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BREAKING SYMMETRY: UNILATERAL SARCOIDOSIS PRESENTING AS A DIAGNOSTIC CHALLENGE

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ABSTRACT

Sarcoidosis is a multisystem granulomatous disorder that classically involves the lungs and intrathoracic lymph nodes in a bilateral distribution. Unilateral disease is rare and may closely mimic malignancy, leading to diagnostic uncertainty. We report the case of a 71-year-old man with a history of coronary artery disease, prior tuberculosis exposure, and a 20–pack-year smoking history, who was found to have pleural-based, calcified pulmonary nodules on screening computed tomography (CT) scan. Initial positron emission tomography (PET) imaging demonstrated no avidity; however, follow-up revealed interval growth and new fluorodeoxyglucose (FDG) uptake, prompting CT-guided biopsy. Histopathology showed chronic inflammation with multinucleated giant cell reaction and focal calcification, consistent with sarcoidosis and without evidence of malignancy. The patient later developed cough and wheezing, with symptoms and imaging findings improving on corticosteroid therapy. This case highlights the diagnostic challenges of unilateral sarcoidosis, the limitations of PET in differentiating granulomatous inflammation from malignancy, and the value of surveillance with targeted biopsy.

KEYWORDS

Sarcoidosis, unilateral, calcified pleural nodules

LEARNING POINTS

- *Unilateral sarcoidosis can mimic malignancy.* Even in the absence of bilateral lymphadenopathy, sarcoidosis should remain in the differential for unilateral, positron emission tomography (PET) fluorodeoxyglucose (FDG)-avid pulmonary nodules to avoid unnecessary surgical interventions.
- *Calcified pleural-based nodules represent an uncommon sarcoid phenotype.* The presence of coarse calcification does not exclude active disease, and interval progression with evolving FDG avidity highlights the need for longitudinal imaging surveillance.
- *Targeted biopsy and measured therapy are key*. In atypical presentations without accessible lymphadenopathy or extrapulmonary disease, percutaneous parenchymal biopsy is essential for diagnosis. Steroid therapy can be highly effective, and a surveillance-first approach can spare patients from invasive procedures.

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INTRODUCTION

Sarcoidosis is a multisystem inflammatory disorder characterized by non-caseating epithelioid granulomas, most commonly involving the lungs and intrathoracic lymph nodes. The disease is thought to result from an exaggerated granulomatous immune response to environmental antigens in genetically susceptible individuals. Activated macrophages, multinucleated giant cells, and T-lymphocyte–driven inflammation produces the characteristic non-necrotizing granulomas.

The clinical spectrum is broad, ranging from incidental radiographic findings in asymptomatic individuals to systemic manifestations such as fatigue, fever, and weight loss. Respiratory symptoms are common and include cough, progressive dyspnoea, and chest discomfort. Extrapulmonary involvement may present with anterior uveitis, cutaneous lesions, or cardiac conduction abnormalities.

Diagnosis requires a compatible clinical and radiographic presentation, histologic confirmation of non-caseating granulomas, and exclusion of alternative granulomatous disorders, including tuberculosis, fungal infections, and malignancy. Chest radiography (CXR) classically demonstrates bilateral symmetric hilar and mediastinal lymphadenopathy (scadding stage I, or the so-called "potato node" appearance). High-resolution computed tomography (CT) scan, the preferred modality, more clearly delineates peri-lymphatic micronodules in the upper and middle lung zones, peri-broncho vascular nodularity, conglomerate masses, and, in chronic disease, fibrotic changes with architectural distortion and traction bronchiectasis.

Although sarcoidosis typically presents with bilateral pulmonary involvement, unilateral or markedly asymmetric thoracic disease is uncommon and may pose a diagnostic challenge. Herein, we describe a case of sarcoidosis with unilateral pulmonary involvement.

CASE DESCRIPTION

A 71-year-old man with a history of hypertension, hyperlipidaemia, coronary artery disease status post coronary artery bypass grafting, and benign prostatic hyperplasia, as well as a 20-pack-year smoking history and prior tuberculosis exposure, was referred to the pulmonary clinic for evaluation of pulmonary nodules incidentally identified on a screening low-dose non-contrast chest CT scan in February 2021. He was asymptomatic at presentation, denying cough, dyspnoea, or weight loss.

On examination, he was afebrile with a heart rate of 84 bpm, respiratory rate of 12 breaths per minute, oxygen saturation of 97% on room air, and blood pressure of 111/70 mmHg. He had an increased anteroposterior chest diameter but exhibited normal respiratory effort. Lung fields were clear to auscultation bilaterally without adventitious sounds.

Initial CT scan revealed a pleural-based left apical nodule with coarse calcification measuring 2.6 × 1.6 × 1 cm *(Fig. 1)*, as well as a circumscribed, partially calcified pleural-based nodule in the lingula measuring 2 × 1 × 3 cm. Multiple scattered 4 mm calcified nodules were also noted in the left upper and lower lobes. The right lung was free of nodules, and there was no mediastinal lymphadenopathy.

Given concern for malignancy, a positron emission tomography (PET) scan was obtained, which demonstrated no fluorodeoxyglucose (FDG) uptake *(Fig. 2)*. A repeat CT scan in November 2021 revealed a moderate left pleural effusion attributed to heart failure, which was resolved with medical therapy. An annual surveillance CT scan in



Figure 1. Initial computed tomography scan showing pleural based left apical nodule with coarse calcification measuring 2.6 × 1.6 × 1.0 cm (red arrow).

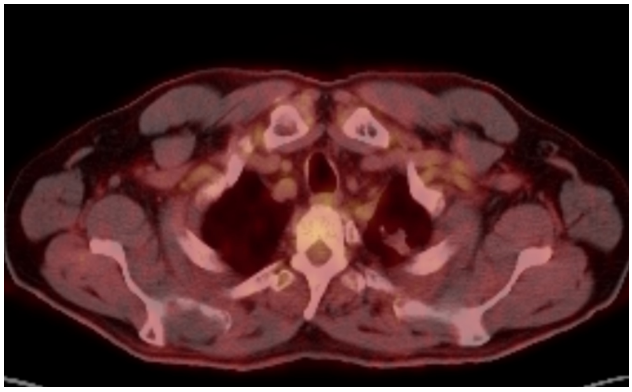


Figure 2. Positron emission tomography scan corresponding with the initial computed tomography scan, which demonstrated no fluorodeoxyglucose uptake.

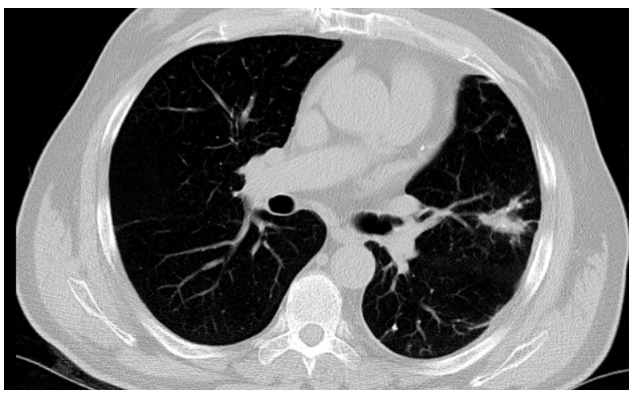


Figure 3. Computed tomography scan of the chest without contrast in 2023 showing interval change with increased nodularity in the left upper lobe, and new infiltrate more voluminous along the medial aspect.

November 2022 demonstrated stable nodules. In 2023, follow-up CT scan demonstrated increased nodularity in the left upper lobe, appearing more voluminous along the medial aspect *(Fig. 3)*. Repeat PET scan showed FDG avidity with a standardized uptake value (SUV) of 4 in a posterior left upper lobe nodule *(Fig. 4)*, a SUV of 3 in an adjacent posterior medial nodule, and mild uptake (SUV 3) in a left lower lobe parenchymal density. CT-guided biopsy of the left upper lobe lesion revealed chronic inflammation with multinucleated giant cell reaction and focal calcification, findings consistent with sarcoidosis and without evidence of malignancy.

At the 6-month follow-up, CT scan demonstrated stable nodularity with calcifications. At one year, however, the nodules had increased in size, and the patient reported a new cough and wheezing. He was initiated on oral corticosteroid therapy, which led to symptomatic improvement and interval regression of nodularity on repeat imaging. Serum angiotensin-converting enzyme and vitamin D levels were within normal limits. The patient continued to improve despite tapering and eventual discontinuation of corticosteroids. Follow-up CT scan demonstrated stable disease *(Fig. 5)*.

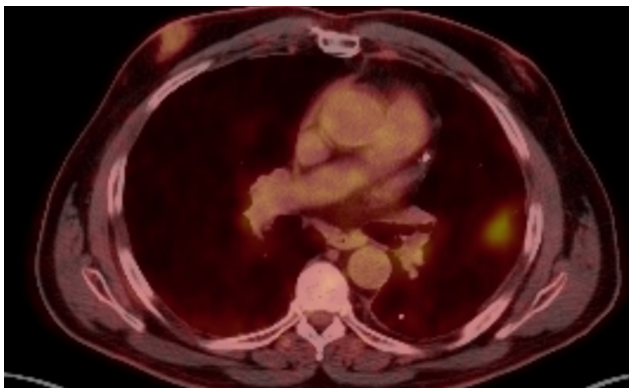


Figure 4. Repeat positron emission tomography scan, in 2023, showed fluorodeoxyglucose avidity with a standardized uptake value of 4 in a posterior left upper lobe nodule.

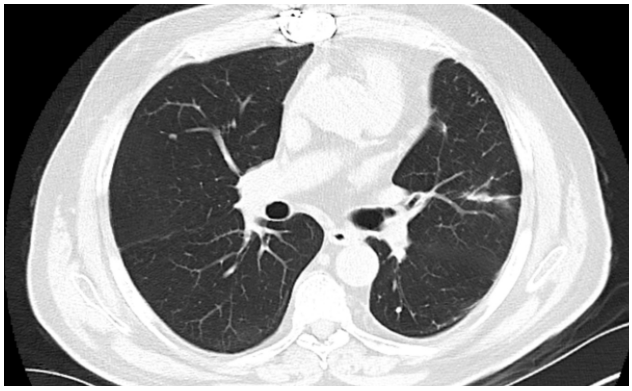


Figure 5. Computed tomography scan in March 2025 showing interval decrease in nodularity after treatment with steroids.

DISCUSSION

Sarcoidosis is classically a bilateral disease of the lung parenchyma and intrathoracic lymph nodes, yet a growing body of literature underscores that unilateral or markedly asymmetric disease, though rare, can complicate diagnosis and management. The case presented here—a 71-year-old man with unilateral pleural-based calcified pulmonary nodules, interval development of PET avidity, absence of mediastinal lymphadenopathy, and eventual steroid-responsive progression—illustrates several ways in which atypical sarcoidosis challenges conventional diagnostic algorithms. Comparison with previously published reports of unilateral sarcoidosis highlights both shared themes and distinctive features.

One of the most consistent themes in the literature is that unilateral sarcoidosis frequently masquerades as malignancy, leading to delays in diagnosis and, at times, unnecessary surgical interventions. Reports describe patients undergoing orchiectomy for unilateral scrotal masses later found to be sarcoid granulomas[1], parotidectomy for presumed neoplasms[2], and mediastinoscopy or open lung biopsies for hilar tumour masses with suspicious PET uptake[3]. Similarly, our patient's nodules evolved morphologically over two years and became FDG-avid on PET, prompting biopsy to exclude malignancy. Unlike some of the aforementioned cases, where surgical resections were performed before the diagnosis of sarcoidosis was established, this case illustrates the value of a staged diagnostic strategy: initial surveillance, followed by minimally invasive CT-guided biopsy once radiographic and metabolic changes raised concern. This approach avoided unnecessary radical procedures while still establishing the diagnosis.

Another point of similarity is the localization of the disease to a single hemithorax. Historical reports have documented unilateral hilar lymphadenopathy[4], and cotton-like unilateral parenchymal opacities that resolved spontaneously[5]. In contrast to these predominantly parenchymal or nodal patterns, the current case was distinguished by pleural-based calcified nodules without associated hilar or mediastinal lymphadenopathy. Calcification in sarcoid nodules has been described, but it is relatively uncommon as a dominant radiographic finding and is rarely emphasized in unilateral presentations[6]. The coarse calcification present from the initial CT scan onward in this patient may have tempered early concern for malignancy; nevertheless, the subsequent increase in size, new nodularities, and avidity underscored the importance of ongoing vigilance.

An especially unique aspect of this case is the temporal evolution of PET findings. The initial PET scan in 2021 was non-avid, which supported a conservative approach. By 2023, however, the same nodules demonstrated SUVs of 3–4, findings that would typically raise suspicion for malignancy. Case reports have highlighted both false-positive PET findings due to sarcoid inflammation and false-negative results when disease activity is low[7,8]. Few, however, describe a longitudinal conversion from non-avid to avid within the same lesions. This pattern suggests waxing and waning granulomatous activity rather than neoplastic transformation and underscores the limitations of PET as a standalone diagnostic tool in granulomatous disease.

The role of pleural involvement further differentiates this case from others. Pleural effusion is a rare manifestation of sarcoidosis, occurring in roughly 2.8% of outpatients, and only a minority of those effusions are directly attributable to sarcoid granulomatous inflammation[9]. Several case reports describe unilateral effusions proven to be sarcoid by pleural biopsy[10]. In our patient, however, the moderate left pleural effusion detected in 2021 was attributed to heart failure and improved with diuretic therapy. Thus, while the imaging suggested a potential sarcoid pleural complication, careful clinical correlation clarified an alternate aetiology. This distinction is clinically important because it avoids misattribution of common comorbid conditions to sarcoid and thereby prevents overtreatment. Histopathologically, the diagnosis was established through CT-guided biopsy, demonstrating chronic inflammation, multinucleated giant cell reaction, and focal calcification without evidence of malignancy. While noncaseating granulomas are the sine qua none of sarcoidosis, the absence of mediastinal lymphadenopathy in this case contrasts with the majority of published unilateral cases, which often still display ipsilateral nodal enlargement[4]. This reinforces the principle that in atypical cases, tissue sampling of pulmonary nodules themselves may be necessary when nodal disease is not present or accessible.

Another dimension worth noting is the lack of extrapulmonary involvement in this patient. The unilateral cases described in the literature frequently include or are dominated by extrapulmonary findings: lachrymal gland enlargement with hemifacial deformity[9], parotid swelling[2], orbital or optic nerve masses[11], or unilateral testicular lesions[1]. These provide alternative biopsy targets and sometimes accelerate recognition of sarcoidosis once malignancy is excluded. In contrast, the patient here exhibited no extrapulmonary sarcoid manifestations, limiting diagnostic avenues and further emphasizing the reliance on thoracic imaging and biopsy.

The clinical course and management also distinguish this case. In several case reports of unilateral sarcoidosis, diagnosis was delayed until symptoms became severe, and treatment was often instituted only after invasive interventions or misdirected therapies[5]. By comparison, our patient was managed conservatively with imaging surveillance until symptoms developed. Once initiated, corticosteroid therapy led to prompt improvement in both respiratory complaints and radiographic findings, with stability maintained even after steroid taper and discontinuation. This outcome highlights the importance of tailoring therapy not only to imaging features but also to the patient's clinical trajectory.

Taken together, these contrasts reinforce broader lessons. First, sarcoidosis should remain in the differential diagnosis of unilateral pulmonary lesions, particularly when biopsy reveals noncaseating granulomas, even in the absence of classic bilateral adenopathy. Second, calcified pleural-based nodules with variable PET activity represent an uncommon but important sarcoid phenotype that can mimic malignancy. Third, careful attribution of associated findings, such as pleural effusions, is critical, since not all thoracic abnormalities in sarcoid patients are due to granulomatous inflammation. Finally, a surveillance-first strategy with selective biopsy may be more appropriate in stable, asymptomatic patients, thereby minimizing invasive interventions without delaying effective therapy when progression occurs.

In conclusion, the present case adds to the spectrum of reported unilateral sarcoidosis by illustrating a rare constellation of pleural-based calcified nodules, biphasic PET behaviour, absence of nodal disease, and subsequent steroid responsiveness. Compared with prior cases—ranging from unilateral hilar adenopathy with fever of unknown origin[4] to unilateral parotid or orbital masses [12]—this patient's disease course underscores the heterogeneity of sarcoid presentations and the diagnostic challenges posed when classic features are absent. Clinicians should recognize that sarcoidosis may mimic malignancy in unilateral thoracic disease, but thoughtful integration of serial imaging, PET interpretation, histopathology, and clinical context can guide appropriate management and prevent unnecessary interventions.

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