

Coexisting sarcoidosis and systemic lupus erythematosus: a case report and literature review

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ABSTRACT

The coexistence of sarcoidosis and systemic lupus erythematosus (SLE) in the same patient has uncommonly been reported. Information on the epidemiology, clinical presentation, and management of this rare association is scarce. We report a 46-year-old Hispanic man who was recently diagnosed with concomitant SLE and sarcoidosis at our institution. A diagnosis of sarcoidosis was established due to the presence of dyspnea, chest pain, fever, and malaise along with bilateral hilar lymphadenopathy and histological evidence of non-caseating granuloma. In addition, he fulfilled the American Rheumatism Association (ACR) criteria for SLE due to a history of photosensitivity, polyarthritides, lymphocytopenia, and positivity of antinuclear antibodies (ANA) and anti-double-stranded DNA (anti-dsDNA) antibodies. He was successfully treated with a combination of oral glucocorticoids, hydroxychloroquine, and methotrexate. In a further step, we conducted an extensive literature review to further investigate into the association of sarcoidosis and SLE. We identified 25 additional published cases. The concurrence of these two conditions may be more common than previously reported, mainly affecting young female adults in the fourth decade of life. The most common manifestation of sarcoidosis was mild pulmonary symptoms whereas SLE presentation was highly variable. Most pa-

tients were positive for anti-dsDNA antibodies. Different therapeutic strategies included oral glucocorticoids, hydroxychloroquine, conventional immunosuppressive drugs and, cyclophosphamide in severe cases. Our study reinforces the need of considering the potential concurrence of sarcoidosis and SLE. Clinicians should be aware of the potential presence of SLE in patients with a diagnosis of sarcoidosis presenting with cutaneous manifestations, cytopenia, renal involvement, and/or positivity for ANA and anti-dsDNA antibodies.

Keywords: Coexistence; Systemic lupus erythematosus; Sarcoidosis.

INTRODUCTION

Sarcoidosis is a multisystemic inflammatory condition of unknown origin, characterized by the presence of non-caseating granulomas. It mainly affects young adults presenting with bilateral hilar lymphadenopathy and lung involvement¹⁻³. However, the clinical presentation of sarcoidosis is highly variable among patients and every organ can be affected. Sarcoidosis is a more complex disease than previously thought and it can even coexist in the same patient with other autoimmune diseases including SLE⁴.

The incidence and prevalence of sarcoidosis vary across different ethnicities and geographical areas, being more frequent in Scandinavian countries and among African Americans⁵. In Spain, the cumulative annual incidence rate of sarcoidosis is 1.36 per 100,000 inhabitants⁶. Interestingly, Brito-Zerón *et al.*⁴ found in a Spanish cohort of 218 patients that sarcoidosis was associated with other systemic inflammatory diseases in 6.4% of patients. The annual incidence rate of SLE in Northern Spain is 3.6 per 100,000 population⁷. However, the coexistence in the same patient of sarcoidosis and SLE has been infrequently reported⁸.

We describe a case of a patient with the simultaneous

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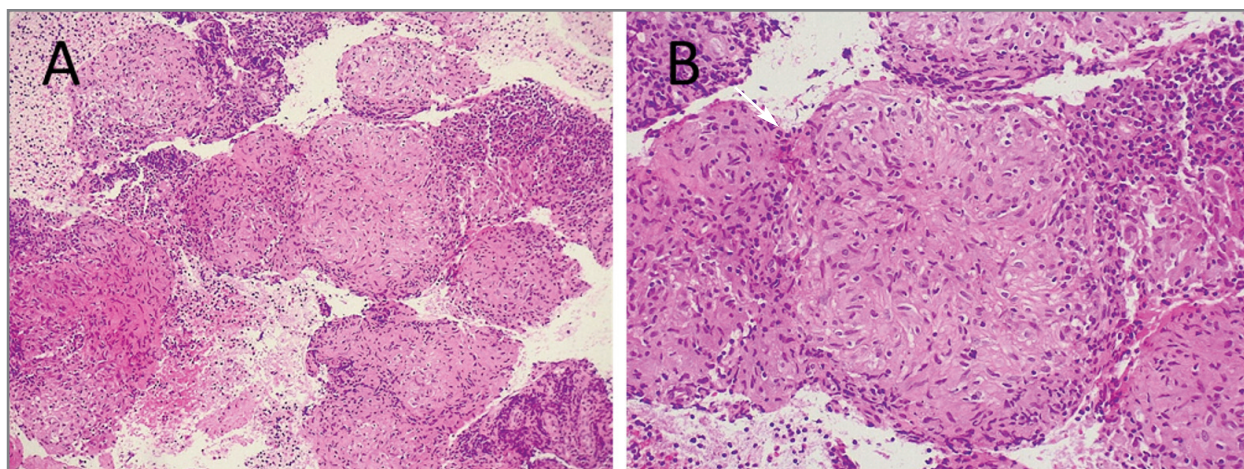


FIGURE 1. Lymph node in subcarinal location, obtained by endobronchial ultrasound (EBUS). Histological sections showed scarce lymphoid tissue and abundant well-shaped granulomas (A). A higher magnification showed granulomas of sarcoid type. In this regard, granulomas were found to be composed of epithelioid cells and lymphocytes but necrosis was not present (B).

occurrence of sarcoidosis and SLE which highlights the relevance of considering the co-existence of these multisystemic autoimmune diseases.

CASE REPORT

A 46-year-old Hispanic man was admitted to a tertiary care hospital in March 2020 due to a 3-day history of fever, dyspnea, chest pain, malaise, and diffuse arthralgia with early morning stiffness. His past medical, family, and social history was unremarkable, apart from an episode of unilateral peripheral facial palsy successfully treated with glucocorticoids ten years before. Prior to the admission, the patient also reported repetitive episodes of polyarthritis in his wrists and hands, as well as, rash on his face and sun-exposed areas. He also complained of dryness of eyes and mouth for the last two years.

Physical examination showed body temperature of 38.3 °C on admission. Lung and heart auscultation were normal. Neurological, abdominal, and skin examinations were unremarkable. No peripheral lymphadenopathy or hepatosplenomegaly was found.

Laboratory tests yielded the following results: marked lymphocytopenia (400/ μ L; normal 1,000-4,800/ μ L) and elevated C-reactive protein (maximum 9.6 mg/dL; normal < 1mg/dL) with normal procalcitonin (0.1 ng/ml). The serum protein electrophoresis showed hypergammaglobulinemia and elevated IgG (2417.13 mg/dl; normal 700-1,600 mg/dl). Urinalysis

was normal. Serum was positive for ANA (titer 1:1280 with a speckled pattern). Anti-dsDNA antibodies were detected by enzyme-linked immunosorbent assays (ELISA) (167 UI/ml; normal < 40) and *Crithidia luciliae* immunofluorescence test (titer 1:20). Anti-SSa and anti-SSb antibodies were also positive. In contrast, rheumatoid factor (RF), anti-cyclic citrullinated peptides, ANCA, anti-RNP, anti-Sm, and anti-phospholipid antibodies were negative. Complement levels were normal. Serum angiotensin-converting enzyme (ACE), serum calcium, phosphate, and 1,25-dihydroxyvitamin D levels were normal.

As the patient complained of chest pain, ischemic heart disease, musculoskeletal conditions, infections, and other causes of chest pain were excluded first. The chest radiograph followed by a chest computed tomography showed bilateral hilar lymphadenopathy raising the suspicion of sarcoidosis. Transbronchial lymph node biopsy specimens revealed non-caseating granuloma (Figure 1). The tuberculin skin reaction was negative, as well as the interferon-gamma release assays (IGRA). An extensive microbial screening was performed in order to rule out infection. Serological tests, nasopharyngeal swabs, cultures of blood, and lymph node were all negative, including SARS-CoV-2 infection. Peripheral blood smear and flow cytometric immunophenotyping excluded a hematologic condition.

A clinical diagnosis of sarcoidosis was made based on the clinical, radiological, and histopathological features mentioned above, after excluding other infec-

tious or neoplastic conditions. In addition, the patient fulfilled the ACR criteria for SLE due to a history of photosensitivity, polyarthritis, lymphocytopenia, and positivity of ANA and anti-dsDNA antibody. Consequently, the patient was diagnosed with concomitant SLE and sarcoidosis.

The patient was started on prednisone 30 mg day, which was gradually tapered. Within the first week of treatment, fever, chest pain, and arthralgias completely disappeared. Hydroxychloroquine (400 mg/day) and methotrexate (15 mg/weekly) were added for the management of SLE and as glucocorticoid sparing agents.

DISCUSSION

The occurrence of sarcoidosis and SLE together in the same patient has been previously reported. However, the clinical picture resulting from the coexistence of these two inflammatory conditions is not well defined. Data regarding plausible common immunopathological pathways, incidence, clinical presentation, and management of this rare association are scarce.

We conducted an extensive literature search of the Medline databases for articles published up to June 2020, in order to thoroughly describe the association of sarcoidosis and SLE. We identified 25 published cases. Table I summarizes the demographic and clinical features of these 26 patients, including our case. The mean age of the patients was 46.4 ± 16.0 years (ranging from 10 to 82 years). Most patients were female (80.8%).

Sarcoidosis and SLE share some common immunological features. Both conditions show evidence of cellular and humoral immune abnormalities including hyperreactivity of the immune system, hypergammaglobulinemia, defective cell-mediated immune function, and loss of tolerance to self-antigens⁹. The exaggerated immune response in sarcoidosis may lead to the production of various antibodies such as ANA or RF. Positivity for ANA can be found in up to 30% of patients with sarcoidosis without clinical manifestations of SLE¹⁰. However, anti-dsDNA antibodies, which are more specific for SLE, are rarely found in patients with sarcoidosis¹¹.

The incidence of sarcoidosis and SLE occurring together may be underestimated. In this regard, Begum *et al.*¹² found three cases (1%) of coexisting sarcoidosis and SLE in a series of 300 patients with SLE. This sug-

gests that the incidence of sarcoidosis in patients with SLE could be higher than reported, supporting the claim that some common immune processes can be shared by these conditions.

Sarcoidosis and SLE can present with similar clinical manifestations such as polyarthritis, fever, or peripheral lymphadenopathy. In the absence of high suspicion, some symptoms can be attributed to a single condition. However, there are distinctive features that should raise the suspicion of the coexistence of sarcoidosis and SLE. In patients with SLE, pulmonary involvement presenting with interstitial lung disease (ILD), as acute lupus pneumonitis or chronic ILD, is rare¹³. Thus, the coexistence of sarcoidosis should be considered in patients with SLE presenting with ILD and bilateral hilar lymphadenopathy¹⁴. In addition, the presence of granuloma in histopathologic specimens is very uncommon in patients with SLE. Although the possibility of granulomatous lymphadenopathy should be considered in the context of SLE, a secondary diagnosis, including sarcoidosis, should be ruled out¹⁵.

Conversely, renal involvement is not frequently found in patients with sarcoidosis, ranging from 1% to 30-50% depending on different series. Calcium-induced nephropathy and granulomatous interstitial nephritis are the most common renal manifestations of sarcoidosis¹⁶. However, glomerulonephritis has been rarely described. The concurrence of SLE in patients with sarcoidosis should be considered when glomerulonephritis is present, particularly in the presence of other features such as cutaneous malar rash or positivity for ANA and anti-dsDNA.

In our literature review of patients with concomitant diagnosis of sarcoidosis and SLE, pulmonary symptoms were the main manifestation of sarcoidosis, being present in 23 of 26 patients (88.5%). Most of them, 17 of 23 patients, were classified as stage I of sarcoidosis according to the radiological lung involvement. Among them, sarcoid extra-pulmonary involvement was also present in 16 (69.2%) patients including skin (n=10), eyes (n=6), liver (n=3), hypercalcemia (n=3), bone marrow (n=2), muscle (n=1), renal (n=1) and hypothalamus (n=1). Interestingly, 3 (11.5%) patients had only extra-pulmonary manifestations of sarcoidosis, involving central nervous system (n=1), skin (n=1), and liver (n=1). Elevated levels of ACE were found in 11 patients (42.3%).

The clinical presentation of SLE in patients with concomitant diagnosis of sarcoidosis was highly variable, ranging from mild cutaneous manifestations to severe

TABLE I. PUBLISHED CASES OF SARCOIDOSIS AND SYSTEMIC LUPUS ERYTHEMATOSUS ASSOCIATION

Case	Reference	Age/Sex/ Race	Sequence of presentation	SLE features that established a diagnosis	Sarcoidosis features that established a diagnosis	Histopathologic evidence of non-caseating granulomas	Treatment
1	Bratic-Mikes E et al, (1976) [21]	F/ND	ND	Rash.	Central nervous system.	ND	ND
2	Harrison GN et al, (1979) [22]	54/F/ND	SLE sarcoidosis	Polyarthritits, alopecia. Coombs test + ANA+/RF+	Dry cough, rash Radiographic bilateral hilar lymphadenopathy. Hypercalcemia.	Liver and skin	GC
3	Wiesenhutter GW et al, (1979) [23]	20/F/Black	SLE sarcoidosis	Polyarthritits, fever, rash, Raynaud's phenomenon, nephritits. ANA+ Hypocomplementemia Coombs test +	Fever, dyspnea, hepatosplenomegaly. Anemia. Elevated ACE.	Liver and bone marrow.	GC Cyclophos- phamide.
4	Hunter T et al, (1980) [24]	62/F/ Caucasian	Simultaneously	Polyarthritits, sicca syndrome, alopecia, cough, dyspnea. ANA + / anti-dsDNA +/ lupus anticoagulant + Hypocomplementemia Coombs test +		Lung and lymph node	ND
5	Needleman SW et al, (1982) [25]	47/F/ Caucasian	SLE sarcoidosis	Polyarthritits, fever, rash, pleuropericarditits, ANA +/- anti-dsDNA + Coombs test + Hypocomplementemia and lymphopenia	Radiographic bilateral hilar lymphadenopathy Radiographic bilateral hilar lymphadenopathy	Lung and bone marrow	GC
6	Aronson PJ et al, (1985) [26]	82/M/ Caucasian	Simultaneously	Arthralgias, skin rash, oral ulcers, dyspnea, dysphagia, muscle weakness ANA +		Skin and muscle (Skin biopsy showed features of sarcoidal granulomas and lupus occurring in the same lesion.)	GC

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TABLE I. CONTINUATION

Case	Reference	Age/Sex/ Race	Sequence of presentation	SLE features that established a diagnosis	Sarcoidosis features that established a diagnosis	Histopathologic evidence of non-caseating granulomas	Treatment
7	Askari A et al, (1988) [27]	52/F/ Caucasian	SLE sarcoidosis	Polyarthritis, malar rash, photosensitivity, Raynaud's phenomenon. ANA +/- anti-dsDNA +	Dyspnea, uveitis Bilateral hilar lymphadenopathy	Lung	-NSAIDs
8	Soto-Aguilar et al. (1988) [20]	43/F/Black	SLE sarcoidosis	Glomerulonephritis ANA +/-anti-RNP +	Subcutaneous nodules Bilateral hilar lymphadenopathy Elevated ACE	Skin and lymph node	GC
9	Fivenson DP et al, (1989) [17]	41/F/Black	Sarcoidosis SLE	Polyarthritis, oral ulcers, alopecia, rash, oral ulcers, pleuritis, nephritis ANA +/- anti-dsDNA +, anti-SSa+/anti-Sc170+/ FR + Hypocomplementemia	Dyspnea Bilateral hilar lymphadenopathy	Lung	GC HQC AZA Oral gold Cyclophosphamide
10	Enzenauer et al, (1992) [9]	57/F/ Caucasian	SLE + SS sarcoidosis	Polyarthritis, oral ulcers, sicca syndrome, nephritis, hemolytic anemia. ANA+/anti-dsDNA+	Radiographic bilateral hilar lymphadenopathy	Lymph node	NSAIDs
11	Magasic et al, (1993) [28]	43/F/ND	Sarcoidosis SLE	Fever, polyarthralgia, peripheral lymphadenopathy ANA +/-anti-dsDNA+ Hypocomplementemia	Dry cough, iridocyclitis Elevated ACE Membranous and focal glomerulonephritis along with interstitial sarcoid granulomas. Radiographic bilateral hilar lymphadenopathy and interstitial lung infiltrates	Lung and kidney	GC
12	Collins et al (1996) [29]	54/F/India	SLE + APS sarcoidosis pleuropericarditis.	Fever, polyarthritis, oral ulcers, rash, ANA+/anti-cardiolipin+.	Liver dysfunction Hypercalcemia Elevated ACE	Liver	GC AZA

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TABLE I. CONTINUATION

Case	Reference	Age/Sex/ Race	Sequence of presentation	SLE features that established a diagnosis	Sarcoidosis features that established a diagnosis	Histopathologic evidence of non-caseating granulomas	Treatment
13	Schnabel A et al. (1996) [14]	23/F/ND	Simultaneously	Polyarthritis, dyspnea, Raynaud's syndrome. ANA +/- anti-U1RNP + Hypocomplementemia Radiographic micronodular	interstitial changes. Subcutaneous nodules, uveitis Radiographic bilateral hilar lymphadenopathy	Lung	GC HCQ
14	Umeki et al (2000) [19]	60/F/ Japanese	SLE sarcoidosis	Malar rash, photosensitivity ANA +/- anti-dsDNA +/- anti-SSa +	Dyspnea, malaise, skin nodules, sicca syndrome Radiographic bilateral hilar lymphadenopathy Elevated ACE	Skin	-GC
15	Kissling et al, (2002) [30]	75/M/ND	SLE + APS sarcoidosis	Polyarthritis, discoid lupus, sicca syndrome, photosensitivity, alopecia Cerebrovascular accident ANA +/- anti-SSa + / anticardiolipin +	Erythema nodosum	Skin, conjunctiva and sclera	GC HQC
16	Kissling et al, (2002) [30]	53/M/ND	Sarcoidosis SLE + APS	Rash, sicca syndrome, oral ulcers ANA +/- anti-dsDNA +/- anti-SSa/SSb +/- anticardiolipin +	Subcutaneous nodules Radiographic bilateral hilar lymphadenopathy Elevated ECA and hypercalcemia	Skin	GC HQC
17	Begum S et al, (2002) [12]	28/F/ Caucasian and Afro- -Caribbean	SLE sarcoidosis	Rash, polyarthritis Pulmonary embolism ANA +/- anti-dsDNA +/- anti-SSa +	Panuveitis Panhypopituitarism Radiographic ILD Elevated ACE	No evidence	GC Cyclosporine
18	Begum S et al, (2002) [12]	10/F/ Caucasian	Sarcoidosis SLE	Fever, malaise, alopecia, Raynaud's phenomenon, rash, oral ulcers. Lupus nephritis ANA +/- anti-dsDNA +/- anti-Sm +/- anti-RNP + Hypocomplementemia and lymphopenia			

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TABLE I. CONTINUATION

Case	Reference	Age/Sex/ Race	Sequence of presentation	SLE features that established a diagnosis	Sarcoidosis features that established a diagnosis	Histopathologic evidence of non-caseating granulomas	Treatment
19	Begum S et al, (2002) [12]	28/F/ Caucasian	Sarcoidosis SLE	Polyarthralgia, malar rash, Raynaud's phenomenon, oral ulcers ANA+/anti-dsDNA+/ anti-SSa/SSb+ Hypocomplementemia	Erythema nodosum Radiographic bilateral hilar lymphadenopathy		GC HCQ
20	Papaioannides et al, 2004 [18]	49/F/ND	Sarcoidosis SLE	Polyarthralgia, fever, oral ulcers, alopecia, malar rash, photosensitivity. ANA+/anti-Sm+/anti-SSa+ Hypocomplementemia	Dry cough, anorexia, dyspnea Radiographic bilateral hilar lymphadenopathy	Lung	GC HCQ
21	Migita K et al, 2005 [31]	42/F/ Japanese	Simultaneously	Dry cough, photosensitivity, rash ANA+/anti-dsDNA +/anti-SSa+ Hypocomplementemia Elevated ACE Radiographic mediastinal lymphadenopathy and ground glass opacities		Skin and lung	GC
22	Nakayama et al, (2007) [8]	52/F/ Japanese	Simultaneously	Polyarthralgia, uveitis, severe photosensitivity ANA +/ anti-dsDNA +/ anti-Sm + Autoimmune hepatitis Radiographic bilateral hilar lymphadenopathy. Elevated ACE		No evidence	GC
23	Wesemann et al, (2009) [32]	48/F/ND	SLE + APS sarcoidosis	Malar rash, polyarthrits, class IV glomerulonephritis Coronary thrombosis ANA +/ anti-dsDNA +/ lupus anticoagulant +/ anticardiolipin + Dry cough, distinct rash Radiographic bilateral hilar lymphadenopathy		Skin	GC HCQ AZA Cyclophosphamide
24	Wesemann et al, (2009) [32]	46/M/ND	SLE + APS sarcoidosis	Pulmonary embolism ANA + /anti-Sm +/ anticardiolipin +	Dyspnea, dry cough, polyarthralgia Radiographic bilateral hilar lymphadenopathy	Lymph node	GC HCQ

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TABLE I. CONTINUATION

Case	Reference	Age/Sex/ Race	Sequence of presentation	SLE features that established a diagnosis	Sarcoidosis features that established a diagnosis	Histopathologic evidence of non-caseating granulomas	Treatment
25	Khammasi et al. (2015) [33]	45/F/ND	Simultaneously	Dyspnea, polyarthralgia, rash, pericarditis, peripheral lymphadenopathy ANA +/- anti-dsDNA +/- hypocomplementemia Hypercalcemia, elevated ACE Radiographic bilateral hilar lymphadenopathy		Lymph node, liver and skin	GC AZA
26	Present case	46/M/ Hispanic	Simultaneously	Fever, chest pain, polyarthralgia, photosensitivity ANA+/anti-dsDNA+/anti-SSa+ and anti-SSb+ Lymphopenia Radiographic bilateral hilar lymphadenopathy		Lymph node	GC HQC MTX

ACE: Angiotensin Converting Enzyme. ANA: Antinuclear antibodies. AZA: Azathioprine. dsDNA: double-stranded DNA. GC: Glucocorticoids. HQC: Hydroxychloroquine. MTX: Methotrexate. SLE: Systemic lupus erythematosus.

renal involvement. The presence of cytopenia was the most common laboratory abnormality. Noteworthy, most patients (61.5%) were positive for anti-dsDNA antibodies.

According to the data retrieved from the literature review, the disease presentation occurrence of sarcoidosis and SLE was quite variable. In 7 patients both conditions were simultaneously diagnosed. In 12 patients SLE features preceded the diagnosis of sarcoidosis, whereas sarcoidosis was diagnosed before SLE in 6 patients. This information was not available in 1 patient. Some authors suggest that the withdrawal of glucocorticoid therapy may precipitate the development of the second inflammatory condition. In this regard, Fivenson *et al.*¹⁷ and Papaioannides *et al.*¹⁸ reported two patients with sarcoidosis who developed SLE features after stopping glucocorticoids. Likewise, Umeki *et al.*¹⁹ and Soto-Aguilar *et al.*²⁰, described another two patients with SLE who developed sarcoidosis after oral glucocorticoid therapy withdrawal.

Most patients with SLE and sarcoidosis previously reported were successfully treated with a combination of glucocorticoids and hydroxychloroquine. Conventional immunosuppressive drugs were added in some patients, including azathioprine (n=3), methotrexate (n=2) and, cyclosporine (n=1). Three patients received cyclophosphamide due to severe manifestations.

In conclusion, our case reinforces the relevance of considering the potential concurrence of sarcoidosis and SLE. According to our review, this association may be more common among female young adults in the fourth decade of life. Pulmonary manifestations were the most common presentation of sarcoidosis, usually with a favorable prognosis. SLE presentation was highly variable, being anti-dsDNA positivity a distinctive feature in most cases. Therefore, the possibility of the coexistence of SLE in patients with a diagnosis of sarcoidosis should be carefully considered in patients presenting with cutaneous manifestations, cytopenia, renal involvement, and positivity for ANA and anti-dsDNA antibodies.

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