

A Case of Sarcoidosis Transition

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Abstract

A sarcoidosis patient, seen as a paediatric case almost 10 years ago, showed a remittent course of illness for more than 15 years, after which he became a case with active tuberculosis. Treatment with antitubercotics gave dramatic improvement. (*J Infect Dis Antimicrob Agents* 1993; 10: 99-102)

Key words: Sarcoidosis, Tuberculosis, Pulmonary cavity.

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INTRODUCTION

Sarcoidosis is an uncommon disease in Thailand, as well as in most Asian countries (1). To the best of our knowledge, up to the time of this writing, there have been fewer than 50 sarcoidosis cases reported in this country. Most of these were very rarely subjected to prolonged follow-up. This communication describes in chronological order some interesting events that developed during the past 10 years in one of our sarcoidosis patients, a 22-year-old man.

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The Patient

On June 2, 1983 at the age of 12 years, our patient was admitted to the Paediatric Ward of Siriraj Hospital (HN. 103173-26; AN. 1-21100) (2) for generalised massive lymphadenopathy (Figure 1) which he had for six years. He had been unresponsive to antituberculosis treatment. Diagnosis of sarcoidosis was made based on the presence of typical bilateral hilar lymph node shadows on the chest radiograph, characteristic histological appearance of superficial lymph node biopsy, positive Kveim's test and elevated angiotensin-converting enzyme level (2). Administration of prednisolone was then initiated. By the end of three months of treatment, all enlarged lymph nodes had disappeared and the chest



Fig. 1 Showing generalised massive lymphadenopathy seen in June 1983.

radiographic picture became normal. The patient was discharged from the hospital on January 28, 1984 in good physical condition; he was given only multivitamin pills for home medication.

The patient came for follow-up on April 30, 1984 making no special complaint. However, on examination he was found to have lost almost 4 kg of body weight, compared with his weight before leaving the hospital. His inguinal, axillary and cervical lymph nodes were readily palpable but not tender (size 1-5 cm in diameter). No treatment was prescribed at that time.

Then a year later (April 23, 1985) he sought medical attention for the increasingly enlarged but not tender inguinal and axillary lymph nodes (size about 3 x 5 cm); there was no other complaint. Prednisolone (40 mg daily) was prescribed and an appointment was made for him to return in one week. The patient skipped the visit because of improvement in his condition. He did not come for follow-up until April 20, 1989 (4 years later) when another episode of lymphadenopathies occurred; a biopsy from an axillary lymph node again revealed sarcoidosis tissue (No. 04118). The patient was then hospitalised for further treatment in the medical ward on May 16, 1989 (HN. 71309-32; AN. 10639-32) and was discharged on May 31, 1989 after corticosteroid treatment produced a good result. Thereafter the patient was

maintained with prednisolone (5 mg/day) on an out-patient basis.

On October 5, 1992 the patient came to the outpatient clinic complaining of having had progressive swelling on the left side of his neck for two months, with some weight loss, regular evening fever and occasional cough with scanty sputum. He denied a history of tuberculosis contact. On examination, there were palpable discrete lymph nodes over the groin, axilla and sides of the neck; the biggest nodes were on the left side of the neck (3 x 4 cm) (Figure 2). Other systems were within normal limits. A chest radiograph taken on the same day showed discrete opacities over both upper lung fields with multiple small cavities (Figure 3). Chest tomography was performed on November 4, 1992, showing well-formed thick-wall cavities in both upper lung lobes (Figure 4). A tuberculin test again showed strongly positive cutaneous reaction (15 mm of induration).

On November 10, 1992 a series of investigations were undertaken: The haemogram showed haemoglobin concentration of 14.1 g/dl, haematocrit 42.2 per cent, white cell count 8,900 cells/mm³, with the differential counts being 73 per cent neutrophils, 29 per cent lymphocytes, and 1 per cent eosinophils. Serum albumin was 4.4 g/dl (normal 3.5-5.5), total serum protein 7.8 g/dl (normal 6.6-8.7), and serum calcium 8.8 mg/dl (normal 8.1-10.4). Fibreoptic bronchoscopy disclosed no abnormality except a very mildly inflamed tracheo-bronchial tree; specimen bronchoalveolar lavage fluid (BALF) contained 458 cells/mm³ broken down as follows: 5 per cent neutrophils, 8 per cent lymphocytes and 87 per cent alveolar macrophages with a few ciliated



Fig. 2 Showing enlarged lymph node on the left side of the neck (October 1992).

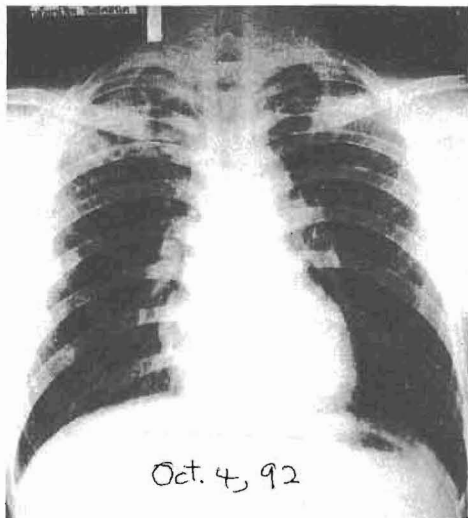


Fig. 3 A postero-anterior chest radiograph taken on October 5, 1992, showing discrete opacities over both upper lung fields with multiple small cavities.

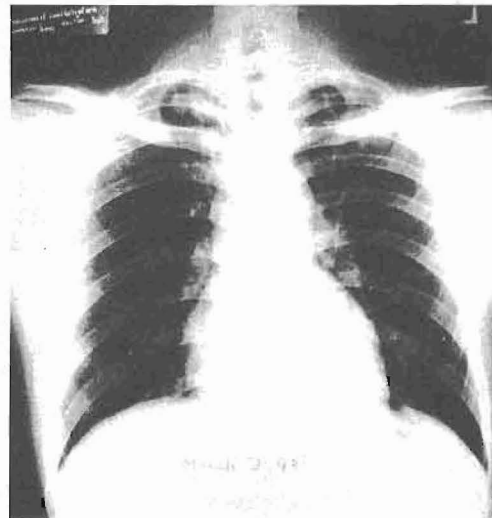


Fig. 5 A postero-anterior chest radiograph taken on March 2, 1993, showing marked clearing of pulmonary lesions.

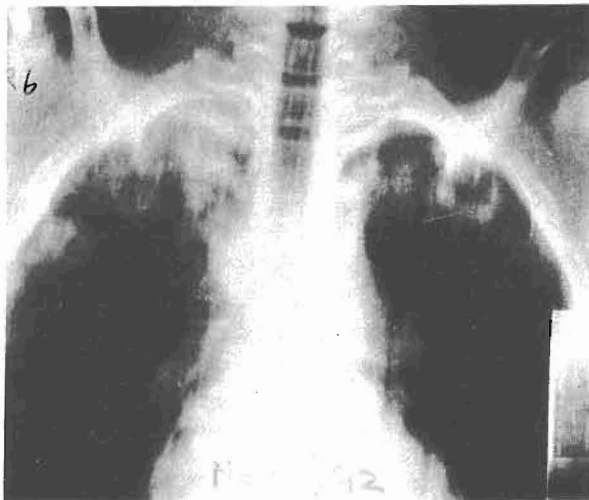


Fig. 4 Chest tomograms taken on November 4, 1992, showing well-formed thick-wall cavities in both upper lung lobes.

columnar epithelial cells; there were no malignant cells and no microorganism was present. Results of studies of T-cell population were as follows: *Blood* contained CD4 (helper/inducer) 455 cells/mm³ (normal 979±358), CD8 (suppressor/cytolytic) 681 cells/mm³ (normal 854±251), CD4/CD8 = 0.67 (normal 1.20±0.45), B cells 134 cells/mm³ (normal 468±253), and NK cells 629 cells/mm³; *BALF* contained B cells 2.12 per cent, T cells 8.15 per cent, NK cells 4.21 per cent, CD4 52.60 per cent, CD8 33.24 per cent, CD4/CD8 1.58.

On November 18, 1992, incision and drainage of the left cervical lymph node lump was performed, yield-

ing about 10 ml of thick yellow pus which was positive for acid-fast bacilli on direct smear and staining; the pus specimen submitted for culture grew *Mycobacterium tuberculosis*. No other microorganism was present. The patient's condition seemed to decline steadily; on November 23, 1992, he coughed up frank fresh blood for the first time. Antituberculosis treatment with rifampicin (600 mg/day), isoniazid (300 mg/day), ethambutol (800 mg/day) and pyrazinamide (1,500 mg/day) was promptly started. Follow-up examination of the patient on March 2, 1993 revealed that most of the enlarged lymph nodes had subsided markedly and a chest radiograph showed marked clearing of pulmonary lesions (Figure 5).

DISCUSSION

The patient described herein was our case of sarcoidosis reported almost 10 years ago (2). We remain firm concerning the diagnosis of our case as then having the most complete features of sarcoidosis ever presented and substantiated of any previous patients encountered in Thailand. Positive skin reactivity to a tuberculin test at that time did not contradict the diagnosis of sarcoidosis, but rather provided evidence in support of a pathogenetic linkage between sarcoidosis and tuberculosis (3).

From an overall picture of the patient's clinical course during the past 10 years, despite some prolonged intervals between visits for follow-up, we conclude that the case had sarcoidosis for more than 15 years (based on the history that he had had lymph node lesions for about

six years before his first visit) with remitting disease for a very long time (9 years after the diagnosis) and that it ultimately became frank tuberculosis.

It may be remarked here that studies of T-cell subpopulations in the blood at the time of active sarcoidosis 10 years ago and in both blood and BALF when tuberculosis developed were not useful in the diagnosis of either sarcoidosis or tuberculosis in our patient.

Although cavitory sarcoidosis in the lung is a known occurrence (4-7), the pulmonary cavitory lesions seen in our patient were most likely tuberculous in nature, despite the negative detection of acid-fast bacilli in bronchoalveolar lavage fluid, as evidenced by the remarkable response of all clinical manifestations to antituberculous and the presence of acid-fast bacilli in the diseased lymph node.

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