

(From the Military Hospital, Medical Department, Copenhagen.
Chief: Kaj Kjerulf-Jensen, M.D.)

PULMONARY EOSINOPHILIC SYNDROME

Two Cases Treated with Prednisone [Meticorten]

By

ERIK ANDERSSON,
Copenhagen

The syndrome of pulmonary infiltration with eosinophilia constitutes a motley group of widely different etiology. An attempt at classifying the individual varieties according to cause may be as follows:

- | | | |
|---------------------------------------|-----------|---|
| A. Known
etiology | Parasites | Schistosoma mansoni (1)
Fasciola hepatica (2)
Filaria (3)
Ancylostoma (4)
Strongyloides (5) |
| | Fungi | Coccidioides (6)
Histoplasma (7, 8) |
| | Bacteria | Brucella (9)
Tubercle bacillus (10) |
| | Virus? | |
| | | |
| B. Allergy | | Bronchial asthma |
| | | Löffler's syndrome (11, 12, 13, 14, 15) |
| | | Pulmonary infiltrate with eosinophilia, prolonged course (16) |
| | | Periarteritis nodosa (17) |
| C. Tropical eosinophilia (18, 19, 20) | | |

The first group comprises the syndrome caused by parasites and infection with bacteria or viruses. This group will probably become the largest and eventually the only one, as some time in the future the other groups will no doubt be incorporated therein, Groups B and C being assumed to be due either to bacteria or viruses. Only, it has so far been impossible to arrive at the specific etiology. It is possible, however, that in bronchial asthma the eosinophilia is not caused by infections, but by the substance to which the organism is allergic. Patients with hay fever, for instance, have shown a marked increase in the eosinophilic cells in the blood in exposure experiments with mixed pollens (21).

Below two cases of pulmonary infiltrate with eosinophilia with a prolonged course are reported.

Case I. A soldier, aged 20, in civil life a joiner. Except for ordinary children's diseases the patient had previously been in good health. He was admitted to the Military Hospital in Copenhagen, as routine chest radiography had revealed an infiltrate of the right lung. Prior to the examination the patient had had a cold for about two weeks, said to have been afebrile, with some unproductive cough. On admission the patient was running low-grade fever, and X-rays showed right-sided pleurisy with some density at the phrenocardial angle. Blood study showed moderately elevated E.S.R., leucocytosis, and eosinophilia about 2,000 per mm³. The patient was treated with penicillin and streptomycin for two weeks without any effect. Control X-rays after this period showed a denser shadow and furthermore a slight shadow basally on the left side. Treated with bed rest for a month. Renewed X-ray examination one month later showed the shadow on the right to be decreasing, but now there was a marked basal shadow on the left with an obliteration of the costophrenic angle. Two month later the right-sided pleurisy had almost disappeared, whereas on the left it was stationary. The low-grade fever, elevated, E.S.R., leucocytosis, and eosinophilia persisted. Sternal puncture revealed nothing abnormally but eosinophilia. Bronchoscopy did not reveal any abnormality. Microscopical examination of bronchial secretion: No tumor cells or tubercle bacilli. Culture of gastric washing and pleural fluid: negative for TB. Thorough allergological study failed to reveal any cutaneous hypersensitivity, and the history gave no clue in this respect. At this juncture the condition was believed to represent a pulmonary eosinophilic syndrome which did not show signs of improvement. It was decided, therefore, to treat the patient with prednisone. During the first two weeks prednisone was administered in a daily dose of 30 mg, in the course of the next 10 days in decreasing dosage and thereafter discontinued. The temperature and blood

findings returned to normal within about two weeks, and the X-ray changes gradually improved in the course of 4 weeks. Upon follow-up examination 3 months after discontinuance of treatment with prednisone, blood examinations and X-ray findings were still normal, and the patient had no complaints whatever. He may, therefore, be considered cured by the prednisone therapy.

Case II. A soldier, aged 20, in civil life a clerk. Previously, he had been in perfect health apart from ordinary children's diseases. He was transferred from the infirmary of the engineer barracks to the Military Hospital, Copenhagen, with right-sided pleurisy. On admission he had a low-grade fever with elevated E.S.R., leucocytosis, and eosinophilia of about 1,500 per mm³. Radiography of the chest revealed an infiltrate basally of the right lung. During treatment with rest, he improved and the temperature returned to normal. After one month control X-rays showed normal appearances, but the elevation of the E.S.R., the leucocytosis, and the eosinophilia persisted. The patient was therefore kept in bed, and two weeks later the low-grade fever returned, and X-rays showed an infiltrate on the right as well as a basal pleurisy with an obliteration of the costophrenic angle. Cultures of gastric washing and sputum were negative for TB. Allergological study with cutaneous tests showed no signs of allergy. Now, treatment with prednisone was started, 20 mg for 8 days and then, in decreasing doses, for another 8 days. The temperature and blood findings returned to normal, and when the treatment was discontinued X-rays of the lungs showed normal appearances. Follow-up examination one month after the treatment had been discontinued showed normal findings, both in the lungs and in the blood. Therefore, this patient too must be considered to have been cured by prednisone.

These two cases are interpretable as pulmonary eosinophilic syndrome of prolonged course. Without prednisone, they would presumably have run an extremely prolonged course with periodical exacerbations. A specific etiology could not be demonstrated. If it was infection, it must have been due to a virus or to a bacterium, resistant to penicillin and streptomycin. The cases can hardly have been what is known as Löffler's syndrome, as this is described as a benign disease of short duration. On the other hand, the two cases are so much alike that they must be presumed to have been of the same etiology. It is difficult to make a primary diagnosis, and treatment with prednisone cannot be commenced until the results of culture for tuberculous bacillus are available. The disease proved extremely sensitive to treatment with cortisone which resulted in rapid cure.

SUMMARY

Two cases of pulmonary eosinophilic syndrome of prolonged course are described; both were successfully treated with prednisone.

BIBLIOGRAPHY

1. *Mainzer, F.*: J. Allergy 10, 349, 1939.
2. *De la Riva, E.*: South M. J. 42, 429, 1949.
3. *Van der Sar, A. & P. Hartz*: Am. J. Trop. Med. 25, 83, 1945.
4. *Whitchouse, F.*: Gastroenterology 9, 303, 1947.
5. *Berk, J. E.*: J.A.M.A. 127, 354, 1945.
6. *Willett, F. M. & E. Oppenheim*: Am. J. M. Sc. 212, 608, 1946.
7. *Editorial*: Calcified pulmonary nodules and fungous infection. Brit. M. J. 883, 1946.
8. *Smith, D.*: Fungus diseases of the lungs. 1947. Charles Thomas, Springfield, Ill.
9. *Elsom, K. & F. Ingelfinger*: Ann. Int. Med. 16, 995, 1942.
10. *Muller, G.*: Clinical significance of the blood in tuberculosis, 1943. The Commonwealth Fund, New York.
11. *Alpher, J.*: J. Allergy 18, 39, 1947.
12. *Depasse, J.*: Le syndrome de Loeffler. G. Doin, Paris, 1939.
13. *Tocker, A.*: J. Allergy 20, 211, 1949.
14. *Loeffler, W.*: Beitr. z. Klin. Tuberk. 79, 368, 1932.
15. — Schweiz. med. Wchnschr. 66, 1069, 1936.
16. *Sheeran, A. D.*: Am. J. Med. 10, 269, 1951.
17. *Wilson, K. & H. Alexander*: J. Lab. and Clin. Med. 30, 195, 1945.
18. *Weingarten, R. J.*: Lancet 1, 103, 1943.
19. *Irwin, J.*: Ann. Int. Med. 25, 329, 1946.
20. *Coutinho, A.*: Ann. Int. Med. 44, 88, 1956.
21. *Dragsted, P. J.*: Acta Allergol. 6, 103, 1953.

Author's address: Serridslevvej 10, Copenhagen Ø, Denmark.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.