

We use this case to describe the procedure and equipment used for whole-lung lavage in an infant, with practical suggestions for performing the technique, including the simultaneous use of 2 endotracheal tubes, one of which was lengthened to allow selective intubation. This system was selected because partial lavage with instillation of smaller aliquots of saline solution via the bronchoscope to the different lung segments is more laborious, although this could be an option in patients with severe respiratory failure who may not tolerate whole-lung lavage.<sup>3</sup>

The trigger in our patient was thought to be a transient dysfunction of alveolar macrophages (responsible, along with type II pneumocytes, for surfactant catabolism) due to immunosuppression, causing occupation of the alveolar space.<sup>4</sup> Another factor associated with this disease in the literature is infection, and in our case, the microorganisms in blood and in bronchoalveolar lavage may have played an important role in determining the course of the patient's respiratory disease.<sup>5</sup>

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### Management Difficulties in a Patient With EGFR-Mutation Positive Lung Adenocarcinoma and Cerebral Metastases<sup>☆</sup>



#### *Difícil manejo en paciente con adenocarcinoma de pulmón con mutación de EGFR y enfermedad cerebral*

To the Editor:

We report the case of a 49-year-old man, who was an occasional smoker. In 2007, a chest radiograph was obtained during an episode of acute bronchitis, which showed a solitary pulmonary nodule, subsequently confirmed on computed tomography (CT). Fiberoptic bronchoscopy did not yield any histological material, so a fine needle aspiration biopsy was performed, which revealed the presence of lung adenocarcinoma. After the case was discussed by the tumor committee, surgical intervention was performed. The pathology study reported adenocarcinoma requiring adjuvant chemotherapy. One year later, contralateral pulmonary and hepatic relapse was revealed on CT. Given the early tumor relapse, the likelihood of the tumor being resistant to chemotherapy, and the clinical characteristics of the patient, the tumor was biopsied again and the sample was sent for molecular analysis, including determination of epidermal growth factor receptor (EGFR) status. This test was positive, reporting an exon 19 deletion, so treatment began with erlotinib. Six months later, the CT showed complete response, subsequently confirmed on PET/CT. The patient continued on the same treatment, with regular assessments, for another 5 years. At that time, as the various radiological tests performed during the previous 5

years showed no evidence of disease, we decided to discontinue treatment under close monitoring. Two months later, the patient presented with headache, vomiting and instability. A head CT was performed, revealing a cerebellar lesion measuring 3×4 cm, with intense perilesional edema. After the possibility of systemic relapse was ruled out by CT and PET/CT, oligometastasis was confirmed and it was decided that the best treatment was local resection of the lesion, in accordance with the various clinical guidelines that advocate local treatment as the best approach in oligometastatic disease, if feasible. Histological analysis confirmed that the lesion was metastasis from the previous lung adenocarcinoma. Development of a T790M resistance mutation was suspected, so the EGFR analysis was repeated. This ruled out secondary resistance and confirmed the presence of the exon 19 deletion. Despite the absence of systemic disease, the high risk of relapse led us to reinstitute anti-EGFR treatment, and the patient currently remains free of both systemic and cerebral disease.

Our case illustrates the difficulties encountered in managing patients with EGFR mutations and predicting their clinical progress. EGFR belongs to the ErbB family of membrane receptors with tyrosine kinase activity. Between 5% and 10% of non-small cell lung cancers have EGFR mutations; they occur more commonly in women and are associated with little or no consumption of tobacco.<sup>1</sup> The finding of a mutation of this type predicts a better response to targeted drug treatment than to cytotoxic chemotherapy. However, not all mutations are the same: exon 19 deletion is the most common and predicts a better treatment response, followed by exon 21 insertion (L858R); finally, exon 20 alterations are associated with drug resistance.<sup>2,3</sup> It should be emphasized here that the typical evolution of this tumor is marked by an initially good response to the inhibitor, followed by the development of secondary resistance, the most common mechanism being the appearance of the T790M mutation.<sup>4</sup>

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In our patient, the excellent response and disappearance of systemic disease led us to question the need to continue with treatment, since no clear directives are currently available. During his off-treatment period, the patient presented cerebral progression. This led us to consider 2 hypotheses: either a T790M resistance mutation had occurred, or the tumor was extremely dependent on the EGFR pathway, and when the drug was withdrawn, breakthrough disease developed with progression in a sanctuary site. After molecular analysis, the second option appears more plausible, and raises once again the issue of the best management of long-term survivors receiving anti-EGFR treatment.

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## Pulmonary Arterial Hypertension Secondary to Partial Anomalous Pulmonary Venous Return in an Elderly Patient<sup>☆</sup>



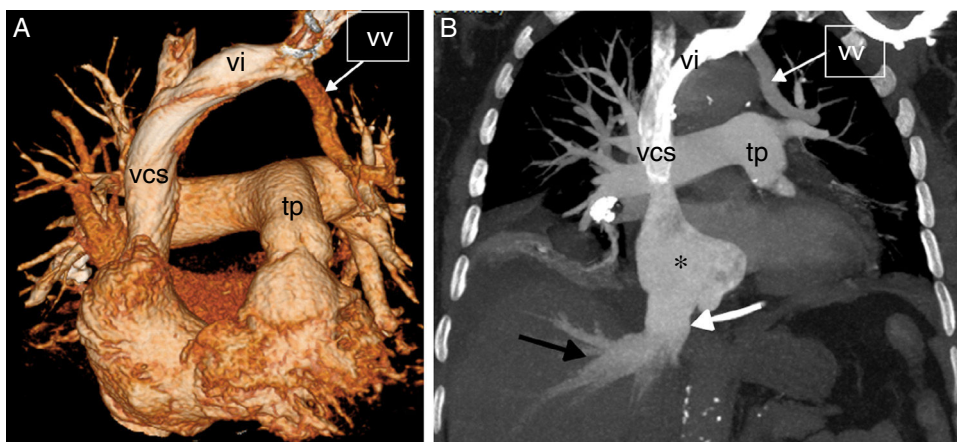
### Hipertensión arterial pulmonar secundaria a un drenaje venoso pulmonar anómalo parcial en una paciente anciana

To the Editor:

Partial anomalous pulmonary venous return (PAPVR) is an uncommon congenital abnormality that can be diagnosed in adult life, although it is more often detected during childhood.<sup>1</sup> It consists of abnormal, incomplete pulmonary venous return to the systemic venous circulation (superior vena cava, azygos vein, coronary sinus, brachiocephalic vein, inferior vena cava, etc.) causing

left-to-right shunt. PAPVR is more common in the right side, and is often associated with other congenital abnormalities, such as heart defects (particularly atrial septal defect), or an abnormally developed airway.<sup>2</sup> We report a case of a patient in her seventies with left PAPVR that began with signs and symptoms of pulmonary arterial hypertension (PAH).

The patient was a 76-year-old woman with no significant medical history who consulted due to progressive dyspnea, edema in the lower limbs, and discomfort in the chest and abdomen. Chest radiography showed cardiomegaly and scant bilateral pleural effusion. Signs of right heart overload were observed on electrocardiogram, and D-dimer was slightly elevated. A chest CT angiogram ruled out pulmonary thromboembolism, but unexpectedly revealed a PAPVR in which the veins of the left upper lobe drained to the brachiocephalic vein via a vertical vein (Fig. 1A). Radiological signs of severe



**Fig. 1.** (A) Computed tomography with 3D volume rendering showing a vertical vein (vv), formed by the confluence of pulmonary veins of the left upper lobe, running toward the brachiocephalic vein (vi); tp: pulmonary artery trunk; vcs: superior vena cava. (B) Coronal computed tomography with maximum intensity projection (MIP) showing signs of arterial/precapillary pulmonary hypertension and tricuspid valve insufficiency: dilation of right atrium (asterisk), inferior vena cava (white arrow) and suprahepatic veins (black arrow) congestion. Note the vertical vein (vv), the brachiocephalic vein (vi), and the superior vena cava (vcs).

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