Suspected mushroom worker's lung – a case report from South Africa

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Mushroom worker's lung (MWL) has been described in workers exposed to mushroom spores or to inhaled antigens (thermophilic actinomycetes) from compost. South Africa is listed as one of the biggest exporters of mushrooms worldwide, yet the incidence of MWL locally has not been documented. We highlight a case of a delayed presentation of suspected MWL owing to non-recognition of the condition and the subsequent improvement in symptoms upon avoidance of the offending antigen. A high index of suspicion is required to ensure that MWL, associated with significant morbidity, is not missed.

Occupational hypersensitivity pneumonitis (OHP) is a non-IgE-mediated allergic reaction to a host of organic materials or low-molecular-weight agents inhaled at the workplace. There are more than 200 antigens known to cause this condition, which can be categorised into bacteria, fungi, enzymes and animal/insect proteins, plant proteins, low-molecular-weight chemicals or metals.[1] Patients presenting with respiratory symptoms are commonly questioned about a farming history or exposure to birds to assess occupational or other allergenic exposure, but exposure to other agents, such as mushrooms, is often not enquired about.

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Mushrooms propagate by disseminating millions of microscopic spores that form in the gills or underneath the mushroom's cap. These spores are either spread by the wind or by animal feeding.[2] Case reports have been published attributing the aetiology of MWL more commonly to the inhalation of spores of the shiitake mushroom (*Lentinus edodes*),[3] bunashimeji (*Hypsizigus marmoreus*),[4] *Pholiota nameko* mushrooms[5] and oyster mushrooms (*Pleurotus osteatus*).[6]

Compared with oyster mushrooms and shiitake mushrooms, button mushrooms (*Agaricus bisporus*) are less likely to release spores as harvesting occurs before the caps open, a process that is associated with spore release.[6,7]

Mushrooms are produced commercially in Europe, the British Isles, the Far East, North America, Australia and South Africa (SA). In 2009, SA was listed as one of the top eleven mushroom exporters worldwide,[8] and yet, to our knowledge, this is the first case report describing MWL in SA. We present a case of an unusually late presentation of MWL owing to a diagnostic delay of several years prior to the patient's referral.

Case report

A 29-year-old man, who had been employed at a local mushroom farm since 2008, was referred to our pulmonology unit with a history of progressive dyspnoea and a chronic productive cough for the past 3 years. His duties at work included planting and harvesting button mushrooms within an enclosed area, as well as making and packing mushroom compost. Although protective respiratory equipment was available to him, he did not use this consistently. Prior to his referral, the management of his symptoms had included the prescription of multiple courses of antibiotics, empirical treatment for tuberculosis (TB) in 2014, and commencement of inhaler therapy for asthma with intermittent salbutamol via a metered dose inhaler (MDI), as well as fluticasone/salmeterol via an MDI twice daily. None of these interventions had alleviated his symptoms of dyspnoea or cough. He also complained of weight loss over the 3 years but was unable to quantify his total weight loss. There was no history of fever or night sweats. He was a non-smoker and had tested negative for HIV previously.

Clinically, he had clubbing of his fingers and toes, and was plethoric. He appeared comfortable with a respiratory rate of 18 breaths per minute, although his oxygen saturation measured by pulse oximetry was 88% on room air. His lung fields were clear and the pulmonary component of the second heart sound was accentuated. He had no features of decompensated cardiac failure. His haemoglobin (Hb) on admission was 19 g/dL with a haematocrit of 59% and a red cell count of 6.3 x 10^9/L, correcting to an Hb of 15.3 g/dL after venesection. He had a normal white cell count of 4.43 × 10^9/L, normal eosinophil count, and normal inflammatory markers with a procalcitonin value of <0.5 μg/L. Serum precipitins to *Aspergillus fumigatus* and other thermophilic actinomycetes were not available at our facility.

His chest radiograph showed a normal cardiothoracic ratio with increased reticular shadowing bilaterally, ill-defined air space opacification of both lung fields, and a prominent pulmonary conus (Fig. 1). Pulmonary function testing showed an obstructive picture with a forced vital capacity (FVC) of 3.1 L (65% of the predicted value) and a forced expiratory volume in 1 second (FEV_1) of 1.58 L (39% of the predicted value), with an FEV_1/FVC of 50.9%. Reversibility of 240 mL and 15% was noted post bronchodilator use. The patient's

Fig. 1. Admission chest radiograph showing increased reticular shadowing and bilateral ill-defined opacities.

diffusing capacity for carbon monoxide was reduced at 37% of the predicted value when corrected for alveolar volume. Whole-body plethysmography showed a residual volume of 203% with a total lung capacity (TLC) of 104% predicted, which was suggestive of air trapping.

A high-resolution computed tomography (HRCT) scan of his chest showed bilateral upper-lobe fibrotic changes, diffuse ground-glass opacities bilaterally with mosaic attenuation in keeping with air trapping. There were bilateral upper-lobe, subpleural, subcentimetre, perilymphatic, non-calcified nodules as well as interlobular septal thickening and intralobular interstitial thickening noted diffusely (Fig. 2). An echocardiogram was ordered which showed a dilated right atrium (47 mm) and right ventricle (48 mm) with an elevated pulmonary artery systolic pressure of 45 mmHg. His ejection fraction was also moderately impaired at 45%.

He underwent an uneventful flexible bronchoscopy with transbronchial biopsy. Both the sputum and bronchoalveolar lavage (BAL) specimens were negative for infective pathogens. Differential cell counts on BAL samples are not available at our facility. His histology results were pending and the patient was discharged with a prescription for domiciliary oxygen and a follow-up date within 2 weeks to review the results, after which a decision regarding medical therapy would be considered. At the time of discharge, he noted improvement in his symptoms and his oxygen saturation measured by pulse oximetry had improved to 92% on room air. He was advised to avoid returning to work until he had been reviewed.

At his follow-up visit, the patient noted worsening in his symptoms with recurrence of dyspnoea and a cough. He had returned to work despite our recommendation and had effectively conducted an unintentional provocation test. His oxygen saturation measured by pulse oximetry had declined to 78% at that visit, with fine crackles noted posteriorly. The bronchoscopic biopsy results were reviewed, which showed alveolar septal walls expanded by fibrosis and

Fig. 2. High-resolution computed tomography chest scan (axial view) showing mosaic attenuation.

interstitial inflammation composed of lymphocytes, histiocytes and plasma cells. There were no eosinophils, foreign body giant cells or granulomas. These were nonspecific features of interstitial pneumonia and fibrosis and although these were not sufficient to confirm hypersensitivity pneumonitis (HP), they were consistent with the overall picture. His clinical improvement while away from work and subsequent worsening on return to the farm was also highly suggestive of OHP. He was commenced on oral prednisone at a dose of 30 mg once daily (0.5 mg/kg) and strongly advised not to return to work.

On subsequent review he had shown further improvement in his symptoms with an oxygen saturation measured by pulse oximetry of 95% on room air one month later. Pulmonary function testing has shown an improvement in his FVC to 4.26 L (90% of the predicted value) and FEV_1 2.28 (57% of the predicted value). At the time of submission, his steroids were being tapered over a period of 3 to 6 months and his case was referred to occupational health for intervention.

Discussion

A review of this case highlighted a few key learning points. This patient was symptomatic for a prolonged period, with chronic hypoxia and resultant secondary polycythaemia, which put him at a greater risk for thrombotic events and possible morbidity. He had been initiated empirically on anti-tuberculosis therapy without microbiological evidence, which was reasonable in the endemic TB region that we live in. However, his failure to respond to therapy should have prompted a search for an alternative cause of his symptoms.

Similarly, the lack of response to asthma treatment and absence of other features to support the diagnosis makes it more likely that his airflow obstruction was a feature of HP rather than a true manifestation of asthma. Approximately 20 - 40% of patients with HP may have nonspecific airway hyper-reactivity, with 5 - 10% of these patients diagnosed with concomitant asthma. The bronchial hyper-reactivity seen in patients with HP may be secondary to bronchiolitis.[1,2]

Furthermore, the delay in recognising the condition allowed him to have repetitive exposure to the offending antigen. The chronicity of his symptoms with features of fibrosis evident on his HRCT chest scan suggested longstanding exposure.

This case draws attention to the diagnostic challenges associated with this condition. There have been many proposed diagnostic criteria for HP. The most commonly utilised criteria, published by Schuyler and Cormier[9] have not been validated. These criteria include the presence of compatible symptoms, evidence of exposure to the offending antigen, symptoms correlating with recurrent antigen exposure, consistent findings on chest imaging, a lymphocyte predominance on BAL fluid and compatible histopathological features.

Inhalational challenges have been utilised in the appropriate setting when alternative procedures have failed to identify the diagnosis of hypersensitivity or causal exposure with accuracy, or when the suspected precipitating agent has not been described as causing OHP. [10]

We postulate that the lack of documentation of cases of MWL in SA is due to this condition being under-recognised or under-reported and further steps should be taken to assess all workers at mushroom-cultivating farms for respiratory symptoms. Farm managers should also ensure that protective respiratory equipment is available and consistently utilised. Ultimately, a high index of suspicion is required and a thorough occupational and exposure history should be obtained in any patient with dyspnoea of unknown cause to ensure that a treatable condition associated with significant morbidity is not overlooked.

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