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A Catastrophic Failure

To the Editor:

"Sick patients die" is a refrain familiar to all clinicians. It can be easily applied to a large number of patients admitted to the intensive care unit. This case represents one of those unfortunate clinical scenarios that we might face in our daily practice.

CLINICAL SUMMARY

A 17-year-old woman presented to the emergency department with a 4-day history of abdominal pain, nausea, and vomiting. Her symptoms started after she received the human papillomavirus vaccine 1 week earlier. She also had dyspnea and substernal chest discomfort. She had no medical or surgical history. She was taking no chronic medications and denied any current use of tobacco, alcohol, or illicit drugs.

On examination, the patient was hypotensive, tachypneic, and tachycardia with marked cool and clammy extremities. Her abdomen was soft and not tender. Lung auscultation revealed no crackles or wheezing. No murmurs were noted on cardiac auscultation. Her skin examination showed no rash.

ASSESSMENT

The patient was admitted to the intensive care unit, where active resuscitation with intravenous fluids, pressors, and wide-spectrum antibiotics was initiated. Laboratory tests showed a creatinine of 1.3 mg/dL, creatinine phosphokinase of 5492 mg/dL, serum aspartate aminotransferase of 831 IU/L, serum alanine aminotransferase of 211 IU/L, and troponin of 1.4. A second-degree atrioventricular block with fusion complex and ST elevation in lead I, aVL, V1 to V3 were seen on electrocardiogram (Figure 1). An echocardiogram showed severe global hypokinesia with an ejection fraction of 20%.

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DIAGNOSIS

Laboratory test results and electrocardiogram and echocardiogram findings indicated that she was in acute cardiogenic shock. The differential diagnosis was acute myocardial infarction, viral or idiopathic myocarditis, giant cell myocarditis, necrotizing eosinophilic myocarditis, and pericardium cardiomyopathy. Because definitive, successful therapy exists for some of these disorders, patients with these conditions must be distinguished from those with fulminant myocarditis. An urgent left-sided heart catheterization failed to show any abnormality within the coronaries. An intra-aortic balloon pump was placed, and aggressive medical supportive treatment was continued. Shortly thereafter, the patient developed respiratory failure that required intubation and mechanical ventilation, followed by ventricular fibrillation and cardiac arrest that was irreversible despite aggressive reanimation. On autopsy, there was diffuse lymphocytic infiltrate in the pericardium, myocardium, and endocardium consistent with acute fulminant lymphocytic panmyocarditis. A histologic image (Figure 2) of the myocardium shows the intense myocardial inflammation and necrosis with disruption of the striated muscle fiber (left side) compared with the preserved striated structure (right side).

MANAGEMENT

Myocarditis, whether fulminant or not, is an inflammatory condition of the myocardium that may be the caus-

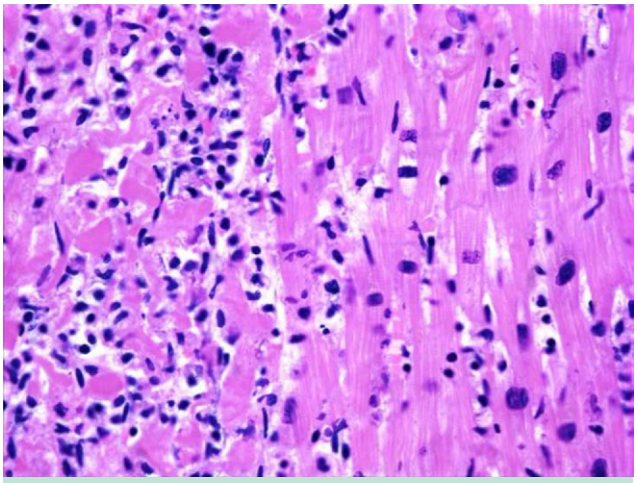

Figure 1 Histologic image showing the infiltration of the myocardium by lymphocytes and the disruption of the striated muscle fiber.

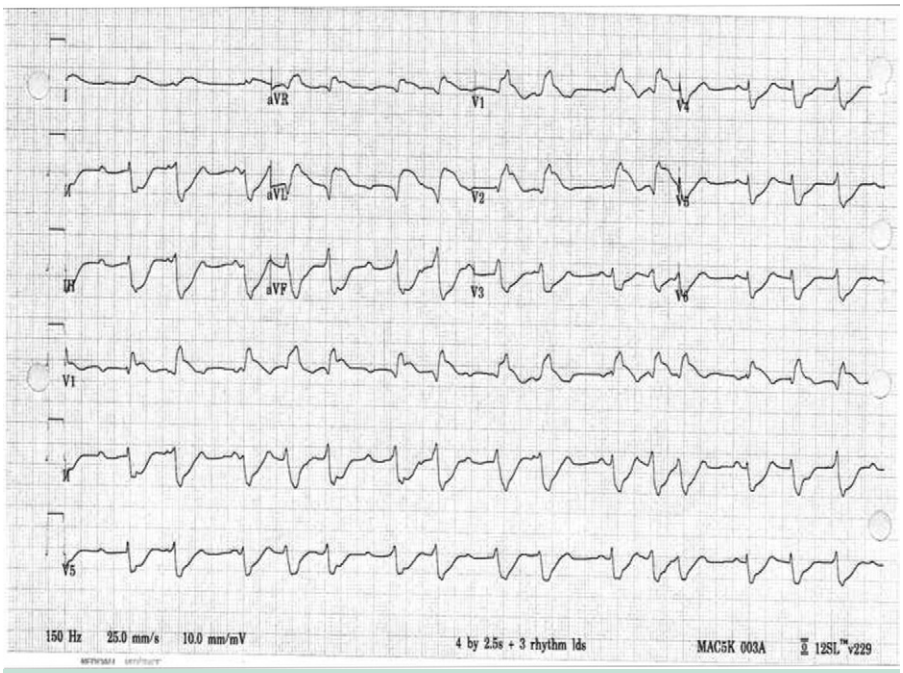

Figure 2 EKG showing the second-degree atrioventricular block with fusion complex and ST elevation in lead I, aVL, V1 to V3.

ative factor in up to 10% of patients presenting with acute-onset heart failure.[1] Patients with fulminant myocarditis commonly present with New York Heart Association class IV symptoms that is often proceeded by flu-like symptoms in 2 to 4 weeks. Our patient had no prodromal symptoms, but the fulminant myocarditis was after she received the human papillomavirus vaccine. No association between the human papillomavirus vaccine and myocarditis was found on review of the literature. Laboratory evidence of multiorgan failure is more common in patients with fulminant myocarditis compared to patients with nonfulminant myocarditis. Prolonged QRS complex and depressed left ventricular ejection fraction were both independently associated with the acute fulminant myocarditis.[2] A complete approach that includes clinical, echocardiographic, hemodynamic, and histologic information is required to distinguish between acute fulminant and nonfulminant myocarditis. There is no specific treatment for fulminant myocarditis. Figure 3 shows an algorithm describing the approach to patients with acute onset of heart failure.[3] The initial treatment is usually supportive with inotropic support; intra-aortic balloon pump and a ventricular assist device should be considered in refractory cases.

CONCLUSIONS

The role of immunosuppressive therapy remains unclear. The long-term survival of patients with fulminant myocarditis is excellent when the disease is diagnosed early and appropriate supportive therapy is initiated quickly.[4]

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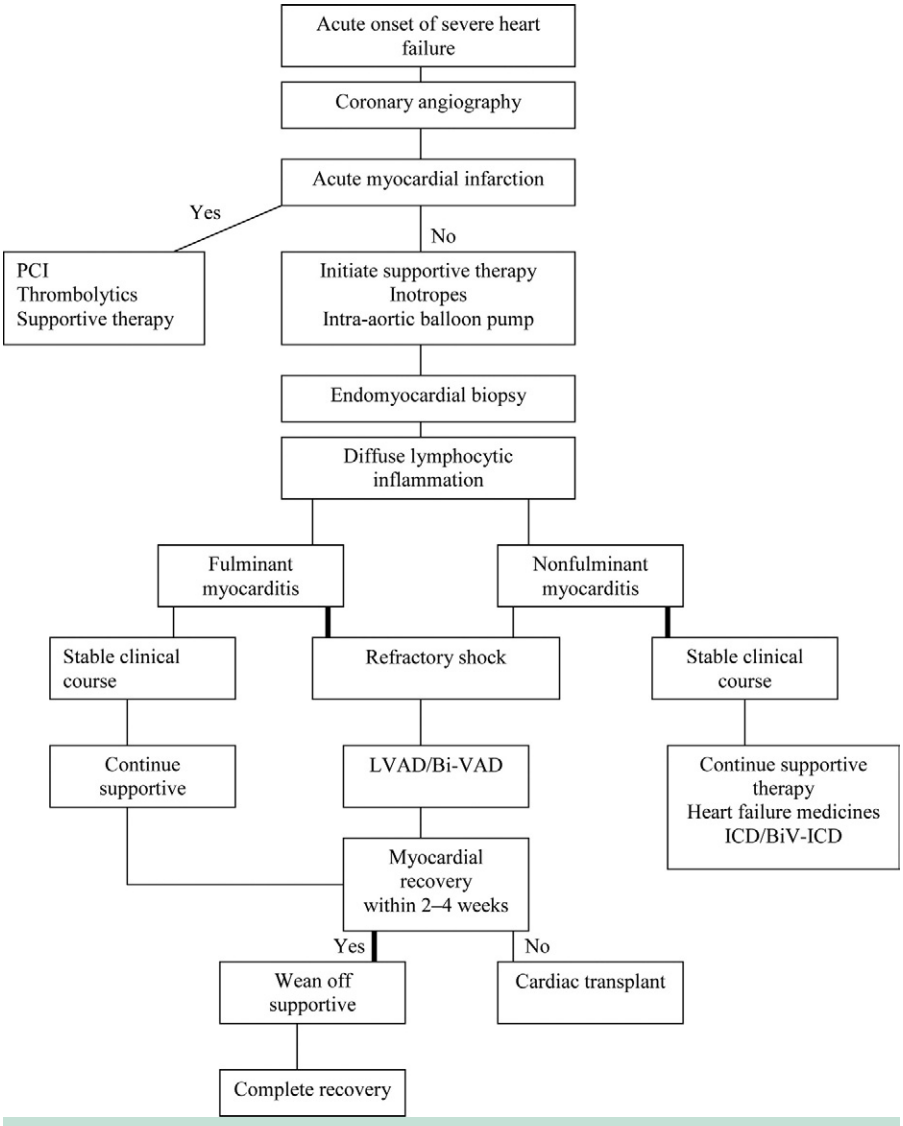

Figure 3 Clinical algorithm adopted from Gupta et al[3] that outlines the management of fulminant myocarditis. Bi-VAD = biventricular assist device; BiV-ICD = biventricular implantable cardioverter-defibrillator; ICD = implantable cardioverter-defibrillator; LVAD = left ventricular assist device; PCI = percutaneous coronary intervention.