nance imaging (MRI),[1,2] helps to visualize the lesion and point to its vascular origin, although these tests are not diagnostic in themselves.

Pericytomas tend to behave unpredictably.[5,6] In the case of our patient, after the initial diagnosis of angioma, the lesion grew slowly without showing any clinical signs or symptoms that would suggest malignancy. Findings that suggest a more aggressive behavior are: size greater than 10 cm (with a 66% probability of metastasis), the existence of more than 3 mitoses per field, necrosis, pleural invasion, and vascular invasion.[7] In our case, despite the size of the lesion (8.5×4.5 cm), the absence of necrosis or mitosis on the pathology study suggested a lack of aggressive behavior up to the time of diagnosis. It is clear however that, since it was a large central endobronchial vascular tumor, the risk of potentially fatal local complications was high.

Treatment of tumors of this type is based primarily on surgical resection of the lesion, if possible.[8] Prior embolization of the feeder arterial branches is always advisable. Postoperative radiation therapy also plays an important role in lesions of this type, while chemotherapy appears to have no clear benefit and is reserved for selected cases and always administered with palliative intent.

In our case, the site of the lesion and the treatments received earlier ruled out surgical resection, so we opted for tumor embolization, implantation of a silicone Dumon Y stent, and external radiation therapy. This prosthesis had to be removed 6 months later due to intractable cough, after which successive bronchial dilation procedures were performed using rigid bronchoscopy, during the last of which massive bronchial bleeding occurred due to rupture of the tumor mass that resulted in the death of the patient.

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Ambulatory Fibrinolysis in the Management of Septated Malignant Effusions☆

Fibrinólisis ambulatoria en el manejo del derrame maligno multiseptado

Dear Editor:

Malignant pleural effusion (MPE) is a complication of advanced cancer, and has an estimated incidence of 1/1000 people per year.[1] It is predicted that the prevalence of MPE will increase in the next few years due to the greater survival of patients with active tumors.

Cure rates in MPE are low, and in most cases the effusion is recurrent. Onset occurs with increasing dyspnea, cough, chest pain, and loss of quality of life, so different therapeutic techniques with palliative intent are used. Pleurodesis was the technique of choice for many years, but tunneled pleural drainage (TPD) is now gaining more prominence in clinical practice.[2–4] In the follow-up of tunneled catheters, formation of fibrinous septa in the interior of the effusion can be observed in up to 14% of patients.[5] This is the result of procoagulant activity and the decline of fibrinolytic activity of MPEs, which contributes to the deposit of fibrin in the pleural space, creating septa that make it difficult to perform pleural effusion drainage in the patient's home. The benefit of urokinase instillation in these cases has been reported by several authors,[6,7] some of whom opt for high doses over prolonged periods.[8] Hsu et al., in 2006, recommended repeated instillations of 100 000 IU urokinase daily for at least 3 days (up to a maximum of 9 days and 900 000 IU urokinase)[9]; in contrast, other authors such as Mishra et al., in 2018,[10] used 3 doses of 100 000 IU urokinase instilled at 12-h intervals for a total dose of 300 000 IU, with monitoring 24 h after the last dose, but reported no significant benefit in the urokinase group.

We present a clinical case treated according to our hospital protocol for septated MPEs that are not effectively drained.

This was a 61-year-old man, referred to the respiratory medicine outpatient department for generalized constitutional symptoms, dyspnea on minimal effort and recurrent pleural effusion. He underwent 2 thoracenteses in the emergency department, for diagnosis and evacuation; a total of 2700 ml lymphocytic exudate was extracted, and cytology was negative for malignancy. In the respiratory medicine clinic, we performed a chest ultrasound which revealed pleural thickening. A computed tomography (CT) scan of the chest was requested, showing grade III/IV right pleural effusion causing right lower lobe atelectasis that contained a 2 cm nodular image and multiple foci of tumor-like pleural nodular thickening. The abdomen was significant for a pathological retroperitoneal lymphadenopathy measuring 2 cm in its greatest diameter. A right pleural ultrasound-guided biopsy was performed and thoracentesis for drainage was repeated (the third within a week, extracting 2000 ml). The pathology study reported renal cell carcinoma metastasis as a primary neoplasm.

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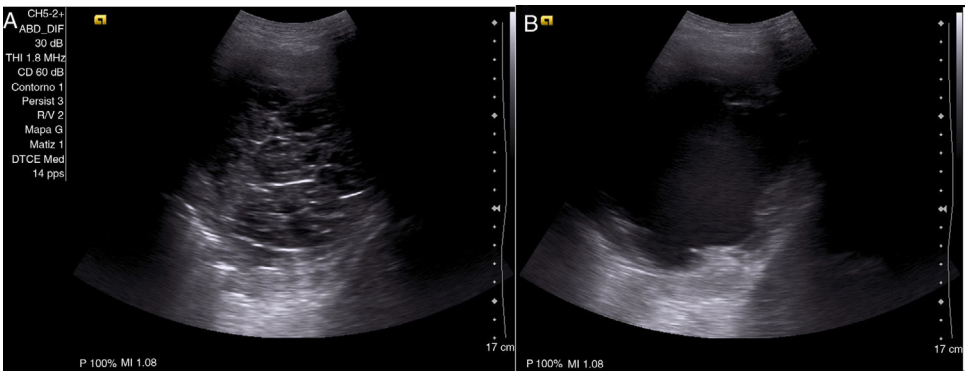

Fig. 1. Patient, 61 years of age, with malignant pleural effusion due to stage IV kidney cancer, presenting with non-draining tunneled catheter. Chest ultrasound showing abundant septa preventing drainage of pleural fluid (A). A single dose of 100 000 IU urokinase was instilled and left to act for 2 h; the thoracic ultrasound was then repeated, revealing pleural effusion containing detritus and lysis of the septa (B). The effusion was than drained, obtaining 750 ml of serosanguineous pleural fluid and subsequent symptomatic improvement.

A diagnosis of MPE due to stage IV kidney cancer was made, and in view of the persisting pleural effusion, we decided, after explaining the different therapeutic alternatives to the patient, to place a TPD catheter (IPC™ Rocket Medical©, Watford, United Kingdom), and both the patient and his family members were instructed how to perform drainage at home. About 30 days after TPD placement, the patient attended the clinic with dyspnea on minimal effort (visual analog scale [VAS]: 8/10, modified Medical Research Council dyspnea grade: III), with ineffective TPD draining. Chest X-ray revealed grade II/IV right pleural effusion, unchanged from previous studies, with the catheter placed correctly in the right hemithorax. A chest ultrasound showed grade II/IV effusion containing multiple septa and detritus (Fig. 1A). An intrapleural instillation of 100 000 IU urokinase was administered and left to act for 2 h, after which chest ultrasound was repeated, according to our protocol; this showed total lysis of the septa and persistent pleural effusion with detritus (Fig. 1B). This effusion was drained, obtaining 750 ml of serosanguineous fluid; no associated complications were reported, and the patient showed significant symptomatic relief.

In October 2017, we implemented our protocol for home-managed MPE that does not drain after connecting the TPD tube to the vacuum bottle. This protocol consists of an initial chest X-ray and pleural ultrasound and, if intrapleural septa are observed on the latter, a single dose of 100 000 IU urokinase is instilled and the patient is reevaluated at 2 h by repeating the ultrasound to visualize the effect of the urokinase (lysis of the septa), and then immediately performing drainage through the tunneled catheter.[11] A third pleural ultrasound is performed to confirm the reduction of the pleural effusion and the absence of immediate complications. The procedure takes less than 10 min from the time of the initial ultrasound to the intrapleural administration of the fibrinolytic agent, and another 10 min between subsequently visualizing septal lysis and draining the effusion. On discharge, the patient is given a contact telephone number to report any possible complications (effusion becoming hemorrhagic, principally, or onset of dyspnea or chest pain).

Fifteen patients have been included in this protocol to date, 53.8% men, with an average age (standard deviation, SD) of 68.5 (13.9) years, and an average (SD) of 584 (199) cc drained after the procedure. Clear symptomatic relief (reduction of >2 points on VAS) was obtained in 73.3% of cases, and no complications have been observed so far.

The dose of urokinase required in MPE is not clearly established, and in our experience a high success rate is achieved in septal lysis with a single dose. As previously mentioned, recent studies published in the literature support the instillation of fibrinolytic agents over several consecutive days with subsequent assessments; however, this approach requires several visits and increases costs, and the patient is obliged to spend more time in the hospital.[12] Since most patients with advanced disease are receiving palliative care, one of the main objectives should be to prioritize the well-being of the patient and reduce the number of visits to the hospital. With this systematic intervention, effective septal fibrinolysis is achieved in a single visit, without affecting the main objective of the procedure, which is to optimize pulmonary reexpansion, reduce pleural effusion, and improve the patient's dyspnea.

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Lung Cancer Invading a Coronary Artery Bypass Graft and Presenting as Refractory Atrial Flutter

Cáncer de pulmón que invade un bypass aortocoronario y produce un flúter auricular refractario

Dear Editor:

Arrhythmias and electrocardiographic abnormalities have been documented to be the first manifestation of cardiac infiltration by different tumors, including primary lung cancer.[1] We describe the case of a lung cancer in the left upper lobe invading a coronary artery bypass graft (CABG) and presenting as refractory atrial flutter (AF). Interestingly, AF was the only clinical symptom prior to the suspicion and diagnostic confirmation of malignancy. Although very rare, supraventricular arrhythmias may be the presenting symptom of lung cancer, and physicians should keep this important differential diagnosis in mind when patients have an unexplained persistent supraventricular arrhythmia, especially in cardiac patients with risk factors for lung cancer.

A 70-year-old ex-smoker man (45 pack-year history of smoking; quit smoking 4 years earlier) presented to our hospital with a new-onset symptomatic AF. His past medical history was significant for coronary artery disease (treated with CABG four years earlier), mitral valve disease (treated with mitral valve replacement at the same time of the CABG), hyperlipidemia, and chronic obstructive pulmonary disease. Since the AF episodes became persistent in spite of medical treatment, a cavotricuspid isthmus (CTI) ablation was planned. The CTI ablation procedure was successful for controlling the patient's AF. A chest radiograph performed at that time showed a subtle left parahilar opacity that was presumed to be of infectious origin (Fig. 1); however, given the absence of thoracic symptoms, a thoracic computed tomography (CT) was recommended. A chest CT was performed 2 weeks later and showed a left hilar mass invading the mediastinum and the left internal mammary artery (LIMA) graft to the left anterior descending coronary artery, consistent with a lung cancer (Fig. 1). The patient denied any chest pain, hemoptysis or coughing in the previous weeks, and an electrocardiogram (ECG) performed at that time (post-CTI) did not show any abnormalities. A bronchoscopic biopsy confirmed a large cell undifferentiated lung carcinoma. A contrast-enhanced whole-body positron emission tomography (PET)/CT detected small liver and adrenal metastases, consistent with a stage IV lung cancer. The patient was started on systemic chemotherapy, but unfortunately the cancer did not respond to treatment. A follow-up PET/CT performed 3 months later showed an increase in the size of the lung mass, a decrease in the diameter of the LIMA graft secondary to an encasement by the surrounding tumor (Fig. 2). Shortly after this follow-up study, the patient presented to the Emergency Department with tachycardia, chest pain, a new ST elevation on ECG in the precordial and lateral leads, and elevation of cardiac troponin levels, consistent with an extensive myocardial infarction. The patient was admitted to the oncology unit and died from cardiac arrest 24 h later.

Arrhythmias and electrocardiographic abnormalities, although rare, have been documented to be the first manifestation of cardiac infiltration by different tumors.[1] AF is well known to be associated with a variety of medical conditions, such as valvular heart disease, coronary artery disease, aging, hypertension, or thyroid dysfunction, among others.[2] However, AF associated with lung cancer has been rarely reported.[3] What makes our case unique compared with previous cases is that AF was the only clinical symptom prior to the suspicion and diagnostic confirmation of malignancy. We hypothesize that the possible pathogenesis of AF occurring in this patient could be the ischemic effect on the electric activity of the heart of the stenotic CABG by the encasing lung cancer as well as the enhanced micro-reentry activity when cancer cells infiltrate the left superior pulmonary vein and/or the left atrium.

Although very rare, supraventricular arrhythmias may be the presenting symptom of lung cancer. We believe that physicians should keep this important differential diagnosis in mind when patients have an unexplained persistent AF, especially in cardiac patients with risk factors for lung cancer.

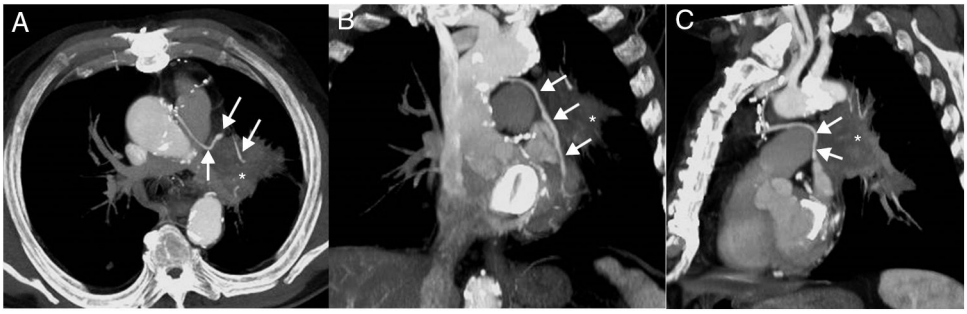

Fig. 1. Axial (A), coronal (B), and sagittal (C) maximum intensity projection (MIP) thoracic CT images (mediastinal window) show a left hilar mass (asterisk) encasing the left internal mammary artery graft to the left anterior descending coronary artery (arrows).