Chronic Eosinophilic Pneumonia in a One-Year-Old Child*

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Although the literature well describes chronic eosinophilic pneumonia in the adult, there are no reports of this entity in the pediatric age group. We describe a child with chronic eosinophilic pneumonia, emphasizing the specific radiologic features, common conditions in the differential diagnosis, and dramatic response to corticosteroid administration, which is a unique feature of this entity.

Since the original description by Loeffler, pulmonary parenchymal disease associated with either blood or tissue eosinophilia has been given a multitude of names constituting a group of ill defined disorders. Crofton et al2 offered a convenient classification of these disorders into (1) simple pulmonary eosinophilic Loeffler’s syndrome, (2) pulmonary eosinophilia with periarteritis nodosa, (3) pulmonary eosinophilia with asthma, (4) tropical pulmonary eosinophilia, and (5) prolonged pulmonary eosinophilia although a proper understanding has been hampered by the terminology.

Carrington and associates3 described nine women with progressive pulmonary infiltrates, inconstant eosinophilia of peripheral blood and pathologic changes consisting of eosinophilic exudation in to the alveoli and interstitium. All the patients showed a dramatic response to corticosteroid therapy. The condition was categorized as chronic eosinophilic pneumonia.

To our knowledge, chronic eosinophilic pneumonia has not been described in children; hence this communication is a report of this entity in a child and a brief review of the literature.

CASE REPORT

A one-year-old black boy was admitted to Downstate Medical Center in March, 1972 with a history of persistent cough and low grade fever. The patient had been hospitalized elsewhere on three separate occasions during the past six months prior to admission with the same complaints. Chest films from the previous hospitalizations showed evidence of mediastinal adenopathy with a large para-tracheal node on the right, as well as prominent hilar nodes. In addition, there were persistent, progressive pulmonary infiltrates throughout.

Figure 1. Frontal chest radiograph demonstrates a diffuse infiltrative pattern especially prominent in the right upper lobe with bilateral hilar adenopathy and a large right paratracheal lymph node.
both lungs with a peripheral distribution, especially in the
right lung.
At the time of admission to our hospital, his temperature
was 37.7°C (100°F). Physical examination revealed râles
and rhonchi over the right lung field. Nose, throat and blood
cultures on several occasions gave negative findings. Repeat
skin tests for tuberculosis, atypical mycobacterial disease and
fungal infections were negative. Kveim test for sarcoidosis
was negative. The patient was treated with ampicillin and
methylene with no response.
The white blood cell count and the differential count on
several occasions during the course of the illness showed
intermittent peripheral eosinophilia.
Repeat chest films revealed a large right paratracheal
node, as well as bilateral hilar and subcarinal adenopathy.
Both lungs showed infiltrates, more on the right than the left,
and there was slight elevation of the minor fissure with some
thickening (Fig 1). Esophagram showed normal findings.
Two weeks following admission, the patient had shown no
response to therapy and open lung biopsy was performed. At
thoracotomy, the upper lobe of the right lung showed diffuse
nodular infiltrates with involvement to a lesser degree of the
middle and lower lobes. A lung biopsy was performed and
the right paratracheal node was resected. Histopathologic
examination of the lung tissue demonstrated acute and
chronic pneumonia, interstitial in nature with numerous
eosinophils (Fig 2). There was no evidence of bacterial,
fungal, tuberculous or parasitic infections. The lymph node
showed follicular hyperplasia. The patient continued to have
clinical evidence of mild respiratory distress with râles and
rhonchi over lung fields, and prednisone therapy was insti-
tuted (2 mg/kg/day). Within one week of treatment, the
respiratory distress cleared and there was complete cessation
of auscultatory findings. At the same time, improvement was
noted on the chest roentgenograms, with a decrease in
infiltration in the right upper lobe and shrinkage of the
subcarinal nodes. Eight months after institution of prednisone
therapy, there was considerable resolution of the infiltrates in
the lungs and the nodal enlargements had disappeared (Fig
3). At this time, prednisone therapy was tapered and finally
discontinued in March, 1973. A six-month follow up showed
an asymptomatic patient with an essentially normal chest x-
ray appearance.

**DISCUSSION**

The features of chronic eosinophilic pneumonia have
been well described by Carrington and associates. In
our patient, the diagnosis of chronic eosinophilic pneu-
monia was established by the presence of chronic pro-
gressive pulmonary infiltrates, intermittent peripheral
eosinophilia and the histopathologic evidence of inter-
stitial and alveolar infiltrates with eosinophils. The excel-
ent response to corticosteroids supported the diagnosis.

Many authors have included in Loeffler's syndrome
diseases which do not fit the original criteria set down by
Loeffler. The use of various different names has added
confusion to the picture. Crofton's classification helps to
correlate roentgenologic with clinical findings and fre-
cently allows a specific diagnosis.

In simple pulmonary eosinophilia, the radiologic
changes are described as migratory, peripheral homo-
genous densities. These, in association with peripheral
eosinophilia and minimal respiratory symptoms, show
spontaneous resolution within a few weeks. The etiology
of this syndrome is thought to be an allergic reaction to
various agents including parasitic nematodes and drugs
such as para-aminosalicylic acid and nitrofurantoin. There
was no evidence to support this diagnosis in our patient.
History and the clinical course in our patient ruled out the possibility of tropical eosinophilia.
Peripheral eosinophilia and transitory pulmonary infiltrates have been described in asthmatics by Ford.
Though periarteritis nodosa has been reported to present with a combination of pulmonary infiltrates and
peripheral eosinophilia, it was not a consideration in our
patient. Prolonged pulmonary eosinophilia with infiltrates has been described in a wide spectrum of condi-
tions including farmer's lung, sarcoidosis, psittacosis,
primary atypical pneumonia and eosinophilic leukemia.
These conditions were very unlikely in our patient.

Prolonged pulmonary eosinophilia with characteristic

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**FIGURE 2.** Histopathology of lung biopsy showing evidence
of acute and chronic inflammation, interstitial in nature, with
numerous eosinophils.

**FIGURE 3.** Frontal chest roentgenogram eight months after
prednisone therapy reveals clearing of the pulmonary infiltrates except in the right upper lobe, with a decrease in the
adenopathy.
clinical and roentgenographic features of unknown etiology is categorized as chronic eosinophilic pneumonia. Radiographically, the characteristics of this entity have been well described in adults and include the finding of dense pulmonary infiltrates which progress with time and are arranged in an unusual pattern in that they are found peripherally rather than centrally. Carrington and associates refer to this as a "photographic negative" of the shadow seen in pulmonary edema. In our patient, the progressive nature of this disease was well documented over an eight-month period. At no time was there evidence of clearing on antibiotic therapy prior to the institution of steroid treatment. It was significant to note the dramatic response to corticosteroid therapy and no recurrence of the disease process after cessation of steroid therapy.

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Control of Hemorrhage in Emergency Pulmonary Resection for Massive Hemoptysis

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Emergency pulmonary resection for hemoptysis during an episode of massive intrabronchial bleeding requires protection of the contralateral lung from aspiration of blood. We describe a method of selective unilateral ventilation applied to 15 patients, without mortality attributable to this factor.

In pulmonary hemorrhage the rate of bleeding into the tracheobronchial tree poses a greater threat to life than the total amount of blood loss. Patients tend to drown in their own blood and asphyxiate rather than exsanguinate. At our institution we operate on all patients who expectorate a minimum of 600 ml of blood in 24 hours or less and are able to tolerate thoracotomy. This aggressive approach resulted in reduction of mortality from massive hemoptysis from over 75 percent in the patients managed conservatively to 18 percent in the patients treated surgically. In the past ten years 64 patients underwent pulmonary resection for massive hemoptysis. In 22 patients hemoptysis persisted after the initial 600 ml of blood had been expectorated, and emergency pulmonary resection had to be performed during an episode of massive intrabronchial bleeding. Early in our experience we utilized Carlens double-lumen tubes to protect the nonbleeding lung from aspiration of blood. Four of seven patients so managed died as a result of massive aspiration of blood during operation. In more recent years, a technique of endobronchial intubation with use of a balloon catheter blocker proved more effective in protecting the contralateral lung from blood

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Figure 1. Right-sided bleeding. (A) Cuffed tube in left main bronchus protects left lung from spillage of blood in early phase of operation. (B) Bronchus of bleeding lobe has been cross clamped and cuffed tube is withdrawn into trachea.