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CASE REPORT

CT-Guided Percutaneous Lung Biopsy in the Diagnosis of Asthma-Associated Chronic Eosinophilic Pneumonia: A Case Report

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Background: Asthma-associated eosinophilic pneumonia (EP) is a relatively uncommon clinical condition characterized by abnormal accumulation of eosinophils in the lung parenchyma or alveolar spaces. Computed tomography (CT)-guided percutaneous lung biopsy, a minimally invasive diagnostic technique, demonstrates a high diagnostic yield for pulmonary diseases. This method offers several advantages, including minimal tissue trauma, procedural simplicity, high positive rate, and an acceptable risk profile for complications, establishing it as a reliable tool for diagnosing both infectious and neoplastic pulmonary conditions. However, the diagnosis of EP remains challenging due to its non-specific clinical presentation and atypical imaging features, often leading to clinical misdiagnosis as pulmonary infection, tuberculosis, or lung cancer. Elevated eosinophil counts in peripheral blood and bronchoalveolar lavage fluid (BALF) provide crucial diagnostic clues. A BALF eosinophil proportion ≥25% is considered highly suggestive of the diagnosis. Nevertheless, when the BALF eosinophil count falls below the diagnostic threshold for EP, lung biopsy serves as a valuable alternative for achieving a definitive diagnosis and facilitating differential diagnosis.

Case: We report a 18-year-old female patient with a history of asthma who was initially diagnosed with pulmonary infection at another hospital. Despite empirical treatment with multiple antimicrobial agents, her condition progressed. Bronchoalveolar lavage revealed an eosinophil percentage of 20% in BALF, which was slightly below the standard diagnostic threshold of 25%. Given the strong clinical suspicion and the subthreshold BALF result, a CT-guided percutaneous lung biopsy was performed at our institution, which confirmed the diagnosis of chronic eosinophilic pneumonia (CEP).

Results: Following the diagnosis of chronic eosinophilic pneumonia, glucocorticoid therapy was initiated. A follow-up chest CT scan at 7 months revealed complete resolution of the pulmonary infiltrates, which was accompanied by the normalization of peripheral blood eosinophil counts. The patient remained disease-free without recurrence until the last follow-up in May 2025.

Conclusion: In asthmatic patients presenting with elevated peripheral blood eosinophils and pulmonary opacities with a predominant peripheral distribution on imaging, secondary eosinophilic pneumonia should be considered. When the bronchoalveolar lavage (BAL) fluid eosinophil count falls slightly below the diagnostic threshold (eg, 20% as in our case), Lung biopsy should be considered to establish a definitive diagnosis, which is critical for guiding subsequent patient management and improving outcomes.

Keywords: CT guidance, percutaneous lung biopsy, asthma, alveolar lavage fluid, Eosinophilic pneumonia

Introduction

Eosinophilic pneumonia (EP) is a pulmonary disorder characterized by the abnormal accumulation of eosinophils within the lung parenchyma or alveolar spaces, typically without significant destruction of the lung architecture. Eosinophilic lung diseases can manifest as either acute or chronic eosinophilic pneumonia. Chronic Eosinophilic Pneumonia (CEP), which can occur at any age and affects both sexes, demonstrates a female predominance. As a rare disease, the estimated annual incidence of CEP is likely less than 1 per 100,000 individuals.[1] Common etiological associations of CEP include bronchial asthma and allergic rhinitis, with studies indicating that a history of asthma is present in 33.3% to 61.4% of CEP patients.[2] CEP is frequently linked to Th2-mediated conditions like asthma, and its pathogenesis involves the activation of Th2 lymphocytes and subsequent eosinophil recruitment driven by cytokines such as IL-5. On CT scans,

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Acute Eosinophilic Pneumonia (AEP) typically presents with bilateral ground-glass opacities, consolidations, interlobular septal thickening, bronchial wall thickening, and pleural effusions. In contrast, the imaging findings of CEP are often more distinctive, frequently demonstrating diffuse peripheral opacities and poorly defined shadows with a predominantly peripheral distribution in the lungs. However, these classic radiographic features of CEP are not invariably present and can overlap with other pulmonary pathologies, frequently leading to misdiagnosis. The diagnosis of eosinophilic pneumonia is primarily based on a combination of clinical presentation, imaging features, and peripheral blood eosinophilia. A BALF eosinophil count of ≥25% holds significant diagnostic value. Nevertheless, in clinical practice, variability in eosinophil count measurements may occur, resulting in some confirmed patients presenting with values below this threshold.

CT-guided percutaneous transthoracic needle biopsy (PTNB) is a minimally invasive diagnostic technique with a high diagnostic yield for various pulmonary conditions, including infectious diseases, neoplasms, and rare lung disorders. In clinical practice, a diagnostic dilemma arises when a patient is highly suspected of having CEP, yet the BALF eosinophil count—such as 20% in the present case—falls below the conventional diagnostic threshold. In such scenarios, histological confirmation often becomes crucial to establish a definitive diagnosis and prevent delays in treatment. CT-guided PTNB can provide clear diagnostic evidence for such atypical cases. We herein report a case of asthma-associated CEP confirmed by CT-guided lung biopsy. This report aims to enhance clinicians' recognition of the atypical presentations of this disease and to delineate a diagnostic pathway when BALF results are inconclusive.

Case Presentation

Clinical Data

General Clinical Data

An 18-year-old female patient, a high school student, presented to the hospital on April 13, 2023, due to persistent cough, sputum production, chest tightness, and shortness of breath lasting over a month. Her symptoms began after catching a cold more than a month prior. Initially, her cough was intermittent, producing white, viscous, and odorless sputum. She occasionally felt febrile but did not measure her temperature. She did not experience chills, sore throat, nasal congestion, rhinorrhea, chest pain, bloody sputum, hemoptysis, nausea, acid regurgitation, epigastric burning discomfort, fatigue, anorexia, night sweats, dizziness, headache, or any other discomfort. She visited a local hospital and was diagnosed with a lung infection. Despite receiving cephalosporin and moxifloxacin anti-infection treatment for two weeks, her condition worsened. Her cough, sputum production, chest tightness, and shortness of breath intensified. Chest CT scans revealed progressive lung lesions, prompting her referral to our hospital for further diagnosis and treatment. She was admitted to our department with a diagnosis of lung infection.

Since the onset of her illness, she has exhibited a poor spirit but maintained a fair appetite and sleep quality. Her urine and stool were normal, and there were no significant changes in her weight. She denied any history of hypertension, diabetes, heart disease, chronic kidney disease, hepatitis, typhoid, other infectious diseases, surgical trauma, blood transfusion, food and drug allergies. Upon systematic review, there were no notable findings in her personal, menstrual, or family history.

Physical and Auxiliary Examination Findings: Temperature

37.7 °C, Pulse: 143 beats/min, Respiration: 23 breaths/min, Blood Pressure: 108/73 mmHg, SpO2: 91% (without oxygen), Weight: 40 kg. The patient presents with normal development, adequate nutrition, a clear state of mind, and chronic illness. There is no enlargement of superficial lymph nodes, no congestion or swelling in the pharynx, and the tonsils are not swollen. Slight cyanosis is noted on the lips. The neck is supple, the trachea is centrally positioned, and there is no jugular vein distension. The liver jugular vein reflux sign is negative, and the thyroid gland is not enlarged. The chest is symmetrical without any deformities, respiratory movements are consistent on both sides, and the intercostal spaces are normal. Bilateral speech tremor is pronounced, but there is no pleural friction. The relative dullness of heart percussion is not enlarged, the heart rate is 143 beats/min with a regular rhythm, and no pathological murmurs are auscultated in the valve areas. The abdomen is flat, without varicose veins on the abdominal wall, tenderness, rebound pain, or muscular tension. Limb muscle strength and tone are normal. Physiological reflexes are present, and pathological

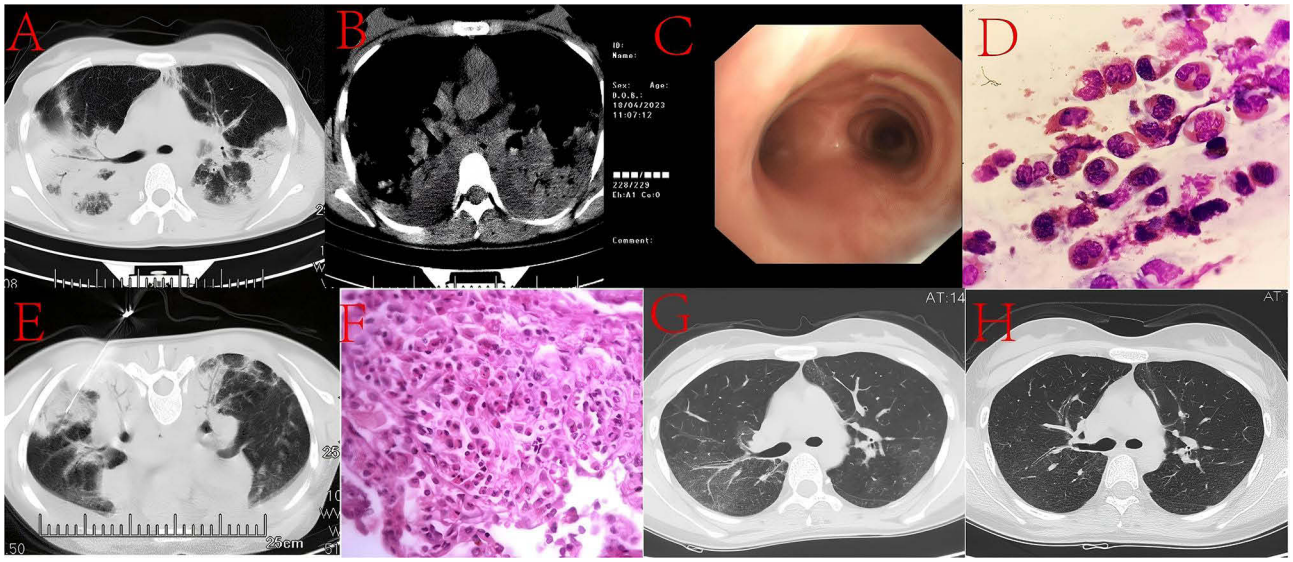

Figure 1 Diagnostic and therapeutic course of eosinophilic pneumonia. (**A** and **B**) Non-contrast chest CT (April 13, 2023) revealed multiple patchy consolidations with heterogeneous density and ill-defined margins in both lungs, predominantly in the right upper lobe and left lower lobe. (**C**) Bronchoscopy (April 18, 2023) showed mild mucosal hyperemia in the trachea and bronchi. (**D**) Bronchoalveolar lavage (BAL) fluid analysis demonstrated a markedly elevated eosinophil count (20%). (**E**) CT-guided percutaneous lung biopsy (April 21, 2023) was performed on the left lower lobe lesion. (**F**) Histopathological examination of the biopsy specimen revealed abundant eosinophils within alveolar spaces, confirming eosinophilic pneumonia. (**G** and **H**) Follow-up chest CT scans (June 13 and September 6, 2023) showed significant resolution (**G**) and eventual complete disappearance (**H**) of pulmonary lesions.

reflexes are absent. Chest CT reveals patchy dense shadows predominantly distributed in both lungs, with uneven density and blurred boundaries, especially in the upper lobe of the right lung and the lower lobe of the left lung. These findings suggest infectious lesions in both lungs and bilateral pleural effusion. It is recommended to exclude tuberculosis and other potential diseases after anti-infection treatment and reevaluation (Figure 1A and B). Results of complete blood count, liver and renal function tests, erythrocyte sedimentation rate, fungal serology, C-reactive protein, and coagulation profile are summarized in Table 1.

Table 1 Laboratory Findings: Complete Blood Count, Coagulation Profile, Liver and Renal Function, CRP and ESR

Test Item	Result	Reference Range	Status ↑ Elevated ↓ Decreased -Ormal
White Blood Cell (WBC) Count	14.23 × 10^9/L	3.5–9.5	↑
Red Blood Cell (RBC) Count	4.34 × 10^{12}/L	3.8–5.1	-
Neutrophils (%)	72%	40 – 75	-
Hemoglobin (HGB)	112 g/L	115 – 150	↓
Platelet (PLT) Count	597 × 10^9/L	125 – 350	↑
Eosinophils (%)	15%	0.4–8.0	↑
Eosinophil Count	2.32 × 10^9/L	0.02–0.52 ×10^9/L	↑
Prealbumin	105.00 mg/L	200 – 400	↓
Total Protein	62.69 g/L	65 – 85	↓
Albumin	29.20 g/L	40 – 55	↓
Urea	3.09 mmol/L	1.7–8.3	-
Uric Acid	399.00 μmol/L	142 – 340	↑
Creatinine	44.10 μmol/L	70 – 115	↓
Alanine Aminotransferase (ALT)	10.00 U/L	9 – 50	-
Aspartate Aminotransferase (AST)	20.00 U/L	15 – 40	-
C-Reactive Protein (CRP)	50.68 mg/L	≤ 10	↑

(*Continued*)

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Table 1 (Continued).

Test Item	Result	Reference Range	Status ↑ Elevated ↓ Decreased -Ormal
Fibrin Degradation Products (FDP)	12.6 μg/mL	0 – 5	↑
D-Dimer	3.35 μg/mL	0 – 0.5	↑
Fibrinogen	4.83 g/L	2 – 4	↑
Erythrocyte Sedimentation Rate (ESR)	120 mm/h	0 – 15	↑
1-3-β-D-Glucan	53.27 pg/mL	0 – 70	-
Galactomannan (Aspergillus Antigen)	0.21	< 0.5	-

Diagnosis and Treatment Process

Following admission, patients underwent symptomatic treatment consisting of oxygen inhalation, amoxicillin clavulanate potassium for anti-infection, doxofylline for bronchodilation, ambroxol for expectoration, and supplemental nutrition, fluids, and electrolytes. Further investigations were performed following admission. Urinalysis and stool routine tests were unremarkable. Sputum acid-fast staining was negative for mycobacteria, and routine bacterial culture of sputum revealed normal flora. Thyroid function tests (five items), a panel of five vasculitis-related antibodies, and a 13-item antinuclear antibody profile were all within normal limits. Abdominal ultrasonography of the liver, gallbladder, pancreas, spleen, and kidneys showed no abnormalities. An electrocardiogram indicated sinus tachycardia. Echocardiography demonstrated mild tricuspid regurgitation with a normal left ventricular ejection fraction. Pulmonary function tests revealed severe mixed ventilation dysfunction with VC MAX 69.9%, FEV1%VC MAX 76.6%, and FEV1 47.7%. The bronchodilation test was positive. Testing for common respiratory viruses and a panel of common parasitic antibodies were performed, and the results are summarized in Table 2. Collectively, the above workup effectively ruled out pulmonary damage secondary to vasculitis or connective tissue diseases. It also excluded COVID-19, pneumonia caused by common influenza viruses, and pulmonary lesions attributable to common parasitic infections.

Table 2 Detection of Common Respiratory Viruses, Serological Tests for Parasitic Antibodies, and Pathogen Nucleic Acids in Bronchoalveolar Lavage Fluid

Test Item	Positive (+) Negative (-)	Test Item	Positive (+) Negative (-)
Influenza A Virus RNA	-	Acid-Fast Stain	-
Influenza B Virus RNA	-	*Streptococcus pneumoniae* DNA	-
Respiratory Syncytial Virus RNA	-	*Staphylococcus aureus* DNA	-
Adenovirus DNA	-	Methicillin-Resistant *Staphylococcus aureus* (MRSA) DNA	-
Human Rhinovirus RNA	-	*Escherichia coli* DNA	-
SARS-CoV-2 (COVID-19)	-	*Klebsiella pneumoniae* DNA	-
Cysticercus Antibody	-	*Pseudomonas aeruginosa* DNA	-
Sparganum Antibody	-	*Acinetobacter baumannii* DNA	-
Toxoplasma gondii Antibody	-	*Stenotrophomonas maltophilia* DNA	-
Echinococcus Antibody	-	*Haemophilus influenzae* DNA	-
Clonorchis sinensis (Liver Fluke) Antibody	-	*Legionella pneumophila* DNA	-
Paragonimus (Lung Fluke) Antibody	-	*Mycoplasma pneumoniae* DNA	-
Schistosoma japonicum Antibody	-	*Chlamydia pneumoniae* DNA	-
Angiostrongylus cantonensis Antibody	-	Non-tuberculous Mycobacteria DNA	-
Cysticercus Antibody	-	*Legionella pneumophila* DNA	-
Sparganum Antibody	-	*Mycobacterium tuberculosis* Complex DNA & RNA	-

The electronic bronchoscope examination was conducted flawlessly, revealing slight congestion of the trachea and bronchial mucosa (Figure 1C). Neither new organisms nor any obstruction were detected within the trachea and bronchial lumen. An alveolar lavage and cell brush examination were carried out in the posterior basal segment, located outside the lower lobe of the right lung. Microbiological analysis of bronchoscopic specimens revealed a negative acid-fast stain of the brush sample. Routine bacterial culture of the BAL fluid indicated the presence of normal respiratory flora. Additionally, nucleic acid testing for a panel of specific pathogens in the BAL fluid was performed, and the results are summarized in Table 2. Collectively, these findings from the bronchoscopic procedures effectively ruled out active pulmonary infection caused by common bacterial pathogens.

However, the surprising finding was the presence of a significant number of eosinophils in the classification and counting of alveolar lavage fluid cells, accounting for 20% (Figure 1D). When combined with the notable increase in eosinophils observed in the patient's routine blood tests, a diagnosis of eosinophilic pneumonia for the lung lesions becomes plausible. A repeat bronchoscopy with BAL for eosinophil count was advised; however, the patient and her family declined the procedure. To establish a definitive diagnosis and exclude conditions such as tuberculosis, organizing pneumonia, and other rare causes of eosinophilia, including eosinophilic granulomatosis polyangiitis (EGPA), a contrast-enhanced chest CT was performed, followed by a CT-guided percutaneous lung biopsy of the left lower lobe lesion on April 21, 2023 (Figure 1E). Histopathological examination of the biopsy specimen revealed abundant eosinophils within the alveolar spaces, confirming the diagnosis of eosinophilic pneumonia (Figure 1F). Based on the confirmed diagnosis, the patient was initiated on oral prednisone acetate 30 mg once daily. Following treatment, her symptoms of cough, chest tightness, and dyspnea significantly improved. A follow-up chest CT on June 13, 2023, demonstrated marked resolution of the pulmonary infiltrates (Figure 1G), accompanied by normalization of the peripheral blood eosinophil count. Consequently, the prednisone dose was tapered to 25 mg once daily. The steroid dose was subsequently reduced by 5 mg per month. By September 6, 2023, a repeat chest CT showed complete resolution of the lung lesions (Figure 1H) with a sustained normal eosinophil count. Prednisone was further tapered to 5 mg once daily and was eventually discontinued in November 2023. Subsequently, the patient was maintained on salmeterol/fluticasone inhalation for asthma control.

Informed consent was obtained from the patient for publication of this case report and accompanying images.

Discussion

Eosinophilic pneumonia (EP) is a lung disease marked by significant eosinophil infiltration in the airway, lung parenchyma, lung interstitium, and alveolar space. While eosinophils in peripheral blood may be elevated or normal.[3] Although the precise pathogenesis of EP remains incompletely understood, current evidence indicates that its central pathophysiological basis is the abnormal accumulation and activation of eosinophils within the lungs. The Th2 immune response is recognized as a key driver in this process. Notably, IL-5, a canonical cytokine intimately associated with eosinophilic inflammation, has garnered significant attention for its pivotal role in initiating and promoting eosinophil survival.[4]

EP is a rare pulmonary disorder characterized by eosinophilic infiltration of the lung parenchyma, which may arise from identifiable triggers or occur idiopathically. Currently, there is no universally accepted classification system for EP. Based on the presence of identifiable underlying causes, some scholars categorize it into primary (idiopathic) and secondary forms.[5] Clinically, EP is classified by disease tempo into: Acute eosinophilic pneumonia (AEP), CEP. Commonly identified triggers for AEP include Löffler's syndrome, drug reactions, and parasitic, fungal, bacterial, or viral infections. In contrast, asthma and allergic rhinitis represent the most frequently associated conditions in patients with CEP.[6]

Clinical Manifestations and Radiographic Features EP exhibits nonspecific clinical presentations, most commonly including cough, sputum production, fever, and chest tightness. Acute exacerbations may progress to dyspnea with respiratory failure, while pleuritic chest pain, bloody sputum, and hemoptysis are rarely observed. Characteristic CT findings: AEP: Ground-glass opacities, consolidations, interlobular septal thickening, bronchial wall thickening, and pleural effusions. On CT AEP typically manifests as bilateral ground-glass opacities, consolidations, interlobular septal thickening, bronchial wall thickening, and pleural effusions. Characteristic radiographic features of CEP typically include

diffuse peripheral opacities and poorly defined shadows with a predominantly peripheral distribution in the lungs.[7] Due to the nonspecific clinical presentations and imaging findings of eosinophilic pneumonia, it is frequently misdiagnosed clinically as lung cancer, lung abscess, or military tuberculosis.[8–11] Furthermore, cases of cryptogenic organizing pneumonia (COP) coexisting with eosinophilic pneumonia have also been documented in the literature.[12]

Diagnosis of EP requires integration of characteristic clinico-radiological features with BALF eosinophilia. Persistent peripheral blood eosinophilia often serves as the primary diagnostic clue for EP. Key diagnostic thresholds: Peripheral blood eosinophils $>1\times10^9$/L (particularly $>1.5\times10^9$/L) provides substantial diagnostic support, BALF eosinophil proportion ≥25% is widely adopted as the diagnostic threshold, BALF eosinophil count ≥40% may serve as a more conservative diagnostic criterion. Diagnostic workflow: When elevated blood eosinophils (even mildly) coexist with compatible radiographic findings, EP should be suspected, prompting BALF eosinophil quantification. In cases with borderline eosinophil elevations but high clinical suspicion, histopathological confirmation via lung biopsy remains definitive.[13] The hallmark pathological manifestation of EP is characterized by extensive eosinophilic infiltration in the pulmonary interstitium and alveolar spaces. Histopathological examination typically demonstrates: Acute and organizing diffuse alveolar damage, Alveolar and interstitial edema, Eosinophilic infiltration involving the interstitium-alveoli interface and bronchiolar walls.[14]

When BALF eosinophil counts fall below the conventional diagnostic threshold (25%), yet clinical suspicion for eosinophilic pneumonia remains, several diagnostic strategies may be considered. These include performing a repeat BAL procedure, initiating empiric corticosteroid therapy, adjusting antimicrobial therapy with re-evaluation, or proceeding to lung biopsy for definitive histopathological diagnosis. Each approach carries distinct advantages and limitations. A repeat bronchoscopy entails procedure-related risks, including hemorrhage, sore throat, hypoxemia, exacerbated cough, hemoptysis, and potential induction of an asthma attack. Furthermore, the repeat BALF eosinophil count might still not reach the 25% threshold, leaving the diagnosis unresolved. In the present case, the initial BALF eosinophil count of 20% was determined manually, introducing the possibility of counting variability and discrepancy from the actual value.

Empiric corticosteroid therapy poses risks of treatment failure, potentially leading to clinical deterioration, alongside steroid-related adverse effects such as peptic ulcer disease, osteoporosis, hyperglycemia, Cushing's syndrome, and fluid retention. Young adults and children face the added risk of avascular necrosis of the femoral head. Moreover, in patients with undiagnosed tuberculosis, steroid use may precipitate disease dissemination, particularly as pulmonary tuberculosis itself can occasionally be associated with eosinophilia.[11] Adjusting or intensifying antimicrobial regimens carries the risk of drug-related side effects and potential delay in correct diagnosis, allowing disease progression. Lung biopsy, while diagnostic, is associated with procedural complications such as pneumothorax, hemoptysis, pleural reaction, and air embolism. After thorough discussion of the risks and benefits of all available options with the patient and her family, informed consent was obtained to proceed with a CT-guided percutaneous lung biopsy. This approach ultimately provided the definitive histopathological confirmation required for diagnosis.

CT-guided percutaneous lung biopsy is a minimally invasive interventional diagnostic technique that frequently plays a pivotal role in the diagnosis and differential diagnosis of diseases. This is achieved by obtaining pathological examination of diseased tissue through puncture biopsy. The technique is characterized by its minimally invasive nature, straightforward operation, short procedure duration, high positive rate, and an acceptable complication rate. It has a long-standing history of clinical application and holds significant value in diagnosing both tumorous and infectious lesions. In a diagnostic study involving 1484 cases of paramediastinal and non-paramediastinal lung lesions, CT-guided percutaneous transthoracic needle aspiration demonstrated a high accuracy rate and an acceptable complication rate.[15]

The patient safely underwent a CT-guided percutaneous lung biopsy, experiencing no complications such as pneumothorax or hemoptysis. Histopathological examination confirmed significant eosinophil infiltration in both the alveolar cavity and interstitium, providing a definitive basis for the diagnosis of CEP. This decision-making pathway indicates that, when clinical presentation strongly suggests EP but BALF results do not meet diagnostic criteria, early biopsy can be an effective strategy. It is superior to diagnostic hormone therapy and repeated bronchoscopy, as it helps to prevent adverse outcomes stemming from misdiagnosis or delayed diagnosis. When differentiating CEP poses challenges, lung biopsy may play a crucial role.[16]

The primary therapeutic approach for eosinophilic pneumonia centers on the administration of glucocorticoids. Regardless of whether the condition is acute or chronic, initiating glucocorticoid therapy promptly following a definitive diagnosis typically leads to a favorable prognosis. Currently, the precise optimal dosage of glucocorticoids remains undetermined; however, treatment may commence with prednisone at a dose of 0.5 mg/kg/day, which can then be gradually tapered over a period of 6 to 12 months, guided by clinical assessments and blood eosinophil counts. The majority of patients require treatment extending beyond 6 to 12 months.[13] This particular patient underwent glucocorticoid therapy for 7 months and experienced a positive therapeutic outcome. Simultaneously, it is crucial to address the underlying cause of eosinophilic pneumonia when identifiable. For instance, if the condition is drug-induced, discontinuation of the relevant medication is necessary; antiparasitic treatment should be administered for parasitic infections, antifungal therapy for fungal infections, antiviral treatment for viral infections, and allergen avoidance for allergic reactions. In recent years, biological agents, such as mepolizumab and benralizumab, have emerged as promising alternatives for patients who are intolerant to hormones, experience frequent recurrences, are glucocorticoid-dependent, or have contraindications to glucocorticoids. Moreover, biological agents appear to offer a safer option for managing the recurrence of chronic eosinophilic pneumonia.[17]

Upon reviewing the diagnosis and treatment of this particular case, we observed that the patient exhibited elevated eosinophil levels in their peripheral blood, along with an increased proportion of eosinophils in alveolar lavage fluid. The final diagnosis was conclusively established through a CT-guided percutaneous lung biopsy. Upon delving into the patient's medical history, it was revealed that they had not utilized inhaled preparations to manage asthma in the months preceding this illness. This omission could potentially result in eosinophil infiltration into the lungs, leading to eosinophilic pneumonia. Notably, during this period, the patient did not experience wheezing symptoms; however, lung imaging examinations revealed large dense shadows in the lungs, which were initially misdiagnosed as infectious lesions. An analysis was conducted to understand why the patient had not used inhaled agents to control their asthma. It was found that patients without wheezing symptoms often mistakenly believed that asthma had not recurred and, consequently, discontinued their inhaled medications independently. This highlights the critical importance of health education for asthma patients to enhance their treatment compliance and ensure the standardized use of inhaled agents. Non-standardized treatment not only poses the risk of drug resistance but also increases the likelihood of secondary related diseases, such as eosinophilic pneumonia.

Conclusion

This case underscores that eosinophilic pneumonia is an uncommon lung condition. When a patient presents with a history of asthma, elevated eosinophil levels in peripheral blood, and chest imaging revealing patchy dense shadows distributed peripherally, chronic eosinophilic pneumonia should remain a consideration in the differential diagnosis, even if the eosinophil proportion in BALF falls below the standard threshold of 25%. CT-guided lung biopsy can serve as a diagnostic cornerstone for critical cases; however, further investigation is warranted to strike an optimal balance between the invasiveness, cost, and diagnostic accuracy of various methods. It is imperative to enhance health education for asthma patients to bolster their adherence to and understanding of the necessity for regular treatment. Simultaneously, the significance of standardized inhaler use for asthma patients must be underscored to effectively manage symptom onset. Inadequate control may prompt eosinophil infiltration into the lungs, potentially precipitating eosinophilic pneumonia.

Study Limitations

Being a single-center case report, this study faces certain constraints in terms of the generalizability of its findings. Firstly, the decision-making process in case management may be influenced by the inherent biases of retrospective analysis. Secondly, the choice of CT-guided percutaneous lung biopsy over repeated bronchoscopy or short-term empirical treatment for establishing the BALF cut-off (20%) was made without a precise quantitative assessment of its cost-effectiveness. Extended follow-up in future studies will be instrumental in assessing the long-term prognosis of patients and the practical value of biological agents in real-world settings. Furthermore, the diagnostic utility of CT-guided percutaneous lung biopsy for CEP in cases where eosinophils in alveolar lavage

fluid do not meet the diagnostic threshold (25%) remains to be validated through a multi-center study with a larger sample size.

Abbreviations

EP, eosinophilic pneumonia; CT, Computed tomography; BALF, bronchoalveolar lavage fluid, CEP, chronic eosinophilic pneumonia; AEP, Acute Eosinophilic Pneumonia; PTNB, percutaneous transthoracic needle biopsy; EGPA, eosinophilic granulomatosis polyangiitis; COP, cryptogenic organizing pneumonia.

Ethics and Consent

The publication of this case report was approved by the Institutional Ethics Committee of Guizhou Aerospace Hospital (Approval No. 2025-LW-11). Written informed consent for publication was obtained from the patient. The paper contains no identifiable patient data or images and adheres to the principles of the Declaration of Helsinki.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors confirm that there are no competing interests.

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