LETTERS TO THE EDITOR

Endobronchial nocardial infection

Further to the recent case report of Drs McNeil, Johnson and Oliver (December 1993;48:1281-2), we have seen another patient with chest infections and endobronchial involvement. This is the fifth reported case. A previously well 25 year old Australian aboriginal man presented with a non-resolving right lower lobe pneumonia. He had a five year history of smoking and had been admitted to a peripheral hospital and treated with intravenous amoxicillin and erythromycin for two weeks. Sputum cultures had subsequently grown Nocardia and he was therefore transferred to our hospital. On admission he was febrile to 38°C, and ascutulation revealed right lower zone crepitations. He produced 200 ml blood-stained sputum per day. Oral trimethoprim/sulphamethoxazole was commenced in a dose of 160 mg/3200 mg per day. The fever settled but sputum production and chest signs persisted. Bronchosopic examination revealed a polypoid “tumour” partially obstructing the postero-basal segment of the right lower lobe. Biopsy material from this revealed filamentous organisms and florid active cavitary inflammation. Culture of the biopsy speci- men confirmed the organisms as Nocardia and bronchial washings also grew Nocardia. Intravenous amikacin was added with resolution of chest signs and cessation of sputum production.

No dissemination of Nocardia was seen after bronchoscopy, possibly because he had been on treatment for 10 days before the procedure. As with the previous case there was no underlying defect of humoral or cell-mediated immunity. However, he consumed an average of 200 mg alcohol per day. He will remain on trimethoprim/sulphamethoxazole for at least nine months.

This case shows once again that endobronchial “tumour” can be due to Nocardia even in patients with normal immune systems.

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Fluticasone propionate v beclometasone dipropionate (BDP) in moderate to severe asthma

I read with interest the paper by L Fabbri et al (August 1993;48:817-23) which compared the above drugs in patients with moderate to severe asthma. The authors found a small (15 l/min) peak expiratory flow (PEF) advantage in the fluticasone propionate group compared with BDP amounting to about 4% of the average morning PEF of these patients. I calculate from figure 1 and table 1 that the morning PEF of the fluticasone group increased from 74% predicted to 81% predicted, whereas in the BDP group PEF increased from 73% to 78% predicted.

The symptom scores were similar on both medications but there was said to be a slight excess of “severe” asthma (not clearly defined) in the group given BDP. There were 13 “severe” exacerbations in the fluticasone group and three “severe” exacerbations in the fluticasone group. However, a further four patients using fluticasone withdrew from the study because of poor asthma control compared with only one patient using BDP and a further 15 patients (all using fluticasone) had to increase their dose of inhaled steroid because of poor asthma control. The total number of patients with poor asthma control was therefore 22 in the fluticasone group and 14 in the BDP group. Neither drug had any significant effect on adrenal function.

The authors conclude that fluticasone may be more effective than BDP and they recom- mend it for long term use in asthma because of the marginal benefits shown in the study, and also because of a theoretically superior side effect profile which was not demonstrated in their own study.

Surely it would be equally logical to con- clude that both groups of patients fared very well on high dose inhaled steroid treatment and any difference between the two groups was disappointingly small considering that the fluticasone group was supposedly given twice the biologically effective dose of inhaled steroid. Most patients would have fared equally well on either preparation although there was a suggestion that a small number of patients who were prone to recur- rent exacerbations might fare better on the more potent drug.

Given that this trial was commercially commissioned and funded, one can understand the enthusiasm for the new agent which is expressed in the discussion. How- ever, the discussion omits to mention that one year’s treatment with fluticasone 1500 µg per day would cost £295 compared with £253 per year for the same dose of BDP. I feel that it would be premature (and very expensive) to transfer patients from BDP to fluticasone on the basis of this trial. I would have difficulty convincing my Pharmacy Therapeutics Committee that a 4% gain in peak flow with no difference in symptom scores or side effects was worth a 265% increase in cost.

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Fluticasone propionate v beclometasone dipropionate (BDP) in moderate to severe asthma

Drs McNeil, Johnson and Oliver (December 1993;48:1281-2) report a case of Nocardia involvement of the bronchus after undergoing bronchoscopy. We have also encountered this finding in another patient.

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Accuracy of CT appearances of fibrosing alveolitis

In answer to the question raised by Drs Selby and Brown (December 1993;48:1289) we agree that the sensitivity and speci- ficity of the high resolution computed tomographic (CT) appearances of crypto-genic fibrosing alveolitis, including only those cases with histopathological confirmation of the diagnosis,3,4 Contrary to their dogmatic assertion that the inclusion of cases without histological confirmation “results in an over- estimate of the diagnostic usefulness of CT scanning”, we have demonstrated the performance of high resolution CT scanning actually improves: sensitivity 91% (compared with 89% for the whole group) and specificity