



Successful management of an unusual case of pediatric inflammatory myofibroblastic tumor: a case report and literature review

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Introduction and importance: Inflammatory myofibroblastic tumor (IMT) is a rare neoplastic condition that primarily affects children and young adults. This case report highlights the challenges in diagnosing and treating pediatric IMT, emphasizing the importance of tailored interventions.

Case presentation: An 8-year-old boy presented with respiratory symptoms and was diagnosed with an IMT located in the left main bronchus. Imaging studies revealed a soft tissue mass and lymph node enlargement. Bronchoscopy and biopsy confirmed the diagnosis. The patient underwent bronchoscopic debulking procedures followed by lobectomy and bronchoplasty due to persistent disease.

Clinical discussion: IMTs pose diagnostic challenges due to their varied clinical presentation and similarities with other neoplasms. A multidisciplinary approach involving pathologists, radiologists, and surgeons is crucial for accurate diagnosis and optimal treatment planning. The pathogenesis of IMTs is not fully understood, but theories suggest an inflammatory response or involvement of the ALK gene. IMTs can affect various organs, each with distinct symptoms. Imaging modalities lack specificity, emphasizing the importance of histopathological examination.

Conclusion: IMTs require individualized treatment approaches based on the location and extent of the tumor. Long-term follow-up is essential for monitoring recurrence and metastasis. Further research is needed to enhance our understanding of IMT biology and develop targeted therapies to improve patient outcomes. This case report underscores the importance of tailored interventions in pediatric IMT cases and highlights the challenges in diagnosis and treatment.

Keywords: ALK gene, bronchoscopy, debulking, histopathology, immunohistochemistry, inflammatory myofibroblastic tumor

Introduction

Inflammatory myofibroblastic tumor (IMT) is an exceptionally rare and distinctive pseudosarcomatous inflammatory lesion that manifests in the soft tissues and visceral organs, predominantly affecting children and young adults. Initially identified in the lung, IMT was initially perceived as a reparative post-inflammatory

condition rather than a neoplastic progression^[1]. Given its relatively low risk of recurrence and metastasis, IMT has been categorized as a neoplastic disease with intermediate biological potential^[2].

While IMTs can emerge across various age groups, a significant majority, more than half, are diagnosed in individuals under 40, with IMTs being particularly prevalent among children under 16, making them the most common pulmonary tumor in this age group^[3]. This incidence appears consistent across diverse ethnic groups, with no notable gender-based variations^[4].

The lung stands out as the predominant site for IMT, constituting the most frequent location. Notably, the occurrence of inflammatory pseudotumors in the lung represents a mere 0.7% of all tumors originating in the bronchus and lung parenchyma^[4]. However, IMT can also manifest in extrapulmonary sites such as the abdomen, retroperitoneum, head, neck, spinal cord, and uterus, adding to the complexity of diagnosis and treatment^[3-5].

Herein, we present a unique case of an 8-year-old boy with an IMT located in the left main bronchus. This case is noteworthy due to the unusual location and the challenges it posed in both diagnosis and treatment. The patient was managed successfully through a series of bronchoscopic debulking sessions followed by targeted lobectomy, illustrating the importance of tailored interventions in pediatric IMT cases. This case report has been reported in line with the SCARE Criteria^[6].

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Case presentation

An 8-year-old boy with a free past medical history presented with symptoms persisting for 20 days, including a mild intermittent cough, dry mouth, progressive dyspnea, and an elevated temperature (38.9°C), on the exam the patient looked tachypneic and had no sign of respiratory distress, the air entry was decreased on the left side and O₂ saturation was 94% off O₂. Despite being treated for a presumed chest infection, there was no improvement over 2 days duration before the admission. Subsequently, the patient developed hemoptysis occurring 3–4 times daily. Diagnostic investigations, including chest X-ray, and abdominal, and chest CT scans, were conducted, followed by a bronchoscopy with biopsy.

Diagnostic efforts included a Chest X-ray that revealed consolidation on the left side of the chest (Fig. 1), while chest CT demonstrated an ill-defined hypodense, heterogeneously enhancing soft tissue mass in the left main bronchus measuring approximately 1 × 1 cm. This mass resulted in complete collapse of the left lung, accompanied by volume loss in the left hemithorax. Additionally, well-defined enhancing lesions were identified at the left hilum, measuring about 15 × 10 mm and 12 × 8 mm, indicative of enlarged left hilar lymph nodes. The right lung exhibited normal findings without evidence of masses, consolidations, pleural effusion, pneumothorax, or pericardial effusion (Fig. 2a and b).

Bronchoscopy findings revealed a fungating mass lesion in the left main bronchus, located 2 cm from the carina, causing complete luminal obstruction. Despite unsuccessful attempts to pass distally using ultra-slim bronchoscopy, the procedure proceeded without complications, allowing for successful biopsy retrieval.

A subsequent bronchoscopy was performed and utilized snare forceps to gradually debulk the entire mass lesion. The mass extended into the left upper lobe bronchus, and as it was progressively removed, the left upper bronchus was opened. However, minimal remaining tissue could not be completely

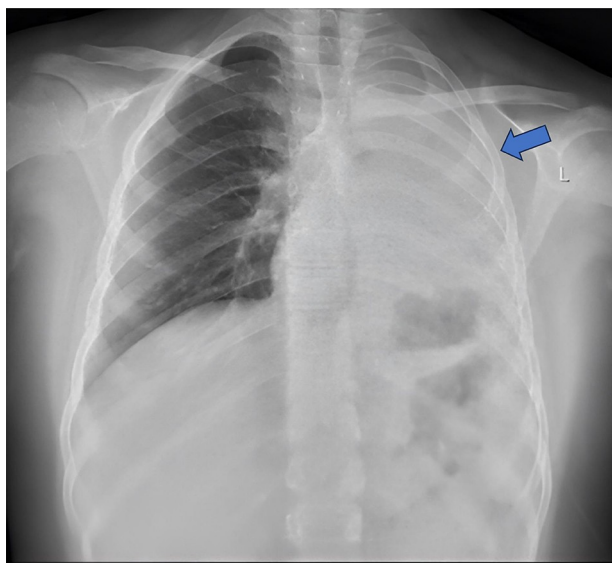


Figure 1. Chest X-ray revealed consolidation on the left side of the chest.

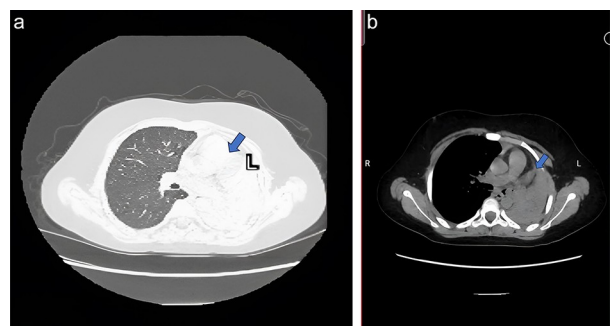


Figure 2. (a) Chest CT revealed an ill-defined hypodense, heterogeneously enhancing soft tissue mass in the left main bronchus. (b) Mediastinal window Chest CT revealed an ill-defined hypodense, heterogeneously enhancing soft tissue mass in the left main bronchus.

excised (Fig. 3a, depicting the left main bronchus before mass debulking, and Fig. 3b, illustrating the left main bronchus after mass debulking). A post-bronchoscopic debulking chest X-ray is presented (Fig. 4).

Further bronchoscopic intervention was necessary to manage residual tumor in the left upper lobe bronchus, identified during a 2-month follow-up. This additional procedure aimed to address the remaining tumor tissue. This procedure was repeated after 1 month, and then again after 4 months coinciding with a comprehensive assessment using a chest CT scan with intravenous contrast. A chest CT scan revealed a relatively well-defined, heterogeneous enhancing lesion in the left lung apex, measuring approximately 3 × 1 cm in axial dimension. This lesion was largely indicative of the previously identified IMT. Both lungs exhibited a normal appearance without evidence of additional masses or consolidations. No notable hilar or mediastinal lymph node enlargement, pleural effusion, or pneumothorax was observed. The heart exhibited normal size without pericardial effusion (Fig. 5).

The histopathological examination as showed (Fig. 6) revealed a diverse composition of inflammatory cells, irregularly arranged fascicular spindle cells, and a predominant plasma cell infiltrate. The inflammatory infiltrate exhibited varying numbers of eosinophils, lymphocytes, and macrophages. Focal myxoid and sclerotic foci were observed in the background stroma, with no evidence of nuclear atypia. The final diagnosis confirmed the presence of an IMT. Immunohistochemistry studies

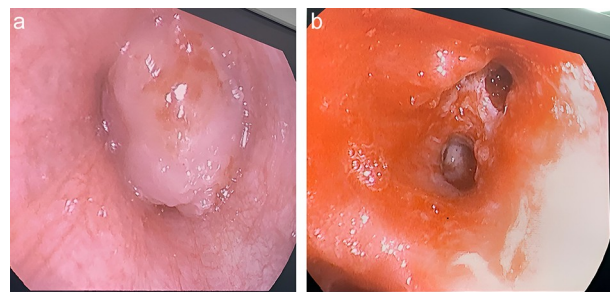


Figure 3. (a) Left main bronchus before mass debulking. (b) Left main bronchus after mass debulking.

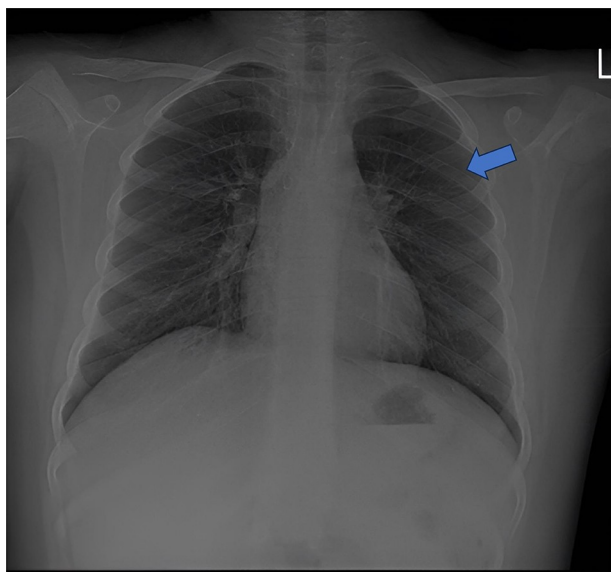


Figure 4. A post-bronchoscopic debulking chest X-ray.

demonstrated strong positivity for vimentin, variable staining for pan-Ck, focal positivity for Alk, and negativity for myogenin immunostain.

On follow up, he has been receiving adjuvant therapy with crizotinib for 7 months. Follow-up PET scans at 3- and 8-months post-operation showed no signs of recurrence. A flexible bronchoscopy at 8 months confirmed an intact stump without local recurrence. The patient remains under the care of the pediatric oncology team, continuing crizotinib therapy according to the protocol. Future follow-ups will include alternating CT scans with IV contrast or PET scans, along with bronchoscopy as needed.

In light of these findings, the thoracic surgery team decided to proceed with a lobectomy and bronchoplasty. This surgical approach was meticulously planned to address the tumor and its implications comprehensively. During the procedure fortunately, no evidence of metastasis was found. However, moderate adhesions involving the left upper lobe were encountered, mainly associated with tumor identification in the left upper



Figure 5. Chest CT scan showed a relatively well-defined, heterogeneous enhancing lesion in the left lung apex.

lobe of the lung and the removal of a sizable lymph node in station 5. Extensive adhesions were found in the hilar area. A tumor involving the upper part of the bronchus was identified, necessitating shaving of the bronchus just at the takeoff to ensure complete removal of the tumor tissue. Identification of residual tumor by frozen section, further shaving as required. The bronchus was then meticulously closed using 3/0 vicryl with several interrupted sutures were performed. Additionally, as part of a thorough exploration and to rule out further spread, lymph node sampling was performed from stations 10 L, 6, and 5. This step was crucial in assessing the extent of the disease and aiding in staging.

Discussion

IMT presents a clinical conundrum, often imitating other benign or malignant conditions without clear patterns. Constitutional symptoms like fever, weight loss, and malaise may be present in some cases, while specific symptoms related to the tumor site are predominant in most instances. The diagnostic assessment of IMT relies on imaging modalities such as ultrasound, CT scans, and magnetic resonance imaging (MRI). However, radiological findings frequently lack specificity, often depicting a solid or cystic mass with various enhancement patterns^[7].

The pathogenesis of IMTs involves multiple theories, one proposing that IMTs represent an inflammatory response to an infection or an underlying low-grade malignancy^[8]. Immunohistochemical analysis often reveals the presence of IgG-predominant and polyclonal plasma cells in IMTs, supporting the concept that IMT is a reactive inflammatory process^[9]. Another theory suggests a significant role of the anaplastic lymphoma kinase (ALK) gene in the pathogenesis of IMTs, based on histological observations in many cases. Approximately, 50% of IMTs exhibit a translocation of the ALK gene^[1], leading to the constitutive activation of tyrosine kinases. The pathogenesis of IMTs, it is essential to consider existing research that supports the proposed mechanisms. One leading theory suggests that IMTs arise as a reactive inflammatory process, with various inflammatory mediators playing a crucial role in tumor development. Gleason and Hornick highlight the presence of inflammatory cells, such as plasma cells and lymphocytes, which contribute to the tumor's characteristic features^[1]. Furthermore, studies by Souid *et al* emphasize the clinical manifestations of IMTs in children, linking them to underlying inflammatory responses. Another significant aspect of IMT pathogenesis involves genetic alterations, particularly the ALK gene^[10]. Research by Butrynski *et al* demonstrates that approximately 50% of IMTs exhibit ALK gene rearrangements, which are associated with constitutive activation of tyrosine kinases, leading to tumor proliferation^[11]. This is supported by the findings of Demir and Onal, who discuss the clinical implications of ALK alterations in treatment outcomes^[12]. Collectively, these studies underscore the multifaceted nature of IMT pathogenesis, involving both inflammatory and genetic components, warranting a comprehensive exploration of these mechanisms in future research.

The clinical manifestation of IMTs varies based on their location. IMTs may present with systemic symptoms like fever, weight loss, fatigue, and malaise, particularly when the tumor is sizable or affects multiple organs^[10]. The diverse organ involvement includes

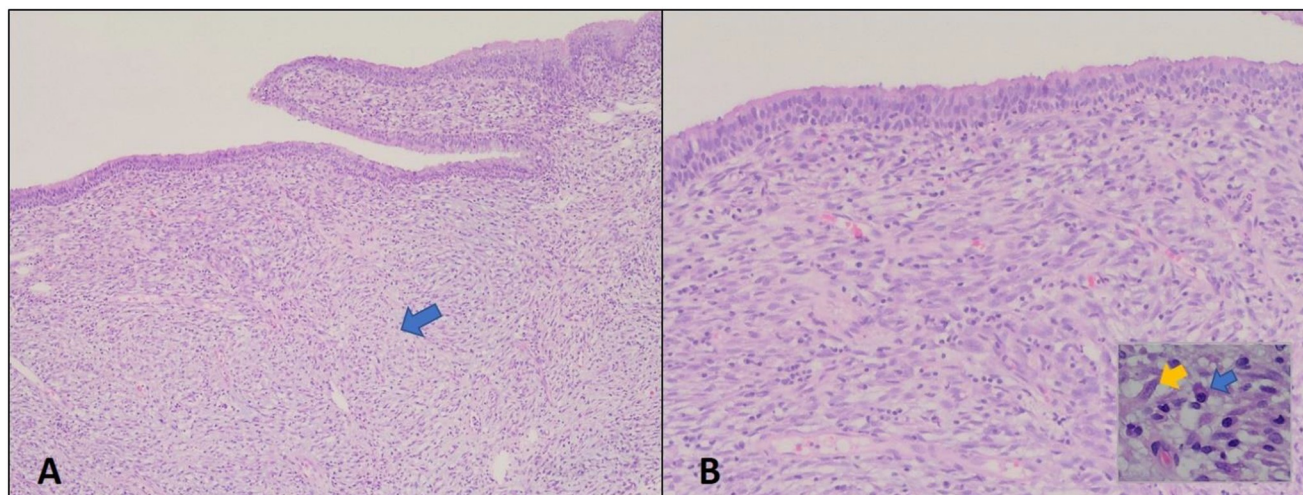


Figure 6. (a) Section shows submucosal spindle cell proliferation (H&E, 4X). (b) The lesion is composed mainly of bland spindle cells admixed with frequent lymphoplasmacytic cells, all set in a loose background (H&E, 10X); the inset shows the bland nature of the spindle cells (yellow arrow), and the admixed inflammatory cells (blue arrow). The spindle cells are immunoreactive for ALK1 (performed at another institution) confirming the diagnosis of IMT.

the lungs, abdomen, liver, gastrointestinal tract, or central nervous system, each exhibiting distinct symptoms. For instance, digestive system tumors may cause gastrointestinal bleeding, bowel obstruction, or abdominal pain, while lung involvement can result in chest pain, shortness of breath, or coughing^[13,14].

The diagnostic approach for IMT involves a combination of clinical evaluation, laboratory tests, medical imaging, biopsy, histopathological studies, and molecular studies. Clinical evaluation encompasses a thorough examination of symptoms, full medical history, and physical findings. Laboratory tests, although nonspecific for IMT, aid in assessing general health and excluding alternative conditions, with elevated erythrocyte sedimentation rate or C-reactive protein potentially indicating an inflammatory process^[15].

Medical imaging techniques, including X-rays, CT scans, MRIs, or PET scans, play a crucial role in visualizing the tumor, determining its location, size, extent, and identifying potential complications or involvement of adjacent structures^[6]. Biopsy procedures, such as core needle biopsy, open surgical biopsy, or image-guided biopsy, are employed to obtain tissue samples for further analysis^[16,17]. Histopathological studies involve microscopic examination to identify characteristic features, including spindle-shaped myofibroblast cells, inflammatory infiltration, and the absence of malignant characteristics. Immunohistochemical staining may be utilized to assess specific marker expression^[18].

Molecular studies, particularly testing for ALK gene rearrangements, may be performed in select cases^[19]. Diagnosing IMT requires careful examination and the exclusion of other potential explanations for similar clinical presentations. Accurate diagnosis often necessitates collaboration between pathologists, radiologists, and physicians. In our case, after the initial debulking, ALK gene sequencing was performed, and the case was evaluated by a multidisciplinary team, including specialists in pediatric oncology, thoracic surgery, and pediatrics. The consensus was to aim for maximal tumor reduction, weighing the options of surgical removal versus continued debulking, followed by adjunctive therapy with crizotinib. However, obtaining crizotinib posed

a challenge due to the complex political and healthcare circumstances in the Palestinian territories.

Complete surgical resection stands as the primary treatment modality for IMTs, with the choice between open thoracotomy or video-assisted thoracoscopy depending on local expertise^[4,20,21]. Thoracotomies are generally reserved for larger lesions and those displaying symptoms of local tissue invasion. A study that was retrospectively scanning patient files, done by Demir, involving 14 patients diagnosed with IMTs revealed that three patients underwent incomplete surgery, while 11 patients underwent full surgery, resulting in a 10-year overall survival of 84.6% and a 10-year disease-free survival of 75.0%^[12]. In our case, the patient had a tumor extending from the left upper lobe bronchus to the main bronchus, causing complete left lung obstruction and atelectasis. Post-first debulking, the tumor was mostly in the left upper lobe, with significant clearance from the main bronchus. Surgery was deferred for two reasons: allowing the collapsed lung time to re-expand and the tumor's close proximity to the bronchus, which might necessitate a pneumonectomy. Hence, further endoscopic debulking and adjuvant therapy were chosen to potentially avoid pneumonectomy in the young patient.

The management of the IMT in this case involved a comprehensive multidisciplinary approach that was crucial for accurate diagnosis and effective treatment planning. Each specialty played a vital role in the process. The pathologist confirmed the diagnosis through detailed histopathological analysis of biopsy specimens, identifying characteristic spindle cells and inflammatory infiltrates, which were pivotal in distinguishing IMT from other neoplastic conditions. The radiologist conducted imaging studies, including chest X-rays and CT scans, revealing the extent of the tumor and its effects on surrounding structures. The radiologist's interpretation guided the surgical team in planning the appropriate intervention, highlighting the need for bronchoscopic debulking. The surgical team, led by an experienced thoracic surgeon, performed several bronchoscopic debulking procedures followed by a lobectomy. Insights gained from the pathologist and radiologist informed the surgical strategy, ensuring that the complete tumor was removed while preserving lung function.

Table 1
Summarized similar case reports of inflammatory myofibroblastic tumors.

References	Author <i>et al</i>	Year	Symptoms	Therapeutic Approach	Outcome
⁴	Lee <i>et al</i>	2005	Chronic cough, weight loss	Complete resection	Recurrence as bilateral consolidated lesions
⁹	Balagobi <i>et al</i>	2022	Burning sensation, lower abdominal pain	Partial cystectomy	Successful resection, discharged on day 3
¹⁶	Hannah <i>et al</i>	2007	Left lung mass	Surgical resected	Recurrence mass
¹⁷	Sachdev <i>et al</i>	2018	Dry cough, breathlessness, weight loss and loss of appetite	Chemotherapy was started as the treatment modality	Reduction in the size of the tumor
²¹	Debonis <i>et al</i>	2021	Incidentally detected nodule	Surgical excision	Disease-free 2 years post-surgery

This collaborative effort underscores the importance of a multi-disciplinary approach in managing pediatric IMTs, optimizing patient outcomes through the integration of expertise from various fields.

For patients ineligible for full surgical resection, such as poor surgical candidates or those with unresectable disease, nonsurgical techniques may be employed. Glucocorticoids, radiation, and chemotherapy have demonstrated variable success in different cases^[22,23]. Non-steroidal anti-inflammatory drugs have shown efficacy in situations where surgical resection is limited due to invasion of essential structures^[24].

Crizotinib, a competitive inhibitor of the ALK tyrosine kinase, has emerged as a potential treatment for IMT. Studies have indicated its effectiveness in patients with ALK gene rearrangements^[11]. In a comparative analysis with chemotherapy, crizotinib significantly increased progression-free survival (PFS), achieving a median PFS of 10.9 months compared to 7.0 months for chemotherapy. Additionally, crizotinib exhibited a higher rate of objective response (74% vs. 45%) and a 95% disease control rate compared to 88% for chemotherapy^[25]. These findings underscore the potential of targeted therapies like crizotinib in the management of IMTs, particularly in cases with ALK gene rearrangements.

This case report is unique for a multitude of reasons. Firstly, the unusual presentation and prolonged time to diagnosis of a left upper lobe tumor obstructing the left main bronchus add to its distinctiveness. Notably, the tumor was specifically localized in the left main bronchus, which is a rare occurrence. Our approach to treatment was multifaceted, involving several bronchoscopic debulking sessions aimed at reducing the tumor size, thereby successfully confining it to the left upper bronchus. This strategy was pivotal in avoiding a more extensive pneumonectomy, as we were able to perform a targeted lobectomy instead. The case also underscores the significance of vigilant long-term monitoring, given the potential for local recurrence and distant metastases in cases of IMT, particularly in pediatric patients (see Table 1). Through this report, we aim to shed light on the diagnostic and therapeutic challenges posed by IMT in young patients, and the imperative role of a comprehensive, interdisciplinary strategy in ensuring precise diagnosis and effective treatment.

Conclusion

In conclusion, IMT poses diagnostic challenges due to its diverse clinical presentation and similarities to other neoplasms. A collaborative effort from a multidisciplinary team is crucial for precise diagnosis and optimal treatment planning. Despite its benign classification, IMT potential for local recurrence warrants the need for vigilant long-term follow-up and monitoring.

a deeper Understanding of the molecular aspects of IMT is vital for specialized and targeted treatment strategies and potentially improving patient outcomes significantly. This case report not only highlights the challenges encountered in diagnosing and treating IMTs but also underscores the imperative for continued research. Such efforts are essential to advance our knowledge of IMTs, leading to more effective management and better prognoses for patients affected by this complex condition.

Ethical approval

IRB approval was not obtained for publication of this case report.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author’s contribution

A.A. and T.M. contributed to writing the first draft. A.A., M.A., A.A., M.A., and Y.A. contributed to the editing of the first draft and writing the final manuscript. S.B. is the pathologist, collection of the histopathological slide. Y.A. supervised the project. All authors contributed to the article and approved the submitted version.

Conflicts of interest disclosure

The authors have no conflicts of interest to disclose.

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Data availability statement

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Provenance and peer review

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References

- [1] Gleason BC, Hornick JL. Inflammatory myofibroblastic tumors: where are we now? *J Clin Pathol* 2008;61:428–37.
- [2] Gros L, Dei Tos AP, Jones RL, *et al.* Inflammatory myofibroblastic tumour: state of the art. *Cancers (Basel)* 2022;14:3662.
- [3] Bahadori M, Liebow AA. Plasma cell granulomas of the lung. *Cancer* 1973;31:191–208.
- [4] Kim JH, Cho JH, Park MS, *et al.* Pulmonary inflammatory pseudotumor: a report of 28 cases. *Korean J Intern Med* 2002;17:252–58.
- [5] Bennett JA, Croce S, Pesci A, *et al.* Inflammatory myofibroblastic tumor of the uterus: an immunohistochemical study of 23 cases. *Am J Surg Pathol* 2020;44:1441–49.
- [6] Sohrabi C, Mathew G, Maria N, *et al.* The SCARE 2023 guideline: updating consensus Surgical CAse REport (SCARE) guidelines. *Int J Surg Lond Engl* 2023;109:1136.
- [7] Cantera JE, Alfaro MP, Rafart DC, *et al.* Inflammatory myofibroblastic tumors: a pictorial review. *Insights Imaging* 2015;6:85–96.
- [8] Sing Y, Ramdial PK. Cryptococcal inflammatory pseudotumors. *Am J Surg Pathol* 2007;31:1521–27.
- [9] Balagobi B, Gobishangar S, Ginige A, *et al.* Inflammatory myofibroblastic tumor: a case report of a rare form of bladder tumor. *Int J Surg Case Rep* 2022;92:106786.
- [10] Souid AK, Ziemba MC, Dubansky AS, *et al.* Inflammatory myofibroblastic tumor in children. *Cancer* 1993;72:2042–48.
- [11] Butrynski JE, D'Adamo DR, Hornick JL, *et al.* Crizotinib in ALK-rearranged inflammatory myofibroblastic tumor. *N Engl J Med* 2010;363:1727–33.
- [12] Demir ÖF, Onal O. Surgical treatment outcomes of pulmonary inflammatory myofibroblastic tumors. *Ann Thorac Med* 2022;17:44–50.
- [13] Chaudhary P. Mesenteric inflammatory myofibroblastic tumors. *Ann Gastroenterol* 2015;28:49–54.
- [14] Pettinato G, Manivel JC, De Rosa N, *et al.* Inflammatory myofibroblastic tumor (plasma cell granuloma). Clinicopathologic study of 20 cases with immunohistochemical and ultrastructural observations. *Am J Clin Pathol* 1990;94:538–46.
- [15] Coffin CM, Watterson J, Priest JR, *et al.* Extrapulmonary inflammatory myofibroblastic tumor (inflammatory pseudotumor). A clinicopathologic and immunohistochemical study of 84 cases. *Am J Surg Pathol* 1995;19:859–72.
- [16] Hannah CD, Oliver DH, Liu J. Fine needle aspiration biopsy and immunostaining findings in an aggressive inflammatory myofibroblastic tumor of the lung: a case report. *Acta Cytol* 2007;51:239–43.
- [17] Sachdev R, Mohapatra I, Goel S, *et al.* Core biopsy diagnosis of ALK positive inflammatory myofibroblastic tumor of lung: an interesting case. *Turk Patoloji Derg* 2020;36:173–77.
- [18] Qiu X, Montgomery E, Sun B. Inflammatory myofibroblastic tumor and low-grade myofibroblastic sarcoma: a comparative study of clinicopathologic features and further observations on the immunohistochemical profile of myofibroblasts. *Hum Pathol* 2008;39:846–56.
- [19] Leuschner I. Inflammatorischer myofibroblastischer tumor [Inflammatory myofibroblastic tumor]. *Pathologe* 2010;31:106–08.
- [20] Sakurai H, Hasegawa T, Watanabe S, *et al.* Inflammatory myofibroblastic tumor of the lung. *Eur J Cardiothorac Surg* 2004;25:155–59.
- [21] Debonis SA, Bongiovanni A, Pieri F, *et al.* ALK-negative lung inflammatory myofibroblastic tumor in a young adult: a case report and literature review of molecular alterations. *Medicine (Baltimore)* 2021;100:e25972.
- [22] Bando T, Fujimura M, Noda Y, *et al.* Pulmonary plasma cell granuloma improves with corticosteroid therapy. *Chest* 1994;105:1574–75.
- [23] Kovach SJ, Fischer AC, Katzman PJ, *et al.* Inflammatory myofibroblastic tumors. *J Surg Oncol* 2006;94:385–91.
- [24] Su W, Ko A, O'Connell T, *et al.* Treatment of pseudotumors with nonsteroidal antiinflammatory drugs. *J Pediatr Surg* 2000;35:1635–37.
- [25] Chuang JC, Neal JW. Crizotinib as first line therapy for advanced ALK-positive non-small cell lung cancers. *Transl Lung Cancer Res* 2015;4:639–41.