KANSAS JOURNAL of MEDICINE

# Air Embolism as a Complication of Lung Biopsy and IV Contrast Administration

Wissam Karam, M.D.[1], Atiyeh Samadi, M.S.[1], Eric Acosta, M.D.[1], Zubair Hassan, M.D.[1,2]

[1]University of Kansas School of Medicine-Wichita, Wichita, KS Department of Internal Medicine

[2]Robert J Dole Department of Veterans Affairs Medical Center, Wichita, KS

*Received Nov. 13, 2022; Accepted for publication Feb. 13, 2023; Published online March 15, 2023*
*https://doi.org/10.17161/kjm.vol16.18682*

## INTRODUCTION

Percutaneous computer tomography (CT)-guided needle biopsy (PCNB) of suspicious pulmonary nodules is a well-established method of obtaining tissue for histopathological examination.[1] Despite its wide use in the United States, this procedure carries with it a set of complications associated with significant morbidity and mortality.[2] Systemic air embolism (SAE) is a rare yet devastating complication of PCNB with longstanding cerebral and cardiovascular effects.

In this report, three cases are discussed. The first case involves an air embolus following CT-guided lung biopsy and leading to acute stroke. The second case was similar, with a CT-guided lung biopsy leading to acute-onset neurologic symptoms. The third case, while it did not involve a percutaneous lung biopsy, highlighted the risk of air embolism with procedures as routine as IV contrast administration for the purposes of obtaining a CT scan.

## CASE REPORT

The first case was a 71-year-old male patient with a medical history significant for hypertension, as well as heavy tobacco and alcohol use, presenting for CT-guided biopsy of a right lower lobe pleural-based mass. During the biopsy, the patient developed a mild cough and hemoptysis, but the procedure was overall well-tolerated. Post-procedural imaging showed no pneumothorax, but demonstrated pulmonary hemorrhage at the site of the biopsy.

A post-biopsy CT scan showed a small amount of air within the non-dependent portion of the mid descending thoracic aorta (Figure 1). The patient was immediately placed in the head down/right-side down position, started on 100% high-flow oxygen, and closely monitored. The CT scan was repeated after one hour showing resolution of the air embolus. Orders were placed for an overnight admission for observation; however, the patient declined and was discharged against medical advice. He was advised to come back if he developed chest pain or shortness of breath.

The following day, the patient presented to the emergency department (ED) for difficulty urinating, lower abdominal pain, and difficulty to ambulate due to lower extremity weakness. On physical exam, the patient had 5/5 motor strength in both upper and lower extremities, as well as intact sensation and 2+ reflexes, however, he exhibited unsteady gait. CT scan of the head showed no evidence of ischemia or hemorrhage. Brain magnetic resonance imaging (MRI) showed multiple focal areas of acute ischemia, predominantly in the posterior left cerebellum and left parietal lobe, as well as punctate foci in the left medullary pyramid, left midbrain, and right parietal lobe (Figure 2). The patient was considered to have an acute cerebrovascular accident secondary to air emboli and was transferred to a nearby stroke center for further management.

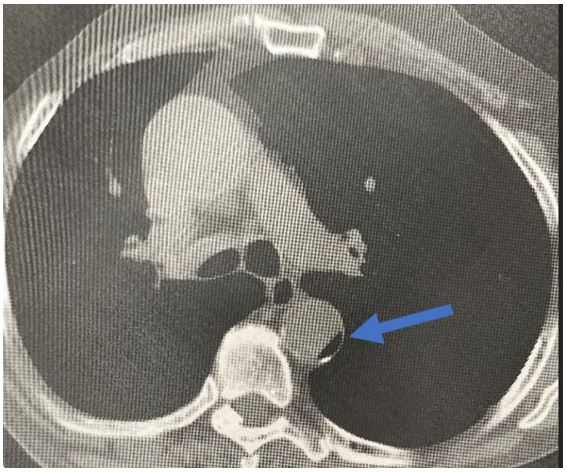

Figure 1. Chest CT scan showed an air embolus within the aorta (blue arrow).

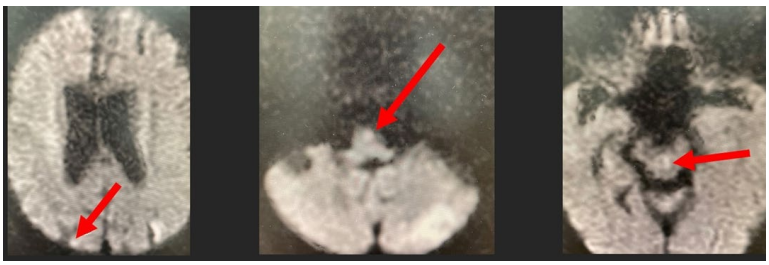

Figure 2. Brain MRI showed multiple areas of acute ischemia (red arrows).

The second case was a 75-year-old male with a past medical history of hypertension and benign prostate hyperplasia who presented to the radiology department for a CT-guided lung biopsy. A previous positron emission tomography had demonstrated a hypermetabolic lesion in the left upper lobe of his lungs. The patient underwent successful CT-guided biopsy of the lesion. Immediately after the procedure, he was unable to move his right arm or squeeze the hand of the bedside registered nurse. Soon after, he developed right lower extremity weakness and expressive aphasia.

A stroke alert was activated, and the patient was transferred to the ED. Upon arrival, the patient's neurological symptoms had resolved. The initial National Institutes of Health Stroke Scale (NIHSS) score was documented to be 0 in the ED. The ED physician consulted a tele-neurologist who recommended against administration of tissue plasminogen activator and recommended medical evaluation for transient ischemic attack (TIA).

CT of the patient's brain revealed no acute intracranial abnormality. The patient was given a 325-milligram (mg) dose of aspirin and was admitted for medical evaluation. He was started on a high intensity statin and clopidogrel in addition to his aspirin. He underwent neurological checks every four hours without any documented neurological deficits.

Review of the patient's medical record revealed a normal carotid ultrasound, which was completed almost a year prior to presentation. This study was ordered for further evaluation of dizziness by his

primary care physician. Post biopsy CT images were reviewed after the stroke alert, and they demonstrated mild expected local hemorrhage, as well as a small anterior pneumothorax. No air embolism was visualized in the pulmonary vessels or the heart. The patient underwent MRI and magnetic resonance angiography (MRA) of his brain and bilateral carotid vessels, respectively.

The MRI of the brain was negative for acute territorial ischemia, hemorrhage, or mass, but it demonstrated a few scattered foci in the periventricular and deep white matter, likely due to chronic microvascular ischemic changes. The carotid MRA demonstrated patent vessels without intraluminal narrowing. An echocardiogram with a bubble study showed normal ejection fraction, normal valvular morphology, and no evidence of a patent foremen ovale.

For the evaluation of risk factors, a complete blood count, complete metabolic panel, fasting lipid panel, and glycated hemoglobin (A1c) tests were obtained. A1c was 6.1%; all other tests were in the normal range. Given the onset and resolution of neurological symptoms and an otherwise negative work up for TIA, the patient was diagnosed with transient neurological deficits secondary to an air embolism. Upon discharge, his clopidogrel was discontinued and his statin dose was lowered to medium intensity due to his age and elevated atherosclerotic cardiovascular disease risk.

The third case involved a 73-year-old male patient with a medical history of alcohol dependence, tonsillar cancer status post radiation in 2010, generalized anxiety disorder, and benign prostate hyperplasia who presented to his primary care physician for annual follow-up. During the visit, the patient reported several weeks of bloating, postprandial vomiting, weight loss, in addition to not having any bowel movements or flatus for a few days. Given these symptoms, the patient was sent to the ED for workup of bowel obstruction. Upon arrival, a CT scan of the abdomen with IV contrast showed partial small bowel obstruction which was managed conservatively with NPO status, nasogastric tube placement, and therapeutic Gastrografin enema.

In addition to these findings, the CT scan showed air in the right ventricle. The cardiothoracic team recommended no surgical intervention at the time. The hospital's cardiology team recommended the Trendelenburg position, which requires placing the patient supine with the head at a 15-30 degree angle below the feet, with close monitoring of the patient's clinical status. The next day and on follow-up imaging, the air embolism in the right ventricle was found to have resolved. In addition, the patient's small bowel obstruction improved, and he had several bowel movements during his hospital stay.

Given the lack of procedures performed on the patient's initial presentation to the ED, and in the absence of otherwise identifiable risk factors, the cause of the air embolism was attributed to the IV access obtained for the purpose of IV contrast administration. Figure 3 is a CT image showing an air embolus in the right ventricle.

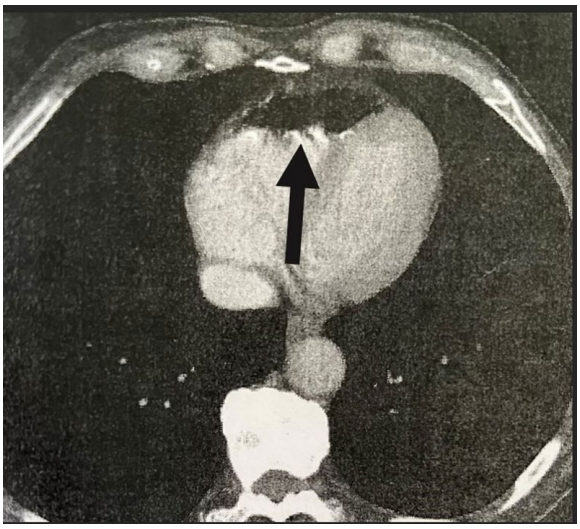

Figure 3. Air embolism was seen in the right ventricle (black arrow).

## DISCUSSION

SAE is classified as venous or arterial depending on the mechanism of gas entry into the bloodstream. Systemic arterial air embolism, rather than systemic venous embolism, is the major concern in percutaneous lung biopsy and occurs when air enters the pulmonary venous system and reaches the systemic circulation.[3] Identified risk factors for development of SAE include depth of the needle in the lesion, number of samples, endotracheal anesthesia, location of the lesion above the level of the left atrium, and prone and right lateral decubitus position of patient.[4,5] Furthermore, case studies have noted patient coughing as a presumed factor that increases the risk of air embolism due to the increased pressure gradient between the airway and pulmonary vein.[6] However, this is an unpreventable risk factor and there were no available data to differentiate the effects of a chronic cough or an acute episode of coughing during the procedure on the risk of an air embolus occurring.

Incidence of clinically apparent SAE was estimated at 0.061%, while clinically silent SAE may be as high as 3.8%.[7] This reported rate was less than the actual rate of SAE most likely because cases are often asymptomatic and not diagnosed.[3] When evident, clinical manifestations of SAE are varied with the most serious concerns being involvement of the cerebral and cardiovascular systems. Typically, patients present with symptoms of end-artery obstruction, such as cardiac arrhythmias, hypotension, drowsiness, dysphasia, stroke-like facial and limb weakness, seizures, and acute dyspnea.

Characterizations of the risk of venous air embolism in patients receiving intravenous contrast have been made. A study of 677 patients who underwent CT with IV contrast observed an incidence of 11.7%.[8] The air emboli varied in size, ranging from less than 1 cm to up to 2 cm in diameter, and found predominantly in the pulmonary artery. Given the small yet quantifiable risk of air embolism with procedures that necessitate intravenous access, knowledge of this phenomenon may reduce time to diagnosis and intervention in the symptomatic patient.

Early recognition of SAE is critical because simple temporizing measures have shown to lead to better outcomes.[2,3] Monitoring patient vitals and starting 100% high-flow oxygen therapy to treat hypoxia, as well as eliminate the gas from the bubbles is critical. Administration of supplemental high flow oxygen aids in the reabsorption of nitrogen

back into the blood, which in turn eliminates gas bubbles, as well as aids in overall oxygenation.

The role of hyperbaric oxygen therapy as first-line therapy is well defined.[2,3] The rationale behind this therapy is to reduce the mechanical obstruction of the embolus, to promote the conversion of nitrogen in the embolus to its soluble form, and to increase oxygen delivery to metabolically active tissues. Additionally, in the setting of arterial embolism, placing the patient in the right lateral decubitus position traps the air in the left ventricle and prevents it from entering the systemic circulation. After providing initial resuscitative and supportive measures, CT of the head and chest can be used to confirm the diagnosis of SAE, as well as rule out other complication of PCNB such as pneumothorax.[3] Approaching complications status-post PCNB with a wide lens can help to capture the diagnosis of SAE and trigger the initiation of a treatment algorithm to promote better outcomes.

## CONCLUSIONS

While procedures like CT-guided lung biopsy and IV contrast administration are indispensable tools for modern medicine, they are not risk-free. In fact, the introduction of any foreign object into the human body for the purposes of diagnosis or treatment carries a risk/benefit profile that needs to be assessed and made clear to the patient in the process of obtaining their informed consent. Although the complication of post-procedural air embolism is not common, its occurrence should be considered a possibility given the wide range of clinical outcomes that can result from it. From being completely asymptomatic, to causing self-resolving symptoms, to being a cause of mortality, air embolism can be unpredictable depending on its location. For this reason, it is vital to keep this differential diagnosis in mind when treating a patient who recently had a procedure done, even if it is as simple and routine as an IV-line placement (the rate of air embolism post IV contrast administration, although asymptomatic in most cases, has been reported to be as high as 23%[9]). In doing so, physicians can identify this entity at an earlier stage and plan accordingly based on the patient's symptoms and clinical status, as well as on the location of the air embolism.

## REFERENCES

[1] Lal H, Neyaz Z, Nath A, Borah S. CT-guided percutaneous biopsy of intrathoracic lesions. Korean J Radiol 2012; 13(2):210-226. PMID: 22438689.
[2] Ramaswamy R, Narsinh KH, Tuan A, Kinney TB. Systemic air embolism following percutaneous lung biopsy. Semin Intervent Radiol 2014; 31(4):375-377. PMID: 25435664.
[3] Hare SS, Gupta A, Goncalves AT, Souza CA, Matzinger F, Seely JM. Systemic arterial air embolism after percutaneous lung biopsy. Clin Radiol 2011; 66(7):589-596. PMID: 21530954.
[4] Freund MC, Petersen J, Goder KC, Bunse T, Wiedermann F, Glodny B. Systemic air embolism during percutaneous core needle biopsy of the lung: Frequency and risk factors. BMC Pulm Med 2012; 12:2. PMID: 22309812.
[5] Monnin-Bares V, Chassagnon G, Vernhet-Kovacsik H, et al. Systemic air embolism depicted on systematic whole thoracic CT acquisition after percutaneous lung biopsy: Incidence and risk factors. Eur J Radiol 2019; 117:26-32. PMID: 31307649.
[6] Fiore L, Frenk NE, Martins GLP, Viana PCC, de Menezes MR. Systemic air embolism after percutaneous lung biopsy: A manageable complication. J Radiol Case Rep 2017; 11(6):6-14. PMID: 29299093.
[7] Marchak K, Hong MJ, Schramm KM. Systemic air embolism following CT-guided percutaneous core needle biopsy of the lung: Case report and review of the literature. Semin Intervent Radiol 2019; 36(2):68-71. PMID: 31123374.
[8] Groell R, Schaffler GJ, Rienmueller R, Kern R. Vascular air embolism: Location, frequency, and cause on electron-beam CT studies of the chest. Radiology 1997; 202(2):459-462. PMID: 9015074.
[9] Ie SR, Rozans MH, Szerlip HM. Air embolism after intravenous injection of contrast material. South Med J 1999; 92(9):930-933. PMID: 10498176

*Keywords: air embolism, image-guided biopsy, contrast media*