Pulmonary Hyalinizing Granuloma☆

Granuloma pulmonar hialinizante

Dear Editor,

Pulmonary hyalinizing granuloma is an uncommon lung disease. Its etiopathogenesis is unknown, although it could be associated with an autoimmune or previous infectious process. The definitive diagnosis is provided by the histopathologic study of the lesion. We describe the case of a patient with a history of tuberculosis and with a pulmonary nodular lesion that was determined to be a metastatic neoplastic process.

Our patient is a 66-year-old male who smoked a pack a day. His pathological history of interest included childhood tuberculosis and necrotizing granulomatous lymphadenitis in a supraclavicular lymph node in 1997 with a PCR that was negative for mycobacteria. The patient presently came to our hospital due to respiratory difficulty and a feverish syndrome that had been evolving over several days. Imaging tests demonstrated the presence of an intraparenchymatous nodule in the lower right lobe that was determined to be a neoplastic process. Surgical resection obtained a segmentectomy piece from the right lower lobe that was 6 cm × 3 cm at the largest diameters. Upon resection, a nodular lesion was observed that was whitish in color with well-defined edges, measuring 9 mm at its maximal diameter. Microscopically, the lesion was non-encapsulated, with well-defined edges, made up of central extracellular laminae that were concentric, eosinophilic, congo red negative, and organized around small-caliber blood vessels (Fig. 1). In the periphery of the lesion, we identified a reactive lymphocytic population made up of lymphoid follicles with reactive germinal centers (Fig. 1). We observed patches of multinucleated giant cells, whose presence was related with an active process of the disease. The PAS, Giemsa and Ziehl-Nielsen stained identified no microorganisms. With these data, the diagnosis was determined to be pulmonary hyalinizing granuloma. This pathology was described for the first time in 1977 by Engleman et al.,[1] who postulated that PHG represented an exaggerated immune response against infectious processes such as tuberculosis and histoplasmosis, likewise demonstrating that sclerosing mediastinitis, retroperitoneal fibrosis, rheumatoid arthritis, posterior uveitis and optic papillitis were diseases associated with PHG because they all presented the same active response against infectious agents. Schlosnage et al.[2] supported this theory by demonstrating the presence of different antibodies and circulating immune complexes in patients with PHG. Some authors,[3,4] related the presence of reactive lymphocyte proliferation in the periphery of the lesion with lymphoproliferative processes such as multiple myeloma, lymphoma and plasma-cell type Castleman's disease. Our case did not present any of these lymphoid pathologies but did present a history of childhood tuberculosis, which would mean that its etiology may have been of the infectious type. Areas of ischemic necrosis, cavitations, bone metaplasia and calcifications are rare.[5] 25% of cases represent a coincidental finding in asymptomatic patients who present uni- or bilateral nodules and whose size does not usually surpass 4 cm in maximum diameter. Its presentation as a single nodule, as the present case, is uncommon, a reason why many times it is misinterpreted as a solitary metastatic nodule. Both the histologic study and immunohistochemistry offer us sufficient data in order to differentiate between the two.
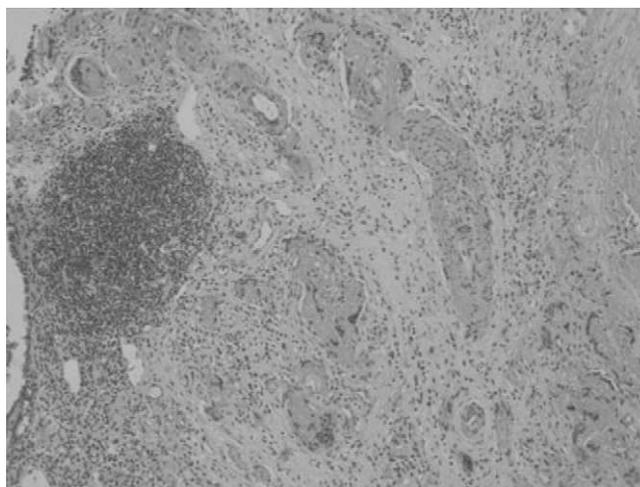

Fig. 1. Histopathologic sample with concentric eosinophilic structures around blood vessels and the peripheral area in which a reactive lymphoid component can be observed.

Within the group of intraparenchymatous primary tumors that present as nodular lesions, we can highlight solitary fibrous tumors, which are subpleural and show positivity for CD34 and BCL-2, and inflammatory myofibroblastic tumors, which usually first affect the pleura and later the adjacent mediastinum. Histologically, they present cell proliferation with myofibroblastic differentiation and chronic inflammatory infiltrates, distributed in a densely sclerosing stroma similar to PHG, although in the latter the stroma is made up of concentric hyalinized structures. Within the benign pathologies that present as intraparenchymatous nodules, the most striking is nodular pulmonary amyloidosis because it presents histologic characteristics similar to our case, but clinically it does not extend to the mediastinum. Although sometimes PHG may present positivity to congo red stain just like amyloidosis, Guccion et al.[4] demonstrated a fibrillar aspect of the deposit of amyloid with electron microscopy, unlike PHG, which is denser and homogeneous. There are other benign pulmonary pathologies that can be included in the extensive list of differential diagnoses, but each one is characterized by presenting specific symptoms and histologic characteristics. Among these are pulmonary rheumatoid nodule disease, Wegener's granulomatosis, sarcoidosis, histoplasmosis and last of all pulmonary eosinophilic granuloma or Langerhans cell granulomatosis.

To summarize, PHG is an uncommon lung pathology included within the large variety of benign or malignant lung lesions. The symptoms are not very specific, the evolution is slow and the possible association with other diseases requires a histopathologic study in order to correctly diagnose this disease.

References

1. Engleman P, Liebow AA, Gmelich PJ. Pulmonary hyalinizing granuloma. Am Rev Respir. 1977;115:977–1008.
2. Schlosnagle DC, Check IJ, Sewell CW, Plummer A, York RM, Hunter RL. Immunologic abnormalities in two patients with pulmonary hyalinizing granuloma. Am J Clin Pathol. 1982;78:231–5.
3. Ren Y, Raitz EAN, Lee KR, Pingleton SK, Tawfik O. Pulmonary small lymphocytic lymphoma. Chest. 2001;120:1027–30.
4. Guccion JG, Rohatgi PK, Saini N. Pulmonary hyalinizing granuloma, electron microscopic and immunologic studie. Chest. 1984;85:571–3.
5. Ramirez J, Mehta JB, Taylor RA, Byrd Jr RP, Roy TM. Symptomatic pulmonary hyalinizing granuloma. South Med J. 1998;91:867–9.

Loubna Boutayeb,* Miriam Gené-hijós, Emilio Mayayo

Servicio de Patología, Hospital Universitario de Tarragona Joan XXIII, Universidad Rovira i Virgili, Tarragona, Spain

* Corresponding author.
E-mail address: lboutayeb@hotmail.com (L. Boutayeb).

Complex Bronchovascular Reconstruction: The Description of a New Segmental Bronchoplasty Technique☆

Reconstrucción broncovascular compleja. Descripción de una técnica novedosa de broncoplastia segmentaria

Dear Editor,

The development of bronchial and vascular reconstruction techniques enables us to carry out lung resections that save the parenchyma. In 1947, Price-Thomas[1] performed the first sleeve lobectomy in a bronchial adenoma. Later, Allison[2] in 1959 reported the first case in a patient with carcinoma while also performing reconstruction of the pulmonary artery that had been infiltrated by the tumor.

Our patient is a 78-year-old male with a smoking history of 80 pack-years who had developed COPD and was being treated with bronchodilators. On the chest CT, we observed a 4-cm tumor in the LUL that was in close contact with the bronchus of the lower lobe. Bronchoscopy demonstrated that the tumor was obstructing the upper left bronchus and extended toward the bronchus of the LLL. On PET, the lesion presented an increased metabolism with bilateral perihilar and hypermetabolic mediastinal lymphadenopathies. Ultrasound-guided bronchoscopy ruled out metastasis in said lymph node stations.

FEV_1 was 1.610 l (61%) and diffusion was 65%. A ventilation/perfusion scintigraphy showed a perfusion of 45% in the left lung.

The surgical approach entailed posterolateral thoracotomy through the 5th intercostal space, observing that the tumor retracted the scissure without overpassing it. Two lobar branches of the pulmonary artery were seen to be infiltrated at their ends; therefore we carried out proximal and distal clamping after anticoagulation with 4000 units of heparin. Later, we proceeded with the sleeve dissection of the main bronchus and bronchus of the LLL. In order to achieve an edge that was free from microscopic infiltration, it was necessary to extend the surgical margin toward the LLL with separation of the bronchus from the sixth segment and the basal pyramid (Fig. 1C). Once the lobectomy was completed, we started to reconstruct the artery with a direct suture and we then approached the bronchus. The bronchial anastomosis was done with a continuous lateral suture between the bronchus of the sixth segment and that of the basal pyramid using a 4/0 reabsorbable suture (Fig. 1F). Previously, we performed a small lateral wedge incision (Fig. 1D and E). Both bronchi joined together were anastomosed to the left main bronchus with a 3/0 reabsorbable suture using individual sutures (Fig. 1G). The discrepancy in calibers was corrected by confronting both bronchi without telescoping. A flap of pericardial fat was placed over the bronchial suture. We carried out lymphadenectomy of the prevascular, aortopulmonary window
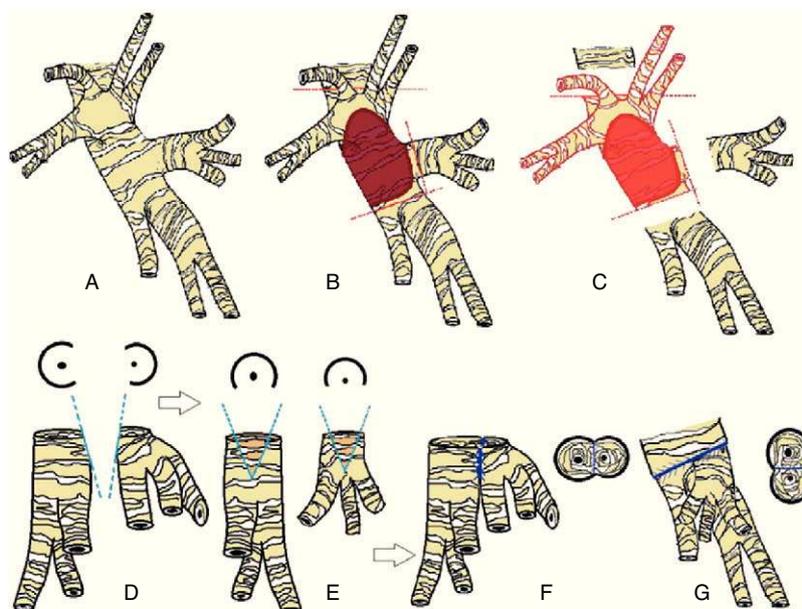

Figure 1. (A) Anatomy of the left bronchus, lateral view. (B) Area of bronchial infiltration. (C) Resection of the affected bronchial area separating the segmental bronchi of the LLL. (D and E) Wedge incision over the bronchus of the basal pyramid and the bronchus of segment six. (F) Lateral anastomosis between segmental bronchi, lateral view. (G) Anastomosis of segmental bronchi to left main bronchus, posterior view.