windows¹³ (serum-filled perspex windows placed over abraded skin). Others, however, have shown normal neutrophil oxidative ability^{14, 15} and normal chemotaxis in vitro. All in all, these probably add up to mild defects in phagocyte function. Lymphocyte function is thought to be normal in Crohn's disease; although there is a specific increase in IgG2

immunocytes.¹⁶ It is not known how many patients with Crohn's have primary antibody deficiency.

In conclusion, we report five patients with primary antibody deficiency who also have Crohn's disease defined radiologically and histologically, and whose symptoms are controlled on steroid therapy.

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Brain parenchymal, subarachnoid racemose, and intraventricular cysticercosis in an Indian man

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Summary

The coexistence of brain parenchymal cysts at various stages of evolution, both intraventricular and subarachnoid racemose, is reported in a patient with neurocysticercosis. The condition has a variety of presentations, depending on the location of the cyst. This case is of particular interest because of the rarity of this condition in India.

Keywords: brain parenchymal cyst; cysticercosis; albendazole

Cysticercosis is caused by cysticercus larvae of *Tenia solium*. The most common manifestation of neurocysticercosis is seizure due to brain parenchymal cysts at various stages of evolution.¹ Subarachnoid and intraventricular cysts are rarely seen in India. In Mexicans, however, they are more common (up to 10% of all cases).² Subarachnoid cysts are of the racemose type, as are the majority of intraventricular ones. A combination of cysticercus cysts in these locations is rather uncommon, especially in the Asian subcontinent. Considering the varied presentations of neurocysticercosis, de-

pending on the location of the cysts, it was felt to be worthwhile reporting this case. Some of the varied presentations and their pathogenesis are also discussed.

Case report

A 50-year-old man presented to the neurology out-patient department with history of left-sided sensorimotor seizures for the last 3 years with a frequency of once every one or two months. There was secondary generalisation at times. There was also a history of complex partial seizures for the last 6 months. These were characterised by episodic altered behaviour, aimless running around, and complete amnesia for the events. He had suffered from recurrent, nonthrobbed left parieto-occipital headaches, each lasting for a few hours, for the last 18 months. Accompanying the headache, he experienced tinnitus in both ears and vertigo, but there was no vomiting or visual symptoms. He gave a history of pork ingestion. He was on a maintenance of dose of phenytoin. He had received albendazole for a month in November 1994.

His general examination was normal. Systemic examination was unremarkable. The

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Accepted 4 August 1998

fundus did not show any papilloedema. There was no neurodeficit or meningeal signs. The haemogram was normal and biochemical parameters were within normal limits. Cerebrospinal fluid (CSF) analysis showed 55 cells (all lymphocytes) with protein 2 g/l, sugar 0.2 g/l (corresponding blood sugar 1.1 g/l), Gram stain was negative and culture sterile. Cysticercus serology (IHA) in blood and CSF was 1:8 (not suggestive). Contrast-enhanced computed tomography (CT) scan of the head showed multiple active parenchymal cysts and an enlarged left sylvian fissure (figure 1). Magnetic resonance imaging (MRI) of the brain showed multiple hypointense lesions on T_1 -becoming hyperintense on T_2 -weighted image, with multiple cysts in the left sylvian fissure with septations (figure 2A and B). No scolex was seen within the racemose cyst. There was an intraventricular cyst in the right posterior horn, seen more clearly on proton density imaging (figure 2C).

In summary, this patient had multiple cysts (cysticercus) in the brain parenchyma, intraventricular region, and a racemose cyst in the left sylvian fissure. In the absence of any acute neurological signs, it was decided to try medical therapy for one month and then re-evaluate the situation.

Discussion

The clinical manifestations of neurocysticercosis are determined by the load of organisms, location of cysts, whether the cyst is dead or alive, and the response of the host.¹ Parenchymal neurocysticercosis occurs most commonly at grey-white matter junctions. Neurocysticercosis is a common cause of late-onset seizure in countries where it is prevalent. Focal seizures are most common, but generalised tonic-clonic seizures are also observed. While parenchymal neurocysticercosis presents with seizures, subarachnoid neurocysticercosis usually presents with meningitis and raised intracranial pressure. Cysticercosis of the basal cistern is severe and frequently fatal. This variant is caused by a racemose form of cysticercosis which may cause obstruction to 4th ventricular outflow.³ Subarachnoid cysticercosis can cause specific neurologic syndromes when large lesions are located in the cerebellopontine angle, sylvian fissure,⁴ or the convexity. Intraventricular cysts are frequently found in association with subarachnoid cysts. The fourth ventricle is the most common site.⁵ They usually present as a syndrome of hydrocephalus and increased intracranial pressure. Free floating cysts in ventricles can cause Brun's syndrome, characterised by intermittent foramen obstruction causing episodic recurrent headaches, vertigo, ataxia and drop attacks.⁵

The racemose form of neurocysticercosis is a collection of transient membranes forming a cluster like a bunch of grapes,⁶ thought to be a forme fruste of cysticercus cellulosae.⁷ Supratentorial racemose cysts are usually found in the sulci. When vesicles break down, an intense inflammatory reaction may occur in the

Figure 1 Contrast-enhanced CT scan of the head showing large CSF density lesion in the left sylvian fissure with incomplete septation. Multiple parenchymal cysts can be seen, some with visible scolexes

Figure 2 Brain MRI scans. (A) T_1 -weighted image, showing the left sylvian fissure containing multiple thin-walled large racemose cysts along with many parenchymal cysts. The lesions are hypointense; (B) T_2 -weighted image showing hyperintensity of the cyst fluid at the left sylvian fissure and multiple parenchymal cysts, some with perifocal oedema; (C) proton density MRI showing right posterior horn of lateral ventricle containing an intraventricular cyst

subarachnoid space, causing arachnoiditis and hydrocephalus.

Cysticercus racemose was first described by Virchow in 1860. It is found in serious cases of cerebral cysticercosis and is characterised by larger size (4–12 cm), absence of scolex and a variable appearance. The coexistence of racemose and cellulosa is rare in Asian countries but not uncommon in Mexico (found in 10% of cases).² The racemose type is more common in the suprasellar area, the cerebellopontine angle and sylvian fissure. The proliferating connective tissue encircles the characteristic bladder wall of the parasite.

Subarachnoid and intraventricular racemose cysts are more easily diagnosed by gross inspection than by microscopy, as they show typical budding of the vesicular wall. In intraventricular cysts there is more intense granulomatous inflammation. The ependymal lining is lost, with subependymal gliosis, ependymitis, and perivascular cuffing.⁸ As the cyst degenerates, granulomatous ependymitis and gliosis are the only residual histological findings.

According to some authors, *cysticercus racemose* may actually be due to a different variety of cestode, including *Tenia solium*, *Tenia multiceps*, and *Tenia serialis*.⁹ In few specimens degenerated scolices have been found, suggesting *T. solium* as the aetiological agent.¹¹

CT scan in subarachnoid cysticercosis most commonly shows hydrocephalus, which may be communicating or non-communicating.¹² There may be contrast enhancement of tentorium and basilar cistern secondary to arachnoiditis and fibrosis. The vessel involvement can lead to infarction. Intraventricular cysticercus is difficult to diagnose by CT scan alone. Obstructive hydrocephalus in the presence of a low-density intraventricular lesion may give a clue. The cysts enhance with contrast.

CT criteria to differentiate between cellulose and racemose cysts

- cellulose cysts are usually located intra-axially, ie, in brain tissue, while racemose cysts are frequently seen in basal cisterns, particularly the suprasellar area and cerebellopontine angle
- racemose cysts are usually bigger, appear as multiloculated cysts resembling 'bunches of grapes', elicit a more intense inflammatory reaction and frequently act as a space-occupying lesion
- in racemose varieties, no scolex is found

MRI is currently the most useful imaging tool for neurocysticercosis. Subarachnoid neurocysticercosis is better seen on MRI than on CT. Similarly, MRI is better for detection of cisternal cysts.¹³ T₁-Weighted images are better for delineating cisternal wall while T₂-weighted images are better in detecting oedema and arachnoiditis. Gadolinium-DTPA contrast in T₁ images enhances leptomeninges, suggesting inflammation. Racemose cysts have a similar signal intensity to that of CSF.¹⁴ Intraventricular neurocysticercosis must be suspected when hydrocephalus is present. MRI is superior to CT scan in detection of intraventricular cyst.¹⁴ Proton density images may better delineate the cyst from the CSF around the cyst,¹³ as shown in the present case.

Ventricular shunting is indicated for hydrocephalus.¹² Cyst extirpation and drainage is indicated for racemose cysticercosis. Intraventricular neurocysticercosis also needs extirpation and/or surgical drainage. A few cases have been treated successfully by albendazole alone.¹⁵

Subarachnoid, intraventricular, spinal and mixed types of neurocysticercosis may behave in a malignant way with worse outcome.¹²

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Internal iliac artery embolisation for intractable bladder haemorrhage in the peri-operative phase

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Summary

Intractable haemorrhage from the bladder wall during transurethral resection of bladder tumour is uncommon but potentially catastrophic. Internal iliac artery embolisation is a minimally invasive technique, which is now widely practised to stop bleeding from branches of these arteries in situations including pelvic malignancy, obstetric and gynaecological emergencies and trauma. We report its successful use peri-operatively, in an unfit, elderly patient with uncontrolled bleeding.

Keywords: embolisation; internal iliac artery; transurethral resection of bladder tumour

Severe haemorrhage from the bladder wall during and following transurethral resection of bladder tumour (TURBT) can be life threatening. Methods of management for uncontrolled bladder bleeding include cystoscopic clot evacuation with cystodiathermy, Helmstein balloon compression,¹ irrigation with alum solution,² and instillation of formalin,³ and may finally culminate in open surgical techniques ranging from simple packing of the bladder to cystectomy and urinary diversion.⁴ Most patients in this situation are elderly and unfit and therefore unlikely to withstand these latter morbid procedures, especially as they require regional or general anaesthesia. Prolonged hypovolaemia and the associated massive blood transfusions that may be required are also poorly tolerated.

Internal iliac artery embolisation has been successfully used in controlling bladder haemorrhage from terminal pelvic malignancy for over 20 years.^{5,6} This case report illustrates its added value in the transurethral peri-operative phase.

Case report

A frail 79-year-old man with superficial, high-grade transitional cell carcinoma of the bladder (G3 pT1) kept on regular surveillance for 3 years, was admitted for transurethral resection under general anaesthesia of recurrent bladder tumour. There was no history of recent haematuria and his haemoglobin level was 10.2 g/dl. Clotting profile was normal.

Cystoscopy revealed an extensive tumour recurrence predominantly on the left side and occupying over 70% of the bladder surface

area. Resection of the tumour was difficult due to excessive bleeding. After 25 minutes, he became cardiovascularly unstable and was transfused with two units of blood. It was not

Figure 1 Pre-embolisation subtraction arteriogram showing selective contralateral catheterisation of the left internal iliac artery with significant vascular supply to the bladder wall tumour

Figure 2 Post-embolisation subtraction arteriogram showing occlusion coils bilaterally within the anterior divisions of the internal iliac arteries

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Accepted 2 September 1998