LETTERS TO
THE EDITOR

Mycobacterial infection in patients infected with the human immunodeficiency 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 was not an immigrant from the subcontinent. The third patient, who was white, developed tuberculosis as a late complication of AIDS. Of the remaining nine patients, one was of sub-Saharan African origin (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 patient was due to the high proportion of ethnic minorities (42%) in whom an increased prevalence of tuberculosis. In England and Wales during 1983 the notification rates of tuberculosis are much higher in the subcontinent and West Indian ethnic population than in sub-Saharan Africa.4 In the United States the incidence of tuberculosis in the white population in 1982 was greater than that of England and Wales,5 but the white population in England and Wales accounted for 8% of the population.

Dr Herbert and colleagues in their discussion suggested only intravenous drug misuse as a cause of the 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 users, reflecting the demographic characteristics 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.6 False negative tuberculin reactions are recognised, however, in HIV positive patients7 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.

P J HORN
D BELL
MURPHY
MW McNicol
Central Middlesex Hospital,
London NW10 7NS


It is now generally accepted that the greater incidence of tuberculosis in patients with AIDS than in the general population is due to breakdown of foci of tuberculous infection acquired before infection with the human immunodeficiency virus (HIV). The incidence of tuberculosis in a group of patients with AIDS will therefore depend mainly on the extent to which the group was infected with tuberculosis before acquiring HIV infection. On this basis the best epidemiological information might be available from the series presented so successfully from the clinical standpoint by Dr M Helbert and others (January 1990;45:45–8). If the series includes any non-white patients it would be helpful to see an analysis of the incidence of tuberculosis by ethnic group. An analysis of the incidence of tuberculosis by broad age groups in the white ethnic group might also be informative, as those over age 50 years in 1983–8, when this series was collected, were found to be at least 40% tuberculin positive when aged 13 years or more in 1949–50, while those under 30 years of age in 1983–8 were less than 2% tuberculin positive when aged 13 years in the early 1970s. Similar information from other series would also be most helpful in assessing the likely impact of the developing AIDS epidemic on tuberculosis in Britain. Separate analysis by age of other mycobacterial infections in patients with AIDS might help to reveal differences in pattern from disease due to Mycobacterium tuberculosis.

VH SPRINGETT
Solihull,
West Midlands B91 2QJ
IAN SUTHERLAND
MRP Biostatistics Unit,
Cambridge CB2 2BW


AUTHORS’ REPLY. Of the 15 patients with tuberculosis and HIV infection, one was of Indian origin (age 43), one was of Afro-Caribbean descent but brought up in Britain (age 21) and one was a white man who had lived in central Africa (age 52). The rest of the patients were white and had lived in Europe all their lives. None of these patients was an intravenous drug user.

M HELBERT
D ROBINSON
D W AVERY
T HELLYER
M McCARTHY
AJ PINCHING
DN MAHI
Human Tumour Immunology Group,
Imperial Cancer Research Fund,
91 Riding House Street,
London WIP 8BT

Assessment of reversibility of airway obstruction in patients with chronic obstructive airways disease

We were interested to read the article by Nisar and colleagues (March 1990;45:190–4) describing reversibility in response to salbutamol and oral prednisolone in patients with chronic airflow obstruction.

We published,1 when analysed according to the criteria used by the Liverpool group do not show the same conclusions, and we are concerned lest chest physicians withhold potentially beneficial treatment in disabled patients with chronic airflow obstruction.

Our reanalysis shows that, of 13 patients who responded to oral prednisolone by increasing their FEV1 by at least 15% and 200 ml, five (38%) showed no response to 10 mg nebulised salbutamol. When we retain our own criteria for response (a 20% improvement in FEV1, or forced vital capacity (FVC) or peak expiratory flow (PEF), over baseline), when analysed according to the criteria used by the Liverpool group do not show the same conclusions, and we are concerned lest chest physicians withhold potentially beneficial treatment in disabled patients with chronic airflow obstruction.

We wish to point out that the number of patients who responded to salbutamol was 13 and the number of patients who showed no response to salbutamol was five. We have reanalysed the data and shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group.

107

We wish to point out that the number of patients who responded to salbutamol was 13 and the number of patients who showed no response to salbutamol was five. We have reanalysed the data and shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group. We have also shown that no statistically significant difference was found when the response to salbutamol was compared with the response to prednisolone when analysed according to the criteria used by the Liverpool group.