Diagnostic Pathology

CASE REPORT Open Access

Acute fibrinous and organising pneumonia presenting with mass-like imaging: a case report

Wei Zhou[1†], Longyun Zhang[1†], Bin Xu[2] and Chuanhai Wang[3*]

Abstract

Objective This case report describes a patient with acute fibrinous and organising pneumonia (AFOP) presenting with mass-like imaging on chest computed tomography (CT), aiming to enhance clinical awareness of this rare disease.

Case presentation A 66-year-old man presented with cough, sputum, chest tightness and weight loss persisting for 1 month. Chest X-ray revealed a space-occupying lesion in the left lung. Further CT imaging demonstrated irregular soft tissue masses in both the upper and lower lobes of the left lung. Although the imaging findings suggested lung cancer, the final pathological diagnosis confirmed AFOP. The patient was treated with methylprednisolone, resulting in substantial improvement of the upper lobe lesion, whereas the lower lobe lesion showed minimal response. Following the addition of mycophenolate mofetil, the lower lobe lesion decreased substantially. Multiple lung biopsies confirmed the diagnosis of AFOP, with no evidence of a malignant tumour.

Conclusions Acute fibrinous and organising pneumonia presents with non-specific imaging findings, and when manifesting as a mass-like lesion, it may be misdiagnosed as lung cancer. Pathological examination remains essential for diagnosis. Close monitoring of the clinical response is crucial during treatment, and the treatment plan should be tailored to individual patient needs.

Keywords Acute fibrinous and organising pneumonia, Mass, Computed tomography, Biopsy

Introduction

Acute fibrinous and organising pneumonia (AFOP) is a rare and distinct histopathological type of interstitial pneumonia [1]. Although the exact prevalence of AFOP is not well established, it is generally considered an uncommon condition and is often regarded as a form of acute lung injury [2].

Acute fibrinous and organising pneumonia can arise as an idiopathic condition, although it may also be associated with a variety of underlying causes [3]. In some instances, the aetiology of AFOP remains undetermined, and such cases are classified as idiopathic. The disease typically presents with non-specific clinical symptoms,

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including cough, dyspnoea and fever, which may overlap with other, more common lung conditions [4].

Due to its non-specific symptoms and variable radiographic presentations, AFOP is frequently misdiagnosed, resulting in delayed diagnosis and inappropriate treatment. Imaging findings often reveal diffuse ground-glass opacities or consolidation and, in rare cases, mass-like lesions that are initially mistaken for malignancy. Pathological examination remains the gold standard for diagnosis, and early recognition of AFOP is vital to ensure appropriate management [5]. The primary treatment for AFOP is corticosteroids, although some patients may require additional immunosuppressive therapies.

Case report

A 66-year-old man presented with cough, sputum, chest tightness and pain persisting for 1 month. Prior to his admission, he had received cefaclor and azithromycin at a local clinic, but with no tangible alleviation. He subsequently underwent a chest X-ray that revealed a space-occupying mass in the left lung, and he was hospitalised for further treatment on 10 September 2023. The results of laboratory investigations were normal.

Computed tomography (CT) imaging demonstrated irregularly shaped soft tissue masses in both the upper and lower lobes of the left lung, characterised by irregular margins, lobulation, spiculation and pleural indentation (Fig. 1). The masses exhibited inhomogeneous enhancement, suggesting an indeterminate nature. The imaging features resembled those of lung cancer, though they were non-specific.

Positron emission tomography-CT (PET-CT) showed high-density lesions in both the upper and lower lobes of the left lung, with increased glucose metabolism (SUV values of 8.8 and 7.4, respectively). Additionally, multiple enlarged lymph nodes were detected in the mediastinum and interlobular spaces, further raising suspicion of lung cancer. It should be noted that our hospital does not currently have a PET-CT scanner. The patient underwent the PET-CT scan at an external institution, and we were unable to obtain the original imaging data. Therefore, we can only refer to the results of the PET-CT scan in our report.

Consequently, pathogenic bacteria culture, Gene-Xpert detection and metagenomic next-generation sequencing of bronchoalveolar lavage fluid and lung tissue were all negative. No tumour cells were found. At this point, lung cancer was given priority consideration. A CT-guided percutaneous lung tru-cut biopsy was then performed on the upper lobe of the left lung. Histological examination revealed that the alveolar cavity was filled with fibrin-like exudate and infiltrated by a large number of inflammatory cells, consistent with the pathological features of AFOP (Fig. 2A). Nonetheless, we considered the limitations of the small biopsy specimen because carcinogenic tissue might not have been taken, and after a multidisciplinary discussion, lung cancer was still not excluded. Therefore, a second CT-guided percutaneous lung tru-cut biopsy was performed in the upper and lower lobes of the left

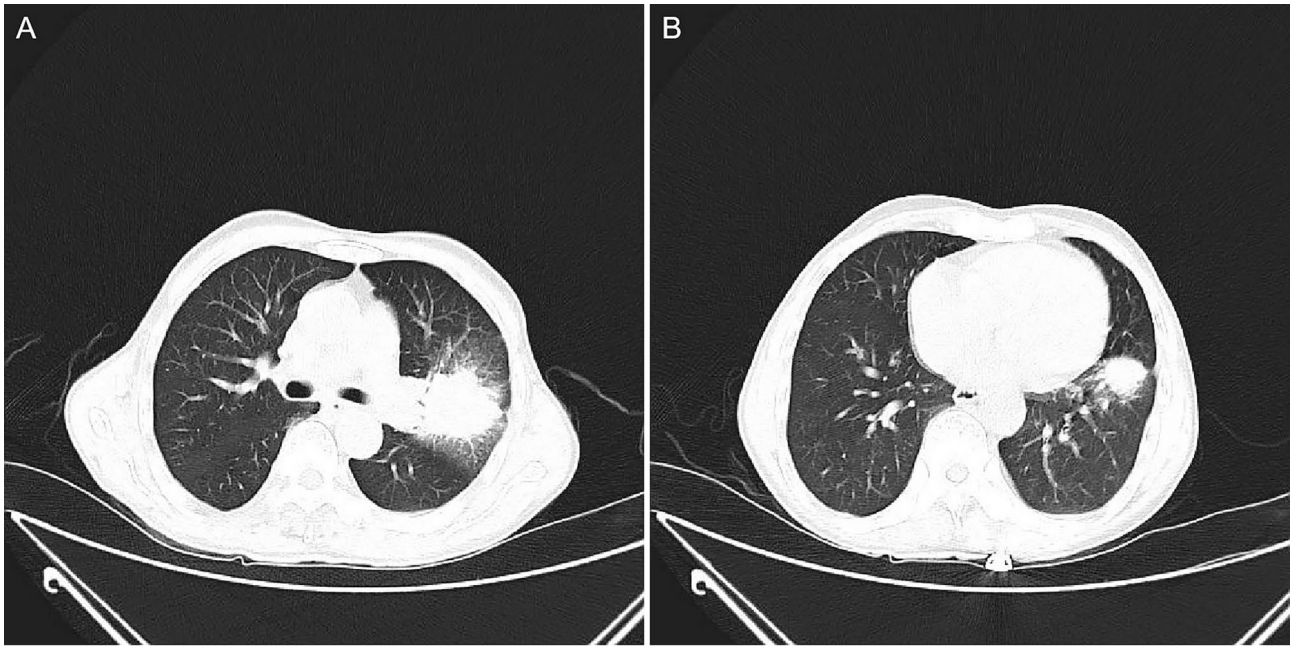

Fig. 1 Initial pulmonary CT images. (**A**) An irregular soft tissue mass measuring approximately 4.7 cm×3.8 cm is seen in the upper lobe of the left lung, with lobulated and spiculated margins and partial bronchioles truncated near the hilum; (**B**) An oval mass is seen in the lower lobe of the lung, measuring about 2.3 × 1.7 cm, with lobulated, spiculated margins and pleural retraction signs

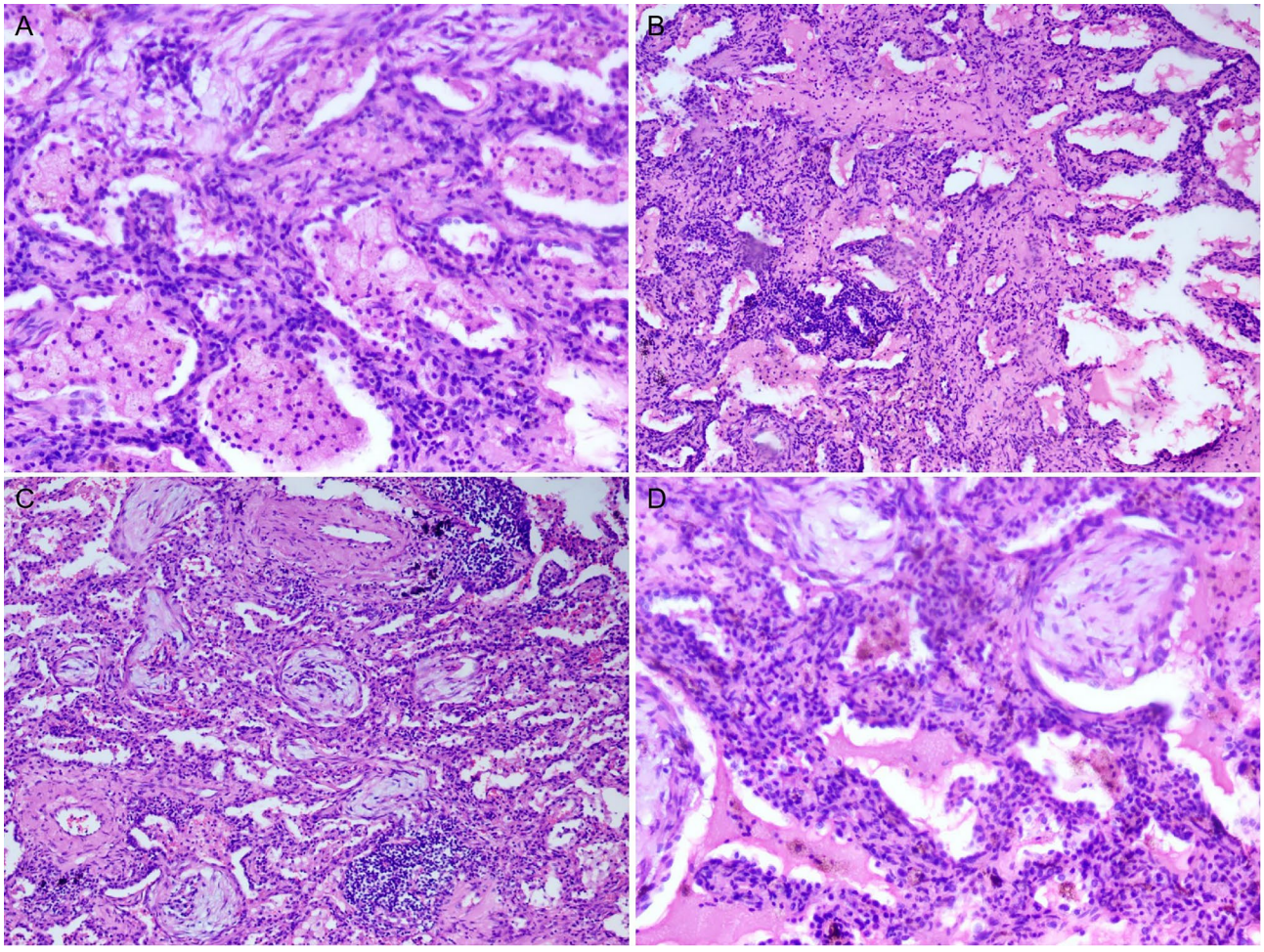

Fig. 2 Results of lung biopsy. (**A**) First lung biopsy (upper lobe of the left lung): Numerous lymphocytes, plasma cells, and neutrophils infiltrate the alveolar cavity, with fibrinoid material in the alveolar cavity (HE×200); (**B**) Second lung biopsy (lower lobe of the left lung): Fibrinoid exudate in the alveolar cavity, with numerous inflammatory cells infiltrating the alveolar septa and varying degrees of alveolar cavity dilation (HE×100); (**C**) Second lung biopsy (upper lobe of the left lung): The alveolar cavity is filled with fibrous tissue. The lung tissue shows widened alveolar septa with numerous lymphocytes, plasma cells, and neutrophils infiltrating, and Masson bodies are seen (HE×100); (**D**) Third lung biopsy (lower lobe of the left lung): Widened alveolar septa with inflammatory cell infiltration, fibrinoid exudate in the alveolar cavity, and local fibrous tissue proliferation (HE×200)

lung, which identified eosinophilic fibrin-like exudate in the alveolar lumen, a small amount of loose connective tissue consisting of fibroblasts, a large number of neutrophils and lymphocytes infiltration, and various degrees of alveolar cavity dilatation (Fig. 2B and C), all consistent with AFOP. Since no tumours were found in either lung biopsy, we considered the diagnosis of AFOP. The patient was treated with 40 mg of methylprednisolone once a day for a duration of 1 week. After this period, the dosage was adjusted based on the patient's clinical condition, with a gradual reduction in steroid dosage. The patient was discharged later after reporting symptom relief.

After 1 month, a follow-up chest CT (Fig. 3) revealed that the mass-like lesion in the upper lobe of the left lung was substantially smaller than before, whereas the mass-like lesion in the lower lobe of the left lung had enlarged to approximately 3.6 × 2.6 cm. The reduction in the upper lobe lesion following corticosteroid therapy was consistent with the diagnosis of AFOP, but the enlargement of the lower lobe lesion was not, prompting consideration of a possible tumour in the lower lobe of the left lung.

The patient was advised to undergo a surgical lung biopsy to further exclude lung cancer, but he refused due to concerns about surgical trauma and risk. We opted for the next best approach, performing a third CT-guided percutaneous lung tru-cut biopsy on the lower lobe of the left lung. Pathological examination (Fig. 2D) revealed fibrin-like exudate in the alveolar cavity, widened alveolar septum and local hyperplastic fibrous tissue, with no evidence of malignancy. Based on three lung biopsies, no neoplastic lesions were detected, confirming that the presence of a tumour could be excluded. We continued corticosteroid therapy for 4 weeks until the symptoms were further relieved.

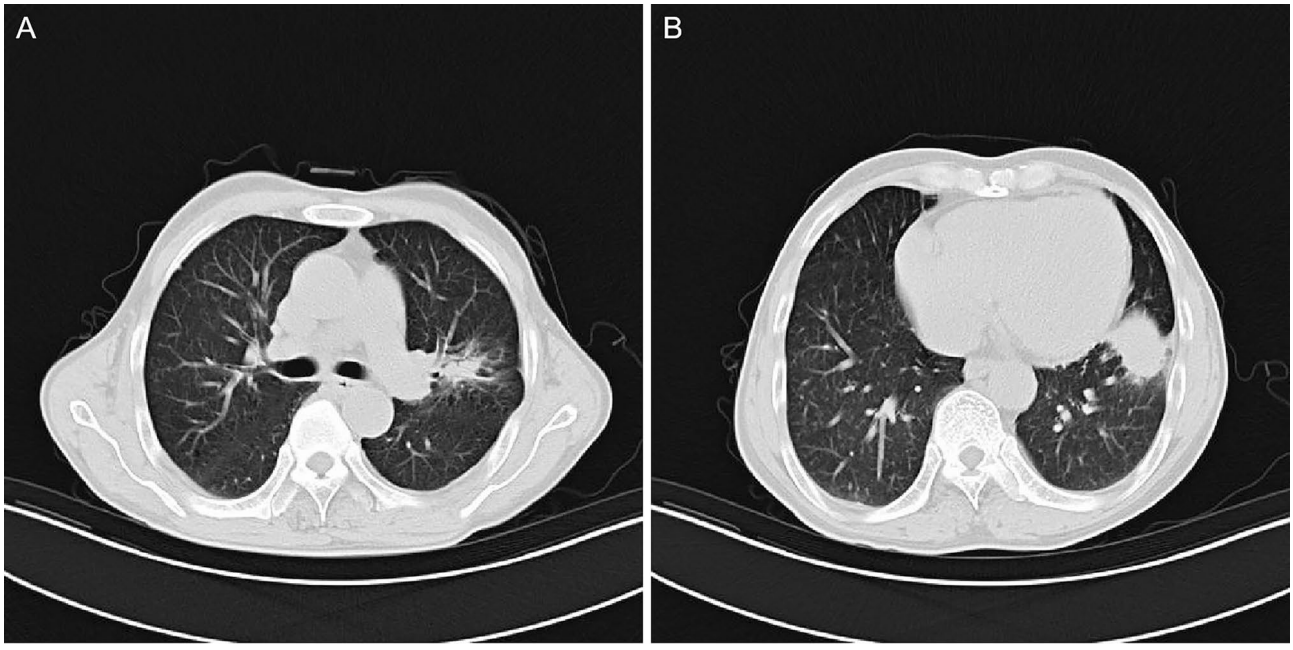

Fig. 3 Follow-up chest CT images one month later. (**A**) The soft tissue in the upper lobe of the left lung has significantly decreased compared to before; (**B**) The soft tissue in the lower lobe of the left lung, measuring about 3.6 cm×2.6 cm, has increased in size compared to before

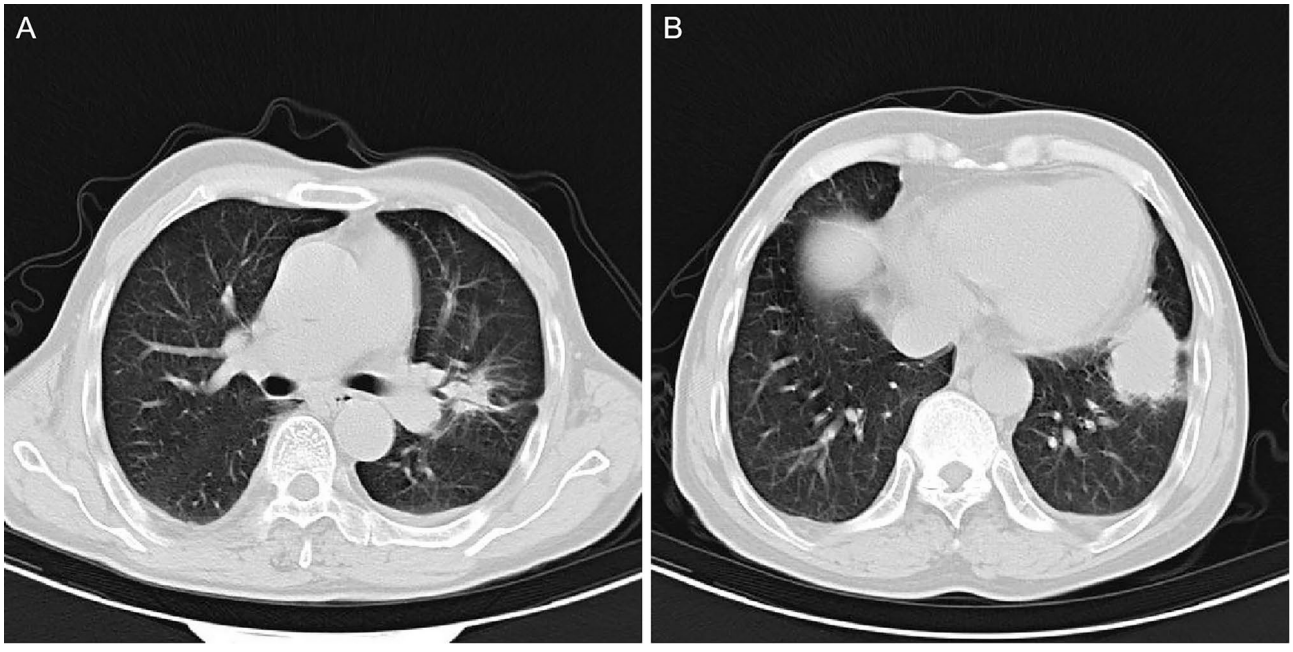

Fig. 4 Follow-up chest CT images two months later. (**A**) Patchy areas of increased density in the upper lobe of the left lung, with surrounding linear shadows, showing no significant changes compared to Fig. 3A; (**B**) An irregular soft tissue density mass is seen in the lower lobe of the left lung, with the largest cross-section measuring about 3.8 × 2.9 cm, which has increased in size compared to Fig. 3B

After 2 months, a follow-up chest CT (Fig. 4) revealed that the lesion in the lower lobe of the left lung had continued to enlarge, measuring approximately 3.8 × 2.9 cm, with a CT value of approximately 51 HU and lobulation at the edge. A patchy density-increased shadow measuring approximately 1.3 × 1.0 cm was observed in the posterior segment of the upper lobe of the left lung.

We considered that the patient's condition was not well controlled by glucocorticosteroids alone, so we added mycophenolate mofetil to manage the mass-like lesion. The bronchial tube running through it appeared

narrowed, with long cord shadows around it. At this time, the space-occupying lesion in the lower lobe of the left lung still showed mild enlargement. We judged that using glucocorticoids alone was insufficient and added 0.5 g of mycophenolate mofetil twice a day.

After 3 months, a follow-up chest CT (Fig. 5) revealed that the mass-like lesion in the lower lobe of the left lung had substantially diminished. Methylprednisolone was gradually decreased, and mycophenolate mofetil was maintained.

After 4 months, the follow-up chest CT showed residual striae-like lesions in the upper and lower lobes of the left lung. The patient's clinical condition was stable, with no recurrence of respiratory symptoms, and the chest CT did not show any worsening. Glucocorticosteroids and mycophenolate mofetil were discontinued after 6 months. At the 1-year follow-up, the patient remained symptom-free, and chest CT (Fig. 6) showed no recurrence of the lesions.

Discussion

The clinical symptoms of AFOP are diverse, with common symptoms including cough, dyspnoea and fever. Other symptoms, such as chest pain, haemoptysis, weight loss and night sweats, may also occur in some patients [6]. Similar to its clinical symptoms, the radiological manifestations of AFOP are non-specific. Common features include multifocal patchy shadows in both lungs, ground-glass opacities, consolidation and air bronchograms [7–10]. In some patients, ground-glass opacities and consolidation may coexist, with these radiological findings distributed along the bronchovascular bundles or in a diffuse random pattern [10]. In certain cases, patients may also present with a halo sign or reverse halo sign, although these features are relatively uncommon [8]. Furthermore, the radiological manifestations of AFOP may vary depending on the underlying cause, such as infection or drug-induced toxicity [9].

Given the non-specific nature of these radiological findings, they cannot provide definitive diagnostic evidence for AFOP. Therefore, pathological examination is often required to confirm the diagnosis. It is extremely rare for AFOP to initially present as a mass in imaging, particularly when the mass exhibits features such as lobulation, spiculation and pleural indentation. This atypical presentation often leads to an initial misdiagnosis of lung cancer, increasing the psychological burden on the patient and their family.

Kobayashi et al. [11] reported a case of AFOP presenting as a solitary lung nodule with a vague edge in the anterior segment of the upper lobe of the right lung, accompanied by an air bronchogram, which was mostly absorbed after 3 months of corticosteroid therapy. Similarly, Wang et al. [12] reported a case of AFOP presenting with multiple nodules on a diffuse ground-glass background in both lungs, with the nodules almost disappearing after 1 month of corticosteroid treatment [12].

Although imaging findings may include diffuse ground-glass opacities or consolidation and rarely mass-like lesions, these features alone cannot confirm a diagnosis of

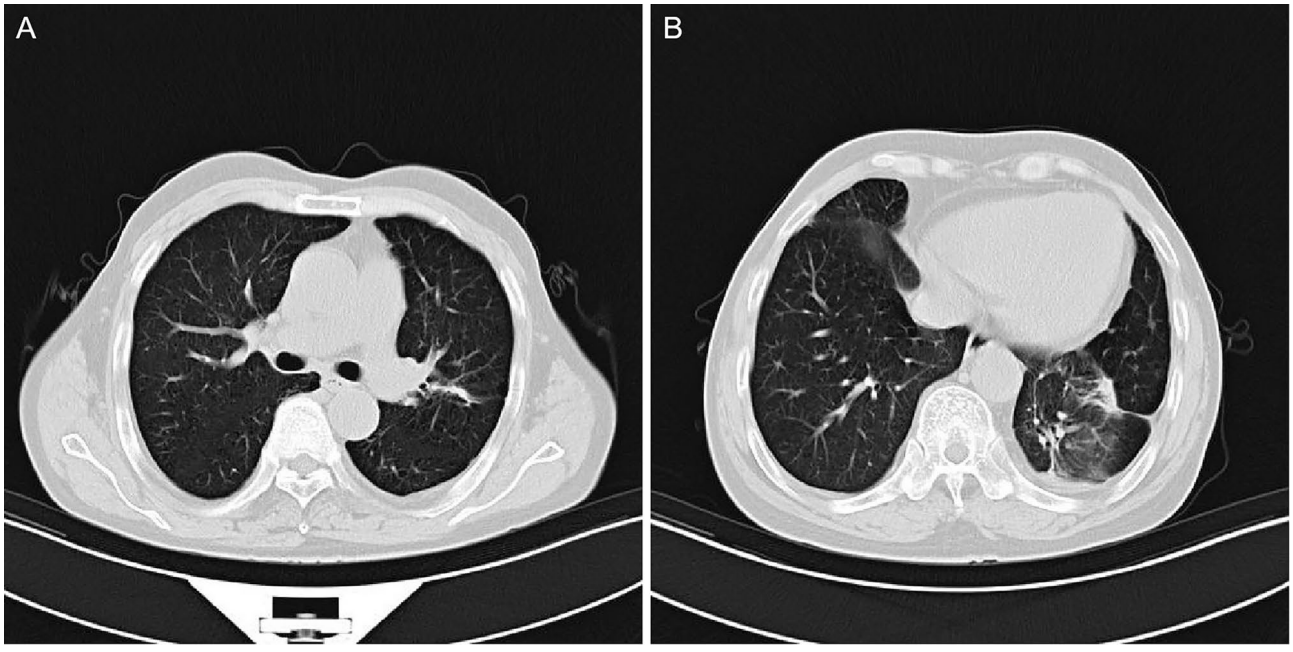

Fig. 5 Follow-up chest CT images three months later. Both the upper lobe (**A**) and the lower lobe (**B**) of the left lung have significantly decreased in size compared to before

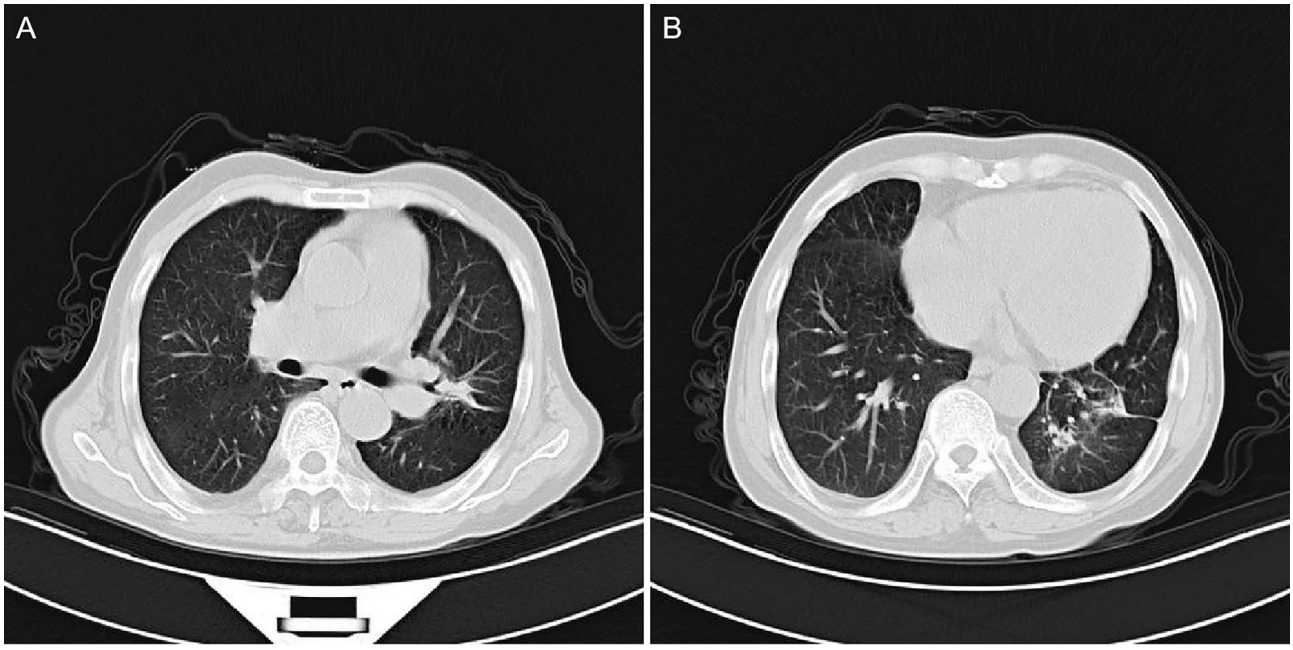

Fig. 6 Follow-up chest CT images one year later. The upper lobe (**A**) and the lower lobe (**B**) of the left lung show residual linear shadows, with no recurrence

AFOP. The gold standard for diagnosing AFOP remains pathological evidence from tissue samples. A diagnosis of AFOP cannot be established solely on imaging features or on the basis of ineffective anti-infection treatment, negative pathogen detection or the presence of features suggestive of organising pneumonia, even when decisions are made by a multidisciplinary team. Imaging is more useful for identifying the lesion location, evaluating treatment effects and assessing prognosis.

It is important to note that making a pathological diagnosis of AFOP in a mass-like lesion is potentially hazardous because this histopathological manifestation can also be seen at the margins of lung cancer. Feng et al. [4] reported a case involving a patient with a CT finding of an occupying lesion in the lower lobe of the right lung and a pathological diagnosis of AFOP obtained through percutaneous fine-needle lung biopsy. After 3 months of treatment with methylprednisolone, the lesion showed slight shrinkage and was eventually surgically resected. Low-differentiated adenocarcinoma was confirmed, with eosinophilic intra-alveolar fibrin balls observed surrounding the cancer mass.

This case highlights the potential pitfalls of relying solely on biopsy findings for the diagnosis of AFOP, as the sampled tissue may not fully represent the entire lesion, particularly if neoplastic foci are missed. Therefore, patients with a pathological diagnosis of AFOP should undergo careful evaluation to exclude the possibility of underlying malignancy, and their condition should be closely monitored during treatment. Additional biopsies may be necessary if the clinical condition progresses or worsens.

In our case, despite multiple CT-guided percutaneous lung tru-cut biopsies demonstrating pathological features consistent with AFOP, the lesion in the lower lobe of the left lung continued to enlarge during treatment with methylprednisolone. This raised doubts about the diagnosis and led us to question whether AFOP surrounding lung cancer might have resulted in a diagnostic error. In such situations, surgical resection of the lesion should be considered to obtain a definitive diagnosis, as it may be the only way to guide appropriate treatment.

The patient refused surgical intervention due to the associated risks, prompting us to perform another percutaneous lung biopsy. The pathological features remained consistent with AFOP, leading us to consider two possibilities: ineffective glucocorticoid therapy or undiagnosed, hidden lung cancer. Based on the available pathological evidence and multidisciplinary discussion, we concluded that glucocorticoid therapy was ineffective and decided to add an immunosuppressive agent.

Currently, there are no standardised treatment guidelines for AFOP; however, glucocorticoids are the most widely used treatment. During glucocorticoid therapy, there is a risk of non-remission or relapse, which may require higher doses, combination with immunosuppressive agents or even lung transplantation [13].

Bhatti et al. [13] reported a case of acute fulminant AFOP that was successfully treated with a combination of glucocorticoids and mycophenolate mofetil. Other immunosuppressive agents, such as cyclophosphamide

and cyclosporine, have also been reported as effective treatments for AFOP [12]. In our case, the upper lobe of the left lung responded well to corticosteroids, but the lower lobe did not. Therefore, we added mycophenolate mofetil, achieving remission, consistent with previous reports in the literature.

In conclusion, AFOP is primarily a pathological diagnosis with limited specificity in clinical manifestations and imaging. When chest imaging shows a mass-like lesion, adequate histopathological specimens should be obtained to exclude malignant tumours, collagen vascular diseases and granulomatous diseases. Close follow-up of the lesion during treatment is essential. The final diagnosis should be based on pathological evidence in conjunction with the response to treatment.

Acknowledgements
No funding or sponsorship was received for this study or publication of this article.

Author contributions
(I) Conception and design: Zhou W. (II) Administrative support: Zhang LY. (III) Provision of study materials or patients: Xu B. (IV) Collection and assembly of data: Wang CH. (V) Data analysis and interpretation: All authors. (VI) Manuscript writing: All authors. (VII) Final approval of manuscript: All authors.

Funding
This research was supported by Shandong Province Medical Health Science and Technology Project (202303020785).

Data availability
All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Shengli Oilfield Central Hospital. Written informed consent was obtained from the all participants for the publication of any potentially identifiable images or data included in this article.

Consent for publication
The manuscript is not submitted for publication or consideration elsewhere.

Competing interests
The authors declare no competing interests.

Received: 20 December 2024 / Accepted: 20 April 2025
Published online: 26 April 2025

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