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Fear of AIDS

SIR: There have been some reports (Miller *et al*, 1985; Jacob *et al*, 1987) of psychological problems among people who are uninfected but fear that they might have AIDS (acquired immune deficiency syndrome). We would like to present a case of acute obsessional neurosis which responded well to clomipramine infusion.

Case report: A 27-year-old married man was admitted to the psychiatric unit as an emergency. He was very anxious and had obsessive ruminations about having developed AIDS. He had been worried about this since he borrowed a razor from a workmate who had had a recent viral illness and then heard rumours that this man might have AIDS.

He went into a local restaurant with his wife for a meal, and the manager of this restaurant later died of AIDS. His wife was presented with a rose by the manager, who pricked his finger on it, and he thought that he might also have pricked his finger on the same rose. He also thought that he might have had sex with the manager in the toilet, but he realised that this was absurd. Nevertheless, he went back to check the toilet in the restaurant to reassure himself that this incident did not occur. He denied having any homosexual experiences, but was worried that he might become homosexual because of these ruminations.

His wife was pregnant at the time, and he was afraid that he might have infected his wife and unborn baby with AIDS. He was self-accusative and thought of cutting his wrist, but was worried in case his wife would not be able to collect the insurance money. He then entertained the idea of committing suicide in such a way as it to appear accidental death. It was at this point that he was admitted to hospital.

There was no personal or family history of psychiatric illness. The patient had an uneventful school career and became a Scientific Officer. He was happily married with two children. He described himself as an "introvert and a worrying type of person". His only past medical history of note was a skull fracture sustained during a road traffic accident at the age of 20, after which he was unconscious for three weeks and later had an isolated epileptic seizure.

On examination he looked very worried and complained of having disturbed sleep, but his appetite was fair. He was well orientated and his memory was intact. HIV antibody tests were negative and EEG was normal.

He was diagnosed as suffering from an acute obsessional neurosis in an anxiety prone personality and commenced on daily clomipramine infusions.

He made a good symptomatic recovery after ten days and remained symptom-free on oral clomipramine (25 mg t.d.s.) eight months later.

This case presents a problem of differential diagnosis of anxiety state, depressive illness, delusional state, and organic condition. Although he exhibited the psychic symptoms of anxiety, the patient lacked the somatic symptoms which makes anxiety state unlikely. His anxious mood and lack of biological features exclude the possibility of a depressive illness. Retention of insight ruled out a delusional condition, and lack of cognitive and memory impairment made an organic condition unlikely. It would appear that the anxiety and some of the depressive features were inter-related to an obsessional illness.

There have been reports of the development of obsessional illness as a consequence of head injury (McKeon *et al*, 1984). In this case, the seven-year interval makes head injury an unlikely cause, but it may have affected his personality. It seems that the predisposed personality and media influence contributed to the genesis of his obsessional illness. This case also highlights the point made by O'Brien (1987) that a wide range of psychiatric illness may present with fear of AIDS, and shows that obsessional illness is one of them.

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Mania in a Case of Eale's Disease

SIR: Eale's disease is characterised by recurrent retinal and vitreous haemmorhages with retinal perivasculitis, predominantly affecting the veins (Duke-Elder, 1967). There have been several reports of associated neurological involvement (Singhal and Dastur, 1976), but psychiatric complications are unreported. In this report we present a man with Eale's disease and neuropsychiatric complications.

Case report: A 34-year-old married male presented to our out-patient department with 2 years history of episodic loss of vision and a one-month history of disturbed behaviour and restlessness. There was no other contributory family or past history. For 2 years he had been having episodic loss of vision, for which he had been on steroids with poor compliance. He had been off steroids for six months prior to consultation. For one month before consultation he had been singing, over-confident, restless, talking excessively, and not sleeping adequately. He was brought in because he was unmanageable at home. He was in hospital for a period of 45 days. Physical examination revealed only light perception in the right eye, with vitreous haemmorhages. With his left eye, vision was 6/12 and fundal examination revealed peripheral gliosis and haemmorhages. A right lateral rectus palsy was present.

Mental status examination revealed increased psychomotor activity, pressure of speech, grandiose delusions of ability and identity, impaired social judgement, intact cognitive functions, and absent insight. He was treated symptomatically with phenothiazines, and the episode remitted about 3 weeks after discharge. Subsequently, he had another similar episode after one year which remitted after 5 weeks; this also required admission.

Routine investigations were within nomal limits: ESR = 24; STS non-reactive; LE cells negative; Rh factor negative; CSF cells nil; sugar 50 mg%; protein 28 mg%; globulins negative. ENMG showed motor nerve involvement in the left lower limb with myelin axon. EEG was within normal limits, and CT scan was normal. Muscle biopsy taken from the gastrocnemius was normal. In the biopsy of the sural nerve there was a minimal increase in the peri and endoneural connective tissue, with a marginal fall-out of myelinated tubules on the K. Pal stain. This was suggestive of an ischemic process.

The aetiology of Eale's disease is not known, although tuberculin hypersensitivity is most widely agreed upon (Dastor & Udani, 1966). Haarr, (1964) has pointed out that the histological appearances of vasculitis resemble those of erythema nodosum.

The diagnostic possibilities include reactive excitement, which is unlikely as the patient did not satisfy criteria for diagnosis of reactive psychosis. The two illnesses could have occurred due to a rare unreported association. The third possibility is of secondary mania (Krauthamer & Klerman, 1978) with Eale's disease. The possibility of steroid psychosis seems unlikely because of the gap of five months between cessation of steroid intake and onset of psychosis. It would be interesting in this case to regard vasculitis as the cause of all three defects; namely, in the encephalon, the peripheral motor nerve, and in the eye.

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Fluvoxamine and Hepatic Function

SIR: Fluvoxamine maleate is a recently introduced antidepressant, marketed as a specific 5-HT reuptake inhibitor (*Drug and Therapeutics Bulletin*, 1988). Caution in prescribing fluvoxamine is recommended for patients with renal or hepatic impairment. I would like to draw your attention to a possible serious side-effect of fluvoxamine therapy.

Case report: A 57-year-old office worker was admitted to hospital for assessment of low mood and suicidal ideation. He had become increasingly preoccupied and withdrawn over the previous three months and had had to stop work because of anxiety symptoms. There was no significant past medical or psychiatric history. There was no history of alcohol abuse from the patient, and this was confirmed by his wife and children. On admission his physical examination and initial investigations, including ECG and chest X-ray, were normal (mean cell volume 90 fl and gamma-glutaryl transferase 50 U/litre). He was started on fluvoxamine (100 mg b.d.). He was maintained on this dose for 18 weeks. There was no significant improvement in his mood, and he remained anxious and preoccupied. A repeat blood screen after 3 weeks of therapy showed a γ-GT of 176 U/litre. When this was repeated the γ-GT had remained elevated at 167 U/litre. The fluvoxamine was discontinued. Abdominal examination and ultrasound confirmed an enlarged liver with echo characteristics of fatty change. After stopping fluvoxamine therapy his liver function tests gradually returned to normal. Five weeks after ceasing fluvoxamine his γ-GT was back to 59 U/litre.

It seems most probable that fluvoxamine was responsible for the deterioration in hepatic function, with the possibility that it was responsible for the observed fatty change. This is the first such reported