Scientific Foundation SPIROSKI, Skopje, Republic of Macedonia
South East European Journal of Cardiology. 2023 Oct 30; 4(1):84-87.
https://doi.org/10.3889/seejca.2023.6065
eISSN: 1857-9361
Category: *Case Report*
Section: *General Cardiology*

OPEN ACCESS

Fulminant Myocarditis Mimicking Myocardial Infarction in a Young Woman: A Challenging Diagnosis and Treatment

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Edited by: Igor Spiroski
Citation: Bahar MA, Karolina W, Rohman MS, Anjarwani S, Prasetya I. Fulminant Myocarditis Mimicking Myocardial Infarction in a Young Woman: A Challenging Diagnosis and Treatment. SEE J Cardiol. 2023 Oct 30; 4(1):84-87. https://doi.org/10.3889/seejca.2023.6065
Keywords: Cardiogenic shock; Chest pain; Hemodynamic support
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E-mail: aswinbahar8@gmail.com
Received: 02-Nov-2023
Revised: 25-Nov-2023
Accepted: 09-Dec-2023
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Funding: This research did not receive any financial support
Competing Interests: The authors have declared that no competing interests exist
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Abstract

BACKGROUND: Fulminant myocarditis is a rare and serious condition with high mortality. It poses diagnostic challenges due to myocardial infarction-like symptoms and rapid deterioration. Timely diagnosis and access to advanced interventions are vital for optimal management and improved outcomes.

CASE PRESENTATION: A 34-year-old woman presented with typical chest pain, ST changes, positive cardiac biomarkers, and impaired left ventricle function. The presumptive diagnosis was fulminant myocarditis complicated with cardiogenic shock, respiratory failure, and acute kidney injury. Leukocytosis and high anti-streptolysin O (ASO) titer suggested a recent streptococcal infection as a possible cause. Advanced intervention limitations hindered patient management. On the second day, the patient had a cardiac arrest.

CONCLUSION: The diagnosis can be challenging due to the similarity of symptoms and signs to myocardial infarction, combined with the rapid deterioration of the patient's condition. Prompt initiation of mechanical circulatory support is vital. However, its limited availability in developing countries hinders routine implementation, leading to unfavorable outcomes when life support measures are unavailable.

Introduction

Fulminant myocarditis (FM) is a severe condition with a high risk of mortality. It progresses rapidly and often requires heart transplantation due to profound heart failure and circulatory failure [1], [2]. A case of fulminant bacterial myocarditis in a young woman initially diagnosed with myocardial infarction is presented.

Case Report

A 34-year-old woman presented to the Emergency Department with chest pain that began 7.5 h prior. The pain, described as a heavy-like sensation on her left chest, occurred while she was sleeping and lasted over 20 min. It radiated to her back and left arm, and she rated its severity as 6/10 on a visual analog scale. The pain improved after treatment at the previous hospital. She experienced shortness of breath, palpitations, and cold sweating. The previous hospital diagnosed her with a heart attack and administered a loading dose of aspirin, clopidogrel, enoxaparin, and norepinephrine. The physician recommended transferring her to our hospital for further treatment.

Eight months prior, the patient gave birth to her third son. During her pregnancy, she experienced high blood pressure. About 5 weeks ago, she was diagnosed with heart failure, with a low ejection fraction (EF) of 25% shown on echocardiography, and subsequently hospitalized for 2 weeks. Two weeks before her recent admission, she had an untreated high fever. She had a family history of thyroid disease.

Clinical examination with the support of a norepinephrine drip revealed normal blood pressure, irregular tachycardia, and tachypnea. Dental caries affected multiple teeth. Bilateral mid and basal rhonchi were heard on chest auscultation. A grade 3/6 systolic murmur was perceptible, originating from the apex. An audible Gallop rhythm was also detected.

The electrocardiography (ECG) showed a 1st-degree atrioventricular (AV) block with a heart rate of 70 bpm. ST elevation was observed in some leads, while ST depression was seen in others. A left ventricular strain pattern was evident in certain leads (Figure 1a). After medication administration at the previous hospital, the ECG exhibited 1sto AV block and premature ventricular contractions (PVCs) occurring in a pattern of bigeminy, originating from the apex of the LV (Figure 1b). Chest radiography showed cardiomegaly and pneumonia (Figure 2).

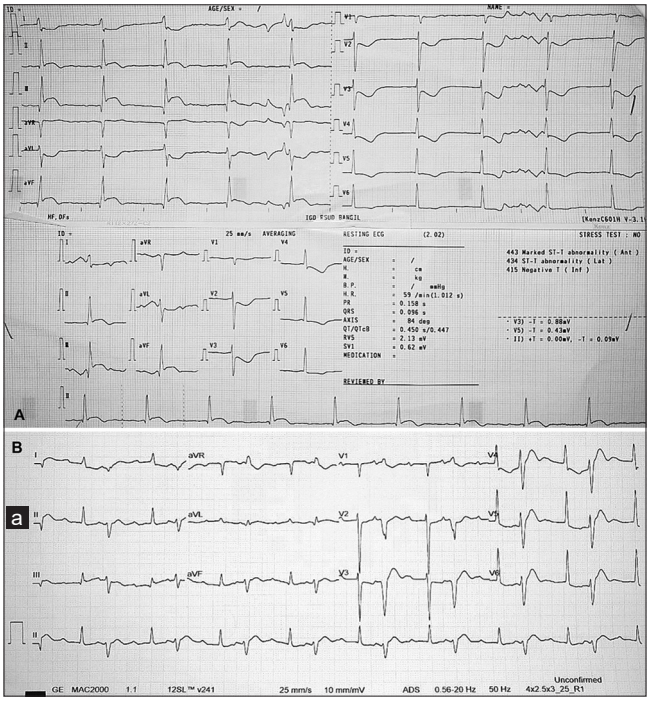


Figure 1: *Findings of the electrocardiography (ECG). (a) The ECG at the previous hospital revealed first-degree atrioventricular (AV) block with a heart rate of 70 bpm, ST elevation in leads II, III, and aVF, ST depression in leads I and aVL, and left ventricular (LV) strain patterns in leads V4-V5. (b) Following the administration of the loading dosage of medications at the previous hospital, the ECG showed first-degree AV block and premature ventricular contractions occurring in a pattern of bigeminy, originating from the apex of the LV*

Laboratory investigations revealed leukocytosis, with the full blood count showing neutrophilia and lymphocytopenia. In addition, elevated levels of troponin I, creatine kinase-myocardial band, N-terminal prohormone of brain natriuretic peptide, C-reactive protein (CRP), and aspartate aminotransferase were observed. Hypokalemia and hypoalbuminemia were noted, along with decreased estimated glomerular filtration rate. Blood gas analysis indicated hypoxemia and respiratory alkalosis. Subsequently, the Jackson-Rees circuit was employed, and the patient was admitted to the cardiovascular care unit.

The patient rapidly deteriorated, experiencing seizures, pulseless ventricular tachycardia (VT), hypoxemia, and severe metabolic acidosis. Cardiopulmonary resuscitation, intubation, and mechanical ventilation were initiated. Inotropic,

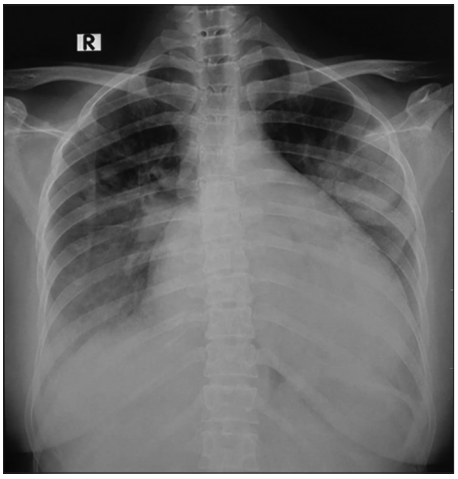


Figure 2: *Chest radiography showed cardiomegaly and pneumonia*

anticonvulsant, sedative, and antiarrhythmic drugs were administered, along with sodium bicarbonate and albumin transfusions. She experienced VT with a detectable pulse, requiring cardioversion to restore sinus tachycardia. Non-sustained VT episodes followed. Post-stabilization, an invasive strategy was implemented, revealing normal coronary arteries. Bedside transthoracic echocardiography (TTE) showed eccentric hypertrophy, global hypokinesia, severely impaired LV function (EF of 24%), and mild mitral regurgitation (Figure 3).

The presumptive diagnosis for the patient was FM complicated with cardiogenic shock, respiratory failure, and acute kidney injury. Heart failure with reduced EF (HFrEF) in NYHA functional class III due to nonischemic dilated cardiomyopathy (DCM) was also considered. Further testing revealed a high titer of anti-streptolysin O (ASO, 800 IU/mL), but no evidence of *Streptococcus* was found in cultures. However, *Pseudomonas aeruginosa* was identified in the sputum culture. As a result, the patient was treated with ceftriaxone and moxifloxacin. Autoimmune etiologies were ruled out, and immunosuppressive agents were not administered. Unfortunately, the patient's condition deteriorated rapidly. The hospital lacked resources such as extracorporeal membrane oxygenation and continuous renal replacement therapy. Regrettably, the patient experienced a cardiac arrest 44 h after admission, and an autopsy was not performed due to the absence of family consent.

Discussion

Diagnosing myocarditis is challenging due to diverse clinical manifestations. In this case, the patient presented with typical chest pain, but the clinical characteristics were distinct from those of patients with coronary artery disease. She was a young woman without typical cardiovascular risk factors. She fulfilled

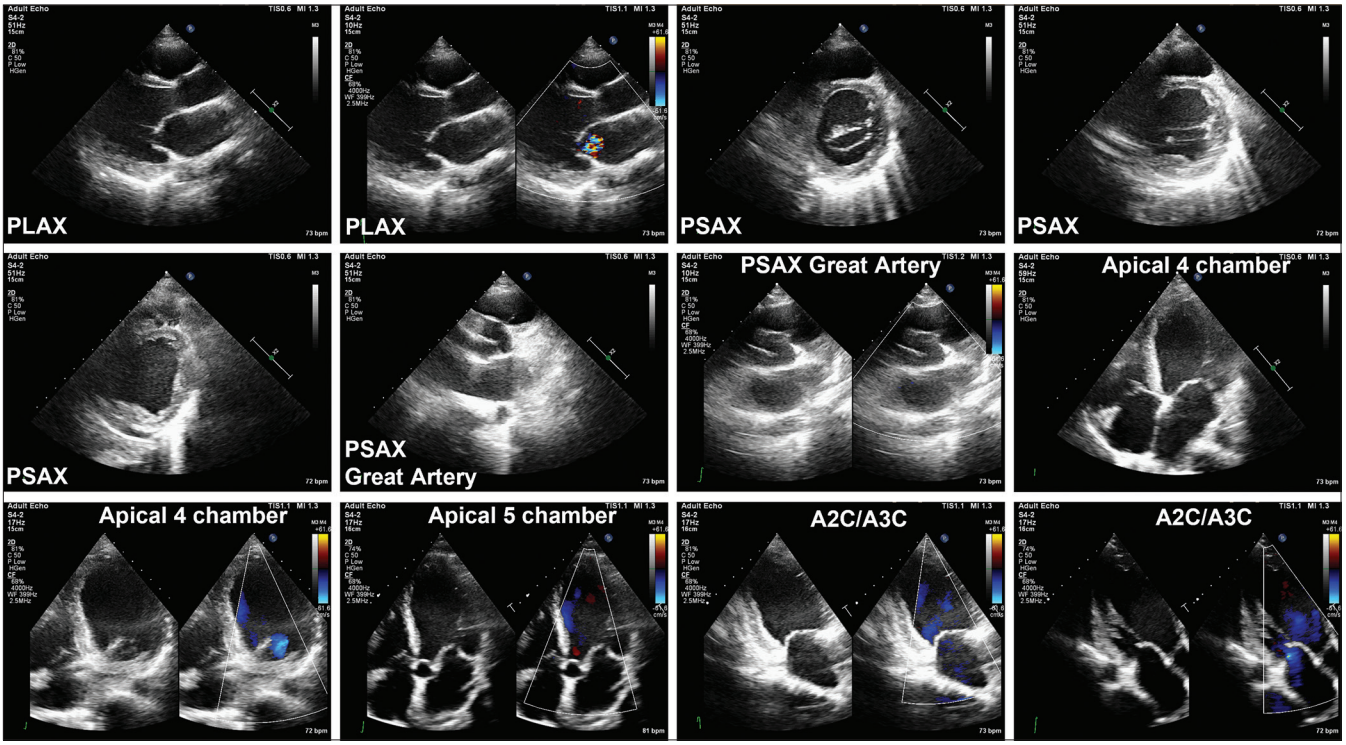


Figure 3: *Bedside transthoracic echocardiogram (TTE) results. TTE showed eccentric hypertrophy, global hypokinesia, severely impaired LV function with an ejection fraction of 24%, and mild mitral regurgitation*

multiple diagnostic criteria suggestive of clinically suspected myocarditis [3], which include acute chest pain, ST wave changes, severe LV dysfunction, and elevated troponin I levels. Despite normal angiography and no identifiable causes, she experienced a rapid progression of heart failure and cardiogenic shock. Her condition met the criteria for FM [1], characterized by acute illness within <2–4 weeks since the onset of symptoms, hemodynamic instability, and the need for inotropic agents and/or mechanical circulatory support (MCS). The observed laboratory abnormalities indicate multi-organ perfusion impairment, including the kidneys and liver.

Echocardiography is the imaging method of choice for the initial assessment of patients suspected to have FM [2]. In this case, besides global hypokinesia and severely impaired LV function, the TTE also showed eccentric hypertrophy and mild mitral regurgitation. Thus, the diagnosis of HFrEF in NYHA functional class III due to non-ischemic DCM was considered. Peripartum cardiomyopathy was ruled out as the cause, as heart failure symptoms appeared 7-month postpartum [4].

To confirm FM, cardiac magnetic resonance imaging is the preferred noninvasive method [2]. However, the patient's unstable condition limited its use in this case. Despite being the gold standard for diagnosing myocarditis [3], endomyocardial biopsy was not performed due to anticoagulation. Instead, laboratory investigations revealed leukocytosis and an elevated CRP level, confirming inflammation. The patient's recent streptococcal infection history, indicated by a high fever 2 weeks prior and a high ASO titer, suggested it as a potential cause. However, no Streptococcus was detected in blood, urine, or sputum cultures, indicating a possible localized or inactive infection. Further testing, such as polymerase chain reaction-based methods for viral genome detection, may be necessary to explore other potential infectious agents.

The patient did not meet the diagnostic criteria for acute rheumatic fever [5], nor clinical domains and criteria for systemic lupus erythematosus according to the 2019 European League Against Rheumatism and the American College of Rheumatology [6]. Antiphospholipid syndrome was excluded as the patient had successfully given birth 3 times without the need for anticoagulant therapy. Hashimoto's disease and Graves' disease were also ruled out as the patient did not present any symptoms and signs associated with the conditions. Moreover, Takotsubo cardiomyopathy was excluded as there was no apical ballooning syndrome on echocardiography.

Early prioritization of hemodynamic and respiratory support is crucial in the management of patients with FM, with careful monitoring of end-organ perfusion. In cases of unresponsive shock, the use of temporary MCS devices should be considered [2]. In this particular patient, we were unable to carry out advanced interventions due to limitations in our facilities.

In this case, we refrained from using immunosuppressive agents due to ongoing

controversy. Limited occurrence and diagnostic challenges in FM hinder randomized controlled trials for evaluating treatment approaches. In general, the 2016 American Heart Association scientific statement discourages empirical immunomodulatory agents as a first-line treatment for myocarditis without a definitive diagnosis [7].

In this patient, ECGs revealed the presence of 1^{sto} AV block, PVCs occurring in a pattern of bigeminy, and episodes of VT. Arrhythmias are frequently observed in FM and can arise from myocardial edema or scar formation. The management of arrhythmias in patients with inflammatory heart disease follows established clinical guidelines, including antiarrhythmic drugs, with a preference for amiodarone and beta-blockers [2]. It is recommended to postpone the implantation of any devices until the resolution of any reversible episodes of acute myocarditis [3].

The precise etiology of the patient's condition remains uncertain, and the inability to perform an autopsy limited further investigation into the case. While this patient passed away, advancements in the treatment of FM have led to a significant reduction in mortality rates, dropping from more than 50% to <5% in distinct clinical centers [8]. Optimal care for critical patients necessitates accessible and well-equipped health-care facilities capable of delivering timely and suitable interventions to maximize outcomes.

Conclusion

FM is a serious condition with high mortality. The diagnosis can be challenging due to the similarity of symptoms and signs to myocardial infarction, combined with the rapid deterioration of the patient's condition. Prompt initiation of MCS is vital. However, its limited availability in developing countries hinders routine implementation, leading to unfavorable outcomes when life support measures are unavailable.

Patient Consent Statement

Written informed consent was obtained from the relative of the patient for publication of this case report, including accompanying images.

Acknowledgment

This case report was one of three selected papers presented at the "Campfire Discussions: Challenging Clinical Cases in Heart Failure" during ACC Asia 2023 on September 29, 2023, in Manila, Philippines.

References

1. Ammirati E, Cipriani M, Lilliu M, Sormani P, Varrenti M, Raineri C, *et al*. Survival and left ventricular function changes in fulminant versus nonfulminant acute myocarditis. Circulation. 2017;136(6):529-45. https://doi.org/10.1161/CIRCULATIONAHA.117.026386
PMid:28576783
2. Kociol RD, Cooper LT, Fang JC, Moslehi JJ, Pang PS, Sabe MA, *et al*. Recognition and initial management of fulminant myocarditis. Circulation. 2020;141(6):E69-92. https://doi.org/10.1161/CIR.0000000000000745
PMid:31902242
3. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, *et al*. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: A position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013;34(33):2636-48. https://doi.org/10.1093/eurheartj/eht210
PMid:23824828
4. Bauersachs J, König T, van der Meer P, Petrie MC, Hilfiker-Kleiner D, Mbakwem A, *et al*. Pathophysiology, diagnosis and management of peripartum cardiomyopathy: A position statement from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. Eur J Heart Fail. 2019;21(7):827-43. https://doi.org/10.1002/ejhf.1493
PMid:31243866
5. Dougherty SD, Carapetis J, Zühlke L, Wilson N. Acute Rheumatic Fever and Rheumatic Heart Disease. St. Louis, USA: Elsevier; 2017. p. 1-343.
6. Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, *et al*. 2019 EULAR/ACR classification criteria for systemic lupus erythematosus. Arthritis Rheumatol (Hoboken, NJ). 2019;71(9):1400-12. https://doi.org/10.1002/art.40930
PMid:31385462
7. Bozkurt B, Colvin M, Cook J, Cooper LT, Deswal A, Fonarow GC, *et al*. Current diagnostic and treatment strategies for specific dilated cardiomyopathies: A scientific statement from the American Heart Association. Circulation. 2016;134(23):e579-646. https://doi.org/10.1161/CIR.0000000000000455
PMid:27832612
8. Hang W, Chen C, Seubert JM, Wang DW. Fulminant myocarditis: A comprehensive review from etiology to treatments and outcomes. Signal Transduct Target Ther. 2020;5(1):287. https://doi.org/10.1038/s41392-020-00360-y
PMid:33303763