Lateral ST-elevation myocardial infarction from systemic air embolism after CT guided lung biopsy

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Arterial gas embolism; Cardiac; Coronary; Iatrogenic; Complications and management

Abstract

(Htay AM, Wilson E. Lateral ST-elevation myocardial infarction from systemic air embolism after CT guided lung biopsy. Diving and Hyperbaric Medicine. 2024 30 September;54(3):233–236. doi: 10.28920/dhm54.3.233-236. PMID: 39288930.) Systemic air embolism is a rare but potentially life-threatening complication of computed tomography (CT)-guided lung biopsy. The largest lung biopsy audits report an incidence rate of approximately 0.061% for systemic air embolism, with a mortality rate of 0.07–0.15%. A prompt diagnosis with high index of suspicion is essential, and hyperbaric oxygen treatment (HBOT) is the definitive management. We report the case of a 44-year-old lady who developed a lateral ST elevation myocardial infarction from coronary artery air embolism following CT-guided lung biopsy for evaluation of a left lung lesion. The biopsy was performed in the right lateral decubitus position, and the patient reported chest pain after coughing during the procedure. The clinician decided to proceed, taking four biopsy samples as no pneumothorax was identified in the intraprocedural CT image. The patient was noted to have hypotension with ongoing chest pain post-procedure. Resuscitative measures were taken to stabilise her haemodynamics, and she was successfully treated with HBOT with total resolution of air embolism. She developed a left sided pneumothorax post-treatment and needed intercostal chest drain insertion. The left lung fully re-expanded, and the patient was discharged home after day two of admission.

Introduction

Computed tomography (CT)-guided needle biopsy of lung lesions is a common procedure used to evaluate lung nodules or lesions. The procedure is relatively safe. The most common complication is pneumothorax followed by other minor complications such as pulmonary haemorrhage and haemoptysis.[1] Major complications such as pneumothorax requiring intervention, systemic air embolism, haemothorax and death are rare.[1] Although systemic air embolism is a very rare complication, most deaths from this procedure have been attributed to fatal air embolism.[2] Systemic air embolisms can present as arrhythmias, seizures, cardiac ischaemia or stroke.[2] We present a case of cardiac ischaemia after a CT-guided lung biopsy.

Case report

The patient provided written consent for publication of her deidentified case details and imaging.

A 44-year-old lady, on evaluation for non-specific chest pain, was found to have a left peripheral lung lesion that required CT guided lung biopsy. Past medical history included hysterectomy for cervical cancer, thyroidectomy for thyroid cancer, and hypertension treated with amlodipine. She was a smoker with a 40-pack-year history.

A 20G Quick-Core® biopsy needle (IZI Medical, Baltimore, USA) was used for the procedure which was performed in right lateral decubitus position. Her pre-procedure vital signs were normal with BP 101/56 mmHg, heart rate 80·min^{-1} sinus rhythm, respiratory rate 18 with a normal oxygen saturation of 97% breathing room air. During the biopsy the patient coughed, and then complained of chest pain with shortness of breath. She became hypotensive with a systolic blood pressure of 61 mmHg with heart rate 65·min^{-1} and an intravenous fluid (crystalloid) 1 L bolus was given. Blood pressure improved to 85/52 mmHg with heart rate 72·min^{-1}, respiratory rate 28·min^{-1}, oxygen saturation of 97% on 2 L·min^{-1} of oxygen via nasal prongs. There was no pneumothorax or pulmonary haemorrhage noted on intraprocedural CT images.

A medical emergency team call was activated from the CT suites because of ongoing hypotension with chest pain. There were no arrhythmias on cardiac monitor throughout the procedure. A large air embolism was identified in the left ventricle on immediate CT chest (Figure 1). A 12-lead electrocardiogram (ECG) showed ST segment elevation in the lateral territory and ST depression in inferior leads (Figure 2); changes which were not present in the pre-procedure ECG.

Figure 1
Coronal computed tomography view showing biopsy needle placement (larger arrow) and air embolism in LV (smaller arrow)

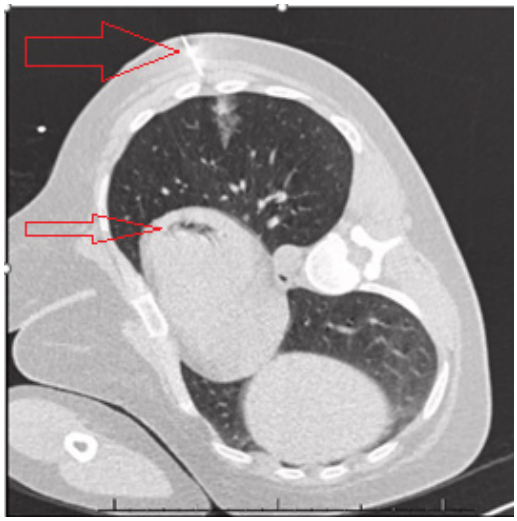


Figure 2
The electrocardiogram after biopsy and prior to hyperbaric oxygen treatment showing ischaemic changes (ST segment elevation in the lateral territory [particularly I and aVL] and ST depression in inferior leads [II, III, and aVF])

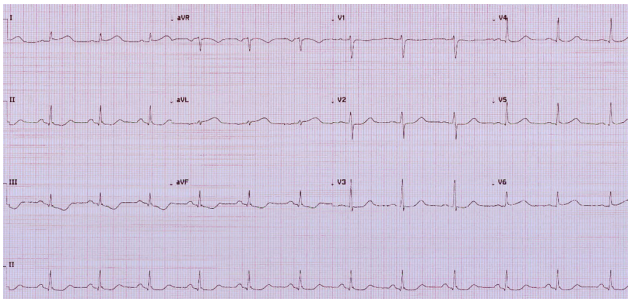
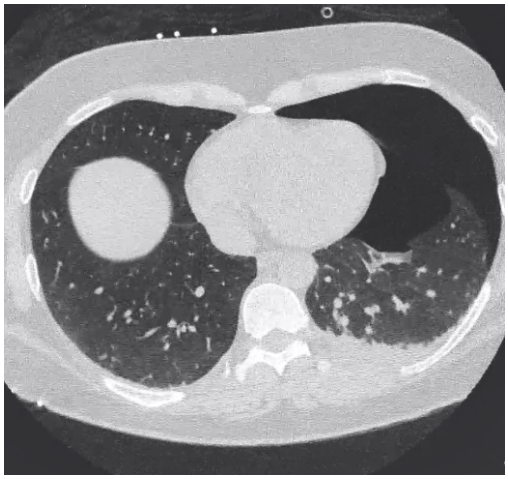


Figure 3
Coronal computed tomography view taken after decompression from hyperbaric oxygen treatment showing a large left pneumothorax but no intracardiac gas



Left ventricle lateral wall and apical hypokinesia was noted on a bedside echocardiogram by the cardiology team. Findings were consistent with a lateral territory ST elevation myocardial infarctionfrom coronary artery air embolism leading to haemodynamic instability.

The patient was given crystalloid fluid boluses up to 2.5 L in total, and two doses of metaraminol (1 mg and 0.25 mg). She was maintained head down in the right lateral decubitus position, keeping the left ventricle up to avoid a systemic air embolism shower. Oxygen was given via humidified high-flow nasal prongs with 50 L flow with 50% FiO_2 by the medical emergency team but was changed to 15 L oxygen with a non-rebreather mask to avoid positive pressure ventilation. An indwelling urinary catheter was inserted before transfer to the hyperbaric unit for hyperbaric oxygen treatment (HBOT).

Compression commenced approximately 90 minutes after air embolism was diagnosed. The patient was compressed to 284 kPa (2.8 atmospheres absolute) over five minutes and breathed 100% oxygen for 45 minutes prior to decompression to surface pressure over 30 minutes with no air breaks.

We chose the treatment table to avoid inert gas (nitrogen) administration to a patient with high risk of pneumothorax and to ensure no decompression requirement for attendants. This provided the capability to decompress at any time in an emergency with the anticipated risks of unstable arrhythmia and cardiogenic shock.

The hyperbaric physician acted as the attendant in-chamber so as to provide the capability of in-chamber intercostal catheter insertion, should she become haemodynamically unstable from a possible expanding pneumothorax on ascent.

The patient complained of left-sided chest pain after finishing decompression, and reduced air entry was noted in the left lung field. A left sided pneumothorax was confirmed by point-of-care ultrasound, with loss of lung sliding. The patient remained haemodynamically stable without needing immediate intercostal catheter insertion. It was therefore decided to return to the CT suite as planned for evaluation of resolution of the left ventricle air collection and quantification of size of the left-sided pneumothorax (Figure 3). The left ventricle air had totally resolved post-HBOT, but she needed an intercostal chest catheter for a large left-sided pneumothorax.

The patient was admitted to the intensive care unit in a stable condition for close observation. The ST segment changes were resolved on serial ECGs. Her initial troponin drawn 40 minutes after initial hypotensive episode with chest pain was normal at 5 $ng{\cdot}L^{-1}$ and peaked at 72 $ng{\cdot}L^{-1}$ 20 hours post-event (normal < 15 $ng{\cdot}L^{-1}$). Her left lung totally re-expanded within 24 hours, and she was discharged home after the second day of admission.

The patient was readmitted on the day of discharge with a recurrent left-sided pneumothorax and large amount of subcutaneous emphysema, following a coughing fit. She was treated conservatively in the Emergency Medical Unit (short stay unit) with 15 L·min^{-1} of oxygen via a non-rebreather mask overnight, and was discharged home the following day, after total resolution of the pneumothorax.

Discussion

COMPLICATIONS FROM CT-GUIDED LUNG BIOPSY

CT-guided lung biopsy is a frequently performed procedure by interventional radiologists, and the rate of common complications are quoted as: pneumothorax (20–35%); pulmonary haemorrhage (4.7–11%); and haemoptysis (2–7%). Systemic air embolism is extremely rare (incidence 0.02% to 0.7%) but may potentially lead to a fatal outcome.[1–4]

MECHANISM OF SYSTEMIC AIR EMBOLISM

The most common mechanism is believed to be that the biopsy needle opens the pulmonary vein to the atmosphere leading to systemic air embolism. Following lung biopsy, fistula formation between the pulmonary vein and the alveoli is another possible mechanism.

RISK FACTORS

Coughing during lung biopsy, positive end-expiratory pressure ventilation, prone position, location of the lesion above the level of the left atrium, and depth of needle in the lesion are documented risk factors associated with systemic air embolism.[4–7] In this case, the lesion was above the level of the left atrium and coughing promoted entrance of air into the pulmonary vein by increasing the pressure gradient between the airway and pulmonary vein.

DIAGNOSIS

Systemic air embolism is a clinical diagnosis, based on a high index of suspicion in patients who develop unexplained hypotension, arrhythmia or new neurological symptoms in the context of lung biopsy. Imaging sensitivity for detecting cerebral air embolism may be as low as 25%.[8] Transoesophageal echocardiography is the most sensitive imaging modality for identification of cardiac air, detecting as little as 0.02 ml·kg^{-1} of air.[9] Imaging is not recommended to make the diagnosis of systemic arterial gas embolism, due to its low sensitivity and its potential to delay definitive management.

MANAGEMENT

Management includes supportive and definitive treatments. Initial supportive measures include maintaining a patent airway, 100% oxygen as first aid, intravenous fluid resuscitation with crystalloids as needed to treat hypovolaemia or hypotension, and inotropes to maintain circulation. Aggressive hydration is unnecessary for isolated arterial gas embolism due to the risk of cerebral and pulmonary oedema. It is recommended to avoid glucose containing intravenous fluid as it can worsen cerebral injury.

Traditionally patients were kept head down with lateral decubitus position for ventricular systemic air embolism, but studies from animal models showed buoyancy had little effect on distribution of air embolism.[10,11] Current consensus is to keep the patient in a supine position to avoid cerebral oedema and to avoid provoking posturally-induced mobilisation of further gas emboli from places like the left atrium or ventricle if cerebral gas embolism is suspected.[12]

High inspired oxygen concentration not only increases nitrogen gradient between bubble and tissue but also improves oxygenation to ischaemic tissue. Administering 15 L·min^{-1} oxygen via a non-rebreather mask is an initial first aid treatment option before HBOT.

Hyperbaric oxygen is the definitive treatment for systemic air/gas embolism. It is indicated for all cases with neurological, cardiopulmonary or other associated clinical abnormalities. Isolated venous gas embolism without arterial gas embolism or clinical manifestations is not an indication for HBOT. Early HBOT is associated with favourable outcome in patients with iatrogenic arterial gas embolism.[13]

Normobaric oxygen therapy is an option with variable results, if HBOT is not available or is contraindicated. In other cases arising from lung biopsy, one who was treated with normobaric oxygen therapy had complete resolution,[14] but another had residual neurological deficit post normobaric oxygen therapy.[15] In a recent third case the patient was initially treated with normobaric oxygen with some improvement, but subsequent deterioration prompted a change in plan and progression to HBOT.[16]

Conclusions

Most systemic air embolism cases related to diving are traditionally treated with US Navy treatment table 6 (Royal Navy table 62), but we treated our patient with modified treatment table with total resolution of air embolism. Decisions should be made on a case-by-case basis, depending on the severity of illness, time to HBOT, and amount of air/gas loading in patients. Further studies should be conducted to confirm if treatment with US Navy Treatment Table 6 is necessary in all cases.

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