Reminder of important clinical lesson

Atrial fibrillation and pneumothorax after transthoracic needle lung biopsy

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An obese 65-year-old male smoker with chronic obstructive pulmonary disease developed an iatrogenic pneumothorax with pulmonary haemorrhage during an elective transthoracic needle biopsy of a pulmonary lesion. Successful re-inflation was achieved with a chest drain which was then removed before transfer to the medical ward. He later developed persistent atrial fibrillation with breathlessness and haemoptysis. He was treated empirically for a pulmonary embolus, which was subsequently ruled out with CT pulmonary angiogram. Serial chest radiographs demonstrated recurrence of his pneumothorax and a chest drain was re-inserted. His atrial fibrillation was erroneously managed as supraventricular tachycardia, which was resistant to vagal manoeuvres and adenosine but later responded to intravenous amiodarone before a further relapse. Upon successful management of the pneumothorax, his atrial fibrillation terminated. This case highlighted the persistent and serious nature of complications posttransthoracic needle biopsy.

BACKGROUND

Lung cancer is a major killer in the world and primary malignancies often present as a solitary pulmonary nodule.¹ With the abundant availability of chest CT, these nodules are diagnosed with increasing regularity.² In USA alone, several hundred thousand pulmonary nodules are diagnosed each year.² However, not all nodules are malignant and a large proportion are found incidentally.² Histological confirmation is therefore required to make definitive diagnoses as well as to stratify patients for palliative or in certain cases, curative chemotherapy and radiotherapy.³ Histology is also important for accurate prognosis. Bronchoscopy and surgical resection are feasible options only in selected individuals.³ In patients contraindicated for surgery or in whom bronchoscopy is inadequate, percutaneous transthoracic needle lung biopsy (PTNB) presents an attractive modality for tissue diagnosis.³ PTNB was first employed in 1883 by Leyden and later by Menetrier in 1886.³ Procedure related complications were prevalent at the time, largely due to the lack of imaging guidance. Later, with the availability of CT guided needle biopsies, complication rates have dramatically reduced, with an increase in diagnostic accuracy.³

Transthoracic needle biopsy complications

The commonest complication of PTNB is pneumothorax, followed by pulmonary haemorrhage. Other rarer complications include air embolism and tumour seeding.⁴ Pneumothorax occurs in up to 62% of cases of lung biopsies.⁵ Predisposing factors include the presence of chronic obstructive pulmonary disease (COPD), small lesions with increased depth from skin multiple punctures traversing the pleura and wider angle of needle insertion.⁴ Pulmonary haemorrhage occurs in up to 27% of transthoracic needle biopsies, which is usually evident by the presence of peri-

lesional opacities on CT or symptomatic haemoptysis. Predisposing factors are similar to those of pneumothorax with an added risk from the presence of pulmonary hypertension.⁴ Life-threatening pulmonary haemorrhages can often be avoided by proper management of coagulation defects and avoiding the heart and major vessels in the needle trajectory.⁴ Air embolism is a rare complication with a reported prevalence of 0.061% and is limited to case reports in the literature.⁶ There are two postulated mechanisms for pulmonary haemorrhage; first, rapid inspiration coinciding with stylus removal when the biopsy needle tip is wedged within a pulmonary vein introduces air into the circulation.⁷ This phenomenon relies on an atmospheric pressure which exceeds that of the pulmonary venous pressure. The second mechanism involves the formation of an arterial-alveolar fistula as the needle traverses both a pulmonary vein and air spaces. Air moves from alveolar spaces into the pulmonary circulation during periods of increased intrapulmonary pressure, for example, coughing and rapid expiration.⁷ Air embolism may cause life-threatening arrhythmias or seizures if cerebral involvement occurs.⁸

Supraventricular tachycardia and atrial fibrillation

Supraventricular tachycardia (SVT) refers to tachyarrhythmias necessitating involvement of atrial tissue or the atrioventricular node (AVN) for its perpetuation.⁹ It has an incidence of 35/100 000 per year and encompasses AVN re-entrant tachycardia (AVNRT), AVRT and atrial tachycardia.¹⁰

SVT usually has an abrupt onset and termination with good prognosis. Many patients have little or no symptoms.¹¹ Since the AVN is involved in the majority of cases, vagal manoeuvres should first be attempted to block atrioventricular conduction and is often sufficient to terminate

Figure 1 Initial CT chest scan showing a 1.2 cm pulmonary nodule in the left lung. This lesion required histological diagnosis. If confirmed malignant, it was staged T1a, N0, M0.

benign episodes of SVT.¹¹ These measures are contraindicated in patients with carotid bruits or known carotid artery disease.¹¹ Cases resistant to vagal manoeuvres may be treated with pharmacological atrioventricular conduction block. In non-asthmatic patients, adenosine is the first line agent recommended by the American Heart Association/American College of Cardiology/European Society of Cardiology (AHA/ACC/ESC), since there is evidence that it terminates both nodal and atrial tachycardias.¹¹ A 12 mg dose of adenosine is reported to revert >90% of SVT.¹¹ In asthmatic patients, reasonable alternatives are calcium channel antagonists such as verapamil or diltiazem.¹¹ Resistant cases or patients with impaired ventricular function may be treated acutely with amiodarone.¹¹ However, amiodarone has no role in the long term management of SVT due to its side effect and toxicity profiles.¹¹ Re-entry tachycardias which involve accessory pathways may be cured with radiofrequency ablation.¹²

Atrial fibrillation (AF) is the commonest type of narrow complex tachycardias.¹⁰ It is classified into 1) paroxysmal, lasting less than 7 days and terminates spontaneously, 2) persistent, lasting more than 7 days and terminates either spontaneously or with cardioversion and 3) permanent, where successful cardioversion is not possible.¹⁰ The treatment of AF involves rate or rhythm control with consideration for thromboprophylaxis.¹⁰ Two landmark studies showed that there are very little differences in terms of long term mortality between rate and rhythm control. These were (AF Follow-up Investigation of Rhythm Control) and Rate Control versus Electrical Cardioversion for Persistent Atrial Fibrillation.¹⁰

CASE PRESENTATION

An obese 65-year-old male heavy smoker with COPD was referred from his local district general hospital for a PTNB of a left upper lobe pulmonary lesion. Prior CT scan

showed a small 1.2 cm spiculated nodule suggestive of primary lung tumour, which if proven malignant was staged as T1a N0 M0 (figure 1). He was taking regular inhaled bronchodilators and his medical history was otherwise unremarkable. He was well on arrival with a body mass index of 32, temperature 36.5°C, blood pressure 140/95 mm Hg, pulse 85 regular, respiratory rate of 16 per min and pulse oximetry 95% on air. On auscultation, there were mild scattered wheezes bilaterally with normal heart sounds. Admission ECG (figure 2a) and chest x-ray were unremarkable. Initial screening blood tests including clotting studies were normal.

Written informed consent was obtained and PTNB was performed under CT guidance with aseptic technique. A standard biopsy needle was used. The procedure was complicated by the development of an anterior pneumothorax, which worsened with withdrawal of the biopsy needle (figure 3). There was concurrent development of pulmonary haemorrhage near the lesion site (figure 3). He was given oxygen via a nasal cannula. A 12 french calibre chest drain was inserted using the Seldinger technique and the left lung was successfully re-inflated (figure 4). The chest drain was then removed and the patient was transferred to the medical ward for observation where he was given a prophylactic dose of low molecular weight heparin (LMWH).

He was well for the next 3 h but subsequently developed sudden onset shortness of breath and haemoptysis. He denied chest pain or sputum production. Initial assessment showed a patient in respiratory distress with an unobstructed airway but unable to complete sentences. He was tachypnoeic (30 per min) with accessory muscle breathing and tachycardic at 150 beats per min. Bedside observations showed temperature of 36°C, pulse oximetry of 85% with a blood pressure of 187/121 mm Hg. Further examination revealed no cyanosis, normal jugular venous pulse, centrally positioned trachea, resonant percussion notes and

Figure 2 A collection of lead II rhythm strips during the patient's stay in hospital. a) Normal sinus rhythm on admission. b) Atrial fibrillation which developed 3 h after lung biopsy, with rapid ventricular response of 192/min. It was resistant to vagal manoeuvres and three successive doses of intravenous adenosine. c) Normal sinus rhythm was achieved with intravenous amiodarone therapy. d) Soon after CT pulmonary angiogram, the patient relapsed back into atrial fibrillation with a ventricular response of 156/min.

expiratory wheezes bilaterally. Heart sounds were normal and abdomen was soft and non-tender. Neurological examination was unremarkable.

Telemetry ECG showed AF with a peak rate of approximately 192 per min (figure 2b), ranging from 150 to 200 per min. His initial arterial blood gas sample showed type 2 respiratory failure (pH 7.35, pCO₂ 6.5 kPa, pO₂ 6.3 kPa, bicarbonate 24.7, base excess 0, oxygen saturation 87.9% on room air). Venous blood sample was normal without anaemia or leucophilia. Serum Troponin I was normal. A portable chest radiograph did not show any obvious pneumothorax or focal consolidation. He was given nebulised salbutamol and ipratropium with oral prednisolone. Oxygen therapy was delivered via a Venturi mask at 28% aiming for oxygen saturation of 88 to 92%. His repeat arterial sample showed slight improvements (pH 7.36, pCO₂ 6.0 kPa, pO₂ 8.2 kPa, bicarbonate 25.4, base excess 0, oxygen saturation 92% on room air). The patient became less breathless following bronchodilator therapy, however, the AF remained.

Given the sudden onset breathlessness, haemoptysis, ECG findings of tachycardia, pulmonary embolus was suspected. The patient was treated empirically with 1.5 mg per kilogram body weight of LMWH. However, he was deemed haemodynamically unstable for transfer to the radiology department for a CT pulmonary angiogram (CTPA). It was felt that the haemoptysis could also be due to a simple complication of PTNB, but this was a diagnosis of exclusion in the face of a possible pulmonary embolus.

Once empirical LMWH was commenced, priority turned to the management of AF. The patient was initially erroneously managed as SVT. Carotid sinus massage and the valsalva manoeuvres were performed without success. Subsequent doses of intravenous adenosine (3 mg, 6 mg, 12 mg) were also ineffective in terminating the aberrant rhythm (figure 2b). Pharmacological cardioversion was attempted. A loading dose of intravenous amiodarone (300 mg) was given which resulted in a moderate reduction in heart rate from 137 to 123 per min over 30 min. A further

continuous infusion of 900 mg successfully cardioverted the AF back to normal sinus rhythm (NSR) (figure 2c).

A CTPA was performed after sufficient control of AF, which demonstrated no evidence of pulmonary emboli. The patient was switched back to a prophylactic dose of LMWH. However, shortly after returning to the ward from the radiology department, the patient reverted back into AF with a ventricular rate of approximately 156 per min. A repeat chest radiograph showed a left-sided pneumothorax (figure 5) and a second chest drain was inserted. Over the next 24 h, his clinical state improved and he spontaneously cardioverted back to SR. He was discharged within a week without any further episodes of haemoptysis.

INVESTIGATIONS

- ▶ Routine blood tests – no evidence of anaemia, clotting abnormalities or infection
- ▶ Arterial blood gas samples – type 2 respiratory failure
- ▶ ECG – AF
- ▶ Chest radiograph – initially unremarkable, later showed left sided pneumothorax
- ▶ CTPA – no evidence of pulmonary emboli.

DIFFERENTIAL DIAGNOSIS

- ▶ AF due to pneumothorax with haemoptysis secondary to procedure related pulmonary haemorrhage.
- ▶ AF and haemoptysis secondary to pulmonary embolus.
- ▶ AF secondary to air embolism.

TREATMENT

Intravenous amiodarone bolus and subsequent infusion for fast AF.

OUTCOME AND FOLLOW-UP

The patient was followed up in the respiratory outpatient clinic. He had an outpatient echocardiogram which was normal with an ejection fraction of 64%. A 24 h

Figure 3 A biopsy sample was taken with under CT guidance, however this was complicated by an anterior pneumothorax with a maximal rim of air 3 cm from the anterior chest wall. There was also evidence of peri-lesional pulmonary haemorrhage in the left lung.

ECG monitor did not show any recurrences of AF after discharge. Histological studies of the pulmonary biopsy revealed squamous cell carcinoma and he was subsequently referred to the oncology department of his local hospital. Subsequent CT scan of his abdomen and pelvis showed liver metastasis and he underwent palliative chemotherapy with radiotherapy.

DISCUSSION

In summary, the patient underwent an eventful PTNB of a solitary pulmonary nodule, which was later demonstrated to be squamous cell carcinoma with abdominal and pelvic metastasis. The PTNB was complicated by a pneumothorax, initially successfully managed with a chest drain but later recurred. There was evidence of procedure related pulmonary haemorrhage demonstrated on CT scan. The patient developed AF which was erroneously managed as SVT. The AF was temporarily cardioverted to NSR with intravenous amiodarone but later relapsed and ultimately terminated with the resolution of pneumothorax. Finally, pulmonary embolus was diagnosed (but later excluded), the diagnosis mimicked clinically by the triad of tachycardia, haemoptysis and sudden onset breathlessness. These are all reminders of important clinical lessons.

Pneumothorax is the commonest complication of PTNB. The reported prevalence is extremely variable between 4%¹³ and 62%.⁵ This is partly due to the fact that the lower estimates reflect the expertise of specialist centres. Most of the studies in the literature have included small sample sizes, reviewing several hundred case note or discharge summaries. These studies tend to be institution specific and therefore fail to provide an accurate representation of universal practice. A recent large scale cross-sectional analysis by Wiener *et al* of 15 865 discharge records in four geographically diverse American states found the

prevalence of pneumothorax to be 15% (CI 14 to 16%), of which 6.6% (CI 6.0 to 7.2%) required chest drain insertion. This is the largest single study to date.¹⁴

The major risk factor for PTNB related pneumothorax is the presence of COPD and more specifically emphysema.¹⁵ Early work by Fish and colleagues suggested a direct correlation between reduced forced expiratory volume in 1 s (FEV₁) and an increased risk of pneumothorax with the need for chest drain insertion.¹⁶ The reported patient had COPD with extremely low FEV₁ of 0.99 l/s (34% predicted) which predisposed him to PTNB related pneumothorax. Small lesion size is also correlated with a higher rate of pneumothorax.¹⁷ It is postulated this is related to an increased number of passes with the biopsy needle before a successful sample is taken.¹⁷ However, this correlation is somewhat controversial as Li and colleagues found no relationship between lesion size and the risk of pneumothorax, using 1.5 cm as a cut off between large and small nodules.¹⁸ Similarly, Yeow *et al* showed that smaller lesion size was linked to an increased rate of pulmonary haemorrhage but not pneumothoraces.¹⁹ The patient presented in this report had a 1.2 cm nodule which is classified as small according to available evidence in the literature which have generally used 1 cm to 1.5 cm as the range for small nodules.^{17–19} Lesions deep to the skin surface leads to higher rates of procedure related pneumothorax, this is due to an increased length of needle trajectory required for biopsy. A multivariable analysis by Rizzo and colleagues suggested a 3% increase in pneumothorax risk with every 1 cm increment of the distance between lesion and skin surface.²⁰ This can be explained by excessive pleural damage due to increased needle trajectory.²⁰ In summary, the reported patient had a history of severe COPD with a small lesion deep to the skin, all of which predisposed him to a procedure related pneumothorax.

Figure 4 A chest drain was inserted which successfully re-inflated the left lung. Pulmonary haemorrhage was clearly apparent.

Pulmonary haemorrhage is the second commonest PTNB related complication, which occurs in up to 27% of cases.⁴ The risk factors are similar to those of procedure related pneumothorax. The presence of COPD,¹⁴ small lesion size¹⁹ and long needle trajectory²⁰ all contribute to increased likelihood of pulmonary haemorrhage in the patient reported. Additional risk factors include deranged coagulation parameters and the presence of pulmonary hypertension,⁴ neither of which applied in this case. Haemothorax is an extremely rare complication of PTNB which was unlikely to apply to this patient since no blood was aspirated from the chest drain.³ Air embolism is another rare complication⁶ and in this case is unlikely in the face of a proven recurrence of pneumothorax on chest radiograph (figure 5). The patient did not have symptoms of coughing which would predispose him to air embolism.

The pathogenesis of AF is complex and remains unclear. AF initiation is related to atrial fibrosis and dilatation, which causes heterogeneous atrial conduction, leading to reduced refractory periods.¹⁰ Two major theories exist regarding the perpetuation of AF; the first is known as automatic focus theory, which proposes discrete areas of automatic impulse firing as the source of continuing re-entry.¹⁰ These areas are often localised to the pulmonary vein-left atrial junctions on electrophysiological studies. The second theory involves propagation of multiple-wavelets in random directions undergoing constant reflections, coalescence and cancellations as they collide. The presence of these wavefronts depends on increased atrial mass, reduced conduction velocities and shortened atrial refractory periods.¹⁰ As long as a threshold of wavelets are present at any given time, AF is maintained.

AF is known to be linked with pulmonary emboli. However, its association with pneumothorax is less recognised. The mechanism is unclear but may be related to an increase in intrathoracic, and therefore intra-atrial pressure due to the pneumothorax. Indeed, raised intrathoracic

pressure in obstructive sleep apnoea (OSA) is connected to AF, which demonstrates support for this theory.²¹ In OSA, there is also repetitive episodes of oxygen desaturation and unstable autonomic tone, both of which are present in persistent pneumothorax.²¹ Moreover, the successful management of these abnormalities has been effective in terminating the associated AF in OSA.²¹ Similarly, there was termination of the AF with the successful management of the pneumothorax in our case. Increased intrathoracic pressure may also exert stress on the pulmonary vein, in which case automatic foci may be considered responsible for the perpetuation of AF. However, such theory is best applied to persistent or permanent AF, where long-term structural remodelling is more likely to be present. While all of these explanations are valid, there are limited data in the literature to provide further support for these theories. AF is also known to be associated with intrathoracic masses and cancer, with resolution following successful curative surgery.²² However, in most cases, the tumours exerted direct pressure effects on the atria, leading to arrhythmogenesis. In our case, the lung nodule was too small to compress the atria, however, the tissue diagnosis of squamous cell carcinoma and the presence of systemic metastases raise the suspicion that the AF induction may also be a paraneoplastic phenomenon.

The erroneous diagnosis and initial management of AF as SVT is certainly a lesson to be learnt. AF is predominantly atrial tissue dependent while SVT also involves the AVN. Vagal manoeuvres and adenosine exert their effects by slowing AVN conduction and exposing p-waves on ECG.¹⁰ While effective in SVT management, these interventions are unlikely to terminate AF. The updated ACC Foundation/AHA Task Force guidelines in 2011 recommends rate control initially with β -blockers, non-dihydropyridine calcium channel blockers, that is, verapamil or digoxin.¹⁰ In the case of persistent symptomatic AF despite rate control, there is a preference

Figure 5 A repeat chest radiograph demonstrated a pneumothorax with a rim of air >4 cm from the chest wall. The red arrow heads indicate the edges of the collapsed lung.

towards restoration of NSR. Amiodarone, flecainide, propafenone are all valid choices.¹⁰ Flecainide is contraindicated in patients with structural heart disease or previous myocardial infarctions due to its arrhythmogenic properties in such individuals. Rhythm control is sometimes preferred in young patients and in cases of hypotension or AF-induced cardiomyopathy and heart failure.¹⁰ Pharmacological management is most effective if initiated within the first 7 days.¹⁰ In our patient, rate control was not carried out due to the consideration of SVT early on. Therefore, rhythm control with amiodarone was the first targeted management of AF. The patient converted to NSR initially however relapsed back into AF as the underlying aetiology (namely the pneumothorax) had not been managed. Complete termination of the AF was observed with successful management of the pneumothorax, which suggests a probable relationship.

Direct current cardioversion (DCC) restores NSR with higher success rates compared to pharmacological management.¹⁰ Immediate DCC is preferred to drug management if there is concurrent myocardial ischaemia, heart failure, symptomatic hypotension, angina or haemodynamic instability.¹⁰ Despite respiratory compromise, our patient did not fulfil these indications. Furthermore, amiodarone was successful in rhythm control therefore electrical cardioversion was not required initially. DCC was later considered once the patient reverted back into AF despite the initial amiodarone infusion. However, the discovery of the pneumothorax necessitated chest drain insertion which ultimately led to the resolution of AF. Had the AF persisted, DCC would have been again considered.

Finally, stroke prevention is recommended in patients with AF by the American College of Cardiology Foundation/American Heart Association Task Force and the CHADS₂ score is used (congestive heart failure, hypertension, age >75, diabetes and stroke = 2 points).¹⁰ Our patient had a CHADS₂ score of 0 with spontaneous resolution of AF. Hence no anticoagulation was recommended and would have been problematic in light of the haemoptysis.

In conclusion, our patient developed a recurrent pneumothorax and AF post PTNB. Both complications were resistant to initial management. This case highlights the challenges faced by clinicians in the postprocedure period and a high index of suspicion is needed for complications with close monitoring required in the hours postprocedure.

Learning points

- ▶ CT guided percutaneous transthoracic needle lung biopsy is a useful method of obtaining histological diagnosis of pulmonary nodules.
- ▶ Pneumothorax and pulmonary haemorrhage are two major complications of percutaneous needle lung biopsies.
- ▶ AF may be associated with postprocedural pneumothorax, which may be difficult to manage.
- ▶ A high index of suspicion for complications is needed in the postprocedure period and close monitoring is mandated in the hours following the procedure.

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Competing interests None.

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