An Eosinophilic Pneumonia Mimicking Lung Cancer on Multiple Imaging Modalities Monitored By CT

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AUTHOR CONTRIBUTIONS
Filippo Montella: The author was responsible for the conception, design, data collection, imaging review, manuscript writing, and final approval of the submitted version.
Antonietta Vitale: The author helped with data collection, imaging review, manuscript writing and analysis.
Ghassan Merkabaoui: The author helped with data collection and data interpretation.
Guido Faggian: The author helped with study design and literature search.
Serena De Luca: The author supervised imaging review, manuscript writing, analysis.

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None

CONFLICT OF INTEREST
None declared.

CONSENT
Yes

HUMAN AND ANIMAL RIGHTS
None

ABSTRACT

Eosinophilic pneumonia (EP) is a rare interstitial lung disease often mimicking other pulmonary conditions. We present the case of a 43-year-old male evaluated for suspected lung cancer due to progressive dyspnea, weight loss, cough. Cytological analysis confirmed an interstitial pulmonary inflammatory process with significant eosinophilic granulocyte infiltration, with no evidence of malignancy. Complete clinical and radiological resolution was achieved within two weeks following corticosteroid and empiric antibiotic therapy. This case highlights the challenging differential diagnosis of EP, especially when mimicking malignancy, emphasizing the crucial role of a complete clinical evaluation based on a multimodality imaging diagnostic assessment and prompt response to corticosteroids for diagnosis confirmation.

CASE REPORT

BACKGROUND

Eosinophilic lung diseases represent a heterogeneous group of rare pulmonary conditions characterized by eosinophilic infiltration of the lung parenchyma. Among these, chronic eosinophilic pneumonia (CEP) is often challenging to diagnose due to its nonspecific clinical and radiological features, which can mimic infectious, inflammatory, or neoplastic conditions. This case is significant because it describes a reversible eosinophilic pulmonary infiltrate that was initially misinterpreted as a malignant lesion, highlighting the importance of a thorough diagnostic workup and the inclusion of eosinophilic lung diseases in the differential diagnosis of atypical pulmonary opacities. By presenting this case, we aim to raise awareness of this rare but treatable condition and contribute to the existing literature by illustrating its radiologic and pathologic features.

CASE REPORT

A previously healthy 43-year-old Caucasian male presented to the Oncology Department of "AOU Federico II" in Naples in January 2025 with suspicion of lung cancer. Over the preceding two months, he had experienced progressive symptoms including dyspnea, unintentional weight loss, fatigue, a persistent cough and multiple episodes of bronchitis which were treated empirically with antibiotics. His past medical history included allergic rhinitis and Basedow-Graves disease. He was a former smoker (15 cigarettes/day for 20 years) but reported no exposure to environmental toxins, tropical diseases, or illicit drugs.

In November 2023 the patient presented with cough and asthenia, without peripheral eosinophilia. A chest radiography revealed a suspicious solitary pulmonary nodule of 11 mm in the lingularregion, raising suspicion for malignancy. A subsequent 18-fluorodeoxyglucose positron emission tomography- computed tomography (**18F-FDG PET/CT)** scan demonstrated a low and nonspecific uptake in the lingular region (SUV**max 1.1**) (Figure 1), excluding the tumor hypothesis. To evaluate the possibility of a potential neuroendocrine tumor too, which can exhibit low FDG avidity, a **68 Gallium-DOTATOC PET** scan was performed three months later that showed no abnormal somatostatin receptor expression in the lesion, effectively excluding a neuroendocrine tumor (Figure 2).

One year later a follow-up chest CT, two areas of consolidation with surrounding ground-glass opacities, one in the posterior segment of the right lower lobe and the other one in the superior lingular segment of the left upper lobe, corresponding to the previously noted pulmonary nodule, which had increased in size and was now incorporated within the consolidation (38x44x48 mm) (Fig. 3-4). Therefore an 18F-FDG-PET/CT scan was repeated and revealed a higher but still non-specific uptake on these areas (SUVmax 5.4 and 3.5, respectively) (Figure 5).

Laboratory evaluation showed:

- White blood cell count: **11.3 x 10^3/µL**
- **Eosinophils**: **6.7% (0.76 x 10^3/µL)**
- Mildly elevated **C-reactive protein (CRP): 123 mg/L % (70-120)**
- **Mildly elevated fibrinogen: 524 mg/dL (160-350)**
- **Tumor markers**: CEA, NSE, CA 19-9, CA 15-3, AFP within normal limits.

The presence of peripheral blood eosinophilia, commonly observed in patients with Chronic Eosinophilic Pneumonia (CEP), was also detected in our patient and should be highlighted as a relevant clinical finding.

To obtain a definitive diagnosis, a CT-guided percutaneous cytology was performed. Cytologic analysis revealed an interstitial pulmonary inflammatory process characterized by a significant infiltration of eosinophilic granulocytes, with no evidence of malignancy. The patient started corticosteroids (betamethasone) and empiric antibiotics (levofloxacin 500 mg twice/day and ceftriaxone 1 g/day for 7 days) therapy, in line with treatment for community-acquired pneumonia (CAP). Within two weeks, the patient experienced complete clinical and radiologic resolution. A follow-up chest CT scan confirmed the disappearance of pulmonary consolidations and the lingular nodule (Fig. 6).

DISCUSSION

Etiology & demographics

EP is a rare and insidious inflammatory lung disorder, accounting for less than 3% of cases of various interstitial lung diseases. EP can be classified as a primary (i.e., acute and chronic eosinophilic pneumonia) or secondary disorder (i.e., allergic bronchopulmonary aspergillosis (ABPA)), parasitic infection or drug reaction as well as eosinophilic vasculitis (i.e. eosinophilic granulomatosis with polyangiitis (EGPA)) [1-4]. The incidence and prevalence of pulmonary eosinophilic disorders are uncommon and vary depending on the specific clinical syndrome and underlying causes.

There are two main types of primary or idiopathic EP [5]:

- Acute Eosinophilic Pneumonia (AEP): a rare condition (9.1 cases/100.000 person-years) characterized by rapid onset of symptoms (typically <1 week). It commonly affects young adult males, particularly following recent changes in smoking habits, without a history of atopic disorder [5, 6]. Several reports have documented an increased incidence of AEP in individuals who have recently begun smoking, and current evidence suggests that exposure to secondhand smoke [7-9].

- Chronic Eosinophilic Pneumonia (CEP): despite its name, CEP can have an acute or subacute presentation and is often recurrent. It most frequently affects middle-aged women (2:1 female/male ratio), with peak incidence occurring between the ages of 30 and 39. Approximately 50% of these patients have a history of allergic disorders, such as allergic rhinitis, asthma, and other atopic conditions [10]. Although it is a rare disease, representing less than 3% of cases of various interstitial lung diseases, CEP is the most common of the eosinophilic pneumonias in no tropical areas where the prevalence of parasitic infection is low [5].

Clinical & imaging findings

In AEP symptoms include fever, progressive dyspnea, cough, and can progress to respiratory failure. AEP generally responds well to corticosteroids and rarely relapses [5]. CEP manifests with prolonged symptoms such as cough, fever, dyspnea, wheezing and night sweats. It may be misdiagnosed as community-acquired pneumonia. Asthma is present in 50-75% of cases and is often severe [10]. Relapse is common. Diagnosis of EP may be established by:

1. Pulmonary opacities with peripheral eosinophilia.
2. Elevated eosinophils in bronchoalveolar lavage (BAL) fluid (> 25%).
3. Lung biopsy showing eosinophilic infiltration [2].

Radiological findings of EP:

In AEP, chest X-rays may show diffuse bilateral reticular densities or alveolar infiltrates, which can mimic cardiogenic edema or acute respiratory distress syndrome (ARDS). Other radiological findings on the High-Resolution Computed Tomography (HRCT) include pleural effusions (found in 60–100% of cases), bronchovascular bundle thickening, bilateral, patchy or random distribution ground-glass opacities and smooth interlobular septal thickening [5, 11]

CEP is associated with migratory or chronic airspace opacities, as well as peripheral upper/middle region-lung consolidations ("photographic negative of pulmonary edema") [12]. HRCT often reveals ground-glass opacities (frequently next to consolidations), linear band-like opacities and subpleural consolidations (typical in the upper/middle -zone predominance) [11]. Less frequent features include nodules, septal thickness and the reverse halo ("atoll") sign [1, 2, 5, 13].

Treatment & prognosis

AEP generally responds well to corticosteroids and rarely relapses. In CEP relapse is common and in these cases corticosteroid therapy is often needed [5].

Differential Diagnoses

Reaching a differential diagnosis between CEP and other lung disease (whether eosinophilic or not) can be difficult because CEP shares many clinical and radiological features with several lung diseases of different etiologies. The easiest differential diagnosis is between AEP and CEP, due to the rapid onset of symptoms, prompt clinical progression, absence of peripheral blood eosinophilia and radiologic evidence of diffuse lung bilateral consolidations. An atypical radiologic presentation of CEP on CT-scan with a focal and irregular consolidation can mimic a lung cancer. In our case the presence of a previous single lung nodule and two speculated-margin consolidations in contact with the pleura immediately led to including CEP in the differential diagnosis with lung cancer [14].

Peripheral blood eosinophilia is a common laboratory finding in patients with CEP, being reported in 80-90 % of cases [5]. In our patient laboratory tests revealed an increased eosinophil count (6.7 %; 0.76 x10^3/μL), supporting the diagnosis of CEP.

Another differential diagnosis of CEP is with autoimmune forms, such as EGPA, formerly Churg-Strauss syndrome. EGPA is a multisystem disease characterized by necrotizing granulomatous and eosinophilic inflammation of small and medium-sized vessels and the respiratory tract. It is often associated with the involvement of extra-pulmonary organs, presenting with peripheral neuropathy, skin lesions, joint pain and gastroenteritis. In ~40% of cases, positivity for ANCA/MPO-ANCA is found. The absence of systemic involvement and recovery without immunosuppressive therapy led us to exclude the diagnosis of EGPA [3, 15]. Furthermore, drug-induced pulmonary eosinophilia (i.e., amoxicillin or sertraline) can also present with respiratory symptoms, pulmonary infiltrates and eosinophilia, but the patient had no history of taking these drugs [16]. Given the lack of recent foreign travel, a diagnosis of helminth or fungal infections was also rejected. Another disease to consider in the differential diagnosis with CEP is ABPA. It is similar to CEP with a previous history of asthma, peripheral blood eosinophilia, and radiographic opacities localized to the upper lung lobes, but requires isolation of Aspergillus fumigatus from BAL or culture [4]. Among infectious diseases, CAP has an uncommon bilateral involvement, but it remains among the possible alternative diagnoses; indeed, our patient was treated pharmacologically with empirical antibiotic therapy for CAP. However, the presence of subacute symptoms (particularly cough, fatigue, fever, and weight loss) along with evidence of consolidation on CT-scan, did not allow for the definitive exclusion of lung cancer, despite the negative functional imaging studies. For this reason, a cytological examination had to be warranted. Our case highlights an atypical presentation of eosinophilic pneumonia with a prolonged course and initial misinterpretation as a possible malignancy. Despite the radiological suspicion, especially due to nodular appearance and delayed onset symptoms, the final diagnosis was confirmed via cytology showing eosinophilic infiltration. The combination of peripheral eosinophilia and the eosinophilic infiltration of the lung parenchyma strengthened the suspicion of EP and should help to distinguish it from malignancy and other infectious causes of pulmonary consolidation. The rapid and complete response to corticosteroid therapy validates the diagnosis of PE. While the radiological features were atypical for classic CEP or AEP, the clinical context and multimodality diagnostic evaluation developed a key role for appropriate diagnosis and management.

TEACHING POINTS

An initial finding of a solitary pulmonary nodule, followed by negative tumor markers and no uptake on subsequent PET imaging scans, should prompt consideration of alternative diagnoses such as infectious or autoimmune pneumonias.

A complete assessment of the patient's medical history and comorbidities along with close follow-up are crucial to guide and confirm the correct diagnosis between the alternative diagnostic hypotheses.

CONCLUSIONS

Eosinophilic pneumonia may present with heterogeneous and potentially misleading clinical and radiological features. Although it accounts for only a small percentage of pneumonia cases (approximately 3%), it should be considered as a possible diagnostic hypothesis, especially in younger patients, even in the absence of symptoms and with atypical , such as peripheral

eosinophilia, radiographic findings. In cases where clinical suspicion is high, diagnostic confirmation with bronchoalveolar lavage (BAL) and/or lung biopsy should be performed.

QUESTIONS

Question 1: Which of the following radiological findings is most typical of Chronic Eosinophilic Pneumonia (CEP) ?

a) Diffuse pleural effusion middle region
b) Peripheral upper/middle region lung consolidations ("photographic negative of pulmonary edema")
c) Diffuse bilateral reticular densities
d) Smooth interlobular septal thickening
e) Reverse halo ("atoll") sign

The most typical radiological findinig is Peripheral upper/mid lung consolidations ("photographic negative of pulmonary edema").

Question 2: In this clinical case, which factor most strongly contributed to the decision to perform pulmonary cytology, despite initially negative functional imaging studies for malignancy?

a) The patient's history of allergic rhinitis
b) The patient's age
c) The presence of a previous nodule and new consolidations with irregular margins in contact with the pleura
d) The high peripheral blood eosinophil count
e) Complete clinical resolution after empiric antibiotic therapy

In this case the presence of a previous nodule and new consolidations with irregular margins in contact with pleura lead us to perform pulmonary cytology.

Question 3: What is the most distinguishing characteristic that differentiates AEP from CEP in most cases?

a) The presence history of asthma
b) The response to corticosteroids
c) The rapid onset of symptoms (< 1 week)
d) The predominance in middle-aged women
e) The common relapse after treatment

AEP is characteristic for its rapid onset of symptoms (<1 week)

Question 4: A common finding on HRCT in eosinophilic pneumonia often adjacent to consolidations, is:

a) Calcified nodules
b) Cavitations
c) Ground-glass opacities
d) Significant traction bronchiectasis
e) Diffuse alveolar infiltrates

A very common finding on HRCT in eosinophilic pneumonia is ground-glass opacity.

Question 5: The described "hilar and pleural continuity/attachment" within the lesions in this case report, in the context of a pulmonary lesion, should primarily prompt consideration of which differential diagnosis?

a) Allergic bronchopulmonary aspergillosis (ABPA)
b) Community-acquired pneumonia (CAP)
c) Lung malignancy
d) Drug-induced pulmonary eosinophilia
e) Eosinophilic granulomatosis with polyangiitis (EGPA)

Hilar and pleural continuity or attachment should primarily prompt consideration of lung cancer.

REFERENCES

[1] Cottin V. Eosinophilic Lung Disease. *Clin Chest Med.* 2016; Sep 37(3): 535-556. PMID: 27514599.

[2] Jeong YJ, Kim KI, Seo IJ, et al. Eosinophilic lung diseases: a clinical, radiologic, and pathologic overview. *Radiographics.* 2007; 27(3): 617-637. PMID: 17495282.

[3] Choi YH, Im JG, Han BK, Kim JH, Lee KY, Myoung NH. Thoracic manifestation of Churg-Strauss syndrome: radiologic and clinical findings. *Chest.* 2000; 117(1): 117-124. PMID: 10631208.

[4] G. P. Patel G. Allergic bronchopulmonary aspergillosis. *Allergy Asthma Proc.* 2019; 40(6): 421-424. PMID: 31690385.

[5] Suzuki Y, Suda T. Eosinophilic pneumonia: A review of the previous literature, causes, diagnosis, and management. *Allergol Int.* 2019; 68(4): 413-419. PMID: 31253537.

[6] M. T. N. Y. M. T. Nakajima M. Cigarette smoke-induced acute eosinophilic pneumonia. *Radiology* . 1998; 207(3): 829-831. PMID: 9609914.

[7] Kamada T, Yamashita Y, Tomioka H. Acute eosinophilic pneumonia following heat-not-burn cigarette smoking. *Respirol Case Rep.* 2016; 4(6): e00190. PMID: 28031826.

[8] F. M. Y. M. U. K. K. S. N. M. M. T. K. M. Shintani H. Acute eosinophilic pneumonia caused by cigarette smoking. *Intern Med.* 2000; 39(1): 66-68. PMID: 10674853.

[9] Philit F, Etienne-Mastroïanni B, Parrot A, Guérin C, Robert D, Cordier JF. Idiopathic acute eosinophilic pneumonia: a study of 22 patients. *Am J Respir Crit Care Med.* 2002; 166(9): 1235-1239. PMID: 12403693.

[10] Marchand E, Etienne-Mastroianni B, Chanez P, et al. Pulmonaires. Idiopathic chronic eosinophilic pneumonia and asthma: how do they influence each other?. *Eur Respir J.* 2003; 22(1): 8-13; PMID: 12882444.

[11] Johkoh T, Müller NL, Akira M, et al. Eosinophilic lung diseases: diagnostic accuracy of thin-section CT in 111 patients. *Radiology,* 2000; 216(3): 773-780. PMID: 10966710.

[12] Gaensler EA, Carrington CB. Peripheral opacities in chronic eosinophilic pneumonia: the photographic negative of pulmonary edema. *AJR Am J Roentgenol.* 1977; 128(1): 1-13. PMID: 401562.

[13] bone RG, Puppo F, Mattar E, Roden AC, Hirani N. Acute and chronic eosinophilic pneumonia: an overview. *Front Med (Lausanne).* 2024; 11: 1355247; PMID: 38711783.

[14] W. M. Allen J. Eosinophilic Pneumonias. *Journal of Allergy and Clinical Immunology: In Practice;* 2018; PMID: 29735405.

[15] W. ME. Pulmonary eosinophilic syndromes. *Immunol Allergy Clin North Am.* 2007; 27(3): 477-492. PMID: 17868860.

[16] G. M. F. N. C. P. Bonniaud P. Pneumopathies interstitielles iatrogéniques [Drug-induced interstitial lung diseases]. *Rev Prat.* 2014; 64(7): 951-956. PMID: 25362778.

FIGURES

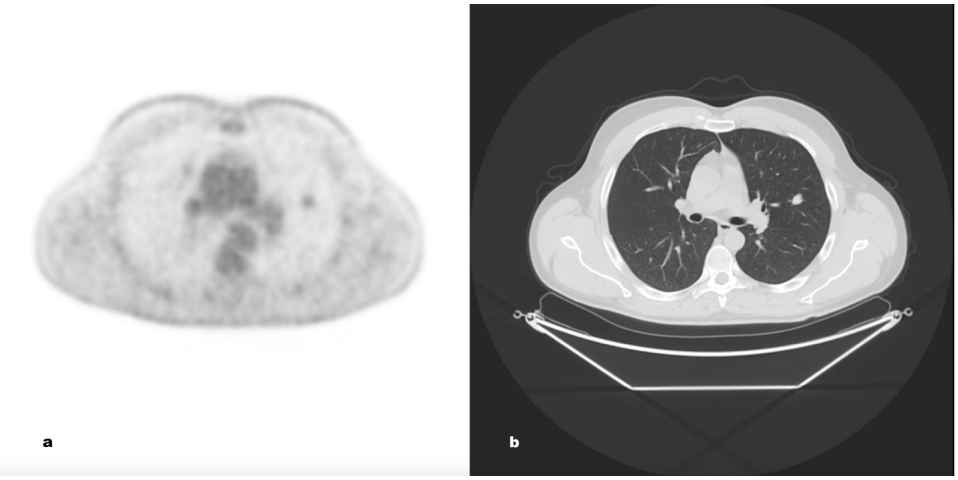

Figure 1: Axial thin-section 18F-FDG PET/CT (1a: PET image; 1b: CT lung window shows a nodular lesion measuring up to 11 mm in the lingular region demonstrating a low and not significant uptake of 18F-FDG. (SUVmax 1.1)

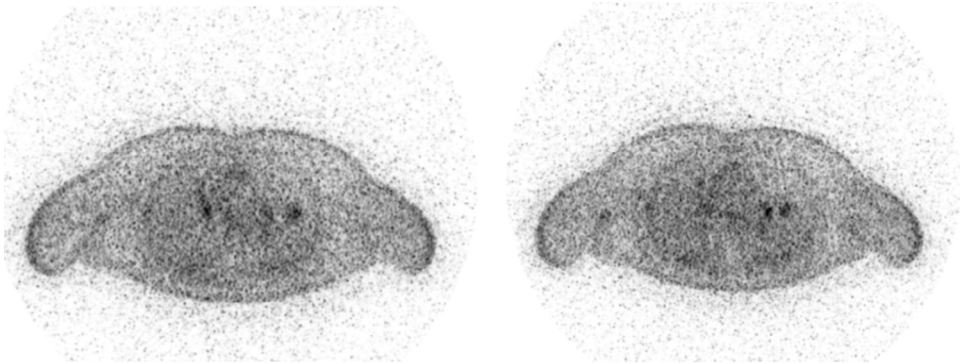

Figure 2: Axial slice from a 68Gallium-DOTATOC PET demonstrates no significant tracer uptake corresponding to known lingular lesion, effectively excluding a neuroendocrine tumor.

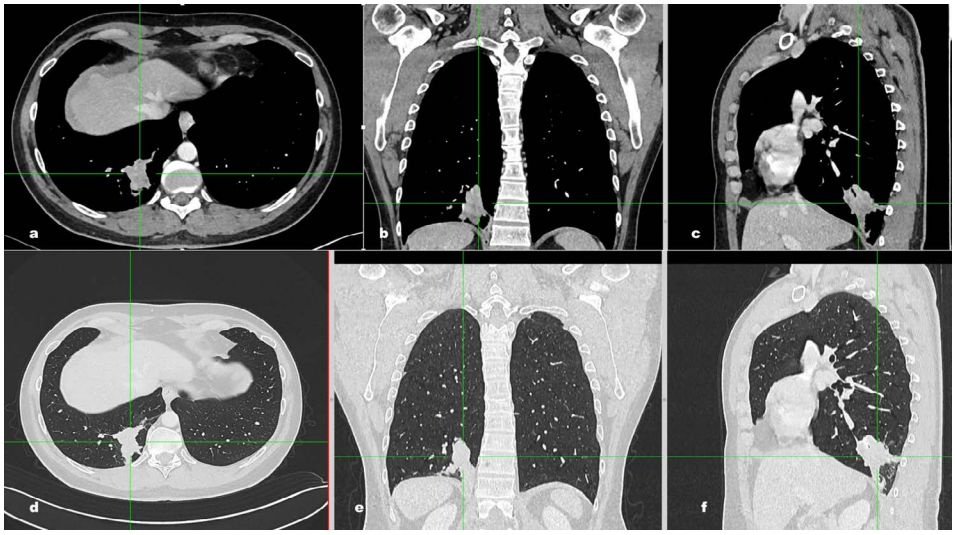

Figure 3: (3a-f) Axial (3a,3d), coronal (3b,3e) and sagittal (3c,3f) thin-section contrast-enhanced CT scan in portal venous phase (3a-c: mediastinal windowing; 3d-f: lung windowing) show a parenchymal consolidation measuring approximately 60x40x40 mm in the posterior segment of the right lower lobe. This lesion is characterized by irregular margins with strands extending to the hilar region and adjacent pleural plane.

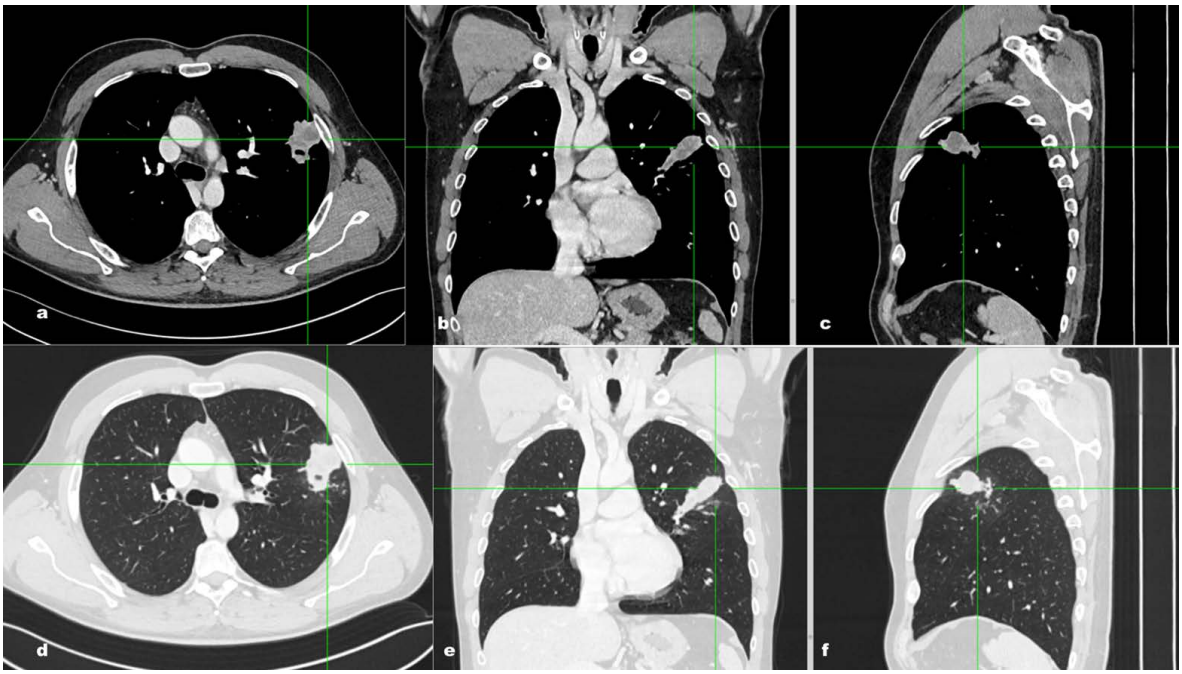

Figure 4: (4a-f) Axial (4a,4d), coronal(4b,4e) and sagittal (4c,4f) thin-section contrast-enhanced CT scan in portal venous phase (4a-4c: mediastinal windowing and lung windowing) shows a parenchymal consolidation measuring 38x44x48 mm in the superior lingular segment of the left upper lobe. This lesion is characterized by irregular margins with strands extending to the hilar region and adjacent pleural plane, it also demonstrates inhomogeneous density due to the presence of colliquative areas.

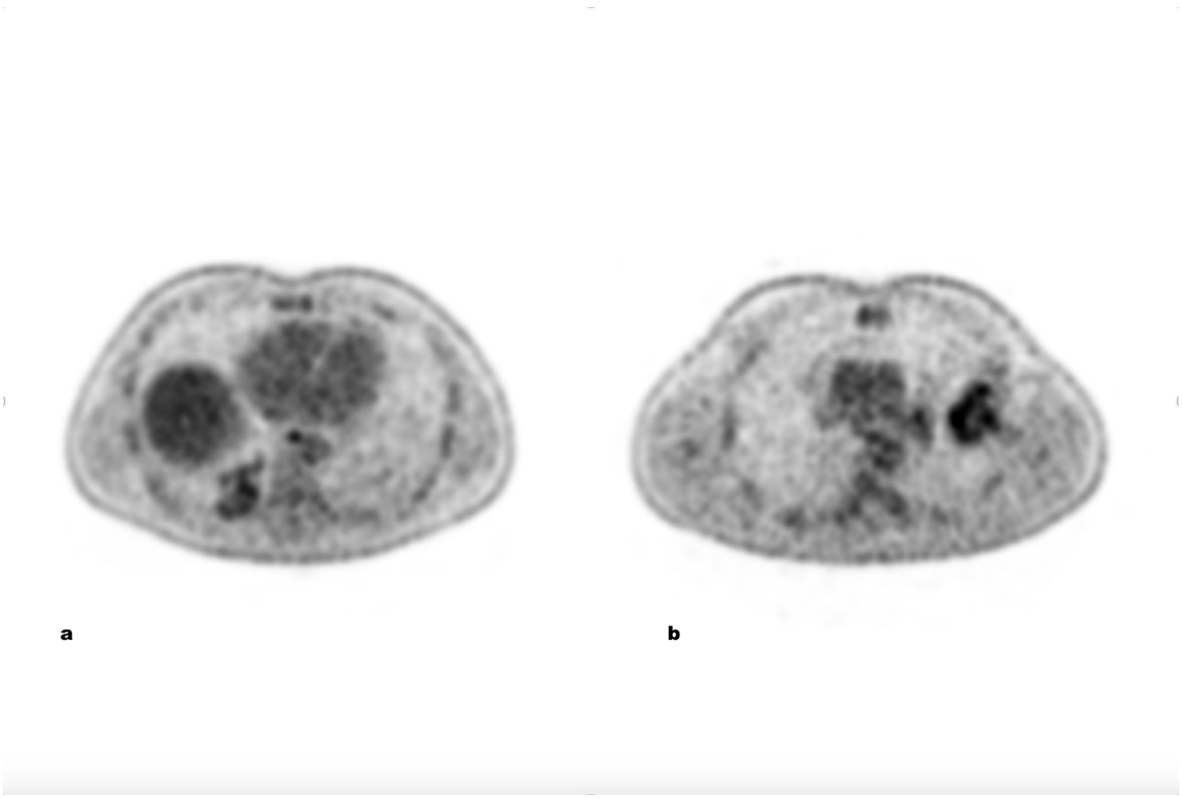

Figure 5: (5a-5b) Axial slice from 18F-FDG-PET/CT scan showed mild uptake on the superior lingular segment of the left upper lobe (SUV max 5.4) (b) and on the posterior segment of the right lower lobe too (SUV max 3.5) (a).

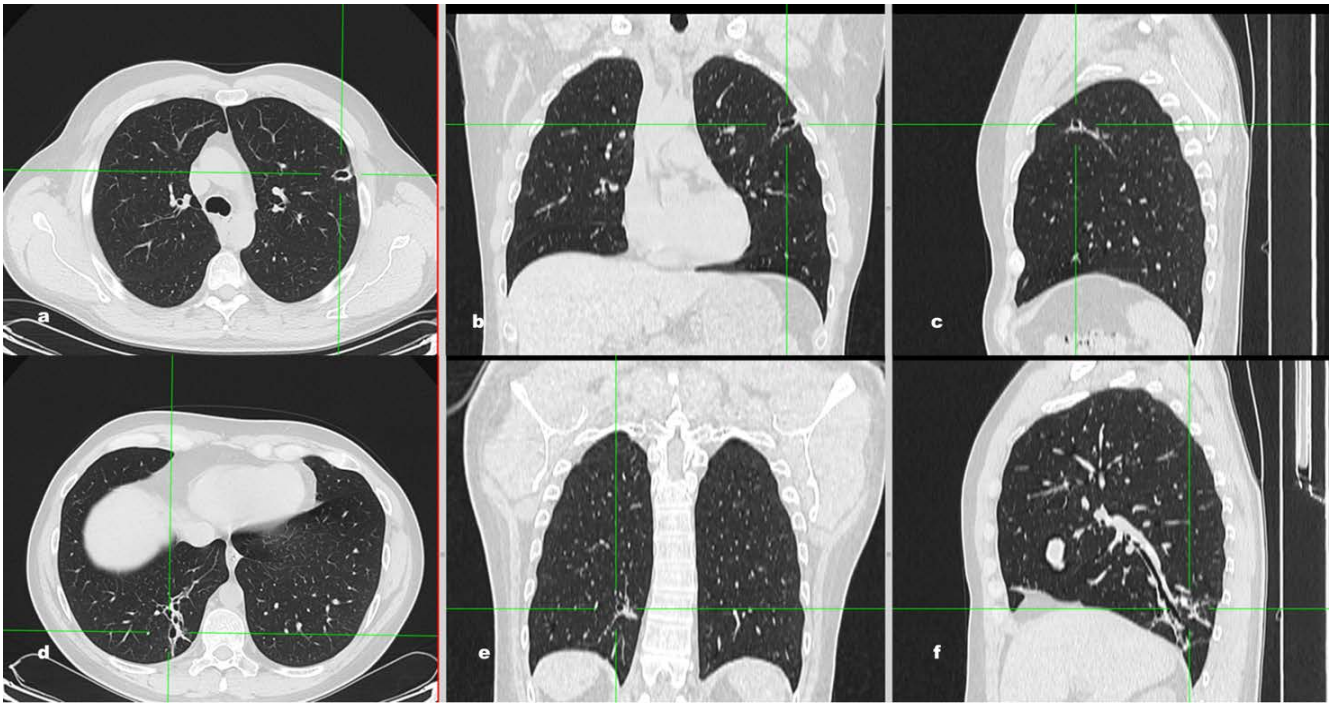

Figure 6: (6a-f) One-month radiological follow-up with axial (6a,6d), coronal (6b,6e) l and sagittal (6c,6f) thin-section CT scan (lung windowing) after pharmacological therapy. The consolidations on the superior lingular segment of the left upper lobe and on the posterior segment of the right lower lobe have disappeared. In both regions some fibrotic changes characterized by small traction bronchiectasis remain.

KEYWORDS

Eosinophilic pneumonia, chronic eosinophilic pneumonia, acute eosinophilic pneumonia, lung cancer, eosinophilia, BAL, photographic negative of pulmonary edema.

ABBREVIATIONS

EP = Eosinophilic Pneumonia
18F-FDG PET/CT = 18-Fluorodeoxyglucose positron emission tomography- computed tomography
CAP = Community-Acquired Pneumonia
ABPA = Allergic Bronchopulmonary Aspergillosis
EGPA = Eosinophilic Granulomatosis With Polyangiitis
AEP = Acute Eosinophilic Pneumonia
CEP = Chronic Eosinophilic Pneumonia
BAL = Bronchoalveolar Lavage
ARDS = Acute Respiratory Distress Syndrome
HRCT = High-Resolution Computed Tomography

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