ticularly when the preparation contains povidone. Unfortunately, our patient died from complications of unnecessary treatment given because the unfamiliar molar units used to report the phenobarbitone concentration were misinterpreted.

ADDENDUM—Since writing this report we have encountered a further case of serious but eventually reversible respiratory failure in a 46 year old man after aspiration of Medicoal.

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Lyme disease with acute purulent meningitis

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Cases of Lyme disease with neurological manifestations have recently been reported in Britain[1,2] and were well documented in the original reports of this disease from Connecticut.[3] These neurological complications consist of a chronic lymphocytic meningitis often associated with cranial and peripheral neuritis. We report a case of Lyme disease with the undescribed complication of acute purulent meningitis.

Case report

A 69 year old man presented in May 1987 with a two month history of cough, haemoptysis, and pleuritic pain. He was under regular review for a transitional cell tumour of the bladder and had suffered a myocardial infarction six months earlier. A chest radiograph showed left upper lobe consolidation, but sputum cultures were negative. Appearances at bronchoscopy were normal, and transbronchial biopsy specimens showed an interstitial pneumonitis. Seven days after bronchoscopy he was readmitted to hospital with severe headache, photophobia, a fever of 38·5°C, and severe neck stiffness. The peripheral white cell count was $16{\cdot}8\times10^9$/l (89% polymorphs), and lumbar puncture yielded a purulent cerebrospinal fluid containing 2750×10^9 polymorphonuclear leucocytes per litre: protein concentration was raised at 1·6 g/l and glucose reduced at 2·1 mmol/l (blood glucose 8·2 mmol/l). No organisms were seen on Gram, auramine, or Ziehl-Neelson staining. Latex coagglutination testing (Inverclyde Biologicals) of the cerebrospinal fluid was negative for pneumococcal capsular antigen, cultures of cerebrospinal fluid and blood remained sterile, and serological testing for viruses and *Mycoplasma pneumoniae* gave negative results.

He was given ampicillin and chloramphenicol for 14 days and antituberculous treatment (rifampicin, pyrazinamide, and isoniazid) for the first six days. A repeat lumbar puncture after seven days showed a fall in the polymorphonuclear count to 1020×10^9/l. He developed a transient right facial palsy. Serial computed tomograms showed the development of an area of infarction in the right parietal region. A repeat chest radiograph showed resolution of the original consolidation. He made a partial recovery, but three months later, having returned to the care of his general practitioner, he remained disorientated, ataxic, and incontinent. Serological tests at this stage, arranged by his general practitioner, showed IgM antibodies to *Borrelia burgdorferi* at a titre of 1/512 by indirect immunofluorescence. Retrospective examination of serum obtained at the time of his acute meningitis gave negative results, while serum taken five months later showed IgG but not IgM antibody at a titre of 1/256, indicating a recent infection occurring around the time of the meningitis. He was negative for antinuclear antibodies, rheumatoid factor, and leptospira antibodies, and gave negative results to *Treponema pallidum* haemagglutination and Venereal Disease Research Laboratory tests. Six months after the initial presentation, when he had almost fully recovered, he developed a peripheral neuritis with a right foot drop, which improved after treatment with intravenous benzylpenicillin 12 MU daily for two weeks. He recalled having had several tick bites while working on his farm but gave no history of a rash or arthropathy.

Comment

Our patient probably had Lyme disease complicated by acute pyogenic meningitis, though this has not been described before. He had the classic triad of neurological manifestations—meningitis, cranial neuritis, and late peripheral neuritis[3]—and his serological findings were consistent with recent infection by *B burgdorferi*. Other known causes of serological cross reactivity such as syphilis and leptospirosis were excluded.[4]

We do not know why this patient responded to infection with *B burgdorferi* with acute pyogenic meningitis, cerebral infarction suggestive of an associated vasculitis, and possibly pneumonitis. The virulence of the infecting organism may be relevant. North American and European strains of *B burgdorferi* differ in their major proteins so we may expect to see differences between the disease in the United States and Britain.[5] Erythema chronicum migrans and arthropathy are less common in European than in American cases.[5] At this stage in our knowledge of Lyme disease in Britain we would draw attention by this report to unusual presentations of the disease, which may reflect differences from the syndrome originally described in Connecticut.

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