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From ventricular thrombosis to cardiogenic shock: Fulminant eosinophilic myocarditis in eosinophilic granulomatosis with polyangiitis

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Introduction: Fulminant eosinophilic myocarditis is a rare, life-threatening manifestation of eosinophilic granulomatosis with polyangiitis (EGPA), potentially leading to severe myocardial dysfunction and cardiogenic shock. Early recognition is particularly challenging in patients with pre-existing coronary artery disease, where inflammatory myocardial injury may be misattributed to ischemic cardiomyopathy.[1]

Case report: 67-year-old woman with bronchial asthma, type 2 diabetes mellitus, and a previous non-ST-elevation myocardial infarction treated with right coronary artery stenting presented with progressive dyspnea, profound weakness, anorexia, and fever. During her previous hospitalization, an apical left ventricular thrombus had been detected despite the absence of overt apical akinesia. Peripheral eosinophilia had also been noted. On admission, she was critically ill, hypotensive (70/50 mmHg), tachycardic (110 beats/min), and hypoxemic (SpO_2 87%). Cardiogenic shock necessitated norepinephrine and inotropic support. Laboratory findings revealed leukocytosis (22.7 × 10^9/L), profound eosinophilia (36%; absolute count 8.17 × 10^9/L), markedly elevated high-sensitivity troponin T (2,020 ng/L), NT-proBNP (2,236 pg/mL), and C-reactive protein (72.3 mg/L). Echocardiography demonstrated severe left ventricular systolic dysfunction (LVEF 30%), myocardial thickening, and minimal pericardial effusion. The constellation of cardiogenic shock, marked eosinophilia, myocardial injury, longstanding asthma, and acral vasculitic manifestations raised strong suspicion of EGPA-associated fulminant eosinophilic myocarditis. Retrospectively, the preceding ventricular thrombosis suggested possible earlier eosinophil-mediated cardiac involvement. Empirical high-dose intravenous methylprednisolone pulse therapy was promptly initiated alongside hemodynamic support. Multidisciplinary evaluation involving clinical immunology guided further diagnostic workup and immunosuppressive management. Corticosteroid-associated psychiatric complications required treatment adjustment. Endomyocardial biopsy was deferred following clinical improvement after individualized risk–benefit assessment, while cardiac magnetic resonance imaging remained pending. Progressive hemodynamic recovery was achieved, with LVEF improving to 40–45% before discharge. Mepolizumab (300 mg subcutaneously every four weeks) was subsequently introduced for long-term disease control. At follow-up, the patient remained clinically well, with resolution of peripheral eosinophilia despite mild residual left ventricular systolic dysfunction.

Conclusion: EGPA-associated eosinophilic myocarditis should be considered in patients presenting with cardiogenic shock and profound eosinophilia, even in the presence of established coronary artery disease. Preceding ventricular thrombosis may represent an early warning sign of eosinophilic cardiac involvement. Prompt corticosteroid therapy, hemodynamic support, and multidisciplinary management are crucial for favorable clinical outcomes.

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