

Allergic bronchopulmonary cladosporiosis in a child

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Since the first description of allergic bronchopulmonary aspergillosis (ABPA) in 1952, many reports in the literature have increased the knowledge about immunologic mechanisms, clinical presentation, diagnostic criteria, and treatment of the disease.¹⁻³ *Aspergillus fumigatus* is the most common fungus involved, but other *Aspergillus* species and other fungi have been reported as causative agents.⁴⁻¹² *Candida albicans*,^{4, 11} *Mucor* spp.,¹¹ *Helminthosporium* spp.,⁵ *Stemphylium* spp.,⁶ *Torulopsis* spp.,⁷ *Curvularia lunata*,^{8, 12} *Rhizopus* spp.,^{10, 11} *Penicillium* spp.,¹¹ *Drechslera* spp.,⁸ and *Pseudallescheria* spp.¹¹ are fungi involved in allergic bronchopulmonary mycosis (ABPM) (Table I).

Diagnostic criteria for ABPA are well established in adult patients,¹ but there are few reports of this disease in children.^{2, 3, 9, 12, 13} We present a case of childhood ABPM caused by *C. herbarum*. To our knowledge, this is the only published case of childhood ABPM caused by this fungus. Remission was achieved by adequate corticosteroid treatment.

CASE REPORT

A 6-year-old child was seen in our outpatient clinic for assessment asthma symptoms. For the last 3 years, he had had several febrile episodes of cough, dyspnea, green-brown sputum production, and wheezing. Chest roentgenograms had shown pulmonary infiltrates on several occasions. He was seen in our hospital's emergency department because of a new episode, and he was referred to our department.

Physical examination revealed disseminated ronchi and wheezes. Laboratory results included a white cell count of 7100/mm³ with an eosinophil count of 1690/mm³. Erythrocyte sedimentation rate was normal. Blood chemistry, α_1 -antitrypsin, and serum immunoglobulin

Abbreviations used

ABPA:	Allergic bronchopulmonary aspergillosis
ABPM:	Allergic bronchopulmonary mycosis

levels and sweat electrolytes were within the normal range. Results of Mantoux test and several blood cultures were negative. Antibodies for *Mycoplasma pneumoniae* and respiratory viruses were not found in serum.

Allergy tests

Skin prick test reactions were positive (wheal: 6 × 7 mm) to *Cladosporium herbarum* and weakly positive (wheal: 3 × 2 mm) to *Lolium perenne* pollen and cat dander. (The control histamine wheal was 5 × 5 mm, and the saline solution wheal was 0 mm.) Intracutaneous tests were immediately positive to *Cladosporium herbarum*, *Aspergillus* spp., *Penicillium notatum*, and *Alternaria tenuis*; and negative to other fungi. All late reactions (6 and 24 hours) were negative.

Total IgE level (measured by microparticle enzyme immunoassay with an IMX system (Abbot Diagnostics, Chicago, Ill.) was 1771 ng/ml.

Specific IgE to fungi were measured by fluoroimmunoassay (CAP system; Pharmacia Diagnostics, Uppsala, Sweden).

Specific IgE was positive to *Cladosporium herbarum* (14.8 kU/L), *Alternaria tenuis* (0.7 kU/L), and *Aspergillus fumigatus* (0.57 kU/L). Specific IgE to other fungi were negative (<0.35 kU/L). We also determined the specific IgE to a serum pool sample from asthmatic patients with positive prick test responses to fungi and without ABPM. IgE values were 7.47 kU/L to *Alternaria tenuis*; 6.7 kU/L to *Aspergillus fumigatus*, and 5.7 kU/L to *Cladosporium herbarum*.

Specific IgG to fungi were assessed by CAP system fluoroimmunoassay (Pharmacia Diagnostics), and results are expressed as percentage of the mean fluorescence value of a control serum. Specific IgG were positive to *Cladosporium herbarum*, *Alternaria tenuis*, and *Aspergillus fumigatus*. Precipitating antibodies measured by immune electrodifussion in agar plaque were found positive only against *Cladosporium herbarum*.

Sputum culture for fungi was negative, but marked

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J ALLERGY CLIN IMMUNOL 1996;97:714-5.

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0091-6749/96 \$5.00 + 0 1/54/68126

eosinophilia was observed in a sputum cell sample. A computed tomographic scan showed central bronchiectasis in the right middle lobe and central and middle bronchiectasis in the left lower lobe. It was not possible to carry out a pulmonary function test because of lack of patient collaboration.

A diagnosis of acute phase of ABPM was established (stage 1). *Cladosporium herbarum* was considered to be the causative agent. A 3-month course of oral corticosteroid therapy (prednisone, 0.5 mg/kg/day) was completed.

Remission of the disease (stage 2) has persisted since completion of oral corticosteroid treatment. Laboratory tests after treatment showed a blood eosinophil count of 980/mm³, and total IgE levels below 1000 ng/ml. No significant changes were found in specific IgE and IgG levels. A computed tomographic scan performed 12 months after the treatment did not show worsening of bronchiectasis. The patient is still under close observation for any new acute episode.

DISCUSSION

ABPM clinically presents with episodic wheezing, blood eosinophilia, elevated serum IgE level, serum precipitating antibodies (IgG), and serum IgE antibodies for fungi antigens.^{1,2} Specific IgE levels to the causative fungus should be higher than twice those found in a serum pool sample of asthmatic patients sensitized to the same fungus but without ABPM.¹

Aspergillus fumigatus is the most common fungus involved in this disease. Other fungi, however, have been reported as causative agents.⁴⁻¹² ABPM has seldom been described in children.^{2,3,9,12,13} This may be due to the correspondence of established diagnostic criteria to an advanced ABPM stage. Only several cases reported in children have met all of the diagnostic criteria for ABPM.^{12,13} Our patient also met all diagnostic criteria for allergic bronchopulmonary cladosporiosis.

It is noteworthy that patients are sometimes sensitized to several fungi that are concomitantly responsible for the same condition^{8,11,13}; in other cases, as in ours, only one of the fungi meets all of the diagnostic criteria.

The diagnosis of childhood ABPM requires a high degree of suspicion. Early diagnosis and aggressive therapy can achieve resolution of pulmonary infiltrates and prevent lung sequelae, such as bronchiectasis, which does not usually disappear after treatment when previously present. Pediatric patients are problematic because only a precise diagnosis may warrant the potential risk of corticosteroid therapy. Moreover, clinical suspicion is critical in children because in some cases all diagnostic criteria may not be present.

TABLE I. Fungi involved in ABPM

<i>Candida albicans</i> ^{4,11}
<i>Mucor</i> spp. ¹¹
<i>Helminthosporium</i> spp. ⁵
<i>Stemphylium</i> spp. ⁶
<i>Torulopsis</i> spp. ⁷
<i>Curvularia lunata</i> ^{8,12}
<i>Rhizopus</i> spp. ^{10,11}
<i>Penicillium</i> spp. ¹¹
<i>Drechslera</i> spp. ⁸
<i>Pseudallescheria</i> spp. ¹¹

In conclusion, we present a new case of childhood ABPM caused by *Cladosporium herbarum*, which meets all accepted diagnostic criteria. Adequate treatment with prednisone achieved remission of the disease. In suspected ABPA with negative test responses to *Aspergillus* species, the possibility of other fungi as causative agents should be investigated.

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