SARCOIDOSIS VASCULITIS AND DIFFUSE LUNG DISEASES 2023; 40 (2); e2023014 DOI: 10.36141/svdld.v40i2.13015

A sarcoidosis patient developing psoriatic arthritis 18 years later: First description

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To the Editor,

Sarcoidosis is a chronic non-caseous granulomatosis disease that clinically usually has mediastinal lymphadenopathy and pulmonary infiltrates. It occurs mainly in adults younger than 40 years of age, with a peak incidence in the third decade of life. The prevalence in men is 5.9 cases and in women is 6.3 cases per 100,000 person-years (1). It may evolve with good outcomes after steroid therapy and remain in remission for several years or decades (2).

The concomitance of sarcoidosis and other autoimmune disease is relatively described in the literature. For instance, sarcoidosis was described to be associated with autoimmune liver diseases, with Hashimoto's disease, Sjögren's syndrome and also ankylosing spondylitis (3,4). Because the exact immunopathogenesis of sarcoidosis is unknown, it is unknown whether these exposures are indeed causing sarcoidosis, making the immune system more susceptible to the development of sarcoidosis, exacerbating subclinical cases of sarcoidosis, or causing a granulomatous condition distinct from sarcoidosis (5). Sarcoidosis may also appear during anti-rheumatic therapy. Some reports have described sarcoidosis following TNF blockers and ustekinumab in patients with psoriatic arthritis (PsA) (6-9). Although, to the best of our knowledge, no patient with sarcoidosis evolving to PsA was described in the literature. Therefore, this article aims to report a patient with sarcoidosis who developed after 18 years of PsA.

A 78-year-old woman with a past medical history of systemic hypertension, under losartan 50mg/day, had a diagnosis of sarcoidosis at 60 years old, characterized by dyspnea, cough, and typical imaging thorax computed tomography with mediastinal lymphadenopathy and mild interstitial lung disease. A transbronchial biopsy confirmed non-caseous granulomatosis diseases, without evidence of tuberculosis of fungi. She was treated with prednisone 60mg twice a day and quickly tapered and had an excellent response. After 8 months, she became asymptomatic, and all lung lesions reduced and then disappeared. When she was 70 years old, fibromyalgia was diagnosed. Furthermore, in 2017, she started with psoriasis vulgaris lesions on her elbows, and onicholysis, and dermatologists confirmed the diagnosis. Her mother had also psoriasis. She was treated with moisture and topical steroids. After three years, she initiated polyarthritis of knees, ankles, and wrists bilaterally. Laboratory tests demonstrated normal cell blood count and blood biochemistry. Erythrocyte sedimentation rate (ESR) of 65mm/1st hour [normal range (nr): < 10mm/1st hour], C-reactive protein (CRP) of 5mg/dL (nr: < 1mg/dL), vitamin D of 28.5 ng/mL (nr: > 30 ng/mL), normal levels of acid alpha1-glycoprotein and normal serum electrophoresis. Rheumatoid factor, antinuclear antibodies, anti-dsDNA, anti-Ro/SS-A, anti-La/SS-B,

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anti-U1RNP, anti-Sm, anti-CCP, anti-gliadin, anti-endomysium, anti-tissue transglutaminase, anti-thyroglobulin, anti-thyroperoxidase, IgG and IgM anticardiolipin and lupus anticoagulant were all negative. Complement levels were normal. Serology for syphilis, HIV 1 and 2, HTLV I and II, hepatitis B and C; IgM for cytomegalovirus, Epstein-Barr, toxoplasmosis, rubella, and herpes 1 and 2 were negative. The X-rays of the affected joints and also the thorax were normal. Spirometry was normal. She was diagnosed as PsA based on the ClASsification criteria for Psoriatic Arthritis (CASPAR) criteria: evidence of joint involvement plus current psoriasis, nail dystrophy, and negative rheumatoid factor (10). She was treated with methotrexate 15mg/week plus folic acid 5mg/week and vitamin D3 10,000Iu/day. After 3 months, she returned and was asymptomatic, ESR reduced to 18 mm/1st hour, CRP was reduced to 0.5mg/dL, and 25-vitamin D normalized in 50 ng/dL. She is currently well, asymptomatic, although the onicholysis remains, no skin lesion or arthritis are noted and sarcoidosis continues in remission, with imaging and laboratory tests within the normal range.

Although the pathophysiological mechanisms of sarcoidosis is yet unknown, the central hypothesis is that it results from genetically susceptible persons to specific environmental agents. It is unknown the prevalence of sarcoidosis associated with another autoimmune disease. The case-control studies give this association's evidence, retrospective cohort studies, or case reports, as the present study. A recent case-control Taiwan study investigated the association between autoimmune diseases in 1,237 sarcoidosis patients compared to 4,948 healthy controls. Interestingly, in 218 (17.6%) patients, this association was observed. Mainly with autoimmune thyroid disease, 11.6%, Sjogren's syndrome (1.54%), and ankylosing spondylitis (3.64%) (11). Many other autoimmune diseases have been reported to co-occur with sarcoidosis on a case report basis, for example, systemic sclerosis and inflammatory myositis (12).

Fibromyalgia was not a bias for the diagnosis of PsA since the patient fulfilled the CASPAR criteria for PsA and had all criteria including arthritis, psoriasis lesions, nail distrophy and negative autoantibodies (10).

In conclusion, this article describes a patient with pulmonary sarcoidosis who developed psoriatic arthritis after 18 years and was successfully treated with methotrexate.

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