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ACUTE HYPERSENSITIVITY PNEUMONITIS ASSOCIATED WITH MUSHROOM WORKER'S LUNG – A CASE REPORT

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ABSTRACT

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis is a non-IgE mediated allergic reaction to inhalation of an allergen. There are more than 200 allergens known to cause this condition, but the list of antigens responsible for the development of HP is constantly expanded.

Dangerous agents are usually inhaled at the workplace, so we may distinguish the following patients at risk of HP: farmers, pigeon breeders, mushroom workers, cheese employees or even malt workers. Mushroom worker's lung (MWL) has been described in

workers exposed to mushroom spores or to inhaled antigens from compost (mostly thermophilic actinomycetes).

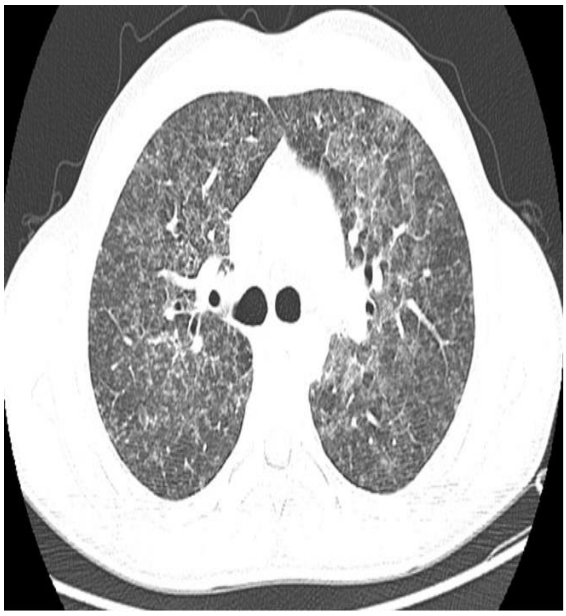
KEYWORDS: acute hypersensitivity pneumonitis, mushroom worker's lung, thermophilic actinomycetes

CASE REPORT

A 31-year-old man was referred to Department of Pulmonology with a history of cough and dyspnea developed 2 weeks after starting work at a local mushroom's farm. His duties at work included planting and harvesting button mushrooms in an enclosed area, as well as making and packing mushroom compost. He did not use protective respiratory equipment, but cessation of exposure to the mushroom compost had alleviated his symptoms to a small extent. Our patient fulfilled special questionnaire asking about other possible expositions to different antigens, but only mushroom exposure was underlined. Past medical and family history was not clinically significant, he had never been hospitalised before, he had no comorbidities.

Physical examination on the day of admission revealed fever (39°C), tachypnea (30 breaths per minute) and central cyanosis. The general state was acute, the patient presented symptoms of respiratory failure. His oxygen saturation measured by pulse oximetry was 87% on room air. We auscultated diffused inspiratory crackles over the lung fields. Also hypoxemia in blood gas analysis drew our attention. His haemoglobin on admission was 14 g/dL with a haematocrit of 44%. He had leucocytosis (white cell count of 20,08 x 10^3/L) and elevated inflammatory markers (C-protein of 115 mg/l). The serum of patient gave a precipitin reaction to an extraction of mushroom compost - *Thermoactinomyces vulgaris* was found (these thermophilic actinomycetes are responsible for the decay of self-heating mushroom's compost). Although no reaction occurred to a variety of hay antigens used in the diagnosis of farmer's lung.

We did not observe spirometry features of obstruction. Whole-body plethysmography showed reduced total lung capacity (TLC), which was suggestive of air trapping. Patient's diffusing capacity for carbon monoxide was also reduced. Chest X-ray revealed thickened stromal lung markings with numerous poorly defined small opacities throughout both lungs (fleeting, micronodular pattern). Also pattern of fine reticulation occurred. A high-resolution computed tomography (HRCT) scan of his chest showed massive, bilateral ground-glass and alveolar opacities. Areas of air trapping were visible. Also hilar and mediastinal lymph nodes were enlarged. The radiologist suggested that radiological abnormalities are compatible with allergic alveolitis (Figure 1 and 2). Our patient underwent flexible bronchoscopy with bronchoalveolar lavage (BAL). Cells obtained from BAL showed increased proportion of lymphocytes (60%).



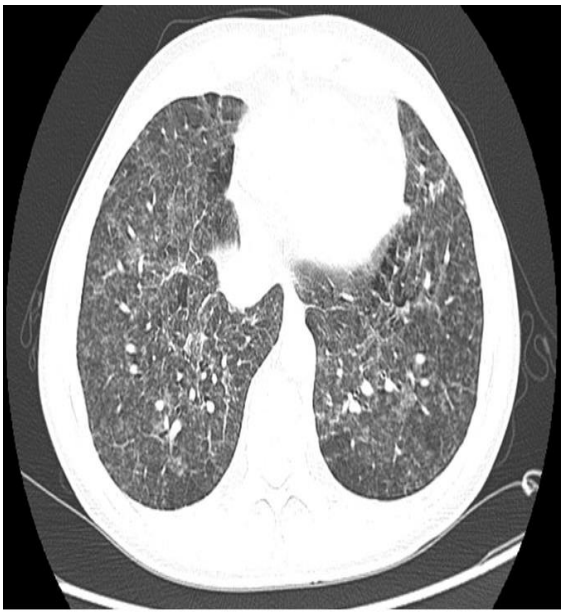


Figure 1 *Figure 2*

High-resolution computed tomography chest scans showing mosaic attenuation and reticular pattern. Hilar and mediastinal lymph nodes are enlarged. We might also observe a lower zonal predilection, which is characteristic for acute hypersensitivity pneumonitis.

Taking into account clinical picture, characteristic HRCT features and BAL lymphocytosis we diagnosed acute hypersensitivity pneumonitis associated with mushroom worker's lung. We administered methylprednisolone by intravenous way and in a few days – orally. Our treatment lasted one month. The patient changed his workplace, because we strongly advised to avoid returning to work at mushroom's farm. One month later, the symptoms subsided, changes in his chest X-ray regressed and lung function tests improved markedly - the patient presented clinical recovery.

DISCUSSION

Hypersensitivity pneumonitis (HP), or extrinsic allergic alveolitis, is an inflammatory disorder of the lung caused by insistent inhalation of antigens in a susceptible host. Clinical presentations of HP have classically been defined as acute, subacute, and chronic. The distinction between acute and subacute HP is often difficult, that is why some authors distinguish only acute and chronic form of the disease [1, 2].

Acute hypersensitivity pneumonitis (AHP) is an immunologic lung disease with variable clinical presentation resulting from lymphocytic and frequently granulomatous inflammation of the peripheral airways, alveoli, and surrounding interstitial tissue [3,4]. The pathogenesis of the disease is complex: specific IgG antibodies are produced, macrophages secrete numerous cytokines (eg. TNF-α, Il-1), cell-mediated response is developed and production interferon-ϒ is intensified. After subsequent exposures episodic lung inflammation is developed, immune complexes form and neutrophils influx. Particularly, the disease develops as the result of a non-IgE-mediated allergic reaction to a variety of organic or low-molecular-weight agents e.g. bacteria, fungi, enzymes and animal/insect proteins, plant proteins, chemicals or metals [5, 6, 7].

There are no established criteria for the diagnosis of AHP. Many offers have been proposed so far, but have not been validated. Cormier et al. [8] and Schuyler et al. [9] underlined that the fundamental components of AHP diagnosis are correlations between exposure and the presence of antibodies against suspected antigen, clinical picture (influenza-

like symptoms), increase in the number of neutrophils and lymphocytes in bronchoalveolar lavage and significant improvement after cessation of exposure and deterioration after re-exposure.

Mushroom worker's lung (MWL) has been described in workers exposed to mushroom spores or to inhaled antigens from compost. MWL was originally reported in the second half of the twentieth century by Bringhurst et al. [10] and Sakula et al. [11]. The main source of antigens is proliferation of thermophilic actinomycetes (e.g. *Micropolyspora faeni*, *Thermoactinomycetes vulgaris*) or spores of Japanese fungi (e.g. *Pholiota namcho*, *Pleurotos ostreatis)*. Mushroom cultivation requires high temperatures (approximately 60 °C) and humidity of 100%. These conditions favour the development of actinomyces. Crushing the substrate and mixing with the mycelium results in the dust rise, which causes the symptoms of the disease among sensitive and unprotected people. Improved ventilation and protective respiratory equipment may reduce exposure to these mushroom antigens. A genetic predisposition to HP has been suggested based on several lines of evidence [12].

Jackson et al. suggested that ccute hypersensitivity pneumonitis connected with mushroom worker's lung is characterised by severe dyspnea and dry cough developing within few hours after initial exposure. An influenza-like syndrome may occur (fever, chills, headache, chest tightness, muscle pain). During severe form of HP symptoms of respiratory failure might appear. Tachypnea and tachycardia can be found in physical examination, also symmetrical inspiratory crackles are auscultated over the lung fields. Wheezing is rarely present. Described symptoms may be confused with viral or bacterial infections, and patients are often treated initially with antibiotics [13, 14].

In the opinion of Selman et al. [15] the first step in the evaluation of a patient with suspected HP is a detailed history of potential occupational, avocational, or domestic exposures. Sometimes it helps to use a standardized questionnaire. Leucocytosis, neutrocytosis and elevated C-protein level are common laboratory abnormalities. The presence of serum precipitins (specific serum immunoglobulin G antibodies) is only evidence of exposure, but their role in the pathogenesis of HP is uncertain. Positive results can be seen in exposed, but asymptomatic individuals. Diagnosis of acute HP is confirmed when we have clinical symptoms after exposure to the detected antigen and increased amount of precipitins. Pulmonary function tests do not have any characteristic diagnostic pattern. Patients with acute HP usually have features of restriction in spirometry and reduced diffusing capacity of carbon monoxide (DLCO) [16].

According to Hansell et al. [17] and Adler et al. [18] studies in the radiological background micronodular pattern may be identified in the lower and middle lung zones on chest radiograph, but the chest film is frequently normal. In high resolution computed tomography (HRCT) the acute phase of HP is dominated by air space abnormalities with no features of fibrosis. There are usually homogeneous ground-glass or nodular opacities which are typically bilateral and symmetric. HRCT may also reveal focal areas of low attenuation or decreased perfusion associated with air trapping and typical mosaic perfusion pattern might be visible. Periodically a lower zonal predilection is seen. The combination of segmental or lobular air trapping, normal lung, and ground glass changes can give a head cheese sign [19].

Bronchoalveolar lavage (BAL) reveals an inflammatory process in the alveoli. It is the most sensitive tool to detect acute HP, but not always necessary especially in patients with a convincing exposure history and typical HRCT findings. The profile of the cells from BAL depends on the time after exposure. We usually observe an increase in the number of cells. In acute HP there is marked BAL lymphocytosis: the percentage of lymphocytes can reach up to 70% and CD8 lymphocytes predominate. In our patient we have also observed increased proportion of lymphocytes (60%), which confirmed our diagnosis. What is more, BAL

lymphocytosis can also be seen in organizing pneumonia and nonspecific interstitial pneumonia, but not usually at this high level [20, 21, 22].

A lung biopsy can be obtained via transbronchial lung biopsy (TBLB), often at the time of a BAL, transbronchial cryobiopsy (TBCB), or video-assisted lung biopsy. The diagnosis of acute HP often relies on histopathology. The histopathological triad of HP includes:

1. chronic cellular bronchiolitis with a peribronchial infiltration of lymphocytes
2. small, poorly-formed noncaseating granulomas located near respiratory or terminal bronchioles
3. chronic cellular pneumonitis with a patchy mononuclear cell infiltration (predominantly lymphocytes and plasma cells) of the alveolar walls.

Also a trial of antigen avoidance that leads to resolution of symptoms and radiographic opacities lends supportive evidence to a diagnosis of HP [23, 24].

The first-line treatment for patients with acute symptomatic HP connected with mushroom worker's lung is to avoid costant exposure to the mushroom's compost. Oral glucocorticoids should be taken, usually the initial dose is the equivalent of prednisone 0,5 to 1 mg/kg per day (up to 60 mg/day); the initial dose is maintained for about 2 weeks, as we have done in presented patient [25, 26].

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