A race against time: A case report of fulminant myocarditis leading to cardiogenic shock

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Background:
Fulminant myocarditis (FM) is the most severe form of acute myocarditis (AM), an inflammatory heart condition often triggered by viral infections or autoimmune disorders. It manifests as an uncommon syndrome encompassing various clinical presentations, including severe acute heart failure (HF), cardiogenic shock (CS), ventricular arrhythmias, or cardiac arrest. FM carries significant morbidity and mortality. This case report highlights the importance of early identification of FM, initiating end-organ support, and ensuring timely transfer to specialized facilities with mechanical circulatory support capabilities to improve patient outcomes and reduce complications.
Case presentation:
A previously healthy 40-year-old male presented to the Emergency Department (ED) with chest pain and worsening dyspnea over the past week. In the ED, he displayed diaphoresis, tachycardia, hypotension, and severe respiratory distress. An initial ECG showed sinus tachycardia with ST elevation and Q-waves in anterior leads. Point-of-care echocardiography (ECHO) revealed global hypokinesia and severe left ventricular (LV) dysfunction, with an estimated ejection fraction (EF) below 30%. A provisional diagnosis of cardiogenic shock was established, and he was promptly initiated on noninvasive positive pressure ventilation (NIV) and inotropic support. Initial lab tests indicated significantly elevated troponin and lactate levels, along with increased inflammatory markers, a high white blood cell count, and abnormal kidney and liver function. Due to inadequate improvement with NIV, he was intubated and ventilated in the ED. An urgent coronary angiography (CAG) was performed due to suspicion of acute coronary syndrome (ACS), but it showed non-obstructed coronary arteries. An intra-aortic balloon pump (IABP) was inserted as bridging therapy. He was admitted to the Cardiac Intensive Care Unit (CICU), where a formal ECHO revealed low stroke volume and normal LV size, leading to the diagnosis of FM. Viral titers and pan cultures were unremarkable. Cardiac magnetic resonance imaging (MRI) was not pursued; instead, efforts focused on maintaining end-organ perfusion with IABP and inotropes. On the second day of admission, an early transfer to a specialized center for Veno-arterial (VA) Extracorporeal membrane oxygenation (ECMO) was initiated, with the receiving team cannulating the patient at the primary facility. Subsequently, he underwent an endomyocardial biopsy, which confirmed the diagnosis of giant cell myocarditis. He received pulse-dose steroids and immunosuppressive therapy in addition to being on mechanical circulatory support, and his condition continued to improve significantly.
Conclusion:
This case underscores that myocarditis, even in its severe forms like FM, can be cured. Early recognition is vital for improving the disease's natural course and prognosis. Emergency Medicine physicians, often the first to encounter FM, should remember that the early initiation of inotropes and mechanical circulatory support are life-saving tools in its treatment and can potentially lead to improved long-term outcomes.