CASE REPORT

Lung adenocarcinoma masquerading as refractory *Klebsiella pneumoniae*

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SUMMARY

We report the case of a middle-aged man where a diagnosis of *Klebsiella pneumoniae* obscured the underlying malignancy. The patient was hospitalised for management of a presumed refractory community-acquired pneumonia with radiological features of right lower lobe consolidation. Bronchoscopy did not identify an endobronchial lesion and washings grew *K pneumoniae*. CT-guided fine-needle aspirate samples did not detect any malignancy. However, despite appropriate antibiotic treatment there was no improvement in the patient's clinical condition. Consequently, a CT-guided lung core biopsy was performed to obtain more tissue for histopathology, which was diagnostic of primary lung adenocarcinoma. This case highlights the need to continue to investigate a patient who is not progressing as clinically appropriate to their original diagnosis.

BACKGROUND

Community acquired pneumonia (CAP) is readily recognisable by the symptoms of cough, chest pain and sputum production in conjunction with radiological findings of pulmonary infiltrates and/or consolidation. However, there are many non-infectious mimics of CAP.¹ Some of the common non-malignancy mimics of CAP include systemic lupus erythematosus pneumonitis, sarcoidosis, recurrent aspiration pneumonia, alveolar haemorrhage and bronchiolitis obliterans with organising pneumonia.¹ The malignancy mimics of CAP can be due to primary bronchogenic adenocarcinoma, lymphoma or postobstructive pneumonia.¹

Our report illustrates that in patients with CAP who fail to respond to appropriate antibiotic therapy, it is important to consider an underlying malignancy and obtain an adequate tissue biopsy and pathological examination.

CASE PRESENTATION

Our patient was a 55-year-old man who was referred by his family doctor to our institution for evaluation and management of a 3-month history of chronic productive cough with clear sputum, febrile illness and a 1 kg weight loss. The patient had completed two courses of oral antibiotics with no noticeable improvement in symptoms. The patient denied any history of haemoptysis, night sweats, chest pain or dyspnoea. There was no history of recent overseas travel or exposure to sick contacts.

The patient was of East Asian descent and had migrated to Australia over 10 years ago from a South-East Asian country. He was previously

healthy with no comorbidities. He denied taking any regular medications and was not aware of any allergies. Prior to hospital presentation he had been working as a mushroom farmer and lived rurally with his wife and two children. There was no significant family history of any medical illness. He identified himself as a never-smoker.

INVESTIGATIONS

At the time of initial presentation, the CT of the chest demonstrated a dense right lower lobe pulmonary opacity (figure 1). Blood tests revealed mild leucocytosis 11.5×10^9 (4–11) and an elevated C reactive protein (CRP) 45 mg/L (<5). Hepatitis B surface (HBs) antigen, hepatitis C antibody and HIV antigen/total antibody tests were negative. Immunoglobulin levels (IgG and IgE) were within normal limits. Sputum analysis identified 3+ growth of *Klebsiella pneumoniae*. Antibiotic sensitivity testing demonstrated sensitivity to amoxicillin/clavulanate, cefazolin, gentamicin and co-trimoxazole. The patient underwent a diagnostic bronchoscopy, which did not identify an endobronchial lesion. Bronchial washings from the right lower lobe grew *K pneumoniae*. In order to exclude an underlying malignancy, a CT-guided fine-needle aspiration was performed on the right lower lobe mass, which contained mixed inflammatory cells consistent with reactive change and no malignant cells were detected.

DIFFERENTIAL DIAGNOSIS

Working diagnosis: *K pneumoniae*.

Differential diagnosis: Bronchogenic carcinoma; cryptogenic organising pneumonia.

Figure 1 Diagnostic chest imaging performed in the patient. Representative slice of CT scan of the thorax showing a right lower lobe consolidation.

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TREATMENT

The patient was treated with amoxicillin with clavulanic acid for 15 days and reviewed in clinic. During follow-up, the patient continued to have intermittent febrile episodes, worsening dyspnoea, reduced exercise tolerance, fevers and loss of weight. A repeat chest X-ray showed worsening right lower lobe changes and patchy changes in the left lower lobe.

Serial sputum cultures grew *K pneumoniae* sensitive to amoxicillin. Despite three courses of antibiotics, the patient had not improved and continued to deteriorate clinically.

Infectious diseases were consulted who recommended a 4-week treatment of bactrim 800 mg/160 mg twice daily for treatment of his *K pneumoniae*, with a further plan of intravenous antibiotics and consideration of a lobectomy if improvement was not seen.

OUTCOME AND FOLLOW-UP

Since the patient was not responding appropriately to antibiotics, a decision was made to repeat the lung biopsy with the intention of obtaining more tissue for histological examination. A CT-guided core biopsy was performed of the right lower lobe mass. Histopathology of the biopsy sample showed a well-differentiated adenocarcinoma (figure 2) with positive staining for thyroid transcription factor 1 (TTF-1) suggesting that the tumour was a lung primary. Molecular testing was performed on the tumour sample to check for the presence of an epidermal growth factor receptor (*EGFR*) mutation, which was surprisingly negative. The patient was referred to medical oncology and commenced on platinum-based doublet chemotherapy. After receiving two cycles of chemotherapy, it was noted that the patient was developing disease progression and was subsequently started on gefitinib, an *EGFR* tyrosine kinase inhibitor as second-line therapy. However, he continued to deteriorate and eventually died of his disease 9 months after his initial presentation.

DISCUSSION

Many pulmonary and systemic medical disorders can mimic the clinical and radiological pattern of CAP. Primary bronchogenic adenocarcinoma is a well-recognised mimic of bacterial CAP.² This is because adenocarcinoma grows along the septa of the lung and does not distort the pulmonary architecture or cause endotracheal obstruction.¹ Radiologically, this can present as lobar infiltrates and/or consolidation, making it difficult to distinguish from CAP. Indeed, there have been a few reports in the literature of patients with primary lung adenocarcinoma who

Figure 2 A microscopic section of lung core biopsy which is a H&E section with $\times 20$ magnification demonstrating cells consistent with adenocarcinoma originating from a lung primary, with lining columnar cells featuring some mucinous change and nuclear atypia.

were initially diagnosed to have CAP.^{1–3} The reports describe patients with apparently non-resolving CAP, despite being on appropriate antibiotics and a diagnosis of malignancy was established on histological examination of lung biopsy.

It is now recognised that bacterial colonisation of the bronchial airways is common in patients with lung cancer. Laroumagne and coauthors recently reported the results of their prospective study which evaluated the prevalence and nature of bronchial colonisation in patients at the time of diagnosing lung cancer.⁴ They found that potential bacterial pathogens were found in 48.1% of samples and most of the pathogens were Gram-negative bacilli. The study also reported that bacterial colonisation in patients with lung cancer was associated with worse survival. Interestingly, there have been recent reports of an association between *Klebsiella* pyogenic liver abscess and occult colorectal adenocarcinoma.⁵ Whether such a relationship also exists between *K pneumoniae* and lung adenocarcinoma requires further research.

In our patient, the diagnosis of lung adenocarcinoma was potentially delayed because at the time of initial presentation he underwent a CT-guided fine-needle aspirate. The diagnosis of malignancy was established when a core biopsy of the suspicious right lower lobe mass was performed. This is not surprising since core biopsy needles can obtain more tissue sample for histological evaluation, thus reducing the chance of the needle missing the site of pulmonary pathology. Indeed, Anderson *et al* report in a study comparing the diagnostic utility of CT-guided fine-needle aspiration versus core biopsy for pulmonary lesions, the yield of core biopsy was significantly higher compared to fine-needle aspiration (93% vs 78%, $p < 0.005$).⁶

Our patient's clinical presentation was unusual because without bronchial obstruction, bronchogenic adenocarcinomas are not accompanied by febrile episodes. It is quite likely that our patient had pulmonary infection superimposed on the underlying primary bronchogenic adenocarcinoma. Another surprising finding was that our patient's tumour was *EGFR* mutation negative. He had many of the clinical phenotype features associated with *EGFR* mutation positive lung adenocarcinomas (Asian and never-smoker). Several studies have now demonstrated that in the overwhelming majority of primary lung tumours in this cohort of patients are *EGFR* mutation positive.⁷

Learning points

- ▶ In patients with clinical and features suggestive of community-acquired pneumonia, empirical treatment of antibiotics should be followed by clinical assessment to ensure complete resolution.
- ▶ Primary lung adenocarcinoma can mimic non-resolving community acquired pneumonia.
- ▶ The identification of gram negative bacteria in biological specimens does not necessarily exclude the diagnosis of malignancy as bacteria frequently colonise the bronchial airways in patients with lung cancer.
- ▶ In clinical situations of non-resolving consolidation, a targeted core biopsy may be preferred to a fine-needle aspirate as a means of ruling in malignancy and ruling out other pathologies.

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