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Figure 1. Above, therapeutic thoracocentesis procedure aided by wall suction. Below, angiocath fenestrated with a scalpel at its distal third and connected to the suction tube by means of a 3 ml syringe.

Acinar Cell Carcinoma of the Lung

Carcinoma de células acinares de pulmón

To the Editor:

A 55 year old male, smoked 2 packs of cigarettes and drank 100 g of alcohol per day, with a history of arterial hypertension, grade IV chronic arterial ischemia in lower limbs, chronic bronchitis, alcoholic and deficiency neuropathies, twice admitted to hospital to be studied for constitution syndrome. The first time, in May 2005, the patient underwent fibrogastroscopy, fibrocolonoscopy, abdominal ultrasound and thoracoabdominal computerized tomography (CT), and a 1 cm indeterminate nodule was found in the lingula. For this reason he was admitted again in December 2006, undergoing another CT scan which showed an area of consolidation of pseudonodular morphology in the lingula, with an air bronchogram inside, of 2 cm in diameter. Both hospital admissions were attributed to disorders due to alcohol dependence. To test the lesion in the lung he was referred to the pneumology department, which recommended a PET scan and functional breathing tests, which the patient refused to undergo for personal reasons. He went to hospital after suffering progressive dyspnoea for 10 days, during which time he suffered no fever and there was no increase in his typical cough. On arrival at the casualty department he was normotensive, afebrile, showed signs of chronic alcohol abuse, and had cardiac arrhythmia at 100 bpm, general hypophonesis in left hemithorax, pain in right hypochondrium, hepatomegaly to 3 fingers breadth, and bilateral horizontal nystagmus. The electrocardiogram revealed auricular fibrillation at 126 bpm, QRS axis at 0° and left bundle branch block. Analysis showed a high MCV and parameters of inflammatory activity. The chest x ray revealed a mass in the left parahilar region with a loss of left lung volume. A chest CT scan revealed a mass of about 3 cm in the upper left lobe, with peribronchovascular thickening in the ipsilateral pulmonary hilus and significant mediastinal and contralateral lung hilar adenopathy. These findings strongly suggested pulmonary neoplasia affecting the ipsilateral and contralateral mediastina, and left-sided pleural effusion with nodular thickening of the pleura of the left costophrenic sinus, suggestive of tumour implants (fig. 1). A diagnostic thoracentesis provided exudative pleural fluid, according to Light's criteria. The study was completed with a bronchoscopy, which showed extensive, very friable lesions covering the whole of the left bronchial tree, suggesting infiltration and resulting in complete occlusion of the lingula; in the right bronchial tree the same, but more diffused, lesions were observed from the main bronchus. Both the results

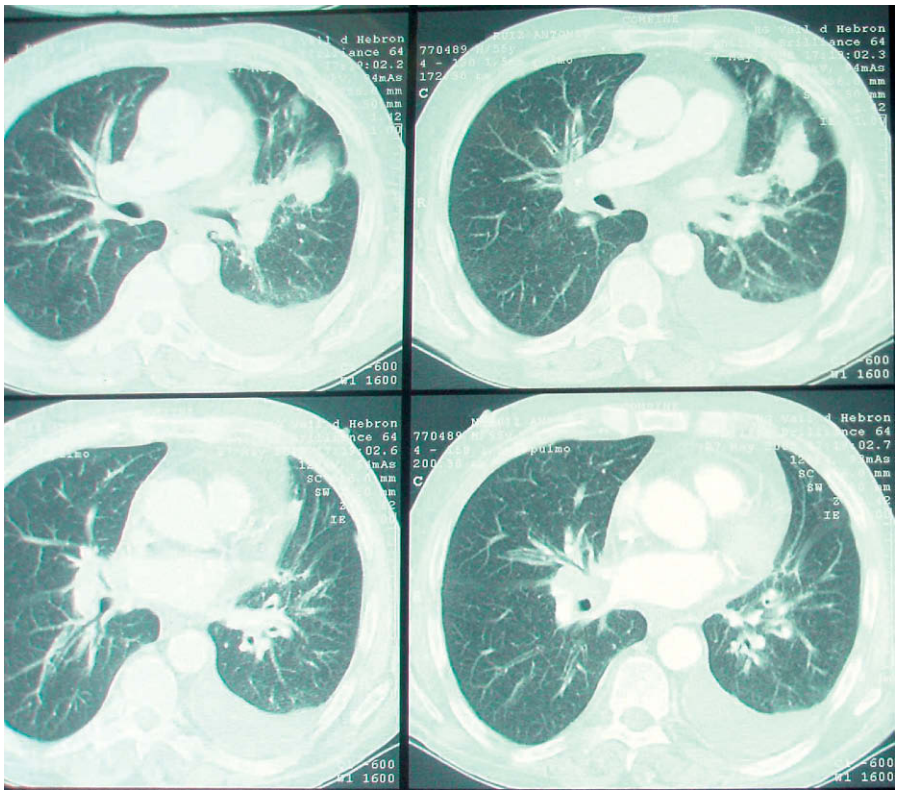

Figure 1. Chest CT (2008): mass of approximately 3 cm in ULL. Peribronchovascular thickening in the ipsilateral pulmonary hilus. Significant mediastinal and contralateral hilar adenopathies. Left-sided pleural effusion with nodular thickening of the left costophrenic sinus.

from the biopsy of the left main bronchus and the cytological study of the pleural fluid were positive for acinar cell carcinoma (Fechner tumour). The chest tumour committee considered that the patient should only be given symptomatic and palliative treatment. There was a progressive increase in the patient's dyspnoea. A chest x-ray revealed a white left lung, secondary to atelectasis and pleural effusion. Evacuation thoracentesis was carried out, but with no subsequent re-expansion. The patient was transferred to a palliative care centre, where he died a few weeks later.

There are tumours which are histologically identical to salivary gland neoplasias which occasionally arise in the tracheobronchial tree and lungs. The most commonly found are cystic adenoid carcinomas and mucoepidermoid carcinomas, followed by mixed tumours. Oncocytomas and acinar cell carcinomas are extremely rare. They constitute less than 1% of primary lung tumours. Although their growth is typically slow, they often invade adjacent tissue and can metastasize, often after several years.[1,2] Acinar cell carcinomas (ACC), first described by Fechner et al in 1972, are mainly found in the parotid gland, but occasionally have other origins, such as the small salivary glands of the anterior nasal mucosa, paranasal sinuses, jaw, chest, lymphatic glands or lungs.[1,2] It originates in pluripotential cells of mucosa and serosa glands of the submucosa of the tracheobronchial tree (analogous to major and minor salivary glands).[3] The diagnosis of ACC of the lungs requires a histological study, as it can not be based on clinical symptoms or x-rays alone. A simple x-ray or CT scan usually shows a well-delimited nodule, so it can be confused with a benign lesion.[3] After diagnosing ACC, it is very important to rule out the existence of a primary origin in a salivary gland (parotid, above all), since it is characterized by an indolent course and can metastasize after prolonged latency.[1] Furthermore, it must be taken into account that the lungs, followed by the bone, are the main sites of haematogenous dissemination of salivary gland ACC.[3,4] Histological, immunohistochemical and electron microscope examinations are important for the definitive diagnosis. Regarding the histological examination, the stains are characterized by sheets of acini of large polyhedral cells, with a round, uniform nucleus and abundant granular cytoplasm. As for the immunohistochemical study, there is a series of immunoreactivity patterns which guide the diagnosis. The differential diagnosis must be established, mainly with other endobronchial lesions in which the neoplastic cells have a granular cytoplasm, such as oncocytic carcinoid tumours, granular cell tumours of the bronchi and metastatic renal-cell carcinomas. A differential diagnosis must also be established with primary adenocarcinomas of the lung with clear cell changes, benign clear cell tumours (sugar tumour) of the lung and bronchial oncocytomas.[1-3,5] Regarding the prognosis, the adverse factors have been described as the presence of mitotic activity, perineural invasion and lymph node metastasis.[2,6] Therefore, a potential malignancy must be taken into account, which is why the WHO changed the term tumour for carcinoma in 1991, based on its potential for local recurrence and metastasizing.[1,6] Among the cases described, this would seem to be the first death reported. Previously published cases were treated with resection of the trachea or lung, together with staging of the lymph nodes, which seems to produce good results. However, the role of radio- and chemotherapy as either an initial or adjuvant therapy for a more advanced stage of the disease is unknown.[1,3,5,6]

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