It is anticipated that AIDS as the content for psychiatric illness will continue to increase in frequency, as will the number of AIDS-related / functional and organic syndromes. Although only one patient was referred by the AIDS counsellor, the author would like to recommend that psychiatrists are always invited to liaise closely with those who perform AIDS counselling. It is hoped that this may help to prevent disastrous sequelae (O'Brien, 1987).

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Organic Reaction in AIDS

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A case of organic brain syndrome in a patient with AIDS is described. The implications for medical and psychiatric services are discussed and problems highlighted.

The psychiatric complications of AIDS have become increasingly recognised (Nichols, 1985; Faulstrich, 1987) since the original description of the clinical features of the disease in the USA in 1981 (Masur *et al*, 1981; Gottlieb *et al*, 1981). These usually consist of psychogenic reactions (Holland & Tross, 1985), similar to those in other life-threatening disorders, such as anxiety and depression. However, there are often also feelings of rejection by relatives and friends, and a sense of isolation.

Afflicted patients frequently suffer a variety of complications in the central nervous system (Carne & Adler, 1986). Although some of these are wellrecognised opportunistic infections, much more common is a progressive encephalopathy that has not previously been described in other immunosuppressed patients (Navia & Price, 1986). In adults, this encephalopathy characteristically manifests as a subacute dementia accompanied by motor dysfunction (Snider *et al*, 1983); however, a variety of other organic reactions accompanied by mood change have been described (Nurnberg *et al*, 1984; Hoffman, 1984), although this is one of the first cases reported in the British literature.

Case report

A 35-year-old homosexual man was admitted under section 2 of the Mental Health Act 1983 to a general hospital in September 1985, following an episode of disturbed behaviour, accompanied by incoherent conversation, disorientation in time and space, and visual hallucinations. Previous history revealed he had been diagnosed as HIVantibody seropositive eight months earlier, in New York. He had been treated for *Pneumocystis carinii* pneumonia, and treated with interferon for Kaposi's sarcoma four months before admission. There was no previous psychiatric history or family history of psychiatric illness. He was in possession of amphetamines on admission, but a urine drug screen was negative and there was no other history of drug abuse. His behaviour in hospital continued to be erratic, disinhibited and distractible. He was elated in mood. He had pressure of speech and visually hallucinated, e.g. he was seeing rabbits in the room, and misidentified people around him. His mental state fluctuated over this threemonth stay. One month after admission he remained restless and distractible, but with no evidence of pressure of speech. There was evidence of patchy disorientation in time and long- and short-term memory loss.

In hospital he was treated with major neuroleptics. However, severe Parkinsonian side-effects were experienced with the use of haloperidol and chlorpromazine. Thioridazine was the only drug tolerated by the patient. During the final month of his stay, his short-term memory improved, but he had no memory of the circumstances of his admission into hospital or his length of stay. He appeared slightly grandiose and said, "I must be the only man cured of AIDS. I've had over 1000 partners." On discharge his cognitive status was intact, but his mood slightly elevated, and he retained grandiose ideas. Ten months later he died, with a normal mental state and cognitive function.

Biochemical and haematological profiles, urinalysis, and a computerised-tomography (CT) head scan were all normal. Analysis of CSF revealed a slightly elevated protein level at 0.49 g/l, glucose 3.4 mmol/l, polymorphs $<1/mm^3$, lymphocytes 2/mm³ and a red cell count of 3000/mm³, but the CSF was not tested for HIV antibody. An EEG showed irregular 7–8 Hz alpha-activity, a moderate amount of non-focal theta-activity and diffuse beta-activity, suggestive of a mild diffuse cortical disturbance.

Discussion

The clinical picture presented by this patient conforms to the DSM-III diagnosis (American Psychiatric Association, 1980) of organic mental syndrome. The precipitant here may have been the HIV infection, as there was no evidence of an opportunistic infection, and all investigations including CSF examination were essentially normal. However, it is also possible that this was a functional psychiatric illness, although there was no previous or family psychiatric history, and there was only a single episode of illness. Although there was evidence that he had been associating with amphetamine abusers, the clinical picture described is not typical of an amphetamine psychosis, which tends to be short lived and paranoid in type. In cases such as these, investigations should include full biochemical and haematological profiles, urine screen for drugs, chest X-ray, CT scan, EEG and CSF examination to exclude organic causes.

Previous case reports have described severe memory deficit accompanied by thought disorder, grandiose delusions and dysphoria (Rundell *et al*, 1986). These are associated with dilatated ventricles on CT, and progressive deterioration in clinical picture to dementia and death. In the patient described here, however, there was an apparent resolution of the cognitive deficit and a normal CT scan. In this instance the diagnosis of HIV-antibody seropositivity antedated the psychiatric symptoms, but in at least a few described cases, the psychiatric illness antedated the diagnosis (Kermani et al, 1984; Gabel et al, 1986; Halevie-Goldman, 1987; Navia & Price, 1987). With the increasing prevalence of HIV carriers, this diagnosis should be considered in any person presenting with an organic psychosis in a high-risk group. If the prevalence increases as predicted, it could have serious implications for psychiatric services. As well as patients presenting with an acute psychosis, dementia is becoming increasingly recognised in terminal stages of AIDS, and this may present further demand on psychiatric beds which are currently being reduced in number (Davies, 1988).

The case described highlights several issues concerning management for the group of patients presenting with an acute psychosis. This patient was nursed in isolation in a general hospital with the constant supervision of psychiatric nurses. Those who present with a psychosis that may be of organic origin, and who are known to be HIV-antibody seropositive, should ideally be investigated in a medical ward to identify any organic lesion and its treatability. However, patients with a previous history of psychiatric illness who become HIV positive, and HIV-positive sufferers who develop an acute psychotic illness, may best be nursed in a psychiatric hospital by nurses with experience in dealing with psychiatric patients.

Patients need to be nursed in isolation if there is a particular risk of spreading infection (Geddes, 1986), e.g. those with profuse diarrhoea, or those with a psychosis who are likely to be violent. There are particular risks in treating patients with a psychosis who are HIV positive-these include possible transmission of the virus due to blood spilled in a violent incident or from self-injury; injuries sustained while giving injections; and an increased risk of infection by sexual intercourse with the patient who is disinhibited. These risks may be minimised by taking precautions when handling patients, and barrier nursing. It is also important to provide support and education for staff when handling HIVpositive patients, and to offer advice and information about factors contributing to HIV infection (Nichols, 1985).

As psychiatric complications in patients who are HIV positive and those with AIDS become more common, resources in psychiatric units will have to be provided to meet the needs of the patients.

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Gilles de la Tourette's Syndrome Amelioration Following Acute Akinesia During Lorazepam Withdrawal

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A case of Gilles de la Tourette's syndrome (TS) is described, in which a state of akinesia developed during an attempt to withdraw lorazepam by diazepam substitution. This was followed by sustained amelioration of the TS symptoms.

Aetological theories of Gilles de la Tourette's syndrome (TS) have used both psychological and biological models (Meyer & Rose, 1986). Successful treatment of TS with haloperidol (Shapiro & Shapiro, 1968) led to the suggestion that dop-aminergic overactivity may be implicated (Snyder *et al*, 1970), although there is no direct evidence to support this hypothesis.

We report a case of TS in which clinical amelioration followed an acute akinetic state which coincided with an attempt to withdraw the patient from lorazepam.

Case report

A 47-year-old, married, Caucasian man was initially referred in June 1984 with a 14-month history of agitation, decreased concentration, irritability and depression; he repeatedly banged his forehead with his fist and scratched his face. There was no obvious precipitating factor. His premorbid personality was described as anxious and shy. A diagnosis of depressive illness was made and he was treated with dothiepin (50 mg t.d.s.). His depressive mood improved but his other symptoms remained. He was treated with behavioural psychotherapy over subsequent months, but his symptoms persisted, and he