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 more common in immunocompromised patients. We describe a HIV patient who suffered a relapse of cryptococcal meningitis with blindness and bilateral deafness.

A 22-year-old man presented to the emergency room with malaise and paresthesia in the hands and feet. His medical history included HIV infection which became total deafness two years earlier, had tested positively for HIV-antibodies for seven years. One year before admission, cryptococcal meningitis was diagnosed. Fluconazole as treatment (400 mg daily) was administered. Two months later he stopped the prophylaxis. A relapse of cryptococcal meningitis was diagnosed two weeks later. He was treated again with fluconazole and was again discharged with a prophylactic dose. Ten months later he was admitted with a severe headache, malaise, paresthesia in the tongue, dysarthria 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 C neoformans was cultured. The minimum inhibitory concentration by methods previously described for fluconazole was 0.39 μg/ml. CSF cryptococcal antigen titre was 1:8.

The differential diagnosis included an intracranial drug abuse. skull series revealed a patent sphenoidal sinus and Lowenstein culture of CSF was 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 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. Oculopalpebral reflex was absent. 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.

Funduscopic 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 or 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.

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 not 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, 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. Despite the raised intracranial pressure, the patient was treated with amphotericin B and his vision returned to normal and he was discharged.

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.

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

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 surgery there was no inflammation within the peritoneal cavity but the appendix was normal macroscopically. No Meckels diverticulum was seen, however, a large intraperitoneal abscess cavity was identified. Autopsy revealed the presence of a duodenal diverticulum from the right iliac fossa. The abscess cavity was drained and a 2 cm x 2 cm perforation was found on the convexity of the second part of the duodenum proximal to the duodenal diverticulum. At a simple closure of the cavity was carried out and the patient made an uneventful postoperative recovery.

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.4 Traumatic duodenal perforation has an incidence of 1–7% with blunt trauma or 1.7–5% in penetrating injury.5 Blunt trauma may lead either to immediate perforation which requires emergency surgery or 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 in duodenal ulceration.6 These may be primary and are found on the second and third part of the duodenum, usually on the convex border. Secondary diverticulitis occur on the first part of the duodenum and are the result of scarring following duodenal ulceration. Most diverticulitis are asymptomatic, complications, however, include perforations and hemorrhage and obstruction.7 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.8 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.

AKHTAR M KHAN
ALAN P DICKSON
Booth Hall Children's Hospital, Blackley, Manchester M8 7AA, UK

Correspondence to: Akhtar M Khan, 40 Alwen Avenue, Birkenhead, Huddersfield HD2 2SJ, UK


Reference


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A. M. Khan and A. P. Dickson

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