January 2020 Critical Care Case of the Month: A Code Post Lung Needle Biopsy

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A 67-year-old man with a history of stage IIA rectal adenocarcinoma post neoadjuvant chemoradiation presented with a near code event after elective CT guided biopsy of an enlarging left lower lobe lung nodule. The patient became bradycardic and profoundly hypotensive immediately after the CT guided biopsy with the following vital signs: Systolic BP < 90 mmHg, HR 40/min sinus bradycardia, SpO_2 on 100% oxygen non rebreather was 90%. Telemetry and EKG showed ST elevation in the anterior leads. He complained of vague arm and leg weakness and tingling, but did not lose consciousness or suffer a cardiac arrest.

A CT scan was performed about 2-3 minutes after the patient deteriorated (Figure 1).

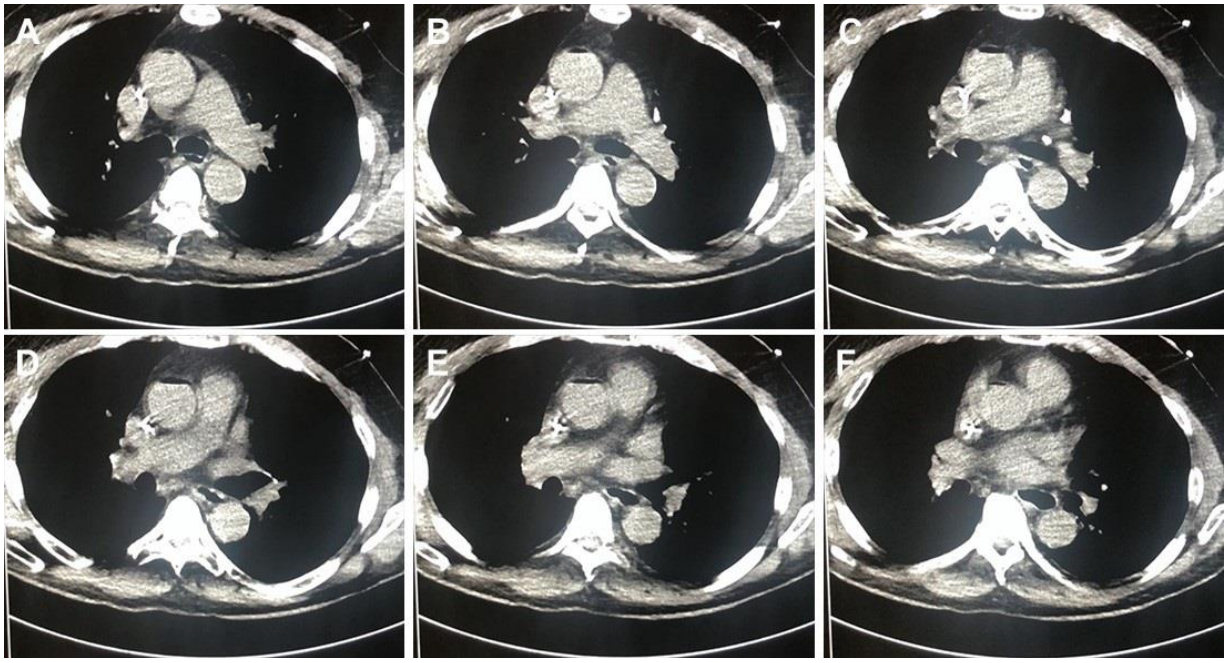

Figure 1. Representative images from CT scan in soft tissue windows.

What ***radiographic finding*** likely explains the patient's clinical deterioration?

1. Arterial air embolism
2. Pneumothorax
3. Pulmonary edema
4. Pulmonary Embolism
5. Venous air embolism

Correct!
1. Arterial air embolism

There is air in the proximal ascending aorta and possibly coronary arteries (Figure 2).

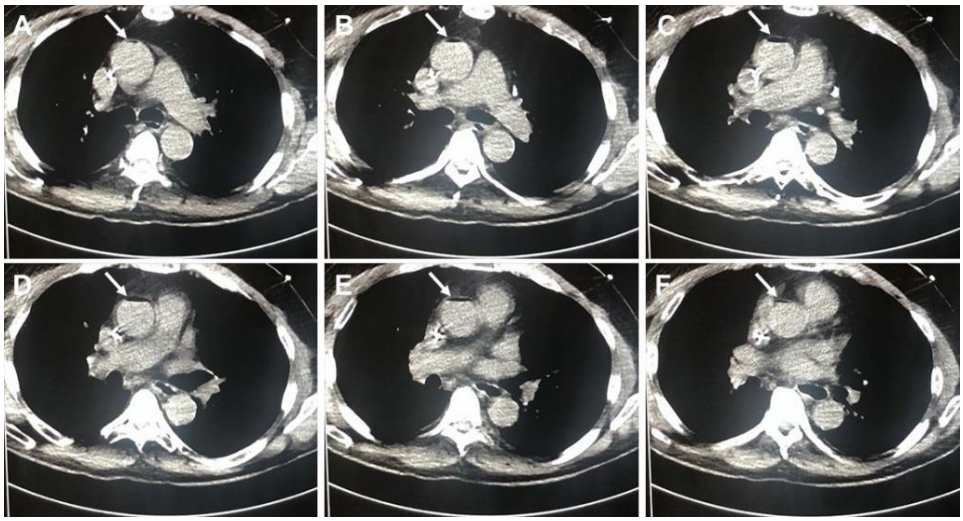

Figure 2. CT images showing air in aorta (arrows).

While there was a small pneumothorax in the left apex, this cannot be seen in the CT scan mediastinal windows. Whether there was air in the coronary arteries is not completely clear but clearly the air in the aorta had to traverse the sinus of Valsalva in the aortic root, from whence the left and right coronary arteries arise. Then low BP may have been due to either transient air emboli obstruction in the aortic outflow tract, and/or air emboli causing obstruction in the coronary arteries leading to myocardial ischemia.

The bradycardia and ST changes could be explained by air in the right coronary arteries, but no right sided EKG was obtained. Air may also have entered both left and right coronary circulations. Since the patient had EKG and troponin evidence consistent with an ST elevation MI, it is likely that some air entered one or both of the coronary circulations or at least occluded them long enough to cause ischemia.

Although this was a non-contrasted CT, a pulmonary embolism in the traditional sense is not likely, and there was no pulmonary edema on exam or in the lung windows (not shown). A pneumothorax was present but was too small to explain the shock and did not warrant chest tube placement.

What is or are the ***possible mechanism(s)*** of the above pathology post biopsy?

1. Bronchus/alveoli and the bronchial artery or its branches
2. Bronchus/alveoli and the bronchial vein or its branches
3. Bronchus/alveoli and the pulmonary artery or its branches
4. Bronchus/alveoli and the pulmonary vein or its branches
5. Direct air entry from the biopsy needled into any of the above vessels

Correct!
4. Bronchus/alveoli and the pulmonary vein or its branches

For air to end up in the aorta, it has to have entered the left heart first. This was a lung biopsy and the needle in approximation to the aorta.

Air entering the *pulmonary arteries* would end up getting lodged in a branch of this circulation, but probably would not make its way through the lung past the alveoli, into the pulmonary veins and back to the left heart.

Air entering the *bronchial arteries* would similarly end up getting stuck in smaller branches and again, it is not likely that the air would go the capillaries between bronchial artery and bronchial veins then end up in the right sided systemic circulation. If it did, the only way it could get into the aorta would be a right to left intra-cardiac shunt.

Air entering the *pulmonary veins* would get back to the left atrium and thence into the systemic circulation.

Air entering the *bronchial veins* would end up in the azygos vein on the right side or the hemiazygos vein on the left side and thence into the superior vena cava and right atrium, thence back into the right ventricle and pulmonary arteries. Without a right to left shunt, such air should not end up in the left side of the heart.

It is possible that air could have entered from the biopsy needle rather than from a bronchus or alveoli, but is less likely because the appropriate biopsy technique keeps fluid in the syringe at all times and gentle continuous suction to prevent this very event from occurring.

What is the ***ideal patient positioning*** for such a complication?

1. Left lateral decubitus
2. Reverse Trendelenburg
3. Supine
4. Trendelenburg
5. It makes no difference

Correct!
3. Supine positioning

Wherever the air in the sort ends up, nothing good will result. Most clinicians and patients would probably want to avoid stroke and air in the brain, or carotid circulation compared air in the peripheral arterial circulation. Supine positioning is favored over Trendelenburg positioning given arterial blood flow is more forceful than venous blood flow and air bubbles are propelled forward by the arterial blood despite head down positioning.

The usual thought that many critical care physicians may consider, is to place the patient in the left side down, feet up position. This is the mantra for venous air embolism from placing a central line in the superior vena cava or internal jugular veins, and is hoped that such a position will keep the air emboli away from the right ventricular outflow tract and lung, long enough to break up or be aspirated by an adventurous critical care fellow or experience IR/cardiologist.

What is a ***definitive therapy*** for such a complication once the patient has been stabilized?

1. Anticoagulation
2. Aspiration of air
3. Chest compressions
4. Hyperbaric oxygen therapy
5. Supportive therapy

Correct!
4. Hyperbaric oxygen therapy

Hyperbaric oxygen therapy (HBOT); provides O2 at pressures higher than atmospheric and at 100% providing supra physiologic levels of systemic hyperoxia. This allows for the creation of large gradients for nitrogen displacement from the air bubble which will reduce the air bubble size and the degree of mechanical obstruction. This would not be as helpful if the emboli occurred while the patient was already breathing 100% oxygen

An EKG revealed ST segment elevation in V1-V4 and cardiac troponin was elevated at 8.0 ng/mL (normal <0.4 ng/mL).

How would you ***manage*** the patient's ST-elevation myocardial infarction (STEMI)?

1. Anti-platelet therapy
2. Heparin drip
3. Left heart catheterization with PCI
4. Stenting of the left anterior descending artery
5. Supportive care, 100% F_IO_2

Correct!
5. Supportive care and 100% oxygen

Supportive care with 100% F_IO_2 only versus traditional therapies for STEMI given the underlying pathophysiology is not that of plaque or coronary thrombus.

Somewhat surprisingly, the patient did well. He was intubated electively as he was deteriorating and to put him on true 100% oxygen (non-rebreathing masks do not provide 100% oxygen in most situations). HBOT was sought, but no beds available, so he was kept on 100% oxygen despite P_aO_2 of > 100 mmHg for 24 hours. He was extubated in two days with no clear neurological sequelae, but a diagnosis of adenocarcinoma of the rectum with pulmonary metastases was made after reviewing the biopsy.

Presumably the air bubbles were broken up by the turbulence of the blood flow and gradual dissolution of the oxygen and CO_2 into the blood. It is unlikely that a true randomized, double-blind placebo, controlled, multi-center study will be conducted and clinicians will have to rely on theory, anecdote and perhaps common sense.

Reference

1. Malik N, Claus PL, Illman JE, et al. Air embolism: diagnosis and management. Future Cardiol. 2017 Jul;13(4):365-78. [CrossRef] [PubMed]