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Microsurgical Treatment of Epilepsy with Parenchymal Neurocysticercosis*

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Summary: Parenchymal neurocysticercosis is the most common form of neurocysticercosis in the central nervous system (CNS), which mainly causes epilepsy and usually responses well to routine medications. However, there are appreciable cases of relapses refractory to medical treatment. We investigated microsurgical treatment of epilepsy with parenchymal neurocysticercosis. Nine cases of epilepsy caused by parenchymal neurocysticercosis from 2002 to 2018 were analyzed retrospectively. Cysts in 7 cases were completely removed. No case died of operation and no new dysfunction of the nervous system was observed after surgery. Among the other 9 cases, 8 cases became seizure-free or controlled by medicine according to the postoperative follow-up for 6 months to 9 years. One case was lost for follow-up. It was suggested that epilepsy with parenchymal neurocysticercosis can usually be controlled after routine medications. However, surgery is still indicated in some cases and careful microsurgery is associated with satisfactory clinical outcomes in appropriately selected cases.
Key words: epilepsy; parenchymal cysticercosis; neurocysticercosis; microsurgery

Neurocysticercosis is the most common parasitic infection of the human central nervous system (CNS), and a leading cause of acquired epilepsy worldwide, especially in developing countries[1–7]. Neurocysticercosis results from ingestion of the eggs of Taenia solium, the pork tape worm. The oncospheres hatch in the intestine, penetrate the intestinal wall and disseminate to several body tissues, showing strong tropism to the CNS. The clinical manifestations of neurocysticercosis are non-specific and varies due to the parasite itself and/or an inflammatory reaction around degenerating cysts in the CNS. It may cause epilepsy, chronic meningitis and hydrocephalus, depending on the number of lesions and the developmental stage of the cysticercus. Epilepsy is the most common clinical manifestation and more frequently observed in patients with parenchymal neurocysticercosis than in those with subarachnoid or ventricular disease, occurring in 50% to 80% of patients[7–9].

Neurocysticercosis has been classified depending on the location of cysts, its clinical presentation, prognosis and cyst viability[8, 10–12]. In the parenchymal neurocysticercosis, the parasite lodges in the brain parenchyma as single or multiple cysts forming clumps. Occasionally, the cyst may continue to grow and produce a tumor-like syndrome and mass effect. Growth of a parenchymal cysticercus is not a common event and may be life-threatening, which deserves active management, either with antiparasitic drugs or by surgical excision. Meanwhile, pericystic inflammation can result in a granuloma formation. Finally, the cyst is processed by the cellular response, and its remnants either are not detectable by imaging or become calcified lesions. Epilepsy can attack during all these development stages and usually be the most frequent clinical presentation of parenchymal neurocysticercosis. It commonly presents with simple partial seizures, partial complex seizures, simple partial seizures with secondary generalization or generalized seizures, which usually can be controlled with antiepileptic drug therapy. Both praziquantel (a heterocyclic isoquinolone) and albendazole (an imidazole) have been shown to offer a reasonable and effective treatment for parenchymal cysticercosis. However, a prospective study showed that up to 50% of patients with parenchymal neurocysticercosis successfully treated with cysticidal drugs had relapses after withdrawal of antiepileptic drugs[13]. Taking into consideration of acute mass effect and intractable

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seizure recurrence, surgery still has a role in the management of some patients.

In this study, we reported 9 cases suffering from epilepsy with parenchymal neurocysticercosis and receiving microsurgical treatment. The characteristics, treatment, and outcomes of these cases were well analyzed.

1 MATERIALS AND METHODS

1.1 General Information

Nine cases (7 males, and 2 females) with parenchymal neurocysticercosis were operated on in Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, China from 2002 to 2018. The patients were aged from 20 to 59 years (mean, 36.9 years) and suffered with courses of disease varying from 1 month to 9 years.

1.2 Epidemiology and Clinical Manifestation

The cases included in this study were selected from 96 cases (9.4%) of parenchymal neurocysticercosis and the total 221 cases (4.1%) of neurocysticercosis who were hospitalized during the same period. All the 9 cases suffered from chronic headache and seizures, of which, 4 were general seizures, 3 were partial, and 2 were mixed. The first manifestation was epilepsy in 6 cases. The rest 3 cases were hospitalized for intracranial hypertension (headache, nausea, and vomiting), 2 of which were initially misdiagnosed as brain tumors. Of these operated cases, 5 cases had taken two or more kinds of antiepileptic drugs for more than two years, which, however, failed to control the seizures. One case refused to take long-term antiepileptic treatment. Intracranial hypertension occurred in one case due to acute mass effect. Two cases were misdiagnosed as brain tumors preoperatively.

1.3 Preoperative Auxiliary Examination

Cranial computed tomography (CT) and magnetic resonance imaging (MRI) were performed on all patients. The findings of CT and MRI usually differed depending on the parasite's location and its stage of development, as neurocysticercosis may be detected in multiple locations of CNS, and cysticerci may occur at different development stages. In the 9 cases, parenchymal lesions were mainly found in the temporal lobe (3 cases), the frontal lobe (2 cases), the parietal lobe (2 cases) and the occipital lobe (2 cases). Low density was present on CT images. On the MRI scanning, it showed long T1 as well as isointense on T1-weighted sequences and isointense to hypointense with or without a central area of hyperintensity on T2-weighted sequences. Evident edema was noted surrounding the lesion in 5 cases on CT or MRI plain scanning, especially T2-weighted sequences (fig. 1). On contrast enhancement scanning, enhancement of the cystic wall was present, and clear boundaries were noted between lesions and normal tissues, as well as brain edema. In one case, calcified cysticerci appeared on CT as small hyperdense nodules without perilesional edema or abnormal enhancement after contrast medium administration.

As all the operated cases suffered from seizures, every patient underwent interictal electroencephalographic (EEG) examination preoperatively. Perilesional abnormal discharges were recorded in 7 of 9 cases.

Enzyme-linked immunosorbent assay (ELISA) was performed to detect anticysticerca antibody preoperatively in 6 cases and postoperatively in 3 cases. However, only one case with polycystic lesions was detected positively. The rest cases with a single or two cerebral cysts or with calcification showed negative results.

1.4 Microsurgery

Operation process was regular with craniotomies performed at the sites closest to the lesions. The bone flap was designed often larger than that in usual craniotomies to have adequate access to the epileptogenic cortex. We detached lightly along the pia mater under intraoperative ultrasound, which guided surgical procedures for localization in real time. When the yellowed cyst lesion was exposed, we camped it with micro-tumor clamp and detached with "waterjet". It was important that the lesion should be resected completely without strong and rough detachment,

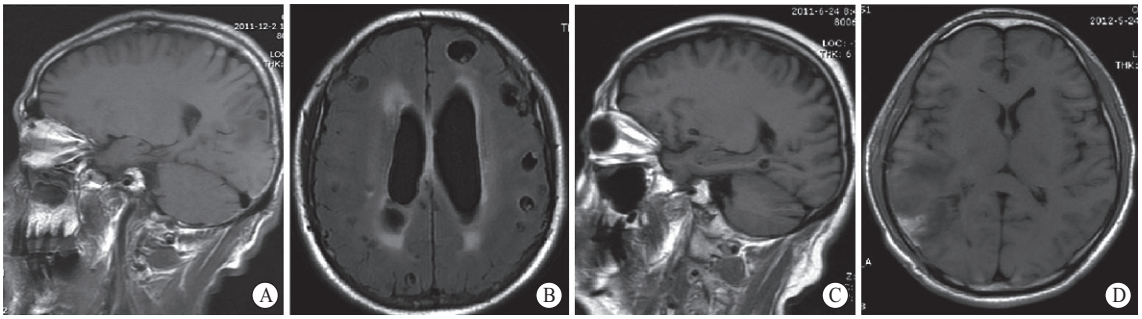

Fig. 1 A: T1-weighted sequence shows evident edema surrounding the lesion; B: T2 flair shows multiple cysts with isointense to hypointense with or without a central area of hyperintensity; C: T1 sequence shows a lesion with hypointense in the temporal lobe; D: T1 sequence shows a single giant cortical cyst exhibiting pseudotumor with edema.

leaving the brain tissue intact.

For the cases with growing parenchymal cysticercus, which might cause mass effect, the goals of microsurgery were removal of the scolex and the cystic wall in brain parenchyma without damaging normal brain tissues. Especially, for the cases with ring enhanced cisternal lesions, every care was taken to avoid spillage of cyst contents to avoid the possibility of severe meningitis. For the cases with calcified cysticerci, the removal of lesion with abnormal discharges was the main operation.

After resection of the lesion, electrocorticography (ECoG) with subdural electrodes or frame electrodes was performed. If abnormal discharges were recorded within "noneloquent" areas, we enlarged the cortical resection to normal cortex on ECoG. However, if abnormal discharges were recorded in the eloquent area, we performed subpial transaction additionally according to the ECoG recordings.

1.5 Postoperative Treatment

For all the patients with epilepsy, antiepileptic therapy was continued for 1–2 years, until they were seizure-free and the interictal EEG demonstrated no epileptiform abnormalities. Corticosteroids were used in postoperative short-term, especially for the cases with brain edema or other reactions after operation emerged. Conventional antiparasitic therapy was offered for the positive cases tested with anticysticerca antibody.

2 RESULTS

Cranial CT or MRI was performed within 48 h after surgery for all the patients. Seven cases in this series underwent complete removal of cysts. One case with calcified cysticerci and one case with polycystic lesions underwent successful removal of seizure focus according to intraoperative ECoG. All the removals were pathologically confirmed to be cysticercosis (fig. 2). None of them died of operation and no new neurological dysfunction was observed after surgery.

Postoperative follow-up was done for 6 months to 9 years. Among the other 9 cases, 8 cases became seizure-free or controlled by medicine with no epileptiform discharges re-detected in their EEG. One case was lost for follow-up.

3 DISCUSSION

3.1 Epidemiology of Neurocysticercosis

There is few comprehensive epidemiologic studies that could define the precise prevalence of cysticercosis[14]. However, there are indirect data, mostly from medical institutions, which point out the conspicuous presence of neurocysticercosis as a cause of the most common parasitic infestation of the human brain. And this intracranial parasitic infestation accounts for a leading cause of acquired epilepsy, especially in developing countries[4, 6, 7, 12, 15, 16]. In the developed world, a recent increase in the number of patients with neurocysticercosis was noted, mainly due to the growing number of immigrants from endemic areas[17–20].

Two hosts are involved during the complex life cycle of Taenia solium, of which, humans are the only definitive hosts for the adult tapeworm. The brain parenchyma is the most frequently affected CNS site. Parenchymal cysticercosis can be divided into different pathologic stages (the vesicular stage, the colloid vesicular stage, the granular nodular stage and the calcified stage) depending on cysticerci development and different forms (active form, transitional form and inactive form) according to criteria of viability[10, 21]. The stage and classification establish the correlation between the different forms of neurocysticercosis and its clinical manifestations, and can be used for planning therapeutic strategies.

3.2 Clinical Manifestations of Parenchymal Neurocysticercosis

The clinical pleomorphism of parenchymal neurocysticercosis mainly depends on individual differences in the number and location of the lesions within the brain parenchyma and on variations in the severity of disease activity. The most common clinical manifestation of parenchymal neurocysticercosis is seizures, which may represent the primary or sole manifestation of the disease in almost more than a half of patients[7–9, 22–24]. Focal neurological dysfunctions

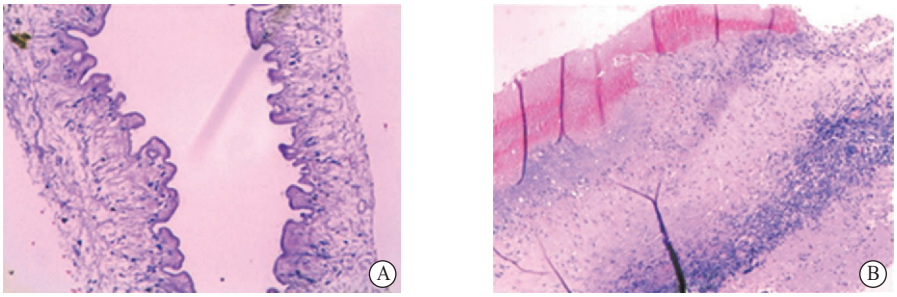

Fig. 2 Representative image showing the body of a parasitic worm (A: HE, ×400), and hemorrhage necrosis and inflammatory cell infiltration (B: HE, ×100)

have been described in up to 20% patients with neurocysticercosis and are related to the size, number and location of the parasites[25, 26]. These manifestations usually follow a subacute or chronic course, resembling that of a brain tumor with large subarachnoid cysts compressing the brain parenchyma[27]. Another common clinical manifestation is intracranial hypertension, associated or not with seizures or focal neurological lesions. The most common cause of this syndrome is hydrocephalus, which may be either related to cysticercotic arachnoiditis, granular ependymitis, or ventricular cysts[27–29].

The complex characteristics of cysticercosis explain the variable clinical manifestations of the disease. Nevertheless, seizure is the most usual and frequent clinical manifestation in a large proportion of neurocysticercosis with parenchymal granulomas or calcifications, which might be the most frequent finding on imaging studies. Especially, epileptogenesis in patients with calcified neurocysticercosis has been a subject of debate. Calcified cysticercus larva with perilesional abnormality is thought to be responsible for seizures in patients with neurocysticercosis while some calcified cysts without perilesional abnormality are still associated with seizures. Thus, calcified neurocysticercosis lesion which causes seizures might has different pathophysiologic basis[30]. Recent data suggest that calcified cysticerci are not clinically inactive nor pathologically inert lesions, as they may cause recurrent seizures when parasitic antigens trapped in the calcium matrix are exposed to the host immune system due to a process of calcification remodeling[31, 32].

3.3 Diagnosis and Differential Diagnosis of Parenchymal Neurocysticercosis

Despite the advances in neuroimaging and immune diagnostic tests, the diagnosis of parenchymal neurocysticercosis is still a challenge in many patients, because clinical manifestations are nonspecific, most neuroimaging findings are not pathognomonic, and some serologic tests have low sensitivity and specificity. What's more, the complexity of manifestations of neurocysticercosis makes its diagnosis difficult. A set of diagnostic criteria are proposed based on objective evaluation of clinical, neuroimaging, immunological, and epidemiological data[11, 33, 34]. This set includes four categories of criteria—absolute, major, minor and epidemiologic. These criteria provide two degrees of diagnostic certainty: definitive diagnosis and probable diagnosis[35].

The accurate diagnosis is based on the combination of clinical, epidemiologic, radiographic, and immunologic information. Nevertheless, the prevalence of misdiagnosis is still high on clinical grounds, especially for single parenchymal neurocysticercosis. Useful diagnostic clues may derive from epidemiological data, careful neuroimaging studies and highly specific immune diagnostic tests. Of the 9 patients reported on here, 2 cases were misdiagnosed as having gliomas, which, however, were found by pathologic demonstration of the cysticercosis. Therefore, improvement of diagnosis of neurocysticercosis is imperative.

3.4 Microsurgical Indications and Techniques

The treatment approach and response depends on the location of the pathology. For patients with confirmed parenchymal neurocysticercosis, pharmacotherapy should be performed in routine. The treatment modalities available to patients include: cysticidal agents (to kill larvae), corticosteroids (to decrease or prevent the inflammatory reaction) and antiepileptic drugs (to prevent or decrease the severity and number of seizures)[12]. Guidelines for treatment of neurocysticercosis must be individualized related to the number and location of lesions, as well as depending on the viability of the parasites within the nervous system. As demonstrated by numerous experiments and clinical practices, praziquantel and albendazole are effective antiparasitic drugs[36–38]. Seizures should be treated in a similar manner to other causes of secondary seizures. First-line antiepileptic drugs usually prove effective in this type of seizures. After resolution of the parasitic infection with normalization of imaging studies, a majority of patients can eventually discontinue antiepileptic drugs with seizure-free[11]. Corticosteroids are the primary management for cysticercosis arachnoiditis or encephalitis, as they may decrease neurological symptoms caused by the death of the parasite[8]. In general, most patients will recover after routine medications.

However, it must be remembered that some forms of parenchymal neurocysticercosis should not be treated with cysticidal drugs[11, 12, 35]. For example, patients with refractory seizure due to parenchymal neurocysticercosis should undergo microsurgery to remove the epileptogenic focus. In addition, a fraction of patients with acute cysticercotic encephalitis, which may be exacerbated with cysticidal drugs, should undergo craniectomy to improve malignant intracranial hypertension. Surgery is not indicated in all the cases of parenchymal neurocysticercosis and when performed, the microsurgical indications should include: (1) a single giant cortical cyst or large clumps (maximal lesion diameter greater than 2 cm) exhibiting acute mass effect, provided that the lesion is surgically accessible; (2) lesions that, though small, hinder cerebrospinal fluid circulation or compress important functional regions; and (3) pseudotumor (edema) refractory to medical treatment, progressively worsening symptoms (uncontrolled intracranial hypertension or seizures)[11, 12, 29, 35, 39].

The surgical approach in the treatment of parenchymal neurocysticercosis depends on the number, size and location of the cysticerci and the

anatomic-pathologic characteristics of the infection. The following points must be defined: (1) the number of cysts (single or multiple); (2) the size of cysts (>2 or <2 cm in diameter); (3) the location of the cysts (in the functional region or nonfunctional; Is the lesion surgically accessible?); (4) the biological stage of the parasite; and (5) the secondary pathological conditions produced in CNS by the presence of the cysts. As refractory seizures are the main complain for surgical treatment in our operated cases, intraoperative ECoG proved to be particularly important. In order that the intraoperative ECoG recording is not disturbed by medication, the anticonvulsants will usually be stopped 1 to 2 days before the operation. Some anesthetics that affect the EEG recording greatly should be avoided. The bone flap is usually designed larger than that in usual craniotomies to have adequate access to epileptogenic cortex. When the dura is incised, the spotted lesion can often be seen under the arachnoid. Intraoperative ultrasound can play a real time guide for surgical localization. We place electrodes marked with numbers around the lesion, record ECoG from the lesional to perilesional regions, write down the numbers of the electrodes that record spikes or sharp waves and draw a picture of the cortex area with abnormal ECoG. On the surface of the lesion or around the lesion, sporadic or dense spikes, sharp waves or polyspikes can be recorded through several electrodes. We resect the lesion and surrounding cortex, until there are no further epileptiform discharges on the ECoG recording.

The goals of microsurgery are to remove the epileptic focus with more significance, as well as the full cysts if feasible. During surgery, leakage of cyst contents should be prevented to avoid the possibility of severe aseptic meningitis, especially, for the cases with ring enhanced cisternal lesions. Leakage of the cyst fluid should be followed by repeated irrigation with normal saline[39–41]. Epileptic pathological focus namely "epileptogenic lesion" is believed to cause indirectly or directly epileptiform discharges on EEG and complete removal of the epileptogenic region can result in seizure freedom. However, the epileptogenic region does not always overlap the epileptogenic lesion, and there may be one or more functional epileptic foci within or at the edge of the lesion, even in the remote cortex[42, 43]. Thus, to control the seizure caused by parenchymal neurocysticercosis, not only the lesion itself, but also these epileptic foci should be removed. When the epileptic focus was located in the eloquent cortex, we performed multiple subpial transection, which transected the level short cortex fibers, and obstructed the extensive cortical synchronization caused by focal epileptic discharges.

Conflict of Interest Statement

There is no conflict of interest in this study.

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