

## Diagnosis of rheumatoid arthritis based on clinical and serological findings

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### ABSTRACT

In this report, we report a case who was hospitalized in the internal medicine clinic with pneumonia. Based on clinical and serological findings, the definitive diagnosis of rheumatoid arthritis was made and the inflammatory process was suppressed with steroid therapy.

**Keywords:** Inflammatory arthritis, interstitial lung disease, rheumatoid arthritis, symmetrical polyarticular.

Although rheumatoid arthritis (RA) is a chronic systemic inflammatory disease with severe joint involvement, extra-articular involvement has been reported with a rate of 17.8 to 50% in various studies.<sup>[1,2]</sup> Pulmonary involvement is one of the common findings of RA and can cause serious mortality and morbidity.<sup>[3]</sup> Rheumatoid lung disease may present with different clinical manifestations such as pleuritis, pleural effusion, pneumonia, interstitial lung disease, pulmonary nodules, and bronchiectasis. In this article, we report a case of RA presenting with pneumonia.

### CASE REPORT

A 75-year-old female patient who was evaluated in the emergency department with complaints of weakness, generalized body pain, and cough was initially hospitalized in the internal medicine clinic to investigate the etiology of acute renal failure and pneumonia. On further investigation, the patient complained of not being able to walk for two months. It was learned from the patient's history that she was followed for 15 years for coronary artery

disease and for 12 years due to hypertension. On physical examination, suprapubic tenderness, coarseness in bilateral lung sounds, and bilateral crepitant rales were detected. There was pain and tenderness in the right and left shoulder joints, right and left elbow joints, and all right and left metacarpophalangeal joints. Laboratory examinations were as follows: white blood count (WBC):  $27.5 \times 10^9$  u/L, neutrophil:  $23.9 \times 10^9$  u/L, hemoglobin: 9.9 g/dL, mean corpuscular volume: 79 fL, platelet count:  $525 \times 10^9$  u/L, urea: 163 mg/dL, creatinine: 2.6 mg/dL, sodium: 132 mmol/L, potassium: 5.89 mmol/L, chloride: 106 mmol/L, calcium: 8.7 mg/dL, phosphate: 4.5 mg/dL, magnesium: 2 mg/dL, procalcitonin: 1.53 µg/L, C-reactive protein (CRP): 345 mg/L, erythrocyte sedimentation rate (ESR): 129 mm/h, and ferritin: >450 µg/L. Arterial blood gas analysis results were as follows: pH: 7.28, partial pressure of carbon dioxide: 31.8 mmHg, bicarbonate: 15.5 mmol/L, lactate: 0.8 mmol/L, and potassium: 5.8 mmol/L. Thoracic computed tomography (CT) showed a 3-cm-thick loculated effusion in the right pleural

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space, and pleuroparenchymal, locally fibrotic changes in the upper, middle and lower zones in both hemithoraces, and signs of reticulation in the subpleural area, compatible with new-onset interstitial lung disease. The pleural fluid sampling revealed no growth in the pleural fluid culture, and pleural fluid biochemical examinations were as follows: lactate dehydrogenase: 952 U/L, glucose: 48 mg/dL, sodium: 142 mmol/dL, albumin: 1.7 g/dL, WBC:  $0.6 \times 10^3/\mu\text{L}$ , and neutrophil count:  $0.3 \times 10^3/\mu\text{L}$ . It was initially thought that the significant increase in inflammatory markers at the time of hospitalization might be due to urinary tract infection and pneumonia. Urinalysis and urine culture were performed. Piperacillin-tazobactam 2.25 g q.i.d. and 2 mL/kg/h intravenous hydration were started. Since leukocyte and nitrite were negative in the urinalysis and no growth was detected in urine culture, the preliminary diagnosis of urinary infection was ruled out. Control CRP values at 48 and 96 h of antibiotherapy were 274 mg/L and 259 mg/L, respectively. The control ESR was 118 mm/h. Since the desired acute phase reactant response could not be obtained despite antibiotherapy and the complaints of joint pain continued, rheumatological markers were analyzed and the results were as follows: rheumatoid factor (RF): 1150 U/mL, myeloperoxidase-antineutrophil cytoplasmic antibodies (ANCA): negative, C3: 1.53 g/L, C4: 0.48 g/L, anti-cyclic citrullinated peptide (anti-CCP):  $>200$  U/mL, c-ANCA (-), p-ANCA (-), anti-double-stranded deoxyribonucleic acid: 5.5 IU/mL, and antinuclear antibody: negative. With possible RA diagnosis according to the rheumatology recommendation, 4 mg methylprednisolone was started. Since anti-hepatitis B core protein antibody immunoglobulin G was found to be positive, entecavir 0.5 mg was started for hepatitis B prophylaxis. After 4 mg methylprednisolone, control CRP and procalcitonin values decreased. The methylprednisolone dose was increased to 8 mg. The decline in the CRP values continued (CRP: 259.8 mg L-118.1 mg/L-67 mg/L-46.6 mg/L). Renal function tests and metabolic acidosis of the patient regressed after hydration and antibiotherapy. Antibiotherapy was discontinued on the seventh day, due to the decrease in joint pain and regression of inflammation markers under 8 mg methylprednisolone treatment. She

was discharged with the recommendation of rheumatology outpatient clinic control. At one month after discharge, ESR was 66 mm/h and CRP was 25 mg/L.

A written informed consent was obtained from the patient for all diagnostic and therapeutic procedures.

## DISCUSSION

The experience gained from basic and clinical research over the last two decades has been enlightening for the diagnosis and management of RA. In an appropriate clinical picture, when RA is suspected, requesting anti-CCP and RF as prognostic biomarkers provides a great advantage in the diagnosis.<sup>[4]</sup> It has been shown that positivity in biomarkers such as RF and anti-CCP may precede the clinical diagnosis of RA.<sup>[5]</sup>

Initially, mostly symmetrical involvement of the small joints of the hands and feet, and normal physical activity following joint stiffness within 1 h of waking up in the morning are supportive for the diagnosis of RA.<sup>[4]</sup> In our case, in addition to polyarticular symmetrical small joint involvement, anti-CCP and RF positivity, which may indicate an active RA disease, also supported the diagnosis.

Extraarticular manifestations are important in the clinical course of RA. Interstitial lung disease associated with RA is considered one of the major causes of mortality, although it is a rare extra-articular manifestation of RA.<sup>[6]</sup> Uncontrolled RA disease is thought to be one of the main risk factors for the development of interstitial lung disease associated with RA, as in our patient.<sup>[6]</sup>

The symptoms of interstitial lung disease associated with RA exhibit a wide spectrum ranging from asymptomatic to fatal acute interstitial pneumonia, while the most common symptoms are exertional dyspnea and dry cough. Pulmonary hypertension and subsequent hypoxic respiratory failure may develop in the advanced stages of the disease.<sup>[6]</sup>

In our case, the fact that acute phase reactants did not regress with antibiotherapy, and no growth was found in the urine and pleural fluid cultures, rheumatological diseases were considered in the differential diagnosis. The regression in

acute phase reactants after methylprednisolone treatment and the decrease in joint pain of the patient day by day were explained by RA in the light of clinical and serological findings. On the other hand, the lung CT findings were also found to be compatible with the initial stage of interstitial lung disease.

In conclusion, the presence of symmetrical polyarticular inflammatory arthritis, the marked increase of clinical, specific serological, and inflammatory markers, and clinical and laboratory response after steroid therapy seem to be supportive of the diagnosis of RA.

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