A Patient With Chronic Cough And Fever: A Case of Extensive Sarcoidosis; A Notorious Mimicker of Tuberculosis

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ABSTRACT

Background

Sarcoidosis is a chronic systemic granulomatous disease that closely mimics tuberculosis (TB) in clinical, radiological, and histopathological features. In TB-endemic regions like Nepal, this overlap frequently leads to misdiagnosis and delayed appropriate treatment.

Case Presentation

A 53-year-old male construction worker from India presented with 8–9 months of intermittent cough, low-grade fever, mild chest pain, and exertional dyspnea (MMRC Grade II). He had a history of TB contact and had already completed 8 months of anti-tubercular therapy (ATT) with incomplete and transient improvement. Investigations showed elevated ESR, negative sputum Gene Xpert, negative Mantoux test, and restrictive pattern on spirometry. CT chest revealed bilateral consolidation, fibrotic bands, fine nodules, and ground glass opacities. Bronchoscopy with BAL showed airway inflammation and an elevated CD4:CD8 ratio. CT-guided lung biopsy confirmed non-caseating granulomas with extensive fibrosis, establishing a diagnosis of pulmonary sarcoidosis (Scadding Stage IV). The patient responded well to pulse methylprednisolone followed by oral prednisolone, with significant symptomatic improvement at two-week follow-up.

Conclusions

This case underscores the need to maintain sarcoidosis as an active differential diagnosis in TB-endemic settings, particularly when clinical response to ATT is suboptimal. Histopathological confirmation remains essential to avoid prolonged, inappropriate treatment.

Keywords: Sarcoidosis; tuberculosis; Myobacterium tuberculosis; Gene Xpert; granuloma; Human Leukocyte Antigen.

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INTRODUCTION

Sarcoidosis is a chronic systemic inflammatory disorder of unknown etiology, characterized by the accumulation of T lymphocytes, mononuclear phagocytes and the formation of noncaseating granulomata in affected tissues.[1] It has a variable clinical presentation and disease course that depends on the organs involved. Virtually any organ system can be involved extending from eyes, skin, joints, the reticuloendothelial system, musculoskeletal system, heart, kidney, and central nervous system. The granuloma in sarcoidosis is characterized by a core of monocyte-derived epithelioid histiocytes and multinucleate giant cells with interspersed CD4+ T lymphocytes.[2] Most evidence suggests that the development of the disease is similar to other granulomatous diseases of known cause. That is, some antigen(s) enter the host and are phagocytosed by antigen-presenting cells (APCs), predominantly macrophages or dendritic cells. The APCs process the antigen and subsequently present it via human leukocyte antigen (HLA) class II molecules to a restricted set of T-cell receptors on T lymphocytes, primarily of the CD4+ class. The immune reaction begets polarization of the T lymphocytes to a Th1 phenotype, followed by cellular recruitment, proliferation, and differentiation leading to formation of the sarcoid granuloma[3,4] Genetic Study has determined the role of HLA the pathogenies of the disease with certain HLA types like HLA DRB1*1101, HLA-DqB1, HLA-DR B1*03 showing increased susceptibility for the disease with HLA-DQB1*0201 associating with a good prognosis in studies done Dutch and British patients, although it is difficult to ascertain which is the responsible allele.[5–8] These studies have highlighted the issues of population stratification and the need for careful clinical phenotyping in association studies of sarcoidosis. Despite the uncertainty about a potential mechanistic relationship, it is well established that tuberculosis and sarcoidosis share a number of similar clinical, radiographic, and histopathologic features in developing countries and is often misdiagnosed. We present a case of a male with cough and fever, diagnosed as Sarcoidosis at our center who was initially treated in lines of pulmonary tuberculosis with sub optimal improvement

Case Report

A 53 year old man from India presented to our outpatient department with complains of cough on and off for 8-9 months associated with low grade fever, intermittent in character for the same duration, no chills, rigor, no specific aggravating or relieving factors .He also complained of mild chest pain, nonspecific, generalized, not associated with respiration, no specific aggravating or relieving factors. Upon further inquiry he also reported shortness of breath, MMRC grade II for the same duration. There was no history of smoking, tobacco chewing or alcohol ingestion. He worked as a construction worker with more focus on iron welding work as a blacksmith. There was positive history of contact with tuberculosis in the family as well as two of the neighbors during the last 1 year.

However he reported no episodes of hemoptysis, weight loss or night sweats. With the above mentioned complains he had visited a local clinic in India where he was treated in line of tuberculosis. The patient received Anti tubercular therapy for 8 months. He noted that his symptoms initially resolve for a few months but again reappeared, thus he came to our center.

At our center, his investigations were within normal limits except for a raised ESR at 63.

Chest Xray showed patchy areas of heterogenous opacities and fibrosis, distributed throughout the both lung fields. Sputum analysis was done which was normal and culture showed no growth of any bacteria. Sputum gene Xpert for PTB was sent which was also negative. A Mantoux reading was also negative.

Spirometry performed on the patient showed significant restrictive deficit. Thus with a possibility of Interstitial lung disease , a chest CT was done which showed areas of extensive consolidation with surrounding fibrotic bands and randomly distributed fine nodules and areas of ground glass opacity in bilateral lower lungs. (figure 3)

In view of the CT findings, a serum ACE levels

was sent which was normal and serum calcium and vit D also came back into levels. A bronchoscopy with BAL was also performed on the patient which revealed inflamed mucosa thought the airways. (figure 2). The BAL fluid was collected and was sent for CD4:CD8 ratio which revealed increased suggestive for sarcoidosis.

Ophthalmological consultation was done to rule out any evidences of ocular sarcoidosis so as dermatological consultation was done to look for any cutaneous features of sarcoid. A screening ECG and ECHo also came back normal.

In view of the above findings, a lung biopsy was planned in discussion with Interventional Radiology team. Under CT guidance, a biopsy was obtained from the left and right lower zones and sent for Histopathological examination. The event was complicated by an iatrogenic pneumothorax which resolved under conservative management with high flow oxygen.

The patient was treated with IV pulse of methylprednisolone 500mg for 3 days then tapered to prednisolone 40mg. His Symptoms significantly improved and fever also subsided. He was discharged on Prednisolone 40mg along with LABA-ICS (Formoterol- Budesonide) inhaler and other supportive medicines.

On his follow up after 2 week, he reported significant improvement in his symptoms with resolution of cough and significant increase in exercise tolerance. There was no fever noted during the treatment period as well as after discharge. His biopsy report was obtained which read areas of extensive fibrosis surrounding few non caseating granulomas; suggestive of Sarcoidosis with extensive lung fibrosis. The patient is kept on tapering doses of steroid and is under regular follow up.

DISCUSSION

Also known as Besnier–Boeck–Schumann disease, The annual incidence of sarcoidosis varies between 1 and 15 per 100,000 depending on the region. The rates are lowest in Eastern Asian countries (0.5–1 per 100,000), higher in North America and Australia and highest in Northern European (Scandinavian) countries9–12

In context of Nepal, the exact data on the prevalence remains unknown largely due to underreporting, misdiagnosis and unapproachable health care to the vast majority of the rural population but studies done in past have shown that it is prevalent in a large number of populations.13 The presentation of sarcoidosis varies depending on what organ system is involved. The lungs are the most commonly affected organ in the disease process, with more than 90% of patients exhibiting pulmonary symptoms. The liver, spleen, bones, skin, heart, and nervous system may also be involved.14 Clinical presentations most commonly include unexplained or persistent cough, dyspnea, chest pain or fever. A handful of patients may present with acute presentation of multiple erythema nodosum and arthritis addition to the pulmonary findings what is called as Lofgren syndrome).15 Extrapulmonary findings depends on the organ involved with heart failure, conduction abnormalities, ocular manifestation as uveitis and skin findings being more common.16,17 Diagnosis of sarcoidosis relies heavily on the basis of suspicion in part of the treating physician in a tuberculosis endemic country like ours. Compatible clinical findings with radiological and Histopathological evidences account for the cornerstone in the diagnosis with radiological imaging the primary clue in guiding the treating physician.18 Scadding described a typical pattern on the chest Xray which we still rely on as the first and most easily accessible imaging modality in the cases.

Table 1. Scadding scale for staging of pulmonary sarcoidosis

Stage	Chest radiograph Findings
I	Bilateral Hilar lymphadenopathy
II	Pulmonary Infiltrates and Bilateral hilar lymphadenopathy
III	Pulmonary Infiltrates
IV	Pulmonary fibrosis

In retrospective analysis our patients chest Xray lied on stage on stage IV disease. CT findings of pulmonary sarcoidosis include peri lymphatic micronodules, predominantly in the mid and

upper lung zones, symmetrical intrathoracic lymphadenopathy, where advanced cases are characterized by fibrosis, traction bronchiectasis, honeycombing, and fibrocystic changes. The relation between tuberculosis and sarcoidosis is an area of emerging research since the pathogenesis of sarcoidosis has been understood. In 1960, Scadding reported a high proportion of sarcoidosis cases that developed overt tuberculosis in the chronic stage of the disease, and the development of sarcoidosis in a minority of tuberculosis cases. In his series of 230 cases, he added that tuberculous bacilli were found at some stage in 29 or 13% of patients.[19] Saboor et al. found mycobacterium tuberculosis (MTB) DNA in half of sarcoidosis lung samples and identified non-MTB DNA in an additional 20%.[20] Mootha et al.6reported a high prevalence of mycobacterial DNA in sarcoidosis samples compared to control in a population in India.[21,22] In a meta-analysis spanning over two decades (1980–2006) including 31 studies, 874 patients with sarcoidosis employing molecular methods, predominantly polymerase chain reaction (PCR), to detect mycobacterial DNA in biological samples (blood, BAL, and tissue biopsies) from patients with sarcoidosis; a positive signal rate of 26.4 (23.6–29.5% was found). The pooled odds ratio for detecting mycobacteria in sarcoidosis samples versus controls was 9.67 model) and 19.49[22] However despite the possible etiologies, efforts to culture or isolate the mycobacterium from the sarcoid lesions have largely been unsuccessful.[23] However in a retrospective longitudinal cohort study done by hung et al, they found that patients with tuberculosis exhibited an 8.08-fold higher risk of developing sarcoidosis than the non-tuberculosis patients over 5 years of observation drawing the possibility of a dysregulated T cell response to the various mycobacterium antigens and enzymes resulting in initiation of a systemic inflammatory response resulting in the formation of the granuloma and the systemic manifestations.[24]

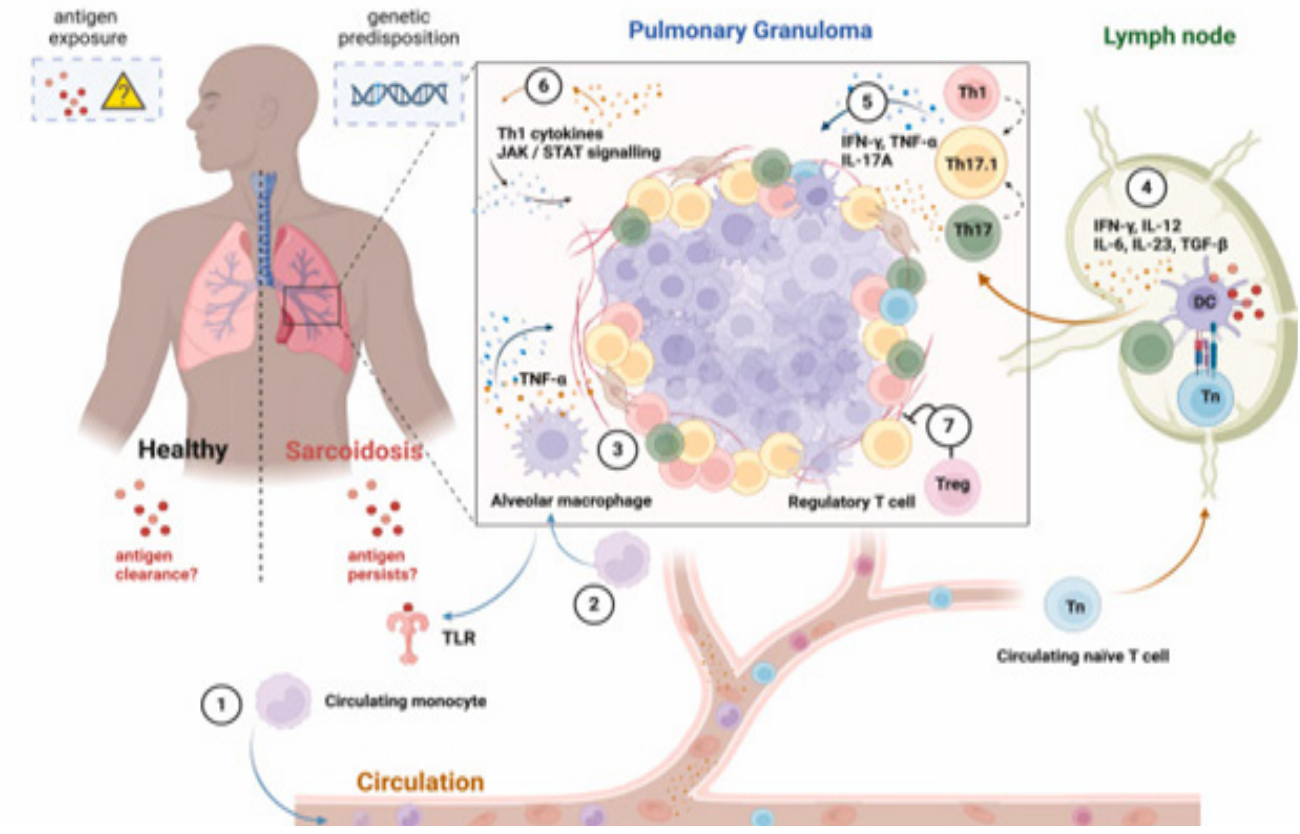

Figure 1. Immunopathogenesis of Sarcoidosis showing the classical antigen- host response in formation of the pulmonary granuloma

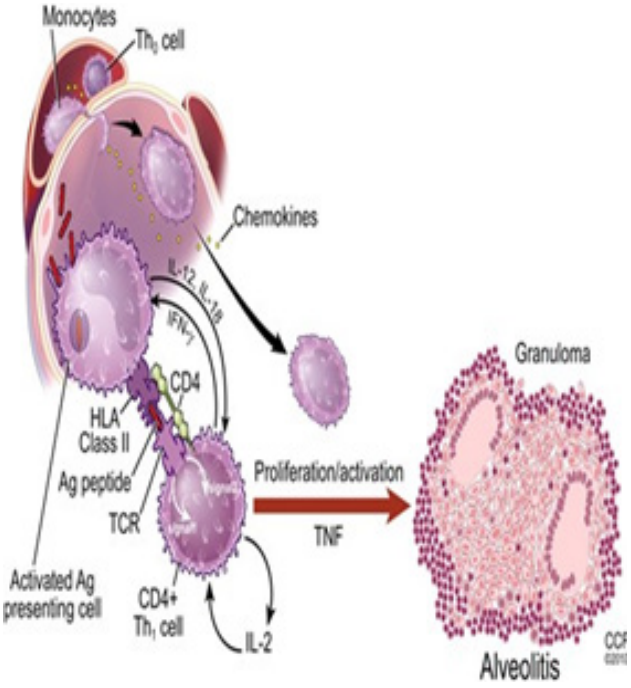

Figure 2. Inflammatory response of Sarcoidosis airway showing formation of granuloma

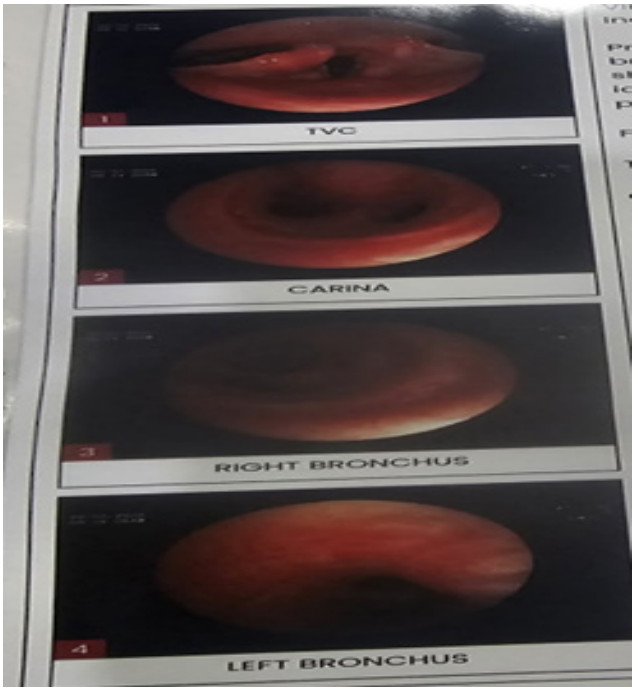

Figure 3. Bronchoscopy showing airway showing formation of granuloma inflammation

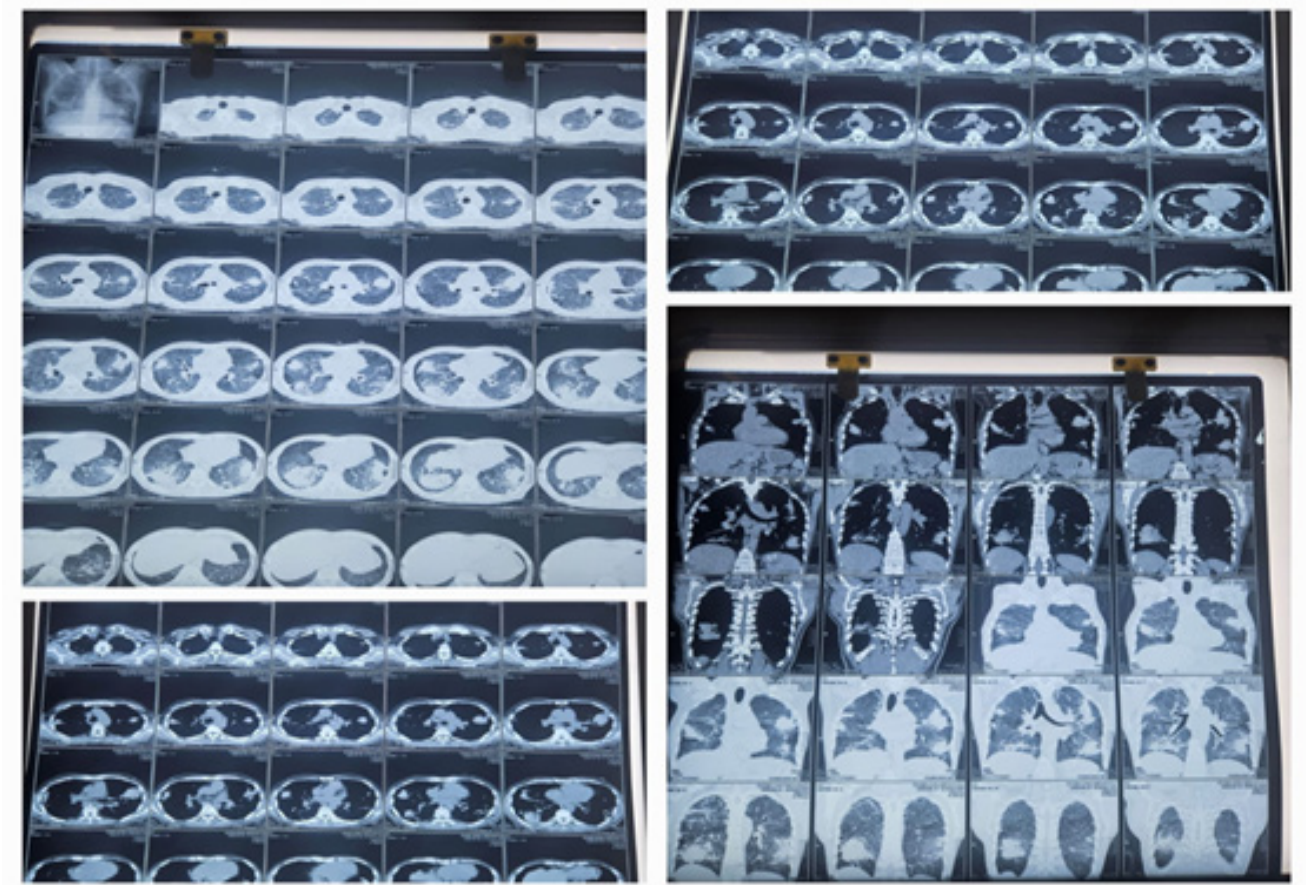

Figure 4. CT Chest of the patient showing areas of extensive consolidation with surrounding fibrotic bands and randomly distributed fin nodules and areas of ground glass opacity in bilateral lower lungs

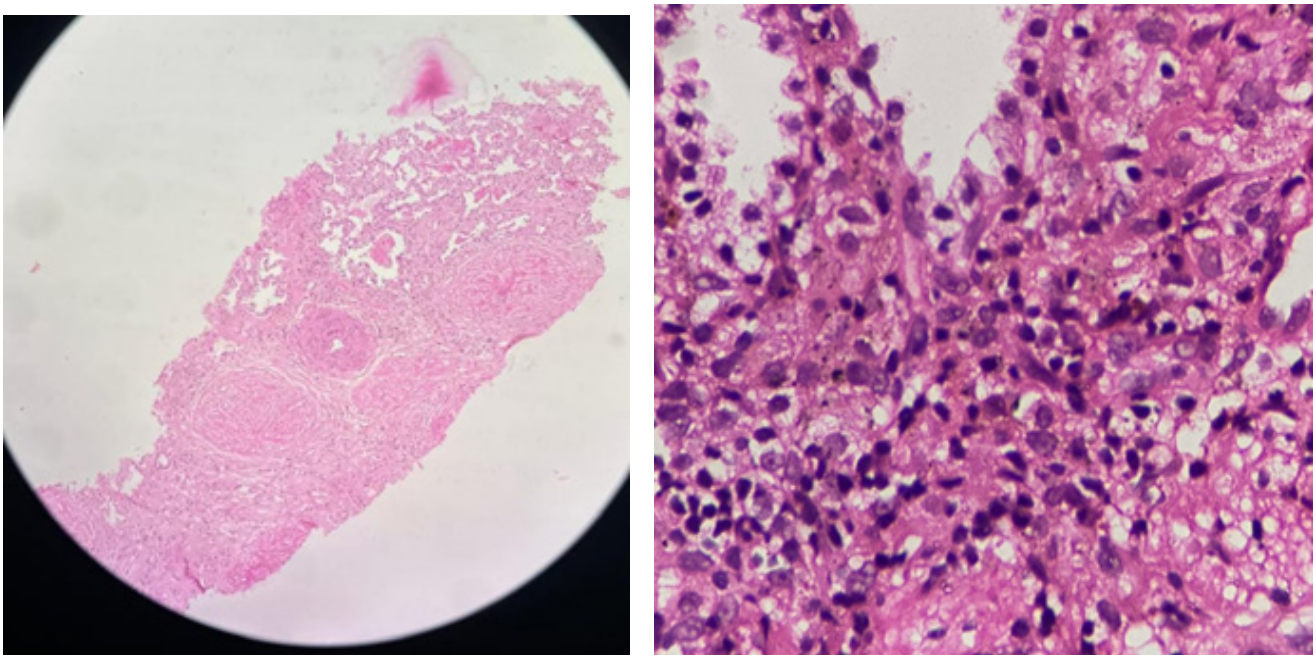

Figure 5. HPE slides showing extensive fibrosis with ill formed granulomas with epitheliod cells (Focused) favoring Sarcoidosis as possible etiology

CONCLUSIONS

In a tuberculosis endemic country like ours the possibility of Sarcoidosis should always be born as a differential in patients presenting with a chronic history of cough and fever. There have been numerous case reports where the two diseases coexist in the same patient thus a high index of suspicion is necessary on the part of the clinician.[25–27]

Conflicts of Interest: The authors declare that they have no conflicts of interest related to this study.

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