Mycobacterial infection in patients infected with the human immuno-deficiency virus

Dr M Helbert and colleagues (January 1990;45:45–8) reported a 6% incidence of Mycobacterium tuberculosis in a case series of 207 patients with AIDS and 34 patients with the AIDS related complex. At Central Middlesex Hospital we have been concerned in the care of 12 patients with AIDS, three of whom had M tuberculosis. Two developed tuberculosis a few months before the diagnosis of AIDS; one was an immigrant from sub-Saharan Africa and the other an immigrant from the Indian subcontinent. The third patient, who was white, developed tuberculosis as a late complication of AIDS. Of all the 199 patients in our patient base, nine patients had Tuberculosis (one of whom developed an infection with Mycobacterium avium-intracellulare), one was West Indian, and the other six were white.

The United States has shown that M tuberculosis in patients with AIDS is much more common in groups which have a higher prevalence of previous tuberculous infection.1,2 A recent prospective study of intravenous drug users provided strong support for the idea that tuberculosis in these patients is a consequence of reactivation of latent tuberculous infection rather than a primary infection.3 We believe that the high prevalence of M tuberculosis in our patients was due to the high proportion of ethnic minorities (42%) in whom there is an increased prevalence of tuberculosis. In England and Wales during 1983 the notification rates of tuberculosis in Africans and the Indian subcontinent and West Indian ethnic populations were estimated to be 24 times and four times, respectively, that of the white population.4 In sub-Saharan Africa the risk of infection with tuberculosis is also substantially greater than that of England and Wales.5 Dr Helbert and colleagues in their discussion considered only intravenous drug misuse as a cause of increased prevalence of tuberculosis within their population. No mention was made of the ethnic background of their patients. We believe that this is important in the assessment of M tuberculosis in HIV infected patients. Of the 23 HIV positive patients attending Central Middlesex Hospital, nine are from sub-Saharan Africa or the Indian subcontinent and three are intravenous drug misusers, reflecting the demography of the local population.6 It has recently been suggested that in the United Kingdom people infected with HIV who have not had BCG vaccination should be tuberculin tested and offered prophylactic treatment if they are positive.7 False negative tuberculin reactions are recognised, however, in HIV positive patients8 and in symptomatic patients must be combined with a chest radiograph and appropriate bacteriological specimens. The demography of our population suggests that tuberculosis is likely to be a continuing problem. We intend to implement routine tuberculin testing in our patients who have not had BCG vaccination and to offer antituberculous treatment or prophylaxis as necessary.