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ORIGINAL PAPER

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# Bird breeder's disease: a rare diagnosis in young children

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**Abstract** Bird breeder's lung disease is the most common form of hypersensitivity pneumonitis and is a rare entity in young children. We report three cases of children under 7 years of age in whom this diagnosis was confirmed early in the course of the disease. Three children aged 4.4 to 6.5 years presented with dry cough lasting for more than 1 month, dyspnoea, variable loss of appetite, weight loss, fatigue, fever and mild signs of respiratory distress. Chest X-ray films and CT scans showed a bilateral micronodular infiltrate. All three patients had strongly suggestive bronchoalveolar lavage fluid findings with lymphocytosis; two had elevated cell counts and decreased CD4/CD8 ratios. Lung biopsy confirmed the diagnosis in all children. Contact with allergens was identified in all children: two had spent holidays close to a farm in the previous month and one was living next to a pigeon house. In all children, avian precipitins were positive. The symptoms rapidly resolved after allergen avoidance and treatment with oral prednisone. Corticoid treatment was given between 11 and 15 weeks. One child relapsed and required long-term low-dose corticotherapy for 1 year. Lung function tests were normal in all three patients, 3.9 to 5.7 years after diagnosis. *Conclusion:* Bird breeder's lung disease is a rare entity but should be considered in young children presenting long lasting cough. While rapid allergen exclusion and start of treatment can avoid the evolution into irreversible lung fibrosis, clinical and biological evolution should be monitored carefully even after stopping corticoid treatment because of the possibility of relapse.

**Keywords** Bird breeder's disease · Bronchoalveolar lavage · Child · Hypersensitivity pneumonitis · Treatment

**Abbreviations** BAL: bronchoalveolar lavage · BBD: bird breeder's disease · DLCO: diffusion lung capacity for carbon monoxide · HP: hypersensitivity pneumonitis · VAT: video-assisted thoracoscopy

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## Introduction

Although very rare, bird breeder's disease (BBD) is the most common form of hypersensitivity pneumonitis (HP) in children [9]. Pigeon breeder's disease was described for the first time in adults in 1965 by Reed et al. [16] and in children in 1967 by Stiehm et al. [19].

This immune-mediated interstitial lung disease is caused by repeated inhalation of avian antigens in susceptible individuals. Repeated inhalation of avian antigens stimulates humoral immunity by forming immune complexes and cellular immunity by activating lymphocytes T, mainly of the cytotoxic type. Typical clinical manifestations are cough, dyspnoea, fever, fatigue, loss of appetite and weight following exposure. Traditionally, there are four forms of clinical presentation (acute, subacute, chronic and hyperacute); subacute presentation is the most frequent [5].

BBD is very rare in children [9], particularly in young children. We report three cases of young children in

whom a diagnosis of BBD was made early in the acute phase. Treatment with oral prednisone was started immediately. None of these children developed fibrosis although biological alterations persisted in some of them 2 to 4 years after initial diagnosis.

## Case reports

Case 1

A 6.5-year-old boy presented with a 1.5 month history of dry cough, weight loss of 1 kg and an episode of fever of 39°C. He was unsuccessfully treated by his paediatrician with oral roxithromycine, salbutamol inhalation and various antitussive drugs. On admission, physical examination revealed erythematous areas with papulae on both cheeks, tachypnoea at rest (40 breaths/min), an oxygen saturation of 83% in room air and some bilateral crackles on auscultation. A careful past history revealed that the symptoms appeared after the boy had spent a 2-week holiday on a farm where he was in close contact with poultry and other farm animals.

Blood cell count and CRP were normal and different serologies (including *Mycoplasma pneumoniae*, *Chlamydia trachomatis* and *psittaci*, *Legionella pneumophila* and HIV) were negative. An intradermal tuberculin Mantoux test was also negative. Blood lymphocyte phenotyping was normal. The serum IgE level was within the reference range. Screening of specific IgE against common allergens ( *Graminae*, *Alternaria*, birch, hazel, *Artemisia,* plantain, acarid, cat, dog and *Aspergillus*) by the RAST method was negative. Anti-nuclear and anti-DNA antibodies were also negative. Blood immunoelectrophoresis and Ig concentrations showed an elevation of IgG (13.19 g/l, reference range for age 5.35–10.9 g/l) and IgM (2.15 g/l, reference range for age 0.32–1.38 g/l) and normal IgA. Blood precipitins were positive for hen, pigeon and *Aspergillus fumigatus* and strongly positive for hay and parakeet. A chest X-ray film displayed bilateral diffuse reticulonodular lung opacities with a bronchogram in some areas (Fig. 1). The macroscopic appearance on bronchoscopy was compatible with a diffuse non-specific mild tracheobronchitis. The cell count was at the upper limit ($45\times10^4$ cells/ml) with lymphocytosis (lymphocytes 62%, macrophages 26%, granulocytes 12%) and a decreased CD4/CD8 ratio (0.56) were found in bronchoalveolar lavage (BAL) fluid. Ziehl, Grokott and Prussian blue staining were also negative. Malignancies were excluded by bone marrow aspiration and testing for malignant cells and abdominal echography. A lung biopsy performed by video-assisted thoracoscopy (VAT) of the right inferior lobe showed inflammatory infiltrate centered on the terminal bronchioli, interstitial pneumonitis and rare multinucleated giant cells, that were compatible with HP. Masson-Trichrome staining showed slight subpleural and alveolar duct fibrosis.

Allergen avoidance and introduction of oral prednisone (1 mg/kg/day) allowed rapid oxygen withdrawal, disappearance of the symptoms and regression of interstitial infiltrates as seen on a chest X-ray film. Prednisone was tapered down after 1 month and stopped after 3 months. Six months later, the chest film still showed a slight micronodular diffuse infiltrate. Avian precipitins became negative 8 months after the end of exposure and 6 months after onset of treatment. The first interpretable lung function test was performed 9 months after diagnosis and was normal. At this time, a

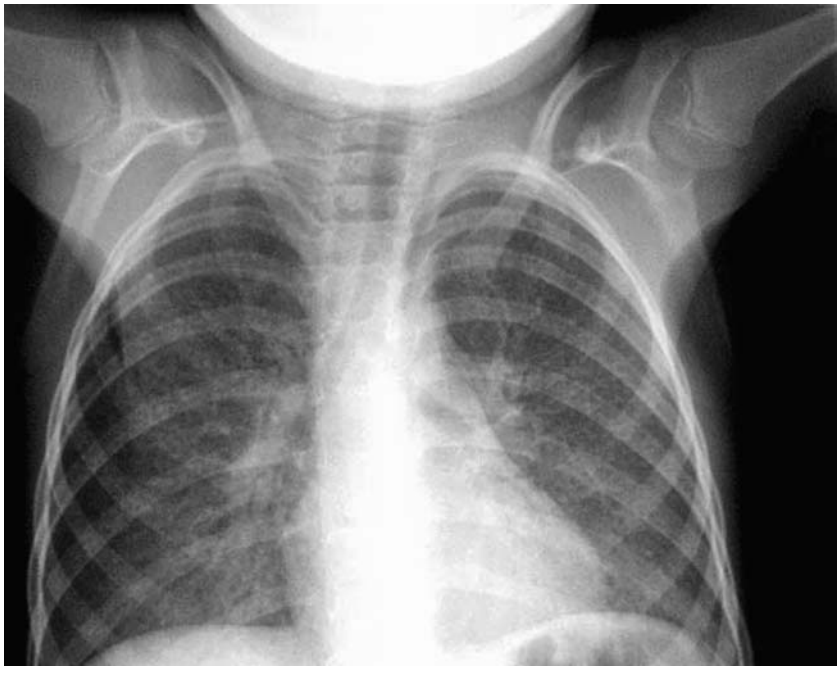

**Fig. 1** Chest X-ray film demonstrating a reticulonodular pattern in both lungs (Case 1)

chest CT scan was normal. The last check-up occurred 5.7 years after diagnosis and the child was still asymptomatic with a normal lung function and CT scan. All avian precipitins remained negative 4.8 years after diagnosis.

## Case 2

A 4-year and 5-month-old girl was referred for a cough lasting for 1.5 months and dyspnoea with wheezing episodes. On examination, the oxygen saturation was within the reference range, the respiratory rate was 30/min and lung auscultation was normal. The initial diagnosis, based on clinical symptoms and a chest X-ray film, was infectious interstitial pneumonia with bronchial hyperreactivity. She was treated by her paediatrician with oral clarithromycin, inhaled salbutamol and fluticasone, without clinical improvement. The second chest X-ray film showed an increasing diffuse micronodular infiltrate predominant on both lung bases and she was referred to our paediatric pulmonology outpatient clinic for further investigations.

Medical history revealed that the symptoms began during a holiday in an old house with poultry, pigeons and other birds nearby. Past medical and family history was positive for atopy. The clinical examination was normal. Laboratory screening was performed as described for Case 1 (blood cell count, CRP, serologies including HIV, intradermal tuberculin Mantoux test, antinuclear factor, serum Ig and precipitins concentrations). In this case, serum IgG levels were markedly elevated (17.4 g/l, reference range for age 5.35–10.9 g/l) and serum IgE just above normal (89.1 IU/ml, +2 standard deviations for age 68.9 UI/ml). Blood precipitins were positive for parakeet and *Aspergillus fumigatus*. A chest CT scan confirmed a diffuse micronodular interstitial infiltrate. Lung biopsy performed by VAT was decided because of normal cellularity and normal CD4/CD8 ratio in BAL and performed in the left upper lobe. It showed a diffuse interstitial pneumonia with a predominant centrolobular pattern. Peribronchiolar inflammation and several granuloma without fibrosis were also present. Histochemical staining for *Mycobacterium tuberculosis*, fungi and search for iron by Prussian blue staining were negative. All these findings strongly supported the diagnosis of BBD.

Allergen avoidance and oral prednisone (1.5 mg/kg/day) were started. Three weeks later, the cough disappeared and dyspnoea on exercise was improved. The dose of prednisone was tapered gradually. One and a half months after diagnosis, the girl was completely asymptomatic and is still at last follow-up. Three and a half months after diagnosis all precipitins became negative and prednisone was stopped and replaced by inhalation of fluticasone (250 μg twice daily for 10 months), which was administered seasonally because of atopy and wheezing episodes; however, therapeutic compliance was poor. Six months after diagnosis, the chest CT scan was normal, but blood IgG remained elevated (13.20 g/L). BAL monitoring showed a persistent lymphocytosis (lymphocytes 48%, macrophages 29%, neutrophils 23%) with a normal CD4/CD8 ratio (1.10). In this case, complete allergen avoidance could not be achieved, because, despite our advice, the family returned to their holiday home. Three years and 9 months after diagnosis, blood IgG was again elevated (18.0 g/l) and precipitins were positive for parakeet and pigeon but the CT scan was still normal. At the last follow-up, 4.7 years after diagnosis, lung function tests were normal.

## Case 3

A 5-year and 4-month-old boy presented with a history of dry cough, especially during the night, dyspnoea, restlessness and activity impairment lasting for 3 weeks. Treatment with inhaled salbutamol, budesonide and cromoglycate resulted only in partial relief of symptoms. Clarithromycine was added for 2 weeks because of poor improvement. He was first admitted to a primary care hospital because of hypoxaemia (88% oxygen saturation in room air). A chest X-ray film and CT scan revealed a diffuse bilateral reticulonodular infiltrate (Fig. 2). The boy was then transferred to our hospital for further investigations.

Environmental history revealed that the family lived close to a pigeon house and that the child used to play nearby. Family history was positive for atopy. On admission, the patient was tachypnoeic (40 breaths/min), hypoxaemic (required 0.5 l/min oxygen to reach 98% saturation), had slight sternal retraction, intermittent grunting and mild inspiratory basal crackles. Laboratory screening and BAL were performed as in Cases 1 and 2. Blood precipitins were positive for pigeon and canary and markedly positive for parakeet. No lung biopsy was performed at this stage because of strong serological and clinical evidence of BBD.

Avoidance of the allergen by moving to a new house and oral prednisolone therapy (2 mg/kg/day) together with inhaled budesonide (200 μg twice a day) were rapidly followed by resolution of oxygen dependence. Oral corticotherapy was gradually decreased after 10 days (1 mg/kg/day) and stopped after 11 weeks. Inhaled budesonide was continued. Cough disappeared after 1 month of therapy and dyspnoea on exercise improved. A moderate restrictive syndrome was found on lung function tests (vital capacity 59% of predicted value). Measurement of diffusion lung capacity for carbon monoxide (DLCO) was impossible to perform due to lack of collaboration. The interstitial infiltrate diminished on a CT scan, but did not completely disappear.

Six weeks after prednisolone withdrawal, clinical worsening was observed with recurrent tachypnoea and increasing dyspnoea on exercise necessitating readmission to hospital. Quantification of blood immunoglobulins was still normal and precipitins were

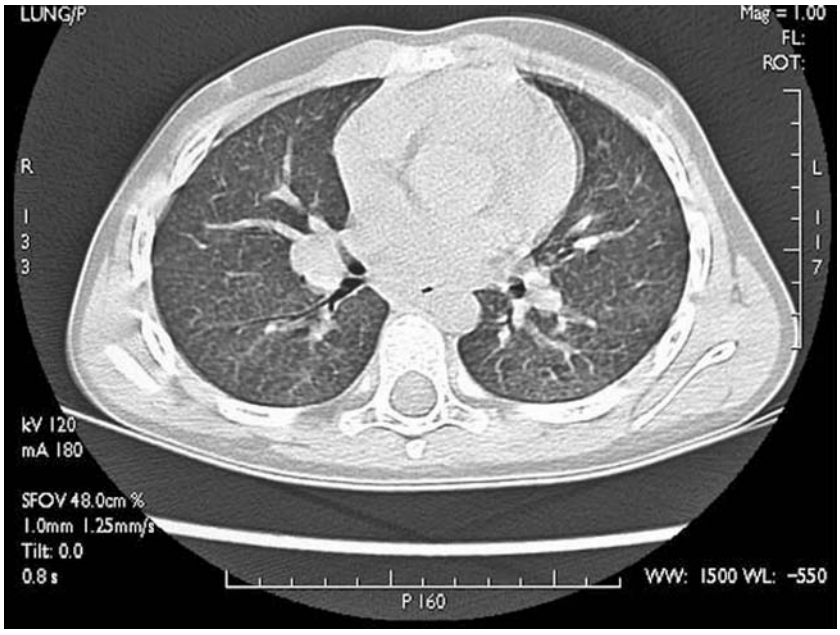


**Fig. 2** High-resolution CT scan showing diffuse ground-glass attenuation with fine nodules (Case 3)

unchanged. Blood lymphocyte typisation and stimulation test were normal. A chest CT scan was unchanged. The BAL cell count was still elevated ($50\times10^4$ cells/ml) with persistent lymphocytosis (lymphocytes 29%, macrophages 24%, granulocytes 46%) and reduced CD4/CD8 ratio (0,14). All BAL cultures remained negative. Relapse was suspected and it was decided to perform a lung biopsy by VAT (in lingula and left lower lobe) to substantiate the diagnosis of BBD before starting long-term steroid therapy. An important interstitial and peribronchial lymphoid infiltrate, granuloma and foci of obliterative bronchiolitis confirmed the diagnosis (Fig. 3). Here again, staining for fungi and acid-fast bacilli was negative. Prednisolone was reintroduced at 1 mg/kg per day showing a rapid clinical improvement. Nearly 2 months later, the boy was asymptomatic and the prednisolone dose was slowly reduced and stopped after 1 year. Nineteen months after the relapse, the cough reappeared but a CT scan was entirely normal at this time. This cough disappeared suddenly after a few months and a psychogenic origin was suspected. At the last follow-up, 4 years after diagnosis, the boy was asymptomatic and lung function tests were normal.

## Methods

Clinical history and signs when the diagnosis was made are summarised in Table 1. Laboratory tests and bird exposure were analysed in all patients (Table 1 and Table 2). The three patients were screened for the following tests: blood cell count, CRP, immunoglobulins

**Table 1** Clinical features of the three cases

| | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| Age (years) | 6.5 | 4.4 | 5.4 |
| Symptoms: | | | |
| Dry cough > 1 month | + | + | + |
| Dyspnoea on exercise | - | + | + |
| Dyspnoea at rest | - | - | + |
| Fever (> 38.5°C) | + | - | - |
| Signs: | | | |
| Tachypnoea | + | - | + |
| Hypoxia | + | - | + |
| Crackles | + | - | + |
| Type of exposure | Holiday on a farm | Holiday on a farm | Living next to a pigeon house |

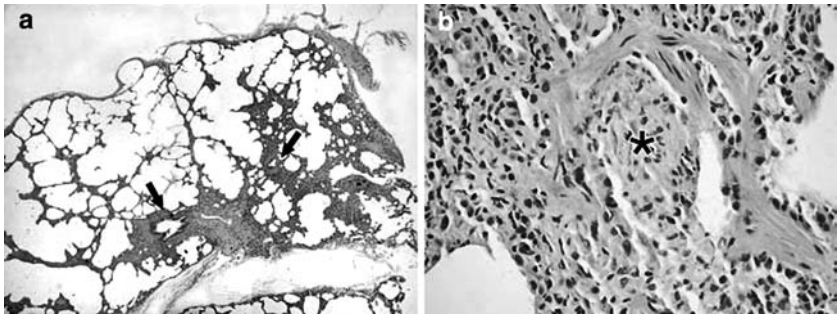


**Fig. 3** **a** Histology of lung biopsy of Case 3 at relapse showing interstitial pneumonia with peribronchiolar predominancy ( *arrows*, original magnification ×20) and **b** foci of obliterative bronchiolitis (*asterisk*, original magnification ×200)

**Table 2** Results of laboratory investigationsat diagnosis

| | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| IgG (g/l) | 13.19 (increased) | 17.4 (increased) | 6.93 (normal) |
| Precipitins: | | | |
| Parakeet | + + | + | + + |
| Pigeon | + | - | + |
| Canary | - | - | + |
| Hen | + | Not available | Not available |
| *Aspergillus fumigatus* | + | + | - |
| BAL: | | | |
| Cell number ($\times 10^4$/ml) | 45 (normal) | 5 (normal) | 75 (increased) |
| Lymphocytes (%) | 62 (increased) | 44 (increased) | 77 (increased) |
| CD4/CD8 ratio | 0.56 (low) | 1.62 (normal) | 0.35 (low) |

**Table 3** Results of lung function tests at the latest clinical evaluation

| Lung function tests (% of theoretical value) | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| Time interval since diagnosis (years) | 5.7 | 4.7 | 3.9 |
| Time interval since oral steroid stop (years) | 5.5 | 4.4 | 2.6 |
| Total pulmonary capacity | 100 | 93 | 97 |
| FVC | 92 | 93 | 92 |
| DLCO (corrected for Hb) | 94 | 76 | 87 |
| FEV1 | 94 | 98 | 92 |
| FEV1/FVC | 100 | 107 | 100 |

(Ig) and precipitins quantification, intradermal tuberculin screening, standard chest X-ray, chest CT scan, bronchoscopy with BAL, lung biopsy performed by VAT and lung function tests. Avian precipitins were determined by immunoelectrophoresis using birds' serum as antigen, according to the method described by Reynaud et al. [17]. Cell number in BAL was considered normal when the value was included between 5 and $50\times10^4$ cells/ml; indeed, normal values depend on the age of the child and a great variation has been reported between different studies [7]. Lymphocytosis was considered when over 10% of total cell number [7].

Diagnosis of BBD was assessed on clinical presentation, positive history of bird exposure, positive avian precipitins, lymphocytosis in BAL fluid and lung biopsy. Response to treatment and evolution of symptoms and laboratory tests were studied. Lung function tests at the last clinical evaluation are summarised in Table 3. Duration of follow-up for the three patients was 3.9, 4.7 and 5.7 years respectively.

## Discussion

We report here three cases of BBD in very young children in whom the diagnosis was made early after the onset of symptoms. Interestingly, no other diagnosis of HP was suspected during this period of time in older children.

The diagnosis of BBD was based on clinical presentation, bird exposure, radiological findings, analysis of BAL fluid, positive blood precipitins and good response to treatment. The three patients had a diffuse reticulomicronodular infiltrate with ground-glass shadowing on the chest X-ray film and CT scan. BAL fluid displayed lymphocytosis in all three children and two of them had an increased cell count and reduced CD4/CD8 ratio. Typical elevated cellullarity with predominant lymphocytes and reduced CD4/CD8 ratio lower than 1 have been classically reported in adults with HP but not always in children [15]. Total blood IgG were elevated in two of the three patients, reflecting B- and T-lymphocyte activation. Positive avian precipitins were found in all three patients. Two of them (cases 1 and 2) also had positive precipitins against non-avian antigens like *Aspergillus fumigatus*. This can be explained by a non-specific cross-reaction phenomenon. Interestingly, positive precipitins can also be found in normal individuals; therefore, in this case, a positive precipitin result does not confirm HP but reveals a sensitisation [12,13]. BBD is diagnosed if the clinical presentation and complementary investigations corroborate the disease. In our three patients, birds as causal allergens of HP were confirmed by the fact that symptoms were associated with bird exposure and that they rapidly improved under steroid treatment. Moreover, avoidance of birds was followed by a quick resolution of symptoms. It is interesting to note that two out of the three children were exposed for a short period of time (holidays), suggesting that long exposure is not necessary to develop symptoms. Moreover, diagnosis delay was very short (between 1.5 and 2.5 months).

Lung biopsy is not considered mandatory by everyone to prove the diagnosis of HP; however, because of the young and atypical age at clinical presentation of the disease, we decided to perform it to confirm the diagnosis and to exclude other diseases [3]. VAT is now a well mastered technique which allows a minimal discomfort compared to open lung biopsy performed by thoracotomy. It was fully warranted in case 3 because of relapse after corticoid withdrawal to confirm the diagnosis before starting corticotherapy again. Lung biopsy confirmed the histological triad: (1) interstitial pneumonia with predominance of lymphocytes and peribronchiolar distribution, (2) non-necrotising granuloma (not constant [5]) and (3) foci of obliterative bronchiolitis (not constant [5]).

Because of the young age of our patients, we could not perform lung function tests at the disease onset to demonstrate the typical restrictive syndrome or the decrease in DLCO.

BBD is usually described in adults. It has been reported that the prevalence of pigeon breeder's disease among pigeon breeders is between 0.5% and 22% [18]. Diagnosis is often made late when irreversible fibrosis is already present. This entity is very rare in children [9]

and less frequent than in adults [18]. There are no epidemiological data concerning BBD in this age group; however, for HP, the average age at clinical presentation in childhood is 10 years [8]. In the literature, the youngest described patient suffering from HP without a more specific diagnosis was 8 months old [6] and there is only one reported case of pigeon breeder's disease in a 3-year and 10-month-old boy [20]. Pigeons are the most common allergens causing HP in children followed by parakeet, canaries and other birds [8]. Sources of allergens are bird excreta, blood and feathers [10,14].

It is likely that HP only develops in susceptible individuals. When exposed to a given antigen, only susceptible persons (up to 50%) become sensitised, and only a few of them (5% to 15% of all the exposed individuals) will develop the disease [8]. Familial cases of BBD have also been described [1,8], suggesting that genetic factors might play a role in the type and extent of immune response leading to the disease. Indeed, polymorphism of the 5' promoter region of the tumour necrosis factor α gene has been reported to be related to individual susceptibility to pigeon breeder's disease [2]. It has also been suggested that adjuvant factors such as non specific lung inflammation during viral infection may play a role in the development of the illness [1,21].

On follow-up, case 1 had a simple evolution with rapid normalisation of clinical signs and laboratory investigations. Case 2 remained asymptomatic but still presented signs of disease activity on laboratory parameters. Case 3 had a clinical relapse after steroid therapy withdrawal necessitating steroids for 1 year. Evolution thereafter was good. In our three patients, the very short delay between onset of symptoms and diagnosis (1.5 to 2.5 months) may probably explain the good and rapid evolution without development of lung fibrosis. On follow-up, all had a normal CT scan and normal lung function tests. Cases 2 and 3 showed persistently elevated IgG levels and positive precipitins 2 to 4 years after the initial diagnosis of HP, although no pulmonary disease activity could be detected by clinical signs, CT scan and lung function tests. This suggests that overproduction of IgG in response to allergen can persist for a long time after allergen avoidance. Early recognition of BBD allows rapid allergen avoidance and introduction of steroid treatment that probably prevent the development of lung fibrosis. Evolution of HP in children when treated adequately is usually favourable [8]. However it is not yet clear whether the young age of the children is also a good prognostic factor and this should be taken into account in further studies. It has been suggested that children suffering from interstitial lung disease might respond more favourably to treatment and this could be explained by the nature of wound healing after primary injury during lung growth and development [4].

Treatment consists of total and definitive allergen avoidance and systemic corticotherapy. Allergen exclusion alone has not proven to be efficient in the literature. Corticosteroids are generally administrated in HP because they are effective in improving symptoms, lung function and radiological abnormalities [11].

The clinical relapse of case 3 could be explained by a more intense and prolonged exposition to allergen, since the boy lived next to a pigeon house, whereas cases 1 and 2 were exposed only shortly during holiday periods. This prolonged exposition could be responsible for a more active form of the disease, necessitating a longer course of corticotherapy.

Based on our experience with these 3 patients, we think that early recognition of the disease and rapid introduction of treatment can probably avoid evolution into irreversible lung fibrosis.

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## References

1. Bourke SJ, Dalphin JC, Boyd G, McSharry C, Baldwin CI, Calvert JE (2001) Hypersensitivity pneumonitis: current concepts. Eur Respir J 18[Suppl. 32]: 81–92
2. Camarena A, Juarez A, Meija M (2001) Major histocompatibility complex and tumor necrosis factor-α polymorphisms in pigeon breeder's disease. Am J Respir Crit Care Med 163: 1528–1533
3. Clement A (2004) Task Force on chronic interstitial lung disease in immunocompetent children. Eur Respir J 24: 686–697
4. Clement A, Henrion-Caude A, Fauroux B (2004) The pathogenesis of interstitial lung diseases in children. Paediatr Respir Rev 5: 94–97
5. Delacourt C (1999) Alvéolite allergique extrinsèque. Arch Pédiatr 6[Suppl. 1]: 83–86
6. Eisenberg JD, Montanero A, Lee RG (1992) Hypersensitivity pneumonitis in an infant. Pediatr Pulmonol 12: 186–190
7. ESR Task force on bronchoalveolar lavage in children (2000) Bronchoalveolar lavage in children. Eur Respir J 15: 217–231
8. Fan LL (2002) Hypersensitivity pneumonitis in children. Curr Opin Pediatr 14: 323–326
9. Grech V, Vella C, Lenicker H (2000) Pigeon breeder's lung in childhood: varied clinical picture at presentation. Pediatr Pulmonol 30: 145–148
10. Grimfeld A, Gaultier Cl, Baculard A, Le Moing G, Tournier G, Gerbeaux J (1981) Pneumopathies par hypersensibilité chez l'enfant. A propos de 5 cas. Sem Hop Paris 57: 1267–1272
11. Leland LF (2002) Hypersensitivity pneumonitis in children. Curr Opin Pediatr 14: 323–326
12. Levenson T, Patterson R (1996) Chronic cough in a child. Ann Allergy Asthma Immunol 76: 311–316
13. Lindemann H, Keller F, Velcovsky HG (1982) Exogene allergische Alveolitis im Kindesalter. Ergeb Inn Med Kinderheilkd 50: 1–30
14. Patel AM, Ryu JH, Reed CE (2001) Hypersensitivity pneumonitis: current concepts and future questions. J Allergy Clin Immunol 108: 661–670
15. Ratjen F, Costabel U, Griese M, Paul K (2003) Bronchoalveolar lavage fluid findings in children with hypersensitivity pneumonitis. Eur Respir J 21: 144–148
16. Reed CE, Sosman A, Barbee A (1965) Pigeon-breeder's lung: a newly observed interstitial pulmonary disease. JAMA 193: 81–85
17. Reynaud C, Slosman DO, Polla BS (1990) Precipitins in bird breeder's disease: how useful are they? Eur Respir J 3: 1155–1161
18. Severien C, Artlich A, Jonas S (1998) Die Taubenzüchterlunge im Kindesalter. Klin Pädiatr 210: 413–417

19. Stiehm ER, Reed CE, Tooley WH (1967) Pigeon breeder's lung in children. Pediatrics 39: 904–915
20. Wolf SJ, Stillerman A, Weinberger M, Smith W (1987) Chronic interstitial pneumonitis in a 3-year-old child with hypersensitivity to dove antigens. Pediatrics 79: 1027–1028
21. Zacharisen MC, Schlueter DP, Kurup VP, Fink JN (2002) The long-term outcome in acute, subacute and chronic forms of pigeon's breeder's disease hypersensitivity pneumonitis. Ann Allergy Asthma Immunol 88: 175–182