Rifampicin induced pneumonitis or bronchogenic spread of tuberculous empyema through a bronchopleural fistula?

We read with great interest the article by Kunichika et al which describes a probable case of pneumonitis induced by rifampicin.

However, we think that the case needs further clarification.

We note that on admission the chest radiograph did not show a simple left pleural effusion but an air-fluid level, suggestive of a bronchopleural fistula. A bronchopleural fistula associated with tuberculosis usually follows trauma or a surgical procedure, but it can also occur spontaneously in patients with longstanding tuberculous empyema because the tuberculous process establishes an open pathway between the bronchus and pleura. Tuberculous empyema may be present for a long time with few clinical symptoms, and patients may come to clinical attention only when undergoing a routine chest radiograph or after a bronchopleural fistula or empyema develops. The chest radiograph on admission also showed a thickened pleura and loss of volume of the left lung, suggesting longstanding pleural disease. The authors do not specify the characteristics of the pleural fluid of the patient.

In the chest radiograph taken on day 9 we think that the shadows in the right lung follow an alveolar rather than an interstitial pattern (an air bronchogram may be present). A possible alternative explanation for these findings might be the bronchogenic spread of a tuberculous empyema through a bronchopleural fistula. The drainage of the pleural fluid should theoretically have prevented this, but we note that there was still a small amount of pleural fluid on day 9 and therefore it is possible that some fluid could have passed through a bronchopleural fistula. A steroid induced reduction in the inflammatory response associated with this spread might explain the clinical and radiographic improvement. BAL lymphocytosis would be consistent with this hypothesis. The CD4/CD8 ratio in the BAL fluid cannot reliably differentiate between this possibility and lung induced pneumonitis. Drug induced disease can be associated with either high or low CD4/CD8 ratios in BAL fluid. The CD4/CD8 ratio in pulmonary tuberculosis is variable, although in most patients it is within the normal range. The results of the drug lymphocyte stimulation test are highly suggestive of rifampicin induced pneumonitis, but it cannot be considered 100% specific.

Furthermore, we think that the antituberculous chemotheraphy regimen needs to be clarified. As we understand it, the patient was treated with streptomycin for about 2 months. Single drug treatment in tuberculosis is not appropriate because it is associated with a high risk of drug resistance. Also, isoniazid and ethambutol (without streptomycin) were later administered for another 6 months. This seems a rather short course of treatment for a case of smear positive tuberculosis, unless an initial (2 months) course with at least three drugs was implemented. In fact, chronic tuberculous empyema with bronchopleural fistula can result in treatment failure, probably because the thick pleural walls can limit penetration of drugs into the pleural space.

The long term course of the patient needs to be described in more detail. The authors only state that within 6 months there was no recurrence of the abnormal shadows on the chest radiographs, which seems a somewhat brief description of this interesting case.

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References

Authors’ reply
We would like to thank Drs Golpe and Mateos for their interest in our case report and their concerns regarding the diagnosis and antituberculous drugs used to treat the patient. We would like to answer their questions and further clarify the case.

We agree that the possibility of a bronchopleural fistula causing a tuberculous empyema should be considered. We performed a thoracocentesis on day 2, as mentioned in the paper, which explains the reduction in the pleural effusion on the chest radiograph performed on day 9. The pleural fluid was not consistent with a tuberculous empyema. The acid fast staining of the pleural fluid was negative and the culture failed to grow Mycobacterium tuberculosis. Pleural biopsy specimens were taken which did not show features of tuberculous pleuritis. The thickened pleura and volume loss of the left lung suggest late sequelae of pulmonary tuberculosis.

Drs Golpe and Mateos put forward the possibility that the new radiographic findings in the right lung were the result of bronchogenic spread of a tuberculous empyema. As stated earlier, the pleural fluid was not consistent with a tuberculous empyema. In addition, the radiographic findings of the right lung did not indicate a consolidative process. This was confirmed by a chest CT scan which showed an interstitial and non-segmental pattern. Furthermore, bronchoalveolar lavage (BAL) was performed on the right lung and the BAL fluid was negative for acid fast bacilli and for Mycobacterium tuberculosis. This further supported our theory of a drug induced pneumonitis causing the patient’s new signs and symptoms rather than the bronchogenic spread of tuberculosis.

We agree that the antituberculous drug regimen was temporary. However, for this particular patient we weighed up the risks and benefits and felt this was the safest choice. Because of our suspicion that rifampicin was the causative agent (based on the increased drug lymphocyte stimulation test index of 370%), we could not rechallenge this high risk patient with the same drug. We designed his regimen on the basis that he is an elderly patient and may not tolerate certain side effects of antituberculous chemotherapy.

We followed the patient closely for 1.8 months during which time he showed no evidence of drug failure or recurrence. He has continued his medical care at another hospital and they have confirmed his continued health.

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A meeting organised by Professor A Newman Taylor and Dr P Cullinan will be held on 31 October 2003 at the National Heart and Lung Institute, Dovehouse Street, London SW3 6LY.

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