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PULMONARY IMPAIRMENT IN RHEUMATOID ARTHRITIS

Marcus Vinicius Petruco^{1,*}, Adriane Mesquita Petruco¹, Ana Júlia de Lima Nogueira¹, Bruna Flávia Tristoni¹, Marina Laís Barreta¹

1.Centro Universitário Ingá, Maringá (PR), Brazil.

*Corresponding author: marcus.adriane@gmail.com

BACKGROUND

Rheumatoid arthritis (RA) is a systemic inflammatory autoimmune disease where joints have traditionally been the primary target. Extra-articular manifestations are frequent and may affect the lungs from mild to extensive and severe injuries. The diagnosis of rheumatoid lung disease can be difficult, with many confusing factors, which make the subject a challenge. The objective of this work is to report a case of RA with pulmonary involvement evolved with respiratory failure, demonstrating the different diagnostic possibilities as well as the therapeutic approaches.

CASE REPORT

A 57-year-old female patient reported dry cough for 4 months, with recurrent purulent sputum in the last month, tiredness, dyspnea and fever of 39 °C. During this period, she used azithromycin, levofloxacin and amoxicillin + clavulanate without improvement. History of RA (anti-CCP > 200 IU/mL), using methotrexate for more than one year without joint signs. She denied smoking, comorbidities and relevant family cases. On physical examination, she was afebrile, acyanotic, with peripheral oxygen saturation (Spo2) of 88%, respiratory rate of 20 irpm and sparking subcrepitating rales on pulmonary auscultation. She was admitted with broad-spectrum antibiotic therapy and oxygen therapy. Chest CT revealed multiple confluent opacities associated with the diffuse bilateral ground-glass pattern. The pulmonary condition evolved with persistence, opting to perform open lung biopsy, which showed anatomopathological findings compatible with BOOP, subacute lung injury and PINE, suggesting involvement by the underlying disease (RA). The patient underwent pulse therapy with methylprednisolone 1 g for 3 days, with clinical and laboratory improvement, and was discharged with oral corticosteroids and supplemented with oxygen, mycophenolate mofetil capsules was introduced at a dose of 2 g a day. In the first outpatient return in 30 days, she did not present respiratory complaints, obtaining complete weaning from oxygen and gradual reduction of the corticoid. A new chest CT scan in 60 days showed almost complete regression of the pulmonary changes, maintaining only the use of mycophenolate mofetil capsules.

CONCLUSION

The reported case demonstrates the diagnostic challenge of pulmonary involvement in RA, where an infectious condition, use of methotrexate and the underlying disease itself competed as the cause of pulmonary changes. Chest tomography and lung biopsy were decisive for making therapeutic decisions, with appropriate treatment and favorable evolution despite their severity.