

Farmer's lung in infants and small children

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A boy and a girl, 10 weeks and 3 years of age, respectively, were admitted to our department with low temperature, dry cough, fatigue and weight loss. In both patients pulmonary X-rays showed diffuse, bilateral, micronodular infiltrations, and sparse signs of fibrosis. Serum IgG and blood eosinophils were abnormally high. After a stay in hospital for 3 weeks, the patients recovered slowly. However, after a few days at home, they were readmitted with the same symptoms. Family histories revealed that the children lived on farms with huge grain magazines and dryers, where moist grain and straw were stored. Massive amounts of mould spores were cultured from the residential areas, and, in addition, the male patient had an elevated titer to *Micropolyspora faeni* and the female patient, elevated titers to *Thermoactinomyces vulgaris*, *Micropolyspora faeni*, *Aspergillus fumigatus* and *Alternaria alternans*. The patients and their families moved from the farms and, for approximately a year, have been without lung symptoms. Farmer's disease in infants and small children is extremely rare. However, the incidence may be increasing due to the tendency in latter years to decentralize grain dryers and store moist grain and straw in big magazines, often close to residential areas.

Key words: allergic alveolitis; farmer's lung; hypersensitivity pneumonitis; infants; small children.

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Farmer's lung (FL) is defined as a condition of allergic alveolitis caused by inhalation of mould spores, such as *Micropolyspora faeni* and *Thermoactinomyces vulgaris*, often grown in moist grain and straw stored in large magazines (5, 12, 16, 17). FL is a serious disease, occasionally leading to progressive fibrosis resulting in chronic lung disease (15). FL is extremely rare in children (2, 7, 8, 13). The aim of the present study is to draw attention to the disease and to describe the clinical picture of the syndrome and the diagnostic difficulties in children.

CASE REPORTS

Case 1

A 10-week-old boy was admitted to our department with tachypnoe and dry cough

during which he became cyanotic. He had lost weight and ran a low temperature. The boy was the second child of healthy parents and was born after a normal pregnancy and delivery. Symptoms started at the age of 6 weeks and slowly progressed to the actual status. On admission lung stethoscopy revealed fine crepitation bilaterally, and chest X-ray showed micronodular infiltrations scattered diffusely over both lung fields. Laboratory tests showed blood eosinophilia $2539 \times 10^6/l$ ($50-700 \times 10^6/l$). Serum immunoglobulins were all elevated, IgG 17.9 g/l (3.0-8.0 g/l), IgA 1.26 g/l (0.03-0.60 g/l), IgM 3.76 g/l (0.10-0.60 g/l), except serum IgE which was normal for age. Serum trypsin and lipase activity and three quantitative sweat electrolyte tests were normal. Stools cultured for pathogenic organisms were negative and cultures from lower airway secretions grew normal saprophytes.

During the patient's 3 weeks stay in hospi-

tal, he recovered without medication and a follow-up chest X-ray showed regression of the infiltrations. A few hours after coming home, however, he was readmitted with the same symptoms. Now the family history revealed that the farm owned by the family was based on grain production, and a grain store in connection with a grain dryer was situated close to the living quarters.

On suspicion that the boy's symptoms were caused by an immunological reaction to mould spores, laboratory tests were performed for precipitating IgG antibodies to mould spores, and a titer of 4 to *Micropolyspora faeni* was found (ALK, Copenhagen, normal 1,000 donor pool: 0). In addition, culturing of samples (ALK, Copenhagen) from the residential property showed massive growth of actinomyces species. However, RAST for cow milk, animal danders and several mould spores were negative.

The family moved to other residence and for 6 months the boy has been well. He has a normal chest X-ray, but the titer of 4 to *Micropolyspora faeni* still remains, for how long and why is still to be investigated.

Case 2

The 3-year old girl was admitted to our department with tachypnoe, dry cough, cyanosis and loss of weight. The patient was the only child of healthy parents and the pregnancy and delivery had been unevenful. At 10 months of age she had her first attack with tachypnoe, fever and dry cough and was treated several times with antibiotics without success. On admission, lung stethoscopy revealed fine crepitation bilaterally and chest X-ray showed diffuse scattered, micronodular infiltrations with sparse signs of fibrosis in both lung fields. Laboratory tests showed severe blood eosinophilia $1722 \times 10^6/l$ ($30-600 \times 10^6/l$) and serum IgG was raised to 16.8 g/l (4.5-12.3 g/l). However, IgA, IgM and IgE were normal for age. Attempts to culture parasites from her stools were unsuccessful. Two consecutive quantitative sweat electrolyte tests were normal. Culture from lower airway

secretions grew *Branhamella catarrhalis* and *Haemophilus influenzae*, but treatment with ampicilline was without effect.

She gradually recovered over a period of 3 weeks, and a follow-up chest X-ray showed regression. However, already a few days after discharge, she was readmitted with the same symptoms. On this occasion, her family history revealed that she lived on a farm. A grain store in connection with a grain dryer was situated close to the living quarters.

Due to the suggestion that her lung symptoms might be caused by inhalation of mould spores, investigations for precipitating serum IgG-antibodies to mould spores were carried out. This resulted in a positive titer of 8 to *Thermoactinomyces vulgaris*, of 4 to *Micropolyspora faeni* and *Aspergillus fumigatus*, and a titer of 32 to *Alternaria alternans* (ALK, Copenhagen, normal 1,000 donor pool: 0). In addition, a culture of samples (ALK, Copenhagen) from the residence showed massive growth of mould spores of the above species.

She was no longer allowed to visit the grain store or dryer, and the family moved to another house, 1 kilometer away. For 12 months she has been well and her chest X-ray has been normal for the last 6 months.

Skin prick tests were performed for pollen, animal danders, house dust mites, and several mould spores, with negative results (Pharmacia, ALK, Copenhagen). RAST to the same allergens, including the mould spores mentioned above, were negative. Ten months after her first admission to the department, investigations of precipitating serum antibodies to *Thermoactinomyces vulgaris*, *Micropolyspora faeni*, *Aspergillus fumigatus* and *Alternaria alternans* were repeated. On this occasion, the results were negative.

DISCUSSION

FL is a syndrome, usually divided into three subgroups. Firstly, an acute condition, which occurs 4 to 8 h after massive exposure to mould spores, generated from moist grain and straw, clinically inseparable from *influenzae* with high fever, chills, headache and a dry

cough. Chest X-rays are usually normal, and the patient recovers without treatment within a few days (1, 4). Secondly, a less acute condition is found after daily but slight exposure to mould spores, characterized by weight loss, low-grade fever, fatigue and coughing, often followed by cyanosis and difficulty in breathing. Lung stethoscopy reveals fine crepitation, and chest X-ray shows bilateral, diffused scattered, micronodular infiltrations. If, in such a patient, exposure to mould spores is interrupted, he usually recovers within a few months (1, 4). Thirdly, a more irreversible form, followed by increasing signs of obstructive lung disease and pulmonary fibrosis, probably due to prolonged (years) exposure to mould spores (1, 4, 6). In both the acute and subacute conditions there is often hyperimmunoglobulinemia G in serum and granulocytosis, followed by blood eosinophilia (3). In patients with FL, IgE-mediated disease (Type I reaction) is rare (10, 11). However, in adults with bronchopulmonary aspergillosis, Type I reactions occur simultaneously with Type III reactions (3).

The pathogenesis of the FL syndrome is not obvious. However, the existence of precipitating IgG-antibodies in serum to mould spores points to a Type III reaction taking place in the lungs of these patients (3, 10). In addition, some have claimed that a Type IV reaction is also present (3, 9, 10). On the other hand, precipitating antibodies in serum to mould spores are also found in otherwise healthy individuals, especially farmers working with grain and straw (3, 10, 14).

The incidence of FL in infancy and early childhood is unknown, but probably extremely rare, and to our knowledge, only five cases have been reported (2, 7, 8, 13). However, in recent years, although we have no data to support the theory, the incidence may have increased, partly because of a growing tendency all over Europe to decentralize grain magazines and dryers, and partly due to a tendency among farmers to store moist grain and straw in the vicinity of the living quarters. Thus people living on the farms risk massive exposure to mould spores.

The only effective treatment for FL besides systemic steroids is to remove the child from the source of generating mould spores. This implies serious social as well as economic problems for the families involved.

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