CIRUGÍA ESPAÑOLA

Original article

Inflammatory pseudotumor and myofibroblastic inflammatory tumor. Diagnostic criteria and prognostic differences[☆]

Rommel Carrasco Rodríguez,[a,] Eva María García Fontán,[a] Montserrat Blanco Ramos,[a] Laura Juaneda Magdalena Benavides,[b] Daniel Otero Lozano,[a] Milagros Moldes Rodriguez,[a] Miguel Angel Cañizares Carretero[a]*

[a] *Servicio de Cirugía Torácica, Hospital Álvaro Cunqueiro, Vigo, Pontevedra, Spain*
[b] *Servicio de Anatomía Patológica, Hospital Álvaro Cunqueiro, Vigo, Pontevedra, Spain*

ARTICLE INFO

ABSTRACT

Introduction: Inflammatory pseudotumor (IPT) and inflammatory myofibroblastic tumor (IMT) are two very rare entities that were formerly included in the same category; however, today they are considered two different diseases due to the neoplastic origin of the IMT. Our objective is to share our experience in the management of these two types of tumors that we must take into account in the differential diagnosis of pulmonary masses or nodules.
Methods: Thirteen patients with a pathological diagnosis of IPT and IMT who underwent surgery between 2008 and 2019 were retrospectively studied. We recorded the pre and postoperative information of each one, as well as the survival analysis.
Results: Of the 13 patients, 8 were men and 5 women. The mean age of presentation was 53,5 years. An atypical segmentectomy was performed in 6 patients; a lobectomy was necessary in 6 and a pneumonectomy in 1 case. In all cases a complete resection was achieved. Diagnosis was possible thanks to histology, immunohistochemical (IHQ) and fluorescent in situ hybridization (FISH) techniques determining the expression of IgG4 and the rearrangement of ALK, respectively. After a median follow up of 49 months, we didn't find any locoregional or distant recurrence in the patients studied.
Conclusion: IPT and IMT are rare tumors with a very good prognostic. The diagnosis of both entities is based mainly on specific anatomopathological techniques. Surgery has, in most cases, both a diagnostic and therapeutic role.

Seudotumor inflamatorio y tumor miofibroblástico inflamatorio. Criterios diagnósticos y diferencias pronósticas

R E S U M E N

Palabras clave:
Seudotumor inflamatorio
Tumor miofibroblástico
inflamatorio
Cirugía

Introducción: El pseudotumor inflamatorio (PTI) y el tumor miofibroblástico inflamatorio (TMI) son dos entidades muy poco frecuentes que se incluían antiguamente en la misma categoría; sin embargo, en la actualidad se consideran dos enfermedades diferentes debido al origen neoplásico del TMI. Nuestro objetivo es compartir nuestra experiencia en el manejo de estos dos tipos de lesiones que debemos tener en cuenta en el diagnóstico diferencial de masas o nódulos pulmonares.

Métodos: Fueron estudiados retrospectivamente 13 pacientes con diagnóstico anatomopatológico de PTI o TMI intervenidos entre los años 2008 y 2019. Registramos la información pre y postoperatoria de cada uno, así como el análisis de supervivencia.

Resultados: De los 13 pacientes, 8 eran varones y 5 mujeres. La media de la edad de presentación fue de 53,5 años. En 6 pacientes se practicó una segmentectomía atípica, en 6 fue necesario realizar una lobectomía y en 1 caso una neumonectomía. En todos los casos se consiguió una resección completa. El diagnóstico fue posible gracias a la histología, técnicas de inmunohistoquímica (IHQ) y de hibridación fluorescente in situ (FISH) determinando la expresión de IgG4 y el reordenamiento de ALK, respectivamente. Tras una mediana de seguimiento de 49 meses no se observaron datos de recidiva locorregional ni a distancia en los pacientes estudiados.

Conclusión: El PTI y el TMI son tumores poco frecuentes con muy buen pronóstico. El diagnóstico de ambas entidades se basa principalmente en técnicas anatomopatológicas específicas. La cirugía tiene, en la mayor parte de las ocasiones, un papel tanto diagnóstico como terapéutico.

Introduction

The term 'inflammatory pseudotumor' (IPT) was first described in 1939[1] to identify a heterogeneous group of fibroinflammatory diseases, both reactive and neoplastic in origin. Since then, these tumors have received many names (plasma cell granuloma, pseudosarcomatous fibromyxoid tumor, inflammatory myofibrohistiocytic proliferation, etc)[2,3], all of which reflect their varied cellular composition. Different hypotheses have been posed about the origin of these lesions. Melloni et al.[4] suggest a disproportionate inflammatory reaction to an underlying infectious process. Thus, the possible association with the Epstein-Barr virus[5,6] and herpes virus type 8[7] has also been contemplated.

Inflammatory myofibroblastic tumors (IMT) are characterized by a proliferation of myofibroblasts that are closely related to an inflammatory infiltrate of plasma cells, lymphocytes and eosinophils[8]. They may occur in any anatomical region but show a predilection for the lungs, soft tissue, and abdominopelvic cavity of children and young adults[9,10]. In the past, IMT were classified as benign lesions, although they are currently considered neoplasms of intermediate biological potential due to their ability to recur and produce metastasis[3].

In the past, both IPT and IMT were included in the same category due to similar morphological characteristics from a pathological standpoint, such as fibroblastic-myofibroblastic proliferation and the presence of inflammatory cells. In fact, recently published studies have frequently confused the 2 entities[11,12]. Currently, however, they are considered 2 different tumors due to the neoplastic origin of IMT[3]. Given the small number of cases published in the literature to date, we present our experience from the last 11 years based on a series of patients diagnosed with IPT and IMT who were treated surgically by our service, analyzing diagnosis, treatment and survival.

Methods

From 2008 to 2019, we operated on a total of 13 patients with the final diagnosis of either IPT or IMT. All patients underwent a preoperative fiberoptic bronchoscopy examination. In cases of peripheral location, transthoracic fine-needle aspiration (FNA) was performed for diagnosis. In the 13 cases, the definitive diagnosis of IPT or IMT was reached by pathological study of the resected piece. The data used in the study were collected retrospectively in a coded and anonymized computer database, complying with current Spanish legislation regarding personal data protection and after having received the approval of the hospital ethics committee.

The following variables were studied: age, sex, comorbidity (tobacco consumption and cancer history), clinical manifestations, location of the lesion and morphology on computed tomography (CT) scan, size of the lesion in millimeters, standardized uptake value (SUV) on positron emission tomography (PET), performance of FNA or bronchoscopy,

surgical approach (video-assisted surgery or thoracotomy/sternotomy), type of surgical resection (lobectomy, atypical resection or pneumonectomy), postoperative hospital stay, postoperative complications, immunohistochemical analysis (vimentin, desmin, cytokeratin, smooth muscle actin, EMA, IgG4, and ALK), molecular biology techniques (FISH), and survival.

The statistical analysis was performed using the SPSS® v.24.0 for Windows. Global results are expressed as absolute and relative frequencies for qualitative variables. For quantitative variables, the mean, median and range were used. A P-value <.05 was considered statistically significant.

Results

Among the 13 patients included, 9 cases were pathologically diagnosed as IPT and 4 as IMT. There were 8 males (61.53%) and 5 females (38.46%), with a predominance of males in the IPT group (6/3). The mean age of presentation in the series was 53.5 years, with a median of 48 years (range: 11–82 years), which was 60.1 years in the IPT group and 47.8 years in the IMT group (P = .243). The analysis of comorbidities identified 6 patients (46.15%) who were smokers at the time of diagnosis, while 2 patients had a history of cancer (germ cell tumor and pancreatic gastrinoma).

With regards to clinical presentation, only 2 patients were symptomatic at the time of diagnosis (one case of IMT with dyspnea and one case of IPT with hemoptysis), while in the remaining cases the finding was incidental. No significant laboratory abnormalities were detected in any of the patients studied. By analyzing the radiological characteristics of these tumors on CT, we were able to verify that the lesions were predominantly solid with smooth edges (12/13 cases), and only one case presented cavitation; the median tumor size measured radiologically was 22 mm (range: 8–135 mm). No patient presented signs of tumor invasion of neighboring structures on the imaging studies. PET showed pathological uptake in all the patients studied, with a mean maximum standardized uptake value (SUVmax) of 4.3 g/mL for IPT and 2.6 for IMT (P = .198). The lesions were mainly located in the lower lobes (4 left and 3 right), with a predominance of IPT (71.42%). Although endoscopic samples were taken in 12 of the 13 patients who underwent fiberoptic bronchoscopy (92.30%), the sample was insufficient to reach a diagnosis in all cases. FNA was performed in patients with peripherally located lesions, although it was not diagnostic in any of the cases.

In terms of the surgical technique performed, 6 patients underwent wedge resection (4 IPT and 2 IMT) after intraoperative pathological confirmation of the mass, which was reported as benign disease. In 6 patients, lobectomy was necessary due to the location of the lesion (4 IPT and 2 IMT), and in one patient (IPT) right pneumonectomy was performed through a median sternotomy. This case was an 11-year-old patient who was treated surgically in collaboration with the pediatric surgery service due to his clinical presentation (hemoptysis and hemorrhagic shock), as the thoracic CT scan had revealed a large tumor occupying the entire right hemithorax with signs of intratumoral bleeding, hemothorax and mediastinal displacement (Fig. 1).

Complete tumor resection was achieved in all cases. The most used approach was video-assisted surgery (61.53%). Mean postoperative hospital stay was 3.2 days (range: 3–6 days), with no postoperative complications during this period.

For the pathological study of the resection pieces, tissue sections fixed in formaldehyde and embedded in paraffin were used. Hematoxylin-eosin staining and immunohistochemical (IHC) techniques were performed for vimentin, desmin, cytokeratins, smooth muscle actin, epithelial membrane antigens, IgG4/IgG and ALK (Figs. 2A and B, Figs. 3A and B). Translocation for the ALK gene was also analyzed using the FISH technique (Fig. 3C). The definitive diagnosis of IPT or IMT was based on histopathological, immunohistochemical, and cytogenetic criteria. Four cases were classified as IMT and 9 as IPT. The median tumor size measured in the resection specimen was 14 mm (range: 9–85 mm). The 4 patients diagnosed with IMT were positive for ALK, while the 9 diagnosed with IPT showed IgG4 expression in associated plasma cells (IgG4/IgG ratio), this being one of the diagnostic criteria followed. The main differences between IPT and IMT in our series are listed in Table 1.

After a median follow-up of 49 months, no signs of locoregional or distant recurrence were observed in the patients studied. Only one patient diagnosed with IPT died during follow-up due to an unrelated cause.

Discussion

IPT generally affects older or elderly adults with a mean age of 60–65 years, showing a predominance in men (60%–80%)[13], as we observed in our patients. Meanwhile, some studies have shown that IMT more frequently affects children and young adults[14,15]. However, our series showed a significantly higher mean age (53.5 years). This is explained by the fact that our service does not treat the pediatric population, with the sole exception of the aforementioned case that required our urgent collaboration. Furthermore, we treat a large portion of a rural population with an above-average mean age, which, together with the indolent nature of the symptoms, could explain why our mean age is higher than reports published in the literature. In any case, the small sample size does not allow conclusions to be drawn in this regard.

The clinical presentation of both tumors depends on the location of the lesion and is usually nonspecific (cough, dyspnea, fever, and chest pain). Regarding IPT, Zen et al.[16] found that 53% of patients with pulmonary or pleural involvement had no symptoms at the time of diagnosis, and these patients had been diagnosed incidentally. In our series, more than 80% of patients with IPT were asymptomatic. Some 15%–30% of IMT can present as a constitutional syndrome, with thrombocytosis, polyclonal hyperglobulinemia, elevated erythrocyte sedimentation rate, and growth retardation in children[9]. Out of the 4 patients diagnosed with IMT in our series, only one presented symptoms (hemoptysis), and the preoperative blood test was strictly normal.

The confirmation diagnosis of these tumors is performed by histopathological characterization. In our series and for all cases of both IPT and IMT, the diagnosis was reached through
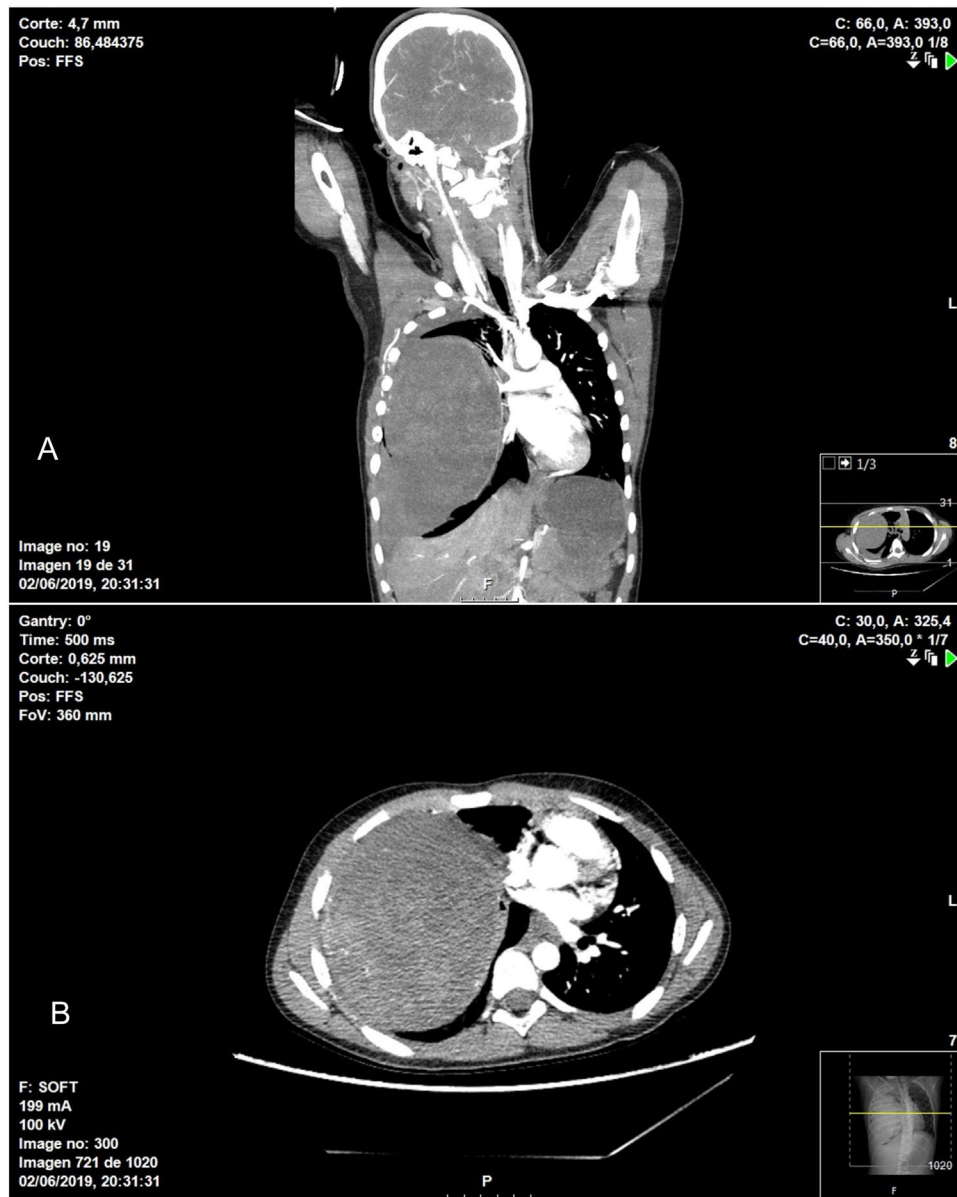
Fig. 1 – Chest CT showing heterogenous intrapulmonary mass associated with moderate hemothorax occupying practically the entire right hemithorax, causing significant mass effect, with complete collapse of the ipsilateral pulmonary vessels in addition to displaced mediastinal structures: A) coronal image; B) axial image.

pathological study of the resection piece after surgery. The suggested cut-off point for the histopathological diagnosis of IPT is the presence of more than 50 IgG4-positive plasma cells per high-power field, IgG4/IgG ratio >0.4[8,17] and serum IgG4 levels greater than or equal to 135 mg/dL[18], although the latter may be altered in other diseases and in up to 5% of the healthy population.

Although preoperative determination of IgG4 may be of interest in screening for this disease, the non-specific nature of the symptoms and imaging tests mean that it does not seem to be a cost-effective diagnostic method, except in cases with a high index of suspicion. In addition, 50%–70% of IMT present translocation in the ALK gene on the short arm of chromosome 2 (2p23) that results in aberrant expression of the ALK

protein in myofibroblasts, which can be detected by immunohistochemistry or fluorescent *in situ* hybridization (FISH)[19–21]. ALK positivity could serve as an exclusion criterion for IPT. However, ALK negativity does not rule out IMT since, as mentioned above, in less than half of IMT, ALK can be negative in IHC or FISH.

Several studies have shown the presence of positive plasma cells for IgG4 in the lymphoid accumulations of IPT, indicating that this type of cells could be related to its pathogenesis[3,22,23]. Likewise, a chromosomal rearrangement has been described that is present in more than half of IMT[8,9,24]. These findings support that IMT and IPT should be considered 2 separate diseases, with different pathogenesis and histological/molecular characteristics.
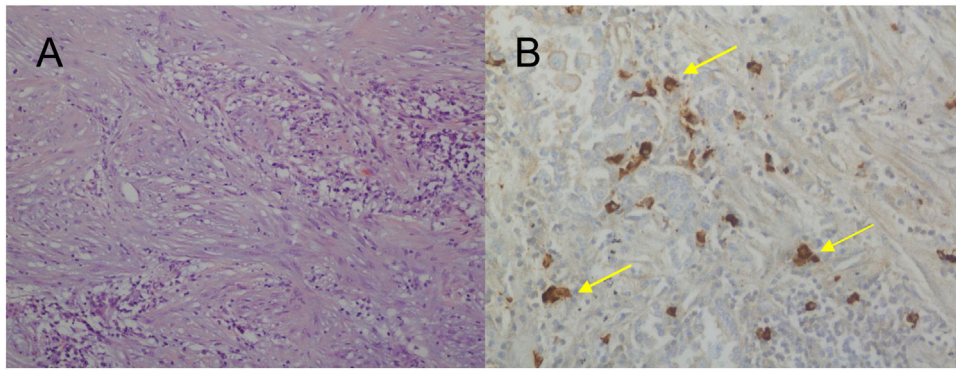
Fig. 2 – A) H-E: proliferation of fibroblasts with no signs of malignant disease and chronic inflammation, interspersed with a predominance of plasma cells; B) Immunohistochemistry: positive plasma cells for IgG4 (yellow arrows).
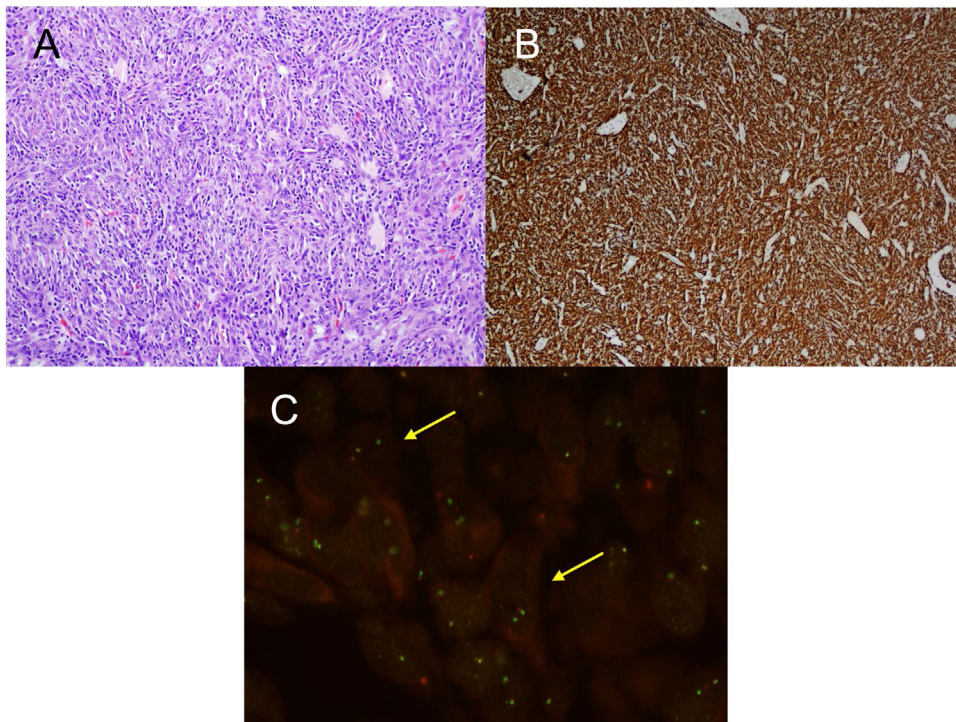

Fig. 3 – A) H-E: fibrohistiocytic cell proliferation, which is organized in short or storiform fascicles, accompanied by prominent inflammatory infiltrate. No necrosis or vascular permeation is observed; B) IHC: immunohistochemistry stain positive for ALK; C) FISH: translocation for ALK gene (yellow arrows).

The natural course of both diseases is unknown since there are no long series published regarding the disease or its treatment. In the case of IPT, the goal of treatment is to prevent fibrosis and irreversible organ damage. Initial therapy with corticosteroids has been shown to be effective for both pulmonary and extrapulmonary disease[25,26]. The same is true for treatment with immunosuppressants[27], including rituximab for cases resistant to corticosteroids and other immunosuppressants[28,29]. In our cases, no corticosteroid therapy was applied prior to surgery because no preoperative diagnosis was available in any patient. Although reports of

spontaneous resolution have been published[30], many authors recommend surgery if the patient does not present contraindications for both diagnosis and treatment, showing 5-year and 10-year survival rates in patients with complete resections of 96% and 90%, respectively[24,31]. Our cases have presented zero mortality without recurrence, as all of them had complete resection.

With regards to IMT, in the past it was classified as a benign lesion, although it is currently considered a neoplasm of intermediate biological potential due to its ability to recur and produce metastasis[3,32,33]. In our series, however, it had a more

Table 1 – Characteristics of the series.		
	IPT	IMT
Age at presentation (years)	60.1	47.8
Sex		
Males	6	2
Females	3	2
Comorbidities		
Tobacco consumption	4	2
Cancer history	2	—
Clinical manifestations		
Dyspnea	—	1
Hemoptysis	1	—
Mean SUV_{max} (mg/dL)	4.3	2.6
Surgical approach		
VATS	6	2
Thoracotomy	2	2
Sternotomy	1	—
Surgical technique		
Lobectomy	4	2
Atypical segmentectomy	4	2
Pneumonectomy	1	—
Median tumor size (mm)[a]	17	14
Immunohistochemistry		
IgG4	9	—
ALK-1	—	4

IPT: inflammatory pseudotumor; IMT: inflammatory myofibroblastic tumor; VATS: video-assisted thoracoscopic surgery.

[a] Measured in the pathological study specimen. As mentioned previously, in less than half of IMT, the expression of ALK can be negative in IHC or FISH.

favorable biological behavior, but the small number of cases makes it necessary to be cautious in this analysis. In any case, surgery is the first-choice treatment in IMT[10,34], with a recurrence rate of less than 2% after complete resection. Even so, no differences were observed in cases of incomplete resection[9], and the 10-year recurrence-free period was 80%[35]. Surgery is also the treatment of choice in recurrences, as long as there are no contraindications[36].

In conclusion, IPT and IMT are rare lesions with different pathogenesis as well as clinical and histological characteristics. Problems may arise when making a differential diagnosis with other lung neoplasms since the diagnosis is based on specific pathological techniques. Surgery has both a diagnostic and therapeutic role; after complete resection, good survival rates and a recurrence-free period are achieved.

Conflict of interests

The authors have no conflict of interests to declare.

REFERENCES

1. Brunn H. Two interesting benign lung tumors of contradictory histopathology. J Thorac Surg. 1939;9:119–31.
2. Gleason BC, Hornick JL. Inflammatory myofibroblastic tumours: where are we now? J Clin Pathol. 2008;61: 428–37.
3. Bhagat P, Bal A, Das A, Singh N, Singh H. Pulmonary inflammatory myofibroblastic tumor and IgG4 inflmmatory pseudotumor: a diagnostic dilemma. Virchows Arch. 2013;463:743–7.
4. Melloni G, Carreta A, Ciriaco P, Arrigoni G, Fieschi S, Rizzo N, et al. Inflammatory pseudotumor of the lung in adults. Ann Thorac Surg. 2005;79:426–32.
5. Loughlin P, Brady A, Devlin E, McManus DT, Spence RA. Epstein-Barr virus positive inflammatory pseudo-tumour of the spleen: a case report and literature review. Int J Surg Case Rep. 2014;5:186–8.
6. Lewis JT, Gaffney RL, Casey MB, Farrell MA, Morice WG, Macon WR. Inflammatory pseudotumor of the spleen associated with a clonal Epstein-Barr virus genome. Case report and review of the literature. Am J Clin Pathol. 2003;120:56–61.
7. Gómez-Román JJ, Sánchez-Velasco P, Ocejo-Vinyals G, Hernández-Nieto E, LeyvaCobián F, Val-Bernal JF. Human herpesvirus-8 genes are expressed in pulmonary inflammatory myofibroblastic tumor (inflammatory pseudotumor). Am J Surg Pathol. 2001;25:624–9.
8. Coffin CM, Dehner LP, Meis-Kindblom JM. Inflammatory myofibroblastic tumor, inflammatory fibrosarcoma, and related lesions: an historical review with differential diagnostic considerations. Semin Diagn Pathol. 1998;15:102–10.
9. Coffin CM, Watterson J, Priest JR, Dehner LP. Extrapulmonary inflammatory myofibroblastic tumor (inflammatory pseudotumor). A clinicopathologic and immunohistochemical study of 84 cases. Am J Surg Pathol. 1995;19:859–72.
10. Panagiotopoulos N, Patrini D, Gvinianidze L, Woo WL, Borg E, Lawrence D. Inflammatory myofibroblastic tumour of the lung: a reactive lesion or a true neoplasm? J Thorac Dis. 2015;7:908–11.
11. Osnaya H, Zaragoza TA, Escoto JA, Mondragón MA, Sánchez RD. Tumor miofibroblastico inflamatorio (pseudotumor inflamatorio) ocasionando abdomen agudo. Rev Chil Cir. 2014;66:264–8.
12. Leiva-Salinas C, Domingo ML, Flors L. Tumor miofibroblastico inflamatorio: un diagnóstico confuso. Arch Bronconeumol. 2010;46:106–7.
13. Yi ES, Sekiguchi H, Peikert T, Ryu JH, Colby TV. Reprint of: pathologic manifestations of Immunoglobulin (Ig) G4-related lung disease. Semin Diagn Pathol. 2018;35:347–51.
14. Roscher AA, Endlich HH. Inflammatory pseudotumors of the lung. Int Surg. 1970;53:268–78.
15. Chan JK, Cheuk W, Shimizu M. Anaplastic lymphoma kinase expression in inflammatory pseudotumors. Am J Surg Pathol. 2001;25:761–8.
16. Zen Y, Inoue D, Kitao A, Onodera M, Abo H, Miyayama S, et al. IgG4-related lung and pleural disease: a clinicopathologic study of 21 cases. Am J Surg Pathol. 2009;33:1886–93.
17. Deshpande V, Zen Y, Chan JK, Yi EE, Sato Y, Yoshino T, et al. Consensus statement on the pathology of IgG4-related disease. Mod Pathol. 2012;25:1181–92.
18. Masaki Y, Kurose N, Yamamoto M, Takahashi H, Saeki T, Azumi A, et al. Cutoff values of serum IgG4 and histopathological IgG4+ plasma cells for diagnosis of patients with IgG4-related disease. Int J Rheumatol. 2012;2012580814.
19. Cessna MH, Zhou H, Sanger WG, Perkins SL, Tripp S, Pickering D, et al. Expression of ALK1 and p80 in inflammatory myofibroblastic tumor and its mesenchymal mimics: a study of 135 cases. Mod Pathol. 2002;15:931–8.
20. Cook JR, Dehner LP, Collins MH, Ma Z, Morris SW, Coffin CM, et al. Anaplastic lymphoma kinase (ALK) expression in the inflammatory myofibroblastic tumor: a comparative

immunohistochemical study. Am J Surg Pathol. 2001;25:1364–71.

21. Yousem SA, Shaw H, Cieply K. Involvement of 2p23 in pulmonary inflammatory pseudotumors. Hum Pathol. 2001;32:428–33.

22. Janik JS, Janik JP, Lovell MA, Hendrickson RJ, Bensard DD, Greffe BS. Recurrent inflammatory pseudotumors in children. J Pediatr Surg. 2003;38:1491–5.

23. Peretti M, Radu DM, Pfeuty K, Dujon A, Riquet M, Martinod E. Surgical resection of pulmonary inflammatory pseudotumors: long-term outcome. Asian Cardiovasc Thorac Ann. 2017;25:440–5.

24. Kubo N, Harada T, Anai S, Otsubo K, Yoneshima Y, Ijichi K, et al. Carboplatin plus paclitaxel in the successful treatment of advanced inflammatory myofibroblastic tumor. Intern Med. 2012;51:2399–401.

25. Kamisawa T, Okazaki K, Kawa S, Ito T, Inui K, Irie H, et al. Working Committee of the Japan Pancreas Society and the Research Committee for Intractable Pancreatic Disease supported by the Ministry of Health, Labour and Welfare of Japan. Amendment of the Japanese Consensus Guidelines for Autoimmune Pancreatitis, 2013 III. Treatment and prognosis of autoimmune pancreatitis. J Gastroenterol. 2014;49:765–84.

26. Khosroshahi A, Wallace ZS, Crowe JL, Akamizu T, Azumi A, Carruthers MN, et al. International consensus guidance statement on the management and treatment of IgG4-related disease. Arthritis Rheumatol. 2015;67:1688–99.

27. Kobayashi H, Shimokawaji T, Kanoh S, Motoyoshi K, Aida S. IgG4-positive pulmonary disease. J Thorac Imaging. 2007;22:360–2.

28. Campbell SN, Rubio E, Loschner AL. Clinical review of pulmonary manifestations of IgG4-related disease. Ann Am Thorac Soc. 2014;11:1466–75.

29. Carruthers MN, Topazian MD, Khosroshahi A, Witzig TE, Wallace ZS, Hart PA, et al. Rituximab for IgG4-related disease: a prospective, open-label trial. Ann Rheum Dis. 2015;74:1171–7.

30. Mandelbaum I, Brashear RE, Hull MT. Surgical treatment and course of pulmonary pseudotumor (plasma cell granuloma). J Thorac Cardiovasc Surg. 1981;82:77–82.

31. Cerfolio RJ, Allen MS, Nascimento AG, Deschamps C, Trastek VF, Miller DL, et al. Inflammatory pseudotumors of the lung. Ann Thorac Surg. 1999;67:933–6.

32. Fletcher CDM, Unni KK, Mertens F. World Health Organization classification of tumors. Pathology and genetics of tumors of soft tissue and bone. Lyon: IACR Press; 2002: 10.

33. Meis JM, Enzinger FM. Inflammatory fibrosarcoma of the mesentery and retroperitoneum. A tumor closely simulating inflammatory pseudotumor. Am J Surg Pathol. 1991;15:1146–56.

34. Hammas N, Chbani L, Rami M, Boubbou M, Benmiloud S, Bouabdellah Y, et al. A rare tumor of the lung: inflammatory myofibroblastic tumor. Diagn Pathol. 2012;7:83.

35. Lawrence B, Perz-Atayde A, Hibbard MK, Rubin BP, Dal Cin P, Pinkus JL, et al. TPM3-ALK and TPM4-ALK oncogenes in inflammatory myofibroblastic tumors. Am J Pathol. 2000;157:377–84.

36. Morar R, Bhayat A, Hammond G, Bruinette H, Feldman C. Inflammatory pseudotumour of the lung: a case report and literature review. Case Rep Radiol. 2012;2012214528.