

Delayed pneumothorax after CT-guided percutaneous fine needle aspiration lung biopsy

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Abstract

Two patients are described who developed pneumothoraces more than 24 hours after computed tomography (CT) guided percutaneous fine needle aspiration lung biopsies. The pneumothoraces required treatment in both cases. Such delayed pneumothorax after lung biopsy is extremely unusual. Patients should be warned of the possible occurrence of this complication and instructed to seek medical help if they develop chest pain or breathlessness.

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Keywords: complications, lung biopsy, pneumothorax.

Percutaneous fine needle aspiration biopsy is a well established method for diagnosing lung lesions.¹⁻³ Although generally a safe and well tolerated procedure, pneumothorax is a relatively common and potentially serious complication.^{4,5} We describe two patients who developed significant pneumothoraces more than 24 hours after percutaneous fine needle aspiration lung biopsy.

Case reports

CASE 1

A 56 year old woman presented with a one month history of right sided Jacksonian-type seizures. She was a life long smoker with no past medical history of note. Physical examination was unremarkable. A computed tomographic (CT) scan of the brain revealed a single enhancing lesion in the left hemisphere with surrounding oedema in keeping with a metastasis. A chest radiograph showed a small mass in the left upper lobe. A chest CT scan confirmed the presence of a 2 cm mass in the left upper lobe adjacent to the aortic arch which was thought most likely to represent a primary bronchial carcinoma. A fine needle (22 gauge) aspiration biopsy specimen of the left upper lobe mass was taken percutaneously under CT guidance and revealed adenocarcinoma cells. No pneumothorax was seen on the CT scan performed immediately after the procedure, nor on chest radiographs taken at one and four hours after the procedure. The patient was discharged home the same day, but re-presented 26 hours after the lung biopsy with a

sudden onset of left chest pain associated with minor exertional dyspnoea. A chest radiograph at this time showed a moderate left pneumothorax which was aspirated with good effect. Over the subsequent months she received symptomatic benefit from cranial radiotherapy, dexamethasone, and anticonvulsants but progressively deteriorated and died 10 months after the start of her illness.

CASE 2

A 68 year old male smoker presented with a one week history of intermittent claudication of his right leg. A chest radiograph performed as part of his routine assessment showed enlarged right paratracheal nodes and a 3 cm lobulated mass in the right upper lobe. A chest CT scan confirmed the presence of a mass within the anterior segment of the right upper lobe and large volume mediastinal and right hilar lymphadenopathy. Bronchoscopic examination was normal and cytological examination of bronchial brushings and washings did not show malignant cells. CT guided percutaneous fine needle (22 gauge) aspiration biopsy of the mass revealed small cell lung carcinoma. A CT scan taken after the procedure did not show a pneumothorax, neither did chest radiographs taken at one and four hours after the biopsy. He was discharged home the same day. Approximately 36 hours after the biopsy he became acutely breathless but did not seek medical attention. His dyspnoea improved over the next 48 hours but then worsened and he was re-admitted four days after the biopsy. A chest radiograph showed a right pneumothorax which was successfully managed by intercostal chest drain insertion. He is currently well and undergoing chemotherapy.

Discussion

Radiologically guided percutaneous fine needle aspiration biopsy is a well established technique in the diagnosis of lung lesions. Sensitivities in the detection of malignancy in excess of 90% are repeatedly obtained.^{2,3} It is routinely performed as an outpatient procedure.⁵ Pneumothorax is the most common and potentially serious complication. Rates of pneumothorax of about 25% are common^{6,7} although most do not require treatment. In a study of 673 patients in whom transthoracic fine needle aspiration biopsy was performed Perlmutter and colleagues⁶ did not have a single case of pneumothorax occurring more than four hours after the procedure. They recommended that a chest radiograph should be taken at one and four hours after such biopsies in outpatients.

In our institution CT guidance is preferred over fluoroscopic guidance in percutaneous transthoracic needle biopsy for those lesions which are poorly seen or considered inaccessible at fluoroscopy and for lesions adjacent to major cardiovascular structures, either hilar or mediastinal. The pneumothorax rate appears to be higher with CT guidance than that commonly reported with fluoroscopy.⁸ This is likely to be due to the increased time

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that the biopsy needle remains in the lung, although the increased sensitivity of CT scanning compared with chest radiography in the detection of pneumothorax must be contributory. The two patients we describe both had CT scans performed at the level at which the needle crossed the pleural surface immediately after the biopsy. In neither of these was a pneumothorax apparent. Their delayed pneumothoraces were not therefore due to enlargement of an initial pneumothorax not visible on chest radiographs taken one and four hours after the procedure. Although neither of our two patients gave a history of sudden exertion or coughing prior to the onset of symptoms, it may be that their delayed pneumothoraces were secondary to displacement of small pleural blood clots following the biopsy procedure.

Delayed pneumothorax (of more than 24 hours) has been reported as a complication of transbronchial lung biopsy⁹ and also of subclavian vein catheterisation.¹⁰ The occurrence of pneumothorax in two patients more than 24 hours after the procedure emphasises the importance of considering this diagnosis even

at a delayed stage. Every patient who undergoes this procedure should be warned of the importance of seeking medical attention should they develop increased breathlessness or chest pain after discharge.

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Neuromuscular blockade with acute respiratory failure in a patient receiving cibenzoline

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can be responsible for neuromuscular blockade, and should therefore be used with caution in patients with neuromuscular and respiratory diseases or with impaired renal function.

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Keywords: cibenzoline, neuromuscular blockade, acute respiratory failure, diaphragm paralysis, phrenic nerve stimulation.

A host of drugs can interfere with the contraction of respiratory muscle at several levels¹ including impairment of neuromuscular transmission. The latter can result in life threatening episodes of respiratory failure,² particularly in patients with pre-existing myasthenia gravis and other neuromuscular diseases, and in patients with a reduced respiratory reserve or a decreased ability to eliminate drugs.^{1,2}

We report a case of acute respiratory failure with electrophysiological evidence of neuromuscular blockade involving the diaphragm in a patient with chronic renal failure receiving cibenzoline, a class Ic antiarrhythmic agent.³ This case appears to be the first report of cibenzoline-induced neuromuscular blockade. The initial report of cibenzoline-induced hypoglycaemia⁴ mentions the association of bradycardia, central nervous system depression, and respiratory distress but no details are given.

Case report

CLINICAL HISTORY

The patient was a 75 year old man with end stage chronic renal failure from polycystic renal disease, undergoing continuous ambulatory peritoneal dialysis (CAPD) and receiving oral

Abstract

Cibenzoline is a class Ic antiarrhythmic agent that can be used to treat supra-ventricular arrhythmias. A case is reported of cibenzoline overdose in a patient with impaired renal function, leading not only to the usual cardiac and metabolic symptoms (bradycardia and hypoglycaemia), but also to a myastheniform syndrome with acute respiratory failure. Neuromuscular blockade was demonstrated by repetitive supramaximal stimulation of the median nerve, and diaphragmatic involvement was evidenced by applying the same protocol to the phrenic nerve. Muscle strength recovered as serum cibenzoline levels decreased, allowing the patient to be weaned from the ventilator. This observation suggests that cibenzoline, like other antiarrhythmic agents,