Case report

Air embolism following bronchoscopy with fine needle aspiration: An unexpected complication

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ABSTRACT

Flexible fiberoptic bronchoscopy with fine needle aspiration is a common procedure, useful in the diagnosis and assessment of lung disease. There are known complications associated with such a procedure that are well documented in the literature. However, there are only four cases of air embolus following fine needle aspiration during bronchoscopy described in the literature. Due to the varying clinical manifestations of the complication, it remains underrecognized by the clinical community and was not described at all by the most recent British Thoracic Society 2013 statement on bronchoscopy. The following two case reports describe incidences where air emboli ensued following bronchoscopy with fine needle aspiration. They examine four notable, and arguably avoidable, risk factors that can exacerbate an air embolus and offer guidance on both imaging and treatment for any physician faced with a corresponding clinical picture.

1. Introduction

Flexible fiberoptic bronchoscopy with Fine Needle Aspiration (FNA) of lymph nodes is a widely accepted and safe procedure which is useful in the diagnosis and assessment of lung disease. Minor complications include vasovagal spasm, bleeding and arrhythmias with an incidence of 6.5% [1]. Major complications are much less common but include respiratory arrest, pneumonia, pneumothorax and major bleeding occur at 0.5% [2]. Systemic air embolus is an extremely rare complication of computed topography (CT) guided transthoracic biopsy, reported to be around 0.02%–0.07% [3–6]. In a prospective study of complications following Flexible Fiberoptic Bronchoscopy in 908 patients there were no instances of neurological events [7]. To our knowledge only five cases of air embolus following fibroptic bronchoscopy have been reported [8–13] (Table 1). Furthermore, in the recent British Thoracic society statement 2013 on bronchoscopy there was no mention of air embolus as a complication of fibroptic bronchoscopy [14]. We are describing two cases of air embolus causing a cerebral vascular accident because we feel that this complication is under recognised and under reported and can potentially be fatal [10].

2. Case report 1

A 69 year old gentleman was referred to the rapid access unit for recurrent chest infections. It was subsequently noted on CT of the thorax that the patient had an enlarged right hilar lymph node measuring 2.5 × 1.6 cm, with multiple mediastinal lymph nodes, as well as evidence of centrilobular and paraseptal emphysema (Fig. 1). He was subsequently referred for bronchoscopic evaluation using a standard (adult size) bronchoscope. FNA was performed using 1.1 mm 19-gauge needle.

During the procedure the patient became hypoxic and unresponsive. Hemiparesis was evident on the left side. A CT brain and CT Intracranial Angiogram, at the time of the event, was reported as no evidence of acute ischaemia or intracranial occlusion. Although, in retrospect, it appears that there was effacement of the cerebral sulcus on the right side, in keeping with acute ischaemia.

The only finding was of air passing through the base of the skull, along the lacerum segment of internal carotid artery (Fig. 2).

Alt was noted that the chest tube was noted to be oscillating with respiration and was no longer bubbling, suggestive of a resolution of the air leak. There was no evidence of bleeding in the chest tube or in the tube site. Moreover, the patient was conversing and had a complete resolution of her shortness of breath and chest pain.

Despite absence of identifiable clot in the middle cerebral artery on the cerebral angiogram the decision was made to thrombolysis the patient based on the severity of the hemiparesis and the identifiable onset of the neurological symptoms. Almost immediately after the thrombolysis was administered haemoptysis ensued. This was further complicated by the patient developing generalised tonic-clonic seizures. Rapid intubation secured the airway.

A repeat CT chest was performed identifying new onset mediastinal haemorrhage (Fig. 3) and evidence of diffuse pneumomediastinum (Fig. 4) which was managed conservatively.
Haemoptysis resolved within 2 hours spontaneously and seizures ceased in 24 hours after Phenytoin loading. A further 24 hour period saw the patient extubated, regaining full consciousness, with no cognitive impairment and minimal residual left-sided hemiparesis. Repeat CT brain showed some subtle effacement of the gyral pattern in the right cervical hemisphere over the vertex but no low-density change in brain parenchyma. However, subsequent Magnetic Resonance Imaging (MRI) found right cerebral hemisphere watershed ischaemia with foci of acute infarction, consistent with the left-sided hemiparesis demonstrated. It was concluded in retrospect that this was an air embolus (Fig. 5).

Stroke rehabilitation was performed, and full recovery was made within two weeks. A repeat CT thorax showed no progression of his mediastinum lymphadenopathy, thus excluding any possibility of malignant lesion of the node. Unfortunately, there were no facilities to enable hyperbaric oxygen treatment at this hospital and by the time the patient was stable enough for transfer, their neurological status had improved considerably.

3. Case report 2

69-year-old male with a history of productive cough and recent admission for pneumonia went on to have a CT thorax that reported as a 3 × 2.1 cm soft tissue mass at the right hilum with adjacent hilar lymphadenopathy and mild pulmonary fibrosis on CT thorax (Fig. 6). Due to a history of bowel cancer, with sigmoid colectomy two months prior, there was a concern for metastases to the lungs. FNA was performed using 1.1mm 19-gauge needle.

The patient became hypoxemic on the table and became unresponsive. Reversal of Midazolam sedation was attempted with no marked response. Similarly, to the first case, there were no facilities to enable hyperbaric oxygen treatment at this hospital. However, the patient did regain consciousness, but suffered a left-sided hemiparesis.

CT brain and CT angiogram intracranial showed marked evidence of pneumocephalus in the right middle cerebral artery (MCA) and within the extra-axial space on the right side, characteristic of air emboli (Fig. 7).

Similarly to the first case, the patient made a full recovery with the assistance of a physiotherapist and occupational therapist within a week and due to a lack of adequate facilities, the patient was not treated with hyperbaric oxygen therapy but rapid neurological improvement was observed clinically.

4. Discussion

A small number of air emboli cases feature in the literature, but it is said that the number is underreported as asymptomatic patients will go undiagnosed [6,15]. We feel that our two cases are instructive in this respect as they highlight this potential, albeit rare, risk of bronchoscopy. In doing so, physicians will become increasingly aware of it but also aware that a full recovery can be made. There is a wide spectrum of possible presenting symptoms that a patient can manifest with, thus making diagnosis difficult [3,4]. In fact, in our first case we felt that the patient had suffered an acute ischaemic cerebrovascular accident, that was not a direct consequence of the bronchoscopic procedure. The patient was thrombolysed as per acute stroke guidelines as we did not recognise air embolus as aetiology until we reviewed the case retrospectively [16]. Therefore, it is vital that physicians are mindful of and

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**Abbreviations**

- CAAE: Cerebral Arterial Air Embolism
- COPD: Chronic Obstructive Pulmonary Disease
- CT: Computed Topography
- FNA: Fine Needle Aspiration
- MCA: Middle Cerebral Artery
- MRI: Magnetic Resonance Imaging

**Table 1**

Table of who developed CAAE following diagnostic flexible fibreoptic bronchoscopy.

<table>
<thead>
<tr>
<th>Reference Number</th>
<th>Author and year</th>
<th>Personal Characteristics</th>
<th>Procedure</th>
<th>Bleeding</th>
<th>Diagnosis, treatment and outcome of CAAE</th>
<th>Lesion of Infarction</th>
<th>Oxygen delivery</th>
<th>Seizure</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[11]</td>
<td>Dhillon et al., 2004</td>
<td>Age 55</td>
<td>TBNA, TBLB</td>
<td>50ml</td>
<td>Left-hemi</td>
<td>Right frontal</td>
<td>NBO2</td>
<td>(+)</td>
<td>100% recovered</td>
</tr>
<tr>
<td>[10]</td>
<td>Azzola et al., 2010</td>
<td>Sex Male</td>
<td>TBNA</td>
<td>N/A</td>
<td>Right-hemi</td>
<td>Bilateral</td>
<td>HBO2</td>
<td>(+)</td>
<td>Almost improved</td>
</tr>
<tr>
<td>[16]</td>
<td>Azzola et al., 2010</td>
<td>Underlying Disease COPD</td>
<td>N/A</td>
<td>N/A</td>
<td>Left-hemi</td>
<td>Bilateral</td>
<td>Intubation</td>
<td>N/A</td>
<td>Dead</td>
</tr>
<tr>
<td>[12]</td>
<td>Ragey et al., 2013</td>
<td>Smoking history (pack years) N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Left-hemi</td>
<td>Bilateral</td>
<td>HBO2</td>
<td>N/A</td>
<td>Dead</td>
</tr>
<tr>
<td>[13]</td>
<td>Goto et al., 2014</td>
<td>Suspected Disease Lung Cancer</td>
<td>N/A</td>
<td>N/A</td>
<td>Left-hemi</td>
<td>Bilateral</td>
<td>HBO2</td>
<td>N/A</td>
<td>Dead</td>
</tr>
</tbody>
</table>

N/A, not available; COPD, chronic obstructive pulmonary disease; IPF, idiopathic pulmonary fibrosis; TBNA, transbronchial needle aspiration, HBO2, hyperbaric oxygen; NBO2, normobaric oxygen.

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**Fig. 1.** Enlarged right hilar lymph node measuring 2.5 × 1.6cm.
can recognise the symptoms of a potential air embolus. The onset of neurologic symptoms (confusion, personality change, dizziness, visual disturbance, or paraesthesia); delayed recovery from anaesthesia; cardiovascular instability and the presence of air bubbles in retinal vessels on fundoscopy, are some quick and non-invasive indicators that should alert the doctor to the possible occurrence of an air embolus [5]. The second case on the other hand was immediately recognised as an air embolus and was consequently managed accordingly from the start. It is important to note that both patients, despite demonstrating very significant neurological deficits, recovered completely despite no targeted intervention directed against the air embolus.

It is not yet clear how an air embolus is induced while performing FNA during bronchoscopy. The literature yields some hypotheses on causation related to the FNA procedure. First, air may enter the pulmonary system through the needle. If atmospheric pressure exceeds pulmonary venous pressure, air emboli can traverse down the pressure gradient and establish themselves in the pulmonary veins. This can occur if the patient were to inhale deeply during procedure. Second, if internal airway pressure distal to the scope were to rise, the risk of embolus is thought to increase. For example, actions that mimic valsalva manoeuvre, such as coughing and straining, during the procedure, can create a sudden pressure increase distal to the needle, thus inducing embolus [3–6,17–20]. A potential learning point for physicians is to consider increasing sedation in patients who show signs of airway resistance during the procedure, including coughing, straining or deep breathing, to reduce risk of embolus. It is of note that in both our cases the patients had severe episodes of coughing during the performance of the FNA procedure. In addition, patients with chronic obstructive pulmonary disease (COPD) or those on positive pressure ventilation should also be considered for increased sedation as distal pressure gradient
may be exacerbated due to increased air trappings [17].

A further hypothesis suggests that a needle may penetrate simultaneously at an air-containing space, such as a nearby pulmonary alveolar space or bronchus, and a nearby pulmonary vein, which can create a communicating fistula [3–6,17–20]. Lastly, radiology literature suggests an association between the size of the needle used and increased incidence of air emboli [6].

Diagnosis of an air embolus is primarily clinically suspected, but can be difficult. However, second to clinical suspicion, brain and CT scan can provide a definitive diagnosis by showing air bubbles in the cerebral vessels, aorta, pulmonary veins and left atrium and ventricle [21]. However, a pivotal point is that an air embolus less than 1.3 cm may not be detected by CT brain or CT intracranial angiogram and will only be seen on MRI. Therefore, to be aware of the need for an MRI, as opposed to a CT brain or intracranial angiogram, if you experience a patient with clinical symptoms that allude to a cerebral vascular accident is vital to obtain definitive exclusion criteria. Case 1 is a clear example of where the air embolus was initially overlooked due to the embolus size likely being < 1.3 cm and thus not appearing on CT imaging.

If patients are correctly diagnosed as having an air embolus as opposed to a cerebral vascular accident, correct treatment can be initiated and is shown to decrease mortality rate to 7% [5,6]. Hyperbaric oxygen therapy is considered the gold standard for treatment of systemic air embolism [22]. By breathing 100% oxygen increases ambient pressure above that of the atmosphere and consequently the size of gas bubbles decreases. This is because the increased ambient pressure compresses the bubble (air embolus), reducing its size. Secondly, it also induces systemic hyperoxia which has the following effects. 1) Oxygen replaces Nitrogen in the air bubble by diffusion. 2) A large quantity of oxygen can then dissolve into the plasma. 3) Increased oxygen is also diffused out into the tissue [18]. In addition, the patient can be placed in the left lateral decubitus position with a lowered head [5,6,15,20].

In summary, air embolism is an underrecognized and potentially disastrous complication of FNA during bronchoscopy.

Summary conflict of interest statement

The authors declare no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2018.09.012.

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* has provided final approval of the version to be published;
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