



Sarcoidosis mimicking lymphoma; a case report

Shakiba Hassanzadeh¹ , Mohammadreza Khosravifarsani^{2,3*} 

Abstract

Sarcoidosis is a granulomatous disorder that mostly involves the lungs. Bilateral hilar lymphadenopathy (LAP) and lung infiltration are commonly observed in these patients. However, other organs such as the skin, liver, and eyes may also be involved. The exact cause of sarcoidosis is still unknown. Sarcoidosis is diagnosed with non-caseating, non-necrotic epithelioid granulomas on histology and its typical clinical and radiological presentations. However, other differential diagnoses of sarcoidosis such as lymphoproliferative disorders and infectious diseases should be excluded. We herein report a case of a 47-year-old male who was initially diagnosed with lymphoma but upon further diagnostic workup, sarcoidosis was established as the definite diagnosis.

Keywords: Sarcoidosis, Lymphoma, Leukemia, Lymphadenopathy, Lymphoproliferative diseases, Hypercalciuria, Hypercalcemia

Please cite this paper as: Hassanzadeh S, Khosravifarsani M. Sarcoidosis mimicking lymphoma; a case report. *J Parathyroid Dis.* 2021;9:e11175.

Copyright © 2021 The Author(s); Published by Nickan Research Institute. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Introduction

Sarcoidosis is a granulomatous disorder that may involve individuals of any age, gender, and ethnicity. The prevalence and incidence of sarcoidosis have been reported to be approximately 4.7-64 and 1-35.5 per 100 000 individuals each year, respectively. However, the exact cause of sarcoidosis is still unknown (1,2).

Lungs are the most involved organ in patients with sarcoidosis. Bilateral hilar lymphadenopathy (LAP) and lung infiltration are commonly observed. However, other organs such as the skin, liver, and eyes may also be involved. Dyspnea, cough, chest pain, wheezing, and fatigue are the most common symptoms of sarcoidosis. Other constitutional symptoms such as muscle pain, arthralgia, fever, weight loss, and headaches may also be observed (2-7).

Sarcoidosis is diagnosed with non-caseating, non-necrotic epithelioid granulomas on histology in addition to the typical clinical and radiological presentations. However, other differential diagnoses of sarcoidosis such as lymphoproliferative disorders and infectious diseases should be excluded (2,3).

We herein report a case of a 47-year-old male who presented with fatigue, weight loss, abdominal pain, increased urinary frequency, hypercalcemia, and decreased glomerular filtration rate (GFR). He was initially diagnosed with lymphoma but upon further diagnostic workup, sarcoidosis was established as the definite diagnosis.

Case Presentation

A previously healthy 47-year-old male was referred to our clinic for further evaluation of a possible diagnosis of lymphoma. He had a few months history of fatigue, weight loss, abdominal pain, and increased urinary frequency. On initial evaluation, hypercalcemia, hypertension, and decreased GFR were detected. His past medical history was negative except for a COVID-19 infection about a year ago. Lung lesions had been detected on his lung imaging during his COVID-19 infection and he was recommended to follow up after recovering. However, he did not follow up as advised. His family history was positive for hypertension in his parents.

Laboratory tests showed elevated levels of erythrocyte sedimentation rate (1st hour) (50 mm), C-reactive protein (7.5 mg/L), blood urea nitrogen (18.1 mg/dL), creatinine (2.14 mg/dL), and calcium (12.6 mg/dL). There were decreased levels of vitamin D3 (21.83 ng/mL) and parathyroid hormone (5.03 pg/mL). Serum levels of sodium and albumin were 141 mEq/L and 4.57 g/dL, respectively. Urinalysis showed trace protein (10 mg/dl), 4+ blood (70-150/ μ L), 5-9 white blood cells/hpf (high-power field), many red blood cell (RBC)/hpf, 2%, dysmorphic RBCs, calcium oxalate crystals, few bacteria/hpf, and 1+ leukocyte esterase (Table 1). In addition, the patient had an increased level of beta-2 microglobulin (6.65 mg/L). However, hepatitis B surface antigen, hepatitis B core antibody (immunoglobulin (Ig) M), hepatitis C virus antibody, and human immunodeficiency virus (HIV) (1 and 2)-antibody and antigen were negative (Table 1).

Received: 3 October 2021, Accepted: 8 December 2021, ePublished: 18 December 2021

¹Division of Pathology, Nickan Research Institute, Isfahan, Iran. ²Cancer Prevention Research Center, Isfahan University of Medical Sciences, Isfahan, Iran. ³Department of Internal Medicine, Shahrekord University of Medical Sciences, Shahrekord, Iran.

***Corresponding author:** Mohammadreza Khosravifarsani, Email: drmohammadkhf@gmail.com, Khosravifarsani@med.mui.ac.ir

■ Implication for health policy/practice/research/medical education

Sarcoidosis is a systemic granulomatous disorder that involves multiple organs. It is diagnosed by its clinical presentations, radiological features, and non-caseating granulomas on histology. Other possible diagnoses such as lymphoproliferative diseases, infections, and malignancies should be excluded.

Furthermore, serum protein electrophoresis showed an albumin/globulin ratio of 0.92 (low) with decreased albumin (48.0%) and increased beta 2-globulin (7.8%) and gamma-globulin (25.9%). The absolute concentration for total protein was 8.1 g/dL, albumin was 3.9 g/dL (low), beta 1-globulin was 0.6 g/dL (high), beta 2-globulin was

0.6 g/dL (high), and gamma-globulin was 2.1 g/dL (high) (Table 2 and Figure 1). Serum protein immunotyping revealed the presence of weak IgG kappa and lambda; non-monoclonal (Figure 2).

Evaluation with ultrasound revealed 3-4 lymph nodes in the left side of the neck (up to 10.5 mm) suggestive of LAP. There was also one cyst (diameter of 4.5 mm) in the upper lobe of the right thyroid. Several lymph nodes (up to 10 mm) with reactive appearances were observed in the axillary region. However, there was no evidence of LAP in the axillary regions of both sides. Ultrasound revealed moderate splenomegaly (spleens span of 174 mm). Multiple hypochoic masses (up to 12 mm) were observed in the spleen and were recommended to further evaluate for lymphoma. One stone (size of 5 × 5 mm) was also

Table 1. A summary of the patient's laboratory results

Test	Result/Unit	Reference
ESR (1 st hour)	50 mm (High)	0-15
CRP	7.5 mg/L (High)	<6
WBC	6.67 10 ³ /μL	4-11
Neutrophil	60.80%	40-80
Lymphocyte	20.20 % (Low)	20.5-51.1
Monocyte	14.70 % (High)	2-10
Eosinophil	3.90%	0-4
Basophil	0.40%	0-2
RBC	5.04 10 ⁶ /μL	4.2-6.1
Hemoglobin	14.4 g/dL	14-18
Hematocrit	43.8%	42-52
MCV	86.9 fL	80-97
MCH	28.6 pg	27-31
MCHC	32.9 g/dL	32-37
Platelet	199 10 ³ /μL	150-450
BUN	18.1 mg/dL (High)	7-21
Creatinine	2.14 mg/dL (High)	0.7-1.4
eGFR	35.6 mL/min/1.73 m ²	Stage 3b (Moderate to severe loss of RF: 30-44)
Uric acid	10.7 mg/dL (High)	3.5-7.2
Calcium	12.6 mg/dL (High)	8.6-10.3
Phosphorus	4 mg/dL	2.6-4.5
Sodium	141 mEq/L	136-145
Potassium	4.35 mEq/L	3.5-5.0
Urinalysis		
Color	Yellow	-
Appearance	Semi Turbid	Clear
Specific gravity	1.007	1.005-1.030
pH	6.0	5-8
Protein	Trace	Trace: 10mg/dl
Ketones	Negative	-
Blood	Positive (++++)	Positive (++++):70-50/uL
Glucose	Negative	-
Bilirubin	Negative	-
Urobilinogen	Normal	-
WBC/hpf	5-9	1-2
RBC/hpf	Many	1-2
Dysmorphic RBC	2%	Negative
Squamous EP/hpf	3-5	1-2

Table 1. Continued

Test	Result/Unit	Reference
Path cells/hpf	Negative	-
Granular cast	1-2	-
Crystals/hpf	Calcium oxalate	Negative
Others/hpf	Negative	-
Bacteria/hpf	Few	Negative
Leukocyte esterase	Positive (1+)	Negative
Nitrite	Negative	-
Mucus	Negative	-
LDH	325 U/L	<480
Albumin	4.57 g/dL	3.5-5.2
Vitamin D3	21.83 ng/mL	Insufficient: 10-29
PTH	5.03 pg/mL	11-67
PT-Patient	12.9 Second	12-15.0
PT-Control	13.0 Second	12-13.3
PT-Activity %	100 %	-
INR	1.00	-
aPTT	28.9 Second	25-42
Procalcitonin	0.142 ng/mL	<0.5 (low risk)
Wright test (SAT)	Negative (Titer)	<1/80
Coombs Wright	Negative (Titer)	Negative
T4 (Total Thyroxin)	9.6 μg/dL	Adult male (6.6-10.8)
TSH	2.27 μIU/ML	Adult: 0.39-6.16
Beta-2 microglobulin	6.65 mg/L (High)	1-3
HBsAg	0.507	Nonreactive:<0.9
HBcAb (IgM-ECLIA)	<2.0 Index	Nonreactive:<10
HCV Ab (ELISA)	0.224 Index (Negative)	Nonreactive:<0.9
HIV (1 and 2)-Ab and Ag (ELISA-4 th generation)	0.207 Index	Nonreactive:<0.9

Abbreviations: Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), White blood cells (WBC), Red blood cell (RBC), Mean corpuscular volume (MCV), Mean corpuscular hemoglobin (MCH), Mean corpuscular hemoglobin concentration (MCHC), Blood urea nitrogen (BUN), estimated Glomerular filtration rate (eGFR), Lactate dehydrogenase (LDH), High-power field (hpf), Epithelial (EP), Renal function (RF), Parathyroid hormone (PTH), Prothrombin time (PT), International normalized ratio (INR), Activated partial thromboplastin time (aPTT), Standard tube agglutination test (SAT), Thyroid stimulating Hormone (TSH), Hepatitis B surface antigen (HBsAg), Hepatitis B core antibody (HBcAb), Immunoglobulin M (IgM), Enhanced chemiluminescence immunoassay (ECLIA), Hepatitis C virus antibody (HCV Ab), Human immunodeficiency virus (HIV), Enzyme-linked immunosorbent assay (ELISA), and Antibody and antigen (Ab and Ag).

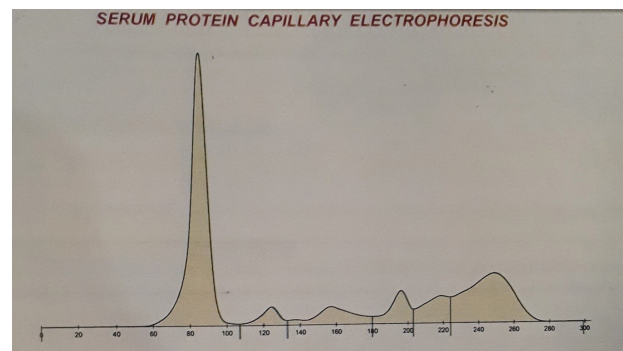
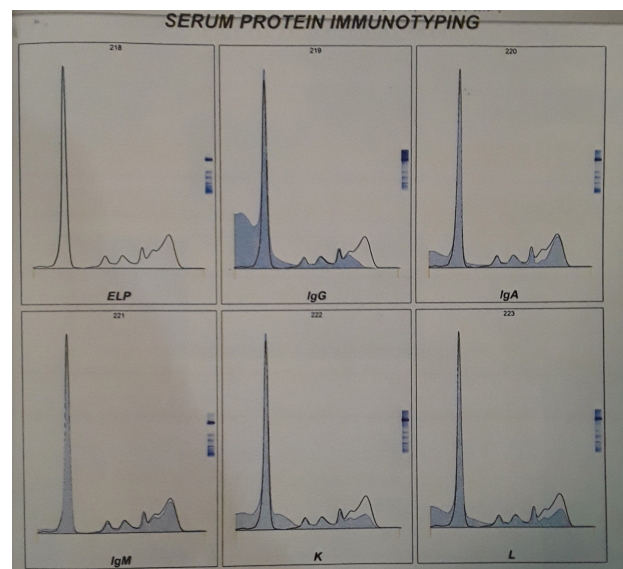
Table 2. The results of the patient's serum electrophoresis

Test	Result	Unit	Reference
Protein electrophoresis (serum)			
Albumin/globulin ratio	0.92 (Low)	Ratio	≥1
Concentration %			
Albumin %	48.0	%	55.8-66.1
Alpha 1-globulin %	3.7	%	2.9-4.9
Alpha 2-globulin %	7.7	%	7.1-11.8
Beta 1-globulin %	6.7	%	4.7-7.2
Beta 2-globulin %	7.8 (High)	%	3.2-6.5
Gamma-globulin %	25.9 (High)	%	11.1-18.8
Absolute concentration			
Protein (Total)	8.1	g/dL	6-8
Albumin	3.9 (Low)	g/dL	4-4.8
Alpha 1-globulin	0.3	g/dL	0.2-0.4
Alpha 2-globulin	0.6	g/dL	0.5-0.9
Beta 1-globulin	0.6 (High)	g/dL	0.3-0.5
Beta 2-globulin	0.6 (High)	g/dL	0.2-0.5
Gamma-globulin	2.1 (High)	g/dL	0.8-1.4

detected in the distal right ureterovesical junction (UVJ). Furthermore, a total colonoscopy was performed due to the patient's abdominal pain and weight loss which was normal. Enhanced computerized tomography (CT) scan of abdomen and pelvis showed significant splenomegaly and mild hepatomegaly with homogeneous parenchyma without any abnormal mass. Splenomegaly in addition to the findings in the lung and mediastinum as mediastinal and subclavian LAP and bilateral pulmonary adenopathy was suggested to be in favor of leukemia/lymphoma than sarcoidosis (Figure 3). However, it was also noted that splenomegaly may occur in about 30% of chronic or subacute cases of sarcoidosis as well.

Consequently, the patient was referred for staging utilizing positron emission tomography (PET)/CT scan with ¹⁸F-fluorodeoxyglucose (F-FDG) with the impression of lymphoma. The result showed multifocal FDG avid reticulonodular changes throughout the lungs, numerous FDG avid enlarged thoracic lymph nodes, enlarged spleen with diffuse increased FDG uptake, several FDG avid abdominopelvic lymph nodes, and several small FDG avid subcutaneous lesions. The ¹⁸F-FDG PET/CT scan reported that the findings were in favor of lymphoproliferative disorders and recommended excisional biopsy from mediastinal lymph nodes. In addition, a few calcified stones were also detected in the left kidney and proximal ureter (Figure 4).

Histological evaluation of the core needle biopsy of the splenic mass showed several granulomas composed of epithelioid histiocytes, lymphocytes, and multinucleated giant cells. However, there was no necrosis and no acid-fast bacilli with Ziehl-Neelsen staining. Non-necrotizing granulomatous inflammation was established as the diagnosis. Histological evaluation of the core needle biopsy of the supraclavicular lymph node showed a lymph node tissue with nodal effacement by small granulomas

**Figure 1.** The image of the patient's serum protein capillary electrophoresis.**Figure 2.** Serum protein immunotyping: Presence of weak immunoglobulin G (IgG) kappa and lambda; Non-monoclonal.

composed of epithelioid cells with scattered Langhans giant cells and lymphocytes. Foci of calcification were seen. However, there was no necrosis and no evidence of malignancy. Therefore, a diagnosis of non-necrotizing granulomatous inflammation (sarcoid type) was established.

Evaluation of bone marrow aspiration and bone marrow biopsy showed normo-cellular marrow with tri-lineage hematopoiesis without any evidence of lymphoproliferative disorders. Flow cytometry and immunophenotyping of the bone marrow showed no excess myeloblast and the lymphocytic gate consisted about 7% of the total gate which were mostly T-cells with normal expression of pan-T-markers and normal CD4/CD8 ratio. B-cells showed no aberrancy (details in Table 3).

A second pathological evaluation of the core needle biopsies of the supraclavicular lymph and splenic mass reported granulomatous inflammation containing several Schaumann's bodies and suggested that the findings were highly characteristic for sarcoidosis. Consequently, a final diagnosis of sarcoidosis was established and the patient

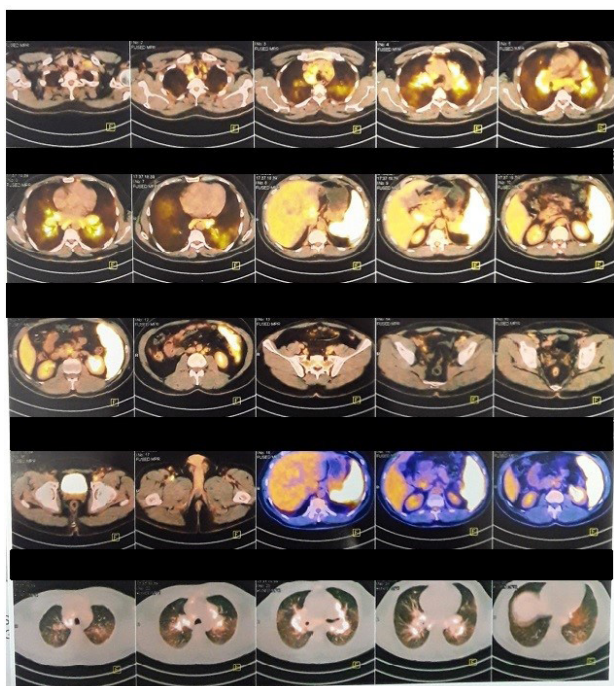


Figure 3. Enhanced computerized tomography (CT) scan: significant splenomegaly and mild hepatomegaly with homogeneous parenchyma without any abnormal mass.

was referred to a rheumatology subspecialist for further management of his condition.

Discussion

Sarcoidosis is a systemic granulomatous disease that is caused by T-helper responses and involves multiple organs (2,3). Antigens activate macrophages and, in turn, stimulate immune responses through T-cells and macrophages. Consequently, extensive cytokines, chemokines, and oxygen radicals are released that cause sarcoidosis. The involved organs contain epithelioid granulomas formed by activated macrophages. Granulomas are surrounded by CD4 T-cells and may contain Langerhans cells with Schaumann and asteroid bodies (3-6,8).

Sarcoidosis mostly occurs in the third and fourth decades of life and is more common in females and some ethnicities such as African-Americans (7,9). In Iran, the mean age of patients with sarcoidosis has been reported

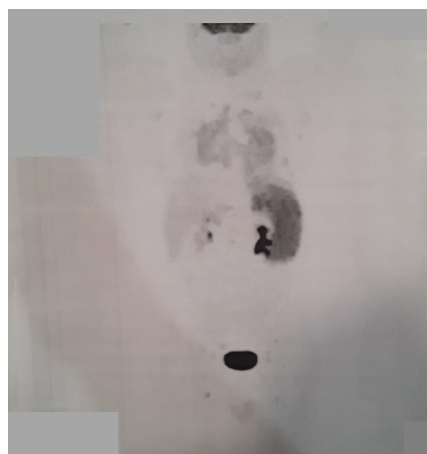


Figure 4. PET/CT scan with 18F-FDG (positron emission tomography/computerized tomography scan with 18F-fluorodeoxyglucose). Multifocal FDG avid reticulonodular changes throughout the lungs, numerous FDG avid enlarged thoracic lymph nodes, enlarged spleen with diffuse increased FDG uptake, several FDG avid abdominopelvic lymph nodes, and several small FDG avid subcutaneous lesions. Few calcified stones were detected in the left kidney and proximal ureter.

to be 42.8 ± 9.8 years with a female predominance (about 63.5%) (10). It has been reported that the incidence is higher in northern European compared to southern European countries (3). The mortality rate of sarcoidosis is about 2-4% which is mostly due to lung complications (lung fibrosis and respiratory failure) and occasionally, sudden cardiac death due to cardiac complications (3).

The exact etiology of sarcoidosis is still unknown. However, it has been suggested that it is a multifactorial disorder involving different genes that stimulate an immune response following triggers with specific antigens. Sarcoidosis is a polygenic disorder and is associated with HLA-A, HLA-B, and HLA-C (class I) and HLA-DP, HLA-DQ, HLA-DR (class II). In addition, there seems to be involvement of genotypes and environmental factors. However, less than 10% of sarcoidosis cases have been reported to be familiar (3,5,6).

Lungs are the most involved organs in sarcoidosis (in over 90% of the cases). However, other organs may also be involved. Therefore, a comprehensive workup is required to search for extra-pulmonary involvements

Table 3. The results of the multicolor flow cytometry

Marker	% Of total	Lymphocytic gate	Blastic gate	Result
CD5	-	22%	-	-
CD19	-	4%	-	-
CD5/CD19	<1%	1%	-	Negative
CD34	-	-	35%	-
CD117	-	-	35%	-
CD34/CD117	1%	-	27.5%	Negative
CD3	-	22%	-	-
CD3/CD4	-	63%	-	-
CD3/CD8	-	33%	-	-
CD2	-	25%	-	-

(3, 4). Dyspnea, cough, chest pain, and wheezing are the most common signs of sarcoidosis (2). The most common clinical presentations of sarcoidosis in Iranian patients include cough (59.6-77.3%), dyspnea (37.4-61.7%), weight loss (50%), fever (28.7-42.5%), fatigue (5%), chest pain (18.3%), arthralgia (16.2%), skin involvement (12.3%), and eye involvement (4.8%). Hypercalcemia and erythema nodosum have been reported in 2.8% and 7.1% of the patients, respectively. Increased serum ACE levels were observed in 77.4-83% of the patients and about 65.3% of the patients had normal pulmonary function tests (PFTs). The restrictive pattern was more common than the obstructive pattern in Iranian patients with sarcoidosis (10, 11). Jayakrishnan et al reported that some constitutional symptoms were more common in Iran compared to other Middle Eastern countries, such as fever (42.5%) and weight loss (50%). In contrast, fatigue (<5%) was less common in patients with sarcoidosis in Iran (2). The present case presented with fatigue, weight loss, decreased GFR, and hypercalcemia.

The lung complications of sarcoidosis include pulmonary artery hypertension, pulmonary infiltration, restrictive lung involvement, airflow obstruction, decreased diffusion capacity, interstitial lung complications, lung fibrosis, and respiratory failure. In addition, there are usually hilar and mediastinal LAPs but about 20% of the patients present with peripheral LAPs (the cervical, axillary, or inguinal lymph nodes). Neurosarcoidosis may involve the central nervous system (CNS) or the peripheral nervous system. The most common manifestation of neurosarcoidosis is meningeal involvement of the CNS. The pituitary may also be involved and present with diabetes insipidus. Peripheral neuropathy or muscle involvement may present with polymyositis, chronic myopathy, chronic fatigue, muscle nodules, or Bell's palsy. Anterior uveitis (acute or chronic iridocyclitis), posterior uveitis, uveoparotitis, dry keratoconjunctivitis are commonly observed in patients with sarcoidosis. There may be thyroid impairment, parotid enlargement, xerostomia, xerophthalmia, erythema nodosum, Löfgren syndrome, and lupus pernio. Cardiac involvement in patients with sarcoidosis could include heart failure or myocardial granulomas. Hepatomegaly and splenomegaly are common in patients with sarcoidosis. Kidney complications may include renal calculi, cortical or medullary granulomas, and renal impairment. Some patients may experience arthralgia or bone involvements and the radiological features of bone involvement vary. Patients with sarcoidosis may present with increased levels of 1,25-dihydroxy vitamin D, hypercalciuria, and hypercalcemia (3, 4, 12-26).

Sarcoidosis is a disorder that does not have a pathognomonic clinical feature but involves multiple organs. The diagnosis of sarcoidosis is established by its clinical presentations and radiological features in addition to a histological report of non-necrotic, non-caseating granulomas on a biopsy specimen. In addition,

other possible diagnoses should be excluded (such as lymphoproliferative diseases, infections, interstitial lung diseases, storage disorders, and malignancy (2,3). The present case presented with clinical features of lymphoma but on further workup was diagnosed with sarcoidosis.

It has been reported that the active inflammation of the lung parenchymal in patients with sarcoidosis is better detected by PET/CT scan than high-resolution CT. Studies have reported that patients that had lung parenchyma involvement in PET/CT scans, had lower lung function tests, diffusion test results, and forced vital capacity (FVC)% (27-29). In addition, PET/CT scans can show extra-thoracic involvement in patients with sarcoidosis (such as lymph nodes, bone marrow, and solid organs). Extra-thoracic involvement has been reported to be present in 27.5% to 75% of the cases (27,29). Moreover, Kemal et al reported that the neutrophil/lymphocyte ratio, PET/CT scans, maximum standardized uptake value (SUVmax), and diffusing capacity for carbon monoxide (DLCO) % could predict the disease progression. They showed that after a one-year follow-up, the mediastinal lymph nodes had a progression rate of 60% in those patients that had a SUVmax value of 14 or more in their PET/CT scans (27). Furthermore, Schimmelpennink et al recommended using SUVmax of the lung parenchyma to monitor the response to infliximab therapy in patients with pulmonary sarcoidosis. They reported that a SUVmax of more than 7.5 before treatment with infliximab predicted a 5% response in FVC and a SUVmax >9.2 predicted a 5% response in DLCO corrected for hemoglobin (DLCOc) (30).

The common treatment options for sarcoidosis include corticosteroids (prednisone) as the first-line treatment medications. The second-line agents include methotrexate, hydroxychloroquine, leflunomide, azathioprine, and mycophenolate. Infliximab and adalimumab are the third-line agents used in the treatment of sarcoidosis (31). Based on the reports from Iran, around 46.2%-44.9% of patients with sarcoidosis showed clinical or radiological improvement indicating a good prognosis (2, 10, 11). Furthermore, it is important to follow up with the patients for complications such as osteoporosis and osteopenia as long-term complications of treatment with steroids to avoid unnecessary bone fractures or stone formations (17,32).

Conclusion

Sarcoidosis is a systemic granulomatous disorder that involves multiple organs. It is diagnosed by its clinical presentations, radiological features, and non-caseating granulomas on histology. However, possible diagnoses such as lymphoproliferative diseases, infections, and malignancies should be excluded.

Authors' contribution

Conceptualization, Validation, Resources, Supervision, Project

Administration, MK; Writing—Original Draft Preparation: SH; Investigation, Data Curation, Writing—Review and Editing, Visualization: MK & SH.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This manuscript followed the principals of the World Medical Association Declaration of Helsinki. Written informed consent was taken from the patients for its publication. Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

Funding/Support

None.

References

- Valeyre D, Prasse A, Nunes H, Uzunhan Y, Brillet PY, Müller-Quernheim J. Sarcoidosis. *Lancet*. 2014;383:1155-67. doi: 10.1016/s0140-6736(13)60680-7.
- Jayakrishnan B, Al-Busaidi N, Al-Mubaihsi S, Al-Rawas OA. Sarcoidosis in the Middle East. *Ann Thorac Med*. 2019;14:106-15. doi: 10.4103/atm.ATM_227_18.
- Bargagli E, Prasse A. Sarcoidosis: a review for the internist. *Intern Emerg Med*. 2018;13:325-31. doi: 10.1007/s11739-017-1778-6.
- Hunninghake GW, Costabel U, Ando M, Baughman R, Cordier JF, du Bois R, et al. ATS/ERS/WASOG statement on sarcoidosis. American Thoracic Society/European Respiratory Society/World Association of Sarcoidosis and other Granulomatous Disorders. *Sarcoidosis Vasc Diffuse Lung Dis*. 1999;16:149-73.
- Moller DR, Chen ES. Genetic basis of remitting sarcoidosis: triumph of the trimolecular complex? *Am J Respir Cell Mol Biol*. 2002;27:391-5. doi: 10.1165/rcmb.2002-0164PS.
- Prior C, Knight RA, Herold M, Ott G, Spiteri MA. Pulmonary sarcoidosis: patterns of cytokine release in vitro. *Eur Respir J*. 1996;9:47-53. doi: 10.1183/09031936.96.09010047.
- Kouranos V, Jacob J, Wells AU. Severe Sarcoidosis. *Clin Chest Med*. 2015;36:715-26. doi: 10.1016/j.ccm.2015.08.012.
- Chen ES, Song Z, Willett MH, Heine S, Yung RC, Liu MC, et al. Serum amyloid A regulates granulomatous inflammation in sarcoidosis through Toll-like receptor-2. *Am J Respir Crit Care Med*. 2010;181:360-73. doi: 10.1164/rccm.200905-0696OC.
- Duchemann B, Annesi-Maesano I, Jacobe de Naurois C, Sanyal S, Brillet PY, Brauner M, et al. Prevalence and incidence of interstitial lung diseases in a multi-ethnic county of Greater Paris. *Eur Respir J*. 2017;50. doi: 10.1183/13993003.02419-2016.
- Foumani AA, Akhoundzadeh N, Karkan MF. Sarcoidosis, a report from Guilan (an Iranian northern province) (2001-09). *Sarcoidosis Vasc Diffuse Lung Dis*. 2015;31:282-8.
- Amoli K. Sarcoidosis: A report from 310 patients with sarcoidosis in Iran. Teymoozade Tabib Publication. Back to cited text.
- McDonnell MJ, Saleem MI, Wall D, Gilmartin JJ, Rutherford RM, O'Regan A. Predictive value of C-reactive protein and clinically relevant baseline variables in sarcoidosis. *Sarcoidosis Vasc Diffuse Lung Dis*. 2016;33:331-40.
- Baughman RP, Papanikolaou I. Current concepts regarding calcium metabolism and bone health in sarcoidosis. *Curr Opin Pulm Med*. 2017;23:476-81. doi: 10.1097/mcp.0000000000000400.
- Kefella H, Luther D, Hainline C. Ophthalmic and neuro-ophthalmic manifestations of sarcoidosis. *Curr Opin Ophthalmol*. 2017;28:587-94. doi: 10.1097/icu.0000000000000415.
- Tugal-Tutkun I. Systemic vasculitis and the eye. *Curr Opin Rheumatol*. 2017;29:24-32. doi: 10.1097/bor.0000000000000345.
- Birnbaum AD, French DD, Mirsaeidi M, Wehrli S. Sarcoidosis in the national veteran population: association of ocular inflammation and mortality. *Ophthalmology*. 2015;122:934-8. doi: 10.1016/j.ophtha.2015.01.003.
- Bargagli E, Olivieri C, Penza F, Bertelli P, Gonnelli S, Volterrani L, et al. Rare localizations of bone sarcoidosis: two case reports and review of the literature. *Rheumatol Int*. 2011;31:1503-6. doi: 10.1007/s00296-009-1315-7.
- Carter JM, Howe BM, Inwards CY. Conditions Simulating Primary Bone Neoplasms. *Surg Pathol Clin*. 2017;10:731-48. doi: 10.1016/j.path.2017.04.012.
- Agrawal A, Sahni S, Iftikhar A, Talwar A. Pulmonary manifestations of renal cell carcinoma. *Respir Med*. 2015;109:1505-8. doi: 10.1016/j.rmed.2015.10.002.
- Mehta D, Lubitz SA, Frankel Z, Wisnivesky JP, Einstein AJ, Goldman M, et al. Cardiac involvement in patients with sarcoidosis: diagnostic and prognostic value of outpatient testing. *Chest*. 2008;133:1426-35. doi: 10.1378/chest.07-2784.
- Culver DA, Ribeiro Neto ML, Moss BP, Willis MA. Neurosarcoidosis. *Semin Respir Crit Care Med*. 2017;38:499-513. doi: 10.1055/s-0037-1604165.
- Ogawa Y, Saraya T, Fujiwara M, Takizawa H. Massive Neurosarcoidosis. *Intern Med*. 2017;56:2537-8. doi: 10.2169/internalmedicine.8217-16.
- Hebel R, Dubaniewicz-Wybieralska M, Dubaniewicz A. Overview of neurosarcoidosis: recent advances. *J Neurol*. 2015;262:258-67. doi: 10.1007/s00415-014-7482-9.
- Said G. Sarcoidosis of the peripheral nervous system. *Handb Clin Neurol*. 2013;115:485-95. doi: 10.1016/b978-0-444-52902-2.00027-8.
- Levitt DG, Levitt MD. Protein losing enteropathy: comprehensive review of the mechanistic association with clinical and subclinical disease states. *Clin Exp Gastroenterol*. 2017;10:147-68. doi: 10.2147/ceg.s136803.
- Cunha BA, Sivarajah T, Jimada I. Sarcoidosis with fever and a splenic infarct due to CMV or lymphoma? *Heart Lung*. 2017;46:394-6. doi: 10.1016/j.hrtlng.2017.05.009.
- Kemal CT, Aylin OA, Volkan K, Seda M, Recep B, Can S. The importance of PET/CT findings and hematological parameters in prediction of progression in sarcoidosis cases. *Sarcoidosis Vasc Diffuse Lung Dis*. 2017;34:242-50. doi: 10.36141/svldl.v34i3.5299.
- Mostard RL, Verschakelen JA, van Kroonenburgh MJ, Nelemans PJ, Wijnen PA, Vöö S, et al. Severity of pulmonary involvement and (18)F-FDG PET activity in sarcoidosis. *Respir Med*. 2013;107:439-47. doi: 10.1016/j.rmed.2012.11.011.
- Cremers JP, Van Kroonenburgh MJ, Mostard RL, Vöö SA, Wijnen PA, Koek GH, et al. Extent of disease activity assessed by 18F-FDG PET/CT in a Dutch sarcoidosis population. *Sarcoidosis Vasc Diffuse Lung Dis*. 2014;31:37-45.
- Schimmelpennink MC, Vorselaars ADM, Veltkamp M, Keijsers RGM. Quantification of pulmonary disease activity in sarcoidosis measured with (18)F-FDG PET/CT: SUVmax versus total lung glycolysis. *EJNMMI Res*. 2019;9:54. doi: 10.1186/s13550-019-0505-x.
- Gerke AK. Treatment of Sarcoidosis: A Multidisciplinary Approach. *Front Immunol*. 2020;11:545413. doi: 10.3389/fimmu.2020.545413.
- Minisola S, Cipriani C, Occhiuto M, Pepe J. New anabolic therapies for osteoporosis. *Intern Emerg Med*. 2017;12:915-21. doi: 10.1007/s11739-017-1719-4.