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 a HIV patient who suffered a relapse of cryptococcal meningitis with blindness and bilateral deafness.

A 23-year-old man, who was an intravenous drug user until two years earlier, had tested positively for HIV-anabodies for seven years. One year before admission, cryptococcal meningitis was diagnosed. Fluconazole as treatment and after was again discharged with a probiotics. Ten months later he was admitted with a strong headache, malaise, and tenderness in the tongue, dysartria and fever. Blood cultures were negative. A cranial computed tomography (CT) scan was normal. Serum cryptococcal antigen by latex agglutination was 1/1280. Cerebrospinal fluid (CSF) pressure was not measured. CSF showed lymphocytic pleocytosis, Indian ink stain was positive and Cryptococcus neoformans was cultured. The minimum inhibitory concentration by methods previously described by fluconazole was 0.59 μg/ml. CSF cryptococcal antigen was 1/1,024. Indian ink stain 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. Ophthalmologic 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 invasion6 or compression of the optic nerve, secondary to high intracranial pressure7 or inflammatory adhesions characteristic of arachnoiditis. Rapid onset of blindness is probably due to direct involvement of the optic nerve, either by infarction 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 al8 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 been described.3 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 al9 reported a HIV-negative patient with bilateral deafness, ataxia, 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 al9 have postulated that the mechanism could be a reduced CSF outflow, possibly due to increased outflow resistance.

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Comment

Perforation of the duodenum is rare in children and is usually associated with either blunt or penetrating trauma, Zollinger–Ellison syndrome or as a complication of duodenal diverticulum. Spontaneous duodenal perforation has an incidence of 1–7% with blunt trauma or 1.7–5% in penetrating injury. Blunt trauma may lead either to immediate perforation which requires emergency surgery or later to a 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.4,5 Autopsy series suggest that the incidence of duodenal diverticulum 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 diverticulum occur on the first part of the duodenum and are the result of scarring following duodenal ulceration. Most diverticula are asymptomatic, complications, however, include perforations, bleeding and obstruction.6 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.7 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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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. At urgent laparotomy, a duodenal haematoma was found in the peritoneal cavity but the appendix was normal macroscopically. No Meckells diverticulum was seen, however, a large intraperitoneal abscess cavity was identified extending up to the duodenum and the right iliac fossa. The abscess 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 diverticulum. A simple closure was carried out and the patient made an uneventful postoperative recovery.