Pneumonia-associated inflammatory myofibroblastic tumour: a case report

Xiuxin Mo[1], Yuchun Zhuang[1], Liming Zhang[1], Chengcheng Chen[2] *

[1] Department of Thoracic Cardiovascular Surgery, Weifang Second People's Hospital, Weifang, China
[2] Department of Radiology, People's Hospital of Rizhao, Rizhao, China

* Corresponding author: chengcheng9987@163.com

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Case presentation

A 42-year-old woman was admitted to Weifang Second People's Hospital on 20 June 2024 following an incidental finding of a pulmonary nodule (21 × 27 mm^2) during a routine physical examination 1 year previously. Although serial imaging over the following year showed stable size and morphology, suggesting a benign nature, malignancy remained possible. The patient had no significant medical, family, or psychosocial history, and denied tobacco and alcohol use. Preoperative evaluation included fine-needle aspiration cytology, which revealed spindle cells with lymphoplasmacytic infiltration. Contrast-enhanced chest computed tomography demonstrated a lobulated left upper lobe nodule with heterogeneous enhancement and partial bronchial obstruction (Fig 1). Magnetic resonance imaging of the brain and abdominal ultrasound showed no metastasis. Based on the above investigations and considering the patient's financial circumstances, a positron emission tomography scan was not performed. Tumour marker levels were within the normal range—neuron-specific enolase: 13.06 ng/mL, carbohydrate antigen 19-9: 9.76 U/mL, carcinoembryonic antigen: 1.54 ng/mL, cytokeratin 19 fragment: 1.23 ng/mL, and squamous cell carcinoma antigen: 0.51 ng/mL. Thoracoscopic left upper lobectomy was performed on 22 June 2024. Histopathology revealed proliferating spindle myofibroblasts/fibroblasts with lymphoplasmacytic infiltration and focal mucin deposition (Fig 2). Immunohistochemistry confirmed inflammatory myofibroblastic tumour (IMT): positive for cytokeratin, vimentin, smooth muscle actin (SMA), and epithelial membrane antigen; STAT6 (signal transducer and activator of transcription 6) negative with a Ki-67 index of 30%. The patient recovered well, with no recurrence at 3-month follow-up, although long-term surveillance was recommended.

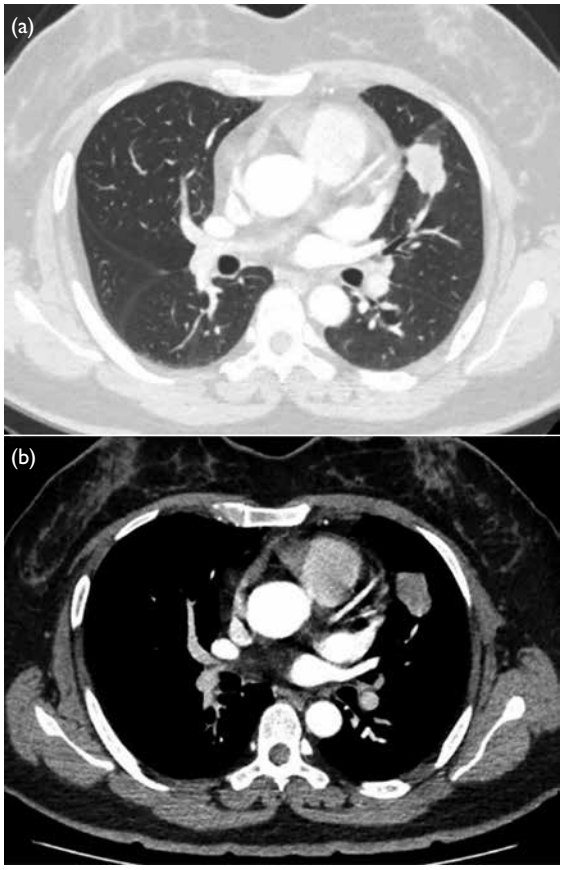

FIG 1. (a) Plain computed tomography imaging of the left upper lobe demonstrates a 21 × 27 mm^2 lobulated nodule with well-defined margins. (b) Contrast-enhanced scan reveals marked heterogeneous enhancement of the lesion and occlusion of the adjacent proximal bronchus

Discussion

Inflammatory myofibroblastic tumour, originally termed inflammatory pseudotumour (IPT) in 1939, has been reclassified through molecular insights from a reactive proliferation to a true neoplasm.[1] Although IPT remains a non-neoplastic inflammatory lesion with regression potential, IMT is now defined as a clonal neoplasm composed of myofibroblastic spindle cells within a plasma cell/lymphocyte/eosinophil-rich stroma. This distinction is crucial clinically since IMT exhibits local invasiveness and recurrence risk, unlike IPT's benign course.[2]

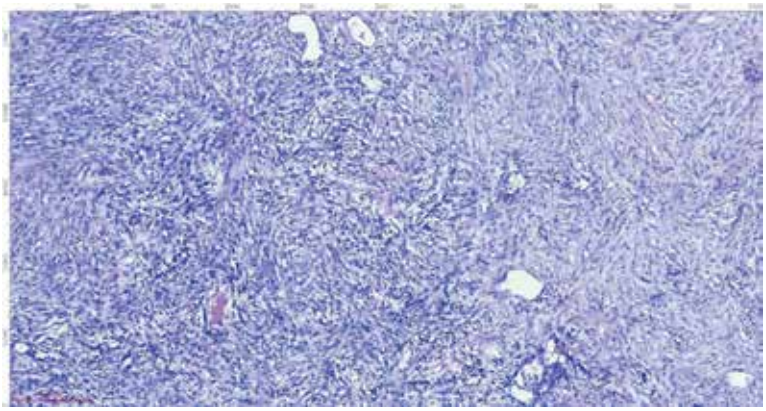

FIG 2. Histopathological examination of the left upper lobe nodule revealed proliferating spindle-shaped myofibroblasts/fibroblasts accompanied by abundant lymphoplasmacytic infiltration (haematoxylin and eosin staining, ×20)

Inflammatory myofibroblastic tumour is a rare mesenchymal neoplasm that primarily affects children and young adults, with lower incidence in adults.[3] Its broad anatomical distribution most commonly involves the lungs (0.7% of pulmonary tumours)[4] and the abdomen/mesentery/retroperitoneum; rare sites include the oesophagus, cardiac chambers, and adrenal glands. As a borderline malignancy, recurrence rates differ by site (pulmonary 2% vs extrapulmonary 25%), with less than 5% risk of distant metastasis.[5] Symptoms vary anatomically: pulmonary cases may present with cough or haemoptysis (including incidental detection), abdominal lesions may cause pain or obstruction, while systemic symptoms include fever and weight loss. Pulmonary IMTs, as observed in our patient, may present with cough, atypical chest pain, haemoptysis, or dyspnoea, although incidental detection during routine health screening, as in our case, is not uncommon.

The non-specific radiological features of IMT pose significant diagnostic challenges, necessitating histopathological confirmation. In our patient, the nodule was identified during a routine physical examination 1 year prior to admission, and serial imaging demonstrated stable lesion size. This supported a benign nature but did not entirely exclude malignancy. Although minimally invasive techniques such as fine-needle aspiration biopsy and bronchoscopic sampling are often attempted, these methods frequently yield insufficient tissue for definitive diagnosis. Complete surgical resection therefore remains the gold standard for both diagnostic confirmation and therapeutic intervention.

Histopathological examination typically reveals spindle-shaped myofibroblastic proliferation within variable stromal matrices (myxoid, collagenous, or calcified patterns), accompanied by a polymorphic inflammatory infiltrate. In our patient, the histopathological features were consistent with IMT, showing proliferating spindle-shaped myofibroblasts/fibroblasts with abundant lymphoplasmacytic infiltration and focal mucin deposition. The diagnosis was further supported by immunohistochemical findings, including positivity for cytokeratin, vimentin, SMA, and epithelial membrane antigen, although anaplastic lymphoma kinase (ALK) and STAT6 were negative.

Molecular studies have identified chromosomal 2p23 translocations in approximately 50% of IMT cases, leading to constitutive activation of ALK pathways.[6] This genetic aberration correlates with tumour aggressiveness and local recurrence, supporting IMT's classification as a true neoplasm rather than a reactive pseudotumour. Immunophenotypically, most IMTs express mesenchymal markers such as ALK (cytoplasmic/membranous), caldesmon, desmin, and SMA, with ALK reactivity aiding differentiation from histological mimics. Notably, our case showed an atypical immunoprofile with SMA positivity and ALK negativity, reflecting the phenotypic heterogeneity and the need for comprehensive molecular profiling in challenging cases.

Therapeutic strategies for IMT depend on disease stage and resectability. For localised lesions, complete surgical resection (R0 margins) achieves a 2% recurrence rate, whereas incomplete resection (R1/R2) increases recurrence risk to 60% ($P<0.01$).[7] In our patient, thoracoscopic left upper lobectomy was performed with negative surgical margins, and no tumour recurrence was observed during the initial 3-month postoperative follow-up. Nonetheless, longer-term surveillance is recommended to confirm the absence of tumour recurrence. Non-resectable or recurrent cases require multimodal approaches, including radiotherapy (45-50 Gy), platinum-based chemotherapy, and ALK inhibitors for ALK-positive subtypes.

Emerging molecular insights have identified *ALK* rearrangements as key oncogenic drivers, positioning ALK-targeted therapies as both diagnostic and therapeutic tools.[8] Clinical trials have demonstrated the efficacy of crizotinib: an initial phase 1 study (NCT01121588)[9] achieved a 42.9% partial response rate in refractory paediatric/young adult IMTs (n=7), while cohort expansion (n=14) improved the overall response rate (ORR) to 86% (36% complete responses). Japanese studies corroborate these findings, with 100% ORR (1 complete response, 2 partial responses) in *ALK*-rearranged IMTs treated with crizotinib or alectinib.[10] Nonetheless, therapeutic heterogeneity (ORR: 36%-100%), small

sample sizes, and geographical bias necessitate standardised multicentre trials to validate efficacy and durability.

In summary, IMT represents a rare borderline neoplasm with intermediate malignant potential, distinct from the historically described IPT. The present case highlights the importance of accurate histopathological and immunohistochemical diagnosis, particularly in immunophenotypically atypical lesions, and underscores the need for long-term follow-up to monitor for potential recurrence.

Author contributions

Concept or design: X Mo.
Acquisition of data: Y Zhuang
Analysis or interpretation of data: L Zhang.
Drafting of the manuscript: X Mo.
Critical revision of the manuscript for important intellectual content: C Chen.

All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

All authors have disclosed no conflicts of interest.

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Ethics approval

This study was approved by the Ethics Committee of Weifang Second People's Hospital, China (Ref No.: KY2024-077-01) and was conducted in accordance with the Declaration of Helsinki. The patient provided written informed consent for participation and publication of this case report, including the accompanying clinical images.

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