

Case Report

Curious Cavities: An Atypical Presentation of Pulmonary Sarcoidosis

Nikhita Kalra^{1*} , Duaa Mohamed¹ , Ma Del Carmen Valdes Bracamontes² , Riddhi Chauhan² , Jenelle Cole³ , Rosa Arancibia² 

¹Department of Internal Medicine, SUNY Downstate Health Sciences University, USA

²Department of Pulmonary and Critical Care, SUNY Downstate Health Sciences University, USA

³Department of Pathology, SUNY Downstate Health Sciences University, USA

*Correspondence author: Nikhita Kalra, MBBS, Department of Internal Medicine, SUNY Downstate Health Sciences University, USA;

Email: kalranikhita@gmail.com

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Abstract

Sarcoidosis is a multisystemic granulomatous disorder that usually presents with bilateral hilar lymphadenopathy and micronodules in a perilymphatic distribution. It is unusual to find ground glass opacities, consolidations and cavities on imaging in the absence of superimposed infections. These atypical findings are difficult to classify in any category of the Scadding staging system. Cavitory lesions can develop in advanced sarcoidosis due to chronic granulomatous inflammation and ischemia. Ground glass opacities are formed due to the confluence of granulomas or fibrotic lesions.

Sarcoidosis can be confirmed by the presence of non-caseating epithelioid cell granulomas on Endobronchial Ultrasound (EBUS) guided Transbronchial Needle Aspiration (TBNA) of the thoracic lymph nodes. We present an atypical case of pulmonary sarcoidosis with gradual progression from ground glass opacities to peribronchial nodules and cavities of different sizes in the absence of any associated symptoms.

Keywords: Sarcoidosis; Primary Cavitory Sarcoidosis; Ground Glass Opacities; Scadding Staging; Lung Cavities

Abbreviations

HRCT: High-Resolution Computed Tomography; GGO: Ground Glass Opacities; EBUS: Endobronchial Ultrasound; TBNA: Transbronchial Needle Aspiration; CXR: Chest X-Ray; PCS: Primary Cavitory Sarcoidosis; DVT: Deep Venous Thrombosis; PE: Pulmonary Embolism; CT: Computed Tomography; BAL: Bronchoalveolar Lavage

Introduction

Sarcoidosis is a granulomatous disorder that primarily affects the lungs and lymphatic system, but can involve extrapulmonary sites. The diagnosis is confirmed by clinical and radiological findings, histological evidence of non-caseating granulomas and exclusion of other granulomatous diseases [1]. Imaging modalities provide valuable diagnostic insights but have limited use in monitoring treatment response. The most widely adopted system was developed by Scadding based on chest X-ray findings and subsequently modified by DeRemee in 1983 [2]. It classifies sarcoidosis into five stages based on the chest X-ray findings. Stage 0 has a normal chest X-ray. Stage I involves bilateral hilar lymphadenopathy with normal lung fields. Stage II has both hilar lymphadenopathy and pulmonary infiltration. In stage III, infiltrates are seen without lymphadenopathy and stage IV indicates advanced disease with pulmonary fibrosis.

The Scadding system is helpful for prognostication. Patients may not exhibit stepwise progression according to the Scadding staging, but the probability of recovery decreases as the stage advances [1,3]. Its limitations include a lack of reproducibility, poor correlation with pulmonary function tests and exclusion of atypical radiological presentations like cavitory sarcoidosis [1]. The Muers scoring system was introduced in the late 1900s by the International Labor Organization (ILO) primarily for pneumoconiosis. It has been modified for use in sarcoidosis. It scores lung shadows based on Reticulonodular (R), Confluent (C),

Mass-like (M) and Fibrotic (F), but requires more skilled training and is a less practical approach [1,4]. A more effective way to assess the response to therapy is to compare chest X-rays taken before and after the intervention [4]. High-Resolution Computed Tomography (HRCT) of the chest has better sensitivity than CXR and is the gold standard for the detection of parenchymal abnormalities. It has been used to assess the disease findings in sarcoidosis but not particularly applied for monitoring response to therapy [4].

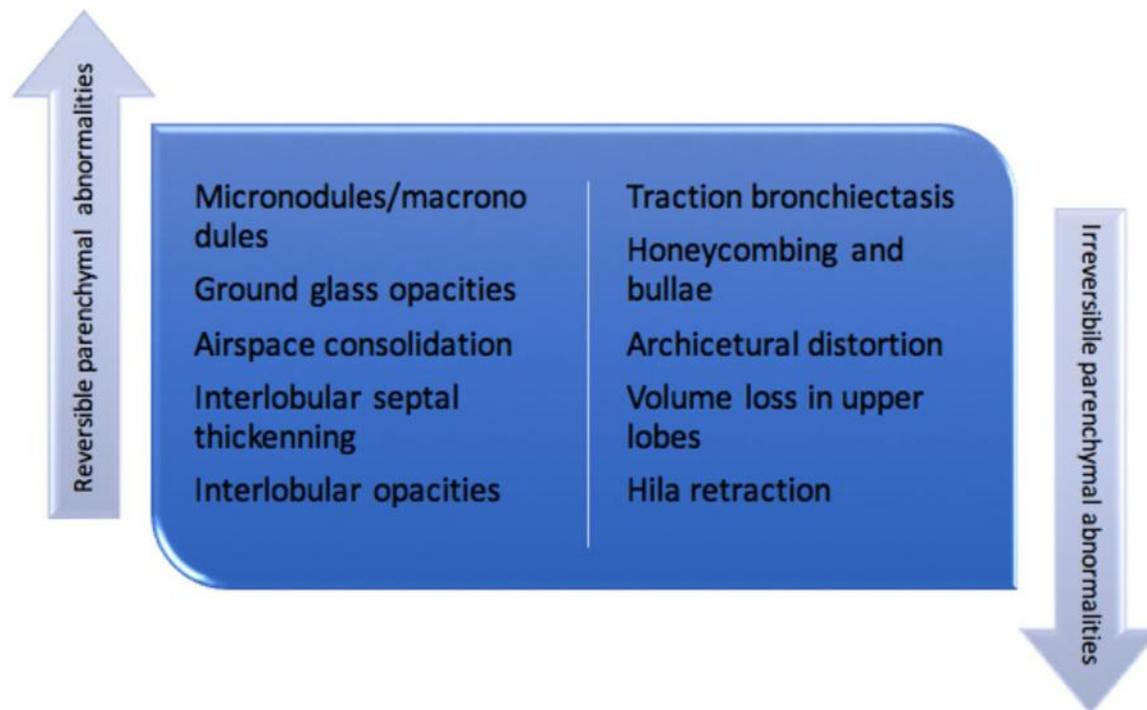


Figure 1: Reversible and irreversible parenchymal abnormalities of pulmonary sarcoidosis [2].

The most commonly seen HRCT radiological findings in sarcoidosis are bilateral hilar lymphadenopathy and micronodules in a perilymphatic distribution with predilection to the upper lobes [1,3,5,6]. They are often seen to some extent even in uncommon presentations of sarcoidosis. Some of the atypical findings include Ground-Glass Opacities (GGO), cavitations, unilateral lymphadenopathy and pleural or airway abnormalities [1,5,6]. Fig. 1 shows the various HRCT findings seen in pulmonary sarcoidosis [2]. Primary Cavitory Sarcoidosis (PCS) is only reported in about 2% of the patients and is generally associated with an active and severe disease [6]. Cavitory lesions can develop in advanced sarcoidosis due to chronic granulomatous inflammation and ischemia. It is rare for asymptomatic patients to present with such incidental findings. Cavities can resolve with corticosteroid treatment or progress to hemoptysis, pneumothorax and superimposed infections [7]. Ground glass opacities are formed due to the confluence of granulomas or fibrotic lesions. They usually present earlier in the disease course and are often found in association with micronodules and enlarged lymph nodes [1].

We present an atypical case of pulmonary sarcoidosis with gradual progression from ground glass opacities to peribronchial nodules and cavities of different sizes in the absence of any associated symptoms.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore, was exempt.

Case Presentation

A 35-year-old woman presented to the emergency department in April 2025 with a four-day history of left lower extremity pain and swelling. She also reported experiencing night sweats for two weeks, associated with fatigue and intermittent productive coughing. She denied any weight loss, recent travel, sick contacts and occupational exposures. Her medical history was significant for asthma and multiple episodes of Deep Venous Thrombi (DVT) in bilateral lower extremities and a Pulmonary

Embolism (PE). An extensive workup for thrombophilia had been unremarkable in the past. During this admission, she underwent thrombectomy for an extensive left ilio-femoral DVT.

CT chest was negative for PE but showed diffuse ground-glass and tree-in-bud opacities in a peribronchial distribution and multiple bilateral peribronchial cavities, the largest in the left upper lobe (measuring 4x3x3 cubic centimeters), as shown in Fig. 2-4. There were multiple enlarged subcentimeter bilateral hilar, mediastinal and subcarinal lymph nodes, as well as scattered bronchiectasis. These findings showed marked interval changes compared to her previous CT scans. A CT scan of the abdomen from 2018 showed patchy ground-glass opacities, most prominent in the left lower lung lobe. A CT chest from 2023 revealed cylindrical bronchiectasis with a mild tree-in-bud appearance in the bilateral upper lobes, more prominent in the left lung. CT chest from February 2025 showed bilateral lower lobe segmental PE and a 4 cm thick-walled left upper lobe cavity (Fig. 5). Her symptoms during prior years had only been related to her DVTs, but she had had no respiratory complaints or undue fatigue. No further pulmonary evaluation was done on prior admissions.

The differential diagnosis for her pulmonary findings included infections, autoimmune vasculitis, sarcoidosis and malignancy. Infectious workup for tuberculosis, *Mycobacterium avium-intracellulare*, aspergillosis, histoplasmosis and *Legionella* was negative. Autoimmune workup was negative. She underwent Endobronchial Ultrasound (EBUS) guided Transbronchial Needle Aspiration (TBNA) of the mediastinum, subcarinal and hilar lymph nodes and Bronchoalveolar Lavage (BAL). Notably, her lymph nodes were less than 1 cm in diameter. BAL was negative for infection, cell count revealed 3% lymphocytes and cytology was negative for malignancy. TBNA from her lymph nodes revealed non-caseating epithelioid cell granulomas (Fig. 6), confirming the diagnosis of sarcoidosis. She was started on oral prednisone and is now following up in the pulmonary clinic.

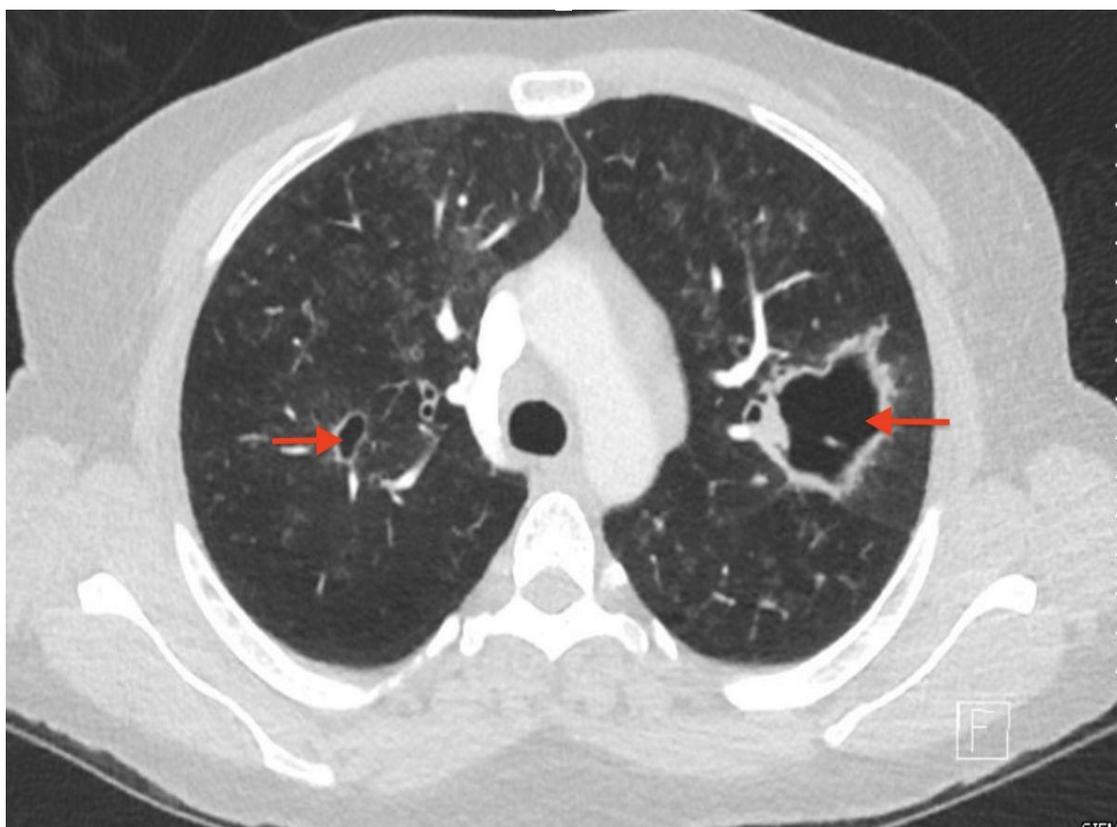


Figure 2: Computed Tomography (CT) of the chest from this admission in April 2025 showing multiple bilateral peribronchial cavities, the largest in the left upper lobe measuring 4 cm (red arrows).



Figure 3: Computed Tomography (CT) of the chest from this admission in April 2025 showing a ground glass opacity (red arrow).

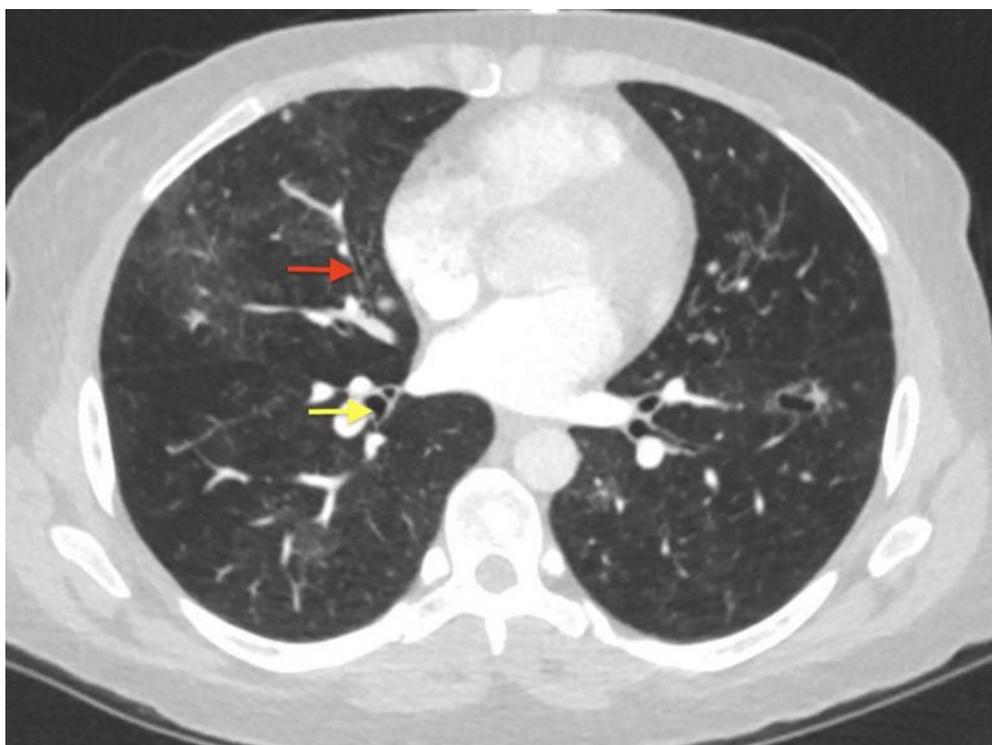


Figure 4: Computed Tomography (CT) of the chest from this admission in April 2025 showing traction bronchiectasis (red arrow) and cylindrical bronchiectasis (yellow arrow).

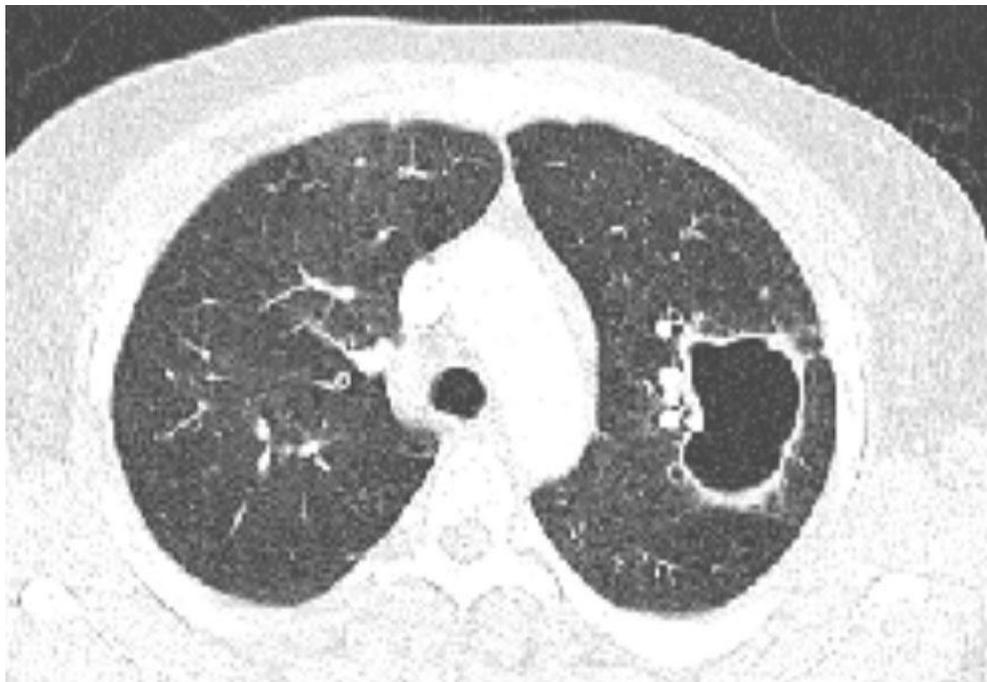


Figure 5: Computed Tomography (CT) of the chest from February 2025 showing a 4 cm thick walled left upper lobe cavity.

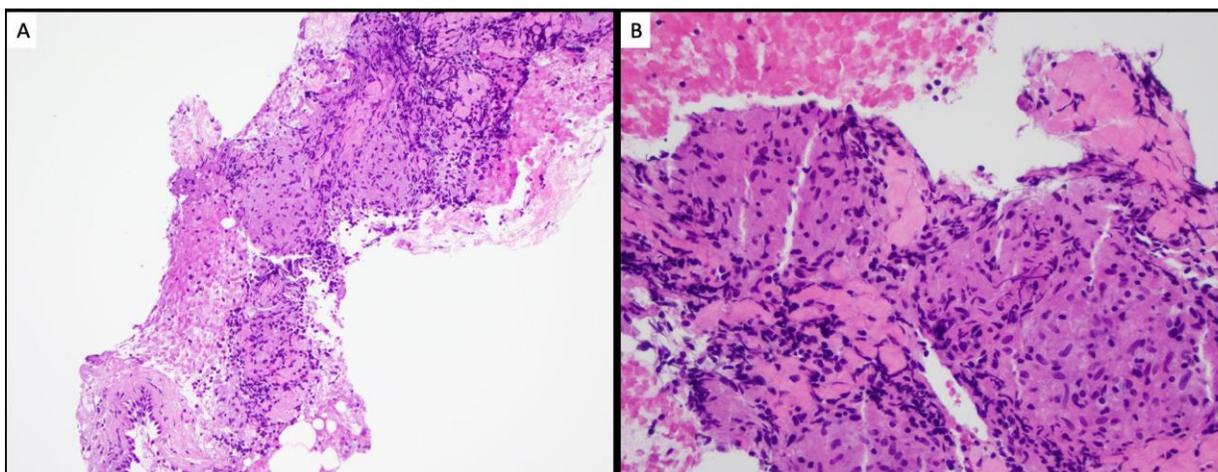


Figure 6: Histomorphologic features of non-necrotizing granulomas in the thoracic lymph node biopsy. Hematoxylin and eosin stain showed well-formed granulomas consisting of epithelial histiocytes with a lymphocytic infiltrate (Image A shows 20X magnification and image B shows 40X magnification).

Discussion

Sarcoidosis is a systemic granulomatous disease of unknown etiology that predominantly affects the lungs and the lymphatic system, though it can involve virtually any organ in the body [7]. In the United States, the estimated prevalence is approximately 60 cases per 100,000 individuals. Peak incidence typically occurs between 30 to 40 years of age in men and 50 to 60 years of age in women. The disease is believed to result from an environmental trigger in an individual with a genetic predisposition, leading to a sustained immune response and the formation of non-caseating granulomas [8].

Sarcoidosis is characterized by the formation of non-caseating granulomas in the affected tissues, most commonly the lungs. The hallmark of the disease is an exaggerated immune response, driven by CD4+ T helper cells, to an unidentified antigen. This response leads to the recruitment of macrophages and lymphocytes, which form granulomas—a collection of epithelioid histiocytes surrounded by lymphocytes and often containing multinucleated giant cells. In pulmonary sarcoidosis, these granulomas predominantly form in the interstitium along bronchovascular bundles, alveolar septa and subpleural regions [9,10]. Over time, the chronic presence of granulomas triggers a fibrotic remodeling process. This is due to persistent inflammation and

cytokine production (such as TNF- α , IFN- γ and IL-2), which stimulate fibroblast activation and collagen deposition. The lung parenchyma, including alveoli and small airways, undergoes distortion due to both granuloma infiltration and progressive fibrosis. This causes architectural disruption, reduced gas exchange and restrictive lung physiology. Notably, inflammation and subsequent fibrotic scarring can significantly weaken alveolar walls and bronchial structures [9].

Cavity formation in sarcoidosis, although relatively rare compared to other granulomatous diseases, such as tuberculosis, can result from progressive parenchymal destruction and a combination of pathophysiologic mechanisms. Granulomas may coalesce into larger nodules that undergo central necrosis due to ischemia, persistent inflammation or secondary infection. This necrotic tissue may liquefy and drain through the airways, leaving behind air-filled cavities. Additional proposed mechanisms include bullae formation secondary to airway obstruction or fibrotic traction, cystic bronchiectasis from necrotic hyaline material and granulomatous inflammation mediated by cytokines such as TNF-alpha, which can contribute to tissue necrosis. These cavities are often associated with radiologic features such as peribronchovascular nodules, fibrocystic changes, bronchiectasis and non-resolving consolidations. In primary cavitory sarcoidosis, cavitory lesions may be seen in the absence of significant fibrosis, as seen in our case [9,11]. Radiologically, these cavitory lesions are often thick-walled, round and located in the upper lobes and they may present early or develop over time [12]. Diagnosing primary cavitory sarcoidosis requires a comprehensive exclusion of infectious and neoplastic causes. Workup typically includes bacterial, fungal and mycobacterial cultures, as well as cytological analysis [11].

The prevalence of ground glass opacities in sarcoidosis ranges from 16% to 83%; they are typically found earlier in the disease course than in later stages. GGOs are a result of the confluence of granulomas or fibrotic lesions and are often found with hilar lymphadenopathy and/or nodules [1]. Isolated GGOs are considered highly unlikely to be due to sarcoidosis.

This case illustrates a rare manifestation of sarcoidosis: progression of GGOs to peribronchial nodules and pulmonary cavities. The initial CT chest showed patchy GGOs, most prominent in the left lower lung lobe, which progressed to cylindrical bronchiectasis with mild tree-in-bud appearance in bilateral upper lobes in five years. Over the next two years, a single cavity formed, which subsequently progressed to multiple bilateral peribronchial cavities, in addition to diffuse ground-glass opacities and peribronchial nodules. At this time, she also had subcentimeter hilar and mediastinal lymph nodes that were sampled by EBUS-guided TBNA, leading to the confirmation of sarcoidosis on histopathology.

Literature reports a low prevalence (approximately 2.2%) of cavitory disease in sarcoidosis, typically in advanced fibrocystic stages or with superimposed infection [10,12]. However, our patient's cavitation occurred in the absence of significant fibrosis or infection. Cavitations don't fit into the traditional Scadding staging system. This discrepancy highlights the need to reconsider staging criteria to account for atypical but clinically significant presentations such as cavitory disease.

Other studies have also described pulmonary cavitory sarcoidosis, where unilateral or bilateral pulmonary cavities are found on imaging, often in conjunction with or preceded by nodules. The cavities resolved spontaneously or with prednisone therapy [13,14]. Another case showed the presence of consolidations with lung cavities [15]. In all these cases, including ours, diagnosis was confirmed by histopathology due to the atypical radiological presentation. In our case, GGOs preceded the formation of nodules and cavitations by five to seven years and the fate of the cavities is yet to be seen.

Although cavitory sarcoidosis is itself an unusual presentation, it is rare to find such progression of sarcoidosis in the absence of any symptoms.

Conclusion

This case highlights an atypical progression of sarcoidosis from ground glass opacities to peribronchial nodules and cavities of varying sizes, in the absence of any associated symptoms. Additionally, it highlights a current limitation in the existing radiographic staging system for sarcoidosis, which is based solely on chest X-ray findings and does not account for cavitory lung involvement. It would be beneficial to consider incorporation of pulmonary cavitory findings. This case could help identify such a constellation of findings early in the course of the disease for other patients. We also hope that HRCT can be considered in the staging of pulmonary sarcoidosis as it will help evaluate disease progression.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Informed Consent

Informed consent was not obtained as it is a case report (no identifiable patient information used).

Consent for Publication

Informed consent was not obtained as it is a case report (no identifiable patient information used).

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None.

Author's Contribution

All authors have contributed equally to this work and have reviewed and approved the final manuscript for publication.

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