Short reports

Mixed allergic bronchopulmonary fungal disease due to *Pseudallescheria boydii* and *Aspergillus*

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Abstract

A 24 year old asthmatic woman with mixed allergic bronchopulmonary fungal disease due to *Pseudallescheria boydii* and *Aspergillus* is reported. No previous cases due to *P boydii* have been described. This patient provides evidence that fungi other than *Aspergillus* species may cause the condition.

Allergic bronchopulmonary fungal disease is characterised by asthma, eosinophilia, infiltrates on the chest radiograph, raised IgE levels, precipitating antibodies, and immediate skin reactivity to the causal fungus.¹ Its management differs from that of simple asthma and frequently requires prolonged use of corticosteroids to minimise development of proximal bronchiectasis.

Aspergillus species are the most common fungi implicated in allergic bronchopulmonary fungal disease, but more recently other fungi, including *Curvularia*, *Drechslera*,² *Helminthosporium*, *Candida albicans*, *Stemphylium*, *Torulopsis*, and *Bipolaris*^{1,3} have been reported. Mixed fungal infection has been recognised less frequently.²

Pseudallescheria boydii is a common soil fungus that is increasingly reported as a human pathogen,⁴ but has not previously been implicated in allergic bronchopulmonary fungal disease. We report a case due to *P boydii* and *Aspergillus* in an asthmatic patient.

Case report

A 24 year old atopic woman with asthma was admitted to hospital with a four month history of two prolonged episodes of cough producing purulent sputum. Broad spectrum antibiotics were unhelpful. The patient had lived in non-tropical rural areas of Western Australia and

Figure 1 Chest radiograph at admission.

her only medication was inhaled salbutamol and the oral contraceptive pill.

On admission she had a cough producing green-brown sputum with obvious plugs. Her asthma appeared stable, she was afebrile, and she had bronchial breathing over the right upper zone. Chest radiographs four months earlier showed a left upper lobe infiltrate extending from the hilum. Subsequent radiographs showed changing recurrent shadows and when she was admitted there was considerable right hilar shadowing (fig 1). The forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) were 2.2 and 2.9 litres, (predicted 3.0 and 3.6 l). A full blood count showed eosinophilia (1.7×10^9 cells/l; normal $< 0.4 \times 10^9$) and the total IgE was greater than 4000 IU (normal < 300 IU). Skin prick testing was performed with reagents from the Hollister Stier Laboratories, Washington. In addition a freeze dried cytoplasmic extract of *P boydii* was prepared by the method described by Mackenzie *et al*⁵ and reconstituted to 20 mg of freeze dried extract/ml of distilled water for skin prick testing. The patient had skin weal responses as follows: *Aspergillus fumigatus* (6 mm), *A terreus* (7 mm), *A niger* (3 mm), *A nidulans* (3 mm), *Alternaria* sp (8 mm), *Helminthosporium* (9 mm), *Curvularia* sp (12 mm), *Stemphylium botryosum* (10 mm), and *P boydii* (7 mm). The skin reaction to 1.0 mg/ml of histamine was 6 mm. Precipitating antibodies were detected by the double immunodiffusion technique⁵ with cytoplasmic extracts of the fungi, the patient's serum, and a control solution of hyperimmune rabbit anti-serum. Antibodies were detected in the patient's serum on two occasions one month apart. Initial assessment showed the presence of *P boydii* (two bands), *A fumigatus* (three bands), and *A flavus* (one band) and on the

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Figure 2 Typical conidia borne singly at the tips of conidiophores.

second occasion *P boydii* (three bands), *A flavus* (one band), and *A terreus* (one band). *Aspergillus* species did not cross react with *P boydii* in immunodiffusion studies using hyperimmune rabbit antisera.

At bronchoscopy plugs of sputum were aspirated from the right upper lobe. A bronchoalveolar lavage specimen showed a high neutrophil (76%) and eosinophil count (12.5%) (normal: neutrophils < 2%, eosinophils < 0.5%). Four bronchoscopy specimens and one sputum specimen were cultured at 25°C and 37°C on five media. All specimens showed large amounts of septate branching mycelia and grew multiple colonies of *P boydii*, which were floccose, smoky grey on the surface, and blackish on the reverse. The fungus grew initially in the anamorph state, *Scedosporium apiospermum* (fig 2), but was later induced to produce ascomas on water agar. Occasional colonies of *A terreus* were also grown.

The patient was treated with prednisolone 40 mg per day for six weeks, which was decreased over a further four weeks and then stopped. Salbutamol was continued and theophylline added. The symptoms improved rapidly and four weeks later a chest radiograph showed almost complete clearance of the infiltrates. Results of ventilatory capacity tests also improved (FEV₁ and FVC being 2.8 and 3.5 l).

Discussion

Pseudallescheria boydii belongs to the class Ascomycetes. It has a worldwide distribution, particularly in soil, where it is a natural saprophyte. It has also been isolated from decaying

vegetation, poultry and cattle manure, and water.⁴ In addition to causing maduromycosis (madura foot) it has increasingly been reported as a pathogen at other sites⁴ and in disseminated infection.⁶

Disease due to *P boydii* has been infrequently reported and this may reflect problems with its identification. *P boydii* resembles other fungal species in tissue specimens and may be difficult to distinguish from *Aspergillus* species. Cultures definitely identifying the fungus are thus required for a diagnosis. Serological antibody testing and skinprick testing may also be helpful, particularly if allergic disease is suspected.

The lung is the most common extracutaneous site of infection, 31 cases of pulmonary infection having been reported.⁴⁻¹⁰ Manifestations of infection have included asymptomatic coin lesions, lung mycetoma, necrotising pneumonia with associated bronchiectasis or abscess formation, transient colonisation, and an infected pleural effusion (one report). Of these, mycetoma was the most common. Of the patients reported, only five neither had underlying lung disease nor were immunosuppressed.⁴⁻¹⁰ No cases of allergic bronchopulmonary fungal disease due to *P boydii* have been reported.

Aspergillus species were the initial fungi to be recognised as causing allergic bronchopulmonary fungal disease when the condition was first described in 1952 and remain the most common fungi implicated in the disease. Mixed allergic bronchopulmonary fungal disease has been described² but seems uncommon. Our patient had six major and two minor criteria for the diagnosis of allergic bronchopulmonary

fungal disease, on the basis of Greenberger's criteria for allergic bronchopulmonary aspergillosis.¹ The strongly positive precipitin bands and skin test responses to *P boydii* and the heavy growth of the fungus confirm its likely pathogenicity in this patient. Additionally, the growth of *Aspergillus terreus*, with a positive skin test response and precipitins, is consistent with mixed allergic bronchopulmonary fungal disease.

Our patient also had positive skin test reactions and precipitins in response to other fungi, a finding common in asthmatic patients, individuals who do not have allergic bronchopulmonary fungal disease. The clinical significance of these reactions is not clear and their presence may in part be due to cross reactivity between common fungi. Cross reactivity between *Aspergillus* species exists, though in this patient cross reactivity of *P boydii* with *Aspergillus* seems unlikely, as shown by immunodiffusion tests using rabbit antisera.

This patient's condition supports the concept of "allergic bronchopulmonary fungal disease" as opposed to "allergic bronchopulmonary aspergillosis", as an increasingly wide range of fungi are being shown to cause a syndrome identical to that commonly

associated with *Aspergillus* species. In suspected allergic bronchopulmonary fungal disease where all tests for *Aspergillus* give negative results the possibility of other fungi, such as *P boydii*, as causative agents should be investigated.

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Mediastinitis and bilateral thoracic empyemas complicating adult epiglottitis

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Abstract

A 32 year old woman developed life threatening mediastinitis and bilateral empyemas as a complication of adult epiglottitis. She recovered completely.

Mediastinitis following deep neck infections was well recognised in the preantibiotic era. In Pearses's series of 110 cases the main cause was perforation of the oesophagus.¹ Twenty three cases resulted from abscesses in the oropharynx. Mediastinitis complicating oropharyngeal infections is now uncommon.

Acute epiglottitis in adults is an uncommon but important condition.²⁻⁴ This is the first reported case of acute epiglottitis complicated by mediastinitis and thoracic empyema.

Case history

A previously fit 32 year old woman presented with a few hours' history of sore throat and dysphagia followed by progressive dyspnoea. On examination she had a temperature of 37.6°C, inspiratory stridor, a muffled voice, drooling saliva, and a tender neck. A lateral

neck radiograph showed a swollen epiglottitis, but the chest radiograph was normal. Flexible laryngoscopy showed swelling of supraglottic structures. The leucocyte count was $22.4 \times 10^9/l$ with neutrophilia. Blood cultures were sterile. Serological examination of paired specimens showed no appreciable rise in the titre of antibodies to viruses or atypical bacteria. She was treated with intravenous chloramphenicol and hydrocortisone followed by dexamethasone. She gradually improved and was afebrile by the fifth day.

On the sixth day she developed a constant left anterior chest pain, increasing breathlessness, and haemoptysis. A pleural rub was present at the left base. The chest radiograph showed a small left pleural effusion. She was given intravenous heparin for a presumed pulmonary embolus but her condition deteriorated. Subsequent radiographs showed widening of the mediastinum, an increasing left pleural effusion, and a small left sided pneumothorax. On the eighth day one litre of foul smelling pus was aspirated from the left hemithorax. This grew mixed anaerobes and *Streptococcus milleri*. Anticoagulation was discontinued and she was treated with benzylpenicillin, gentamicin, and metronidazole. Computed tomography showed fluid and gas in the anterior and posterior mediastinum and confirmed the presence of bilateral pleural effusions. No oesophageal leak was detected from a gastrografin swallow. Intercostal chest drains were introduced into both pleural cavities. Transcervical exploration and drainage of the mediastinum was performed. After these procedures mechanical ventilation was required for three days and was complicated by paroxysmal fast atrial fibrillation.

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