Case Report

Beware of positive pressure: coronary artery air embolism following percutaneous lung biopsy

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A B S T R A C T

Patients undergoing percutaneous lung biopsy are at risk of developing a systemic air embolism. Air embolism may manifest as a catastrophic iatrogenic event with ischemic insult to the end organs, with sites of least resistance such as coronary and cerebral circulation the most susceptible. We review the available literature and present a case of iatrogenic air embolism during computed tomography guided percutaneous lung biopsy under general anesthesia. Management, outcome, and periprocedural factors that may have contributed to the complication are discussed.

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Introduction

Computed tomography (CT) guided percutaneous lung lesion biopsy is an indispensable diagnostic tool. Complications related to this procedure include pneumothorax, pulmonary hemorrhage, tumor seeding, and systemic air embolism (SAE), with latter reported incidence of 0.06% [1]. SAE may manifest as a catastrophic iatrogenic event with ischemic insult to the end-organ sites of least resistance, such as coronary or cerebral circulation. We present a case of a patient who suffered an iatrogenic coronary air embolism during CT-guided percutaneous lung biopsy manifesting as a heart block.

A 65-year-old man referred for biopsy of a right middle lobe nodule. Because of the proximity of the nodule to the diaphragm and major fissure, the case was performed with patient positioned right side up 30° and anesthesia with endotracheal positive airway pressure to facilitate an optimal access window by hyperexpanding the lung and controlling respiration (Fig. 1).

A 20-gauge coaxial system was directed into the lesion followed by 24-gauge needle aspirates. Attempts to redirect the access needle into the lesion resulted in moderate iatrogenic pneumothorax (Fig. 2A) necessitating placement of an 8-French pigtail catheter connected to wall suction into the pleural space.
with near complete resolution of pneumothorax (Fig. 2B). Procedure was completed after 5 fine-needle aspirates.

At the completion of sample collection, telemetry revealed intermittent third degree heart block with junctional rhythm in the 30 bpm range (telemetry strip unavailable). Transcutaneous pacing was instituted with capture at a rate of 60 bpm. Delivered oxygen was increased to 100%, and the table was placed in Trendelenburg. With these interventions, the patient remained hemodynamically stable without the need for additional supportive therapies.

Postprocedural CT scan following needle removal revealed iatrogenic air embolism in the segmental pulmonary vein draining the biopsy site (Fig. 2C) with nondependent air collecting in the superior right pulmonary vein (2D) and aorta (Figs. 2D and 3). Additionally, air was noted in the entirety of the right coronary artery (RCA), conus branch, proximal, mid, and distal RCA including 2 acute marginal, posterior descending, and posterolateral branches (Figs 4A-E). Heart block was likely a manifestation of air embolism to the atrioventricular nodal branch. Ten-minute and 20-minute delayed CT scans of the chest demonstrated gradual decrease with complete resolution of air contained within the aorta and coronary arteries 40 minutes later. CT imaging of the head did not reveal gas in the intracranial vasculature.

With resolution of intracoronary air, the rhythm returned back to normal sinus, and transcutaneous pacing was discontinued. General anesthesia was discontinued, and the patient was extubated. On examination, patients only complaint was reproducible pain at the chest tube site. He denied other chest pain sensation, headache, abdominal pain, weakness, and confusion. Follow-up 12-lead electrocardiogram showed borderline (mild prolongation of PR interval to 220 ms) first
degree atrioventricular block but no significant ST changes (Fig. 5). Troponin I was: 0.401 (+8 hours) → 0.625 (+12 hours) → 0.248 (+30 hours; reference ranges 0.000–0.034 ng/mL). Transthoracic echocardiogram revealed no intracardiac shunt and a preserved left ventricular ejection fraction of 55%. Magnetic resonance imaging of the brain ruled out acute infarct. The patient was discharged from the hospital 3 days later after resolution of pneumothorax.

In follow-up, the patient was diagnosed with stage IA T1b N0 M0 non–small-cell lung cancer, treated with stereotactic body radiation therapy with no evidence of neoplastic disease on 3-year follow-up.

Discussion

The proposed mechanisms for development of an air embolism in the context of percutaneous lung biopsy include open cannulation of the pulmonary vein or fistula creation between an airspace and pulmonary vein [2].

There have been approximately 90 reported articles referencing SAE in the literature over the past 4 decades associated with percutaneous lung biopsy. Retrospective observation study of approximately 600 patients calculated a higher radiological incidence for SAE of 3.8% and clinically apparent incidence of 0.49%, with SAE-specific mortality of 0.16% [3]. Reported independent risk factors for incidence of SAE include depth of the needle in the lesion, endotracheal anesthesia, location of the lesion above the left atrium, and prone position of the patient [3,4].

Endotracheal intubation with positive-pressure mechanical ventilation was used in this case to provide superior access window for the biopsy, avoiding the diaphragmatic dome, however, likely contributed to the air embolism.

The patient had sustained air embolism of the aorta and RCA, which clinically manifested as transient third degree

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Fig. 4 – (A–E). Embolized air in the entirety of the RCA, including conus branch (B), proximal, mid, and distal RCA, including 2 acute marginal and posterior descending (C), and posterolateral branches (D, E).

Fig. 5 – Twelve-lead electrocardiogram following cessation of transcutaneous pacing. Mild prolongation of PR interval. No significant ST segment changes.
atrioventricular block and periprocedural myocardial infarction. The anterior position of the RCA predisposes it to air embolism in setting of intraaortic air. Expected anatomic air embolization routes include RCA when supine, left coronary artery with right lateral decubitus positioning, right common carotid artery irrespective of the patient’s position, and left common carotid artery when prone [5].

Management of the aortic and coronary air embolism is mainly based on anecdotal experience. Successful conservative management included early recognition and hyperoxygenation with FiO₂ of 1.0 to promote air resorption by drawing gas nitrogen into soluble form. Trendeleburg positioning may be useful to prevent cerebral embolization if air trapped in the left ventricle. Muth et al. analysis yielded simplified approach with recommendations for flat, supine positioning for all SAE [6]. Additional treatment options to consider include hyperbaric oxygen by increasing dissolved O₂ deliver to tissues, pharmacologic coronary vasodilatation, direct percutaneous and/or endovascular aspiration of air from the aorta, or coronaries, or emergent cardiac bypass [1–15].

Reported case supports current literature that positive pressure ventilation should be avoided during percutaneous lung biopsy. Furthermore, our patient’s outcome without lasting morbidity is in accordance with conservative management of air embolism as an acceptable treatment option.

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