Cerebral Cysticercosis Presenting as a Ring Enhancing Lesion with Partial Seizures
(A Case Report and Review of Literature)

Vijay K Kak, FRCS (Eng & Edin), FAMS*
Adel A Al-Jishi, MRCPI**
Sumant G Daftary, DMRD (Eng)***
Kishore V Nadkarni, MD****

ABSTRACT

A case presenting with partial motor seizures and a ring enhancing lesion on CT is described. The lesion was proven to be a cysticercus granuloma, the first such case in the State of Bahrain. These patients should be managed by anticonvulsant medication alone and monitored by serial CT. The relevant literature is reviewed.

Patients presenting with seizures, partial or generalised, and a single enhancing lesion on computerised tomography (CT) pose a problem in diagnosis and management. These lesions have been reported from all over the world with increasing frequency[1-5]. The two most commonly considered diagnoses in such patients from endemic areas are tuberculoma and cysticercus granuloma. However, similar patients presenting in non-endemic areas are liable to be misdiagnosed. Since both these conditions are amenable to medical therapy, it becomes imperative to try to reach a diagnosis prior to initiation of appropriate therapy.

The present communication describes the management of a patient with partial motor seizures and a ring enhancing lesion on CT, proven to be a cysticercus granuloma.

THE CASE

A 30 year old Indian male was admitted to the neurology service following a partial motor seizure involving the right side, starting from the face, with aphasia lasting about 5 minutes. He had postictal right supranuclear facial paresis. The rest of the clinical examination and routine laboratory investigations were normal. CT scan revealed a hypodense lesion in the left posterior frontal region, which showed ring enhancement on contrast administration, with surrounding oedema; there was no midline shift (Fig 1). He was started on diphenylhydantoin

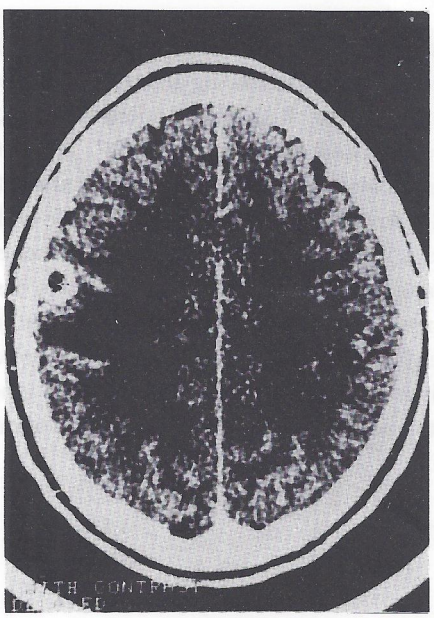

Figure 1: *Contrast enhanced CT scan showing a ring enhancing low attenuation lesion with surrounding oedema.*

* Consultant Neurosurgeon
** Consultant Neurologist
*** Consultant Radiologist
**** Consultant Pathologist

Departments of Surgery, Medicine, Radiology & Pathology
Salmaniya Medical Centre
State of Bahrain

(DPH) 300 mg daily and antitubercular therapy (ATT - rifampicin, INAH, pyrazinamide, ethambutol) with the tentative diagnosis of a tuberculous granuloma. Evidence of hepatic dysfunction appeared a week later, with serum bilirubin 3.6 mg dl^{-1}. ATT was discontinued and his liver function tests gradually improved over the next 4 weeks. He continued to have seizures, and the DPH was increased to 400 mg daily. CT scan, repeated 4 weeks after admission, showed reduction of surrounding oedema but no appreciable change in the lesion morphology.

The patient was referred to the neurosurgery service during the phase of hepatic dysfunction. A diagnosis of cysticercosis was entertained and he was scheduled for surgery after the liver function tests had returned to normal. At craniotomy, the dura was found adherent to the brain surface in the posterio-inferior frontal region. A hard subcortical nodule was felt in the lower part of the precentral gyrus. It was approached through a neighbouring sulcus and totally excised. The postoperative course was uneventful and he was discharged on the twelfth postoperative day. Cut surface of the excised lesion showed it to be cystic, lined by a thin glistening membrane with an attached white nodule. It was surrounded by a soft tissue wall measuring 2.5-4.0 mm in thickness. Histopathological examination of the lesion showed a largely intact parasite including the body cavity, suckers and hooklets, typical of cysticercus cellulosae (Fig 2). This was surrounded by necrotic tissue debris. The adjacent brain tissue showed inflammatory reaction and reactive gliosis.

DISCUSSION

CT abnormalities in simple partial seizures have been reported in 24-70% of cases whereas similar changes are less frequently seen in complex partial seizures[6]. The aetiological lesions reported have included tuberculomas, cysticercosis, solitary metastases, microabscesses, cavernous haemangiomas, focal meningoencephalitis, gliomas, sarcoidosis and larva migrans.

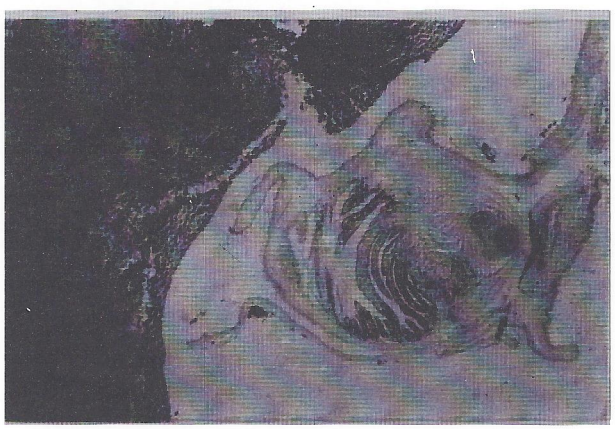

Figure 2: *Microphotograph showing the cavity containing the parasite, surrounded by necrotic tissue debris and brain tissue showing inflammatory reaction. (Haematoxylin and eosin, X 160).*

Ring or disc enhancing hypodense lesions in focal epilepsy have not been described in western series. Bhargava and Tandon[2] from India first reported these lesions as being tuberculomas on the basis of histology in some cases and association with tuberculous meningitis in others, and advised ATT. Goulatia et al[3] presented evidence that most of these lesions disappeared when treated with anticonvulsants alone, thus casting doubt on the tuberculoma hypothesis. Ahuja et al[1] suggested breakdown of the blood brain barrier around the lesion during a seizure as the cause of enhancement seen on CT.

The aetiology of a single ring enhancing lesion in patients with partial seizures can only be confirmed by surgical excision. Of all the pathologies reported for these lesions, the commonest based on biopsy evidence has been cysticercosis. Rajshekhar et al[7] presented clinical and CT criteria to distinguish small cysticercus granulomas from small tuberculomas in patients with epilepsy, with histologically proven lesions and suggested a management algorithm for patients with seizures and solitary small enhancing lesions on CT. They recommended that such patients without evidence of raised intracranial pressure (ICP) or focal neurologic deficit be managed with anticonvulsant medication only. A repeat CT after 8-12 weeks would show resolution of the lesion in up to two-thirds of the patients. If the size of the lesion enlarges to more than 20mm, an excision biopsy was recommended. Stable lesions could be further monitored. Serological testing for cysticercus antibodies could be useful if the result is positive. The above strategy is not only appropriate in endemic areas but also in developed regions of the world, if judiciously applied[5].

Neurocysticercosis is the commonest helminthic infestation of the central nervous system with high prevalence rates in Mexico, South America, Eastern Europe, India, China and Southern Africa[8,9]. However, with present day increase in travel and immigration, cases are being reported from non-endemic areas such as the USA, UK, and Australia[4,5,9,10]. The ring or disc enhancing lesion represents the dying parasite with attendant inflammatory reaction, which evolves rapidly to an inactive granuloma or disappears entirely[5]. Since most inflammatory cysts in neurocysticercosis involute, antiparasitic therapy is unnecessary as the parasite is already dead[5,7,11,12].

We would, therefore, suggest that such patients should be managed by anticonvulsant medication and serial CT. No cysticidal therapy is required. If the CT lesion persists beyond 8-12 weeks or shows enlargement, an excision biopsy should be performed. Hepatotoxicity related to ATT, as in our patient, is another reason for an accurate diagnosis prior to specific treatment.

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