Tuberculous pleural effusion occurring during corticosteroid treatment of sarcoidosis

SIR,—The patient whose case Drs AJ Knox, AG Wardman, and RL Page reported (August 1986;41:651) belongs to a group which we suspect is larger than the number of published reports would suggest. Patients with clinical and histopathological features concordant with a diagnosis of sarcoidosis concurrently with evidence of mycobacterial infection are not infrequent; in Britain they are seen especially among recent immigrants from the Indian subcontinent and the Caribbean.

In discussion of such cases it is essential to keep in mind the different implications of the diagnosis of sarcoidosis, which categorises clinicopathologically, and of tuberculosis, which since the time of Koch has been categorised aetiologically. In their brief discussion the authors imply that the only interpretation of their case is that the patient was first attacked by the demon sarcoidosis and then by the demon tuberculosis. They seem to regard it as so unthinkable that both the granulomatous and the exudative features of the disease in their patient might be related to mycobacterial infection that they do not mention this possibility.

Chapters 3, 23, and 25 of our book, to which they refer, are relevant to the conceptual and factual aspects of this problem. Claims that the responses to corticosteroid and antimycobacterial treatment observed in their patient discriminate between tuberculosis and sarcoidosis in such "mixed up" cases cannot be sustained. Clinical manifestations of undoubted mycobacterial disease may be suppressed by corticosteroids; and in some cases, especially of indolent reactions to mycobacterial infection, response to antimycobacterial treatment may be apparent only after the addition of corticosteroids. Thus an initial response to corticosteroid treatment is no evidence against a role for mycobacteria in causation. Although the automatic response to the finding of mycobacteria is now the initiation of appropriate chemotherapy, it should be remembered that before antimycobacterial drugs were available most mycobacterial pleural effusions resolved spontaneously.

We certainly agree that *Mycobacterium tuberculosis* should be sought in a pleural effusion occurring in a patient with sarcoidosis, just as in other patients and in other material from sarcoidosis patients; but we try to keep an open mind about the implications of this finding for the aetiology of sarcoidosis. We are ready to admit not only that we do not know what sarcoidosis is due to but also that we are not certain in some instances what it is not due to.

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*** This letter was sent to the authors, who reply below.

SIR,—We welcome the comments of Professor Scadding and Dr Mitchell on our paper. It is well known, of course, that the granulomatous and exudative features of tuberculosis may be initially suppressed by corticosteroid treatment, and to suggest that we had not considered this explanation for the initial illness in our patient is a trifle uncharitable in the light of our discussion.

The point they make, of course, is that while it may have been reasonable to make a diagnosis of sarcoidosis initially, this diagnosis is cast in some doubt in the light of the subsequent development of a tuberculous effusion. This point is well taken and we had considered it carefully. We concluded, however, that the prompt and sustained suppression of clinical and radiological signs of disease with continuous 10–15 mg of corticosteroids per day over a period of three years taken in conjunction with the other evidence, which included non-caseating granulomas found by transbronchial lung biopsy with no growth of myobacteria, a negative Mantoux test response, and a raised serum angiotensin converting enzyme level, suggested that the initial diagnosis was sarcoidosis and not tuberculosis.

The point made by Professor Scadding and Dr Mitchell is important because what they appear to be implying is that relatively extensive tuberculosis (diffuse interstitial shadowing and bilateral hilar gland enlargement) can be suppressed completely by corticosteroids alone for three years without the aid of antituberculous drugs. We have not seen this and if it can occur it clearly has major implications for our management of "supposed" sarcoidosis.

We were pleased that they concurred with the basic message of our short report, which was that the development of a pleural effusion in subjects with sarcoidosis should prompt a diligent search for tuberculosis.

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