

Disseminated Granulomatous Disease: TB or sarcoidosis – A Diagnostic and Therapeutic Challenge

By Anamitra Hait

Case Study:

Disseminated Granulomatous Disease: TB or sarcoidosis – A Diagnostic and Therapeutic Challenge

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Abstract

The overlapping presentation of tuberculosis (TB) and sarcoidosis poses significant diagnostic and therapeutic challenges, particularly in endemic regions. This case study details the diagnostic journey and management of a 52-year-old female patient with disseminated granulomatous disease, highlighting the complexities of differentiating TB from sarcoidosis and addressing overlapping features. A multidisciplinary approach and judicious use of investigations and treatment strategies led to a favourable clinical outcome.

keywords : Tuberculosis; Sarcoidosis; Granulomatous inflammation; Overlapping diseases; Anti-tubercular therapy (ATT); Non-caseating granulomas; Histopathology; Bronchoalveolar lavage (BAL); Whole-body PET-CT; Isoniazid-induced psychosis; Multisystem involvement; Diagnostic challenges; Serum ACE levels; Endemic regions; Aspergillosis.

5 Introduction

Tuberculosis (TB) and sarcoidosis are granulomatous diseases that often create diagnostic dilemmas, particularly in regions like India where TB is endemic. TB is caused by infection with *Mycobacterium tuberculosis*, a pathogen that triggers granuloma formation as an immune containment strategy. On the other hand, sarcoidosis is a multisystem inflammatory disease of unknown etiology, characterized by the formation of non-caseating granulomas. Despite being fundamentally different in origin, these two diseases share overlapping clinical, radiological, and histopathological features, making their differentiation highly challenging in TB-endemic settings.

15 India accounts for the highest burden of TB globally. The World Health Organization (WHO) reported in 2022 that India contributed to nearly 30% of global TB cases, with millions of new cases annually (WHO, 2023). Sarcoidosis, though less prevalent, has seen an increase in reported cases in India, particularly with improved diagnostic facilities. Sarcoidosis is thought to result from an exaggerated immune response to environmental, infectious, or occupational antigens, including potential triggers such as *Mycobacterium tuberculosis* antigens (Rastogi et al., 2020). Both diseases primarily involve the lungs, but systemic manifestations involving lymph nodes, skin, eyes, and other organs are also common, further complicating the diagnostic process [1,2].

Comparison of Key Features: Tuberculosis vs. Sarcoidosis

Feature	Tuberculosis (TB)	Sarcoidosis
Causative Agent	<i>Mycobacterium tuberculosis</i>	Unknown; potential triggers include environmental and infectious antigens

Global Burden	High in TB-endemic countries, including India	Less common but increasingly diagnosed globally and in India
Histological Findings	Caseating granulomas with central necrosis	Non-caseating granulomas; multinucleated Langhans and foreign body giant cells
Primary Immune Response	Th1-mediated immune response	Th1/Th17-mediated immune dysregulation
Clinical Symptoms	Fever, night sweats, weight loss, chronic cough	Persistent fever, fatigue, shortness of breath, skin lesions
Radiological Features	Cavitary lesions, pulmonary nodules, pleural effusion	Bilateral hilar lymphadenopathy, interstitial infiltrates, diffuse nodules
Serological Markers	None specific	Elevated serum ACE levels, hypercalcemia
Treatment	Anti-tubercular therapy (ATT)	Corticosteroids and immunosuppressants

Epidemiology of Tuberculosis in India

India remains a high-burden TB country, with a substantial share of global cases. The burden of the disease is aggravated by socio-economic disparities, delayed diagnosis, and multi-drug-resistant TB (MDR-TB). The table below provides insights into the TB burden in recent years:

Year	Estimated TB Cases (Millions)	Global Share (%)	Deaths (Thousands)	MDR-TB Cases (Thousands)
2020	2.6	26%	410	124
2021	2.9	28%	450	130
2022	3.0	29%	480	140

Source: WHO Global Tuberculosis Report, 2023

This high burden of TB poses a unique challenge for diagnosing sarcoidosis in India, as TB is often the first differential diagnosis in patients presenting with granulomatous disease.

Shared and Distinct Diagnostic Features

Feature	Tuberculosis (TB)	Sarcoidosis
Onset	Gradual, often following exposure to <i>M. tuberculosis</i>	Insidious, with no identifiable infectious agent
Lymph Node Involvement	Caseating necrosis in cervical and mediastinal nodes	Non-caseating necrosis in hilar and mediastinal nodes
Pulmonary Involvement	Cavities, nodules, infiltrates, and pleural effusion	Diffuse pulmonary infiltrates and nodules
Cutaneous Manifestations	Lupus vulgaris, scrofuloderma	Erythema nodosum, plaques, and papules

Laboratory Markers	No specific markers; elevated ESR	Elevated ACE, hypercalcemia, and normal ESR
Microbiological Findings	Positive sputum AFB or culture, CBNAAT	No identifiable microbial agent
Response to Treatment	Significant improvement with ATT	Improvement with corticosteroids

Pathophysiology and Immune Response

The immune response in TB and sarcoidosis shares similarities, particularly in the formation of granulomas. However, their underlying mechanisms differ. In TB, the infection triggers a dominant Th1-mediated immune response, leading to the activation of macrophages and the formation of granulomas with central necrosis. This response is aimed at containing the *M. tuberculosis* bacilli and preventing their dissemination [3,4]. Conversely, sarcoidosis involves an exaggerated Th1/Th17 response, leading to the formation of non-caseating granulomas. This immune dysregulation is not directed at any specific pathogen, making the etiology of sarcoidosis more enigmatic (Bhalla et al., 2017).

Diagnostic Challenges in TB-Endemic Regions

Diagnosing sarcoidosis in India is particularly challenging due to the high prevalence of TB, which mimics many features of sarcoidosis. Radiological findings such as bilateral hilar lymphadenopathy and pulmonary infiltrates are common to both conditions. Furthermore, non-caseating granulomas seen in sarcoidosis can also be present in TB, especially in its atypical or early presentations [5,6]. The use of corticosteroids, the mainstay of sarcoidosis treatment, can exacerbate latent TB infections, leading to reactivation. This necessitates careful exclusion of TB through microbiological tests such as CBNAAT, sputum AFB, and culture before initiating sarcoidosis therapy (Mediouni et al., 2023).

Key Diagnostic Tools

Diagnostic Modality	Role in TB	Role in Sarcoidosis
Chest X-Ray	Pulmonary infiltrates, cavitation	Bilateral hilar lymphadenopathy
High-Resolution CT (HRCT)	Detects nodules, pleural effusion	Identifies interstitial infiltrates, nodules
Bronchoalveolar Lavage (BAL)	Elevated neutrophils; positive for <i>M. tuberculosis</i>	Elevated CD4/CD8 ratio in sarcoidosis
Histopathology	Caseating granulomas	Non-caseating granulomas
Serological Tests	Non-specific	Elevated serum ACE and hypercalcemia
CBNAAT/AFB Sputum	Confirms TB diagnosis	Negative; excludes TB

This case study illustrates the diagnostic challenges encountered in managing a patient with disseminated granulomatous disease and the therapeutic strategies employed to address overlapping TB and sarcoidosis.

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Case Presentation :

A 52-year-old woman presented to the clinic with a six-week history of progressive shortness of breath, occasional cough, and low-grade fever. She also reported appetite loss but denied significant weight loss. Her medical history included diabetes mellitus and hypothyroidism, both managed with oral medications.

Clinical Examination:

- **General Findings:** Pallor.
- **Lymphadenopathy:** Cervical lymph nodes (anterior triangle) and inguinal lymph nodes were enlarged.
- **Respiratory System:** Bibasilar crepitations were noted.
- **Other Systems:** Some skin lesions in nape of neck and both legs

Diagnostic Workup : The patient underwent a series of investigations to identify the underlying cause of her symptoms.

Table 1: Summary of Investigations

Investigation Type	Findings	Remarks
Blood Investigations		
Complete Blood Count (CBC)	Hb 9.0, TLC Normal, No Eosinophilia	Suggestive of chronic disease.
Liver Function Test	Normal.	Excludes hepatic involvement.
Kidney Function Test	Normal.	Excludes renal pathology.
Electrolytes	Normal.	No metabolic derangement.
Serum ACE	99 U/L (normal: 12-68).	Elevated; indicative of sarcoidosis.
Microbiological Workup		
Sputum for AFB/CBNAAT	Negative.	Tuberculosis not confirmed.
Cultures (Blood)	No growth.	Excludes bacterial sepsis.
RT-PCR for COVID-19	Negative.	Rules out COVID-19.
Malarial Parasite	Not detected.	Excludes malaria.
Dengue NS1 Ag, IgM, IgG	Negative.	Excludes dengue.
Widal Test	Negative.	No enteric fever.
Aspergillus Galactomannan (BAL)	5.04 (negative < 0.5).	Suggestive of possible aspergillosis.
CBNAAT for TB (BAL)	Undetected.	Pulmonary TB ruled out.
Histopathology and Imaging		
Inguinal Lymph Node Biopsy	Epitheloid granulomatous lymphadenitis. But no central necrosis or AFB seen. Negative for fungal stain 16.	Suggestive of granulomatous disease. But no definitive etiology found.
Skin Biopsy	Epitheloid granuloma, Langhan's giant cell, foreign body giant cell.	Consistent with sarcoidosis.
Lung Biopsy	Non-caseating granuloma with lymphocytic infiltration.	Suggestive of sarcoidosis. Also ruled out lung carcinoma
Bone Marrow Biopsy	Non-contributory. Negative for malignancy, AFB, fungal stain, hemoparasites.	No significant pathology.
Whole-Body PET-CT	Disseminated granulomatous disease involving lungs, skin, and lymphoreticular system. But SUV was not high enough,	Suggestive of multi-system involvement.

	suggestive of non malignant pathology. There was evidence of osteolytic lesion in cranium and femur but SUV value was low.	
CT Scan	Micro- and macro-nodules in bilateral lungs, absence of cavitation.	Consistent with TB or sarcoidosis.

1. X-ray of chest :

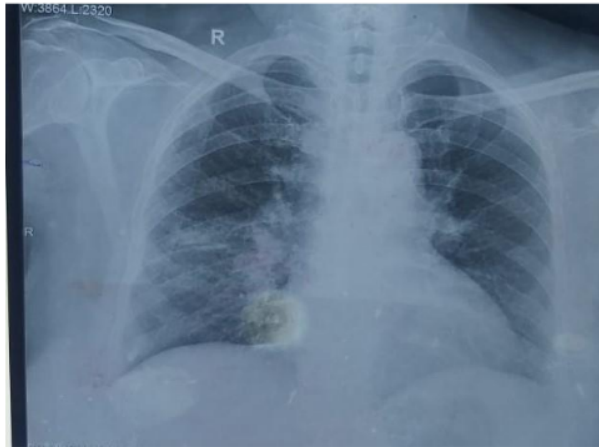


Figure 1: Initial Chest X-Ray Findings

The chest X-ray demonstrates bilateral lung infiltrates and prominent bilateral hilar lymphadenopathy, a critical finding supporting the suspicion of disseminated granulomatous disease. The infiltrates are indicative of pulmonary involvement, commonly seen in tuberculosis and sarcoidosis, whereas hilar lymphadenopathy is more common in sarcoidosis.

2. **Chest CT Scan Findings** : The computed tomography (CT) scan of the chest reveals multiple bilateral pulmonary nodules with areas of micro- and macro-nodularity and tree in bud appearance with hilar lymphadenopathy. These findings are indicative of diffuse pulmonary involvement, commonly associated with granulomatous diseases like tuberculosis and sarcoidosis.

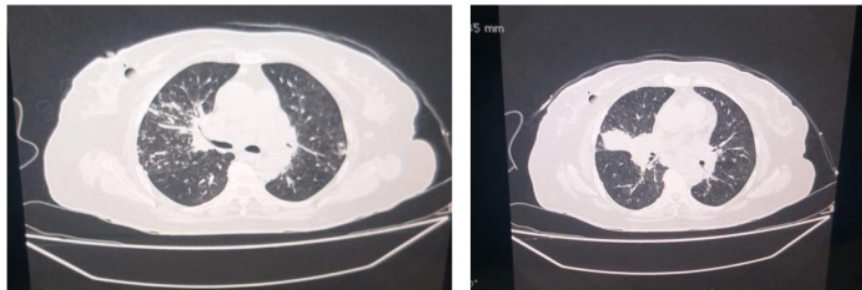


Figure 2: Chest CT Scan Findings Pre- Treatment

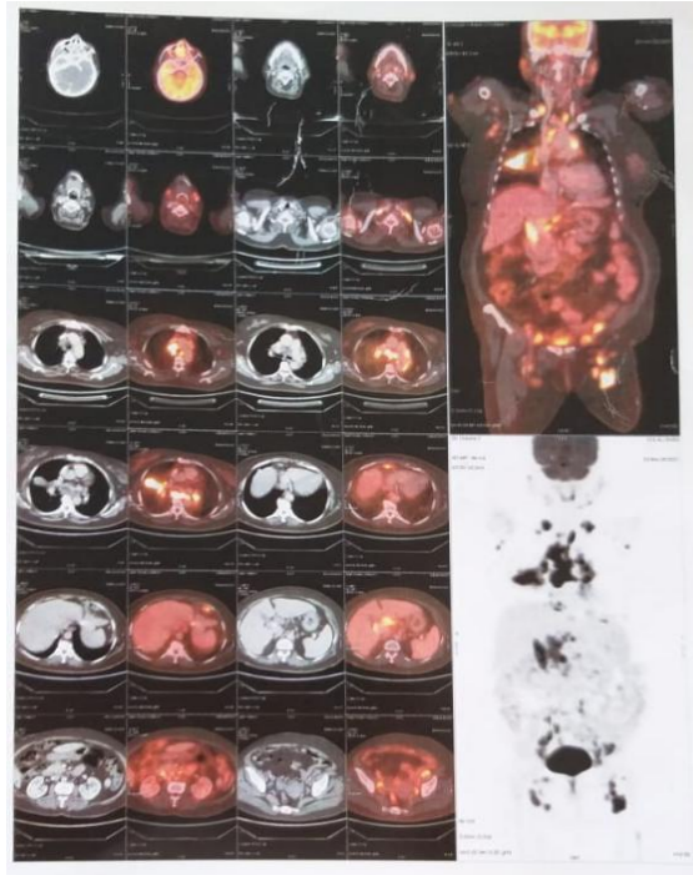
3. Skin Biopsy Findings:



Fig no 3 : Skin lesions in back of the patient

The image depicts skin lesions on the patient's back, consistent with disseminated granulomatous involvement. These lesions were biopsied and revealed epithelioid granulomas with Langhans giant cells, further supporting the diagnosis of granulomatous disease with features of sarcoidosis. There was no central necrosis or AFB and negative for Fungal stain and malignancy also.

4. **PET-CT Scan:** It showed disseminated granulomatous disease affecting multiple organ systems, including the lungs and lymph nodes.



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Fig 4 : PET CT SCAN of the patient

The PET-CT scan reveals hypermetabolic lesions in multiple organs, including the lungs, lymph nodes, nasopharyngeal mucosa, ethmoid sinus, and bone marrow. There was lytic lesion in cranium, nasopharynx and femur left side. But SUV value was not high enough ruling out malignancy. These findings are indicative of disseminated granulomatous disease, strongly suggestive of sarcoidosis or tuberculosis with systemic involvement. But osteolytic lesions further complicated the diagnosis. It was necessary to rule out plasmacytoma and Langerhans cell Histiocytosis. Xray also confirmed osteolytic lesions in cranium and left femur (Fig no 9 and 10). Biopsy from osteolytic bone lesion of left femur also showed predominantly granulomas with minimal sclerosis of bone tissue.

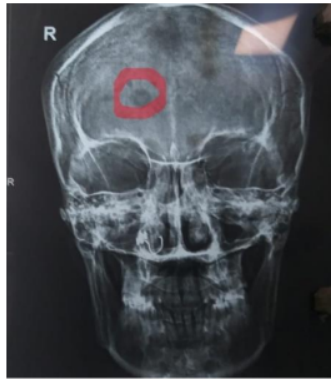


Fig no 9 : Skull X ray



Fig no 10 : X ray of left femur

5. Bone Marrow examination and MRI Pituitary was done which ruled out plasmacytoma and Langerhans cell Histiocytosis. Serum protein electrophoresis, Free Light Chain assay, IHC all were negative and was suggestive of granulomatous pathology.

Differentials we made :

1. **Disseminated fungal infection** due to involvement of lung, skin and lymph node probably histoplasmosis vs aspergillosis. But no bone marrow involvement or pancytopenia excluding histoplasmosis. Keeping increased beta galactomannan in BAL antifungal agents were started.
2. **Disseminated tuberculosis** but all CBNAAT and biopsy were negative for TB.
3. **Sarcoidosis** but serum ACE was not very high and BAL fluid lymphocytosis was not prominent.
4. **Langerhans cell histiocytosis** in view of lytic bone lesions with granuloma but no pituitary involvement and all biopsy were negative for staining and IHC
5. Other rare granulomatous lesions
6. **Carcinoma lung** due to multisystem involvement but no biopsy or histopathology showed any kind of malignancy. All cancer biomarkers were negative.

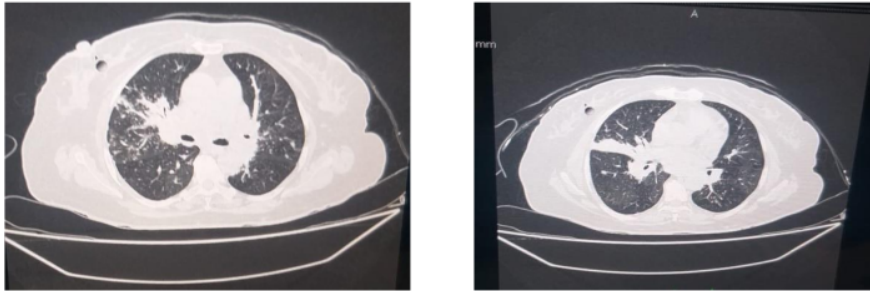
Treatment and Management

The patient presented with features of disseminated granulomatous disease, necessitating a multifaceted treatment approach. Given the diagnostic uncertainty, empirical therapies were initiated based on clinical suspicion and investigation results.

Initial Treatment

- **Antifungal Therapy:** Based on elevated Aspergillus galactomannan levels (BAL fluid: 5.04), antifungal treatment was initiated. However, the fever persisted, and the patient showed no significant improvement.
- **Symptomatic Treatment:** Supportive care included antipyretics and medications for appetite stimulation. Despite these measures, her clinical status remained unchanged.

Figure 5: Post-Treatment Images:



Description: Comparative radiological findings before and after antifungal and steroid therapy.

Definitive Diagnosis and Therapeutic Modifications

Following additional investigations, including biopsy findings and PET-CT results, the diagnosis of overlapping **tuberculosis with tubercular sarcoidosis** was established. This was based on:

- Favourable age group
- Symptoms like chronic cough and low grade fever
- Multiple palpable lymphadenopathy in different body parts
- **3** raised ESR
- **Histological evidence of non-caseating granulomas** in lung and **skin** biopsies.
- Elevated serum ACE levels indicative of sarcoidosis.
- Radiological findings consistent with disseminated granulomatous disease.
- Lack of microbial evidence supporting infectious etiology in BAL or sputum.
- Although osteolytic lesions are rare without osteomyelitis, there are reported case of cranial and long bone lytic lesions in sarcoidosis due to chronic granulomatous disease load.(thirunavukaruru et al.)

Tubercular Sarcoid is a relatively newly identified entity and as formulated by following diagnostic criteria (shah et al):

1 ge 25 to 45 years
No gender predisposition

History of previous tuberculosis with adequate ATT
History of close contact with TB or family h/o TB or association with Type 2 DM
Asymptomatic or with mild fever, anorexia or loss of weight
Chronic dry non productive cough, breathlessness on exertion
Involvement of multiple lymph nodes at multiple locations
Raised ESR
Increased ACE level
Tuberculin test : positive or negative
Sputum positive or negative for TB
Culture negative of TB
Biopsy showing non caseating or caseating granuloma
X ray showing bilateral hilar and right paratracheal lymphadenopathy, absence of cavitation
CT scan showing micro and macro nodules in peri-bronchovascular region, subpleural interstitium and interlobular septa.
No expected response to ATT medication
Dramatic response to steroid and resolution of symptoms

Treatment Plan:

1. Anti-Tubercular Therapy (ATT):

- A four-drug regimen was initiated, including isoniazid (INH), rifampicin, pyrazinamide, and ethambutol, to target the tuberculosis component.

2. Corticosteroids:

- Prednisolone (1mg/kg/day on tapering dose) was introduced to mitigate inflammation associated with sarcoidosis.
- **The dramatic improvement in symptoms (e.g., fever resolution, appetite restoration) supported the Tuberculous sarcoidosis diagnosis.**

Complications During Treatment

While the patient initially showed significant improvement, she developed **neuropsychiatric symptoms** characterized by:

- Nocturnal agitation.
- Incomprehensible screaming episodes.

Comprehensive evaluations, including liver function tests, kidney function tests, and CT brain scans, were normal. The symptoms were attributed to **isoniazid (INH)-induced organic psychosis**, a known but rare side effect of ATT.

Revised Management:

- INH was discontinued from the ATT regimen.
- Levofloxacin was added as an alternative drug to maintain the efficacy of the treatment protocol.

- Psychiatric symptoms resolved following this modification.

Response to Treatment The patient demonstrated remarkable clinical and radiological improvement following the revised regimen:

1. Clinical Outcomes:

- Resolution of fever and pulmonary symptoms.
- Regression of lymphadenopathy.
- Improved appetite and weight gain.

2. Radiological Outcomes:

- Follow-up CT scans and Chest X RAY showed a reduction in lung nodules and lymphadenopathy.
- PET-CT scans indicated decreased granulomatous activity.

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Figure 6 **Follow-Up Whole-Body PET-CT Scan**

The PET-CT scan conducted after 2 months of treatment showed a significant reduction in hypermetabolic activity across multiple organ systems, indicating a favourable response to the combined anti-tubercular therapy (ATT) and corticosteroid treatment. Persistent but reduced metabolic activity is evident in some regions, suggesting partial resolution of granulomatous inflammation and establishing continuation of steroid medications.

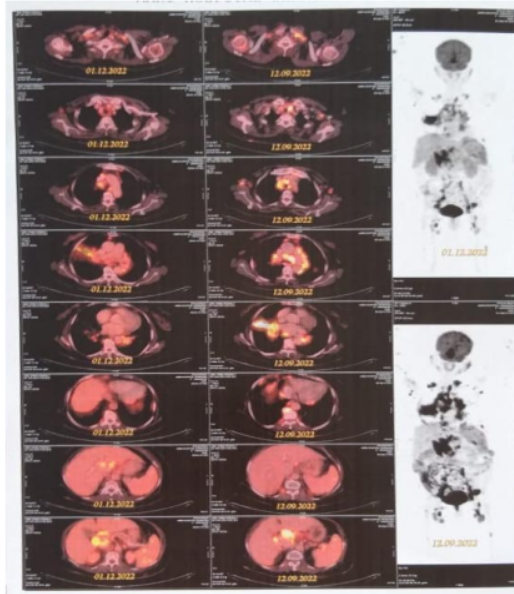


Figure 6 **Follow-Up Whole-Body PET-CT Scan**

Figure 7: Repeat Biopsy Histopathological Findings

This microscopic image from the repeat biopsy demonstrates epithelioid granulomas with Langhans giant cells and lymphocytic infiltration. These findings are consistent with granulomatous inflammation and support the diagnosis of overlapping tuberculosis with sarcoidosis.

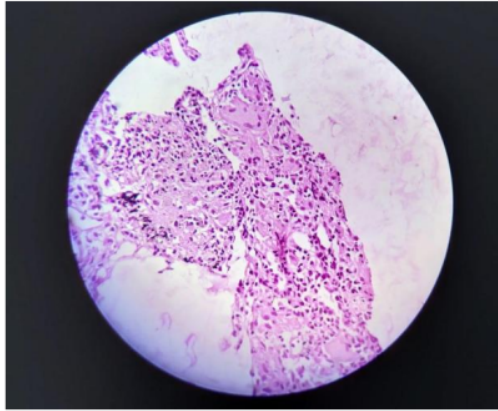
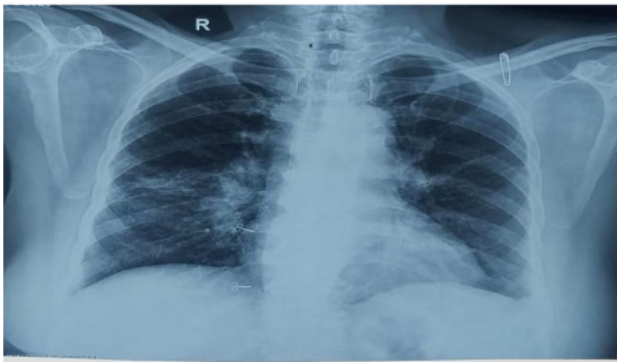


Fig no 8 : Repeat Chest Xray



Repeat Chest X ray showed obvious improvement of bilateral infiltration with hilar lymphadenopathy .

Discussion

This case highlights the challenges of diagnosing and managing overlapping tuberculosis (TB) and sarcoidosis, particularly in a TB-endemic country like India. The coexistence of these conditions is rare but has been increasingly recognized. Both diseases have overlapping clinical, radiological, and histopathological features, which make diagnosis and treatment complex.

Diagnostic Challenges and Relevance

Clinical Features

The patient presented with fever, lymphadenopathy, and lung involvement, all of which are seen in both TB and sarcoidosis. TB, caused by *Mycobacterium tuberculosis*, is usually

associated with caseating granulomas due to tissue necrosis. Sarcoidosis, on the other hand, is characterised by non-caseating granulomas caused by immune dysregulation. However, in TB-endemic regions like India, atypical TB can also present with non-caseating granulomas, making it difficult to differentiate between the two conditions (Shah et al., 2009). In this case, elevated serum ACE levels suggested sarcoidosis, but their lack of specificity in TB-endemic areas required careful consideration (Mediouni et al., 2023) [7].

Histopathology and Imaging

Histopathological findings in this patient, such as non-caseating granulomas in the lung and skin and lytic lesion biopsies, pointed towards sarcoidosis. However, granulomas without necrosis can also occur in TB, especially in early or atypical cases (Sharma & Mohan, 2021). Imaging findings like bilateral lung nodules and hypermetabolic lesions seen on CT and PET-CT scans were consistent with granulomatous diseases but were not definitive for distinguishing TB from sarcoidosis (Carbonelli et al., 2020). Shah et al. (2009) highlighted the importance of combining clinical, histopathological, and radiological findings to arrive at an accurate diagnosis [8,9].

Pathogenesis of Overlap

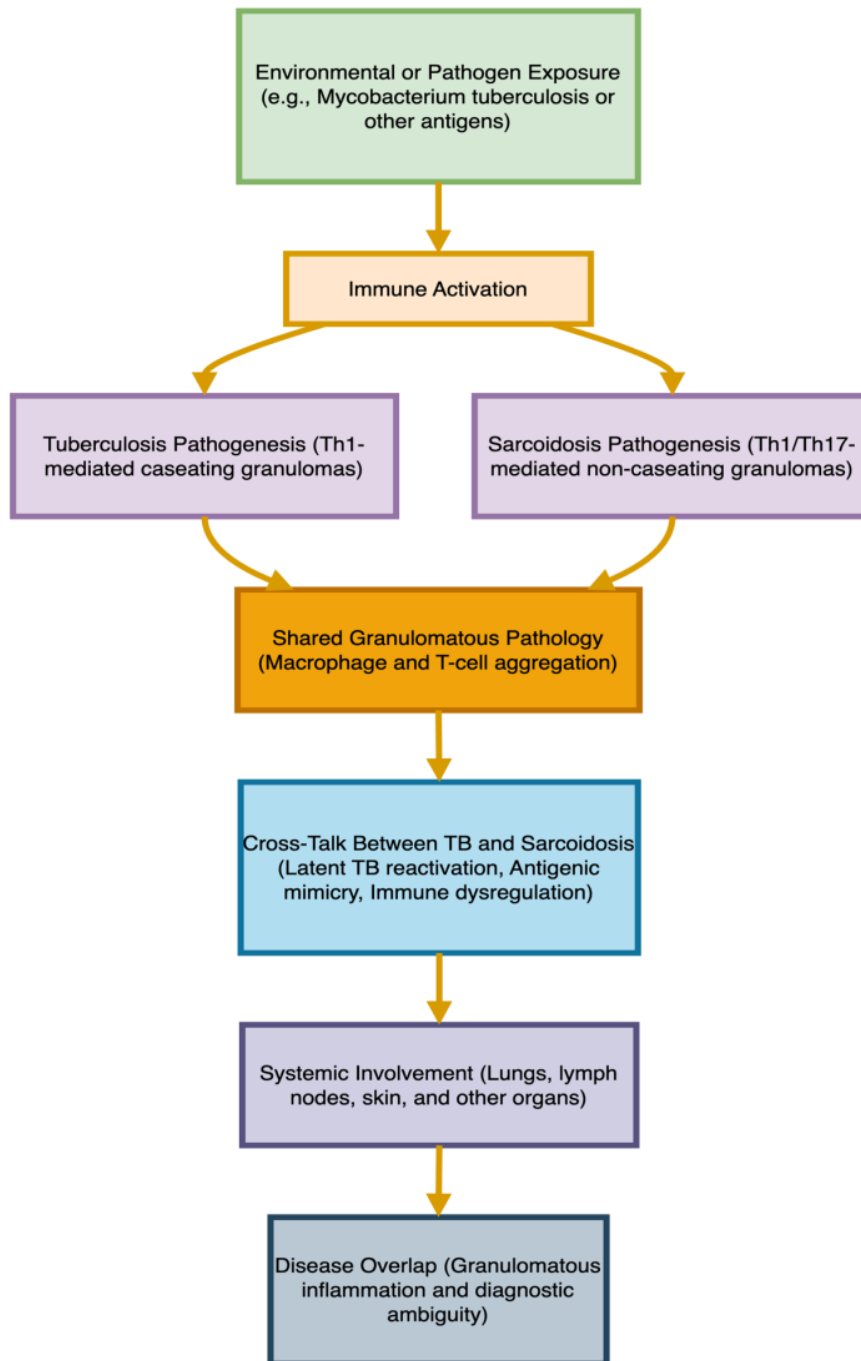
The co-occurrence of TB and sarcoidosis in this patient can be explained by their shared immune mechanisms. TB triggers a Th1-mediated immune response aimed at containing the infection through granuloma formation. Sarcoidosis involves an exaggerated Th1 and Th17 immune response to an unidentified antigen, also resulting in granuloma formation (Rastogi et al., 2020).

Trigger and Interplay

The overlap between TB and sarcoidosis may not be coincidental but rather a result of interconnected mechanisms. Shah et al. (2009) described "tuberculous sarcoidosis," where TB may act as a trigger for sarcoidosis in susceptible individuals. Mediouni et al. (2023) discussed how TB antigens might drive sarcoidosis-like immune responses, resulting in granulomatous inflammation in multiple organs. In this case, systemic involvement of the lungs, lymph nodes, and skin reflects these shared immune pathways [10].

Latent TB Reactivation

The use of corticosteroids to treat sarcoidosis can suppress the immune system and potentially reactivate latent TB. Mediouni et al. (2023) reported a case where sarcoidosis developed after nasal TB, highlighting how TB might create conditions conducive to sarcoidosis development. This interaction underscores the complexity of treating overlapping diseases.



Possible Mechanism of Pathogenesis(Figure 8)

The pathogenesis of overlapping tuberculosis (TB) and sarcoidosis begins with distinct initiation pathways, followed by immune activation and complex interplay between the two conditions. In TB, infection by *Mycobacterium tuberculosis* triggers the formation of

granulomas, which serve as an immune containment strategy to restrict the spread of the pathogen. In sarcoidosis, however, the process is initiated by unknown antigens, leading to an exaggerated immune response characterized by granuloma formation without the presence of a specific pathogen. The immune cascade differs between TB and sarcoidosis. In TB, the dominant Th1 response activates macrophages, resulting in the formation of granulomas with necrotic cores caused by the persistence of *M. tuberculosis*. In contrast, sarcoidosis involves an enhanced Th1/Th17 response, which drives the formation of non-caseating granulomas. These granulomas persist due to chronic inflammation, even in the absence of identifiable pathogens, creating a unique pathological environment distinct from TB. Pathogenic interplay occurs when these conditions overlap. In cases of latent TB, immune suppression caused by corticosteroids, commonly used in sarcoidosis treatment, can lead to reactivation of the TB infection. Additionally, TB antigens may stimulate cross-reactive immune responses that mimic sarcoidosis-like granulomatous inflammation, further complicating the differentiation between the two conditions. As these granulomatous diseases progress, they may coexist due to shared immune pathways. Both conditions lead to systemic granuloma dissemination, involving multiple organs such as the lungs, lymph nodes, and skin. This progression highlights the intertwined nature of their pathogenesis, emphasizing the importance of a comprehensive approach to diagnosis and management in such overlapping cases [11].

Therapeutic Implications

Treating overlapping TB and sarcoidosis requires a carefully balanced approach. Anti-tubercular therapy (ATT), including isoniazid, rifampicin, pyrazinamide, and ethambutol, was initiated in this case to address the infectious component. **The patient's improvement started after starting ATT—marked by resolution of fever and reduction in lymphadenopathy—confirmed the diagnosis of TB. But radiological improvement and complete resolution of symptoms were taking time. Corticosteroids were added to manage the inflammatory response in sarcoidosis, leading to further dramatic symptomatic and radiological recovery.**

Managing Drug-Related Complications

The patient developed neuropsychiatric symptoms due to isoniazid-induced psychosis, a known side effect of ATT. Replacing isoniazid with levofloxacin allowed the continuation of effective treatment while resolving the psychiatric symptoms. This highlights the need for tailored treatment plans based on patient tolerance, as also noted by Agarwal et al. (2016).

Sequential Treatment Approach

Shah et al. (2009) and Mediouni et al. (2023) recommend a sequential approach, starting with ATT to address TB, followed by corticosteroids for sarcoidosis. This ensures control of the infection while minimizing the risk of reactivating latent TB. The favourable outcome in this patient underscores the effectiveness of this approach [12].

Relevance of Case in Broader Context

The diagnostic and therapeutic challenges faced in this case align closely with the findings of Shah et al. (2009) and Mediouni et al. (2023). The systemic involvement, including lung nodules, lymphadenopathy, and skin lesions, reflects the shared pathophysiology of TB and sarcoidosis. Diagnostic approaches emphasised in these studies, such as integrating histopathology, imaging, and clinical evaluation, were instrumental in managing this case.

This case also highlights the need to consider TB and sarcoidosis not as distinct entities but as a continuum of granulomatous diseases in TB-endemic regions. The immunological interplay described in these studies supports the hypothesis that TB antigens may trigger or exacerbate sarcoidosis, as seen in this patient.

Conclusion

This case demonstrates the diagnostic and therapeutic complexities of overlapping TB and sarcoidosis. The shared immune mechanisms, including granulomatous inflammation, systemic involvement, and immunological dysregulation, make differentiation challenging. A sequential treatment approach combining ATT and corticosteroids proved effective, underscoring the need for a tailored, multidisciplinary strategy. Such cases reinforce the importance of recognising "tuberculous sarcoidosis" as a possible continuum rather than two distinct conditions.

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