PULMONARY EOSINOPHILIA

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Case History

An alert and intelligent ten year old girl, who had never left Malta, was referred to the Chest Clinic in September 1961 with the diagnosis of "Consolidation right upper lobe? Primary Complex" *(Fig. 1)*. The Mantoux Test was negative on two occasions at one month's interval, and before coming to the clinic she was given Streptomycin and Isoniazid and a temperature which she had when first seen took three weeks to settle down.

She had had hay fever and bronchial asthma for some years, but gave no history of other allergic manifestations, other illnesses, or of known contact with tuberculosis. There was no family history of allergy, and her asthma had no seasonal incidence, was usually worse at night, but was not provoked by any special factor.

On examination, nothing of note could be detected, except for some expiratory ronchi over both lungs. She had no enlarged glands, and the liver and spleen were not enlarged. There was no history of diarrhoea, and she never had worms in the stools.

Her urine contained no proteins or sugar, the Serum Agglutination Tests were negative, and the sputum revealed some eosinophils, a mixed flora, but no acid fast bacilli. A blood count and picture showed an eosinophilia of 10% of a total white count of 9,100 p/c. mm; there was no anaemia, and the E.S.R. (Westergren) was 15mm/1st hour.

The Total Serum Proteins were 8.62 g/100ml, albumin 4.5 g/100 ml. The electrophoretic pattern was that of a chronic inflammatory process: a significant increase in gamma globulins, and an absolute increase in the albumin, alpha—1 and alpha—2 globulins. The Tuberculin Skin Test was repeated, using the Heaf Technique, and this again turned out negative, and the result of the gastric contents for tubercle bacilli, now available, was also negative.

An X-Ray of the chest taken at this time, that is, one month after the first one, showed that the shadow in the right apex had cleared up almost completely *(Fig. 2)*.

At the beginning of January 1962, she developed an itchy papulo-purpuric eruption mainly in the lower limbs, with some fever, but no abdominal colic or joint pains. A blood count and picture showed nothing of note (eosinophils 6%), but a chest X-Ray showed some mottling this time at the base of the right lung and at the left mid-zone *(Fig. 3)*.

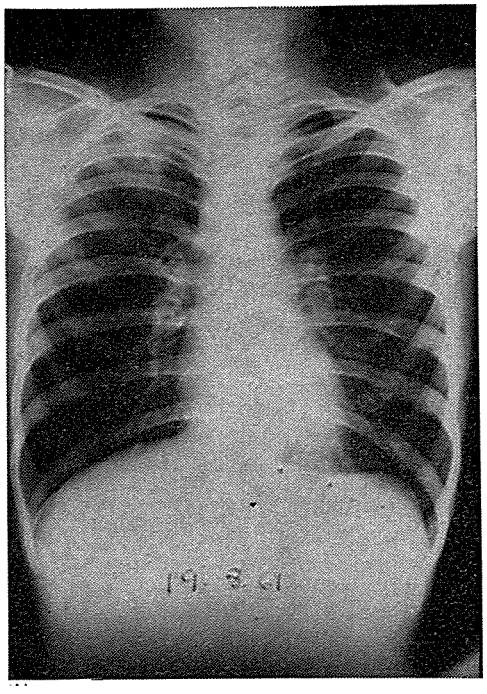

Fig. 1

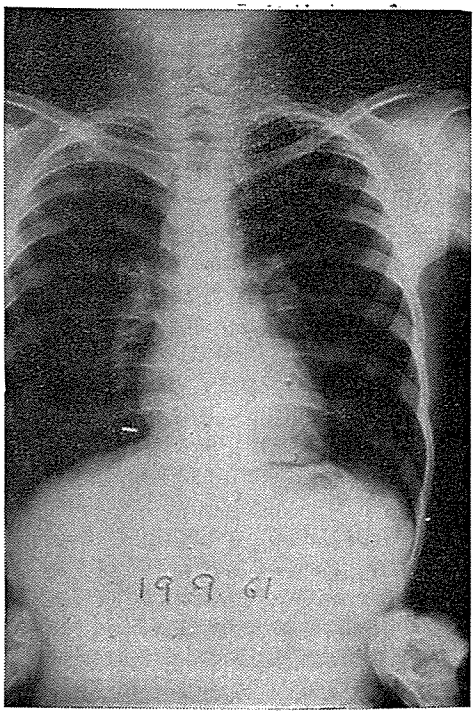

Fig. 2

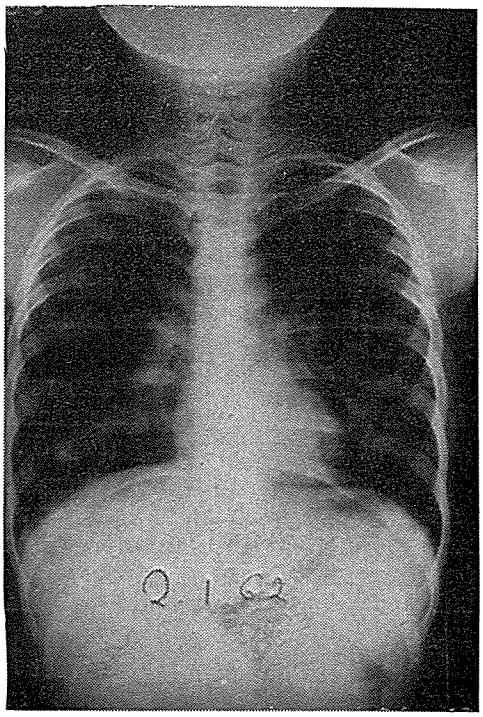

Fig. 3

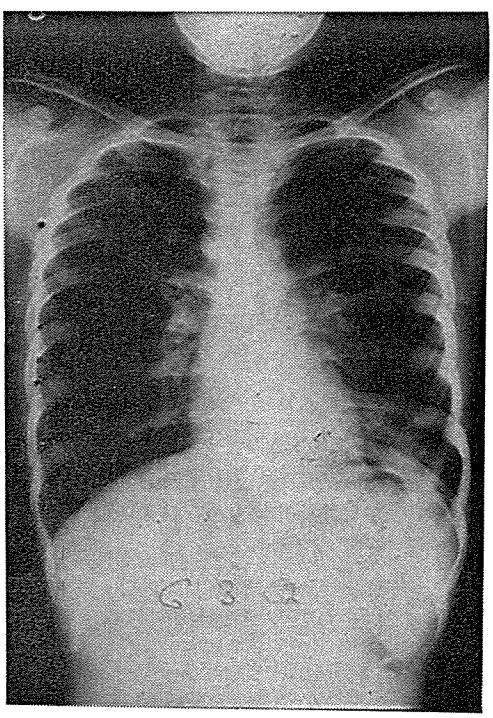

Fig. 4

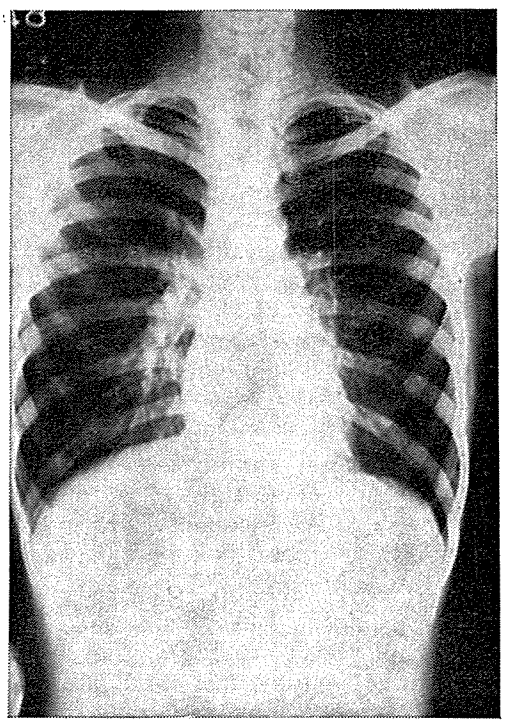

Fig. 5

As the diagnosis by now was getting reasonably clear, she was told to continue with anti-spasmodics as her asthma had returned, was reassured and told to call again for follow-up.

Her next visit was at the beginning of March 1962. She then had had a two days low grade fever with some cough. The chest X-Ray now showed an area of "consolidation" at the left base *(Fig. 4)*. No antibiotics were given and she was told to return after four weeks.

When the chest X-Ray was repeated a month later, sure enough the shadow at the left base had cleared up *(Fig. 5)*.

She continued to have asthmatic episodes, but the chest X-Ray remained clear. In October 1964, an area of "patchy consolidation" appeared in the right midzone and took three months to clear up completely.

She was last seen in August 1966, when, apart from a thin "fibrotic" strand at the site of the original lesion in the right apex, the lungs were clear.

Discussion

The salient positive features in this patient were: a history of asthma, the presence of other allergic manifestations, eosinophilia in the blood and in the sputum, and recurrent pulmonary shadows. On the negative side, there was a repeatedly negative Tuberculin Skin Test, no acid fast bacilli in the sputum or gastric contents, no history of diarrhoea or worm infestation, no exposure to drugs before her present illness, and she had never been away from Malta.

The positive findings, especially the X-Ray changes, cannot be explained by the presence of straight-forward asthma, and the negative evidence rules out other possibilities, especially tuberculosis.

Ordinary bronchial asthma can certainly produce X-Ray changes in the chest, but these would be the result of obstructive emphysema and atelectasis, which may be temporary or permanent, depending on the type, severity and chroinicity of the seizures and on the age of the patient. Permanent pulmonary changes in the chronic asthmatic would show hyperillumination of the lung fields, with prominent hilar shadows, and sometimes bullous formation in the lung parenchyma and blebs at the periphery. Spontaneous pneumothorax may occur.

One form of pulmonary hypersensitivity which fits in exactly the case described are recurrent and prolonged pulmonary infiltrations, with eosinophilia and asthma, a variant of the Pulmonary Eosinophilias.

In 1927 Peshkin, without mentioning the eosinophilia, noticed "subacute pulmonary infiltrations" in asthmatic children (Rubin E. H. and Rubin M., 1961), but the syndrome was first brought to general attention by Loeffler in 1932 (Loeffler, W., 1932). However, in "Loeffler's Syndrome", symptoms are absent or few and last less than eight weeks, bronchial asthma is not a feature, though tightness in the chest has been occasionally recorded, and the pulmonary infiltrations are fleeting and transient and disappear within a period of six to twelve days.

Since Loeffler's original description, similar syndromes have been reported (Crofton et al., 1952) where the illness has been varied, longer and more severe, and did not fulfil the criteria set down by him (Loeffler, W., 1936).

Except during the attacks of asthma, the physical signs in the lungs may be minimal. There is usually some cough, headache, catarrh and a fever. The circulating eosinophils may form as much as 70% of the total white count, but in some cases they may be within normal limits, and the degree of eosinophilia does not appear to be related to the extent of the pulmonary involvement. Eosinophils may be present in the sputum, nasal discharge and other body fluids.

Histological examination of the pulmonary lesions at postmortem (Broch A, 1943, quoted by Crofton *et al.* 1952; Bayley *et al.* 1945), after pneumonectomy (Buckles, M. G., and Lawless, E. C., 1950), or through needle biopsy (Christoforidis, A. J., and Molnar, W., 1960) has led to a

better understanding of the pathological changes. The areas of "infiltration" consist of patches of non-bacterial eosinophilic pneumonia. There is stuffing of the alveoli with lymphocytes, plasma cells and eosinophils, the capillaries are distended, and the bronchi are intensely infiltrated with neutrophils and eosinophils. A variable degree of necrotizing vasculitis, especially in the small arteries, may be present and this determines the intensity and the permanence of tissue damage. Exudation may also occur in any of the serous cavities.

Differential diagnosis

The pulmonary "infiltrations" may be mistaken for tuberculosis, especially in adults and in children with a positive Tuberculin Skin Test. The difficulty is more likely to arise when the pulmonary shadows are bilateral and stay in one part of the lung for a long time. The possible absence of eosinophilia would make the differential diagnosis more difficult. Many of the patients subsequently proved to have Pulmonary Eosinophilia were originally diagnosed as having tuberculosis (Frimodt-Moller, C., and Barton, R. M., 1940, Karan, A. A., and Singer, E., 1942, Crofton, J. W., Livingstone, J. L., Oswald, N. C., Roberts, A. T. M., 1952).

The "pneumonias", especially the so-called "atypical", may be hard to exclude at the onset of the illness, but the response to drugs in some and the subsequent behaviour of the pulmonary shadows in others make the diagnosis clear.

Drug-induced eosinophilic infiltration of the lung may create a problem, but awareness of its possible occurrence and careful questioning will help. Removal of the offending drug will be followed by lasting recovery.

Finally, Tropical Pulmonary Eosinophilia has to be seriously considered, as the chronic type would give the same clinical and radiological sequence of events as the "idiopathic" variety. It is mainly found in India and Ceylon, but sporadic cases have been encountered in other countries. As far as is known it has not been observed in Malta, though the writer has seen it locally in a visiting Indian sailor. The disease is caused by parasites, i.e. spirochaetes, trichinella, amoeba, liver-fluke and filariae. The presence of parasites in the stools or sputum, positive cold agglutinins and serological tests, and the favourable response to diethylcarbamazine (Danaraj, T. J., 1958; Baker et al., 1959) will clinch the diagnosis.

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