

Shattered rhythms: The wrath of fulminant myocarditis

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Abstract

Fulminant myocarditis is a rare and life-threatening condition characterized by rapid hemodynamic deterioration and severe ventricular dysfunction. This report details a 33-year-old female with high-grade fever, syncope, and progression to complete heart block and ventricular arrhythmias. Despite transvenous pacing and antiarrhythmic therapy, the patient rapidly progressed to refractory cardiogenic shock and succumbed within hours due to severe left ventricular dysfunction. This case report emphasizes the need for early recognition, aggressive management, and the potential role of mechanical circulatory support in fulminant myocarditis to improve outcomes in this devastating condition.

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Introduction

Fulminant myocarditis represents a unique subset of acute myocarditis, marked by a dramatic clinical course and variable prognosis. Specific aetiologies, such as giant cell myocarditis, cardiac sarcoidosis, and eosinophilic myocarditis, frequently exhibit a fulminant phenotype. Due to its rapid clinical deterioration, timely and precise management is essential, but this is often challenging in resource-limited settings where advanced diagnostic and therapeutic tools, such as cardiac MRI and Mechanical Circulatory Support (MCS), may be unavailable. There is no definitive diagnostic modality; however, cardiac MRI, endomyocardial biopsy, and clinical presentation provide valuable diagnostic clues. The advent of MCS devices has significantly advanced treatment approaches, promising improved outcomes in fulminant myocarditis [1].

Case report

A 33-year-old female with no prior comorbidities presented with a seven-day history of high-grade fever, chills, rigors, dyspnea, and cough with borderline blood pressure requiring inotropes. Preliminary evaluation with ECG revealed sinus tachycardia and a 2D echo revealed global LV hypokinesia and severe Left ventricular dysfunction with LVEF of 30%. Initial laboratory investigations revealed elevated troponin with elevated NT-Pro-BNP suggestive of myocardial injury and acute heart failure, anemia (Hb: 9 g/dL), and elevated TSH (7.8 mIU/L), and mild renal dysfunction. The patient was admitted to the Intensive Care Unit and was treated for Heart Failure. A cardiac MRI was done, and it showed significant mid-myocardial Late Gadolinium Enhancement (LGE) in the LV free wall, sparing the endocardium and epicardium. While mid-myocardial LGE is commonly associated with myocarditis, it can also be seen in other conditions such as sarcoidosis, dilated cardiomyopathy, and Anderson-

Fabry disease. In this case, the findings were interpreted in conjunction with the clinical presentation, including recent febrile illness, elevated troponin, and rapid LV dysfunction. The imaging met the revised Lake Louise Criteria, supporting a diagnosis of probable myocarditis (Figures 1-3). Patient was started on IV Methylprednisolone 1 g daily for three days and then started on Oral prednisolone 1 mg/kg/day. Four days into her illness, she experienced two syncopal episodes and was found to have bradycardia with a heart rate of 30/min. An ECG revealed a complete heart block (Figure 4) with a wide-complex escape rhythm, and rising troponin levels indicated myocardial injury. A transvenous temporary pacemaker was placed, stabilizing her initially. A formal Transthoracic Echocardiogram (TTE) was performed after temporary pacemaker placement, which revealed the same moderate to severe LV dysfunction and no additional complications. Sarcoidosis was considered due to mildly elevated ACE levels (61 U/L), but further testing, including CT chest, EBUS, and node biopsy, was not feasible due to the patient's rapid deterioration. Giant cell myocarditis, though rare in young patients, was also considered, given the aggressive course and refractory ventricular arrhythmias. A comprehensive evaluation could not be conducted due to the rapid progression of the disease. However, viral myocarditis remained the most likely etiology based on clinical presentation, negative H1N1 swab, and absence of autoimmune markers (ANA: 1:40, dsDNA <30 IU/mL) (Table 1). After TPI, she developed slow ventricular tachycardia (130/min) (Figure 5), which persisted despite multiple attempts at overdrive pacing. Amiodarone was initiated (150 mg IV bolus, followed by 1 mg/min for 6 hours, then 0.5 mg/min for 18 hours), but VT remained refractory. Lidocaine (1.5 mg/kg IV bolus, followed by 1 mg/min infusion) was added, but the arrhythmia remained refractory. Overdrive pacing was attempted but was discontinued after the onset of Polymorphic Ventricular Tachycardia (PMVT). Given the fulminant nature of myocarditis, catheter ablation was not attempted, as the arrhythmogenic substrate was likely diffuse rather than focal, reducing the likelihood of procedural success. The patient continued to deteriorate into refractory cardiogenic shock despite maximal inotropic and antiarrhythmic therapy. She experienced cardiac arrest and could not be resuscitated. Referral for mechanical circulatory support (ECMO) was considered, but stabilization for transfer was not possible given the rapid decline. Post-mortem biopsy was not performed due to lack of consent.

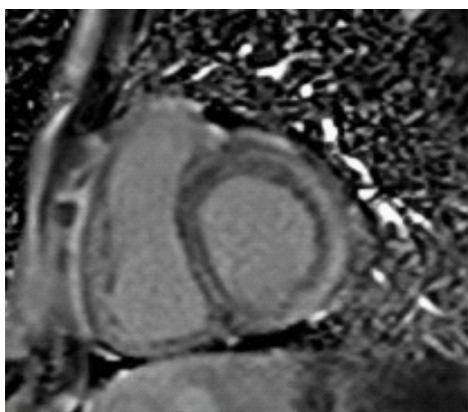


Figure 1: Short axis phase contrast T1 sequence shows mid myocardial late gadolinium enhancement along the left ventricular free wall with sparing of endo & epicardium.

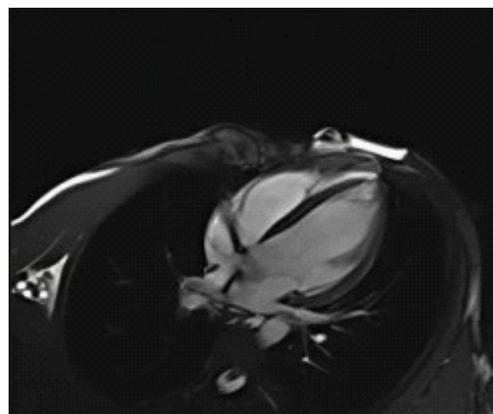


Figure 2: Four chamber view axial T2 White blood sequence shows Hyperintensity in the myocardium along the Left ventricular free wall, suggestive of Edema.

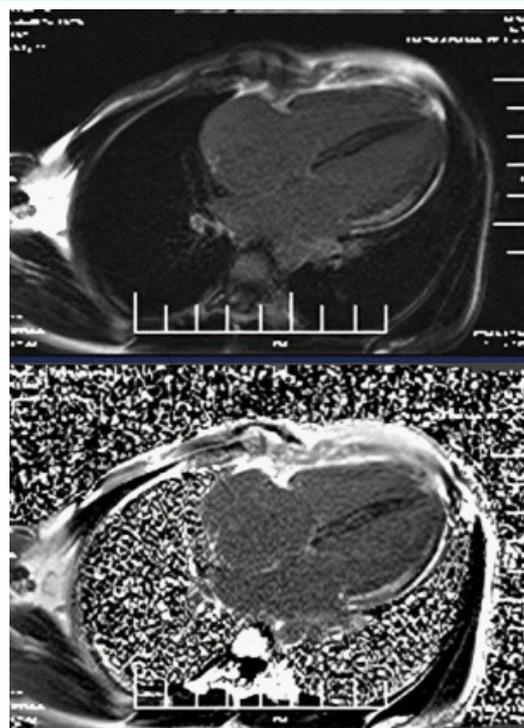


Figure 3: Upper image - 4 chamber post contrast T1 vibs sequence, Lower image - 4 chamber phase contrast T1 sequence - shows mid myocardial late gadolinium enhancement along left ventricular free wall with sparing of endo & epicardium.

Discussion

Fulminant myocarditis is a severe manifestation of myocarditis, characterized by rapid hemodynamic decline necessitating aggressive intervention. Lieberman et al. (1991) first described fulminant myocarditis by its acute symptom onset, histological evidence of multiple foci of active myocarditis, and a binary outcome of complete recovery or death. (2) However, this definition, reliant on Endomyocardial Biopsy (EMB), initially excluded eosinophilic and giant cell myocarditis, leading to underreporting. Updated criteria proposed by Ammirati et al. (2017) include acute symptom onset within 2-4 weeks, hemodynamic instability, and the need for MCS [3]. Fulminant myocarditis often remains underdiagnosed due to its rapid progression. Autopsy studies reveal myocarditis to be a significant cause of sudden cardiac death, especially in young individuals. Fulminant myocarditis differs with respect to garden variety myocarditis due to abrupt symptom onset, severe left ventricular dysfunction, cardiogenic shock, and life-threatening arrhythmias such as ventricular tachycardia or heart block. Non-fulminant myocarditis

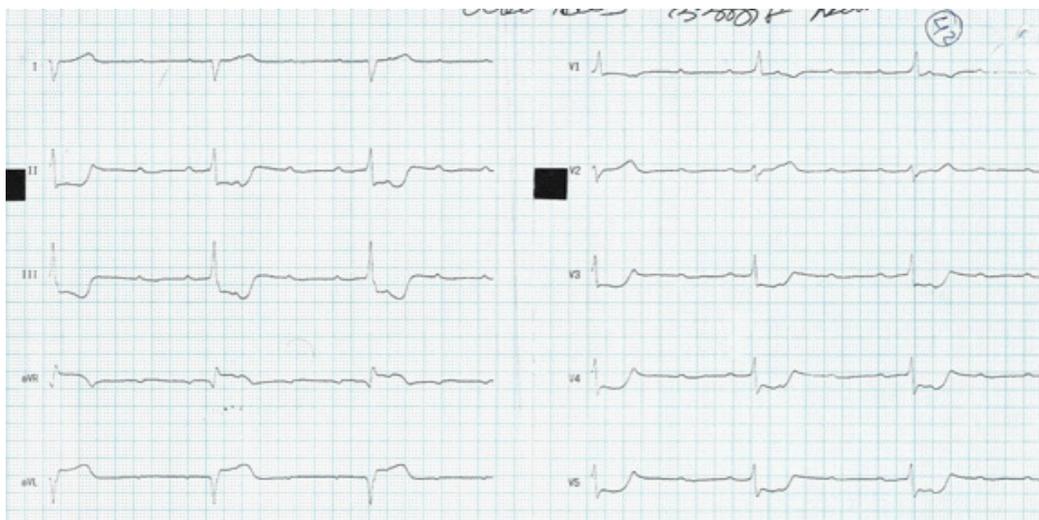


Figure 4: Showing complete heart block with wide complex escape rhythm with a heart rate of roughly 35 beats/minute

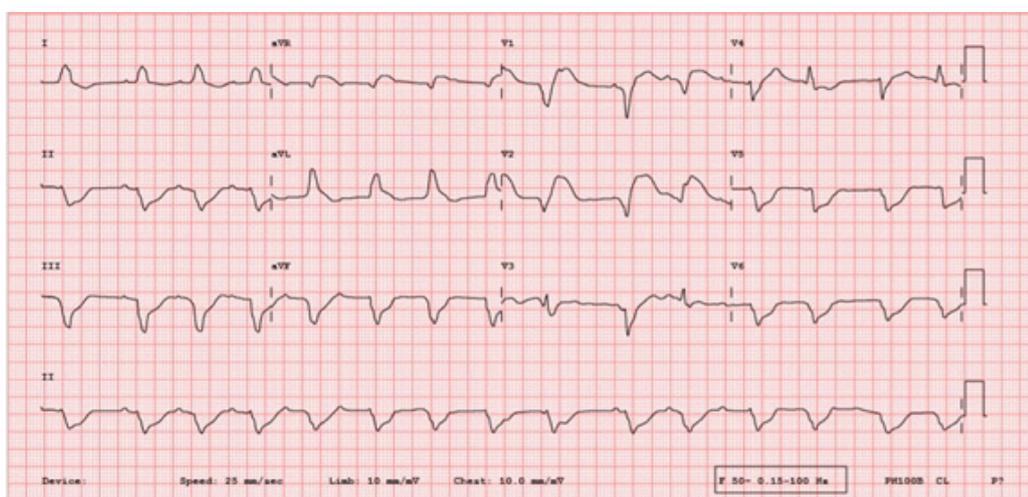


Figure 5: Slow ventricular tachycardia at a rate of 100/min, AV dissociation, and one conducted beat.

progresses more gradually with milder symptoms. Survivors of fulminant myocarditis may experience dramatic left ventricular function recovery, although long-term ejection fraction remains lower compared to non-fulminant cases (Table 2) [4-6].

Fulminant myocarditis can arise from various etiologies, broadly classified into viral, autoimmune, toxic, infiltrative, and bacterial causes. Viral myocarditis remains the most common, with Coxsackievirus, Parvovirus B19, Influenza (H1N1), and Adenovirus frequently implicated. Autoimmune and inflammatory causes include Systemic Lupus Erythematosus (SLE), sarcoidosis, giant cell myocarditis, and eosinophilic myocarditis. Toxic myocarditis may result from drug-induced injury (e.g., anthracyclines, checkpoint inhibitors, cocaine) or hypersensitivity reactions. Infiltrative myocarditis, such as sarcoidosis, amyloidosis, and Anderson-Fabry disease, can lead to chronic myocardial dysfunction. Bacterial and other infectious causes, including Lyme disease, Chagas disease, and diphtheria, are less common but remain important considerations in endemic regions [7]. Management of fulminant myocarditis requires early and aggressive intervention. Recognizing fulminant myocarditis and early referral to advanced centers is critical for improving outcomes. In our case, several clinical findings necessitated early referral, such as persistent hypotension despite initial stabilization efforts and escalating NT-proBNP levels (6000 pg/mL),

indicating severe myocardial stress, Complete heart block, and subsequent refractory ventricular arrhythmias, including polymorphic VT. These parameters align with the “I-NEED-HELP” mnemonic (Table 3) and underscore the need for early MCS therapy. MCS, such as VA-ECMO or LVAD, plays a pivotal role in bridging patients through the acute phase or to transplantation [8,9]. Outcomes in cardiac conditions vary significantly based on etiology and access to advanced care. Although in-hospital mortality remains high, survivors often demonstrate significant recovery of left ventricular function, particularly during the initial weeks after onset. Despite this, barriers such as the high cost of Mechanical Circulatory Support (MCS) devices, limited availability of trained personnel, and inadequate infrastructure pose significant challenges. To address these issues, initiatives could focus on developing cost-effective MCS alternatives, such as portable or simplified ECMO systems, and implementing robust training programs for healthcare professionals. Additionally, establishing regional hubs equipped with advanced resources and fostering international collaborations could improve accessibility and outcomes in underserved areas (Table 4). Routine heart failure therapies should be initiated after stabilization, following guideline-directed medical therapy. Incorporating structured referral criteria and practical advice for resource-limited settings can significantly improve early detection and management. Establishing standardized protocols

Table 1: Relevant laboratory investigations.

Parameter	Patient value	Normal range
Hemoglobin (Hb)	9 g/dL	12-16 g/dL
Creatinine	1.5 mg/dL	0.6-1.2 mg/dL
TSH	7.8 mIU/L	0.4-4.0 mIU/L
Troponin I	1.2 ng/mL	<0.04 ng/mL
Sodium	145 mmol/L	135-145 mmol/L
Potassium	3.2 mmol/L	3.5-5.0 mmol/L
Magnesium	1.8 mg/dL	1.7-2.2 mg/dL
NT-proBNP	6000 pg/mL	<125 pg/mL
Total Bilirubin	2.1 mg/dL	0.1-1.2 mg/dL
Albumin	5.6 g/dL	3.5-5.0 g/dL
ALT	210 U/L	10-40 U/L
AST	180 U/L	10-40 U/L
ANA (Titer)	1:40	≤1:40 (negative)
Anti-dsDNA	<30 IU/mL	<30 IU/mL (negative)
Serum ACE	61 U/L	8-52 U/L
H1N1 Swab	Negative	Negative
Covid 19	Negative	Negative

Table 3: "I-NEED-HELP" mnemonic for advanced heart failure referral.

Indicator	Description
I	Inotropes needed for hemodynamic support
N	NYHA Class III-IV symptoms
E	End-organ dysfunction
E	Ejection Fraction severely reduced (<20%)
D	Device support escalating (e.g., ICD, CRT)
H	Hospitalization for heart failure
E	Edema, refractory
L	Low blood pressure
P	Progressive decline

based on accessible clinical signs and tools, such as reliance on echocardiography and ECG findings, can bridge the gap where advanced diagnostics are unavailable [10].

Conclusion

Fulminant myocarditis is a medical emergency requiring rapid diagnosis and aggressive management. Mechanical circulatory support, especially VA-ECMO, has become a vital therapeutic option. Despite significant advancements, fulminant myocarditis remains associated with high mortality and long-term morbidity, emphasizing the need for continued research and early intervention strategies.

Table 2: Difference between fulminant and non-fulminant myocarditis.

Parameters	Fulminant myocarditis	Non-Fulminant myocarditis
Onset of Symptoms	Distinct onset of symptoms within 2-4 weeks	Indistinct onset of symptoms
Heart failure Symptoms	Class 4 often	Class 2-class 4 (variable)
Course	Rapidly progressive downhill course without intervention	Variable without intervention
Hypotension in acute stage	Often present with a need for inotropes and vasopressors	Maybe present uncommonly
Cardiogenic Shock in acute stage	Often present, need for IABP, VAD, VA-ECMO	Not present
Arrhythmia	VT and high-grade AV blocks are more common	Usually, sinus tachycardia, VT, and heart block are less common
EMB	Class 1 indication	Only needed if LVSD persists. GDMT when a systemic disorder is suspected.
Ejection fraction on admission	Severe LV dysfunction is very common	Severe LVSD is not as common in acute stages
Need for Heart transplant	More	Not as common as FM
In hospital Mortality rate	Higher	Lower
Ejection fraction in Long term Follow up	Dramatic improvement but overall EF is lower than NFM	It may normalize, may be static, but overall EF is higher than FM

Table 4: Constraints in developing world for managing fulminant myocarditis.

Constraint	Description	Possible Solutions
Cost of MCS devices	High costs limit availability	Develop cost-effective, portable alternatives
Infrastructure	Limited facilities for advanced care	Create regional hubs for specialized treatment
Trained Personnel	Shortage of trained healthcare providers	Provide robust training programs and workshops
Diagnostic Tools	Lack of advanced imaging like MRI and biopsy	Focus on clinical and basic diagnostic parameters
Referral Pathways	Delayed recognition and transfer	Develop structured referral networks

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