

Abstract

Beyond Chest Pain: Pericarditis as the Initial Manifestation of Systemic Reactivation in a Patient with Systemic Lupus Erythematosus

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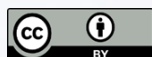
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Introduction: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterized by periods of remission and disease activity, with the potential to involve multiple organs simultaneously or sequentially. Cardiovascular involvement is common, with pericarditis being one of the most recognized cardiac manifestations. In patients with previously diagnosed SLE, the development of pericarditis may represent disease reactivation and precede the recognition of involvement of other organs. **Case Report:** We present the case of a 19-year-old female patient with a previous diagnosis of SLE, under regular follow-up and treated with hydroxychloroquine, prednisolone, and azathioprine, who had experienced recurrent episodes of chest pain in the preceding weeks. She presented to the emergency department because of worsening symptoms, including asthenia, fever, and severe retrosternal chest pain characterized as sharp and non-radiating, aggravated in the supine position and relieved by sitting upright. On admission, she was in poor general and nutritional condition, febrile and tachycardic, with a blood pressure of 104/56 mmHg. Laboratory tests revealed moderate microcytic hypochromic anemia (hemoglobin, 8.8 g/dL), leukocytosis (11,940 cells/mm³), and negative troponin I. Electrocardiography revealed sinus tachycardia, diffuse ST-segment elevation, and PR-segment depression, findings consistent with acute pericarditis. Chest radiography showed no significant abnormalities. Transthoracic echocardiography demonstrated preserved global biventricular systolic function, mild pericardial thickening, and circumferential pericardial effusion. The combination of chest pain with characteristic pericardial features, typical electrocardiographic changes, and pericardial effusion established the diagnosis of acute pericarditis. During further investigation, abnormalities in renal function suggestive of possible renal involvement by SLE were also identified, raising the possibility of systemic disease activity initially manifested by pericardial involvement. Given the unavailability of intensive immunosuppressive therapy with pulse therapy, the oral corticosteroid dose was increased, combined with ibuprofen and colchicine, while maintenance SLE therapy was continued. The patient remained hospitalized in the Cardiology Department for four days, with favorable clinical evolution, and was discharged in improved condition with follow-up arranged in the Cardiology and Rheumatology clinics. **Conclusion:** Acute pericarditis may constitute the initial manifestation of systemic SLE reactivation, including in patients receiving regular treatment and follow-up. In this case, pericardial involvement preceded the identification of renal abnormalities suggestive of involvement of a second organ, reinforcing the pos-

sibility of multisystem disease activity. Recognition of the characteristic features of the chest pain, together with electrocardiographic and echocardiographic findings, was essential for establishing the diagnosis. This case also highlights the therapeutic challenges associated with managing lupus disease activity in settings with limited availability of intensive immunosuppressive therapy.



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