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Hippocampal sclerosis in association with neurocysticercosis

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ABSTRACT – *Objective.* To discuss the possible mechanisms underlying a dual pathology combining neurocysticercosis and hippocampal atrophy, illustrated by the observation of four patients with epilepsy. *Case reports.* The first patient presented at the age of four years with a first episode of status epilepticus, presumably due to an inflamed, calcified, parenchymal cysticercus granuloma. Thereafter, he had occasional seizures. Routine MRI undertaken several years later revealed unilateral hippocampal atrophy and sclerosis. Two other patients with initial imaging evidence of active neurocysticercosis located close to the hippocampus, and occasional seizures, developed ipsilateral hippocampal sclerosis. The seizure disorder of our fourth patient, with medically intractable epilepsy, was initially attributed to a calcified cysticercus granuloma. Clinical description, video-EEG telemetry and imaging work-up suggested a diagnosis of mesial temporal lobe epilepsy due to hippocampal sclerosis.
Conclusions. Definitive conclusions as to the underlying mechanisms cannot be derived from the present, retrospective series of only four patients. However, the following suggestions can be made: 1) seizures due to neurocysticercosis may constitute the initial precipitating illness for the development of hippocampal sclerosis, 2) the hippocampus may be involved in host brain inflammation and gliosis in response to a nearby, degenerating cysticercus, 3) the seizure focus formed by the degenerating cysticercus, engenders epileptogenic changes in the hippocampus through kindling, and 4) the two conditions may coexist purely by chance. Systematic and prospective, serial MRI evaluations of hippocampal structures in patients with neurocysticercosis should contribute to further clarify the underlying mechanisms.

Key words: neurocysticercosis, hippocampal sclerosis, seizure, epilepsy, dual pathology

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Neurocysticercosis (NCC) is the most common parasitic infection of the brain, and is the leading cause of epilepsy in the developing world. It is endemic throughout Latin America, India, Asia, and now the southwestern United States (Garcia *et al.* 1993, Medina *et al.* 1990, Singh *et al.* 2006, Ong *et al.* 2002, DeGiorgio *et al.* 2005). There is consensus that the majority of seizures due to NCC are provoked by host brain inflammation in response to degenerating larval cysticerci (Carpio *et al.* 1998, Garcia *et al.*

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2003). In the majority of cases, the prognosis for control of the seizure disorder is good. Our experience at a tertiary care-epilepsy clinic revealed that NCC was diagnosed in roughly 35% cases of a sample of 1026 patients, which included all types of seizure disorders seen consecutively at the clinic (Singh *et al.* 2006). The finding of NCC upon imaging studies was significantly associated with the occurrence of a single seizure; the probability of diagnosing NCC decreasing progressively with increasing number of seizures prior to presentation. Seizures recur in roughly one-half of all individuals presenting with a first seizure due to NCC (Carpio and Hauser 2002), and for a small number of cases, seizure control can be an enduring concern, at times to the point of being refractory to medical treatment.

Recently, there have been reports (Leite *et al.* 2000, Wichert-Ana *et al.* 2004; Terra-Bustamante *et al.* 2005, da Gama *et al.* 2005) of an association between NCC and hippocampal sclerosis (HS). The finding of this association is important because HS is one of the major causes of temporal lobe epilepsy, the most common form of poorly controlled focal epilepsy. Although these reports do not indicate a causal association, they emphasize the incidental coexistence of the two conditions and that surgical management of HS may be a realistic option despite the presence of NCC lesions.

We report four patients, identified by retrospective chart review, from a cohort of patients followed at an epilepsy clinic in Northwest India. We propose mechanisms that may underlie the association between the two conditions, and which may influence the refractoriness of seizures to medical treatment in some patients with NCC.

Case reports

Patient 1

A four-year-old boy presented with the onset of two seizures over an eight-month period. The first episode comprised of nausea and vomiting, chewing mouth movements, gaze deviation to the right and apparently, clonic movements of the right upper extremity culminating in a generalized tonic-clonic seizure. The entire seizure lasted for 45 minutes. The boy was evaluated in an acute care facility, where he remained unconscious for the next five hours. Once seizures were recognized, antiepileptic drug treatment was initiated with carbamazepine at a dose of 10 mg/kg/day.

The second seizure occurred eight months later. Magnetic resonance imaging (MRI) of the brain was performed and demonstrated a focal, nodular-calcified lesion measuring about 10 mm in diameter with peripheral contrast enhancement, and surrounding edema in the left temporo-occipital region *(figure 1A)*. The abnormality was interpreted as being consistent with a calcified cysticercus granuloma with perilesional edema. At this time, imaging was not specifically oriented to evaluate hippocampal morphology.

The boy remained seizure-free over the next three years. A non-contrast-enhanced MRI was repeated, and revealed a persisting focal calcification, with no surrounding edema in the left temporo-occipital region *(figure 1B)*. Carbamazepine treatment was tapered and discontinued over the next three months. Eighteen months later, the boy developed a severe headache accompanied by vomiting and an episode of unresponsiveness lasting for 10 minutes. Carbamazepine treatment was reinstated, but there occurred another two seizures in succession over the next two months with similar clinical presentation, *i.e.*, headache, vomiting and unresponsiveness. A control MRI study revealed a left temporo-occipital calcification with surrounding edema *(figure 1C)*. Carbamazepine was substituted with oxcarbazepine (15 mg/kg/d) and no further seizures were reported.

During the following period, there were repeated complaints of worsening scholastic performance and new-onset memory impairment. A contrast-enhanced MRI study with oblique, coronal, contiguous cuts perpendicular to the long axis of the hippocampus was performed two years later. The calcified lesion persisted with no contrast enhancement or surrounding edema; however, left hippocampal atrophy with hyperintensity of the left hippocampus was noted *(figure 1D)*. A 2-hour, awake and sleep EEG examination did not reveal any epileptiform or non-epileptiform abnormalities. Cysticercus serology using the electro-immunotransfer blot (EITB) assay (Centre for Disease Control, Atlanta, USA) was negative. Antiepileptic drug treatment was continued.

Patient 2

This 47-year-old man presented to us 10 years after his first seizure. History revealed that six seizures occurred over the first five months, before he elected to take treatment with phenytoin-sodium upon the advice of a physician in the remote area that he lived in. He recollected the occurrence of a warning, prior to each seizure, but was unable to describe the aura any further. According to a description given by a witness, the seizure itself comprised of behavioral arrest lasting 5-10 minutes.

A neurosurgeon was consulted at around the time of the initial symptoms, who noted bilateral papilloedema and ordered a computed tomographic (CT) brain scan. The latter revealed multiple parenchymal granulomas in the left frontal, right temporal and right cerebellar regions *(figures 2A,B)*. Presumably, a diagnosis of multiple, degenerating, brain parenchyma NCC was made as the subsequent treatment comprised of phenytoin-sodium and corticosteroids. The exact dose and duration of the latter could not be ascertained, but apparently, initial treatment was given in the form of intravenous dexamethasone, and

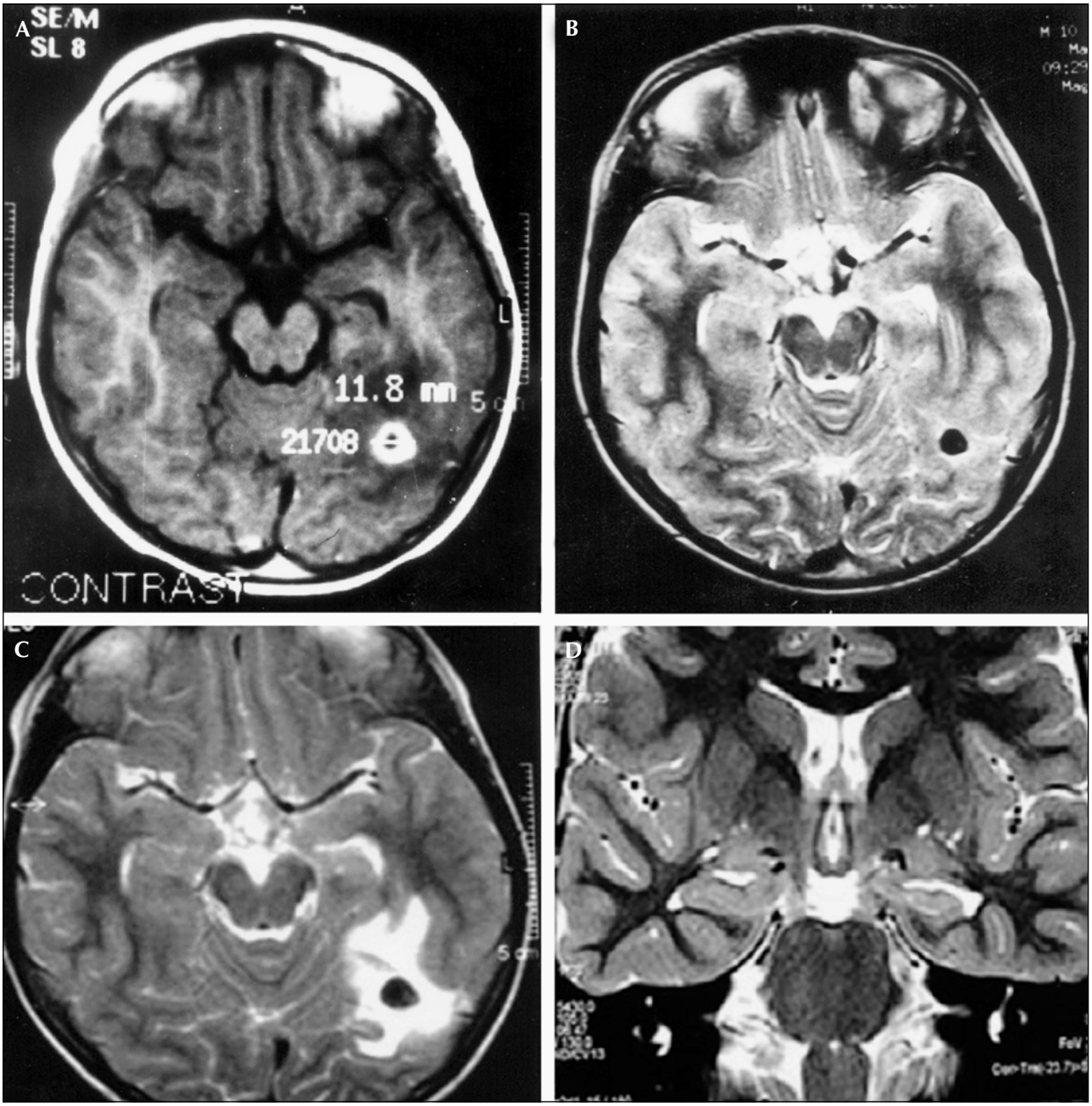

Figure 1. Serial MRIs in case 1. **A**) A calcified lesion with surrounding edema and ring-like peripheral contrast enhancement on initial gadolinium-enhanced T1 imaging. **B**) Follow-up T2 image showing resolution of edema. **C**) Further follow-up imaging upon recurrence of seizures showing reappearance of edema on a T2 image. **D**) Oblique coronal images showing the hyperintensity of, and smaller size of the left hippocampus.

oral corticosteroids were administered for the next two months. Routine hematological and biochemical investigations performed at that time were non-contributory.

The patient remained seizure-free over the next five years and hence subsequently stopped his antiepileptic medication. Seizures recurred soon thereafter at monthly intervals. These persisted until five months prior to presentation, whereupon, he re-initiated treatment with phenytoin and became seizure-free again. Seizures were characterized by a non-specific aura followed by loss of contact. During this period, he reported severe memory impairment that interfered with his day-to-day activities. Upon

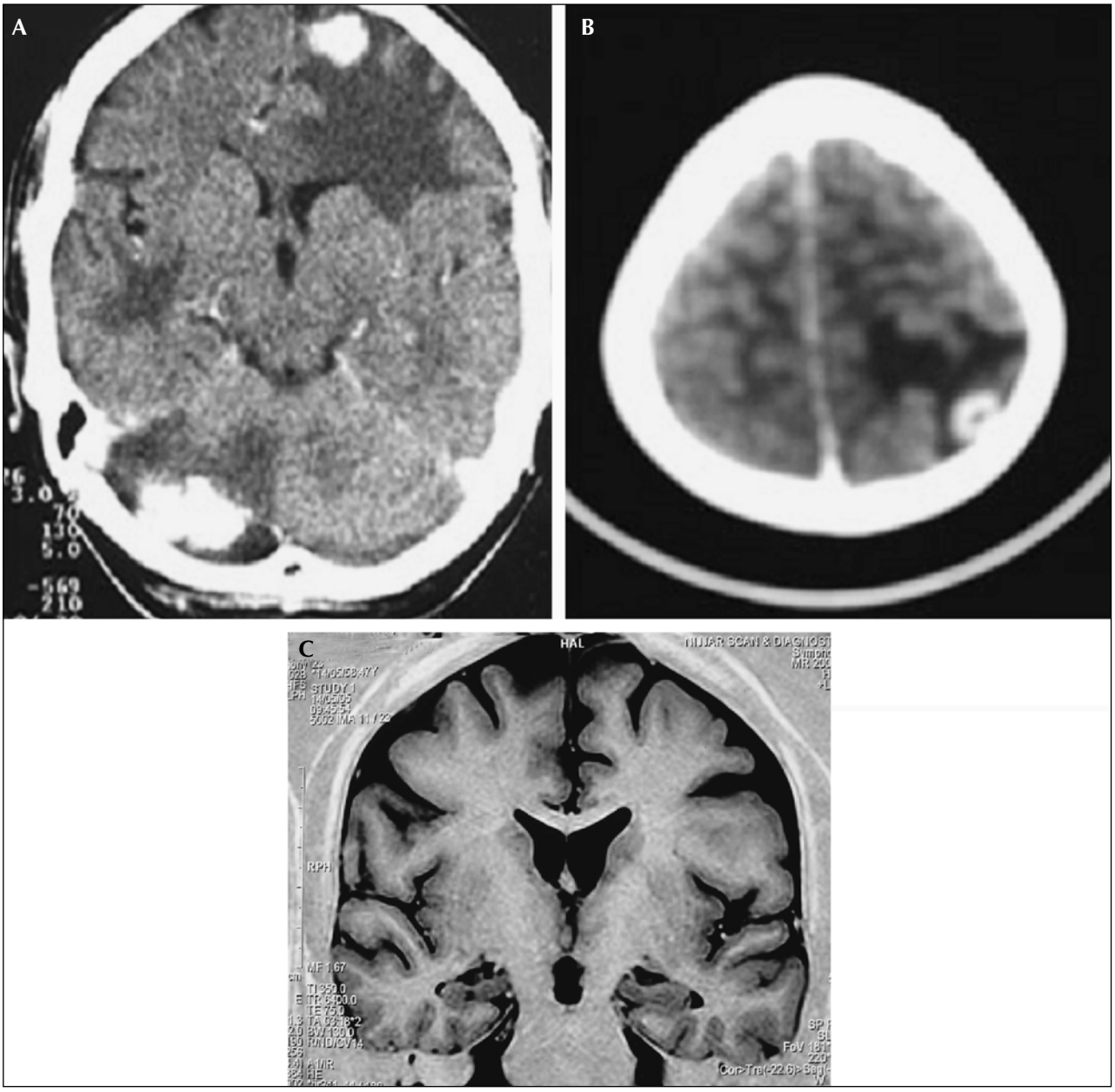

Figure 2. Case 2. **A, B**) Initial contrast enhanced CT sections showing multiple enhancing parenchymal lesions with surrounding edema. **C**) Follow-up MRI, T1 coronal oblique sections showing the smaller size of the hippocampus on the right side.

presentation, hematological and routine biochemical investigations were unremarkable. However, a chest X-ray revealed bilateral reticulo-nodular opacities in both lung fields. He had no respiratory complaints though, and pulmonary function tests were unremarkable. Assay of anti-cysticercus antibodies (electro-immuno transfer blot; Center for Disease Control, Atlanta, USA) in the serum was positive. Magnetic resonance imaging of the brain revealed multiple calcified lesions with right-sided hippocampal atrophy *(figure 2C)*.

Patient 3

This 30-year-old man developed fever and cough 28 months prior to presentation. A diagnosis of miliary tuberculosis was made on the basis of a chest X-ray that revealed miliary mottled shadows in both entire lung fields, findings that were considered to be consistent with pulmonary tuberculosis. Antitubercular treatment with a four-drug regimen comprising of rifampicin (15mg/kg/d), isoniazid (5 mg/kg/d), ethambutol (15 mg/kg/d) and pyrizinamide (15 mg/kg/d) was started.

Three months later, whilst although, fever and respiratory symptoms had improved, the patient developed a seizure. A brain CT scan revealed multiple parenchymal granulomas with surrounding edema. Cerebrospinal fluid examination was also conducted; cytological and biochemical examinations were unremarkable and a polymerase chain reaction (PCR) test for *Mycobacterium tuberculosis* (Ranbaxy Labs., Mumbai, India) was negative. Phenytoin-sodium (5 mg/kg/d) and prednisolone (1 mg/kg/d) were added to the antitubercular regimen. Prednisolone was tapered over the next two months. Stereotactic biopsy of the lesions was offered but was declined by the patient.
About eight months after the first seizure, the patient began to complain of a non-progressive decrease in memory. Over the next four months, several serial seizures ensued, for which serum phenytoin levels were checked and found to be within the usual range. Additional antiepileptic treatment with clobazam was prescribed. Follow-up MRI was performed one year after the first seizure and revealed persistent granulomas in the right mesial temporal lobe (involving the parahippocampal gyrus), right cerebral peduncle, superior temporal gyrus, both parieto-occipital lobes, brainstem and cerebellum *(figures 3A,B)*. Subsequently, the patient had occasional breakthrough seizures involving the right upper extremity, though it could not be decided on the basis of historical data, whether these comprised of clonic seizures or automatisms. Several serial MRIs were performed at four months intervals; the granulomas persisted in all of these investigations, although about 21 months after the initial seizures, two of the granulomas in both parieto-occipital locations had calcified, edema around the remainder had decreased and there were no new lesions. The latest imaging was also oriented to study the hippocampal anatomy; whilst the granuloma and the edema surrounding it in the mesial temporal location appeared to be resolving, the right hippocampus appeared atrophic on T-1 weighted images and hyperintense on T2- weighted *(figures 3C,D)*.
Two years following the intial seizure, a CSF examination was repeated; PCR for *M. tuberculosis* was again negative. Antibodies against *T. solium* cysticercus antigens were demonstrated in the serum using the CDC EITB assay. A repeat chest X-ray revealed complete resolution of the earlier lesions with no miliary shadows. The Montoux test revealed a positive reaction, measuring 20 mm at 72 hours. Toxoplasma serology, HIV serology (ELISA), serum angiotensin-converting enzyme assay and liver function tests were non-contributory. About 30 months after the first seizure, the patient continued to have occasional seizures whilst on a combination of phenytoin-sodium and levetiracetam at optimal doses; seizures occurred once in 3-6 month period, and comprised of possible automatisms involving the right upper extremity. His antitubercular treatment was stopped after two years.

Patient 4

This left-handed, young woman had her first apparently generalized tonic-clonic seizure at the age of nine years. A brain CT scan was performed and it revealed a calcified granuloma in the left occipital head region *(figure 4A)*. The latter was assumed to be the cause of her epileptic seizure and antiepileptic drug treatment with carbamazepine was instituted. Subsequently, she was lost to follow-up but presented eight years later at the age of 17 years. During the intervening period, she had had frequent seizures that remained refractory to several antiepileptic drug trials. Her current seizure frequency was one to two per week and they often occurred in clusters.
She would often recall an aura comprising of an ascending epigastric sensation or rarely, a whole body sensation. This was followed by behavioral arrest with an unusual stare and mouth automatisms, lasting for about one minute. There was minimal post-ictal confusion. Her birth and family history were unremarkable; there were no antecedent events suggestive of an initial precipitating illness.
Treatment upon representation comprised of oxcarbazepine (30 mg/kg/d), lamotrigine (3 mg/kg/d) and clobazam (20 mg/d). Neurological examination was unremarkable. Interictal EEG revealed bi-temporal spike and sharp wave discharges, predominantly from the right side. Video-EEG captured six seizures with similar semiology and ictal EEG. Semiology comprised of a loud cry, lip smacking, right upper limb automatisms and dystonic posturing of the left upper limb. Ictal EEG revealed rhythmic slow activity, with onset in the right anterior temporal region. Brain MRI revealed right-sided hippocampal sclerosis *(figure 4B)*. Neuropsychological examination revealed impairment in verbal memory tests, but full scores on non-verbal tests with the Postgraduate Institute-Memory test (a standard Indian vernacular version of the Weschler Memory Scale- Revised). A right anterior mesial temporal resection was performed; there were no seizures over a post-operative observation period of 14 months.

Discussion

All four cases described herein, presented with imaging evidence of active or inactive NCC; however, imaging evaluations undertaken later for medically-refractory epilepsy disclosed the presence of HS. The coexistence of HS and another epileptogenic brain lesion is commonly referred to as "dual pathology". Dual pathology has been widely recognized in the context of HS and a variety of extra-hippocampal lesions, including malformations of cortical development, arteriovenous malformations, and tumors (Cendes *et al.* 1995, Alsaadi *et al.* 2003). The association between HS with NCC has been reported only recently (Leite *et al.* 2000, Wichert-Ana *et al.* 2004, Terra-Bustamante *et al.* 2005, da Gama *et al.* 2005, Chung *et al.* 1998, Kobayashi *et al.* 2001). The cases described here

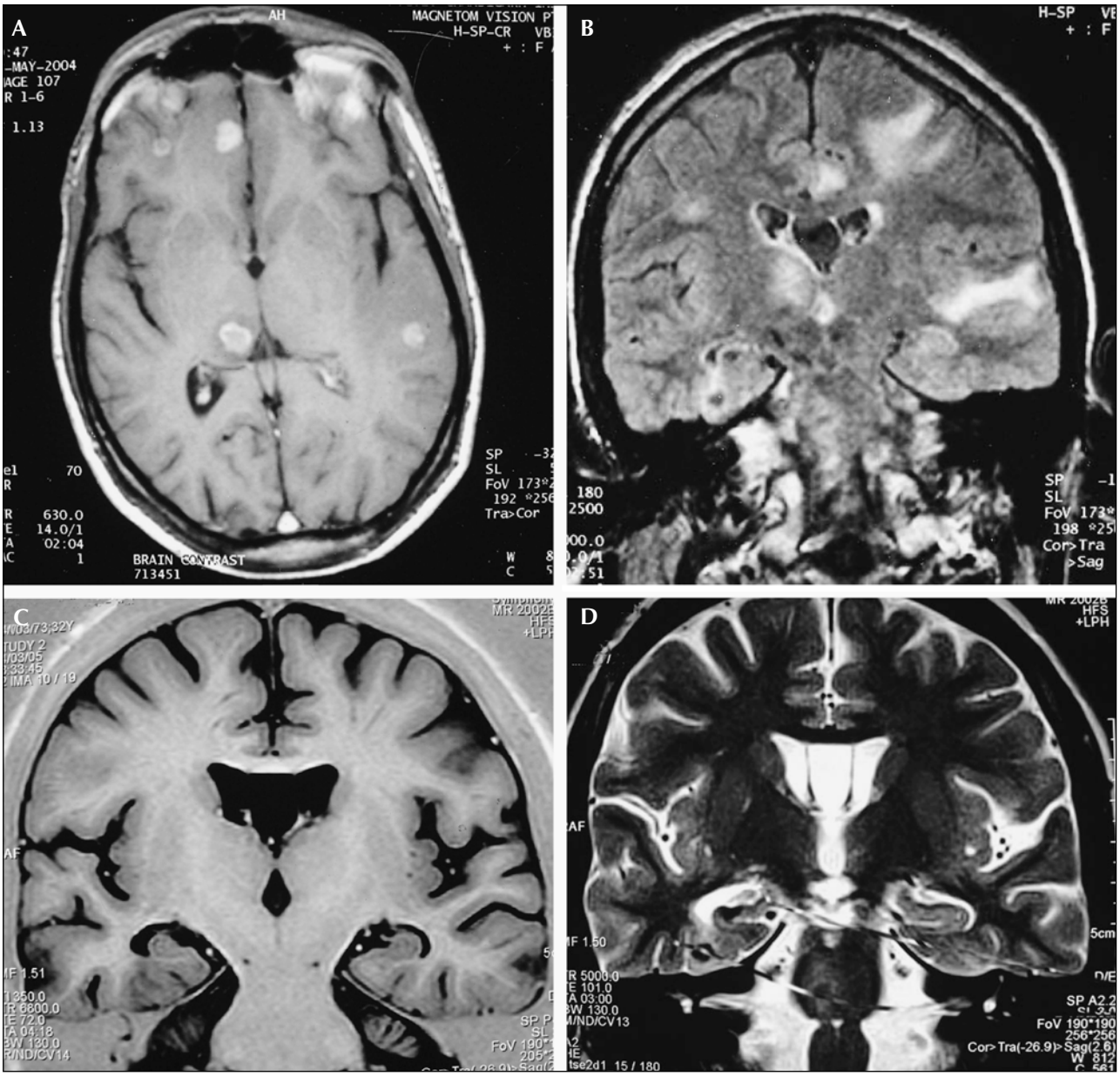

Figure 3. Case 3. **A, B**) Initial images, gadolinium-enhances T1 weighted image showing multiple enhancing granulomatous lesions (**A**) and FLAIR coronal image (**B**) emphasizing the edema around the lesions. Note the lesion in the right para-hippocampal gyrus (**C, D**). Follow-up T2 (**C**) and T1 (**D**) coronal oblique sections showing right hippocampal atrophy (**C**) and hyperintensity (**D**). Note the calcified lesion in the right para-hippocampal gyrus (**C**).

suggest several different mechanisms by which there may (or may not) be a cause-effect relationship between the two conditions. These mechanisms are discussed below.

One likely overriding hypothesis is that seizures due to NCC constitute an initial precipitating illness, leading to the development of HS. Although, the initial precipitating illness is usually a prolonged or complex febrile seizure, the occurrence of status epilepticus due to NCC in a susceptible age group (as illustrated by patient 1) may constitute an antecedent event for the development of HS. Incidentally, the occurrence of status epilepticus has been described in 2% of cases with NCC (Del Brutto *et al.* 1992).

Patients 2 and 3 suggest that the presence of an inflamed, degenerating granuloma in close proximity to the hippocampus may be responsible for the development of HS. We hypothesize that the hippocampus may be directly affected by the inflammatory response and resulting gliosis

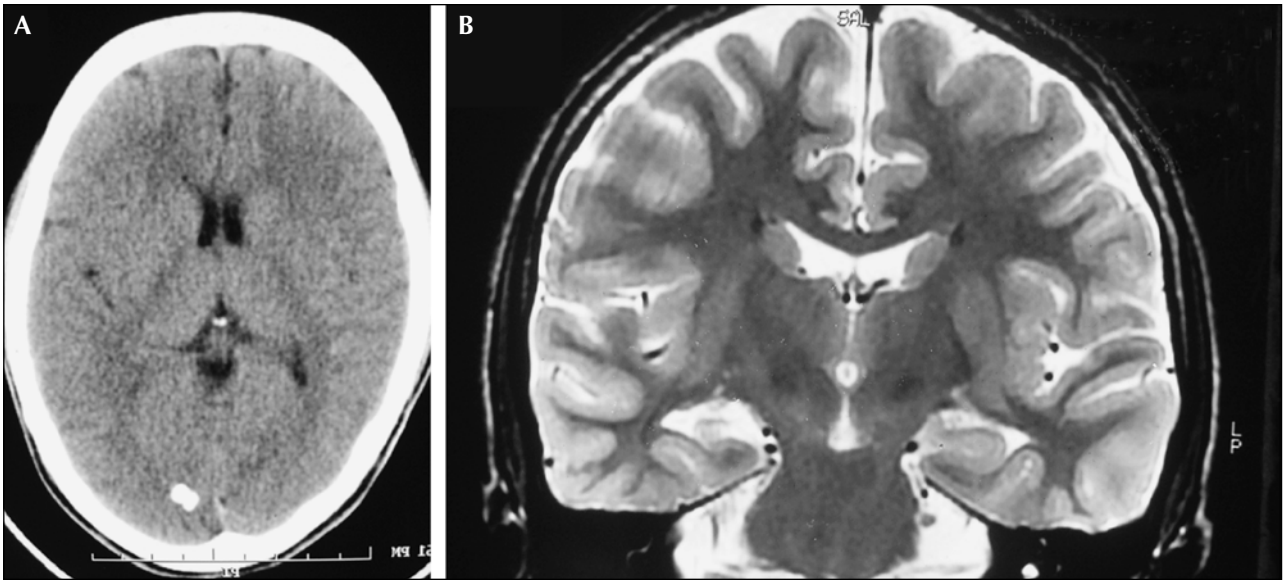

Figure 4. Case 4. **A**) Initial CT revealed the right occipital calcification, possibly a sequela of earlier cysticercus granuloma. **B**) Coronal oblique FLAIR image performed several years later showing the dilated right temporal horn with hippocampal hyperintensity.

that develops around a degenerating cysticercus in its vicinity. In a single, previously reported case of hippocampal resection for medically refractory epilepsy, a calcified lesion was found bordering the edge of the hippocampus. Histopathology revealed abundant corpora amylacea within the hippocampus, suggesting that the hippocampus was affected by the inflammatory and gliotic reaction to a topographically-related, degenerating cysticercus (Chung *et al.* 1998).

Another hypothesis is that the occurrence of inflammation in the vicinity of the hippocampus results in clinical or electrographic seizures within that same hippocampus, leading ultimately to the development of an independent epileptic focus. Experimental studies have suggested that the injection of early, but not late, *Taenia crassiceps*-granuloma material into the hippocampus of mice is highly epileptogenic (Stringer *et al.* 2003). These experiments provide support for a direct involvement of the hippocampus by brain inflammatory responses to degenerating cysticerci. In a larger series describing the coincidental occurrence of calcified cysticerci with HS, about 8% of calcifications were located in the temporal lobe ipsilateral to HS (Leite *et al.* 2000). This therefore, may be only one of several mechanisms responsible for the development of HS in NCC.

The kindling model of epilepsy, wherein application of repeated, subthreshold stimuli lead to the development of seizures, may also be relevant to the occurrence of temporal lobe epilepsy in association with NCC (Goddard *et al.* 1969, Morrell 1985). Limbic structures are particularly susceptible to kindling, and the widespread connectivity of these structures with neocortical regions, where NCC lesions are commonly located, may be responsible for temporal lobe seizures in patients with NCC.

Finally, the occurrence of NCC lesions in association with HS, or *vice versa*, may be merely coincidental. This was suggested in a comparative evaluation of several clinical and histological parameters of HS in a sample of patients with HS with or without calcified brain cysticerci (Leite *et al.* 2000, Wichert-Ana *et al.* 2004, Terra-Bustamante et *al.* 2005, da Gama *et al.* 2005). These authors compared consecutive patients with HS, with and without calcified brain cysticerci, and found no difference in their clinical presentation, neuropsychological characteristics, pathological features as discerned upon histology of resected hippocampii, or surgical outcome. They concluded that the calcified brain cysticerci were merely coincidental findings and that they in no way influenced the development of HS. One of the reasons that the authors did not find any difference in the clinical presentation may be the disparate mechanisms that underlie the association between NCC and HS. We agree with the authors of these studies that in a proportion of cases, NCC and HS may coexist purely by chance. Hippocampal sclerosis is a common substrate underlying epilepsy. Calcified NCC is frequently noted upon imaging studies of both symptomatic and asymptomatic individuals in *T. solium*-endemic regions (Montano *et al.* 2005). Hence, it is likely that the two conditions may occur coincidentally.

Our present series has certain limitations. The causality between NCC and HS cannot be definitively proved. Hippocampal sclerosis was demonstrated only upon follow-up imaging, but our patients were not investigated at onset using a specific epilepsy-protocol evaluating hip-

pocampal structure and volumetry. Secondly, this series is not derived from a prospective and systematic MRI evaluation of all cases with NCC seen at our center. In addition, due to the lack of availability of video-EEG facilities, we were unable to establish whether the hippocampal abnormalities were the source of origin of seizures apart from in Patient 4. Finally, there is a lack of histological evidence for both NCC and HS, as this is not a surgical series. Pertinent in this regard is the pathological basis of the granulomatous condition noted on imaging in Patient 3. The differential diagnosis in this case would include both, tubercular and NCC granulomas; the latter possibility cannot be discounted inasmuch as the CSF PCR for *M. tuberculosis* was negativeon two occasions, cysticercosis serology was positive and that NCC granulomas can spontaneously resolve (Rajshekhar 2001).

In conclusion, there may be several reasons for the observed association of HS and NCC. While a purely coincidental basis for the dual pathology cannot be discounted, we postulate that, among several mechanisms, seizures due to NCC may constitute an antecedent event for HS or that NCC lesions involve the hippocampus either indirectly though kindling or infrequently, directly by being located close to the latter. Neurologists managing seizure disorders in *T. solium*-endemic regions should be aware of this association and should consider that patients with chronic epilepsy related to NCC may harbor dual pathology, and that the epilepsy may be related to HS rather than the NCC lesions. Serial MRIs may be indicated in patients who develop signs and symptoms of temporal lobe epilepsy, or progressive memory impairment. A prospective MRI evaluation, with coronal images of the hippocampal volumes and anatomy in persons with seizures due to NCC is warranted. □

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