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Fulminant myocarditis: a severe manifestation of systemic lupus erythematosus

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Introduction: Myocarditis is a rare manifestation of systemic lupus erythematosus (SLE), affecting 5–10% of patients. It is usually subclinical but fulminant cases have been reported. Common symptoms include fever, tachycardia, chest pain and dyspnoea. Diagnosis is based on clinical presentation combined with diagnostic imaging (echocardiography, cardiac magnetic resonance imaging) and elevated cardiac biomarkers after excluding other causes.

Case description: A 35-year-old man with a history of chronic discoid lupus erythematosus (DLE) was admitted to the Rheumatology Ward due to recurrent fever, unintentional weight loss, fatigue and alopecia lasting six months. Three weeks prior to hospitalization he developed aggravation of skin lesions, facial and lower limbs oedema, muscle weakness, morning joints stiffness, exertional dyspnoea and dry cough. Physical examination revealed decreased breath sounds at lung bases and tachycardia. Point-of-care ultrasound demonstrated myocardial hypokinesis with wall thickening and pleural effusion. Laboratory tests showed elevated aspartate transaminase, creatine kinase, troponin I (TnI) and N-terminal pro-B-type natriuretic peptide (NT-proBNP), as well as decreased complement components C3 and C4. Anti-double-stranded DNA (anti-dsDNA) antibodies were positive (49 IU/ml), while C-reactive protein was relatively low (16.3 mg/l). Hyperferritinemia and hypertriglyceridemia suggested macrophage activation syndrome (MAS), but other criteria were negative. The diagnosis of SLE was confirmed (cSLEDAI-2K = 57). Initial treatment with intravenous methylprednisolone and immunoglobulins (IVIG) was ineffective. A rise in TnI (31–141 ng/l) and markedly elevated NT-proBNP (> 35,000 pg/ml) correlated with a decline in left ventricular ejection fraction (LVEF) to 15%, indicating myocarditis. The patient was referred to the intensive care unit as he required mechanical ventilation and catecholamine support. The first dose of cyclophosphamide resulted in a significant clinical response, so after 10 days the patient returned to the Rheumatology Ward. After one month of hospitalization patient was discharged with mildly reduced LVEF (45%) and no severe symptoms.

Conclusions: This case highlights that acute heart failure in SLE patients should indicate evaluation for myocarditis. Broad immunological and clinical assessment enables timely initiation of immunosuppressive therapy, including high-dose glucocorticosteroids and cytotoxic agents such as cyclophosphamide, to control immune-mediated organ damage.