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ethyl chloride and amyl or butyl nitrite. The aetiology of the immunosuppression must be identified in order to protect the population at risk.

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PRIMARY PNEUMOCYSTIS CARINII AND CYTOMEGALOVIRUS INFECTIONS

SIR,—Pneumocystis pneumonia almost invariably affects the immunosuppressed.^{1,2} Recent reports from the United States^{3,4} have described the occurrence of pneumocystis pneumonia in 61 patients with no clinically apparent immunodeficiency. 51 were homosexuals, and of the 5 patients whose case histories were reported in adequate detail all had evidence of previous or current cytomegalovirus infection (CMV). We report the case of a 49-year-old homosexual male who presented with pneumocystis pneumonia and CMV infection and in whom no underlying immune deficiency was found. We believe this to be the first report of this association in the U.K.

This man was referred to the Brompton Hospital having presented elsewhere with a 3 month history of weight loss, 3 weeks' general malaise, and progressive breathlessness on exertion. He had been fit before this illness and did not abuse drugs or smoke. He was a practising homosexual who travelled to Miami, Florida, annually to visit homosexual friends, the last visit being 9 months before his terminal illness. None of his contacts are known to have been ill at the time of his visit or to have become ill subsequently.

On examination he was clubbed, centrally cyanosed, and had bilateral basal crackles. No other abnormalities were noted. Chest X-ray showed bilateral, predominantly mid and lower zone granular shadows, and the presumptive diagnosis was sarcoidosis. He was started on 30 mg prednisolone per day because of the severity of his illness and attempts were made to obtain histological confirmation. Mediastinoscopy found no lymph nodes. Transbronchial biopsy was diagnostic of pneumocystis pneumonia and steroids were discontinued. He was transferred for further management. On arrival he was febrile, had a sinus tachycardia of 150/min but no new clinical features were noted. Investigation showed Hb 13.6 g/dl, white blood cells $10.6 \times 10^9/l$ (lymphocytes $0.6 \times 10^9/l$), erythrocyte sedimentation rate 3 mm/h; urea, electrolytes, creatinine, and glucose normal; bilirubin 10 $\mu\text{mol/l}$, alkaline phosphatase 438 IU/l, γ -glutamyl transpeptidase 103 IU/l, aspartate transaminase 20 IU/l; total protein normal, albumin 21 g/l, calcium and phosphate normal, IgG 210 IU/ml, IgA 618 IU/ml, IgM normal, C3 normal, C4 normal, antinuclear activity negative, rheumatoid factor negative, HBsAg negative, serum angiotensin converting enzyme normal.

The following antibodies were negative: Wassermann, pneumocystis, influenza A, B, C, adenovirus, respiratory syncytial virus, *Coxiella burnetii*, psittacosis, *Mycoplasma pneumoniae*, cytomegalovirus (both IgM and IgG), coccidioides, histoplasma, and avian and aspergillus precipitins. Sputum was sterile for pathogens and acid fast bacilli. Chest X-ray showed diffuse fine granular shadowing throughout both lung fields. This had become rapidly progressive over a 3 week period. Electrocardiogram showed a sinus tachycardia only. He was ventilated because of increasing restlessness and hypoxia (PO_2 7.79 kPa [58.5 mm Hg], FiO_2 35%) and treated with intravenous co-trimoxazole (14 mg/kg trimethoprim per day). When the association with CMV infection became known, intravenous acycloguanosine was added (10 mg/kg, 8-hourly). His temperature rose at first (to 39.4°C) but subsequently settled although tachycardia persisted. On the 5th hospital day he sustained asystolic cardiac arrest during which he

acquired a left pneumothorax under tension requiring underwater seal drainage. Maintaining adequate PO_2 became difficult despite FiO_2 of 100%. His condition gradually deteriorated and he died on the 10th hospital day. Necropsy revealed pale firm consolidation in both lungs, and histology confirmed pneumocystis pneumonia with masses of the organisms in the alveoli and an interstitial inflammation with lymphocytes and plasma cells. Also there was evidence of CMV infection of the lung with scattered solitary enlarged alveolar epithelial cells showing eosinophilic nuclear and cytoplasmic inclusions. In addition there was evidence of CMV infection of the adrenals.

Pneumocystis carinii pneumonia (PCP) is an uncommon infection which almost always occurs in the immunosuppressed host.¹ Rifkind et al.² reported an association between PCP and CMV infection in immunosuppressed adults. The recent reports^{3,4} of coincidental PCP and CMV infection in homosexual males with no clinically apparent immunosuppression are identical to the presentation of our patient. He was homosexual, travelled to the United States, had histologically proven infection with pneumocystis and CMV and no signs of malignancy or other lesions were found at post mortem. In addition he had an absolute lymphopenia at presentation and negative serology, including CMV titres. It is not known whether homosexual practice transmits some infective agent which predisposes the host to these uncommon infections or whether the contact simply allows spread of CMV with subsequent immunosuppression and opportunistic infection. In support of the latter hypothesis Rinaldo et al.⁵ report transient in vitro evidence of cellular immune dysfunction in previously healthy individuals who acquire CMV infection, although there are no data on cellular immunity in homosexuals with and without CMV antibodies. In another study, Drew et al.⁶ have shown that 94% of homosexual males have CMV antibody compared with 54% of heterosexual males. Our patient, although having normal or raised total IgG, IgM, and IgA had no specific antibody to common viruses or to CMV although he had histological evidence of infection with CMV. We were unable to study delayed hypersensitivity reactions.

We believe this to be the first report of this relationship of disease in the U.K. and suggest that the diagnosis be considered in practising male homosexuals who present with an unexplained respiratory illness.

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METOPROLOL AFTER ACUTE MYOCARDIAL INFARCTION

SIR,—The paper by Dr Hjalmarsen and colleagues (Oct. 17, p. 823) on the use of metoprolol in acute myocardial infarction is of great practical importance and will, no doubt, encourage many physicians to use this treatment. If this is to happen, we need to be reassured that the recommended regimen is safe not only in the milieu of a teaching hospital but also in district hospitals and general practice. Unfortunately we are not told whether any of the adverse reactions, such as hypotension or cardiac failure, were severe enough to have called for emergency or specialised forms of treatment. Profound hypotension after intravenous beta-blockade has been reported from several centres and seems to have been responsible for some of the deaths in the alprenolol study from Denmark.⁷

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