Actinomycotic brain abscess following abdominal suppurati

Sir: Actinomycotic infection of the brain is very uncommon; Actinomyces israelii was isolated from only one of 60 brain abscesses treated surgically. The organism usually reaches the brain by haematogenous spread from an intra-thoracic source. We report a case of an actinomycotic brain abscess which presented 4 months after the drainage of a large abdominal abscess from which anaerobic organisms; but not Actinomyces israelii were isolated.

A 42 year old male clerk had been treated for a series of septic lesions since April 1985. The first was a right heel abscess which resolved after oral penicillin therapy. A left paracolic abscess required drainage in May 1985. The abscess cavity had two components, one of which extended into the subcutaneous tissues over the left loin and buttock. Sigmoidoscopy was normal. He made an excellent recovery without antibiotics over a period of 3 weeks. Culture of the drained pus yielded a heavy growth of Bacteroides fragilis. Two months later, he developed a subungal infection of the right third toe and a "carbuncle" over the left scapula; these resolved after a short course of erythromycin. A week later, he was admitted with headache and left sided weakness.

On examination he was slightly drowsy with a mild pyrexia, normal optic discs, a mild left hemiparesis and extensor plantar responses. The peripheral blood leucocyte count was 17.2 x 10^9/l with 79.1% neutrophils. A chest radiograph was unremarkable. Plasma glucose, creatinine and liver function tests were normal. A CT brain scan revealed space occupation by a diffuse low density lesion in the right frontal lobe which showed ring enhancement after intravenous injection of contrast. A smaller area of low density, which did not enhance, was seen posteriorly in the left hemisphere. Cerebral abscess formation in the right frontal lobe with a separate area of cerebritis in the left occipital lobe was thought likely. Chloramphenicol, fluoxacillin and metronidazole together with dexamethasone produced a dramatic improvement in symptoms over 24 hours. Subsequently, when dexamethasone was withdrawn, he again became drowsy and left sided weakness returned. A repeat CT brain scan showed unaltered appearances and an exploratory craniotomy was therefore performed 7 days after admission. An encapsulated abscess was found and totally excised. Four days after operation all neurological symptoms and signs had resolved.

Histological examination showed that the wall of the abscess was composed of fibroblastic tissue with a heavy chronic inflammatory cell infiltrate. Gram stains of the contained pus revealed positive branching bacilli and culture yielded a growth of Actinomyces israelii, sensitive to penicillin. The initial antibiotic regime was replaced by intravenous penicillin, 12 g daily, which was continued for 6 weeks. Oral amoxyccillin, 6 g daily, was given for a further 6 months. A CT brain scan 2 months after operation showed no features of intracranial infection. The patient remains asymptomatic with no neurological signs after 24 months follow-up.

A thorough search was made for the primary source of infection. Full ENT and dental examination revealed no significant abnormality. A barium enema, however, showed a narrow, non-distensible area in the sigmoid colon where the mucosal pattern was distorted (fig). Tests of polymorphonuclear cell function were performed after steroid therapy had been withdrawn and did not differ significantly from a control. Fasting blood glucose and serum immunoglobulins were within normal limits.

Cases of actinomycotic brain abscesses without an apparent primary source of infection have been reported though some authorities doubt that this occurs. Perhaps owing to the previous treatment with penicillin, Actinomyces israelii was not cultured from the abdominal abscess in our patient, but we believe this was the likely source of infection. Though left sided actinomycotic abdominal abscesses are rare, the organism may gain access to the peritoneal cavity through a diseased sigmoid colon. In one reported case with bacteriological evidence of actinomycotic infection, barium enema abnormalities were very similar to those found in our case. Our case also demonstrates that, although a trial of blind antibiotic therapy may be justified in the initial treatment of brain abscesses, particularly if multiple, accurate bacteriological information should ideally be obtained from operative specimens when clinical and radiological improvement is not evident. Unless this principle is adhered to, unusual intracranial infection will escape diagnosis and the opportunity for curative treatment will be missed.

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Pleural and retroperitoneal fibrosis associated with bromocriptine treatment

Sir: Both retroperitoneal fibrosis\(^1\)\(^2\) and pleural pulmonary fibrosis\(^3\)\(^4\) have recently been reported following treatment with bromocriptine. We describe the first case in which bromocriptine treatment was associated with both conditions, which may be manifestations of the same disease process.\(^5\)\(^6\)

Mr W, born in 1932, developed Parkinson's disease in 1976. He presented in 1985 with a 2 month history of dyspnoea. He had lost about 13 kg in weight in the preceding year and was complaining of pain in the low back, radiating to the genitalia. The only abnormal finding on examination was dullness to percussion with reduced air entry in the lower zone of the left lung. Chest radiographs showed left lower lobe collapse with an overlying pleural reaction. The lack of mediastinal shift raised the possibility that the changes were longstanding. Plasma urea and creatinine and haemoglobin levels were normal but his sedimentation rate (ESR) was 43 mm/h. Bronchoscopy with brush biopsy showed no evidence of tumour. In September 1986 he still had dyspnoea, and back pain and said that he could only walk 50 yards without severe fatigue. He also reported nocturnal urinary frequency and intermittent vomiting. The chest signs and chest radiograph (fig 1) had changed little since the previous year. Blood tests revealed a normochromic normocytic anaemia (9.3 g/dl), ESR 74 mm/h, urea 18.4 mmol/l and creatinine 331 \(\mu\)mol/l. Intravenous urography (IVU) (fig 2) and abdominal CT showed bilateral hydronephrosis with occlusion of the distal ureters. At laparotomy dense retroperitoneal fibrosis (later confirmed histologically) was found extending from the hilum of the right kidney down to the bladder. Bilateral ureterolysis was performed with immediate improvement in renal function.

The patient had received a levodopa formulation in combination with an anticholinergic drug continuously since 1978. Selegiline was added in 1984. He was also given baclofen from 1984–6 in an attempt to improve his dystonia. Bromocriptine was commenced in 1983 and maintained at 30–40 mg daily in divided doses. It was discontinued after the diagnosis of retroperitoneal fibrosis.

We recognise that fibrotic reactions of the kind observed in our patient cannot be unequivocally attributed to bromocriptine. However, the other drugs which he received have never been implicated. Retroperitoneal fibrosis\(^1\)\(^2\) and pleuropulmonary fibrosis\(^3\)\(^4\) have been reported separately (although never in combination) as complications of bromocriptine treatment. To date, all affected patients including our own have received total daily doses of 30 mg or more, for treatment of Parkinson's disease. Bromocriptine is an ergot derivative like

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**References**


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