Letters to the Editor

Chronic diarrhoea associated with Septata intestinalis

Sir, Septata intestinalis is a recently described microsporidium which has been implicated in causing chronic diarrhoea and/or disseminated infections in AIDS patients. Unlike infection with Enterocytozoon bieneusi, the other common microsporidium isolated from stools of AIDS patients with chronic diarrhoea, infection with S. intestinalis responds to treatment with albendazole. To our knowledge this is the first case report of the parasite from the UK.

A 28-year-old man presented with a history of chronic diarrhoea over a period of four months. Initially he passed two stools per day increasing to seven or eight times per day. These stools were watery with no blood or mucus. Prior to admission he began to spike fever and complained of loss of weight from 10 st to 8 st. The patient spent 18 months in Tokyo and then visited Borneo for a short while before returning to the UK six months ago. Whilst abroad he was in good health. On examination he looked thin, dehydrated and febrile and, except for an enlarged right supraclavicular lymph node, there were no other clinical findings. Ultrasonography of the abdomen was normal but the chest X-ray revealed a right supraclavicular lymph node, right paratracheal lymph node and bilateral hilar lymph node enlargement. The right supraclavicular lymph node was aspirated and showed auramine-positive bacilli. The patient denied any risk factors for HIV.

Repeat stool samples were negative for routine culture of bacteria. A routine study of stools for parasites was also negative and therefore stools were sent to the London School of Hygiene and Tropical Medicine for further studies. Diagnosis was first made from unspun faecal samples, which were examined for ova, cysts and parasites (OCP) by formol ether concentration, for cryptosporidium and cyclospora using the phenol auramine and modified Ziehl Neelsen methods and for microsporidia by the Kokoskin strong trichrome method. No OCP or coccidians were detected in the sample, but very scanty microsporidium spores were found. A further sample taken 14 days later was also negative for OCP and coccidians, but contained a larger number of spores, which morphologically were consistent with those of S. intestinalis. As this organism is known to disseminate to other sites in the body, especially the urinary tract, urine samples were obtained from the patient and stained for spores; none were found.

The presence of S. intestinalis in the stool and the auramine-positive bacilli from lymph node aspirate indicated that the patient was immunocompromised. He was therefore advised to have an HIV test which was found to be positive. His CD4 count was 0.06 x 10⁹/l and CD8 count was 0.81 x 10⁹/l, with a significant absolute T helper cell lymphopenia. The patient’s diarrhoea was treated with oral albendazole 400 mg bid for 28 days and subsequently weaned off. The inciden-

tal finding of microsporidal spores in this patient underscores the need for laboratories to examine all faecal samples from patients with chronic diarrhoea for spores, irrespective of known or unknown immunological status. Also, where S. intestinalis is the infecting organism, treatment with albendazole should be given early to prevent possible dissemination to other sites in the body.

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Septata intestinalis
- associated with chronic diarrhoea/dissemination in immunocompromised patients
- responds to treatment with albendazole

Transient ischaemic attack, infectious meningitis, or neurosarcoidosis?

Sir, We report the case of a patient with neurosarcoïdosis who presented with unusual clinical features and cerebrospinal fluid (CSF) alterations.

Case report
A 24-year-old man was admitted for evaluation of a transient episode of aphasia. A few months prior to admission, recurrent early morning nausea developed. He also experienced a transient right facial anaesthesia, and an episode of laryngitis that subsided after a short period of oral corticoid treatment. The patient's general status was excellent. Neurologic examination was normal. Physical examination revealed Perthes-Jüngling osteitis, and maculopapular lesions of the skin exhibiting sarcoid granuloma on biopsy. Electroencephalography showed slow waves over the left temporal area. Brain computed tomography (CT) disclosed slight ventricular

enlargement and a small lacunar infarction in the left internal capsular area. Magnetic resonance imaging (MRI) of the brain revealed a severe leptomeningitis (multiple small nodular hyperintense lesions on T2-weighted images, enhanced by gadolinium). In the right capsulohamatic area, hyperintense lesions on the T2-weighted images were suggestive of an ischaemic process.

CSF, as determined on two different days, contained 85 leucocytes/mm³, with 42% polymorphs and 44% lymphocytes. Cytologic study disclosed numerous altered polymorphs but no carcinomatous cells. CSF glucose level was 10 mg/dl (blood glucose level drawn simultaneously was 90 mg/dl) and protein level was 1780 mg/dl.

An extensive search for an infectious process proved negative. Chest X-ray revealed slight hilar adenopathy, and bronchialvesolar lavage showed marked lymphocytosis.

The patient was treated with prednisone (1 mg/kg daily). During the initial period of treatment, this was combined with antimicrobial chemotherapy. One year later, the patient is doing well. His neurologic status has remained normal, the morning nausea has subsided, CSF has returned to normal, and the brain MRI no longer shows leptomeningitis. Tolerance of treatment is good.

Comment
In this patient, neurosarcoidosis was diagnosed on the basis of skin biopsy, Perthes-Jüngling osteitis, chest X-ray, bronchoalveolar lavage, neurologic symptoms, and meningitis without evidence of neoplastic or infectious disease. The patient's clinical presentation was compatible with transient ischaemic attacks. Such cases have rarely been reported in neurosarcoidosis and must be distinguished from cases involving seizures or the paroxysmal cardiac anomalies associated with sarcoidosis. They might be due to granulomatous arteritis.

Causes of low CSF glucose and high CSF neutrophil count
- infection: bacterial meningitis, fungal meningitis
- other unusual causes: brain abscesses, CMV polyradiculomyelitis in AIDS, amoebic meningitis, chemical meningitis, connective tissue disorders, vasculitis

Learning points
- sarcoidosis may present as a transient ischaemic attack
- meningitis with high CSF neutrophil count should not rule out sarcoidosis

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