

Pleuropericardial Effusion: A Rare Onset of Rheumatoid Arthritis

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Dear Editor,

Pulmonary involvement is one of the most common extra-articular findings in rheumatoid arthritis (RA) where parenchymal lung disease and pleural disease are reported to be up to 79% and 50%, respectively.¹ Recent data suggests that RA-related autoimmunity may initiate in a mucosal site many years before the onset of joint symptoms, suggesting that the lung is one of these mucosal sites.² A 68-year-old male patient was admitted with dyspnea and chest pain that had started a week earlier. Lung sounds had decreased on the middle and lower zone of right lung on auscultation and chest X-ray revealed bilateral pulmonary effusion which was more prominent on the right lung. Pleural fluid reached a thickness of 9 cm on the right and 2 cm on the left, sub-segmental atelectasis, septal thickening and pericardial effusion were observed on chest computed tomography. Laboratory tests marked for normocytic anemia (hemoglobin: 9.0 g/dL), leukocytosis [white blood cell (WBC): 16,200/ μ L] and elevated acute phase reactants, erythrocyte sedimentation rate (ESR): 98 mm/h and C-reactive protein (CRP): 143 mg/dL (normal: 0–5 mg/L). Transthoracic echocardiography showed an ejection fraction of 65%, and a moderate pericardial effusion which measured 14 mm at its widest point. Sputum microscopy, sputum acido-resistant basil and sputum culture tests were negative for the exclusion of tuberculosis and other possible infections. Thoracentesis was performed and pleural fluid was exudative, pleural adenosine deaminase and mycobacterium polymerase chain reaction were negative. In pleural fluid cytology, 60% lymphocytes and 40% polymorphonuclear leukocytes were observed, and no microorganisms or malignant cells were found. ANA was positive at 1/100 titer of chromosomal dense fine speckled (DFS) pattern. Rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibody were positive at high titers 108.5 IU/mL (normal: 0–7 IU/mL) and 195.6 U/mL (normal: <4.5 U/mL), respectively. The patient had no previous history

of arthritis. Rheumatologic examination was unremarkable and there were no features on the hand X-ray. Methylprednisolone was initiated (0.5 mg/kg/day) and pleural and pericardial effusion improved on the control chest X-ray and echocardiography. During the one-year follow-up, the patient did not experience any joint symptoms.

RA causes joint erosions and deformities, more frequently in seropositive cases. It is crucial to diagnose and manage the disease in the “opportunity window” in order to prevent irreversible damage.³ In recent years, growing data suggests that RA may originate from mucosal regions such as the respiratory mucosa, gastrointestinal mucosa or even the urogenital mucosa.² Additionally, extra-articular involvements preceding or occurring without articular manifestations, commonly as interstitial lung disease, have been reported.⁴ To the best of our knowledge, this case is the first pleuropericardial effusion occurring without articular disease and with positive RA-related autoantibodies. Patients with atypical presentation and those with RF and ACPA positivity, as was the case with our patient, may develop joint findings during follow-up. However, it is an issue of debate as to whether these patients should be classified and managed as RA in the initial period with such extra-articular presentations.

Keywords: Pericardial effusion, pleural effusion, rheumatoid arthritis

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