5-fluorocytosine is positive in serum and it is associated with increased trends in Cryptococcus neoformans. We were interested in the epidemiology of this fungal infection in different populations. This is because the lack of correlation between clinical presentation and cryptococcal meningitis is often overlooked in elderly patients.

In this case, however, despite a brain biopsy in an affected area, it was not very useful, because routine haematoxylin and eosin stain was negative for T. gondii, and culture from laboratory animal was necessarily slow. The immunohistological staining with peroxidase anti-peroxidase stain method and specific diagnosis procedure for T. gondii must be recommended, particularly in immunodeficient patients needing a rapid and specific diagnosis, critical to the directing of appropriate and urgent therapy for a potentially curable condition.

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References


Li et al reply

We were interested to see Drs Kurland and Mulder’s comments on age specific incidence rates in motor neuron disease, and note that our findings are in agreement with theirs. Clearly, this implies a common causative factor operating in these widely separated environments and it would be interesting to compare cohort data for these different populations. We agree with Kurland and Mulder that the diagnosis of motor neuron disease is probably often overlooked in elderly patients, thus resulting in an under-estimate of incidence rates in older populations. This is a common limitation of epidemiological data taken from retrospective surveys of case-notes, but in our work we have tried to exclude this factor as far as possible by utilising data from two separate Health Authorities, both with relatively well-organised neurological services. Similar trends were found in the two sets of data.

Cryptococcal meningitis and cerebral toxoplasmosis in a patient with acquired immunodeficiency syndrome.

Sir: We were very interested in the short report of Bahls and Sumi about the documented simultaneous infection of the central nervous system with Toxoplasma gondii and Cryptococcus neoformans in an AIDS patient. We have also observed such an association on the same underlying disease. A 26 year old homosexual man was admitted with fever, cough and meningitis. Transbronchial biopsy, blood and CSF cultures showed Cryptococcus neoformans. CD4/CD8 lymphocyte ratio was under 0.2. Serum sample was LAV/HTLV III antibody positive by two different techniques (ELISA, Western blot). Despite amphotericin B and 5-fluorocytosine in combination, seizures and confusion with right hemiparesis appeared. The cranial computed tomographic scan revealed three ring-enhancing mass lesions. Echoguided neurosurgical puncture of the left parietal mass lesion allowed brain biopsy which showed Cryptococcus neoformans (PAS stain). The patient died 4 weeks later. Toxoplasmosis serological and CSF tests were non-diagnostic.

Brain culture from mice after intraperitoneal inoculation were positive for Toxoplasma gondii.

This new case emphasises the possibility of an infectious agent hiding another. Brain biopsy is indicated in such patients because of the lack of correlation between clinical presentation, CT scan appearance of mass lesions in the central nervous system, isolation of an infectious agent anywhere and the specific diagnosis of mass lesions. In this case, however, despite a brain biopsy in an affected area, it was not very useful because routine haematoxylin and eosin stain was negative for T. gondii, and culture from laboratory animal was necessarily slow. The immunohistological staining with peroxidase anti-peroxidase stain method and specific diagnosis procedure for T. gondii must be recommended, particularly in immunodeficient patients needing a rapid and specific diagnosis, critical to the directing of appropriate and urgent therapy for a potentially curable condition.

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Matters arising

Cryptococcal meningitis and cerebral toxoplasmosis in AIDS: another case report.

Sir: We read with interest the short report of Bahls and Sumi on the association of cryptococcal meningitis and cerebral toxoplasmosis in a patient with Acquired Immune Deficiency Syndrome (AIDS) and we would like to document a further case.

A 34 year old homosexual male was admitted with a 4 months history of slight fever, cough with occasional haemoptysis and weight loss. Three months later he developed a headache. One day before admission the patient became less alert. Neurological examination showed a comatose patient with nuchal rigidity and left facial palsy. The diagnosis of AIDS and cryptococcal meningitis was made by clinical and laboratory evaluation and was treated with amphotericine B and 5-fluorocytosine. After 2 months he developed progressive hemiparesis and CT scan showed multiple, ring-enhancing, low-density lesions. Slightly increased serologic titres against Toxoplasma gondii were detected and therapy with pyrimethamine and sulfonamides was consequently started. In the next week the patient quickly improved, the CT lesions disappeared and he soon became asymptomatic. Unfortunately, after one and a half months, therapy was discontinued because severe pancytopenia had developed, and he progressively experienced again right limb weakness and aphasia. Later, he died of pneumonitis from Pneumocystis carinii. Pathological findings in the central nervous system revealed cryptococcal meningoencephalitis, multiple necrotising abscesses for Toxoplasma gondii and medulla and spinal cord infection by cytomegalovirus.

In our opinion, taking into account that cryptococcus and toxoplasma are both common causes of infection in patients with AIDS, their association may frequently occur. Consequently, we suggest the immediate onset of therapy with pyrimethamine and sulfadiazine if the diagnosis of toxoplasmosis is suspected in patients with cryptococcal meningitis and AIDS. Brain biopsy should be reserved for cases with poor response to treatment because its use is limited by its potential morbidity and the presence of false-negative results.

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Paroxysmal myoclonic dystonia with vocalisations

Sir: Feinberg et al described four patients whom they distinguished from the large majority of patients with Gilles de la Tourette syndrome on the basis of four “atypical” characteristics: (1) paroxysmal bursts of regular, repetitive, rhythmic, stereotypic, coordinated, simultaneous and bilateral myoclonus and vocalisations often with tonic symptoms; (2) presence of hyperactivity and attentional and learning disorders; (3) interference of symptoms with voluntary functioning; and (4) ineffectiveness of haloperidol. I have examined Case 1 and have considered him not to have the lightning-like jerks of myoclonus. Rather, he has virtually all the features of Gilles de la Tourette syndrome. I presented videotapes of his paroxysmal bursts of abnormal movements at the Unusual Movement Disorder Seminar held 29 May 1986, at the meeting of the American Academy of Neurology. The audience of 160 neurologists was in complete agreement that this young man suffered from the tic syndrome, commonly known as Gilles de la Tourette syndrome.

Let me address the four features of the disorder that the authors considered atypical. The first are the “paroxysmal bursts of regular, repetitive, rhythmic, stereotypic, coordinated, simultaneous and bilateral abnormal myoclonus (sic) and vocalisations often with tonic symptoms.” Paroxysmal bursts of stereotypic and coordinated movements are the hallmark of tics, and, in fact, are not encountered in other movement disorders. Paroxysmal dyskinesias of dystonia and chorea are well recognised, and paroxysmal tremor has been reported, but none of these are coordinated sequences of complex movements that are so typical of tics. Vocalisations, also, are a classical feature of the Gilles de la Tourette syndrome, and are only encountered elsewhere in Meige syndrome and as a feature of akathisia. The vocalisations of Case 1 included coprolalia, which is almost diagnostic for Gilles de la Tourette syndrome. Bursts of repetitive, rhythmic, bilateral movements are not commonly seen as part of the motor tic spectrum, but I see no reason why this phenomenology cannot be included within the realm of motor tics. Indeed, a minority of patients seen by me with otherwise typical features of tics have this feature. Tonic symptoms have long been recognised as a feature of tics, and today are commonly referred to as dystonic tics.

It is not clear why Feinberg et al listed “presence of hyperactivity and attentional and learning disorders” as atypical for patients with tics. Several investigators report that attention deficit disorder occurs in approximately 50% of patients with Gilles de la Tourette syndrome. Interference of voluntary functioning by symptoms does occur in Case 1. When he has a burst of the repetitive, rhythmic flexion movements of his arms, he stops speaking, other than occasional vocalisations. There is no loss of contact with the environment; rather, it appears as if his mind is actively and compulsively engaged in other activity, which is what he and other patients with tics who have this symptom inform me. Like bursts of repetitive movements, these simultaneously mind-occupying states should be considered within the spectrum of tics.

The final point raised by Feinberg et al is the ineffectiveness of haloperidol to suppress these paroxysmal motor bursts. But responsibility to medication is not an acceptable criterion for the diagnosis of tics! As an aside, and for a point of information, based on a telephone conversation I had with the patient on 8 May 1986, he considers himself 80% improved on fluphenazine and clonidine. Thus, the former drug, which blocks dopamine, similar to haloperidol, has benefited the patient.

I would like to propose that authors who wish to describe new or variants of movement disorders should also submit a videotape showing the abnormal movements. The demonstration of the videotape can most easily be accomplished in the newly founded journal Movement Disorders which includes a video format in addition to the classical written format. Those interested in this new journal can contact its publisher, Raven Press. I have obtained written permission from Case 1 to publish his videotape, and I will append it as part of the review of tics by Jankovic and Fahn.

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