Deafness and blindness in a HIV-positive patient with cryptococcal meningitis

Sir. Cryptococcosis is the most frequent fungal or yeast infection of the central nervous system (CNS), and meningitis is the most common form of CNS cryptococcal involvement. Although it can be found in healthy subjects, it is usually seen in immunocompromised patients. We describe an HIV patient who suffered a relapse of cryptococcal meningitis with blindness and bilateral deafness.

A 23-year-old man, who was an intravenous drug abuser until two years earlier, had tested positively for HIV-antibodies for seven years. One year before admission, cryptococcal meningitis was diagnosed. Fluconazol as treatment of cryptococcal meningitis was administered. Two months later he stopped the prophylaxis. A relapse of cryptococcal meningitis was diagnosed two weeks later. He was treated again with fluconazol and was again discharged with a prophylactic dose. Ten months later he was admitted with a strong headache, malaise, paresthesia in the tongue, dysarthria and fever. Blood cultures were negative. A cranial computed tomography (CT) scan revealed normal pressure. Serum cryptococcal antigen by latex agglutination was 1:1280. Cerebrospinal fluid (CSF) pressure was not measured. CSF showed lymphocytic pleocytosis, and India ink stain was positive and C neoformans was cultured. The minimum inhibitory concentration by methods previously described was 0.59 μg/ml. CSF cryptococcal antigen was 1:1. A new CT scan and Lowenstein culture of CSF were negative. Amphotericin B (0.8 mg/kg/day) was given as treatment and the fever disappeared; nevertheless in the following 20 days he suffered from ataxia and an increasing hearing loss which became total deafness in six days. He also suffered progressive loss of vision. Ocular examination showed a bilateral enhancement of the optic disk compatible with papillitis. Ophthalmological examination in view of the deteriorated state of the patient it was impossible to do an audiometry, but in the tuning fork test, he did not hear the tone either by air or bone conduction. A new cranial CT scan was similar to the previous one. The patient suffered progressive deterioration of mental status, fever and stiff neck, and he died a week later. Permission for necropsy was refused.

Fundoscopic abnormalities have been found in 53% of HIV-negative patients suffering from cryptococcal meningitis, most commonly swelling of the disc with loss of definition of the margins, usually accompanied by marked haemorrhages around it. Blindness without endophthalmitis can be explained by direct infection or compression of the optic nerve, secondary to high intracranial pressure or inflammatory adhesions characteristic of arachnoiditis. Rapid onset of blindness is probably due to direct involvement of the optic nerve, either by infection or by cryptococcal infection itself. Slow gradual deterioration in vision suggests intercranial hypertension as a likely cause. The progressive loss of vision of our patient suggests the latter mechanism. Denning et al found that in AIDS patients with cryptococcal meningitis cranial CT scan can be normal but intracranial pressure can be high. Involvement of the other cranial nerves in cryptococcal meningitis has also been described. In the case of the optic nerve, palsy of the 8th cranial nerve could be caused by a direct effect on the auditory nerve, either by infection or compression. Rex et al reported a HIV-negative patient with bilateral deafness, strabismus, a slow-onset loss of vision, and bilateral palsies of the 8th, 9th and 11th cranial nerves, but a CT scan revealed hydrocephalus. The possible mechanisms of the raised intracranial pressure are discussed. Denning et al have postulated that the mechanism could be a reduced CSF outflow, possibly due to increased outflow resistance.

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Spontaneous perforation of the duodenum in a 14-year-old

Sir. Delayed presentation of perforation of the second part of the duodenum in the absence of definite preceding trauma or underlying pathology is uncommon in children. We present such a case in a 14-year-old boy initially suspected of having appendicitis.

Case report
A 14-year-old boy presented as an emergency with a 24-hour history of abdominal pain originating in the peri-umbilical area and settling in the right iliac fossa. He felt nauseous and had vomited gastric contents on six occasions. There were no symptoms of genito-urinary infection or dyspepsia. However, on direct questioning, he admitted to minor trauma to the right lumbar region against the edge of a snooker table a week prior to admission. Abdominal tenderness was elicited in the right iliac fossa with rebound and guarding. Appendicitis was diagnosed. Arterial and venous pressures within the peritoneal cavity but the appendix was normal macroscopically. No Meckels diverticulum was seen, however, a large intraperitoneal abcess cavity was identified extending up to the duodenum from the right iliac fossa. The abcess cavity was drained and a 2 cm × 2 cm perforation was found on the convexity of the second part of the duodenum proximal to the duodenal haematoma which can often be treated conservatively. The diagnosis of a duodenal haematoma may be confirmed by computed tomography (CT) or by assessing the passage of bowl contrast material.2 Autopsy studies suggest the incidence of duodenal diverticuli in up to 20%.3 These may be primary and are found on the second and third part of the duodenum, usually on the convex border. Secondary diverticuli occur on the first part of the duodenum and are the result of scarring following duodenal ulceration. Most diverticuli are asymptomatic, complications, however, include peritonitis and obstruction.1 In this case there was no definite evidence of the underlying cause of the perforation except the minor trauma in the right lumbar region a week prior to presentation; the symptoms were not typical of perforation of a duodenal haematoma following trauma.4 Spontaneous perforation of the second part of the duodenum with a delay in the presentation is extremely uncommon in children. This case reinforces the concept that appendicitis may be mimicked by many medical and surgical pathologies.

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