

and IgG-Af and observed in both parameters a progressive decrease related to the clinical improvement of the two patients. We also found a high and significant correlation between IgE-Af and total IgE. Nevertheless, we found no correlation between IgG-Af and total IgE. Thus IgE-Af could be a good indicator of ABPA activity and so could be used as an important aid in the management of ABPA.

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A case of lymphocytic pneumonitis, myositis, and arthritis associated with exposure to *Aspergillus niger*

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CD8 lymphocytic alveolitis has been reported in various disorders.¹ One of the leading causes is hypersensitivity pneumonitis (HP). We describe here a case of CD8 lymphocytic pneumonitis with myositis and arthritis, associated with exposure to *Aspergillus niger*. Up to now, *A. niger* has been described as an agent of chronic necrotizing pneumonia,² bronchial asthma,³ invasive aspergillosis,⁴ and mycetoma.

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Abbreviations used

CPK: Creatine phosphokinase
HP: Hypersensitivity pneumonitis

CASE REPORT

A 50-year-old nonsmoking man was admitted to the hospital in October 1991 because of a 6-week history of exertional dyspnea, weakness, myalgias, dysphagia, and arthralgias. He owned a fishing article store where he prepared bait with several compounds including corn flour, bread crumbs, oat, salt, barley, malt, coconut, polenta, and nitrates; all were mixed with a scoop and then manually packaged.

Physical examination revealed inspiratory dry crackles in both lungs. Painful fingers and stiffness were noted. No rash or adenopathy was found, and results of neurologic examination were normal. No clear amyotrophy

was noted, but the patient had difficulty rising from a chair. Chest roentgenogram showed bilateral infiltrates in the lung bases, confirmed by a computed tomographic scan.

The white blood cell count was $10,770/\text{mm}^3$ with 64% neutrophils, 32% lymphocytes, and no eosinophils. Erythrocyte sedimentation rate was 30 mm/hr. The creatine phosphokinase (CPK) level was 5780 UI/L (normal value: 25 to 190); MB fraction was 2.1%. Hepatic enzyme levels, as well as kidney and thyroid function, were within normal limits. No proteinuria was found. Room air arterial gas showed a pH of 7.44, PaO_2 of 66.6 mm Hg and Paco_2 of 32.8 mm Hg. Pulmonary function tests revealed vital forced capacity of 2.59 L (52% of predicted value), forced expiratory volume in 1 second of 2.22 L (62% of predicted value), and a diffusion capacity for carbon monoxide of 65% of the predicted value. Results of fiberoptic bronchoscopy were normal, and in the bronchoalveolar lavage the total number of cells was $390.10^6/\text{L}$: macrophages 17%; lymphocytes 72% with a CD4/CD8 ratio of 0.13; neutrophils 7%; eosinophils 4%. All bacterial and fungal cultures remained sterile. Results of exhaustive viral serology tests (including the test for human immunodeficiency virus) were negative. IgE was 130 U/ml (normal value < 140 U/ml); and results of tests for anti-JO-1, rheumatoid factor, and antinuclear antibody were negative. Ouchterlony immunodiffusion of the serum revealed two precipitin bands in response to *A. niger* and remained negative for *A. fumigatus*, thermophilic actinomycetes, *Penicillium notatum*, and pigeon serum and stool.

The patient was discharged without any medication and was told to avoid the mixing operations. He could not be removed from his work environment because the store was a part of his house.

Four weeks later, in the absence of major clinical improvement, we decided to perform pulmonary and muscular biopsies. At that time, CPK level had decreased to 1405 UI/L, and the functional and radiologic abnormalities were unchanged.

Electromyography showed mild myogenic disease. Histologic examination of the biopsy specimen of the right rectus femoris showed plurifocal perimysial and endomysial lymphocytic infiltrates with areas of degeneration and regeneration. Pulmonary fragments obtained by pleuroscopy showed interstitial lymphocytic infiltrates and mild fibrosis without granuloma. Vessels were normal. Lymphocytes were CD3 positive (pan-T antigen) on immunohistochemical stainings of both biopsy specimens.

A. niger was isolated from five compounds handled by the patient. A fungal glycoprotein extract was prepared with the isolated strain. For the patient, two precipitating lines against this extract were observed by counter-immunoelectrophoresis, and numerous and intense bands were displayed by sodium dodecylsulfate-polyacrylamide gel electrophoresis IgG immunoblot. Serum from a subject who was free of symptoms and worked in the

same room, as well as a pool of sera from blood donors, were serologically negative.

Methylprednisolone 0.5 mg/kg/day was prescribed, and mixing operations were definitely stopped. Two months later, clinical, biologic, radiologic, and functional abnormalities had disappeared. Corticosteroids were slowly tapered and stopped without any clinical or functional deterioration. However, the patient was lost to follow-up after a few months. Two years later, according to his general practitioner, the patient was free of symptoms, and the chest roentgenogram was normal.

DISCUSSION

The association of myositis, arthritis, and pneumonitis suggests polymyositis after the exclusion of the usual causes of myositis (e.g., viruses, endocrinopathy, drugs) and other connective tissue diseases. However, the presence of a strongly CD8 lymphocytic alveolitis evokes HP.

To the best of our knowledge, only one limited study has reported bronchoalveolar lavage findings in cases of dermatopolymyositis, showing a mixed cellular population of macrophages and lymphocytes in three cases associated with pulmonary fibrosis.⁵ No frank CD8 lymphocytic alveolitis, as described here, has ever been reported in that disease, and the dramatic response to moderate corticotherapy is also an uncommon feature in polymyositis.⁶ In the setting of CD8 lymphocytic alveolitis, the aforementioned immunologic findings lead us to presume that *A. niger* was the offending agent. Despite the fact that the presence of precipitins is a well known marker of exposure to an antigen source (with or without active disease caused by this antigen), positive immunoblot with negative controls, one of which was performed on the serum of the patient's son who worked in the same environment, deserves consideration.⁷ Moreover, the T-lymphocyte infiltrates both in lung and muscle (CD4 and CD8 staining was not available) could suggest the presence of a similar immunologic effector. Interestingly, we noted a spontaneous significant decrease in CPK level 4 weeks after the patient was instructed to stop the mixing procedures.

Nevertheless, HP still remains debatable in the absence of an inhalation challenge (with CPK measurements), which surely would have been of great value.⁸ Unfortunately, the patient declined.

In any case, if we consider the diagnosis of HP, the occurrence of HP caused by *A. niger* as well as that of HP and polymyositis together have never been reported.

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Allergens in school dust:

II. Major mite (*Der p I*, *Der f I*) allergens in dust from Swedish schools

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Several studies have reported that *Dermatophagoides pteronyssinus*, *D. farinae*, and *D. microceras* are the most common house dust mites in Northern and central Europe.¹⁻⁵ Environmental factors such as poor ventilation, dampness, high humidity, and high temperature favor the growth of mites indoors.^{1, 4, 5}

There is increasing evidence that house dust mite allergens are important in the etiology and pathogenesis of asthma.⁶ Asthmatic symptoms improve in patients removed from allergen exposure at home.⁷ However, exposure to indoor allergens in public places is also of importance.^{8, 9}

The most important individual allergens derived from *D. pteronyssinus*, *D. farinae*, and *D. microceras* are the closely related, cross-reacting group 1 allergens, *Der p I*, *Der f I*, and *Der m I*, respectively. These major allergens can be measured in dust by

immunoassays, and the concentrations inducing sensitization and triggering bronchial asthma have been suggested.⁶

Relatively few studies on mite allergen levels in public places have been reported.^{4, 8, 9} In a recent study, the levels of mite allergens in schools in Norway were reported.⁸

The purpose of this investigation was to study the levels of the major mite allergens *Der p I* and *Der f I* in school dust in the Linköping area in central Sweden and to relate these levels to some technical characteristics of the school buildings.

METHODS

Technical data on school buildings and classrooms and information about students and teachers were obtained from administrative persons at each school. Table I demonstrates some characteristics of the schools and classrooms. In total, 29 classrooms in four schools were investigated. The schools were randomly numbered from 1 to 4.⁸

Various cleaning methods were applied to the classrooms studied.⁸ Some information about cleaning frequency and cleaning procedures used for cleaning tables, chairs, and floors is given in Table I.

Dust collection

Dust was sampled from 5 m² of floors and from the horizontal surfaces of tables and chairs from 29 class-

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