LETTERS TO THE EDITOR

Pulmonary complications of HIV disease

We read with interest the review by Drs DG Mitchell and RF Miller (May 1992;47:381-90). They refer to a study published in 1991 that bacterial pneumonia occurred much less frequently than Pneumocystis carinii pneumonia in homosexual men in London over a four year period, in contrast to a high relative frequency in an American population that included a large proportion of intravenous drug users. Our findings suggest that bacterial pneumonias in HIV infected homosexual men in London may be more common than previously reported. We reviewed the case notes of all HIV seropositive patients admitted to our unit over six months (December 1991 to June 1992) in whom a diagnosis of chest infection was made. Sixty three patients were identified, representing 19% of admissions related to HIV infection. Forty one patients were diagnosed as having Pneumocystis carinii pneumonia; ten as having bronchoalveolar lavage and Pneumocystis carinii pneumonia was confirmed in 35 (32 homosexual, three intravenous drug users). Twenty two patients were diagnosed as having bacterial pneumonia. For this survey we adopted the following definition of bacterial pneumonia: the presence of fever (>37.5°C) with new chest symptoms or signs and a new infiltrate on the chest radiograph and responsiveness to antimicrobials (not including anti-Pneumocystis pneumonia cover). Fifteen patients fulfilled these criteria (12 homosexual, three intravenous drug users). Our findings suggest that the ratio of cases of bacterial pneumonia to episodes of pneumocystis pneumonia requiring admission was around 1:2 overall and greater than 1:3 (12:2) in homosexuals, substantially higher than the 1:8 reported by Jeffrey and Miller. The populations studied may not be directly comparable (for instance, our data only apply to patients admitted to hospital), but none the less the results support the possibility that the relative frequency of bacterial chest infections in patients with HIV disease may have increased. Factors responsible for this may include more effective prevention of Pneumocystis carinii pneumonia and an increase in the population of patients with lung damage related to previous chest infection.

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Acute pneumonitis associated with low dose methotrexate treatment for rheumatoid arthritis

We read with interest the paper by Dr MR Hargreaves and others (August 1992;47:628-33). With the increasing use of low dose methotrexate in the treatment of patients with rheumatoid arthritis, drug induced pneumonitis is likely to become more frequently recognised. The correct diagnosis is not always easily made, however, and inappropriate management may be fatal.

We wish to report the case of a 65 year old man with a 20 year history of rheumatoiid arthritis who started taking methotrexate 10 mg weekly in March 1992, at which time his pulmonary function and chest radiograph were normal. He was admitted to hospital with increasing breathlessness and cough over a period of three weeks. His chest radiograph showed a right lower zone shadowing and an electrocardiogram suggested left ventricular ischaemia. He was treated with intravenous diuretics and nitrates but failed to respond and became increasingly hypotensive, with consequent oliguria and acute renal failure. His oxygen saturation (SaO2) fell to 80% despite increasing concentrations of oxygen. Cardiac enzymes were normal, and dopamine was given through a central venous line—to no avail. Antibiotics were not administered.

The possibility of methotrexate pneumonitis causing hypoxia and consequent myocardial depression was considered and he was treated with 500 mg of intravenous methylprednisolone on three successive days. Within 24 hours of his starting on this his SaO2 had risen to 95% and his blood pressure had become normal. Pulmonary function testing at this stage showed a pronounced restrictive deficit and renal function improved and he was started on prednisolone 60 mg daily. He was discharged one week later. At review a month after discharge his renal function and chest radiograph were almost normal. Methotrexate had been permanently discontinued and his pulmonary function is slowly improving with a reducing dose of oral prednisolone.

Pneumonitis due to methotrexate can closely mimic left ventricular dysfunction and failure to suspect it may lead to a fatal delay. Immunosuppressive doses of steroids may be required in severe cases, though infection should first be excluded. Upper lobe abnormality, although unusual, may be especially likely to cause diagnostic confusion. Disproportionate hypoxia with a restrictive spirometric pattern may aid differentiation from primary myocardial disease.

We hope that the accumulating data on methotrexate pneumonitis will alert physicians to the broad spectrum of its presentation.

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Tuberculosis contact tracing: are the British Thoracic Society guidelines still appropriate?

The suggestion made by Dr SF Hussain and colleagues (December 1992;47:984-5) of a review of tuberculosis contact tracing recommendations is timely. The number of
Pulmonary complications of HIV disease.

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