immune haemolytic anaemia in a patient treated with chloropropamide; in vitro testing showed cross reactivity with gibelfamicide and related compounds. Immune haemolysis in vivo caused by gibelfamicide, however, does not seem to have been reported before. The mechanism of haemolysis was compatible with the cephaline type, as a Coombs test gave a positive result with both anti-IgG and anti-C3 and anti-C4. This result is caused by a non-specific binding of serum proteins, including IgG and complement, to a damaged or altered erythrocyte surface. The antiluglobulin test may give a positive result for many months, as was seen in our patient.

Selective IgA deficiency is the most common form of dysgammaglobulinaemia and is, for unknown reasons, often associated with immunological disorders. Whether the IgA deficiency and the drug induced haemolytic anaemia in our patient were connected was uncertain.

We are grateful to Hoechst Pharmaceuticals for generous supplies of gibelfamicide and to Dr SM Wood, of the Department of Health and Social Security, for valuable information.


(Accepted 15 May 1987)

Medical Department B, University of Bergen, Haukeland Hospital, Bergen, Norway.
O B NATAAS, MD, consultant
I NESTHUS, MD, consultant
Correspondence to: Dr Nataas.

Lyme disease presenting as recurrent acute meningitis

The neurological manifestations of Lyme disease have been increasingly recognised in Britain,1 Muhlemann and Wright identifying 21 cases with such symptoms last year in the United Kingdom and Irish Republic.2 The neurological manifestations are fluctuating symptoms of meningoencephalitis with superimposed cranial or peripheral radiculopathy, but although these features are often intermittent, the duration of meningitic symptoms is more like that of chronic meningitis.3 We report on a patient with Lyme disease who presented with recurrent acute meningitis and no history of tick bite, cutaneous eruptions, or other systemic symptoms.

Case report
A 55 year old man was admitted in October 1984 with a history of feeling unwell and occipital headaches for two days. He was feverish, and clinical examination showed only slight stiffness of the neck on extreme flexion. Investigations showed a normal full blood count. Studies of cerebrospinal fluid showed a raised protein concentration (2-4 g/l), a glucose concentration of 5-3 mmol/l, and 240 x 10^6 lymphocytes/100^6 lymphocytes); tests for the presence of bacteria, fungi, and viruses yielded negative results. He was treated with intravenous penicillin 16 megunits daily for seven days and recovered completely within 12 days. Sixteen months later he was admitted with a similar illness, now with slight stiffness of the neck on examination; there was no positive history of tick bite, cutaneous eruptions, or other systemic symptoms.

Seven months later he again presented with similar symptoms of one day’s duration; examination showed a fever (38°C) with slight stiffness of the neck and no other physical abnormality. Cerebrospinal fluid contained 60 x 10^6 leucocytes/1 (95% polymorphs); the protein concentration was 1-8 g/l and glucose concentration 4-2 mmol/l. Cultures for Mycobacterium tuberculosis and tests for fungi and viruses yielded negative results. A repeat examination of cerebrospinal fluid after seven days showed 196 x 10^6 leucocytes/1 (100% lymphocytes), a protein concentration of 1-13 g/l, and a glucose concentration of 4-7 mmol/l. A computed tomogram of the brain showed no abnormality. Intravenous penicillin 16 megunits daily was started on the second day of admission, and he made a complete recovery within the next 24 hours. Treatment was continued for two weeks, and he had no further complications. An indirect immunofluorescence test for antibodies to Borrelia burgdorferi yielded a positive result. Seven weeks after admission his serum IgG titre was 1/256 and IgM titre negative. A further sample of serum 10 days later had an IgG titre of 1/64 and a negative IgM titre.

Comment
Our patient had two symptom free periods of 16 and seven months, respectively, between three episodes of acute meningitis. Serological tests were done only during the last admission. Although we cannot exclude the possibility of re-infection, which is known to occur in Lyme disease,1 we believe that these episodes were reactions to Borrelia burgdorferi as the patient had worked on a road in a deer forest. He had not done this before the second and third episodes, and the presenting symptoms were similar on each admission. The subsequent episodes are therefore unlikely to have been due to reinfection as he did not develop the more common dermatological and systemic symptoms before either of them. Inadequate treatment of the first episode is also unlikely because the intervals between attacks were too long.

Recurrent meningitis is often difficult to diagnose. The natural course of bacterial, fungal, and viral meningitis is well understood, but in some cases it may be exceptionally difficult to establish the source of infection. When only neurological symptoms are present a diagnosis of Lyme disease may not be obvious.2 Patients with recurrent episodes of aseptic meningitis should have serological tests for B burgdorferi even if there is no history of tick bite or cutaneous lesions; if Lyme disease is diagnosed early high dose intravenous penicillin can shorten the duration of meningitic symptoms.

We thank Dr D J M Wright, Department of Medical Microbiology, Charing Cross Hospital, London, for his help with the serological tests for Lyme disease.


(Accepted 13 May 1987)

Wrexham Maelor Hospital, Wrexham, Clwyd LL13 7TD
G S PAL, MB, MRCP, medical registrar
J T BAKER, MS, FRCP, consultant physician
PK D HUMPHREY, DM, MRCP, consultant neurologist (also at Walton Hospital, Liverpool)
Correspondence to: Dr Pal.

Bromocriptine induced impotence in Parkinson’s disease

Increased drive may be common in patients with Parkinson’s disease receiving treatment with levodopa or dopamine agonists.1 The incidence of a true “aphrodisiac” effect, however, is difficult to assess. Frankly hypersexual or deviant behaviour occurs rarely and is usually associated with an acute brain syndrome or history of hypersexuality.2 Though diminished libido is a common feature of Parkinson’s disease,1 impotence related to dopaminergic treatment has not been reported. We describe four men with idiopathic Parkinson’s disease with no history of sexual difficulty who spontaneously complained of impotence associated with dopamine agonist (bromocriptine) treatment.

Case reports
Case 1—A 59 year old man was receiving treatment with incremental doses of bromocriptine over time with levodopa. Although he believed that these episodes were reactions to the drug, the use of bromocriptine was continued. At times daily he experienced inability to sustain erections. When the dose was increased from 45 mg to 60 mg and good control of the parkinsonian symptoms had been gained, he became impotent. In spite of noticeably increased sexual urge he achieved erections only transiently and penetration was impossible. When the dose was reduced to 30 mg daily erectile function returned to normal, with worsening of the parkinsonian symptoms. Subsequent increases in the dose of bromocriptine invariably produced impotence. A combined regimen, however, of levodopa (Sinemet-Plus, one tablet three times daily) and bromocriptine (5 mg three times daily) resulted in good control of Parkinson’s disease and normal sexual drive and erectile function.

Case 2—A 34 year old man was receiving incremental doses of bromocriptine. After four months (22-5 mg daily) he experienced inability to sustain erections. When the dose was reduced to 11:25 mg daily normal sexual function returned.