Giant cystic cerebral cysticercosis

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Abstract

A report of three cases of cerebral cysticercosis, all of them being large cystic lesions on imaging and presenting clinically as intracranial space occupying lesions. All of them presented with features of raised intracranial tension and progressive neurological deficits. Imaging (CT scan & MR imaging) revealed large cystic lesions. There were no other features to suggest the nature of the pathology. All of them were managed surgically. One case was autopsied. The correct diagnosis was established only after surgery and autopsy respectively. These cases showed that giant cysticerci is an important differential diagnosis for large cystic lesions in the endemic areas.

Key words: Cysticerci, Cysticercosis, Cerebral, Cystic lesions, Giant, Surgery

INTRODUCTION

Cysticercosis of the central nervous system (CNS) involves infestation with cysticercosis cellulosae, the larval form of Tenia solium. This is a worldwide and most frequent parasitosis of the CNS. After installing itself in the CNS, Cysticercosis cellulose begins to grow, usually reaching 4-20 mm in the widest diameter when located inside the brain parenchyma. Those developing in the ventricles or in the subarachnoid space usually reach a larger size. They may also take on the form of cysticercosis racemosus, being characterized by a membrane of irregular thickness and absence of a scolex and by usually being clustered in multiple vesicles, interconnected or not, resembling a raceme. These are frequently observed in the ventricles and cisternae, they may also occur in the brain parenchyma. We report here 2 cases of giant cysticerci who were treated surgically and one autopsied case.

CASE REPORTS

Case 1 was a 24 year old, female presented to the emergency services with headache of one year duration. This had increased in severity for the last one month and had been associated with blurring of vision and vomiting. There was no history of seizure and loss of consciousness. On examination she had bilateral papilloedema and a right homonymous superior quadrantanopia. Clinically she was diagnosed to have a left temporal mass lesion. CT scan of the brain showed a large hypodense lesion in the left temporoparietal region, extending up to the ambient and suprasellar cisterns with mass effect and midline shift. The lesion was not enhancing with contrast, also on careful observation a thin septa was seen to be running across the cyst, suggestive of daughter cysts (Figure 1). A preoperative diagnosis of arachnoid cyst or a hydatid cyst was considered. The absence of

FIG 1: CT scan, contrast study, axial section of Case 1 showing a large right temporal cyst with septae seen running across the cyst. There is no enhancement of the wall. There is also no evidence of any thinning of the adjoining calvarial bone as may occur in arachnoid cysts.
thinning of the adjacent calvarial bone was however, atypical of arachnoid cyst. A left temporal craniotomy was performed. The dura and the brain were tense and bulging, and the cyst was not seen on the surface. A corticotomy was done on the inferior temporal gyrus and decompression of the cyst located 1cm deep to the cortical surface was performed. The cyst was of 4cm diameter containing clear fluid and thin membranous tags. Another two greyish opalescent cysts of 2-cm diameter were seen around the perimesencephalic cistern and suprasellar cistern in the medial part of the temporal lobe and these were also removed. Postoperatively the patient had no additional deficits and improved completely.

**Case 2** was a 28-year-old female presented with a history of headache and transient visual obscurations of three months duration. On examination she had papilledema and no other deficits. MR imaging of brain showed multiple coalescing cystic lesions in the left tempo-occipital region hypointense on T1 sequence becoming hyperintense on T-2 weighted images with perilesional edema (Fig. 2a). Contrast enhanced MR showed enhancement of the wall of the cyst (Fig.2b). A possibility of hydatid cyst was kept in mind. The patient was subjected to a left temporo-parietal craniotomy. At surgery, the dura and the brain were tense. An incision was made in the middle temporal gyrus and at a depth of 3cm, a cystic lesion with a capsule was encountered. The plane of dissection was not very well defined with adhesions of the cyst to the surrounding brain parenchyma, the cyst measuring 6cm in diameter, ruptured exuding a proteinaceous gel like material and also delivering smaller thin membranous structures from within.

**Case 3** was a 10-year-old girl presented with mental regression and right motor focal seizures of one-year duration. She developed rapid deterioration of mental functions for the last 30 days, incontinence for 15 days and altered sensorium for 1 day. She made gradual recovery with antiedema measures and anticonvulsants. Cranial CT scan revealed multiple, large cystic lesions in the brain parenchyma, some of them reaching the surface and some closely abutting the ependyma, with marked hydrocephalus. Some of the lesions were hyperdense. In view of multiplicity and unusually large size, cysticercal or hydatid cysts with loculation was considered. The routine haemogram revealed marked diamorphic anaemia of 4 mg% which improved following blood transfusion to 12-mg%. The biochemical parameters were normal. CSF examination revealed mild pleocytosis, with mildly elevated protein, normal glucose and was positive for anticysticercal antibodies. Brain biopsy was carried out for a definitive diagnosis, which revealed a degenerated cysticercal cyst in the subarachnoid space with chronic meningeal inflammation and gliosis. The child improved with medication and was discharged. She however discontinued medications and reported to the emergency services with recurrence of seizures. There she developed a cardiorespiratory
arrest and died. Partial autopsy confined to the examination of the brain was conducted, 4 hours postmortem. Some of the superficially placed cysts were harvested for maintenance in culture (unpublished data).

**Histopathology findings**

The paraffin sections from the biopsied material were stained with Haematoxylin-eosin, periodic Schiff and Masson’s trichrome. In all the three cases, biopsied material revealed a large collagenous wall palisaded by elongated epithelioid cells and large foreign body giant cells some of them having a foamy cytoplasm and chronic inflammatory cells. The inflammation had extended along the wall of the lesion to the markedly gliosed underlying brain parenchyma. Many of the vessels had dense perivascular mononuclear cell infiltration. The luminal aspect of the cystic lesion had large markedly folded, racimose cysticercal cysts in varying degrees of degeneration. Some of them had a scolex (Figure 3) though partly degenerated. The calcareous corpuscles were numerous and calcified. However, the bladder wall, in many places had PAS positive finely villous glycocalyx (Figure 3, inset) and subcuticular cytons, indicating a metabolically viable nature of the cyst. In focal areas, brightly eosinophilic proteinous material was seen deposited on the surface of the bladder wall, representing immunoglobulin deposition, a host defense mechanism.

The large cysticercal cysts harvested from the autopsied case could be maintained viable in tissue culture medium for two days as evidenced by rhythmic contraction of the wall and ductular system observed under microscope (unpublished data). The histology subsequently revealed variable degenerative changes in the scolex and the bladder wall of the cultured cysts.

The autopsied brain was softened; oedematous discoloured, due to the effect of ventilator. Numerous cysticercal cysts were seen in the deep brain parenchyma and some in the cortical ribbon, projecting into the subarachnoid space. The bladder wall has lost the villous glycocalyx and the sudcicular cytons. The ductular system in the bladder wall was markedly dilated, containing protenaceous material. Many focoi of spotty calcification were seen, indicating more viability and advanced degree of degeneration. Some of the large cysts did not have a scolex. One of the cysts in the cerebellum showed ‘evagination’ of the scolex attempting growth of the worm to the adult form. The ependyma was necrotic. Histological examination of the cysts located close to the surface revealed host reaction in the form of chronic inflammation, neovascularisation, hyalinated wall and adjacent parenchymal gliosis.

The deep seated large cysts did not elicit inflammatory reaction, but caused edema probably as a response to antihelmintic therapy and antigen release. The ventricular cysts elicited

![FIG 3: Large cysticercal cyst with scolex and the neck. H.E. 10. Inset: Higher magnification showing distinct microvillous glycocalyx (arrow) covering the cuticle, indicating metabolically active bladder wall. PAS×320.](image)
similar inflammatory reaction as the leptomeningeal ones.

DISCUSSION

Cystercerosis of the nervous system is a disease with varied presentations and may pose complex management dilemmas which may require a combined medical and surgical treatment. The commonest mode of presentation is seizures (65%), increased intracranial pressure (35%) and meningoencephalitis (29%). Less frequently are psychosis; tumor like, stroke like and spinal manifestations. The cases described in this paper presented with increased intracranial pressure. Neuroimaging was suggestive of cystic mass lesions with differential diagnosis of arachnoid cysts, hydatid cyst and abscesses. Cystercerosis was not considered prior to surgery probably because of its giant size. Giant cystercerci have been previously described. It may mimic malignant lesions, parasitic cysts, abscesses or other benign cysts like arachnoid cysts. However the underlying calvaria did not show scalloping indicating a longstanding lesion of an arachnoid cyst. There have been reports of successful treatment with albendazole of these lesions. There were also reports to the contrary with deterioration due to decompensation of the intracranial hypertension. This was in turn due to an inflammatory reaction triggered by cyst degeneration. Previously surgery was thought to be a must for subarachnoid, ventricular, intraoccellar and racemose cystercerci. However, there were also reports of successful medical management. An individualised approach should be taken with factors such as clinical presentation, level of disease activity and the location of the cyst to be taken into consideration.

The factors which may affect the development of giant cystercerci could include genetic and immunological. There has been a report of giant cyst in a patient with HIV infection which may suggest the importance of the host immune status.

REFERENCES


