

Sarcoidosis with bronchial asthma: A case report

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Abstract

Sarcoidosis is a multisystem disorder of unknown etiology usually affecting the respiratory tract and characterized by the formation of immune granulomas in involved organs. Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation. Association between sarcoidosis and asthma is unusual and controversial. A 47-year-old female with clinical diagnosis of bronchial asthma developed skin lesions, bilateral hilar with mediastinal lymphadenopathy and was diagnosed with sarcoidosis on the basis of skin biopsy and serum ACE levels.

Keywords: ACE level; Asthma; Granuloma; Mediastinal lymphadenopathy; Sarcoidosis

Introduction

Sarcoidosis is a multisystem disorder characterized by non-caseating granulomatous inflammation at sites of disease [1-3]. In sarcoidosis, lungs and intra-thoracic lymph nodes are most commonly involved (more than 90%). Chronic skin sarcoidosis is seen in approximately 25% of patients, usually manifesting as plaques or subcutaneous nodules. It generally affects the age group of 25–40 years [4-6]. The pathologic hallmark of sarcoidosis is the presence of discrete, non-caseating, epithelioid cell granulomas. Bronchial Asthma is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation [7]. The occurrence of both sarcoidosis and bronchial asthma in same patient is unusual. However a subgroup of sarcoidosis patients have bronchial hyper-responsiveness and airway obstruction that may respond to bronchodilators. There are some reports that patients with sarcoidosis show bronchial hyper reactivity and tend to develop obstructive pulmonary changes [8-10], while others have reported that this is not the case [11].

Case report

A 47-year-old housewife presented with complaints of recurrent common cold, intermittent breathlessness for two years, and rash over the left upper limb for last one month. Patient had history of intermittent use of antihistaminic (Cetirizine) for last two years. There was no history of hypertension, diabetes or anti-tubercular drug therapy. There was no family history of similar complaints either. On examination patient was stable and she had rash limited over left upper limb which was non tender. Bilateral ronchi was heard on auscultation. On investigations, patient had haemoglobin (Hb)-13gm%, total leucocyte count(TLC)-8000/mm³, differential

leucocyte count(DLC) polymorph cell 60%, lymphocyte 35%, eosinophil 5%, monocyte 0%. Chest X-ray PA view (Fig. 1) was suggestive of bilateral hilar lymphadenopathy. Serum IgE level was 410 IU/ml. Mantoux test was negative. On spirometry patient had FEV1/FVC value 68.4 and FEV1 2.37 L. Skin biopsy from left upper limb was suggestive of multiple discrete naked epithelioid cell granulomas in upper dermis suggestive of sarcoidosis. Serum ACE was 72.4 U/L. CECT thorax [Fig. 2, 3 &4] was suggestive of mediastinal and bilateral hilar lymphadenopathy. Bronchial Asthma diagnosis was made clinically and with spirometry while mediastinal and bilateral hilar lymphadenopathy with cutaneous lesions were supportive for sarcoidosis. She was diagnosed as bronchial asthma with sarcoidosis. Patient was initiated on inhaled medications with long acting β_2 agonist and corticosteroids (Formetrol and Budesonide). For sarcoidosis patient was given oral prednisolone. Patient had improvement in symptoms during course of time.

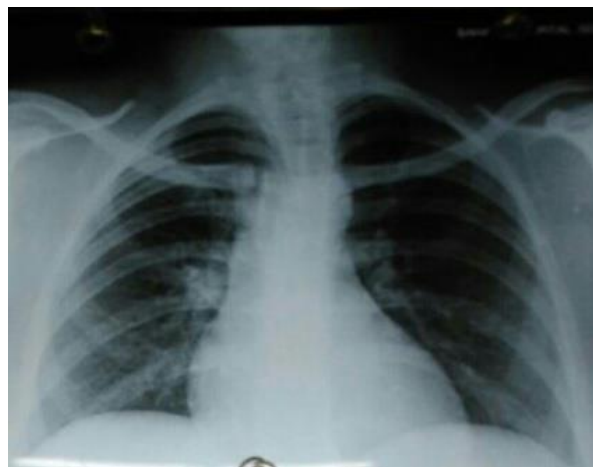


Fig. 1: Chest X-ray PA view suggestive of bilateral hilar lymphadenopathy

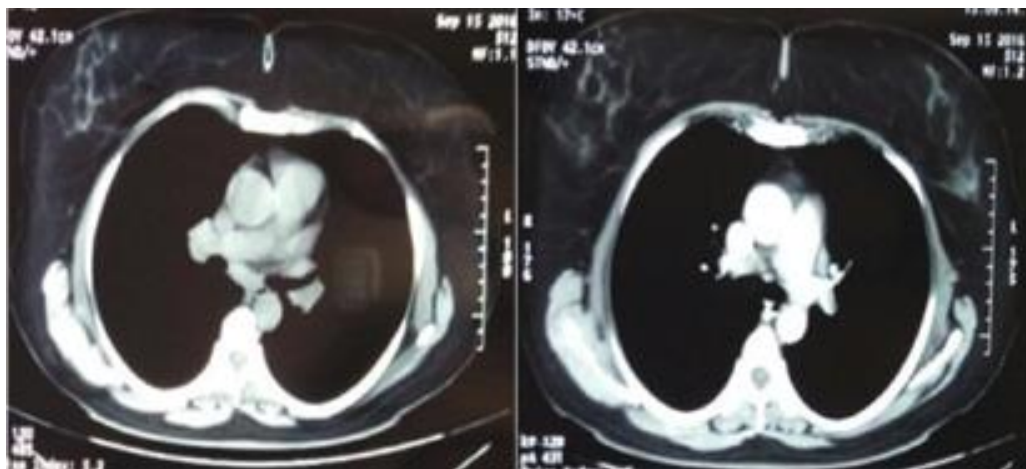


Fig. 2,3: CECT Thorax mediastinal window showing bilateral hilar and mediastinal lymphnode

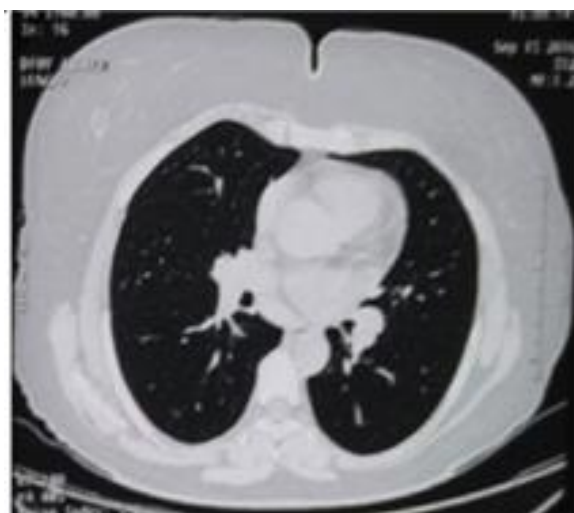


Fig. 4: CECT thorax lung window showing hilar lymphadenopathy

Discussion

Sarcoidosis can lead to bronchial hyper responsiveness but such presentation is unusual [11]. In this case presence of sarcoidosis was established by skin biopsy, bilateral hilar and mediastinal lymphadenopathy, raised serum ACE levels, and negative Mantoux test. However diagnosis of bronchial asthma was made on the basis of clinical and spirometry findings. In sarcoidosis obstructive impairment is as common as restrictive impairment, a subgroup of patients have bronchial hyper-responsiveness and airway obstruction that may respond to bronchodilators. Several mechanisms can be incriminated apart from bronchial distortion related to pulmonary fibrosis, this complication can also result from the direct localization of sarcoid lesions on airways, or more seldom from an extrinsic compression by hilar or mediastinal lymphadenopathy. The pathologic hallmark of sarcoidosis is the presence of discrete, noncaseating, epitheloid cell granulomas. Environmental exposures are linked to sarcoidosis due to seasonal clustering of the disease with a predilection for

winter and early spring months [12]. Sarcoidosis is associated with features of autoimmunity, such as antinuclear antibodies, rheumatoid factor, hypergammaglobulinemia, and immune complexes. Abnormal immunologic reactions present in sarcoidosis cause activation of B lymphocytes to produce immunoglobulin and thus may accelerate IgE-mediated allergic reaction [13]. However, in this case symptoms of bronchial asthma came earlier than of sarcoidosis. A common noxious stimulus triggering pathogenesis of both sarcoidosis and bronchial asthma can be a possible explanation for this case.

Conflicts of interest: None declared

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Consent: Patient's written and informed consent was taken for the publication of case details and accompanying pictures.

References

1. Siltzbach LE, James DG, Neville E, Turiaf J, Battesti JP, Sharma OP, et al. Course and prognosis of sarcoidosis around the world. *Am J Med.* 1974;57:847–52.
2. Neville E, Walker AN, James DG. Prognostic factors predicting the outcome of sarcoidosis: an analysis of 818 patients. *Q J Med.* 1983;52:525–33.
3. Baughman RP, Lower EE, du Bois R. Sarcoidosis. *Lancet.* 2003;361:1111–118.
4. Kern DG, Neill MA, Wrenn DS, Varone JC. Investigation of a unique time-space cluster of sarcoidosis in firefighters. *Am Rev Respir Dis.* 1993;148:974–80.
5. Parkes SA, Baker SB, Bourdillon RE, Murray CR, Raskit M. Epidemiology of sarcoidosis in the Isle of Man: 1. A case-control study. *Thorax.* 1987;42:420–26.
6. Hills SE, Parkes SA, Baker SB. Epidemiology of sarcoidosis in the Isle of Man: Evidence of space-time clustering. *Thorax.* 1987;42:427–30.
7. Global Strategy for Asthma Management and Prevention. Global Initiative for Asthma 2016. Available from URL: <http://www.ginasthma.org>. Last accessed 2017 on January 21.

8. Nowak D, Kanzow G, Meyer A, Magnussen H. Bronchial hypersensitivity in patients with sarcoidosis. *Atemwegs-Lungenkrankh.*1989;6:249.
9. Bechtel JJ, Starr Till, Dantzker DR, Bower JS. Airway hyper reactivity in patients with sarcoidosis. *Am Rev Respir Dis.*1981;124:759.
10. Olafsson M, Simonsson BG, Hansson S-B. Bronchial reactivity in patients with recent pulmonary sarcoidosis. *Thorax.*1985;40:51.
11. Scadding JG. Prognosis of intrathoracicsarcoidosis in England. *BMJ* 1961;4:1165-172.
12. Wilsher ML. Seasonal clustering of sarcoidosis presenting with erythema nodosum. *Eur Respir J.* 1998;12:1197-199.
13. Fanburg BL, Sarcoidosis. In: Wyngaarden JB, Smith LH, editors. *Textbook of Medicine*, Philadelphia, WB Saunders Company, 1988. p. 452.