateral pulmonary edema, and a small bilateral pleural effusion. Transthoracic echocardiogram revealed left ventricular dilation and global left ventricular dysfunction in systolic function with an ejection fraction of 20%. Diffuse myocardial edema and nonischemic late gadolinium enhancement were the findings of the cardiac MRI. Serology studies identified antibodies to Coxsackievirus B3 (IgM) and a 4-fold increase in IgG titer, indicating a long-standing infection. The findings were consistent with viral myocarditis. Confirmatory endomyocardial biopsy requested by the patient showed characteristic features of heavy lymphocytic infiltration and cardiomyocyte necrosis. PCR testing on tissue biopsy confirmed the presence of viral RNA, establishing the diagnosis of fulminant CVB3 myocarditis (Figure 1A, B).

Other diagnostic considerations were integrated into the differential diagnosis, including ACS, takotsubo cardiomyopathy (stress cardiomyopathy), autoimmune cardiomyopathy, pulmonary embolism, hypertension, lung disease, giant cell myocarditis, eosinophilic myocarditis, and toxic cardiomyopathy. Fulminant CVB3 myocarditis with acute severe dilated cardiomyopathy and acute decompensated heart failure (NYHA Class IV) was the final diagnosis. [12] [13] [19] [20]

2.4. Management, complications, and outcomes

2.4.1. Early management

The patient was admitted to the cardiac intensive care unit, where she received intravenous furosemide, a low-dose ACE-inhibitor (initiated when she was hemodynamically stable and tolerated; initially held because of hypotension), and a beta-blocker. She was placed on inotropic support with milrinone to stabilize her vitals. The patient responded to treatment, and the pulmonary edema was resolved within the first 72 hours. However, a frequent transthoracic echocardiogram showed reduced left ventricular function.

2.4.2. Complications

Life-threatening arrhythmia: On day 6, the patient developed sustained monomorphic ventricular tachycardia, requiring electrical cardioversion and subsequent loading with amiodarone. She also developed repeated episodes of non-sustained ventricular tachycardia (VT) and transient high-degree heart block, which was managed with a temporary transvenous pacemaker.

Thromboembolic events: On hospital day 9, the patient developed acute neurological deficits despite receiving appropriate prophylactic anticoagulation. She developed right-sided hemiparesis and aphasia. Further, the CT and MRI of the brain revealed an acute ischemic stroke in the left middle cerebral artery region. A transesophageal echocardiogram revealed the origin of the thrombus responsible for the ischemic episode. Heparin anticoagulation was initiated. On hospital day 12, secondary prevention was achieved with placement of an implantable cardioverter-defibrillator (ICD). On hospital day 14, the patient developed acute dyspnea and hypoxemia, which was most likely due to bilateral pulmonary emboli.

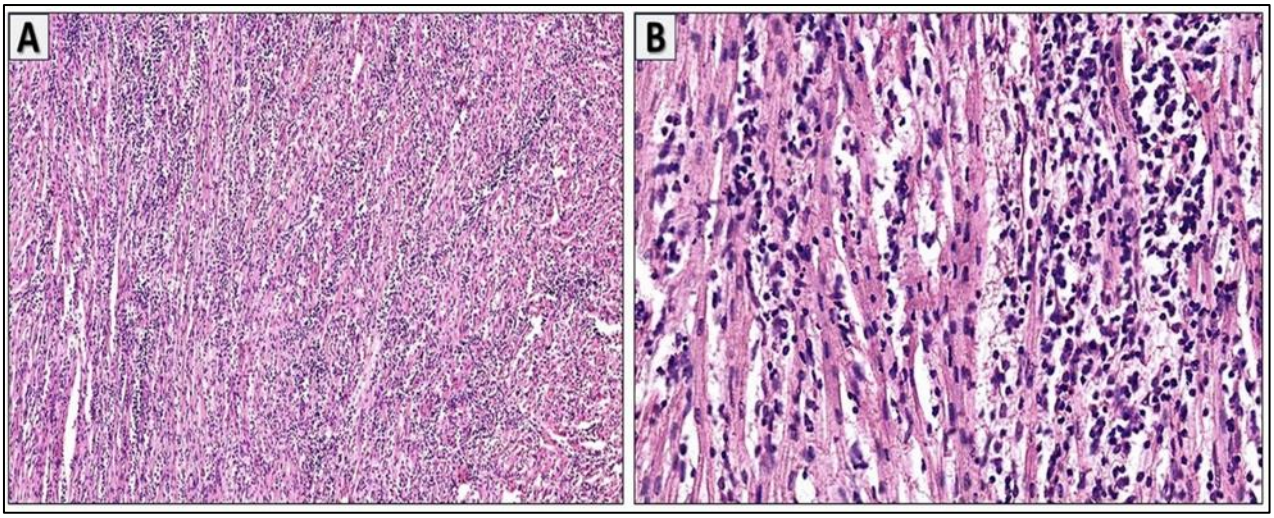
Refractory heart failure: The patient's cardiac function continued to deteriorate regardless of proper medical therapy and continuation of inotropic support. She subsequently developed evidence of multiorgan dysfunction, including progressive renal failure requiring continuous renal replacement therapy (CRRT) and worsening hepatic function.

2.4.3. Follow-up

Medical and surgical therapies for advanced heart failure were considered, including mechanical circulatory support and heart transplantation. However, due to the patient's severe ischemic stroke, neurological deficit, and poor prognosis, she was not considered a surgical candidate. Despite mechanical circulatory and extracorporeal membrane oxygenator (ECMO) support to maintain perfusion, she developed refractory cardiogenic shock exacerbated by sepsis that progressed to multiorgan failure.

Following a conversation with her family, who affirmed that the withdrawal of supportive therapies was in line with the wishes of the patient, life-sustaining therapies were withdrawn. She died on hospital day 28.

This case demonstrates the devastating potential of fulminant viral myocarditis, which can develop into irreversible cardiomyopathy and lethal multisystem complications within a short-term period despite aggressive intensive care management.



1A: Low power view showing prominent diffuse lymphocytic infiltration of the cardiac muscle (H&E stain X20); **1B:** High power view showing mature reactive lymphocytes infiltrating the cardiac muscle with focal necrosis (H&E stain X40)

Figure 1 Histomorphology of the cardiac biopsy with Coxsackievirus B myocarditis

3. Discussion

3.1. Background (History and epidemiology)

Myocarditis is an inflammatory myocardial disease characterized by infiltration of inflammatory cells and related myocyte necrosis that cannot be attributed to ischemia. [10] Myocarditis is classified into three clinical groups: subclinical myocarditis, mild-to-moderate myocarditis, and severe (fulminant) myocarditis. [14]

It has been estimated that worldwide acute myocarditis occurs at a rate of about 10–22 cases per 100,000 people/year and that it contributes to nearly 6–10 percent of non-atherosclerotic etiologies of sudden cardiac arrest (SCA) in young adults. [15] Viruses cause most myocarditis cases, and CVB is one of the most cardiotropic and medically significant viruses of the Picornaviridae family. [9] Over the past decades, myocarditis has gained increasing recognition due to the development of CMR, and molecular diagnostics have enabled the detection of viral genomes and inflammatory activity at an early stage. [6]

There are numerous etiologies of myocarditis, with viral pathogens historically being the most common cause. Common etiologies include Group B Coxsackieviruses (CVB), bacterial or parasitic infections, drug or toxin exposure, autoimmune pathophysiology, and idiopathic causes. [17] Coxsackievirus RNA has been detected in 40-50% of myocardial samples of patients with myocarditis or dilated cardiomyopathy. [17]

CVB3 is the most studied cardiotropic virus. It is closely linked to acute myocarditis and the subsequent development of dilated cardiomyopathy, which is why it has become the focus of serologic studies in the epidemiology and pathogenesis of viral myocarditis. [10]

3.2. Pathogenesis, pathophysiology

CVB3 shows the strongest myocardial tropism of the six serotypes. The virus enters cells through interactions with the Coxsackie-adenovirus receptor and the decay-accelerating factor, both of which are expressed on cardiomyocytes. Consequently, rapid replication and direct my cytolysis take place. [4] The course of the disease proceeds via a biphasic mechanism that involves an initial viremic phase with viral replication and cytotoxicity, followed by an immune-mediated phase, in which molecular mimicry between viral and cardiac antigens activates $CD3^+$ T cells and macrophages, perpetuating inflammation even after viral clearance. [4] Such a cascade may result in ventricular remodeling, thrombus formation over akinetic segments, and arrhythmogenic substrates responsible for life-threatening arrhythmias and thromboembolic complications. [18]

3.3. Comparative analysis of our case with the existing literature.

3.3.1. Clinical/Radiology/Diagnosis/Management

A typical manifestation of fulminant myocarditis (FM), a condition distinct from acute non-fulminant myocarditis, is the patient's presentation, i.e., a flu-like prodrome followed by rapid-onset severe dyspnea and cardiogenic shock. [16] The literature on fulminant myocarditis explains the acute hemodynamic instability that is characterized by hypotension (72/62 mmHg), tachycardia (118 bpm), and the presence of FM as a syndrome of acute, severe inflammation of the myocardium. [14] The initial electrocardiogram findings of diffuse ST-segment elevations and frequent premature ventricular contractions, while suggestive of pericarditis, also pointed toward a diffuse inflammatory process, distinguishing it from the localized injury of ACS. [14]

Transthoracic echocardiography radiologically revealed left ventricular systolic dysfunction of global extent and an ejection fraction of 20%, typical of FM, in which the diffuse nature of the inflammation leads to overall impairment. Moreover, the cardiac magnetic resonance imaging (cMRI) findings of diffuse myocardial edema and nonischemic late gadolinium enhancement (LGE) are typical of acute inflammatory myocardial injury and, together with the clinical history, strongly supported the diagnosis before biopsy. [16]

The diagnostic pathway in this case was complete and adhered to the existing common practice. Coxsackievirus B3 serology (IgM positive and a fourfold rise in IgG titers) was an adequate presumptive test for recent infection. However, endomyocardial biopsy (EMB) revealed active, inflammatory myocarditis, characterized by lymphocytic infiltration, myocyte necrosis, and viral RNA by PCR. A biopsy is a necessary procedure because, in the modern era, it is accepted that, although serology may suggest an etiology, EMB is the gold standard for verifying the diagnosis, the specific phenotype of the inflammatory reaction, and guiding potential immunomodulatory therapy. [9] The patient's demand to have the biopsy, despite the diagnostic serology, finally gave the necessary tissue diagnosis to confirm the fulminant CVB3 etiology and exclude the other critical differentials, such as giant cell or eosinophilic myocarditis, which would demand different immunomodulatory therapy. [19] [20]

3.3.2. Complications and Outcomes

Primary therapy based on conventional management of heart failure (diuretics, temporary withdrawal of ACE/BB, and inotropic support with milrinone) led to a short-term, poor response, which is typical of FM. The acute exacerbation demanded additional interventions, including the placement of an implantable cardioverter-defibrillator (ICD) as secondary prevention following a sustained ventricular tachycardia and the use of mechanical circulatory support (ECMO) as refractory cardiogenic shock treatment. The widespread participation of the conduction system in the inflammatory process was demonstrated by the formation of the complete heart block, requiring temporary pacing and being one of the most familiar and severe complications of diffuse myocarditis. [10]

The thromboembolic complications and the neurological complications were the most impressive and devastating in this case. A slightly more rare but familiar complication of severe dilated cardiomyopathy is the occurrence of an acute ischemic stroke in the left middle cerebral artery territory, which has been demonstrated to be due to a newly formed left ventricular thrombus. Most importantly, this incident occurred even though the patient was undergoing anticoagulant prophylaxis, which presents a very crucial gap in the traditional management processes of this group of high-risk patients. The literature suggests that the coagulation dysfunction is a significant, often undiagnosed feature of FM that necessitates a more sensitive and potentially more aggressive and personalized anticoagulation strategy to balance the risk of bleeding and thrombosis. However, research on specific management strategies is limited [22]. The subsequent development of bilateral pulmonary emboli also made the critical situation of the patient more complicated, and is evidence of the pro-thrombotic condition of the system due to the fulminant inflammatory process and the severe heart failure.

Despite the complete support of ECMO and continuous renal replacement therapy (CRRT) for progressive multiorgan failure, the patient succumbed to refractory cardiogenic shock. High mortality rate in FM, ranging between 40 and 70 percent, is observed in some series, and this is well documented. [21] The patient, however, had very bad neurological impairments because of the stroke and therefore was not fit to undergo advanced treatments of heart failure like a heart transplant, which is a common exclusion criterion that made her prognosis very poor. [23]

This patient’s expiration on day 28 is a good reminder of how devastating complicated FM can be. When the damage to multiple organs and the neurological systems is already irreversible, outcomes are mostly unfavorable, sometimes even with the most vigorous life support interventions. However, the condition can be reversed with prompt, aggressive treatment before irreversible damage is inflicted. [14]

4. What have we learned from this case?

There are multiple critical learning points in this case regarding the diagnosis and management of fulminant VM. First, it solidified the need to have a high index of suspicion of myocarditis in young, otherwise healthy patients with new-onset heart failure despite nonspecific initial symptoms following a viral prodrome. Second, the case reiterated the value of endomyocardial biopsy, which provided definitive CVB3 etiology and inflammatory confirmation, both of which are crucial for future immunomodulatory trials. Most importantly, the case highlighted the devastating and often refractory nature of thromboembolic complications in FM. We see the emergence of a left ventricular thrombus and a stroke, even with the prophylactic use of anticoagulants, which demonstrates that the conventional guidelines are not always adequate. To reduce these disastrous neurological outcomes, a more aggressive, individualized anticoagulation approach, possibly informed by more advanced imaging and coagulation biomarkers, is justified in patients with severe left ventricular dysfunction and evidence of systemic inflammation. Lastly, the rapid development of multiorgan failure and the consequent ineligibility to receive a heart transplantation because of neurological damage highlighted the fact that the time of effective intervention is short, and the results of delayed or improper intervention are often irreversible.

Abbreviations

- Coxsackievirus B3 (CVB3);
- Viral myocarditis (VM);
- Acute coronary syndrome (ACS);
- Cardiac magnetic resonance imaging (CMR);
- Endomyocardial biopsy (EMB);
- Immunohistochemistry (IHC);

5. Conclusion

This case of fulminant CVB3 myocarditis was presented to highlight the diagnostic dilemma, the dynamic and disastrous natural course, and the complex treatment of this emergency. This case is of great importance to the medical community, as it illustrates the necessity of timely, conclusive tissue diagnosis via endomyocardial biopsy to establish the etiology and prescribe individual therapy in the context of cardiogenic shock. Furthermore, and most importantly, it raises awareness of the elevated and potentially fatal threat of thromboembolic events, namely the development of left ventricular thrombus and subsequent stroke that can still happen despite the routine prophylaxis. We hope that presenting this patient's catastrophic course will raise awareness among clinicians about the necessity of becoming more vigilant and aggressive in their monitoring, and possibly implementing more specific and intense anticoagulant strategies to prevent irreversible neurological injury and improve the overall prognosis for patients with fulminant viral myocarditis.

Compliance with ethical standards

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Disclosure of conflict of interest

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

The patient expired, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

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