CASE REPORT

Uncommon condition mimicking non-resolving pneumonia and malignancy on radiography that improves dramatically with a course of steroids

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SUMMARY

A 58-year-old man was seen in a specialist respiratory clinic, with non-resolving pneumonia. Various investigations including CT scan, bronchoscopy and CT-guided biopsy were carried out for suspected malignancy but they could not find any evidence of neoplasia or anything else of significance. The lung mass that persisted for years later resolved with a course of steroids, at which point further investigations for alternative differential diagnoses suggested a surprising conclusion.

BACKGROUND

Non-resolving pneumonia, defined as clinical and radiological evidence of pneumonia that fails to improve after adequate treatment, raises the alarm of a possible malignancy or tuberculosis,¹ and often necessitates harmful and invasive procedures ranging from CT scanning to bronchoscopy to CT-guided biopsy. In a proportion of people, these investigations are unnecessary because there are alternative diagnoses that can be made in specific situations if they are actively suspected.

CASE PRESENTATION

A 58-year-old man was seen in a specialist respiratory clinic, with signs of a non-resolving pneumonia and persistent shadowing in the right mid zone on chest radiographs. He was an ex-smoker and drank alcohol occasionally. Up to this point, he had been on salbutamol and beclometasone inhalers. He denied any dyspnoea, weight loss or haemoptysis. On examination, he looked well with no pallor, jaundice, cyanosis, clubbing or peripheral lymphadenopathy. His chest was clear with no crackles or wheeze. The rest of his examination was unremarkable. Malignancy and tuberculosis needed to be ruled out, so a CT of the thorax was organised, which confirmed a mass followed by a bronchoscopy, which showed no endobronchial lesion, and bronchial washings for acid-fast bacilli (AFB) were negative. A multidisciplinary team (MDT) decision was made to perform a right middle lobectomy—the histology from excised tissue showed only granulomatous inflammation.

The patient continued to have recurrent chest symptoms following surgery, which consisted of cough productive of significant sputum that was green on many occasions, and this raised the suspicion of bronchiectasis. High-resolution CT of the thorax showed bronchial wall thickening in the left

lower zone suggestive of bronchiectasis. At the same time, blood tests were carried out, the results of which were: total IgE 1242 IU/mL (normal range 0–100) and total immunoglobulins were normal. The family reported that when the patient had had chest infections and had taken steroids, he had responded promptly. During a further episode of worsening chest symptoms that included shortness of breath and cough productive of green sputum, the patient was sent for a chest radiograph, which showed left lower zone consolidation (reported as left lingular consolidation). He was placed on a course of steroids and antibiotics, and in follow-up clinic, he felt a lot better and had not had any exacerbation of chest symptoms for months, other than coughing up a small regular amount of phlegm, which would be expected with bronchiectasis. Repeat chest radiography showed complete resolution of the consolidation.

The patient remained well for over a year, with no significant chest symptoms, but then presented with haemoptysis on a daily basis for a period of a week, where sputum was mixed with blood. Urgent chest radiography was performed (figure 1) followed by CT of the thorax (figure 2), which were reported by the radiologist as ‘right upper lobe mass consistent with a lung neoplasm’. The MDT reviewed the images and this time decided to take a watchful waiting approach owing to negative bronchoscopy and histology from

Figure 1 Chest radiograph showing right upper zone opacification.

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Figure 2 CT of the thorax.

previous surgery reporting no malignancy. Follow-up CT scans at 3 months, 1 year and 18 months, showed a persistent right upper lobe lung lesion that was getting slightly larger each time. At this point, the situation was slowly becoming worrisome and the MDT meeting pressed for a CT-guided biopsy, which again did not make the diagnosis any clearer, only demonstrating fibrous tissue with normal lung parenchyma and no evidence of malignancy. The possible differential diagnoses with granulomatous inflammation on histology that should be considered are sarcoidosis and tuberculosis as well as more unusual infections including fungal infections and histoplasmosis. Therefore, the following tests were ordered with their respective results:

INVESTIGATIONS

Total IgE: 3971 (normal range 0–100); aspergillosis precipitins: 156 mg/L (normal range 0–10); and *Aspergillus* radioallergosorbent (RAST) test: positive.

DIFFERENTIAL DIAGNOSIS

A diagnosis of allergic bronchopulmonary aspergillosis (ABPA) was made as the only reasonable option despite it being very uncommon to present with a mass.

Others:

- ▶ Sarcoidosis
- ▶ Tuberculosis
- ▶ Fungal infections
- ▶ Mucus impaction

TREATMENT

The patient was started on a course of steroids for 3 months. Prednisolone was started at 30 mg once daily for 2 weeks, then 25 mg once daily for 1 week, then continued on at 20 mg OD until further review in clinic. The aim was to give the patient prednisolone at a dose of 20 mg and above for a minimum of 12 weeks before tapering it further.

OUTCOME AND FOLLOW-UP

Repeat bloods in 6 weeks showed halving of the total IgE to 1743 and resolution of the right upper lobe lesion on chest radiography (figure 3).

DISCUSSION

The main clue in the history was that the opacification in the right upper lobe improved after a course of steroids. This implies it is likely our patient had a steroid responsive condition such as of inflammatory/autoimmune aetiology. The rate of improvement over 6 weeks and the dramatic resolution of radiological signs suggest it was not malignancy. The histology from lobectomy showed granulomatous inflammation. Bronchoscopy washings were negative for AFB, which would have suggested tuberculosis. This left sarcoidosis as the only common differential other than ABPA. This patient, however, did not show any other signs of sarcoidosis in the lungs nor any extrapulmonary involvement, which is very unusual for sarcoidosis.

Another clue was the total IgE level that was raised early on in this patient's history and then plummeted in the past 6 weeks after steroids were started, as depicted in the graph (figure 4). Although there is currently no consensus on the criteria for diagnosis of ABPA, it is widely agreed that the total IgE count forms one of the major criteria. The Rosenberg criteria can be easily remembered with the mnemonic ARTEPICS:^{2 3}

A Asthma

R Radiological findings: pulmonary infiltrates

T skin Test reactivity to *Aspergillus fumigatus*

E Eosinophilia

P Precipitating antibodies (IgG) in serum against *Aspergillus*

I total serum IgE

Figure 3 Chest radiographs showing (A) right upper zone mass and (B) 6 weeks after starting steroid treatment.

Figure 4 Graph showing serial total IgE counts in this patient from when he was first tested in 2012; when he had exacerbation with haemoptysis in 2014; and the plummeting of the IgE count in the past 6 weeks after starting steroids.

C Central bronchiectasis

S Serum-specific IgE and IgG to *A. fumigatus*

This patient had no formal diagnosis of asthma, although he had a history of bronchodilator use and his spirometry was normal. He also had no eosinophilia and his *Aspergillus* precipitins were originally negative when he first presented, which misled the team to suspect other diagnoses. This example suggests that the criteria for ruling in ABPA as a differential diagnosis should not be tight, although certain features such as history of asthma together with central bronchiectasis would make ABPA a lot more likely. The reliability of the aforementioned criteria for diagnosis of ABPA are discussed in more detail in the article under the ‘further reading’ heading below.

ABPA is a disease that results from an exaggerated immune hypersensitivity reaction to fungus *Aspergillus* sometimes found in the respiratory tract. The pathogenesis of the disease is via airway inflammation and progression to bronchiectasis in later stages. This is in contrast to the other forms of aspergillosis such as an invasive fungal infection and non-invasive saprophytic colonisation of the airways sometimes leading to a ball of fungus in pre-existing lung cavities known as aspergilloma.⁴ Note that the pulmonary mass in this patient could not have been an aspergilloma as it would have been characterised by obvious fungal spores on biopsy and would have left behind an empty lung cavity on resolution.

ABPA is mainly found in people with predisposing lung conditions such as asthma or cystic fibrosis. It is estimated that 2% of all asthma sufferers and 7–14% of corticosteroid-dependent patients with asthma have ABPA.⁴ It is therefore advisable to screen people with uncontrolled asthma for this condition. Interestingly, this patient was a gardener by profession so he likely had exposure to fungus, and although there does not appear to be a link between environmental *Aspergillus* load and the development of ABPA, significant exposure should still be avoided.⁵

Typical radiological findings are fleeting pulmonary infiltrates (especially during acute exacerbations) and thickened inflamed

bronchi ‘Tram line sign’ as well as areas of transient tubular opacities (known as finger-in-glove pattern) due to mucoid impaction causing collapse. Therefore, a high attenuation mucus sign (intensity on CT similar to paraspinal muscles) is an important clue. Later signs may be those of central bronchiectasis and pulmonary fibrosis.⁴ ABPA presenting with a mass is very uncommon. There have been several case reports of such a presentation. Takeuchi *et al* described a 48-year-old warehouse manager with a 30-year history of bronchial asthma who was found to have a high-density mass on chest radiography. He was also initially suspected to have neoplasia and had undergone invasive investigations, but had later improved on corticosteroids.⁶ Agarwal *et al* reported three cases, two of whom were asthmatics, where a large mass was seen on chest radiographs and investigations supported the diagnosis of ABPA. All responded to corticosteroid treatment with resolution of

Learning points

- ▶ Allergic bronchopulmonary aspergillosis (ABPA) is common in patients with asthma and cystic fibrosis but it may occur without prior comorbidities. It is always important to evaluate the patient for comorbidities, for example, with lung function tests and peak flow, because this may help with the diagnosis.
- ▶ ABPA should be identified early to prevent repetitive infections and progressive bronchial injury.
- ▶ ABPA should be considered if granulomatous inflammation is found on biopsy.
- ▶ ABPA may in rare cases present with a pulmonary mass. Have a high index of suspicion.
- ▶ Simple screening blood tests detailed in the Rosenberg criteria may be applied in the first instance before performing further investigations such as skin tests.

radiological features as well as decrease in the total IgE count, which in fact is an established measure of response to therapy.^{3 4}

These cases suggest various mechanisms to explain the formation of lung masses to include mucus plugging with accumulation of distal secretions, large bronchocoeles (dilated bronchi due to mucus) and inflammatory eosinophilic consolidation appearing as a pseudotumour.³ Among the *BMJ Case Reports*, there were no papers on ABPA presenting as a mass, but only cases of fleeting pulmonary opacities^{7 8} and lung collapse.⁹

Contributors AS reviewed the patient's history, met with the patient and wrote article. MM edited the draft and reviewed the final version. He is also the clinician following this patient in his clinic.

Competing interests None declared.

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