onset of asthma and diagnosis. The severe asthmatic reaction presented by the patient during the provocation test, requiring him to remain in the emergency department for 12 h, is also remarkable. Moreover, this reaction occurred after exposure to 0.1 mg/ml of zinc sulfate, a concentration not previously associated with severe asthmatic reactions in provocation tests with metals.[5,12–14] In the 2 cases of OA caused by zinc reported to date, the reaction occurred when doses of 1 and 10 mg/ml, respectively were used, and no severe reactions were observed.[3,4] This observation is in line with the evidence of Meca et al.[7] who suggested that when low molecular weight agents act by non-IgE-mediated mechanisms, the asthma can be more severe.

In conclusion, this case shows that zinc can cause OA by a non-IgE-mediated mechanism, and that this diagnosis must not be excluded when specific IgE and contact skin tests are negative. We also stress the possibility that asthma caused by this agent can be particularly severe, and recommend caution when performing the SBPT.

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## Spontaneous Regression of Pulmonary Emphysematous Bulla☆

### *Regresión espontánea de una bulla enfisematosa pulmonar*

*To the Editor,*

Pulmonary bullae are defined as air spaces greater than 1 cm in diameter with no epithelial wall. They can appear in normal pulmonary parenchyma, or more commonly, in the context of generalized emphysema. The natural course of pulmonary bullae is characterized by progressive growth, but it is not uncommon for them to present long periods of stability. In contrast, spontaneous regression is unusual.[1]

We report the case of a 55-year-old man, former smoker of 50 pack-years who gave up 16 years previously, who attended our hospital in June 2012 with cough, daily expectoration, and dyspnea MRC grade 1, with no significant exacerbations. Lung function tests showed a moderate obstructive pattern, with a forced vital capacity (FVC) of 4890 cc (106%), forced expiratory volume in 1 second (FEV1) of 2740 cc (74%), FEV1/FVC ratio of 56% and normal carbon monoxide diffusion (DLCO). Computed tomography (CT) revealed severe bilateral mixed centrilobular and paraseptal pulmonary emphysema, primarily involving the upper lobes, containing frank areas of pulmonary parenchymal destruction and a large paraseptal emphysematous bulla in the anterior segment of the right upper lobe (RUL), longest diameter 9 cm (Fig. 1). Given these findings and persisting symptoms, surgical bullectomy was proposed, which the patient refused.

In November 2015, a follow-up chest CT was performed, which revealed a nodular image with slightly spiculated margins in the paramediastinal region of the RUL, in close contact with the fat of the anterior line of pleural reflection, measuring 18 × 21 mm in the anteroposterior and transversal diameter on the axial plane, and 4 cm in length on the sagittal plane. Extensive areas of parenchymal pulmonary destruction could still be observed, associated with the prevailing emphysema pattern in the upper lobes. Of particular interest was the disappearance of the large bulla in the anterior segment of the RUL (Fig. 1). Given the suspicion of a malignant solitary pulmonary nodule, a positron emission tomography (PET) was requested that showed a moderately hypermetabolic pulmonary lesions in the RUL, consistent with malignancy, so the lesion was surgically removed. Video-assisted thoracoscopic wedge resection was performed, and pathology study found the lesion to be a residual sclerotic pulmonary nodule, consistent with thrombosed cavernous hemangioma, forming organized dystrophic calcification. In the subsequent lung function tests after resolution of the bulla, no improvement was found on spirometry, with FVC 4600 cc (100%), FEV1 2690 cc (74%) and a FEV1/FVC ratio of 58%.

As mentioned above, the natural course of bullae is progressive growth, to the extent that giant bullae can become so large that they even cause adjacent parenchyma to collapse.[2] Treatment of choice is surgery, which has been shown to improve dyspnea, gas exchange, lung function, and exercise capacity.[3] A few cases of spontaneous regression of an emphysematous bulla have been reported, the first of which was probably that published by Douglas

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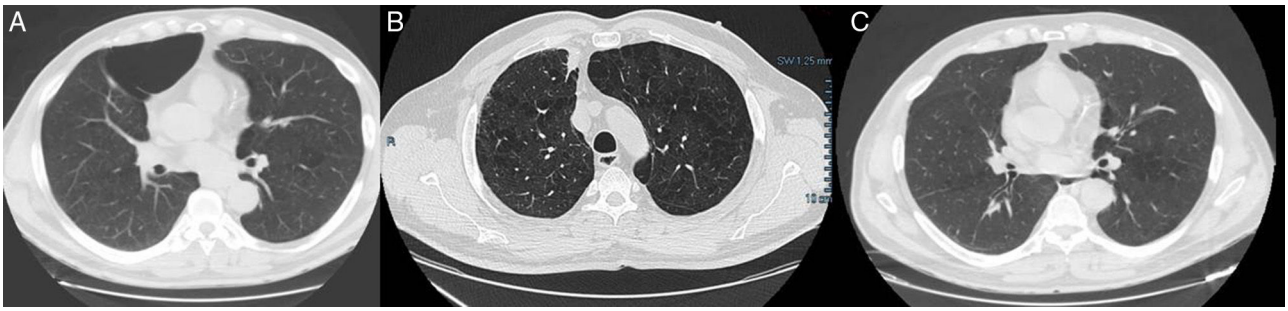


**Fig. 1.** CT images showing a large bulla in the RUL (A), spiculated pulmonary nodule in the RUL, along with resolution of the large bulla in this region (B), and the image after surgical resection of the pulmonary nodule with continued absence of the large emphysematous pulmonary bulla (C).

and Grant in 1957.[4] The process is usually preceded by clinical symptoms consistent with a respiratory infection, manifesting as cough and expectoration, generally with parenchymal consolidation surrounding the bulla and an air-fluid level within. Radiological resolution of air-fluid levels is usually very slow, generally taking more than 70 days, and the use of antibiotics does not speed up the process, so their systematic use is not recommended in asymptomatic patients.[5–7] The causative mechanism is unknown, although most authors suggest that it is due to bronchial obstruction by exudate and inflammation, with subsequent reabsorption of the air-fluid content,[8] or else it might be an inflammatory process within the bulla which causes it to seal.[2] Although in most cases an improvement in lung function has been reported,[9] this might not occur, as in the case described by Wahbi and Arnold in 1996,[10] and in our patient, possibly due to the size of the bulla.

In our case, the mechanism underlying resolution of the bulla is unclear. At no time was there evidence of inflammatory signs on the CT or previous chest radiographs, making this case unusual, since regression was totally asymptomatic, with no associated infection or tumor. The finding of cavernous hemangioma was incidental, and we do not believe that it is associated with resolution of the bulla.

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## Intraventricular Air Embolism Complicating Computed Tomography-Guided Pulmonary Aspiration Biopsy☆

### *Embolia aérea intraventricular como complicación de una punción biopsia pulmonar guiada por tomografía computarizada*

*To the Editor,*

Computed tomography (CT)-guided lung aspiration biopsy is a widely used tool in the histopathological diagnosis of lung lesions.[1] Although complications from this procedure are rare, they are not unknown, and can include pneumothorax, hemithorax, hemoptysis and/or pulmonary hematoma.

We report the case of a patient who developed pneumothorax and left ventricular air embolism after diagnostic aspiration of a pulmonary nodule.

Our patient was a 67-year-old man with a history of perforated colorectal carcinoma, requiring emergency surgery, followed by treatment with chemotherapy and radiation therapy. During the staging CT, a pulmonary nodule measuring 11 mm contiguous with the inferior pulmonary vein was observed. Fine needle aspiration biopsy was performed under general anesthesia, and during the procedure pneumothorax and left ventricular air embolism were visualized. This was a tomographic finding, and the patient was asymptomatic when the complication was discovered. A pleural drainage tube was placed, and pulmonary expansion was immediately observed (Fig. 1). Transthoracic echocardiogram was performed, ruling out coronary and/or ventricular complications. A waiting approach was taken, with echocardiographic studies and hemodynamic monitoring, which remained within normal values. A follow-up CT was performed after 48 h, showing reabsorption of the intracardiac air. The patient progressed without problems and was discharged on day 4 after the procedure.

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