

Cerebral air embolism as an immediate complication of percutaneous lung biopsy

Embolia aérea cerebral como complicación inmediata de biopsia percutánea de pulmón

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Summary

Air embolism occurs when air or gas enters the bloodstream, either venous or arterial. Although rare, this condition is potentially fatal, and its incidence is increasing due to the rise in interventional procedures. In the past, the primary causes of air embolism were trauma and major surgeries. Currently, minimally invasive procedures, such as digestive endoscopies, angiographic studies, percutaneous biopsies, thoracocentesis, and the insertion of central venous or hemodialysis catheters, are the leading causes. The aim of this study is to present a case of cerebral air embolism that occurred immediately after a CT-guided percutaneous lung biopsy, resulting in severe neurological damage and a fatal outcome for the patient.

Resumen

La embolia aérea se produce cuando entra aire o gas en la circulación sanguínea, venosa o arterial. Esta complicación, aunque infrecuente, es potencialmente fatal y su incidencia está aumentando debido al incremento en el uso de procedimientos intervencionistas. En el pasado, las principales causas de embolia aérea eran el trauma y las cirugías mayores. Actualmente, los procedimientos mínimamente invasivos, como endoscopias digestivas, estudios de angiografía, biopsias percutáneas, toracocentesis, y la inserción de catéteres venosos centrales o para hemodiálisis, son las principales causas. En este artículo se describe un caso de embolia aérea cerebral que se presentó inmediatamente después de una biopsia pulmonar percutánea guiada por tomografía computarizada (TAC), que resultó en un grave daño neurológico y desenlace fatal para el paciente.

Case presentation

An 81-year-old male patient with a history of pulmonary tuberculosis treated at age 32 developed right-sided fibrotorax as a complication. He was admitted to the emergency room due to exacerbation of respiratory symptoms, for which a chest computed tomography (CT) scan was performed, revealing fibrotic changes predominantly in the right lung, with a cavitated lesion in the right lower lobe. Hospitalization was indicated due to suspicion of reactivation of pulmonary tuberculosis. Serial sputum smears were performed, with negative results. In addition, the patient underwent a fiberoptic bronchoscopy with bronchoalveolar lavage, whose microbiological and molecular tests were negative for tuberculosis or other microorganisms.

Subsequently, it was decided to perform a percutaneous biopsy of the lesion in the right lower lobe. During the procedure, after performing a CT-guided lung biopsy with a posterior approach using a 17G coaxial system and an 18G semi-automatic needle (Figure 1), the patient suffered left central facial paralysis, atonia in all four limbs, altered consciousness with a Glasgow Coma Scale score of 3/15, bladder sphincter relaxation, and a score of 32 on the National Institutes of Health Stroke Scale (NIHSS).

Given this picture, an emergency simple cranial CT scan was performed, which revealed bubbles distributed in the cerebral sulci, a finding characteristic of cerebral air embolism (Figure 2). Magnetic resonance imaging (MRI) performed 60 minutes after the onset of symptoms identified areas of diffusion restriction due to ischemia in the right frontal, parietal, and occipital regions, associated with areas of magnetic susceptibility in the topography of the cortical veins (Figure 3).

Treatment with high-flow oxygen was initiated, and, given the suspicion of a possible associated thromboembolic event, it was decided to perform

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systemic thrombolysis with alteplase. The patient was transferred to the intensive care unit (ICU), where hyperbaric oxygen therapy was not available.

Twenty-four hours later, a cranial CT scan revealed established infarcts in the territories of the middle and posterior cerebral arteries on the right side, with reabsorption of the air bubbles. Due to the severe alteration in consciousness, a 6-hour video electroencephalographic monitoring was ordered, which showed severe generalized dysfunction with grade IVB encephalopathy (comatose state) without epileptic activity. Given the torpid neurological evolution and the absence of improvement, palliative sedation was initiated. The patient died 48 hours after the start of palliative measures.

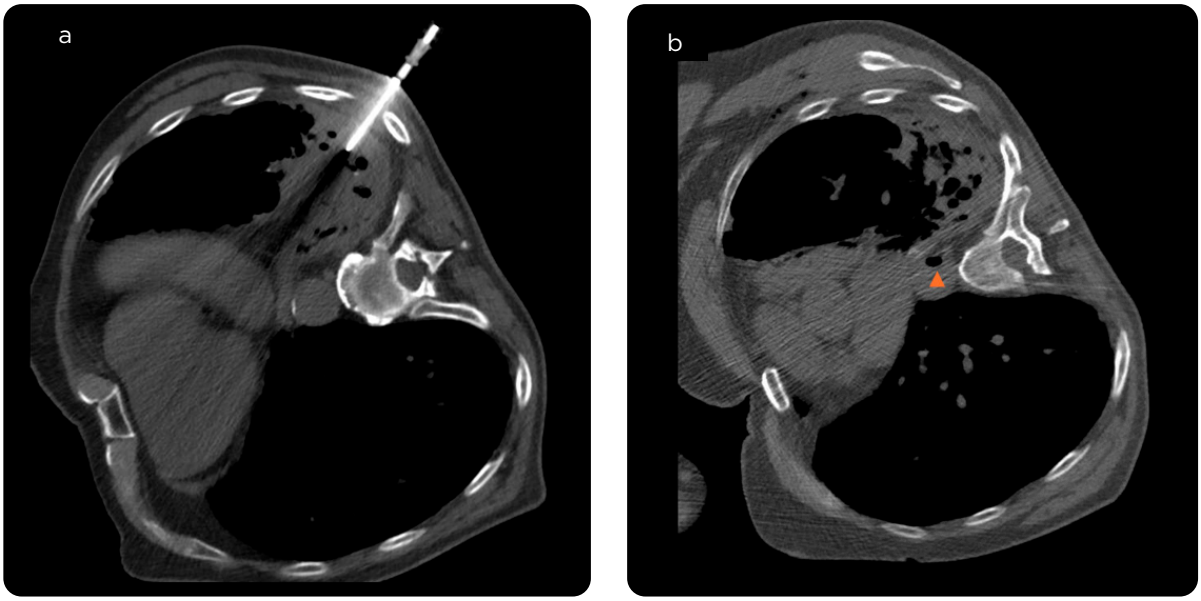


Figure 1. Chest CT scan during the procedure. a) During the biopsy and b) after the biopsy. Shows air in the thoracic aorta (orange arrow).

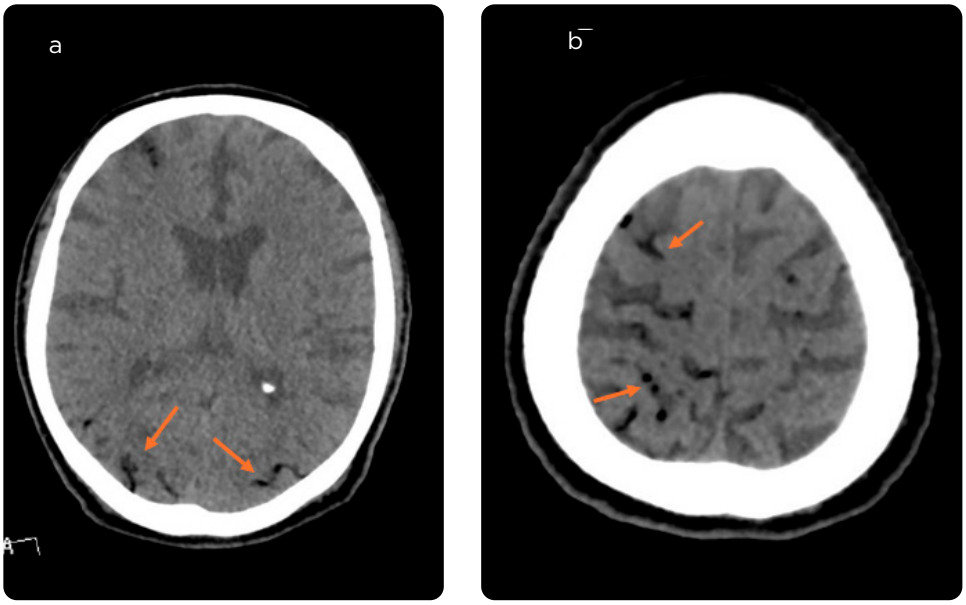


Figure 2. Plain CT scan of the skull performed 8 minutes after the biopsy. a) Slices at the level of the basal ganglia and b) in the convexity: areas of low density in the sulci, projected into the subarachnoid space, predominantly on the right side, indicative of the presence of air (orange arrows).

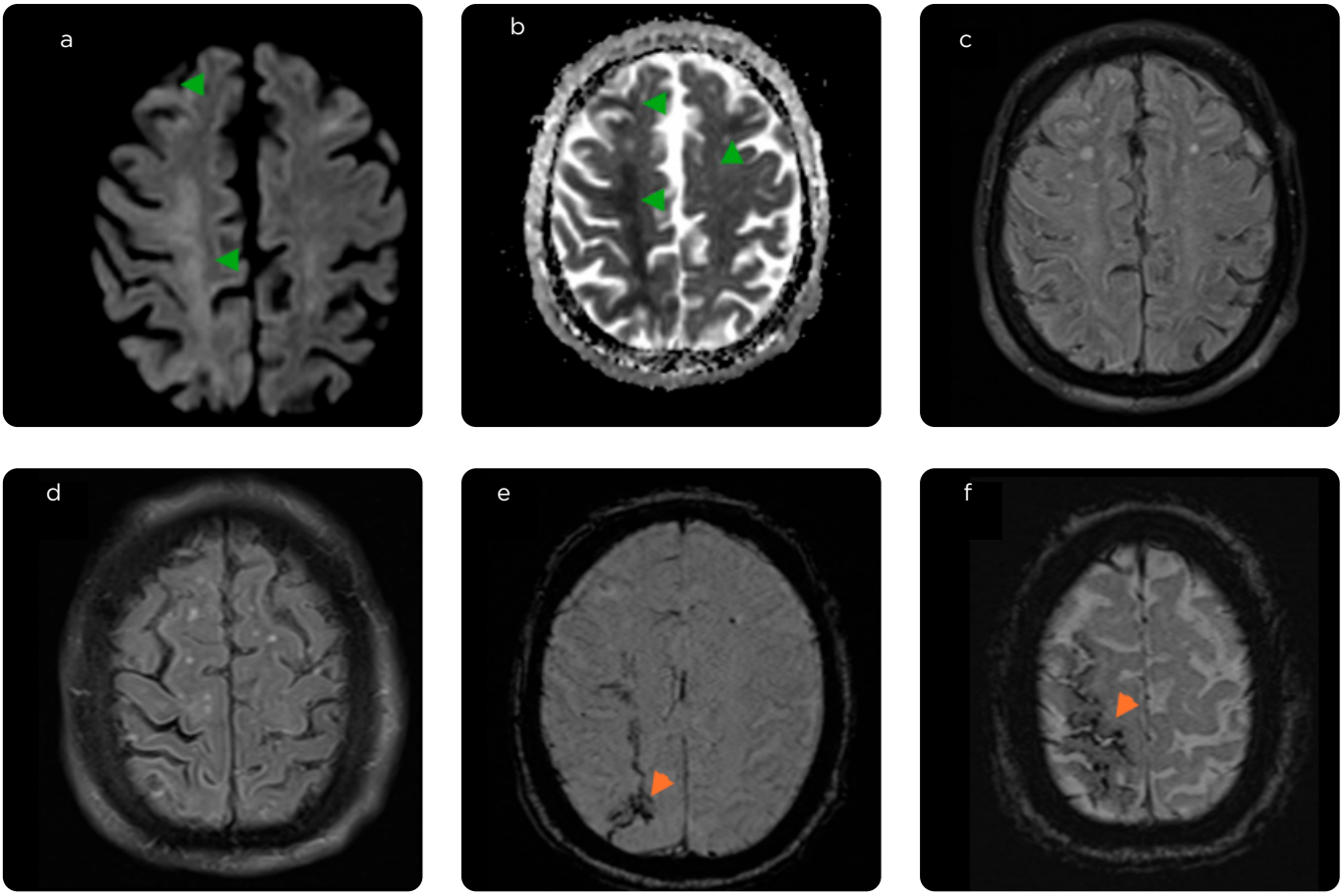


Figure 3. Cranial MRI 38 minutes after the biopsy. a) b1000 sequence and b) corresponding ADC map. Areas of water diffusion restriction in the bilateral frontal and right parietal cortical and subcortical locations (green arrows). c and d) FLAIR (Fluid-Attenuated Inversion Recovery) sequences with T2 information. High-signal punctiform images in the semioval centers and convexity, without representation in DWI (Diffusion-Weighted Imaging), suggesting a hyperacute infarction. e and f) The SWI (Susceptibility-Weighted Imaging) sequence shows low signal intensity in the right frontoparietal subarachnoid space, consistent with known pneumoencephaly (orange arrow), and air in the intravascular space.

Discussion

The true incidence of air embolism is unknown due to a lack of records and robust prospective studies. Reports and case series suggest that this condition may go unnoticed, as it often presents with nonspecific or transient symptoms. A scientific study identified that the anatomical locations most commonly affected by air embolism were the pulmonary artery and right heart chambers (54%), the brain (31.3%), the coronary arteries (12%), and the extremities (1.5%). However, these percentages correspond to the cohort analyzed in that study and cannot be extrapolated to the pathology in general (1). Its diagnosis represents a clinical challenge, as it is an uncommon complication and is not actively sought. In addition, nonspecific and transient manifestations may go unnoticed. Therefore, a high index of suspicion is required to establish a diagnostic pathway and early therapeutic approach.

A prospective cohort study found that this complication occurred in 2.65 per 100,000 hospitalizations, with a mortality rate of 21% per year; central venous catheter insertion is the most common cause (24.3%), followed by thoracic procedures (21%) (2). In the specific case of percutaneous lung biopsy, air embolism has a radiological incidence of 3.8% and a clinical incidence of 0.49% (3).

In a cohort study of 119 patients with iatrogenic air embolism, 45% presented with motor neurological deficits, 30% with seizures, and 39% with Babinski signs. Cerebral air embolism was associated with a high risk of mortality at one year in those with neurological symptoms (2). Symptoms usually appear rapidly, with neurological focalization in 67.7% of cases and coma in 59.5% of them (4). In the case presented, symptoms began almost immediately after the procedure, with a predominance of focal neurological signs and a severe alteration of consciousness. No epileptic activity was documented in the electroencephalographic studies performed during follow-up, but findings suggestive of a comatose state were noted.

A meta-analysis identified several factors associated with the development of air embolism, including lesions in the lower lobes, subsolid lesions, pneumothorax, hemorrhage, and the location of the lesion above the left atrium (5). The only risk factor identified in the patient described was a cavitated lesion in the right lower lobe.

Early diagnosis is crucial for proper management. CT is useful for identifying air bubbles in vascular structures. MRI is more sensitive for detecting changes due to air embolism, showing areas of multifocal restriction in the diffusion sequence and susceptibility changes in

gradient echo sequences (6,7). Findings such as high cortical signal intensity in T1-weighted sequences and loss of differentiation between white and gray matter may indicate early cortical necrosis. In this case, imaging findings allowed for rapid confirmation of the diagnosis, as the distribution pattern of air bubbles in a gyral pattern toward the convexity and along the sulci is characteristic of this entity (8). Additionally, in the retrospective review of post-biopsy images, an intravascular air bubble was documented in the aorta.

Initial management includes continuous monitoring of vital signs, administration of 100% supplemental oxygen, and positioning in the lateral decubitus position. Hyperbaric oxygen therapy is recommended, as it helps reduce the volume of intravascular bubbles and improves oxygenation of ischemic tissues. However, the availability of this therapy is limited (7). Early palliative management has been used in patients with a high comorbidity burden or severe condition (7). In the case of the patient described, supplemental oxygen was administered at an inspired fraction of 100%, and pharmacological thrombolysis with alteplase was performed, according to medical criteria. However, it was not possible to access hyperbaric oxygen therapy. Due to severe neurological dysfunction and deep coma, a palliative management approach was chosen.

Conclusions

In conclusion, air embolism is a rare but serious complication, usually associated with interventional procedures. Its diagnosis requires a high index of suspicion, and early management is crucial to improve clinical outcomes. Although hyperbaric oxygen therapy is recommended, its availability is not always guaranteed, highlighting the importance of timely supportive management and adequate palliative care in the most severe cases.

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