Arch Bronconeumol. 2010;**46(12)**:652-657

ARCHIVOS DE BRONCONEUMOLOGIA

www.archbronconeumol.org

Review Article

The Clinical View through the Archives: The Clinical Notes of 2009

Daniel Samolski,[a] Irene Martín Robles,[b] Diego Castillo Villegas[b,*]

[a] *Servicio de Neumología, Organización de Servicios Directos Empresarios (OSDE), Buenos Aires, Argentina*
[b] *Servicio de Neumología, Hospital de la Santa Creu i Sant Pau, Barcelona, Spain*

ARTICLE INFO

Article history:
Received October 15, 2010
Accepted October 18, 2010

Keywords:
Clinical case
Respiratory disease
Review

ABSTRACT

The Clinical Notes published in 2009 serve as a resource to reflect on clinical aspects relevant to different clinical entities. Through this review an attempt is likewise made to bring the reader closer to the clinical reality of our environment.

La clínica vista a través de Archivos: las notas clínicas del 2009

Palabras clave:
Caso clínico
Enfermedad respiratoria
Revisión

RESUMEN

Las notas clínicas publicadas en el año 2009 sirven como elemento de trabajo para reflexionar sobre aspectos clínicos relevantes de diferentes entidades clínicas. Mediante esta revisión se pretende asimismo acercar al lector a la realidad clínica de nuestro entorno.

Introduction

The case reports published in *Archivos de Bronconeumología* during 2009 differ with respect to previous years as diffuse interstitial lung diseases (ILD) have had more prominence.[1,2] This finding could be merely anecdotal or cyclical, although it could also be related to the growing interest in this pathology. It could also be simply due to the fact that these tend to be the cases that generate the greatest debate in clinical sessions.

In any case, the combined review of published case reports allows us a better perspective of each one. We can therefore extract two initial conclusions:

1. Pulmonary nodules and masses occasionally present a diagnostic and therapeutic challenge, even more so in special situations, such as single or transplanted lungs. A complete and systematic examination is especially important in these circumstances so that all possible alternatives can be taken into account, since the physician must always keep in mind that a differential diagnosis is fundamental for the study of pulmonary nodules.
2. New causative agents for interstitial lung disease are being discovered with increasing frequency. On occasion, the condition presents itself without a known cause, even though it is presumed to exist. As a result, in the study of these diseases, all associated causes must be ruled out (environmental, genetic, systemic, etc.) in order to treat the underlying condition if possible, and avoid its progression.

* Corresponding author.
E-mail address: dcastillo@santpau.es (D. Castillo Villegas).

0300-2896/$ - see front matter

Case Reports

Pleural Effusion and Pulmonary Hypertension in a Patient With Parkinson's Disease Treated with Cabergoline

Although several different studies have demonstrated the adverse effects of cabergoline in the lungs and pleura, the article by Haro-Estarriol et al is the first to show an unknown adverse effect: pulmonary hypertension.[3]

Cabergoline is a dopamine agonist that is used to treat hyperprolactinemia and Parkinson's disease. Several adverse respiratory side effects have been described in association with its use, such as rhinitis, influenza-like illness, pleural effusion, and pulmonary fibrosis. A study published in this journal in 2007 by Villavicencio et al already indicated this toxicity.[4] Furthermore, this medication can cause cardiovascular side effects with potential pulmonary repercussions, such as valvular diseases and heart failure.

In this clinical case, not only did the patient experience well-known adverse effects, but also severe pulmonary hypertension, which had not been reported previously. There is no absolute certainty of a correlation between pulmonary hypertension and cabergoline use. However, they can be associated with one another to a certain degree since there were no other causative agents for this condition and because the patient's symptoms remitted after treatment was suspended.

The main aim of this study is to understand the unwanted effects of cabergoline in the lungs. Special emphasis should be placed on the need for adequately controlling pneumological symptoms in patients being treated with this drug so that its use can be suspended at an early stage when adverse effects appear, or for evaluating other forms of treatment for patients with a greater risk of complications (i.e. those with history of cardio-pulmonary disease). Furthermore, we must emphasize the responsibility of physicians in informing the rest of the medical community about the unknown adverse effects of the drugs existing in the market.

Choriocarcinoma of the Lung

Corpa Rodríguez et al describe an extremely rare case of a primary lung tumor classified as a "choriocarcinoma of the lung".[5] There are no more than 30 cases similar to this one in the medical literature.

The relationship between a choriocarcinoma and the lungs can appear as a metastasis of a gestational trophoblastic neoplasia, or much more infrequently, can manifest as a primary lung tumor.[6] Furthermore, a choriocarcinoma in the form of an anterior mediastinal mass has also been described.[7]

In this case study, the mass showed the same symptoms as any other pulmonary process (cough, chest pain, hemoptysis) and the diagnosis was obtained by a resection of the compromised lobe. β-HCG was measured postoperatively, showing substantially elevated levels for a non-pregnant patient, further supporting the diagnosis.

Even though these tumors are extremely rare, they must be considered as a differential diagnosis in the study of pulmonary masses, especially in young patients and above all women. Measuring β-HCG is simple and could allow early diagnosis when suspecting this condition in non-pregnant patients. Inclusion of this measure as a tumour marker when studying pulmonary and mediastinal masses should therefore be evaluated, especially in young patients.

Surgical Treatment of Pulmonary Lesions in a Single Lung

Although the number of patients was low, the study by Vázquez Pelillo et al opened the door to surgical treatment, including cure criteria, for single-lung patients who had undergone pneumonectomies due to neoplastic processes (whether primary or secondary).[8] This study included 4 patients who underwent segmentectomy or wedge resection due to the finding of peripheral lesions less than 3 cm in diameter. The patients in the study had a FEV_1 value between 29% and 72%, and an FVC between 51% and 89%. The perioperative mortality rate was 0%, and the only complication occurred in a patient with a persistent air leak that was resolved using a chest tube for a longer duration than usual.

In studies that evaluate preoperative conditions of patients considered candidates for pulmonary resection (normally 2-lung patients), FEV1 and DLCO spirometric values are fundamental, both for preoperative and calculated postoperative values (ppo). Van Tilburg et al found that patients with ppo FEV_1>40% are able to undergo resections up to the calculated value.[9] Furthermore, with respect to wedge resections and segmentectomies, Datta et al suggested that these procedures were safe with FEV_1>0.61.[10] According to these two studies, only one of the patients included by Vázquez Pelillo et al should have been excluded from the surgery, but expressed interest in undergoing the procedure even in light of the implied risk.[8] Lastly, Linden et al also described their experience with pulmonary resections with FEV_1<35%, primarily performing wedge resections.[11]

Obviously, in addition to spirometric values, single-lung patients theoretically present a greater risk of perioperative complications than 2-lung patients. Even the anesthetic technique must be modified, according to the study by Vázquez Pelillo et al.[8]

The most important facet of this study, as well as in others described, is the opening of a therapeutic option for patients with primary or metastatic neoplasia that previously were excluded from this methodology, based on the assumption of an elevated perioperative risk of morbidity or mortality, which was not observed in the practice described.

Three Cases of Primary Pulmonary Plasmacytoma

Only 3% of plasma cell tumors appear as extramedullary plasmacytomas.[12] Among these, most are found in the head and neck, especially in the upper respiratory tract tissues. Furthermore, most test positive for monoclonal IgA. In the case of a pulmonary plasmacytoma, this normally is expressed as a nodule, pulmonary mass, or hilar mass.[13]

The series of 3 cases presented by Montero C et al in this journal acquires its relevance from the extreme rarity of this type of tumour.[14] In each of the 3 cases, radiology showed the tumour as a parenchymal mass, and diagnosis was made through biopsies, which were positive for monoclonal Ig (IgG in one case, IgA in the others). Non-pulmonary findings were also used (electrophoretic proteinogram with a monoclonal band in 2 of the described cases). The response to radiotherapy treatment was positive in all cases, along with the first patient's surgery. Only the third case evolved unfavorably following treatment; this was due to an associated infection.

Even though these tumors are extremely rare, it is important to bear them in mind upon finding a pulmonary mass or nodule, even more so when laboratory analyses suggest a hematological disease.

Solitary Pulmonary Nodule: Primary, Metastatic, or Both?

The finding of a solitary pulmonary nodule in a patient with a history of neoplastic disease does not necessarily imply an association with the primary tumour. In a study of 276 patients who underwent a thoracotomy to excise a nodule, Beckers et al found that the nodule was metastatic from the original neoplastic process in 74.4% of cases, a primary malignant pulmonary tumor in 8.6%, and a benign nodule in 15.2%.[15] The article published in this journal by Obeso Carrillo et al described a particular case of a colon adenocarcinoma (metastasis from a known primary tumor) coexisting with a pulmonary adenocarcinoma.[16] The diagnosis was made through an histopathologic study of the mass obtained from a lobectomy. This procedure was indicated for the nodule that was *a priori* just a metastasis from a colon carcinoma according to the findings from a fine needle aspiration. The following key points should be made regarding this rare case: the potential coexistence of tumors of the same type and from different points of origin, even in the same pulmonary nodule; the importance of an detailed histopathological study to determine therapeutic and prognostic factors; the importance of thoracic imaging tests in the staging of colorectal cancer (20% of carcinomas in this area are metastasized at the moment of diagnosis, and are most frequent in abdomen, liver, lungs, and peritoneum lymph nodes)[17]; and the necessity for a histologic confirmation of the positive PET scan findings (in this case, increased uptake in hilar lesions) that allow for a more appropriate surgical technique, or even ruling out this option based on results.

Pulmonary Epithelioid Haemangioendothelioma

Pulmonary epithelioid hemangioendothelioma (PEHE), also known as intravascular bronchoalveolar tumor, is included among the group of rare and infrequent lung tumors, and is considered to be a low-grade sarcoma. It is observed in females, usually less than 40 years of age, and is shown in imaging tests as multiple or single nodules, whether as perivascular or diffuse enlargements of the interlobar septa. Diagnosis is obtained through immunomarkers of a mass biopsy, when positive for factor VIII and CD34.[18]

The case described by Azcarate Perea et al corresponds to an older woman than those described in the literature. Her condition was found incidentally during a routine radiographic examination.[19] The etiological diagnosis required a lung biopsy by video-assisted thoracoscopy. No oncological treatment was prescribed, as there are no treatment standards for this rare condition, it is multifocal disease, and tumor size had barely enlarged in later clinical and CT follow-ups. According to some authors, the treatment for a unifocal disease is surgical excision. Conventional chemotherapy or treatment with interleukin 2 or interferon $\alpha 2\beta$ could be considered for multifocal diseases, such as in this case. A few isolated cases of spontaneous remission have also been described.

Dermatofibroma Metastasising to the Lung: Current Treatment

Dermatofibromas are described as benign tumorous lesions of the skin characterized by a fibroblastic proliferation. They can occur as a result of a traumatic lesion, an insect bite, or can be idiopathic.

Although they are "benign" lesions, several cases have been described in the literature that have developed metastatic lung involvement, which is demonstrated by the clinical case presented by Bermejo Casero et al published in this journal.[20]

This patient had pulmonary nodules in both upper lobes as shown by a radiological examination performed during the preoperative examination for the resection of a relapsing dermatofibroma. These lesions showed hypercaptation in a PET scan, and were synchronous with other small lesions (of few millimeters) that affected the rest of both lungs. A sequential resection of the pulmonary masses was decided on, and the pathology of the masses coincided with the diagnosis of the cutaneous lesion, indicating that they were lung metastases.

The most important part of this clinical case is to recognize that even "benign" entities can lead to pulmonary metastatic involvement. In these cases, not only is a dermatological examination required, but also tests for spread of the disease when potentially metastatic lesions are suspected.

Left Postpneumonectomy Syndrome. Early Endoscopic Treatment

Postpneumonectomy syndrome is one of the many complications that can arise in the late postoperative period following a pneumonectomy.[21,22] The main characteristic of this condition is that it does not show any clinical manifestations until at least 6 months after the surgical procedure and has even been found to appear up to 35 years after surgery. It consists of compression of the trachea and enlarged bronchi as a consequence of the displacement of the mediastinal structures and hyperinflation of the remaining lung. The cases described in the literature have a tendency to occur in right pneumonectomies. The most common symptoms include: dyspnea, coughing, stridor, and recurrent pneumonia. Among the described treatment plans, those procedures that aim to reposition the mediastinum by filling the postpneumonectomy cavity with various non-reabsorbable materials stand out.[21,23,24]

The case described by Rombolá et al is an exception.[25] Following a left pneumonectomy, the patient experienced intermediate bronchial collapse, causing dyspnea and stridor. The problem was resolved by placing a metallic endoprosthesis in the affected bronchus.

It is important to keep this condition in mind since its clinical manifestation can be similar to basic respiratory disease symptoms (COPD for example), necessitating a CT examination when suspected. Based on the findings, diagnostic methods can be complemented with a respiratory endoscopy. A bronchial prosthesis was placed as treatment for this case. Very few similar cases exist in the literature, and the filling treatments described are most frequently used. It would be appropriate to perform comparative studies of the long-term efficacy of both therapeutic methods.

Rapid Resolution of Nitrofurantoin-Induced Interstitial Lung Disease

Several different drugs are well known to provoke pulmonary toxicity. Among these, the prolonged administration of nitrofurantoin can cause interstitial lung disease and irreversible fibrosis. This is a typical condition in elderly patients receiving chronic treatment with this drug as prophylaxis against recurrent urinary tract infections. Histologically, most cases follow a pattern indicative of chronic interstitial pneumonia. Between 10% and 30% of patients respond to corticosteroid treatment to some degree, but complete remissions are infrequent.

Nuñez Viejo et al published a very interesting case of interstitial lung disease associated with chronic treatment with nitrofurantoin.[26]

The radiological examination of the patient indicated irreversible pulmonary fibrosis. However, a transbronchial biopsy indicated predominantly acute to subacute changes. In fact, the clinical and radiological symptoms were resolved rapidly and unexpectedly upon administering corticosteroids.

Previous publications do exist describing cases of apparently irreversible pulmonary fibrosis experiencing a radiological resolution of the disease in elderly patients that had received nitrofurantoin treatment for 3 years.[27] However, this publication is the first case with a histopathologic study in which rapid recovery was observed in severe interstitial lung disease due to nitrofurantoin treatment.

We must point out the importance of closely monitoring patients under chronic nitrofurantoin treatment, especially parameters for respiratory function, so that treatment can be interrupted at the first signs of change. Lastly, we would like to point out the usefulness of transbronchial biopsy in order to evaluate pulmonary lesions, particularly in cases in which pulmonary biopsies are not possible.

Hypersensitivity Pneumonitis Caused by *Mucor* Species in a Cork Worker

As we are aware, the term hypersensitivity pneumonitis (HP) refers to a heterogeneous group of lung diseases that are characterized by an abnormal immune response to antigens present in a wide variety of organic particles.

Villar et al published an extraordinary case in which *Mucor* species were shown to be the cause of HP.[28] To date, the causative agents for suberosis have been shown to be cork proteins, *P frecuentans*, and *A fumigatus*.[29] However, in spite of the fact that *Mucor* species have previously been linked to the development of this disease by the same group of researchers, its role as a direct causative agent was not shown until this case.[29] Without a doubt, it was the excellent clinical study of the patient, which included SBP, BAL, and TBB, that enabled this causality to be demonstrated. It is important to point out that diagnosis can only be made by performing a detailed and systematic examination of the patient, as in this case. Patients should be advised to avoid contact with the causative agent in routine daily life, since this measure goes unnoticed in many cases.[30]

Furthermore, the atypical bronchial obstruction in this case attracts attention from clinical and functional points of view, since it is very rare in HP, despite the evidence that this form of contracting the disease is not infrequent.[31-33] The most common radiological presentation of HP involves ground-glass opacities and micronodules, as well as the appearance of trapped air zones in expiratory tests. However, the findings from a chest CAT scan can be normal in as much as 16% of cases,[34] such as in this publication.

Hypersensitivity Pneumonitis Caused by *Trichoderma viridae*

The article published by Enríquez-Matas et al describes another clinical case related to the publication in the previous section.[35] This case was of HP due to *T. viridae* contamination from an ultrasonic humidifier in the patient's home. This is the first publication of its kind.

T. viridae is the most common of *Trichoderma* species, a saprophytic filamentous fungi that is all over the world. Only a few cases have been described of infection by *Trichoderma* and *T. viridae* in particular,[36] which is what makes this case so unique.

Furthermore, this case reminds us to always inquire about humidifiers during the patient examination for cases of hypersensitivity pneumonia, since they have frequently been associated with causing the disease.

Familial Pulmonary Fibrosis in 2 Mexican Sisters With Hermansky-Pudlak Syndrome

Hermansky-Pudlak syndrome (HPS) is a rare, recessive autosomal disease that is characterized by albinism, hemorrhagic diathesis, and pulmonary fibrosis.[37] Most cases in the medical literature correspond to patients with Puerto Rican heritage, and until now, no cases of HPS with pulmonary fibrosis have been described in patients of Mexican descent. This is the reason for the major relevance of the cases presented by Zamora et al of 2 Mexican sisters with this combination.[38]

Pulmonary fibrosis secondary to HPS has unique characteristics that distinguish it from normal interstitial pneumonia, such as vacuolar degeneration in type-2 pneumocytes, constrictive bronchiolitis, and microscopic honeycombing that is not necessarily found in the periphery, such as occurs in patients with idiopathic pulmonary fibrosis.[39,40] Above all, this case reminds us that interstitial diseases, can, very occasionally, be related to known genetic abnormalities.

Response to Inhaled Granulocyte-Macrophage Colony-Stimulating Factor in a Patient With Alveolar Proteinosis

Pulmonary alveolar proteinosis (PAP) is a rare disease characterized by the accumulation of lipoproteinaceous material in the alveoli derived from alveolar surfactant, which provokes deterioration in gas exchange. Inflammation is not produced in the airway, and lung architecture is preserved.[41] The commonly accepted treatment that has demonstrated the greatest efficacy in these patients is total pulmonary lavage with saline solution in order to extract the proteinaceous material occupying the respiratory tract, for a clinical and functional improvement. The technique is well standardized, although laborious, as it requires general anesthesia and is not without complications.[42]

Despite the above, Rodrígeuz Portal et al present the interesting case of a patient with PAP. After receiving 2 lavages in a short period of time, the patient's symptoms started to deteriorate rapidly instead of showing the expected improvement; this led to the proposal of an inhaled treatment.[43]

Currently, the use of GM-CSF is accepted for PAP, whether administered intravenously, subcutaneously, or by inhalation, although no randomized studies have been performed that evaluate its efficacy. The subcutaneous approach is the most widely used, but the inhaled method has been shown to be safer and more efficient.[44] In this case, after 12 months of treatment, no side effects have been observed, and the clinical response has been positive.

Diffuse Interstitial Lung Disease Related to Peribronchiolar Metaplasia

Peribronchiolar metaplasia is a histological lesion that is characterized by fibrosis and bronchiolar epithelial proliferation, affecting peribronchiolar alveolar septa and terminal bronchioles. It has been considered as a nonspecific tissue reaction secondary to the action of external factors, such as tobacco smoke and microbes.[45] However, the diagnosis is common yet incidental and with uncertain consequences in several different interstitial lung diseases.[46] Recently,

we have learned of several cases of diffuse interstitial lung disease in which the only histological abnormality was peribronchiolar metaplasia.[46]

Cano-Jimenez et al present us with 2 new clinical cases of interstitial lung disease with typical clinical manifestations and characteristic radiological findings in which the only histopathological finding was the presence of peribronchiolar metaplasia.[47] Furthermore, they take the opportunity to comment on the description from the consensus of the American Thoracic Society/European Respiratory Society in 2002 regarding other clinical and pathological phenomena characterized by bronchiolar involvement with or without peribronchiolar fibrosis andoccur with clinical, radiological, and functional respiratory manifestations typical of idiopathic interstitial pneumonia.[48-52]

Firstly, interstitial pulmonary fibrosis centered in the respiratory tract is a clinical disease with a very poor prognosis, associated in most cases with a background of exposure to environmental or occupational organic and inorganic agents. De Carvalho et al described another entity referred to as centrilobular fibrosis, with histopathological characteristics similar to those caused by aspiration of gastric contents in patients with no background of reflux.[50] Another entity described with similar histological characteristics to those of hypersensitivity pneumonitis, except for the absence of interstitial granulomas, is idiopathic bronchiolocentric interstitial pneumonia.[49,52]

Centers of bronchiolar metaplasia were observed in these 3 clinical entities. Furthermore, the possibility exists that these 3 entities, along with bronchiolar metaplasia associated with interstitial lung disease, could be different stages of the same disease with involvement limited to the bronchiolar areas, although we cannot rule out that they are in fact well-differentiated clinical entities.

Conflict of Interest

The authors affirm that they have no conflicts of interest.

References

1. Castillo Villegas D. La colección de mariposas de *Archivos de Bronconeumología*. Arch Bronconeumol. 2008;44:546-50.
2. Sarmiento Méndez LM, Castillo Villegas D. La clínica vista a través de Archivos. Arch Bronconeumol. 2009;45:516-20.
3. Haro Estarriol M, Sabater Talaverano G, Rodriguez Jerez F, Obrador Lagares A, Genís Batlle D, Sendra Salillas S. Derrame pleural e hipertensión pulmonar en un paciente con enfermedad de Parkinson en tratamiento con Cabergolina. Arch Bronconeumol. 2009;45:100-2.
4. Villavicencio C, Ramírez A, Gayete A, Grau S, Orozco M. Toxicidad pleuropulmonar precoz asociada al tratamiento con cabergolina, un fármaco antiparkinsoniano. Arch Bronconeumol. 2007;43:519-22.
5. Corpa Rodríguez ME, Fernández Lahera J, Guadalajara Labajo H, Vázquez Pelillo JC, Nistal Martin de Serrano M, García Sánchez-Girón J. Coriocarcinoma de pulmón. Arch Bronconeumol. 2009;45:153-5.
6. Libshitz HI, Baber CE, Hammond CV. The pulmonary metastases of choriocarcinoma. Obstet Gynecol. 1977;49:412.
7. Knapp RH, Hurt RD, Payne WS, Farrow GM, Lewis BD, Hahn RG, et al. Malignant germ cell tumors of the mediastinum. J Thorac Cardiovasc Surg. 1985;89:82-8.
8. Vázquez Pelillo JC, Corpa Rodríguez ME, Gil Alonso JL, Diaz Agero Álvarez P, Verdu RV, García Sánchez Giron J. Tratamiento quirurgico de lesiones pulmonares en pulmon único. Arch Bronconeumol. 2009;45:252-6.
9. Van Tilburg PMB, Stam H, Hoogsteden HC, Van Klaveren RJ. Pre-operative pulmonary evaluation of lung cancer patients: a review of the literature. Eur Respir J. 2009;33:1206-15.
10. Datta D, Lahiri B. Preoperative evaluation of patients undergoing lung resection surgery. Chest. 2004;123:2096-103.
11. Linden P, Bueno R, Colson Y, Jaklitsch M, Lukanich J, Mentzer S, et al. Lung resection in patients with preoperative FEV_1<35%. Chest. 2005;127:1984-90.
12. Shih LY, Dunn P, Leung WM, Chen WJ, Wang PN. Localised plasmacytomas in Taiwan: Comparison between extramedullary plasmacytoma and solitary plasmacytoma of bone. Br J Cancer. 1995;71:128.
13. Haresh KP, Sharma DN, Prabhakar R, Naik KK, Mannan AA, Julka PK, et al. Plasmacytoma of bronchus treated by radical radiotherapy–a rare case with four and a half years follow up. J Thorac Oncol. 2007;2:980-2.
14. Montero C, Souto A, Vidal I, Fernández MM, Blanco M, Verea H. Plasmocitoma pulmonar primario. Aporte de 3 casos. Arch Bronconeumol. 2009;45:564-6.
15. Beckers F, Ludwig C, Cerinza J, Stoelben E. Pulmonary nodules in underlyng malignant diseases: not necessarily metastases of the primary tumour. Pneumologie. 2009;63:693-6.
16. Obeso Carrillo GA, Rivo Vázquez JE, Cañizares Carretero MA, García Fontan E, Blanco Ramos M, García Tejedor JL. NÓdulo pulmonar solitario: ¿primario, metastasico o ambos? Arch Bronconeumol. 2009;45:567-9.
17. Jemal A, Siegel R, Ward E, Hao Y, Xu J, Murria T, et al. Cancer statistics, 2008. CA Cancer J Clin. 2008;58:71.
18. Cronin P, Arenerg D. Pulmonary epitheloid hemangioendoyhelioma: an unusual case an a review of the literature. Chest. 2004;125:789-93.
19. Azcarate Perea L, Oliveros Acebes E, Moreno Mara N, Salomon Perez R, Vilalta Castel E, González Aragoneses F. Hemangioendotelioma epiteloide pulmonar. Arch Bronconeumol. 2009;45:466-8.
20. Bermejo Casero E, Pérez Alonso D, Quevedo Losada S, López Rivero L. Dermatofibroma y metastasis pulmonares. Tratamiento actual. Arch Bronconeumol. 2009;45:521-3.
21. Regnard JF, Pouliquen E, Magdeleinat P, Sohier L, Gourden P, Gharbi N, et al. Postpneumonectomy syndrome in adults: description and therapeutic propositions apropos of 8 cases. Rev Mal Respir. 1999;16:1113-9.
22. Kelly RF, Hunter DW, Maddaus MA. Postpneumonectomy syndrome after left pneumonectomy. Ann Thorac Surg. 2001;71:701-3.
23. Valji AM, Maziak DE, Shamji FM, Matzinger FR. Postpneumonectomy syndrome: recognition and management. Chest. 1998;114:1766-9.
24. Birdi I, Bughai M, Wells FC. Surgical correction of postpneumonectomy stridor by saline breast implantation. Ann Thorac Surg. 2001;71:1704-6.
25. Rombola C, León Atance P, Honguero Martínez AF, Rueda Martínez JL, Núñez Ares A, Vizcaya Sánchez M. Síndrome posneumonectomia izquierda. Tratamiento endoscopico precoz. Arch Bronconeumol. 2009;45:617-9.
26. Núñez Viejo MA, Fernández Montes A, Velasco Montes J, Gómez-Román JJ, García Ibarbia C, Hernández Hernández JL. Nitrofurantoína y enfermedad pulmonar intersticial de rápida resolución. Arch Bronconeumol. 2009;45:352-5.
27. Sheehan RE, Wells AU, Milne DG, Hansell DM. Nitrofurantoin-induced lung disease: two cases demonstrating resolution of apparently irreversible CT abnormalities. J Comput Assist Tomogr. 2000;24:259-61.
28. Villar A, Muñoz X, Cruz MJ, Morell F. Neumonitis por hipersensibilidad a Mucor sp. en un trabajador de la industria del corcho. Arch Bronconeumol. 2009;45:405-7.
29. Morell F. Alveolitos alérgica extrínseca. In: Morell F, Editor. Pneumológica. Pautas, datos y técnicas en medicina respiratoria (8th. Ed). Barcelona: Masson; 2007.
30. Sogo A, Morell F, Muñoz X. Neumonitis por hipersensibilidad en una planchadora. Arch Bronconeumol. 2009;45:258-9.
31. Small JH, Flower CD, Traill ZC, Gleeson FV. Air-trapping in extrinsic allergic alveolitos on computed tomography. Clin Radiol. 1996;51:684-8.
32. Bourke S, Anderson K, Lynch P, Boyd J, King S, Banham S, et al. Chronic simple bronchitis in pigeon fanciers. Chest. 1989;95:598-601.
33. Bourke SJ, Carter R, Anderson K, Boyd J, King S, Douglas B, et al. Obstructive airways disease in non-smoking subjects with pigeon fancier?s lung. Clin Exp Allergy. 1989;19:629-32.
34. Lacasse Y, Selman M, Costabel U, Dalphin JC, Ando M, Morell F, et al. Clinical diagnosis of hypersensitivity pneumonitis. Am J Respir Crit Care Med. 2003;168:952-8.
35. Enríquez-Matas A, Quince S, Cubero N, Sastre J, Melchor R. Neumonitis por hipersensibilidad causada por Trichoderma viride. Arch Bronconeumol. 2009;45:304-5.
36. Chouaki T, Lavarde V, Lachaud L, Raccurt CP, Hennequin C. Invasive Infections due to Trichoderma species: report of 2 cases, findings of in Vitro susceptibility testing, and review of the literature. Clin Infect Dis. 2002;35:1360-7.
37. Witkop CJ, Nunez Babcock M, Rao GH, Gaudier F, Summers CG, Shanahan F, et al. Albinism and Hermansky-Pudlak syndrome in Puerto Rico. Bol Asoc Med PR. 1990;82:333-9.
38. Zamora AC, Alonso-Martínez D, Barrera L, Mendoza F, Gaxiola M, Carrillo G. Fibrosis pulmonar familiar en 2 hermanas mexicanas con síndrome de Hermansky-Pudlak. Arch Bronconeumol. 2009;45:408-10.
39. Nakatini Y, Nakamura N, Sano J, Inayama Y, Kawano N, Yamakana S, et al. Interstitial pneumonia in Hermansky-Pudlak síndrome: significance of florid foamy swelling/degeneration (giant lamellar body degeneration) of type-2 pneumocytes. Virchows Arch. 2000;437:304-13.
40. Thomas de Montprèville V, Mussot S, Dulmet E, Dartevelle P. Pulmonary fibrosis in Hermansky-Pudlak syndrome is not fully usual. Ann Pathol. 2006;26:445-9.
41. Trapnell BC, Whitsett JA, Nakata K. Pulmonary alveolar proteinosis. N England J Med. 2003;349:2527-39.
42. Claypool WD, Rogers RM, Matuschak GM. Update on the clinical diagnosis, management and pathogenesis of pulmonary alveolar proteinosis (phospholipidosis). Chest. 1984;85:550-8.
43. Rodríguez Portal JA, Rodríguez Becerra E, Sánchez Garrido A. Proteinosis alveolar. Respuesta al tratamiento con factor estimulante de colonias de granulocitos y macrófagos por vía inhalada. Arch Broconeumol. 2009;45:150-2.
44. Wylam ME, Ten R, Prakash UB, Nadrous HF, Clawson ML, Anderson PM. Aeorosol granulocyte-macrophage colony-stimulating factor for pulmonary alveolar proteinosis. Eur Respir J. 2006;27:585-93.
45. Colby TV. Bronchiolitis: pathologic considerations. Am J Clin Pathol. 1998;109:101-9.

46. Fukuoka J, Franks TJ, Colby TV, Flaherty KR, Galvin JR, Hayden D, et al. Peribronchiolar metaplasia: a common histologic lesion in diffuse lung disease and a rare cause of interstitial lung disease. Am J Surg Pathol. 2005;29:948-54.
47. Cano-Jiménez E, Molina-Molina M, Ramírez J, Sánchez M, Aliaga JL, Xaubet A. Enfermedad pulmonar intersticial difusa por metaplasia peribronquiolar. Arch Bronconeumol. 2009;45:57-9.
48. Churo A, Myers J, Suárez T, Gaxiola M, Estrada A, Mejía M, et al. Airway-centered interstitial fibrosis: a distinct form of aggressive diffuse lung disease. Am J Srg Pathol. 2004;28:62-8.
49. Yousem SA, Dacic S. Idiopathic bronchiolocentric interstitial pneumonia. Mod Pathol. 2002;15:1148-53.
50. De Carvalho M, Kairalla R, Capelozzi V, Deheinzekin D, Do Nascimento PH, De Carvalho C. Centrilobular fibrosis: a novel histological pattern of idiopathic interstitial pneumonia. Pathol Res Pract. 2002;198:577-83.
51. Cordier JF. Challenges in pulmonary fibrosis. 2: Bronchiolocentric fibrosis. Thorax. 2007;62:638-49.
52. Muñoz A, Aranda I, Pascual J, Ferrando C. Neumonía intersticial bronquiolocéntrica idiomática: una nueva neumonía intersticial idiomática. Arch Bronconeumol. 2007;43:464-6.