

Systemic air embolism as a complication of percutaneous lung biopsy

Introduction

Systemic air embolism is an uncommon complication of percutaneous transthoracic lung biopsy with the potential for serious neurological and cardiac morbidity. It may ultimately prove fatal. The true incidence is difficult to ascertain. Clinical features vary and range from confusion to stroke, arrhythmia, cardiac ischaemic features, loss of consciousness and death. Prompt recognition and institution of correct management is key to minimizing morbidity.

Discussion

Systemic air embolism is a rare but feared complication of percutaneous lung biopsy. If not rapidly recognized permanent neurological morbidity can occur along with myocardial ischaemia, profound bradycardia (Wu et al, 2011) and death.

Air passes from the pulmonary veins, into the left-sided cardiac chambers, via the aorta into the systemic and coronary arteries. Two mechanisms have been identified (Hare et al, 2011):

1. Formation of a traumatic broncho-venous or alveolar-venous fistula after passage of the biopsy needle

2. Direct inoculation of ambient air via the hub or the dead space of the coaxial needle when its tip is located within a pulmonary venous branch.

The risk of the former is exacerbated during coughing. In the authors' experience systemic air emboli most commonly happen in the immediate post-biopsy period (Bhatia, 2009), rather than intra-biopsy.

Risk factors include prone position of the patient, location of the lesion in the lower lobe, level of the lesion above the level of the left atrium, or a large number of biopsies (Freund et al, 2012).

Minimizing morbidity and mortality relies on early recognition and institution of temporising measures (see below). Certain stereotyped clinical features occur,

CASE REPORT

A 58-year-old man with a significant smoking history was referred with chest pain and weight loss. No cause for these self-limiting symptoms were found but multiple calcified and non-calcified nodules were present on computed tomography of the thorax, the largest measured 2.5 cm. Positron emission tomography demonstrated low-grade uptake (SUV max 2.7) in some of the nodules. Repeat computed tomography 3 months later showed enlargement of some of the nodules.

Percutaneous biopsy of the dominant nodule in the left lower lobe was performed under computed tomography guidance (*Figure 1*). Pre-procedure lung function was not performed as per the authors' local protocol (Abdullah et al, 2016). The patient was positioned in a right semi-decubitus position. A 19 G coaxial needle was used to access the nodule. Fine needle aspiration was performed for microbiology, followed by four 20 G core biopsy samples for histology.

On completion of sampling the coaxial needle was removed with simultaneous saline flush to seal the tract. A tiny localized pneumothorax was present on the final computed tomography image. As the coaxial needle was withdrawn the patient began coughing. He was immediately positioned in the right lateral decubitus position while still on the computed tomography gantry, to aid expectoration and avoid aspiration. He was otherwise asymptomatic, fully alert and oriented. After 2 minutes he was transferred to a trolley, positioned supine and wheeled to the recovery area.

Nursing staff soon became concerned that the patient was acting oddly. On review he was confused, clammy and dysarthric.

Haemodynamic parameters and oxygen saturations were normal. He was immediately turned back to the right lateral decubitus position and high flow oxygen administered via a non-rebreather reservoir mask. Over the following 2 minutes, aphasia and dense left hemiparesis progressively developed. He was transferred back to the computed tomography scanner and a non-contrast computed tomography scan of the brain and thorax was performed, with the patient still in the right lateral decubitus position. A few tiny locules of gas were seen in a non-dependant position in the left ventricle suggesting systemic air embolism (*Figure 2*). No air was seen in the coronary arteries, aorta or brain. No cerebral infarction was seen.

The patient was transferred back to recovery in the right lateral decubitus position and with continuous high flow oxygen. The aphasia and hemiparesis progressively resolved over 30 minutes of conservative therapy with complete recovery of normal speech and motor function. On questioning the patient reported a feeling of tingling in his left arm and leg before the development of further neurological symptoms. He also stated he was fully aware during the episode and could hear everything being said around him (i.e. expressive dysphasia). The tiny pneumothorax resolved over 60 minutes and the patient was discharged as per the authors' institution's early discharge protocol (Tavare et al, 2016), walking out of the department. He was well and asymptomatic on telephone follow up on day 1 and 7 post-biopsy. Histopathology revealed granulomatous inflammation with no evidence of malignancy and the patient had no neurological deficit at clinical follow up 1-month post procedure.

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