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UNEXPLAINED HARMONY; TUBERCULOSIS AND SARCOIDOSIS

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ABSTRACT

Sarcoidosis and tuberculosis are chronic granulomatous diseases with predominant pulmonary involvement. Simultaneous occurrence of sarcoidosis and tuberculosis is a rarely encountered clinical entity. We present two Sri Lankan males with coexisting bacteriologically confirmed tuberculosis and histologically diagnosed sarcoidosis. Careful and timely commencement of anti-tuberculous treatment followed by steroids treatment for sarcoidosis lead to improvement of both conditions. These cases are reported to describe the dilemmas in diagnosis and management of this underrecognized clinical entity.

Keywords: Tuberculosis; Sarcoidosis; Mediastinal lymphadenopathy

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INTRODUCTION

Sarcoidosis is a disease of unexplained etiology, histologically characterized by the presence of non-caseating granulomas [1]. Tuberculosis is an infection caused by Mycobacterium tuberculosis characterized by caseating granulomas. Tuberculosis is treated with standard anti-tuberculosis antibiotic regime whereas sarcoidosis is primarily treated with corticosteroids. Therefore, accurate differentiation of these two conditions is paramount important. They have many overlapping clinical manifestations [2]. Coexistence of the two diseases is rare in medical literature [3,4].

CASE 1

A 57- year old previously well man presented with unintentional weight loss, loss of appetite and shortness of breath for 6 months. He also had dry cough for the same duration. His BMI was 24 kg/m^2. He was not pale. There were no palpable lymph nodes or skin rashes on general examination. Lungs were clinically normal and his peripheral oxygen saturation was 99% on room air. There was no hepatosplenomegaly. His ESR was 88mm/1st hour. Complete blood count, C-reactive protein level, renal and liver biochemistry were normal. Further evaluation with chest radiograph revealed bilateral hilar prominence. Contrast enhanced computed tomography of the chest revealed multiple soft tissue nodules scattered in both lung fields with enlarged mediastinal and bilateral hilar lymph nodes. CECT of the abdomen and pelvis were normal. Serum lactate dehydrogenase level and blood picture were normal. Tuberculin skin test induration was 8 mm. Serum calcium level was normal and angiotensin converting enzyme level was elevated (156 U/L). Sputum examination for acid fast bacilli was negative in three early morning samples. The patient underwent video assisted thoracoscopic exploration for mediastinal lymph node and left upper lobe lung biopsy. Microscopic examination of mediastinal lymph node revealed caseating granuloma formation. Lymph node samples were positive for mycobacterium tuberculosis gene X-pert. Histological examination of left upper lobe lung biopsy revealed non caseating granuloma formation with interstitial fibrosis. Lung biopsy samples were negative for mycobacterium tuberculosis gene X-pert.

CASE 2

A 58- year old man presented with progressive exertional dyspnea (mMRC3) for 1 year. There was an associated dry cough and unintentional weight loss. There was no history of fever, joint pain or night sweats. Three years back he had been investigated for right sided proptosis. MRI performed at that time revealed a well-defined inflammatory lesion measuring 19×36 mm occupying retro-orbital area abutting the posterior wall of the right globe. Tissue biopsy revealed fibrofatty tissues only. Additionally, he had a documented history of right ulnar nerve palsy, apparently resolved spontaneously. Unfortunately, he was defaulted from ophthalmology and neurology clinics after initial visits.

His BMI was 22 kg/m^2. Peripheral oxygen saturation was 98% on room air. He was not pale and there was no finger clubbing. Respiratory system examination was significant for bi basal end inspiratory fine crepitations. Cardiovascular system and abdomen were clinically unremarkable. His complete blood count, C-reactive protein level, renal and liver biochemistry were normal. ESR was 40mm/1st hour. Two-dimensional echocardiogram was normal with preserved bi ventricular function. There was no evidence of pulmonary hypertension. Plain chest radiograph was significant for bilateral hilar shadows and reticular nodular shadows in bilateral lung fields. HRCT chest revealed numerous ground glass density nodules with some conglomeration Spirometry was suggestive of a restrictive pattern of lung impairment. Six-minute walking test showed significant desaturation from 98% to 85%. Angiotensin converting enzyme levels was 95U/L (8-52). Tuberculin skin test induration was 9mm. Patient underwent bronchoscopy and transbronchial biopsy. Histological examination of the bronchial biopsy specimen revealed non caseating granuloma formation suggestive of sarcoidosis. Broncho alveolar lavage was positive for mycobacterium tuberculosis gene X-pert.

DISCUSSION

Aforementioned patients had confirmatory histological and microbiological evidence of two coexisting granulomatous diseases; tuberculosis and sarcoidosis. Coexistence of these two conditions in the same patient is a highly challenging clinical scenario in both diagnostic and management point of view. Starting steroids for sarcoidosis for a patient with a serious underlying infection like tuberculosis will have life threatening complications. Therefore, when there is a clinical suspicion of coexisting tuberculosis and sarcoidosis, it is safe to postpone the commencement of corticosteroid therapy till we get the microbiological confirmation of mycobacterium tuberculosis. Both of our patients were initially commenced on standard anti-tuberculosis treatment and steroid treatment for sarcoidosis was started after two weeks. It was decided to continue both steroid and anti-TB therapy and both of them recovered uneventfully.

We have presented two cases of bacteriologically confirmed pulmonary tuberculosis and pulmonary sarcoidosis diagnosed simultaneously. Is these a coincidence? Or are these diseases etiologically associated? These questions are yet to be answered.

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