GAY COMPROMISE SYNDROME

Sir,—A remarkable outbreak of opportunistic lung infections and/or Kaposi's sarcoma in homosexual men has been reported this year in the United States. 1 2 3 The first report concerned five men with pneumocystis carinii pneumonia. 1 A month later came a report of Kaposi's sarcoma in twenty-six homosexuals, in five of whom opportunistic infections subsequently developed. P. carinii in four and toxoplasmosis in one. Besides their homosexuality these patients had in common a high incidence of past or present cytomegalovirus (CMV) infection. Two later reports 4 5 listed one hundred and eight cases of Kaposi's sarcoma and/or P. carinii pneumonia; 94% of the patients were homosexual or bisexual. We wish to report a case in which Kaposi's sarcoma-like lesions developed in a homosexual man with CMV viruria, followed by P. carinii pneumonia and overwhelming cryptococcal pneumonia.

A 29-year-old man was admitted for evaluation of skin lesions, fever, and an abnormal chest X-ray. 8 months earlier several small, raised, purple, non-pruritic, and painless lesions had appeared on his lower limbs. A biopsy specimen was reported as showing glomus haemangiomas and no therapy was given. 2 weeks before the patient's admission a non-productive cough began, with fevers to 40°C and sweats. The patient was a homosexual who had had many partners during the last few years but no sexual contacts in the 4 months before admission. He had used "recreational" drugs freely, including LSD, mescaline, cocaine, marijuana, nitrous oxide, ethyl chloride, amyl nitrite, and butyl nitrite, but no intravenous drugs or opiates. On admission he was slightly feverish and tachypnoeic. His subsequent progress was complicated by disseminated haemorrhage, and acute renal failure. After 11 days in hospital and chemotherapy, in whom P. carinii and cryptococci were found in the lungs at necropsy, and one of the homosexuals reported by the C.D.C. 1 2 had pneumocystosis and cryptococcal meningitis.

Biopsy of one of the skin lesions showed numerous dilated, irregular small vascular channels lined by mature endothelial cells without spindle cells or fibrosis. Immunofluorescent stains for deposits of immunoglobulins, complement, fibrin, and albumin were negative, as was electron microscopy for virus particles. On the second day in hospital, bronchoscopy was performed, and the washings and a transbronchial biopsy showed P. carinii. On treatment with trimethoprim 20 mg/kg and sulphamethoxazole 100 mg/kg the patient's temperature fell but the pulmonary infiltrates worsened and he required mechanical ventilation by the sixth day. His subsequent progress was complicated by disseminated intravascular coagulation, gastrointestinal and pulmonary haemorrhage, and acute renal failure. After 11 days in hospital and 10 days' treatment with trimethoprim/sulphamethoxazole his respiratory status deteriorated sharply and he died.

Sections of lung obtained at necropsy showed P. carinii in alveoli and large numbers of encapsulated, budding yeasts throughout the interstitium (see figure). Yeasts were also present in hilar and abdominal lymph nodes, liver, spleen, bone marrow, and skin. Subsequent cultures of both lungs and spleen grew Cryptococcus neoformans. A urine specimen taken on admission yielded CMV after 6 weeks' culture. Initial findings in sections from skin lesions and viscera were regarded as consistent with but not diagnostic of Kaposi's sarcoma. These tissues are being studied further by Dr T. Lee and his colleagues.

This case is a paradigm of the newly recognised syndrome of opportunistic infections and/or Kaposi's sarcoma in homosexual males. Because these patients seem to be severely immunocompromised, we have called it the "gay compromise syndrome." P. carinii infection is rare and, before its appearance in homosexuals, was found almost exclusively in malarious or immunodeficient patients. C. neoformans is likewise an infrequent pathogen; it may infect immunodeficient or apparently healthy individuals. Simultaneous infection with these organisms has seldom been reported before. Winslow and Hathaway 5 described a patient with chronic lymphatic leukaemia treated by radiotherapy and chemotherapy, in whom P. carinii and cryptococci were found in the lungs at necropsy, and one of the homosexuals reported by the C.D.C. 1 2 had pneumocystosis and cryptococcal meningitis.

Retrospective examination of specimens obtained on the second day revealed no evidence of cryptocoecosis, and we conclude that this infection began, or at least burgeoned, while the patient was being treated for pneumocystitis. Another implication is that patients with this syndrome, like other severely immunocompromised patients, should be investigated promptly and aggressively. As to the cause of the immunosuppression, one hypothesis invokes CMV infection, which suppresses certain immune responses in animals and man. 6 7 Owing to the high incidence of CMV antibodies and viruria in the homosexual population at large, this hypothesis may be difficult to substantiate. The findings in our patient are consistent with the possibility that CMV infection contributed to immunosuppression. Another possibility is that one of the "recreational" drugs may be immunosuppressive. We speculate that an inhaled agent that depresses pulmonary cellular immune function would provide another plausible explanation. Candidate inhalants in common use include


Section of lung at necropsy.

Note P. carinii in an alveolus (to the left) and numerous budding yeast forms of Cryptococcus in the interstitium (to the right). (Methenamine silver; x about 75).
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

Sr.,—Pneumocystis pneumonia almost invariably affects the immunosuppressed. Recent reports from the United States 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 (CMV) infection. 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 of 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 withdrawn. We are reassured that the recommended regimen is safe not only in the milieu of a teaching hospital but also in district hospitals and general practice.

Sr.,—The paper by Dr Hjalmarson 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 the United Kingdom.

METOPROLOL AFTER ACUTE MYOCARDIAL INFARCTION

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