World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

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Systemic granulomatosis sarcoidosis like: Case report

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World Journal of Advanced Research and Reviews, 2025, 27(03), 1989-1993

Publication history: Received on 26 July 2025; revised on 06 September 2025; accepted on 28 September 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.27.3.3131

Abstract

Many granulomatous diseases can mimic sarcoidosis histologically and in terms of their clinical features. These mimics include infectious granulomatous diseases, granulomatous reactions to occupational and environmental exposures, granulomatous drug reactions, vasculitides and idiopathic granulomatous conditions.

We present here the case of a 50-year-old patient admitted for the management of systemic granulomatosis sarcoidosis like with lung, ORL, lymph node and skin involvement.

Keywords: Systemic Granulomatosis; Sarcoidosis Like; Lung Involvement; Case Report

1. Introduction

The term granulomatous lung disease does not refer to a specific disease entity, but to a wide spectrum of pathologies with variable clinical manifestations and outcomes. Both infectious and noninfectious diseases can be associated with granuloma formation.

Sarcoidosis is the most common granulomatous lung disease. Distinguishing features between sarcoidosis and its mimics requires careful clinical evaluation, laboratory testing, pulmonary function testing and radiological imaging including computed tomography. In most cases, lung biopsy with expert pathological examination of lung tissue specimens is necessary.

2. Case presentation

This is a 50-year-old patient, nonsmoker, treated for pulmonary tuberculosis in 1999 and declared cured, currently admitted for the management of cervical lymphadenopathy with dyspnea.History of the disease goes back 2 years with the onset of continuous stage II dyspnea with dry cough without chest pain, without hemoptysis, wheezing or chest tightness, with no seasonality or triggering factors, evolving in a context of general deterioration and asthenia with unquantified weight loss, prompting the patient to consult a primary care Physician. A cervical ultrasound performed on the patient revealed cervical lymphadenopathy, biopsy was done which returned in favor of epithelioid and giant cell granulomatous adenitis without caseous necrosis. A chest, abdominal, and pelvic CT scan was requested, revealing calcified lymph node formations and mediastinal adenomegaly, with a moderately abundant pericardial effusion.

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A large pleural effusion was present. In the parenchymal window, we note the presence of perihilar parenchymal consolidation, associated with multiple parenchymal nodules and micronodules in both pulmonary hemifields,with subpleural and peribronchovascular distribution.

He underwent pleural puncture and biopsy, which revealed a fibro-inflammatory reaction with no granulomatous lesions or caseous necrosis, and no signs of malignancy with negative fragment culture, negative GenXpert. A fibroscopy with bronchoalveolar lavage (BAL) revealed a mixed paucicellular fluid, with a mixed cell population of macrophages, neutrophils and lymphocytes with a few eosinophilic cells, with no suspicious or tumour cells, a negative BAAR test in the fibroaspiration fluid and a negative expert gene. Tumour markers came back negative (ACE and CA 15.9). Respiratory function test: the walk test starts at 93% and desaturates after 1 minute at 90%, ending at 88%. Spirometry showed a restrictive ventilatory disorder.

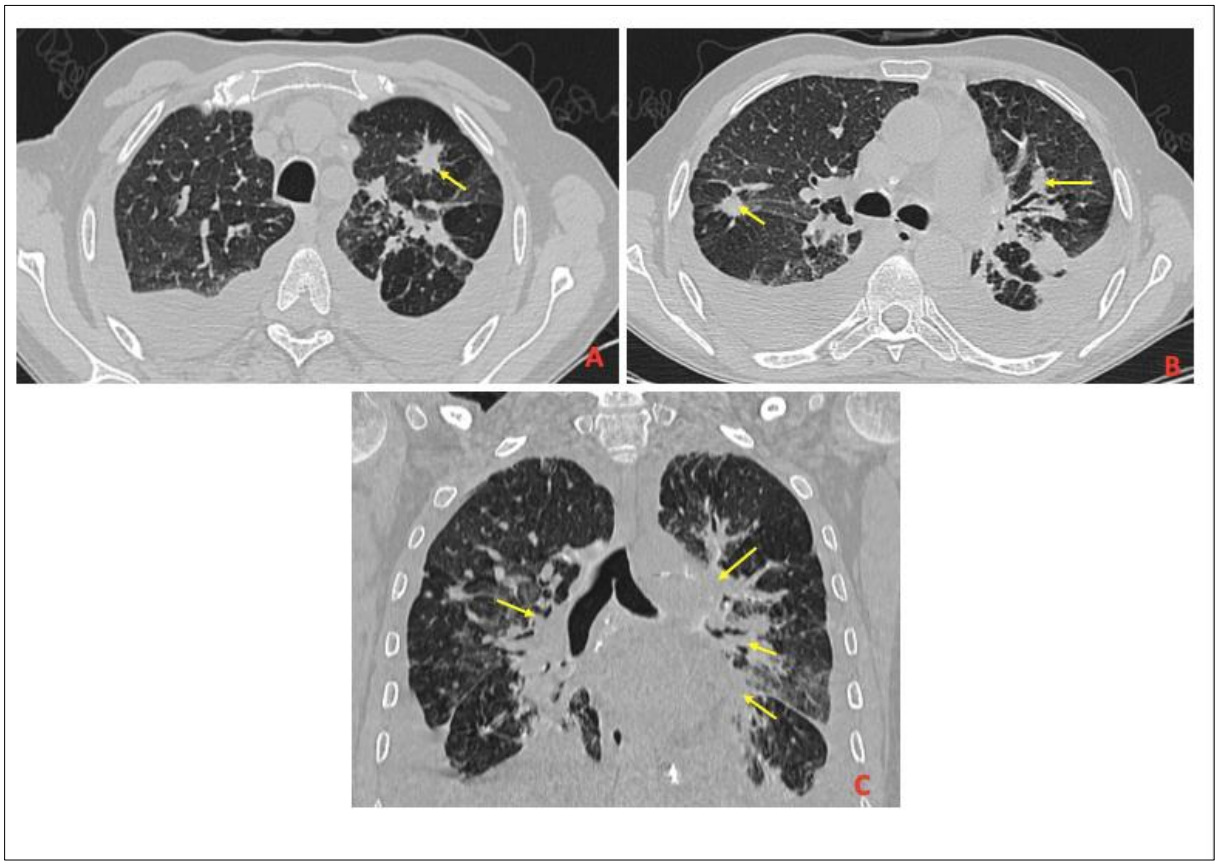

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Figure 1 Axial and coronal reconstruction CT scan in the parenchymal window, show the presence of perihilar pulmonary parenchymal consolidation foci (C), traversed by air bronchograms associated with multiple parenchymal nodules and micronodules in both pulmonary hemifields, in a subpleural and peribronchovascular arrangement, with irregular, retractile contours, and some confluent

The patient also had ulceration of the nasal mucosa with perforation of the septum. The nasofibroscopy was unremarkable and the biopsy showed granulomatous lesions without caseous necrosis associated with necrotising vasculitis, suggesting Wegener's disease. The patient also presented with erythematous plaques on the face and papules, for which the skin biopsy was in favour of Wegener's disease.

The decision was made to complete the biological investigations, which were negative (Anti-neutrophil cytoplasm antibody, C-ANCA antibody, ANTI-P-ANCA antibody), viral and syphilitic serologies, liver work-up, LDH, microglobulin, 24-hour proteinuria, blood and urine phosphocalcium work-up, with an Ig A level of 0.25 (0.7-4). The patient was then admitted to hospital for diagnostic adjustment.

As the biopsies pointed to Wegner's granulomatosis, but the presentation was atypical, with negative ANCA, no renal involvement, and the re-evaluation of the histopathology slides revealed epithelioid cell granulomas without necrosis and infiltration of the vessel wall, which did not fully meet the definition of Wegner's necrotising vasculitis.

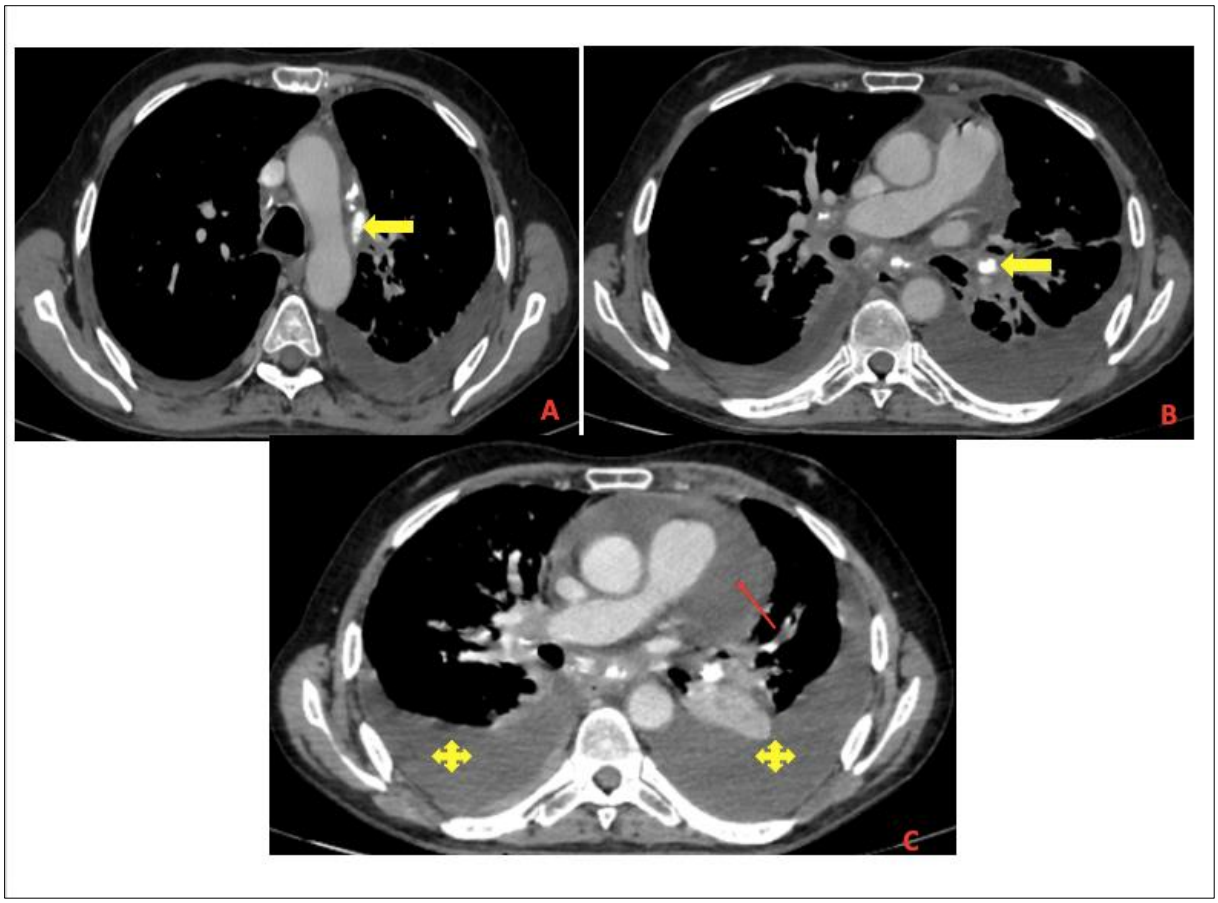

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Figure 2 Axial chest CT scan showing calcified lymph nodes formations and mediastinal adenomegaly (A,B: yellow arrow), with a moderately abundant pericardial effusion, without signs of tamponade (red arrow) associated with a large pleural effusion bilaterally (C)

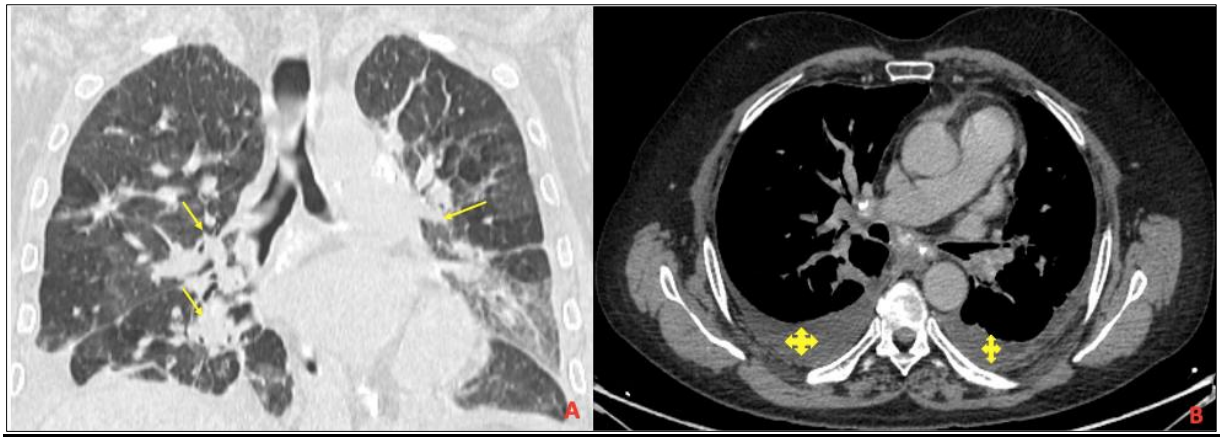

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Figure 3 Coronal reconstruction chest CT scan parenchymal window showing regression of the peri hilar condensation (A: yellow arrow), as well as pleural effusion in mediastinal window(B) after treatment

The final diagnosis was aseptic systemic granulomatosis with bronchial, ORL, lymph node and skin involvement, associated with Ig A deficiency. Corticosteroid therapy was started, and a follow-up thoraco-abdomino-pelvic CT scan showed partial regression of the peri-hilar condensation foci and peri broncho-vascular interstitial infiltrates bilaterally, as well as pleural effusion, which had become moderate in size and more marked on the right.

3. Discussion

The spectrum of granulomatous lung diseases is broad. To diagnose sarcoidosis, other infectious granulomatous lung diseases such as tuberculosis, atypical mycobacterial and fungal infection have to be ruled out. Pulmonary granuloma also evolve in the context of autoimmune diseases such as rheumatoid arthritis, granulomatosis with polyangiitis and eosinophilic granulomatosis with polyangiitis. Furthermore, immunodeficiencies such as common variable immunodeficiency and immune reconstitution syndrome in HIV can be associated with systemic granulomatous inflammation. Finally, occupational lung disease, particularly hypersensitivity pneumonitis, silicosis, hard metal lung, and chronic berylliosis are associated with pulmonary granuloma formation [1-2]. Sarcoidosis is a systemic granulomatous disease of unknown etiology in more than one organ system. For a while thereafter, sarcoidosis was considered a disorder of the skin. However, the multisystem nature of sarcoidosis was soon realised, and it also became clear that the lungs bear the brunt in most patients. The cardinal diagnostic finding on histopathologic examination is the non-necrotising or non-caseating epithelioid cell granuloma. Imaging tests play a role not only in diagnosis but also in management and follow-up.

The basic lesions of sarcoidosis in imaging are: enlargement of mediastinal and hilar lymph nodes is a hallmark of sarcoidosis..On the whole, symmetrical hilar nodal enlargement most often points to a diagnosis of sarcoidosis and away from lymphoma, other malignancies and tuberculosis. Necrosis of lymph nodes is recognised in sarcoidosis but should prompt a search for an alternative etiology, such as TB, nodal calcification, also tends to be bilateral and may have a focal pattern so-called ‘egg-shell’ calcification is also reported. Precise localisation of intra-thoracic lymph nodes on CT may facilitate the planning of endobronchial ultrasound-guided biopsy. Most parenchymal lesions predominate in the upper and middle territories of the lungs, especially the posterior territories of the upper lobes. Micronodules have a perilymphatic distribution; in the subpleural region, at the level of the fissures creating a pearly appearance of the fissure, at the level of the peribronchovascular spaces. They have irregular contours, can merge and often describe the galaxy or cluster sign: center of the dense nodule with a peripheral micronodular halo. On occasion, granulomata coalesce to form larger nodules or masses, sometimes manifesting as a pattern of consolidation. Irregular or nodular peribronchovascular thickening of the bronchial walls may be found [3] .

Differentiating between sarcoidosis as an autonomous disease and sarcoid-like reactions requires considerable efforts. The epithelioid cell granuloma is not equivalent to sarcoidosis because it may be identified in a number of infectious and noninfectious disorders, including neoplastic diseases. At the current state of knowledge, accurate distinction between different causes of epithelioid cell granulomas is in many cases not possible. Its etiology should be sought through careful additional investigations, including the genetic signature of both conditions.

Because vasculitides are systemic disorders, commonly affect the lung and may be associated with granulomatous inflammation, they are occasionally confused with sarcoidosis. However, the clinical presentations and histologic findings of granulomatous vasculitides are usually sufficiently discordant from sarcoidosis that the two diseases can usually be easily differentiated.

In our case, the main differential diagnosis was granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis, wich is a vasculitis of small to medium-sized vessels that usually affects the upper airway, ear/nose/throat, airways, lung parenchyma and kidney. Common extrapulmonary presentations include renal (hematuria), nervous system (polyneuritis), ocular, skin, muscle, and joint involvement, hearing loss, sinusitis, epistaxis, septal perforation, saddle nose deformity, Although most of these presentations have been reported with sarcoidosis, they are relatively rare. Pulmonary involvement with GPA may result in cough, dyspnea, chest discomfort and radiological manifestations include: pulmonary nodules and opacities on lung imaging that are all common presentations of pulmonary sarcoidosis. However, hemoptysis, alveolar hemorrhage and cavitary lung lesions are common presentations of GPA and are rare with sarcoidosis. Anti-neutrophil cytoplasmic antibodies against proteinase 3 (c-ANCA, PR3+) has a high specificity for active GPA. whereas these antibodies are routinely negative with sarcoidosis. Typical histologic features of GPA include necrotizing granulomas in combination with a necrotizing vasculitis , which are uncommon with sarcoidosis [4-5]

4. Conclusion

Many granulomatous diseases can mimic sarcoidosis histologically and in terms of their clinical and imaging features. These mimics include infectious granulomatous diseases, granulomatous reactions to occupational and environmental exposures, granulomatous drug reactions, vasculitides and idiopathic granulomatous conditions. It is important to distinguish sarcoidosis from these mimics, as a misdiagnosis of these diseases may have serious consequences.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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