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Extracorporeal membrane oxygenation associated with steroids as bridge to recovery in cardiogenic shock due to necrotizing eosinophilic myocarditis as first manifestation of Churg-Strauss syndrome

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Introduction: Necrotizing eosinophilic myocarditis (NEM) is a rare condition with poor prognosis. NEM can be the first manifestation of a systemic disease.

Case report: A 25-year-old Caucasian man with a history of allergic rhinitis developed rapidly progressive dyspnoea with severe biventricular dysfunction (left ventricular ejection fraction – LVEF-10%), elevated levels of C-reactive protein and troponin T (4,381 ng/L), evolving in a cardiogenic shock unresponsive to inotropes and intraaortic counterpulsation (IABP). Mechanically assisted ventilation was instituted, and in a hybrid operating room, peripheral veno-arterial extracorporeal membrane oxygenation (vaECMO) was positioned, while transeosophageal echocardiography-guided right ventricle endomyocardial biopsy (EMB) was performed. The histology of diffuse necrotizing interstitial eosinophilic infiltrates, associated with peripheral eosinophilia (64%) supported the diagnosis of NEM. The patient was treated with 500mg iv methylprednisolone daily for 3 days, followed by 1 mg/kg prednisolone daily. Eleven days after initiation of mechanical and inotropic support and steroid therapy, LVEF recovered up to 40% and mechanical supports were removed. Cardiac CT demonstrated the absence of coronary artery involvement. Cardiac magnetic resonance (CMR) confirmed the diagnosis of myocarditis (myocardial oedema on STIR images and at T1-/T2-mapping). Late post-contrast images showed diffuse areas of myocardial enhancement, with typical endomyocardial involvement. CMR also showed a pulmonary infiltrate, which subsequently resolved. At 1 month, EMB showed no eosinophilic infiltrates and diffuse areas of fibrosis. The patient was discharged on oral prednisone, methotrexate and heart failure (HF) treatment; LVEF was 35%. The diagnostic suspect was ANCA-negative Churg-Strauss syndrome (criteria: eosinophilia>10%, extravascular eosinophils, pulmonary infiltrates, allergic rhinitis). Genetic analysis resulted negative for mutation of the FIP1L1/PDGFRA associated with hypereosinophilic syndrome. CMR scan after 3 months showed evolution with LV dilation and LVEF 36%. Wall thickness was reduced, and late enhancement was unchanged.

Discussion: Other conditions that could sustain an acute NEM include a hypersensitivity reaction (DRESS syndrome), nevertheless there are no reports of myocarditis associated with clarithromycin (which the patient had taken before hospital admission) use. Malignant disease, parasitic infections were excluded.

Conclusions: Heart team for advance HF and multidisciplinary diagnostic tools are needed to treat rare but potentially fatal myocarditis as the NEM.