

Case Report

A young female with pulmonary sarcoidosis masquerading as bronchial asthma

Rahman MM^a, Habib FB^b, Kabir AI^c, Khaled SM^d, Rahman S^e, Jahan R^f

ABSTRACT

Sarcoidosis is a multisystem granulomatous disorder that commonly affects the lungs. Accumulation of non-necrotizing granulomas and subsequently fibrosis in the lungs and other organs accounts for the clinical manifestations. A young female patient complaining of shortness of breath, cough and wheezing. First she was treated as asthma for long time and there was no improvement. Then a computed tomography of chest was performed and fine needle aspiration from hilar lymphnode confirmed pulmonary sarcoidosis. She received corticosteroid therapy for six months and her health situation become well.

Key words: sarcoidosis, computed tomographic scans, fine needle aspiration cytology, asthma, serum calcium.

BIRDEM Med J 2026; 16(1): 33-35

DOI: <https://doi.org/10.3329/birdem.v15i1.87662>

INTRODUCTION

Sarcoidosis is a multisystem, chronic granulomatous disease of unknown etiology that commonly affects the lungs¹. After full stop Whereas some patients with pulmonary sarcoidosis are asymptomatic, many report cough, dyspnea, fatigue, unintentional weight loss, and night sweats². Pulmonary sarcoidosis usually presents with nonspecific symptoms and that's why computed tomographic (CT) scans are instrumental for diagnosis. Bilateral, symmetric micronodules in a peribronchovascular distribution with upper and middle lung zone predominance accompanied by bilateral, symmetric hilar lymphadenopathy are the common radiological findings³. We present a case of stage II pulmonary sarcoidosis who was treated initially as a case of bronchial asthma.

CASE REPORT

A 25-year-old lady presented to our hospital with the complaints of dry cough and occasional shortness of breath and wheezing for last 1 year. She had sought repeated medical attention for this and several times she was treated a case of asthma with different types of inhalers with no significant improvement. Our patient was otherwise healthy, with no known chronic diseases and her systemic review was unremarkable. Her vitals were stable and within normal limits and no regional lymph node enlargement was found. Respiratory, cardiovascular, abdominal, and neurological examinations were unremarkable. Laboratory tests showed hemoglobin of 12 g/dL, erythrocyte sedimentation rate of 29 mm/h, serum calcium 8 mg/dl, serum angiotensin converting enzyme 65 U/L (ref. value), sputum for acid-fast bacilli/ gene xpert negative,

Author information

- Mohammed Mirazur Rahman, Assistant Professor, Department of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital, Dhaka, Bangladesh.
- Farjana-Binte-Habib, Assistant Professor, Department of Microbiology, Dhaka Medical College, Dhaka, Bangladesh.
- Ahmed Imran Kabir, Registrar, National Institute of Diseases of the Chest and Hospital, Dhaka, Bangladesh.
- Shaifuddin Mohammad Khaled, Assistant Professor, Department of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital, Dhaka, Bangladesh.
- Samia Rahman, Junior Consultant, Chest Disease Clinic, Kishoregonj, Bangladesh.
- Rawnak Jahan, Pulmonologist, Department of Respiratory Medicine, United Hospital, Dhaka, Bangladesh.

Address of correspondence: Mohammed Mirazur Rahman, Assistant Professor, Department of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital, Dhaka, Bangladesh. Email: mirazrahman41@gmail.com

Received: September 27, 2025

Revision received: October 9, 2025

Accepted: January 20, 2026

Mantoux test 2 mm at ... Chest X-ray showed bilateral hilar lymphadenopathy (Figure 1). CT scan of chest showed multiple enlarged lymphnodes in pre- and para-tracheal and both hilar regions (Figure 2). CT scan also showed diffuse increased attenuation, patchy opacities, septal thickening and centrilobular nodules with branching pattern in most of the segments of both lungs predominantly central region (Figure 3). FNAC of left hilar lymphnode revealed moderate cellular material containing lymphocytes, polymorphs, pulmonary macrophages, a few giant cells, focal collections of



Figure 1. Chest X-ray showed bilateral hilar lymphadenopathy

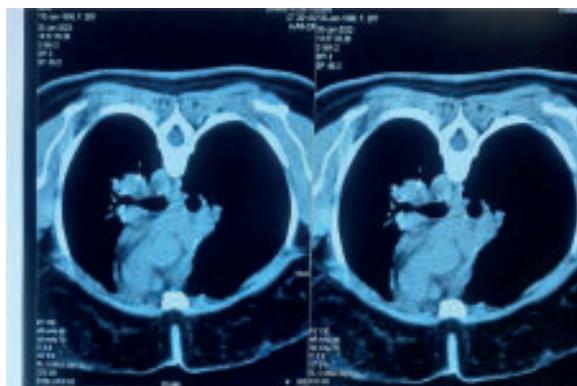


Figure 2. CT scan of chest showed multiple enlarged lymphnodes in pre and para-tracheal and both hilar region



Figure 3. CT scan OF chest showed diffuse increased attenuation, patchy opacities, septal thickening and centrilobular nodules with branching pattern in most of the segments of both lungs.

epithelioid cells without any caseation compatible with sarcoidosis. She was started on treatment with prednisolone 30 mg daily. Within 1 month of treatment, she began to experience improvement of her cough and dyspnea. We tapered the dose to 7.5 mg within next 3 months and continued for 6 months.

DISCUSSION

Sarcoidosis is a multisystem disease of unknown etiology. Lung involvement is very common. Patients with pulmonary sarcoidosis are often asymptomatic at presentation. But symptomatic patients usually report dry cough, dyspnea or nonspecific chest discomfort⁴. Pulmonary symptoms are frequently accompanied by systemic manifestations such as fatigue, malaise, fever, and weight loss⁵. Typical radiological presentation of pulmonary sarcoidosis is bilateral hilar lymphadenopathy but atypical radiographic presentations occur in 15–25% of patients⁶. These findings can include diffuse ground-glass opacities, honeycombing, multiple nodules, or necrotizing consolidations⁶. If the patient does not have extrathoracic involvement in accessible tissues or if a biopsy would be less preferable than a bronchoscopy or transbronchial needle aspiration (TBNA) of radiographically enlarged intrathoracic lymph nodes or lung parenchyma are the next choice of investigation. The characteristic morphologic feature of histopathology of sarcoidosis is the non-necrotizing

granuloma. The sarcoidosis granuloma is a focal, chronic inflammatory reaction formed by the accumulation of epithelial cells, monocytes, lymphocytes, macrophages, and fibroblasts⁷. Our patient presented with typical features of asthma like cough, shortness of breath and occasionally with wheeze which is atypical presentation of pulmonary sarcoidosis. That's why initially she was treated with bronchodilator inhaler. Then we diagnosed this is a case of stage II pulmonary sarcoidosis on the basis of radiological features and histopathology findings. Asymptomatic pulmonary sarcoidosis have low risk of disease progression and usually do not require treatment. A large majority of asymptomatic patients with stage I disease and nodular sarcoidosis will have spontaneous remission, while patients with stage II and III disease have a more variable disease course⁸. Oral corticosteroids are the first line of therapy in patients with symptomatic or progressive disease. Treatment with prednisolone 0.5 mg/kg/day for 4 weeks, then reduced to maintenance dose which will control symptoms and disease progression, should be used for a period of 6-24 months⁹. Methotrexate is a commonly chosen second-line agent because it has been shown to be effective in decreasing the necessary dose of prednisone when used in combination¹⁰. For refractory cases, leflunomide and tumor necrosis factor- α antagonists, such as infliximab, adalimumab or etanercept, have demonstrated efficacy in small observational studies.¹¹

Conclusion

Atypical manifestations of pulmonary sarcoidosis are challenging and also difficult to treat. Here we presented a case of pulmonary sarcoidosis who was initially long term treated as asthma. Only a few case have been reported in patients who has asthma like features but finally diagnosed pulmonary sarcoidosis.

Authors' contribution: MMR was involved in the diagnosis, patient management, manuscript writing and literature review. FBH helped in editing. All authors were involved in evaluation and management of the case.

Consent: Informed written consent was taken from patient regarding publication of this case report. Send the consent paper signed by the patient

Conflicts of interest: Nothing to declare.

REFERENCES

1. Baughman RP, Culver DA, Judson MA. A concise review of pulmonary sarcoidosis. *American journal of respiratory and critical care medicine*. 2011 Mar 1;183(5):573-81.
2. Iannuzzi MC, Rybicki BA, Teirstein AS. Sarcoidosis. *N Engl J Med*. 2007;357(21):2153-65.
3. Criado E, Sánchez M, Ramírez J, Arguis P, De Caralt TM, Perea RJ, Xaubet A. Pulmonary sarcoidosis: typical and atypical manifestations at high-resolution CT with pathologic correlation. *Radiographics*. 2010 Oct;30(6):1567-86.
4. Judson MA. The clinical features of sarcoidosis: a comprehensive review. *Clinical reviews in allergy & immunology*. 2015 Aug;49:63-78.
5. Sharma OP. Fatigue and sarcoidosis. *European Respiratory Journal*. 1999 Apr 1;13(4):713-4.
6. Kachalia AG, Ochieng P, Kachalia K, Rahman H. Rare coexistence of sarcoidosis and lung adenocarcinoma. *Respiratory Medicine Case Reports*. 2014 Jan 1;12:4-6.
7. Ma Y, Gal A, Koss MN. The pathology of pulmonary sarcoidosis: update. In *Seminars in diagnostic pathology* 2007 Aug 1 (Vol. 24, No. 3, pp. 150-161). WB Saunders.
8. Bradley B, Branley HM, Egan JJ, Greaves MS, Hansell DM, Harrison NK, Hirani N, Hubbard R, Lake F, Millar AB, Wallace WA. Interstitial lung disease guideline: the British Thoracic Society in collaboration with the Thoracic Society of Australia and New Zealand and the Irish Thoracic Society (vol 63, Suppl V, pg v1, 2008). *Thorax*. 2008 Nov 1;63(11):1029.
9. Thillai M, Atkins CP, Crawshaw A, Hart SP, Ho LP, Kouranos V, Patterson KC, Sreaton NJ, Whight J, Wells AU. BTS Clinical Statement on pulmonary sarcoidosis. *Thorax*. 2021 Jan 1;76(1):4-20.
10. Judson MA. Advances in the diagnosis and treatment of sarcoidosis. *F1000Prime Rep*. 2014;6:89.
11. Brito-Zerón P, Pérez-Alvarez R, Pallarés L, Retamozo S, Baughman RP, Ramos-Casals M. Sarcoidosis: an update on current pharmacotherapy options and future directions. Expert opinion on pharmacotherapy. 2016 Dec 11;17(18):2431-48.