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Spontaneous Pneumothorax as an Evolutionary Manifestation of Rheumatoid Arthritis: A Case Report and Review of the Literature

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Abstract | **Case Report**

Rheumatoid arthritis (RA) is an inflammatory autoimmune disease that can lead to pulmonary involvement, particularly subpleural nodules. We report the case of a 28-year-old patient with seropositive RA who developed a spontaneous left-sided pneumothorax. Chest CT revealed excavated pulmonary nodules. The evolution was favorable after thoracic drainage. Anti-TNF alpha biotherapy was initiated after exclusion of active tuberculosis. This case illustrates a rare but serious complication of RA. It highlights the importance of pulmonary surveillance in these patients.

Keywords: Rheumatoid Arthritis, Pneumothorax, Pulmonary Nodule.

INTRODUCTION

Rheumatoid arthritis (RA) is a multisystem inflammatory disease characterized by destructive synovitis and extra-articular systemic lesions. The latter include pleuropulmonary manifestations, mainly in the form of diffuse interstitial lung disease, rheumatoid pulmonary nodules or chronic pleurisy [1]. Rheumatoid nodules may evolve into spontaneous pneumothorax. We report the case of a young woman with a long history of rheumatoid arthritis who presented with a spontaneous pneumothorax.

CASE REPORT

A 28-year-old woman was admitted to our clinic as an emergency patient with abrupt onset of dagger-like left chest pain associated with dyspnoea at rest. There was no smoking, occupational exposure or pulmonary tuberculosis in her history. In addition, there was a long history of seropositive rheumatoid arthritis since 2013 initially treated with methotrexate for 2 years, discontinued for ineffectiveness in 2015, then put on corticosteroid therapy for 5 years, and Leflunomide (20 mg/d) for 10 months. The patient reported exertional dyspnea that had been evolving for a year. On admission, examination revealed a left aeric effusion syndrome with supra-sternal tugging. In addition, the patient was experiencing a flare-up of her chronic rheumatism. Osteoarticular examination revealed a peripheral joint syndrome with a joint index of 18 and a synovial index of 7, as well as a spinal syndrome. The thoracic CT scan showed a large, compressible left pneumothorax with foci of condensation excavated in the culmen and left fowler. The blood count showed moderate hyperleukocytosis, predominantly neutrophils, and lymphopenia at 780/mm3. CRP was elevated to 44 mg/l, sedimentation rate was accelerated to 110 mm, cortisolemia was normal, cytobacteriological examination of sputum and urine was negative, and Xpert gene in sputum was negative, as was tuberculin intradermal reaction. Viral serologies were negative. The patient underwent thoracic suction drainage combined with low-flow oxygen therapy and analgesic treatment. Corticosteroid therapy was continued during hospitalization, and she was put on a probabilistic antibiotic regimen of amoxicillin/clavulanic acid 3g daily, with boluses of methylprednisone for 3 days for her rheumatic attack. The evolution was marked by a return of the lung to the wall (Figure2), with normalization of the biological work-up. Two months later, a follow-up chest CT scan revealed left apical scissural nodular thickening with a band of apicodorsal nodular atelectasis, and bilateral apical nodules. Flexible bronchoscopy revealed diffuse 1st-degree inflammation, with no other abnormalities, and staged biopsies showed non-specific chronic inflammatory changes. Bacteriological tests on bronchial aspirates were negative. Spirometry was normal and the 6-minute walk test was maintained at term without desaturation. Treatment with leflunomide was discontinued and anti TNF-alpha biotherapy was started after ruling out tuberculosis.

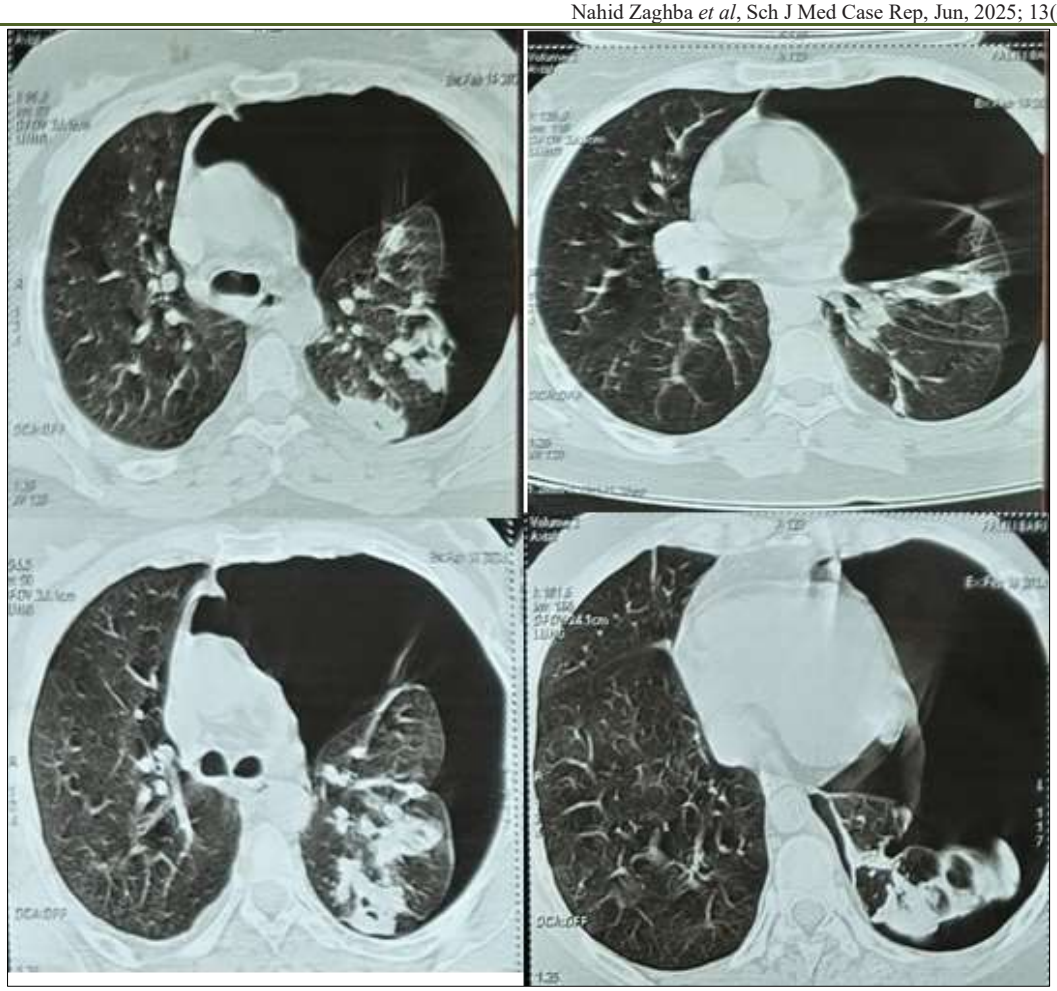

Figure 1: Chest CT scan on admission showing a large compressible left pneumothorax with foci of excavated condensation and pulmonary nodules

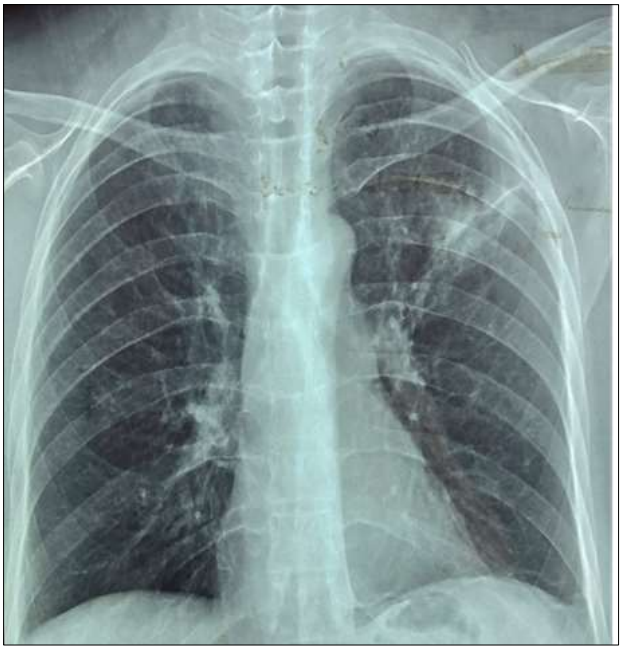

Figure 2: Chest X-ray after pneumothorax drainage, showing the lung returning to the wall

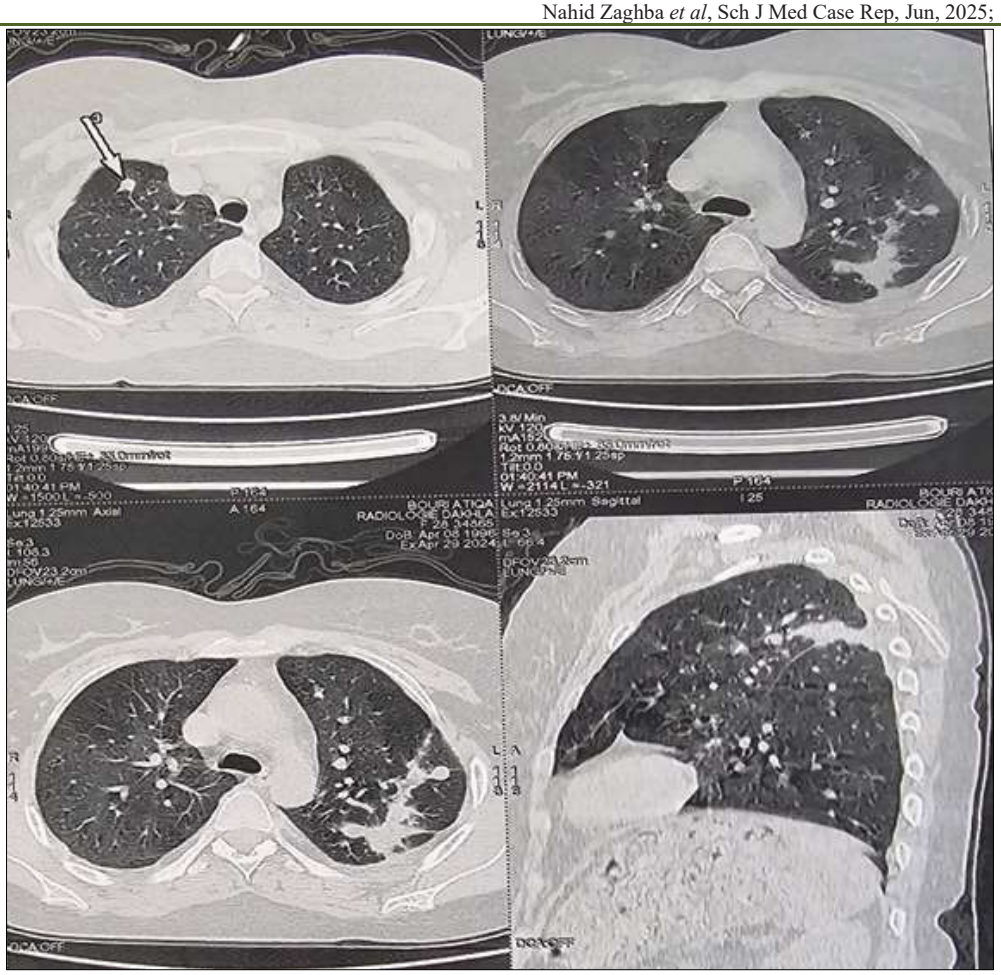

Figure 3: Chest CT scan showing left apical scissural nodular thickening with a band of apicodorsal nodular atelectasis, and bilateral apical nodules

DISCUSSION

Rheumatoid arthritis (RA) is a chronic inflammatory disease characterized by progressive, symmetrical, erosive involvement of small joints. It is the most common autoimmune joint disease, affecting 1% of the general population. Various extra-articular complications may arise, of which pulmonary manifestations are the most frequent, with a prevalence of up to 60% [1, 2]. All components of the respiratory system may be involved: parenchyma, pleura, vessels and airways [2]. Pulmonary rheumatoid nodules are found in 4% to 20% of patients, depending on the series, and are most often subpleural in location. Symptomatic pleural manifestations are less frequent, occurring in 3-5% of cases [1]. Pneumothorax is a rare complication in RA, secondary to rupture of pulmonary rheumatoid nodules. Pneumothorax occurs in less than 0.5% of RA patients [3].

Rheumatoid nodules may appear in the lungs, particularly in patients with long-standing disease and subcutaneous nodules. Their structure is identical to that of cutaneous nodules, and they consist histologically of a central necrotic zone bordered by histiocytes. They are generally located along the interlobular septa or in subpleural regions [2]. Nodules may be single or multiple, ranging in size from a few millimeters to several centimeters. Pathologically, they consist of necrobiotic lesions of giant cells within palisading foci, which produce pro-inflammatory cytokines similar to those of the synovial membrane [1]. The presence of nodules is associated with increased severity of RA and a high risk of vasculitis, hospitalization and mortality [2].

Nodules are typically asymptomatic unless they become cavitated, superinfected or rupture. In such cases, hemoptysis, spontaneous pneumothorax, empyema, pleural effusion or bronchopleural fistula may occur. Uncomplicated nodules may regress spontaneously or improve with standard rheumatoid arthritis treatment, in particular tocilizumab and rituximab [4, 5], and Janus kinase inhibitors, notably bariticinib and tofacitinib, according to recent data in the literature [6].

However, rheumatoid nodules have sometimes been found to enlarge paradoxically with the treatment of rheumatoid arthritis, particularly with methotrexate and the TNF-alpha inhibitors [7], and leflunomide (LEF), The latter is an immunomodulatory antirheumatic drug generally used as a second-line treatment for RA

after failure or contraindication to methotrexate, treatment which our patient had received a few months prior to her first episode of pneumothorax. LEF suppresses the synovial inflammatory reaction and prevents bone destruction in RA. However, numerous respiratory adverse events have been reported, including pneumonia, abscesses, interstitial lung disease and pulmonary nodules [6-9]. Although it is not clear whether these treatments should be discontinued for this reason.

In patients who smoke or have smoked in the past, it is important to differentiate nodules from malignant tumours. Previous imaging studies and Fleischner Society recommendations can be used to guide the evaluation of solitary pulmonary nodules [10]. Positron emission tomography can be used to evaluate nodules ⩾8 mm in diameter; in general, rheumatoid nodules show little or no uptake on positron emission tomography. In general, rheumatoid nodules show little or no uptake on positron emission tomography, although increased uptake may be seen in cases of active inflammation [11].

Table 1: Review of cases of rheumatoid arthritis complicated by spontaneous pneumothorax (RF: rheumatoid factor; ND: not determined), one patient per series

References	Age	Sexe	Duration of disease progression (years)	RF	Underlying lung diseases	Subcutaneous nodules	Lung nodules	Prognosis
Savana *et al*., [12]	40	F	0	Positive	No	No	Multiple	Favorable
Kobayashi *et al*., [13]	67	F	6	Positive	No	No	Multiples, bilateral	Unfavorable
Winne *et al*., [14]	50	F	ND	Négative	No	No	Multiple, bilatéral	Unfavorable
N'Gabou *et al*., [15]	64	F	10	Positive	Yes	No	Multiple	Favorable
Huret *et al*., [16]	74	M	4	Positive	Yes	No	Multiple, bilateral	Favorable
Nishida *et al*., [17]	74	F	14	Positive	No	No	Multiple	Favorable
Kim *et al*., [8]	46	F	4	Positive	No	No	Multiple, bilateral	Favorable
Gaye *et al*., [18]	26	M	ND	ND	No	No	Multiple, bilatéral	Favorable
Our case	28	F	11	Positive	No	No	Multiple, bilatéral	Favorable

The cases of spontaneous pneumothorax on RA described in the literature (Table 1) mainly concern subjects over 40 years of age, unlike our patient who has less. Females predominated. All patients had no subcutaneous nodules. In only one case did pneumothorax precede the articular manifestations of the disease. Our patient was a young woman with seropositive RA who had no subcutaneous nodules or underlying respiratory diseases. She presented with pulmonary nodules complicated by pneumothorax, which appeared 10 months after treatment with LEF.

CONCLUSION

Spontaneous pneumothorax is a rare manifestation of pulmonary lesions in RA. It may be a progressive manifestation or a circumstance of discovery. Its occurrence is considered to be the natural evolution of pulmonary nodules, but others incriminate the immunosuppressive drugs used to treat RA. Rheumatoid arthritis is a condition that should be considered, especially when spontaneous pneumothorax and joint manifestations are present.

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