Effect of Corticosteroids on Deterioration of Endobronchial Tuberculosis during Chemotherapy*

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A 26-year-old man had endobronchial tuberculosis diagnosed on bronchoscopy. He was treated with standard antituberculosis drugs, but the endobronchial lesions deteriorated bronchoscopically. This is believed to be a hypersensitivity reaction to tuberculoprotein. The lesions improved rapidly with addition of corticosteroids.

Chest x-ray appearance at (left) presentation and (right) ten weeks later while on antituberculosis treatment and steroids.

Endobronchial tuberculosis is an important complication of pulmonary tuberculosis. However, its pathogenesis is not established and its clinical course is variable. Corticosteroid treatment has been advocated by some authors but others claimed no benefit. 1,2

We report a patient whose endobronchial tuberculosis deteriorated initially during standard antituberculosis chemotherapy. Prednisone was introduced and rapid clinical, radiologic and bronchoscopic improvement ensued. As far as we are aware, this is the first photographically documented case of endobronchial tuberculosis which deteriorated during chemotherapy and improved after addition of steroids.

CASE REPORT

A 26-year-old man was admitted to the hospital because of fever and dry cough for ten days. Physical examination was normal. No satisfactory sputum specimens could be produced for examination. Tuberculin test was positive. A chest radiograph showed consolidation in the RLL (Fig 1, left). Bronchoscopy revealed inflammation of the carina and right main bronchus with small pale mucosal nodules on its medial wall. The anterior basal segment of the RLL was occluded by necrotic material (Fig 2, a and b). Biopsy showed several granulomas consisting of epithelioid cells, lymphocytes and plasma cells. Ziehl Neelsen staining for AFB was negative. Bronchial aspirate was also negative for AFB (but culture was positive six weeks later).

Antituberculosis treatment with rifampicin, isoniazid, pyrazina-

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...mide and streptomycin was started. The patient's fever subsided in three days and he was subsequently discharged from the hospital. However, cough persisted and a chest x-ray film six weeks later showed no improvement. Repeat bronchoscopy showed progression of the endobronchial lesions. The right main bronchus contained exuberant tissue on its medial wall where small, pale nodules had been observed previously, and the anterior basal segment of the RLL was completely obstructed (Fig 2, c and d). Biopsy of the right main bronchus showed granuloma formation with no caseous material or demonstrable AFB. In view of the rapid enlargement of the endobronchial lesions, prednisone, 30 mg daily, was started. His cough resolved two days later. A repeat chest x-ray examination four weeks later showed partial resolution of the RLL consolidation (Fig 1, b). A third bronchoscopy was performed. There was much improvement and shrinkage of the lesions in the right main bronchus and the anterior basal segment was no longer occluded (Fig 2, e and f). Biopsy of the residual lesions in the right main bronchus again showed granulomas which were negative for AFB. The dose of prednisone was gradually reduced over six weeks.

**DISCUSSION**

Endobronchial tuberculosis has an incidence ranging from 60 percent in autopsy findings to 10 percent on routine bronchoscopy of tuberculosis patients. Its pathogenesis is uncertain. There appear to be three major factors, namely: the effect of mycobacteria, host immunity, and antituberculosis treatment. The interaction between these three factors is complex and any variation in these three factors may result in an altered clinical course. It is an oversimplification to view all cases of endobronchial tuberculosis as a homogeneous group.

Granuloma formation is regarded as the body's defense to capture and destroy the mycobacteria. The key factor in the body's defense is the macrophage, but T and B lymphocytes and lymphokines are also involved. The introduction of antituberculosis treatment would result in hasted killing of the mycobacteria and release of cell wall material (tuberculoprotein), but this may elicit a delayed hypersensitive response from the patient. This response is not considered essential for development of immunity, and some circumstances may even be detrimental. Possible examples are the enlargement of lymph nodes in tuberculous adenitis and the development of intracranial tuberculoma on antituberculosis treatment.

Hypersensitivity to tuberculoprotein released during treatment with antituberculosis drugs probably accounted for the initial deterioration in our patient. Corticosteroids suppress the hypersensitivity response, and this was the likely mechanism for the rapid resolution of the lesions.

Williams et al also described three patients with endobronchial tuberculosis developing during antituberculosis treatment. However, only one of these patients was bronchoscoped before treatment, and biopsy did not reveal any evidence of endobronchial tuberculosis. We believe that our patient is the first photographically and histologically documented case of existing endobronchial lesions deteriorating while on anti-tuberculosis treatment and resolving rapidly when corticosteroids were added. The inconsistent results of corticosteroids reported previously may be related to the pathogenesis of the endobronchial lesions: steroids are likely to be beneficial when hypersensitivity is the predominant mechanism, but are unlikely to be helpful in more advanced cases when extensive fibrosis is present.

**REFERENCES**

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