



Fulminant Lupus Myocarditis Presenting with Cardiogenic Shock and Persistent Complete Atrioventricular Block

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ARTICLE INFO	ABSTRACT
Published Online: 16 July 2026	Fulminant myocarditis is an uncommon but severe cardiac manifestation of systemic lupus erythematosus (SLE), which can rapidly progress to heart failure and life-threatening rhythm disturbances. We report the case of a 33-year-old woman with a recent diagnosis of SLE who developed rapidly progressive cardiac involvement leading to cardiogenic shock and complete atrioventricular block. Her condition required intensive care management, including mechanical ventilation and temporary pacing. Following treatment with high-dose corticosteroids and cyclophosphamide, a marked improvement in ventricular function was observed, with the left ventricular ejection fraction increasing from 20% to 48%. Despite this recovery, the conduction abnormality persisted. This case highlights the fulminant presentation of lupus myocarditis, the reversibility of myocardial dysfunction and the potential persistence of conduction system involvement.
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INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease in which cardiac involvement has been reported in up to half of patients during the course of the disease, and represents one of the leading contributors to morbidity and mortality [1]. While pericarditis is the most common cardiac manifestation, myocarditis is far less frequent, occurring in approximately 5–10% of patients, and is often underdiagnosed because of its non-specific presentation [2].

Fulminant lupus myocarditis represents the most severe form of cardiac involvement, characterized by the abrupt onset of acute heart failure, hemodynamic instability, and, in some cases, cardiogenic shock and life-threatening arrhythmias [2]. Conduction system disturbances, although well described in neonatal lupus secondary to transplacental passage of maternal anti-Ro/SSA antibodies, are increasingly recognized in adults with SLE and may have important prognostic implications when they fail to resolve after recovery of myocardial function [3].

We report a case of fulminant lupus myocarditis in a young woman, complicated by cardiogenic shock and persistent complete atrioventricular block. The case is notable for the dissociation between marked recovery of left ventricular

systolic function under aggressive immunosuppression and the persistence of advanced conduction disease, raising relevant questions regarding long-term electrophysiological management.

CASE PRESENTATION

A 33-year-old woman with no cardiovascular risk factors and a recent diagnosis of systemic lupus erythematosus was admitted for worsening shortness of breath. At first, she only experienced dyspnea on exertion, but over a few weeks, her symptoms progressed to dyspnea at rest. This was accompanied by fatigue, diffuse joint pain, and intermittent low-grade fever.

One month earlier, she had been started on hydroxychloroquine and oral corticosteroids as part of her lupus management.

Initial transthoracic echocardiography showed a preserved left ventricular systolic function, with an ejection fraction estimated at 69% and no regional wall motion abnormalities. Over the next few days, however, the patient's condition worsened rapidly. She developed signs consistent with low cardiac output along with increasing respiratory distress, which ultimately required transfer to the intensive care unit and initiation of mechanical ventilation.

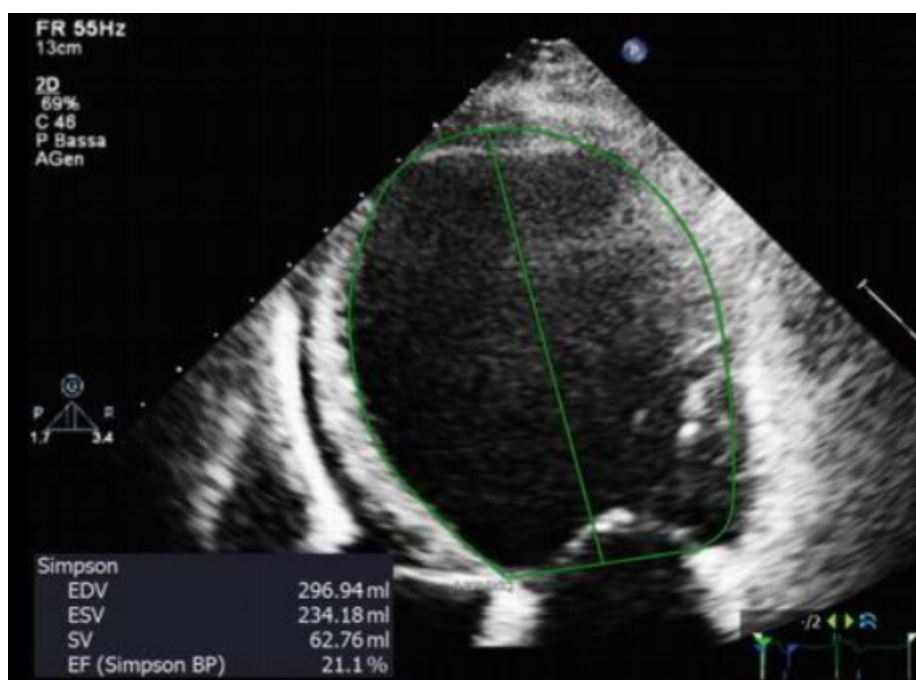


Figure 1: Baseline transthoracic echocardiography demonstrating preserved left ventricular systolic function (LVEF 69%)

A repeat echocardiogram performed at that stage revealed a marked decline in ventricular function, with the ejection fraction dropping to around 20–25%. The hypokinesia was diffuse and involved both ventricles, without any segmental pattern that would suggest an ischemic cause, making an inflammatory process such as myocarditis more likely.

Electrocardiography showed a complete atrioventricular block, with a narrow QRS escape rhythm at approximately 40–50 beats per minute, consistent with a junctional escape. There was also significant QT prolongation (QTc up to 650 ms), along with nonspecific repolarization changes, including biphasic and negative T waves in the lateral and inferior leads.

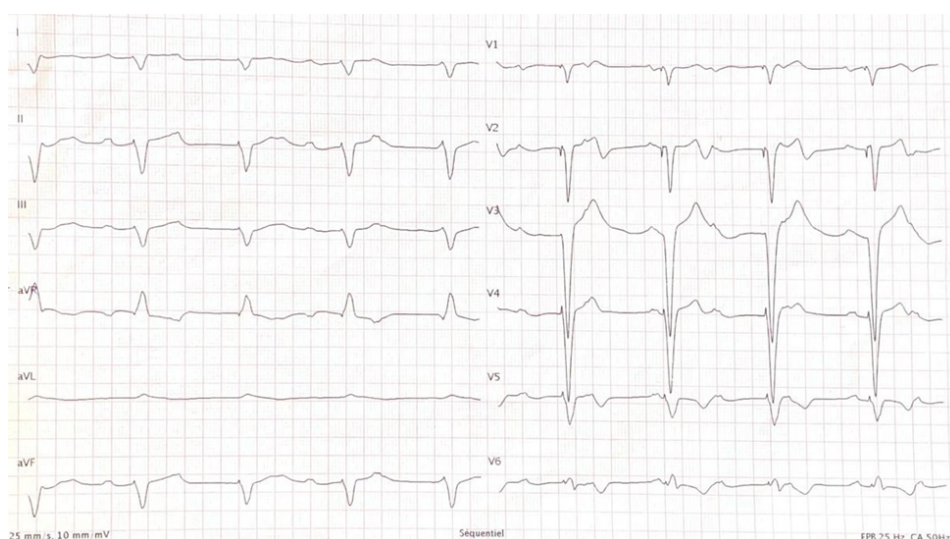


Figure 2: Electrocardiogram showing complete atrioventricular block with junctional escape rhythm.

Chest radiography during the acute phase showed cardiomegaly and bilateral alveolo-interstitial opacities, more pronounced on the right, in keeping with pulmonary edema in the context of cardiogenic shock. These findings gradually regressed as the patient's clinical status improved. In the setting of rapid hemodynamic deterioration and known SLE, fulminant lupus myocarditis was considered the most

likely diagnosis. Treatment with high-dose intravenous corticosteroids was initiated without delay, followed by cyclophosphamide. Hydroxychloroquine was discontinued given the marked QT prolongation. Because of the persistent complete AV block, a temporary transvenous pacemaker was placed.

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Over the following days, the patient showed progressive clinical improvement. Serial echocardiographic assessments demonstrated a gradual recovery of left ventricular systolic function, with the ejection fraction improving from 20% to

30%, then to 42%, and reaching approximately 48% within a few weeks. Right ventricular function also normalized, with improvement in filling pressures.

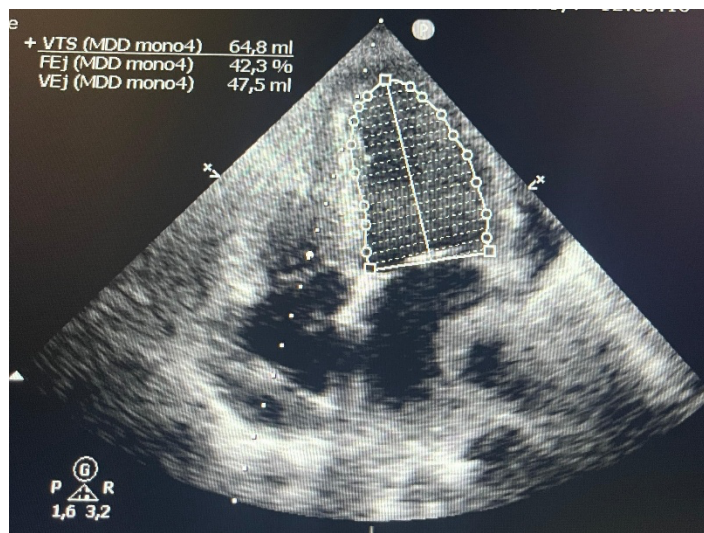


Figure 3: Follow-up transthoracic echocardiography demonstrating partial recovery of left ventricular systolic function (LVEF 42%).

Despite this recovery, the conduction abnormality persisted. Holter monitoring confirmed ongoing complete AV block with a ventricular escape rhythm around 43 beats per minute and clear AV dissociation. No sustained ventricular arrhythmias were recorded. As the patient remained hemodynamically stable and asymptomatic, the decision was made to defer permanent pacemaker implantation and continue close follow-up.

DISCUSSION

This case illustrates a severe and rapidly evolving form of lupus myocarditis. While cardiac involvement is reported in up to 50% of patients with SLE, clinically overt myocarditis remains uncommon and is often underdiagnosed due to its nonspecific presentation [1].

Fulminant myocarditis represents the most aggressive form, characterized by sudden and severe inflammation leading to acute heart failure and, in some cases, cardiogenic shock [4]. In SLE, myocardial injury is primarily driven by immune-mediated mechanisms, including immune complex deposition, complement activation, and inflammatory infiltration of the myocardium [1]. These processes result in myocyte damage and interstitial edema, ultimately impairing cardiac contractility.

The recovery of left ventricular function observed in this case is in line with what is generally reported in fulminant myocarditis when treatment is initiated early [2].

Cardiac conduction abnormalities are more frequently described in the context of neonatal lupus, where they are often permanent [5], although they are well recognized in SLE. In adult patients, these disturbances can sometimes

regress over time. In this case, their persistence raises the possibility of residual structural damage or an incomplete resolution of the inflammatory process.

Among the many possible diagnoses, including viral myocarditis, stress-induced cardiomyopathy, ischemic heart disease, and drug-related cardiotoxicity, the overall clinical picture strongly favored lupus myocarditis. The rapid clinical deterioration, the presence of diffuse rather than regional ventricular dysfunction, and the underlying autoimmune disease all supported this diagnosis [1,6]. Hydroxychloroquine toxicity appeared unlikely given the relatively short duration of exposure and the abrupt onset of symptoms.

Management in such cases depends on prompt recognition and early initiation of immunosuppressive therapy. High-dose corticosteroids remain the mainstay of treatment and are often combined with agents such as cyclophosphamide in more severe presentations [1,7]. At the same time, supportive care plays a crucial role during the acute phase, particularly in patients requiring respiratory support or temporary cardiac pacing.

From a prognostic standpoint, patients who survive the initial phase of fulminant myocarditis often show substantial recovery of ventricular function, as was seen in this patient. However, conduction abnormalities may persist and require long-term monitoring or eventual pacemaker implantation depending on symptoms and recovery [4].

CONCLUSION

Fulminant lupus myocarditis remains an uncommon but particularly severe presentation, with the potential to

deteriorate quickly into cardiogenic shock. In this case, the patient’s clinical course illustrates how timely initiation of intensive immunosuppressive therapy can result in a meaningful recovery of ventricular function. Continued follow-up remains crucial, especially for patients with persistent electrical disturbances.

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