Rheumatoid arthritis presenting as acute myopericarditis

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To the Editor

A 50-year-old man with hypertension, type-2 diabetes, gout, and treated latent tuberculosis infection presented to the Emergency Department with 12 hours of angina. Initial evaluation for acute coronary syndrome and pulmonary embolism were negative. However, 20 hours after hospital admission the patient developed substernal chest pain, 1mm ST-segment elevation in the inferior ECG leads, and repeat troponin was 4.67 ng/mL (normal: < 0.09 ng/mL). Transthoracic echocardiography demonstrated normal biventricular function with no regional wall motion abnormalities. Urgent coronary angiogram revealed angiographically normal coronary arteries and no evidence of plaque rupture, coronary embolization, or dissection. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were 97 mm/Hr and 305.8 mg/L, respectively. The patient was treated for myopericarditis with indomethacin and colchicine, but continued to experience symptoms of pleuritic chest pain, dyspnea, malaise, poor appetite, night sweats, and weight loss over the next several weeks. Cardiac MRI eight weeks after hospital discharge confirmed myocardial inflammation with residual, hemodynamically insignificant pericardial effusion and the interval development of a large left sided pleural effusion (Figure). Laboratory investigation showed persistently elevated ESR (86 mm/Hr) and CRP (63.2 mg/L), positive rheumatoid factor (82 IU/mL), negative antinuclear antibody, and negative Quantiferon-GOLD. Diagnostic thoracentesis
revealed bloody, lymphocyte predominant pleural fluid with negative bacterial, fungal, and viral cultures as well as negative cytology.

The differential diagnosis for exudative pleural effusions includes malignancy, infection, and systemic inflammatory diseases. Bloody pleural effusions are due to underlying malignancy in approximately 50% of cases. Lymphocytic effusions (>80% lymphocytes) can be caused by malignancy (lymphoma), tuberculosis, sarcoidosis, chylothorax, rheumatoid arthritis (RA), and yellow-nail syndrome \(^1,^2\).

Our patient’s pleural fluid cultures and cytology were negative. Adenosine deaminase was low (14.5 U/L) and active tuberculosis was ruled out with three negative acid-fast bacilli sputum cultures. The serum anti-cyclic citrullinated peptide antibody level was found to be markedly elevated (>250 U), establishing the diagnosis of RA.

The patient’s pleural effusion resolved spontaneously and has not recurred during six months of follow-up. Initial laboratory abnormalities, including anemia, thrombocytopenia, acute kidney injury, and elevated inflammatory markers, normalized without immunosuppressive therapy. However, over the following months the patient developed increasing arthralgia, particularly in his wrists and elbows, and was started on azathioprine.

RA is a progressive, systemic inflammatory disease that usually develops insidiously. Cardiac manifestations of RA include pericarditis, myocarditis, and coronary vasculitis \(^3\). Postmortem studies document much higher rates of cardiac involvement than are observed clinically. For instance, while 30–50% of patients with RA have postmortem evidence of pericarditis \(^4\), pericarditis as a clinical manifestation of RA occurs in fewer than 10% of patients \(^3\). Cardiac MRI studies also suggest that subclinical myocardial abnormalities are not uncommon in RA patients \(^5\). Although it is not uncommon for patients with known RA to suffer cardiac or pulmonary complications, it is rare for the initial presentation of RA to be myopericarditis.

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References

Figure. Cardiac MRI demonstrates thickening of the apical segments of the left ventricular myocardium with associated patchy delayed enhancement, as well as diffuse thickening and delayed enhancement of the pericardium.