

Cerebral air embolism after computed tomography-guided percutaneous transthoracic lung biopsy: a case description

Jie Pan^{1#}[^], Danyang Meng^{1#}[^], Liang Chen², Juan Qu¹, Jin Hu¹, Liju Dai¹

¹Department of Neurology, Affiliated Hospital of Jiaxing University, Jiaxing, China; ²Department of Head and Neck Surgery, Affiliated Hospital of Jiaxing University, Jiaxing, China

"These authors contributed equally to this work.

Correspondence to: Liju Dai, MD. Department of Neurology, Affiliated Hospital of Jiaxing University, 1882 Chengnan Road, Jiaxing 314000, China. Email: ourdlj@163.com.

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Introduction

The identification of certain pulmonary lesions via imaging examination is challenging, and peripheral lesions are rarely detected by fiber bronchoscopy. Consequently, computed tomography (CT)-guided percutaneous transthoracic lung biopsy (PTLB) has emerged as a prevalent, rapid, and effective method for the diagnosis of both benign and malignant lung diseases (1,2). However, a rare but serious consequence of CTguided PTLB is arterial air embolism, specifically, a blockage of blood arteries arising from air entering the bloodstream. This study presents a case of acute cerebral embolism caused by PTLB where the patient displayed a significant cerebral embolism in the right hemisphere. The patient subsequently returned to his pre-disease state 3 months after being discharged. This case has the potential to assist radiologists and clinicians in early identification and management of systemic air embolism (SAE).

Case presentation

All procedures performed in this study were in accordance with the ethical standards of the Institutional Review Board of the Affiliated Hospital of Jiaxing University, Jiaxing (No. 2023-LY-127) and with the Helsinki Declaration (as revised in 2013). Written informed consent was provided by the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

The patient was a 47-year-old male office worker who was clinically diagnosed with pulmonary nodules upon admission (Figure 1A). On 27 December 2020, a CT-guided percutaneous lung puncture was performed for the biopsy of pulmonary nodules, with the patient positioned in the left lateral decubitus position on the CT bed and the right chest chosen for needle insertion. Following disinfection and local anesthesia, the nodules in the upper and middle right lobes were biopsied under CT guidance, with a spring coil placed on the edge of the nodule (Figure 1B). The operation was successful, and the post-biopsy CT scan revealed only a small right pneumothorax. However, the patient complained of dizziness and sweating, accompanied by a blood pressure of 102/67 mmHg and a pulse oximetry reading of 96%, indicating the possibility of a pleural reaction. Therefore, 5 mg of dexamethasone was intravenously administered to relieve symptoms. Unfortunately, the patient then experienced left limb paralysis. A subsequent cranial CT scan reflected a locally reduced density in the right cerebral hemisphere (Figure 2A). Physical examination revealed clear consciousness and intact pain sensation, but bilateral gaze towards the right, left peripheral facial paralysis, slurred

^ ORCID: Jie Pan, 0000-0002-8703-4339; Danyang Meng, 0000-0001-5730-0598; Liju Dai, 0000-0002-7790-2286.



Figure 1 CT images of lung before and during puncture. (A) CT scan of the chest before puncture, with nodules in the middle and upper lobes of the right lung indicated by red arrows. (B) CT scan of the chest during puncture, with the needle observed to be white. CT, computed tomography.

speech, tongue extension towards the right, grade 0 left limb muscle strength, National Institutes of Health Stroke Scale (NIHSS) score of 13 points, and modified Rankin scale (mRS) score of 4 points, indicating the possibility of acute ischemic stroke and air embolism. Magnetic resonance imaging (MRI) confirmed the diagnosis of parieto-occipital cerebral infarction (Figure 2B). The patient then received hyperbaric oxygen therapy (HBOT), along with daily administration of enteric-coated aspirin tablets (100 mg) and rosuvastatin (10 mg). Despite these measures, the patient developed secondary epilepsy on 28 December 2020, and thus was prescribed 0.5 g of sustained-release sodium valproate and 0.5 g of levetiracetam twice daily. A chest CT conducted on 30 December 2020 exhibited right pneumothorax, right chest wall and anterior mediastinal pneumatosis (Figure 2C), necessitating closed thoracic drainage. On 14 January 2021, the patient underwent thoracoscopic wedge resection whilst in stable condition. He was discharged on 22 January 2021 in good condition, presenting with clear consciousness, good spirit, blurred left eye vision, positive strength paresis test of the left upper limb, grade V muscle strength of the remaining limb, NIHSS score of 1 point, and normal cardiopulmonary auscultation. The pathology of the pulmonary nodules after the thoracoscopic wedge resection revealed atypical adenomatous hyperplasia of the alveolar epithelium without residual tumor at the incisal edge in the specimen of the right upper lobe and atypical adenomatous hyperplasia with electrocautery changes in the alveolar epithelium, accompanied by fibrous hyperplasia in some areas and no tumor residual on the incisal edge in the specimen of the right middle lobe. By telephone follow-up, the patient was reported to have completely recovered to the pre-disease state three months after being discharged.

Discussion

CT-guided PTLB is a commonly used technique to obtain tissue samples for suspected lung disease that is not amenable to bronchoscopy. Although PTLB is associated with common complications such as pneumothorax and pulmonary hemorrhage, SAE is an infrequent complication that may not be easily detected but poses significant risks to the patient and may only be visualized through imaging in clinical practice (1).

A meta-analysis conducted in 2021 revealed that the overall incidence of SAE after biopsy of thoracic and pulmonary lesions was 0.04-0.08% [95% confidence interval (CI): 0.048–0.128%, I²=45%] (2). However, a retrospective observational study conducted by Freund et al. (3) involving 610 PTLB patients reported a higher incidence rate of 3.8%, although only 0.49% of cases exhibited symptoms, and 0.16% of cases resulted in fatalities. The early definition of SAE was limited to clinically significant events such as cardiovascular failure or neurological manifestations. However, intravascular air may not always be captured through imaging (for example, limited CT range during the perioperative period), or recognized in clinical practice (for example, postoperative imaging shows obvious intravascular air but no clinical symptoms, making it difficult to diagnose in a timely manner), leading to potential underestimation of its occurrence (4). The pathophysiological mechanism of air embolism lies in obstructions to blood flow, leading to hypoxic reactions in the end organs, especially the coronary and cerebral arteries. In contrast to venous air embolism, as little as 0.5-2 mL of air may result in fatal obstruction of the coronary and cerebral arteries in animal studies, respectively, whereas 50 mL of air can produce fatal obstruction of the pulmonary artery. The severity



Figure 2 Brain images of the patient after air embolism and CT images after puncture. (A) Cranial CT on 27 December 2020, showing localized low-density shadows, large areas of poorly defined low-density patchy shadows, and small areas of round shadows surrounded by gas in the right temporo-occipital region. (B) Cranial MRI displayed slightly long-intensity signal in T1 and T2 FLAIR in the right parieto-occipital lobe, and high-intensity signal in diffusion weighted imaging. (C) CT scan of the chest after puncture, presenting right pneumothorax, right chest wall, and anterior mediastinal air accumulation. CT, computed tomography; MRI, magnetic resonance imaging; T1/T2 FLAIR, T1/T2-weighted fluid-attenuated inversion recovery; DWI, diffusion-weighted imaging.

of symptoms and outcomes align with pathophysiology, ranging from mild motor weakness to coma (4).

Understanding the mechanisms of air embolism and identifying the associated risk factors will help to develop and implement effective preventive measures before and during biopsy. There are 3 possible mechanisms that can introduce air into the pulmonary venous system during percutaneous transthoracic needle biopsy (PTNB). The first involves the biopsy needle passing through the pulmonary vein, creating a direct connection with the atmosphere. In this patient, a small amount of gas in the chest CT after the puncture was completed, and the possibility of direct communication between external air and pulmonary veins was low. The second is related with the situation where the needle is inserted into the pulmonary artery, and the air in the pulmonary artery circulation reaches the pulmonary vein circulation through pulmonary microvessels. However, pulmonary puncture rarely leads to gas entering the pulmonary artery (5). The third is associated with the formation of iatrogenic broncho venous fistula when the needle passes through the pulmonary vein and adjacent airways, allowing intrapulmonary air to enter the pulmonary veins through the puncture channel, which is the most possible mechanism of air embolism in this patient. Severe air embolism can still occur in the absence of detectable lung pathology by preoperative chest CT. In the case of

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pulmonary cavities, infections, fibrosis, or pleural adhesions, where the puncture needle pierces adjacent pulmonary veins and bronchi simultaneously, an abnormal anastomosis and a continuous bronchopulmonary vein fistula can be created when the guide wire is loosened and the needle is pulled outward, and the barb in front of the guide wire is hooked on the bronchus and pulmonary vein. In a resting state, the pulmonary vein pressure is higher than the adjacent airway. When patients are nervous or breathe heavily, pulmonary vein pressure may decrease, allowing air to enter the pulmonary vein from the bronchus and subsequently the systemic circulation along the pressure gradient, ultimately reaching the cerebral artery system. As in this case, due to the patient's left decubitus position, gas is more likely to enter the brachiocephalic artery and the right internal carotid artery, resulting in a right cerebral infarction, which also indicates that body posture is related to SAE. Vasospasm caused by the air passing through the blood vessel and platelet thrombi produced by the activation of platelets at the blood-gas interface may play a significant role in vascular blockage, which can be determined by the circulation bubble. Tension pneumothorax possibly has a beneficial impact on the disease course, as distal airway collapse can reduce the volume of broncho venous fistula. In addition, the compression of the pulmonary veins may make blood flow back to the unaffected side, thereby leading to a decrease in cardiac venous reflux (6).

A large multicenter case-control study by Ishii et al. (7) involving 13 centers and 2,216 PTLB surgeries retrospectively reviewed the images obtained during and immediately after surgery and found that several factors significantly increased the risk of SAE, including parenchymal bleeding, lower lobe lesion biopsy, and larger biopsy needle size. Studies have shown that the incidence of air embolism is related to needle size and associated coaxial method because larger needles can increase the risk of pulmonary embolism (8); however, this is controversial (9). To date, there has been only 1 case of symptomatic air embolism caused by a lung puncture in our hospital. We cannot perform statistical analysis to distinguish the probability of air embolism caused by coaxial and noncoaxial methods. It is worth noting that previous studies on the lung structure have confirmed the existence of a straight path from the skin to the lung mass, suggesting that the operator should follow international surgical guidelines and select the shortest needle entry path to the lesion site in order to reduce lung parenchymal damage. The evaluation of surgical indications, full consideration of surgical design,

adequate preoperative communication and accurate and indepth disclosure can further determine the occurrence of air embolism as an intervention complication to avoid medical litigation issues (10).

Ishii et al. found that the lesion in the lower lobe of the lung was a risk factor for air embolism because the vessels were much larger in the lower lobes than in the other lobes (7). Therefore, there is a substantial risk of vein injury and air embolism after surgery in the lower lobes. However, a previous report suggested that air embolism occurred primarily in the prone position in patients with lesions in the lower lobes and that the appearance of air embolism was not related to the location of the lobar (11). The 2 lesions in the patient were located in the middle and upper lobes of the lung, respectively. Therefore, the relationship between lobar location and air embolism needs to be further verified. Other risk factors, such as puncture location above the left atrium, positive pressure ventilation, intraoperative cough, needle depth/length passing through the ventilated lung, number of samples, right lateral and prone positions, needle tip outside the lesion, and pulmonary hemorrhage, are supported by evidence from case-control studies reviewed by Roberts et al. (1). The prone position has been observed to be associated with the occurrence of air embolism and therefore should be deemed a risk factor, particularly with regards to lower lung lesions. In the context of PTNB, some experts have proposed that an ipsilateral dependent position for the patients may be beneficial (3,12), and although some studies have suggested that the supine position is superior to the Trendelenburg position because of the risk of brain edema and seizures (13), more experts recommend a right lying or Trendelenburg position (14,15).

Interestingly, we found a case report of successful treatment of middle cerebral artery air embolism with intravascular aspiration (16). Acute reperfusion therapy with thrombolytic agents or mechanical thrombectomy has not been validated for the treatment of air embolism. In the case of cerebral air embolism, HBOT will replace our traditional acute reperfusion strategy (17). For patients without underlying respiratory disease, early use of HBOT is beneficial, and this treatment can be used continuously until symptoms do not improve further (17,18). If clinical symptoms and/or imaging indicate air embolism, the initial management includes stopping the surgery, initiating cardiopulmonary resuscitation if necessary, administering 100% oxygen, notifying the emergency care team, and commencing transfer to the intensive care unit. HBOT has shown promise in reversing neurological deficits, and application of this therapy should be dependent on clinical symptoms and intravascular gas volume in clinical cases (19). In addition, in situations where hemodynamics is substantially impaired, supplementing intravascular volume and administering intravenous catecholamines ought to be performed concurrently. To prevent the occurrence of air embolism, patients with cerebral artery embolism should also be placed in the Trendelenburg position. Although intravenous injection of lidocaine, corticosteroids, and heparin have been recommended for the management of arterial air embolism, their efficacy presently remains unproven (20).

In conclusion, arterial cerebral air embolism is an uncommon but potentially catastrophic event. This case demonstrates the rare pathogenesis of ischemic stroke after CT-PTLB. PTLB has become a widespread practice in clinical settings, but we must always remain vigilant. Rare and unpredictable events, such as SAE following lung biopsy, can result in serious negative consequences for patient safety. Medical professionals, including clinicians and radiologists, must be aware of this potentially lifethreatening complication and fully prepared to provide prompt and effective emergency treatment as and when required.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims. amegroups.com/article/view/10.21037/qims-23-216/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the Institutional Review Board of the Affiliated Hospital of Jiaxing University, Jiaxing (No. 2023-LY-127) and with the Helsinki Declaration (as revised in 2013).

Written informed consent was provided by the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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