partners held constant, years of homosexual activity but not age was also significant ($p=0.0002$). Number of episodes of venereal disease remained significantly associated even after adjustment for the number of partners. Two subjects were initially anti-HBc positive but seroreverted between 1984 and 1989. Both were anti-HIV positive and had abnormal CD4 to CD8 ratios (0.04 and 0.17) with low CD4 values in 1989.

Discussion

The profile of an association between hepatitis B and a wide range of sexual lifestyle variables indicates that hepatitis B is a sexually transmitted disease among homosexual men. We have analysed these variables in detail in order to contrast the findings with reactivity to hepatitis C virus. Had hepatitis C been readily transmitted sexually we should expect to have seen a characteristic epidemiological profile. We found no direct evidence of sexual transmission of hepatitis C. Nevertheless, although few of our cohort of homosexual men were positive for anti-HCV, the proportion was considerably higher than that among male blood donors (0.3%) of the same age in Denmark (Wantzin *et al*, unpublished data). Yet none of our anti-HCV positive subjects had ever received a transfusion and only one gave a history of intravenous drug use. The absence of parenteral exposure favours the possibility of sexual transmission, although at a much lower rate than in the case of hepatitis B virus or HIV. Rates of seroconversion detected to hepatitis C virus were six to 13 times lower than those recorded to hepatitis B virus and HIV.

One caveat arose from the longitudinal data: the hepatitis C marker was not consistently present among all initially reactive or seroconverting subjects. Loss of anti-HCV has also been described in three patients with resolving non-A non-B hepatitis in a prospective study of transfusion recipients.¹³ We have reported declines in reactivity to hepatitis B virus, sometimes to undetectable levels, with progressive immunodeficiency¹⁴ and also observed the phenomenon with anti-HBc in this study. Not all subjects who lost reactivity to hepatitis C virus in this study were infected with HIV and had abnormal immunity, however, which argues for a different explanation for the phenomenon of seroreversion to hepatitis C virus.

Our observation that seroreversion to hepatitis C virus may be fairly common suggests that the cumulative incidence of seropositivity for the period 1981-9 may have been slightly higher than the 4.1% recorded in this study. Alter *et al*, however, found the mean duration of anti-HCV positivity among subjects

who later seroreverted to be 4.1 years (range 7 months to 11 years).¹³ Hence the number of anti-HCV positive subjects missed in our series was probably small.

The persistence of reactivity in some subjects may indicate that some of these men were chronic carriers of hepatitis C. Although a test for hepatitis C virus in the blood is not available, we note that the highest figures for the prevalence of circulating anti-HCV are reportedly found among patients with chronic non-A non-B hepatitis and that among these patients seroreversion is unlikely to occur.¹³ The high prevalence of anti-HCV in patients with haemophilia and intravenous drug users^{4-6,8} may be explained by these people being chronic carriers or recurrently exposed to hepatitis C virus.

We conclude that hepatitis C virus is not readily spread by homosexual activity, although sexual transmission may occur occasionally. Anti-HCV positive subjects may lose their antibodies and it remains to be seen whether those people are less infectious than persistently seropositive subjects.

This study was supported by the Danish Medical Research Council, the Danish Cancer Society, and the Danish Hospital Foundation for Medical Research.

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(Accepted 18 May 1990)

Diarrhoea due to cryptosporidium infection in thalassaemia major

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Cryptosporidium has been increasingly recognised as a cause of diarrhoea in both immunocompetent and immunocompromised patients with the advent of adequate staining techniques. It is a coccidian parasite, which seems to be a single species able to infect a range of animal hosts.¹ In immunocompetent adults it ranks third in the non-viral causes of gastrointestinal symptoms after salmonella and campylobacter,¹ and in patients with AIDS it is the commonest cause of diarrhoea.

The severity of infection with cryptosporidium varies, and immunocompetent patients may be asymptomatic.² Usually there is an attack of watery, non-bloody diarrhoea with abdominal pain, vomiting, and fever. Temperatures of up to 38.3°C have been reported.² The illness is self limiting, and symptoms may be short lived or persist for many weeks. In immunocompromised subjects the illness is prolonged and debilitating and may be fatal.¹

Case report

A 22 year old woman with β thalassaemia major had been dependent on transfusions since birth and received desferrioxamine for iron overload. She had had a splenectomy at the age of 7, and a test for HIV in March 1989 had yielded negative results. In June she presented to casualty with a 24 hour history of watery diarrhoea, colicky abdominal pain, vomiting, dizzi-

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Br Med J 1990;301:212-3

ness, sweating, and anorexia. Her sister and niece had had similar symptoms five days previously without systemic upset and had not required admission to hospital.

On examination she had a temperature of 39°C, a tachycardia, and minimal abdominal tenderness with active bowel sounds. Haematological and biochemical investigations gave normal results. The ratio of T4 to T8 cells was normal at 2.3 (reference range 1.0-2.5). Cefuroxime was started, desferrioxamine was stopped, and she was given intravenous fluid replacement. Twenty four hours later her temperature was normal. Her symptoms persisted for nine days, and faecal examination showed *Cryptosporidium* sp. Although spiramycin is effective in some cryptosporidium infections, this was not started as her condition was improving. Blood cultures, results of serological examination, and stool samples were negative for yersinia.

Comment

Certain infections have been described more commonly in patients with thalassaemia major than in the general population, including infection with *Yersinia enterocolitica*, which thrives in iron excess³; pneumococcal infections; and mucormycosis. Thalassaemic patients may be more susceptible to infections as a result of underlying immunological abnormalities caused by their treatment. Monocytes in blood from

thalassaemic patients have an intracellular microbicidal defect associated with iron overload, which neither splenectomy nor chelating agents correct.⁴ Furthermore, desferrioxamine inhibits the proliferation of lymphocytes and monocytes and transfusion treatment started early in life might perhaps cause persistent immunological modifications as evidenced by a reversal of the ratio of T4 to T8 cells.⁵

Although the cryptosporidium infection in this patient was self limiting, it was more severe than a concomitant gastrointestinal illness in two members of her family without thalassaemia major. We suggest that in immunocompromised patients with iron overload cryptosporidium infection may be more severe than it is in the general population. We therefore suggest that if patients with thalassaemia major present with diarrhoea and abdominal pain stool samples should be analysed for the presence of cryptosporidium as well as organisms such as yersinia.

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(Accepted 9 May 1990)

Multiple emboli in hydatid disease

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Br Med J 1990;301:213-4

Hydatid disease, usually in the liver or lungs, is not uncommon in west Wales. I report on a sheep farmer in whom a primary hydatid cyst in the interventricular septum caused multiple emboli in the pulmonary and systemic arterial circulation.

Case report

A 57 year old sheep farmer presented with a history of severe chest pain and vomiting followed shortly afterwards by sudden pain in his left leg. He was distressed with a tachycardia and tachypnoea, and there was slight discoloration of his left thigh. Results of investigations were normal apart from a raised white cell count; creatine kinase and β -hydroxybutyrate dehydrogenase activities were normal. An electrocardiogram showed an old inferior myocardial infarction and a wandering atrial pacemaker. The condition of his leg deteriorated progressively, and no pulses were found distal to the femoral pulse, though all other pulses were normal. At embolectomy under local anaesthesia a 5x5 cm sheet of slimy white material was removed from the common femoral artery. This resembled the thick outer layer of a hydatid cyst. All pulses returned, and heparin was started.

The next morning signs of a left hemiparesis were noted and he developed pleuritic chest pain and confusion. Heparin was stopped, his temperature rose, and the tachycardia and tachypnoea returned. A radiograph of his chest showed some fluid in the right horizontal fissure but no other abnormality. An electrocardiogram showed the old inferior infarction, but the rhythm had become a mixture of atrial flutter

with a 2:1 block and atrial fibrillation. His hemiplegia was thought probably to be due to an embolus, and a pulmonary embolus was also suspected. Computed tomography of the head excluded intracerebral bleeding but suggested that the intracranial pressure was increased. Intravenous dexamethasone was given and infusion of heparin restarted. The sudden fever and toxic confusion were considered to be compatible with a ruptured hydatid cyst, and hydatid complement fixation tests were done. Computed tomography of the thorax and abdomen did not show any evidence of hydatid cysts in the chest, liver, or kidneys. Echocardiography, however, showed remnants of a cyst within the interventricular septum causing a ventricular septal defect. The patient improved steadily, and warfarin was started.

Eight days after his admission his chest pain returned and he again developed a fever and became distressed and tachypnoeic. Radiography of the chest and electrocardiography did not show any new changes, and he responded promptly to treatment with diamorphine and hydrocortisone. The hydatid complement fixation tests had given positive results and so albendazole was started. An echocardiogram showed resolution of the interventricular cyst with closure of the ventricular septal defect. Four weeks after his original admission he was well and was discharged taking warfarin and albendazole.

Two weeks later he was readmitted after complaining of gradually increasing pain in the lateral border of his right calf, which was exacerbated by walking. There was slight swelling and tenderness in the peroneal compartment of his right leg. As warfarin had been stopped inadvertently the previous week a spontaneous arterial thrombosis at the site of remnants of a hydatid cyst was thought likely. He was given heparin again and a second course of albendazole. Further emboli were thought unlikely as the cardiac cyst had become fibrous. He returned home one week later taking warfarin. When reviewed three months later he had minor claudication in his right calf but was otherwise well.