# Neurocysticercosis: Focus on Intraventricular Disease

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**Intraventricular neurocysticercosis is of concern because it is associated with a poorer prognosis than is parenchymatous disease. Frequently, associated hydrocephalus occurs, which may recur after treatment. We report on 11 patients with intraventricular cysticercosis (from a larger case series of 33 patients) and evaluate clinical presentations, neuroimaging findings, and responses to treatment, especially of ventricular disease. Intraventricular cysticercosis represented 33% of our cases. Seven patients presented with increased intracranial pressure; four required ventriculoperitoneal shunting. Parenchymatous symptomatic cysticercosis is largely a result of the host inflammatory response, presenting in our series with epileptic seizures in 73% of the patients (tonic clonic generalized seizures occurred in 64% and partial simple seizures in 9%). The prognosis for parenchymatous inflammatory disease is good. We advocate the use of anthelmintic treatment with albendazole in all cases of intraventricular cysts, and if hydrocephalus occurs, then shunt procedures or ventriculostomy is necessary. These patients must be monitored closely for recurrent hydrocephalus.**

A significant increase in the frequency of neurocysticercosis in the United States corresponds to a growth in immigration from areas in which the disease is endemic and to the use of accurate diagnostic neuroimaging methods. Neurocysticercosis is a recognized health problem in certain areas of the United States with a large immigrant population. Parenchymal disease is associated with a better prognosis than is extraparenchymal disease, and several authors have emphasized a high rate of poor outcomes in cases of intraventricular disease [1–4]. We conducted a study at Texas Tech University Health Sciences Center (TTUHSC), in El Paso, a large city on the United States–Mexico border, to evaluate (1) clinical presentations of neurocysticercosis; (2) the wide variety of neuroimaging findings; and (3) management of intraventricular cysts.

## Methods

This study was carried out at outpatient facilities of TTUHSC and at inpatient and outpatient facilities at Thomason Hospital, a 350-bed county hospital nearby. In this case series of 33 patients, 7 patients were identified retrospectively and 26 prospectively. They were treated from June 1990 to June 1995. Data were analyzed with the aid of the SPSS 6.1 spreadsheet program (SPSS, Chicago). This series included all patients who presented to TTUHSC and Thomason Hospital and for whom the diagnosis of active and inflammatory neurocysticercosis was made. Patients with only inactive calcified lesions were not included. Information collected about patients included age, sex, demographic data, clinical presentation, neuroimaging findings, immunodiagnostic results, and treatment management. Multiple admissions and visits were counted only once.

We used the criteria of Carpio et al. [5] to classify neurocysticercosis into active, inflammatory, and inactive calcified lesions (table 1).

*Active (vesicular, viable) cysts.* Such cysts (figures 1 and 2) are seen on $T_1$-weighted MRI sequence as rounded cysts of low intensity signal without surrounding ring enhancement. The fluid inside the cysts have an MRI signal similar to that of CSF. The scolex inside the cysts can sometimes be visualized on $T_1$ as a high-intensity signal dot because of its high fat content. $T_2$-weighted imaging and proton density sequences also help one to visualize the scolex. On a CT scan, active cysts are hypodense without ring enhancement.

*Inflammatory (involutional, transitional, colloidal) cysts.* These cysts (figure 3) are seen in the parenchyma of the brain as hypodense-hypointense cystic lesions surrounded by an enhancing annular capsule and pericystic edema similar to an abscess on both CT and MRI [6–8]. CT sometimes shows concurrent small calcifications.

*Inactive calcified lesions.* These lesions (figure 4) are the end-stage of the cysts. There are no cystic or inflammatory lesions; only rounded single or multiple calcifications are present.

### Other Diagnostic Procedures

ELISA of serum from all patients was done. Enzyme-linked immunoelectrotransfer blotting (EITB) of serum and CSF was done for 15 patients. For nine patients, lumbar puncture was contraindicated; for six patients, neuroimaging was considered sufficient for establishing the diagnosis. An electroencephalo-

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**Table 1.** Classification of neurocysticercosis.

| Feature | Active | Inflammatory | Inactive |
|---|---|---|---|
| Cysts in brain parenchyma | Asymptomatic unless in crucial areas of the brain; hypodense on CT; CSF signal on MRI | Symptomatic (seizures); appearance of abscess on CT and MRI | One or more calcifications per cyst; symptomatic if in crucial area of the brain |
| Cysts in subarachnoid space | Asymptomatic; hypodense on CT; CSF signal on MRI | Meningeal reaction; CSF changes; calcifications; CSF signal on MRI | Calcifications |
| Cysts in ventricles | Symptomatic if they obstruct CSF flow; hypodense on CT; CSF signal on MRI | Meningeal reaction; CSF changes; hydrocephalus; CSF signal on MRI | No calcifications; late hydrocephalus |

gram (EEG) was performed for 32 patients. Tissue diagnosis was performed for four patients.

The type of treatment given varied with the location of the cysts, clinical status of the patient, patient's preference, presence of hydrocephalus, and attending physician's preference and experience. Treatments included administration of anthelmintic drugs with steroids, surgical removal of the cysts, ventriculoperitoneal shunts, combinations of these methods, and symptomatic treatment only.

## Results

There were 33 patients with neurocysticercosis, predominantly males (78%) and natives of El Paso (60%); all were Hispanics, and the mean age was 34 years (range, 16–70 years).

*Clinical presentation.* Tables 2 and 3 summarize the clinical presentations of the 33 patients. Epileptic seizures occurred in 24 patients (73%); 21 of them (64%) had tonic clonic generalized seizures, and 3 had partial simple seizures (9%). The neurological examination findings were normal in 22. Two patients presented with transient hemiparesis. Symptoms and signs of increased intracranial pressure (ICP; headaches, drowsiness, vomiting, papilledema) occurred in nine patients (27%); two of them had meningeal irritation.

*Neuroimaging.* Nineteen patients (58%) had multiple cystic lesions (figure 5), located in the subarachnoid space, basal cisterns, and brain parenchyma, with a predilection for the parietal and frontal lobes. In most cases inflammatory parenchymatous cysts coexisted with active cysts and inactive calcifications (figure 6). A group of 11 patients (33%) with multiple lesions also had coexisting intraventricular cysts (figure 7), with concurrent hydrocephalus in 7; the other 4 patients did not have ventricular enlargement. A single parenchymatous cyst (figure 3) was present in 14 patients (42%), in the parietal region of 11 and in other areas of the brain in 3.

*Clinical and neuroimaging correlations.* Table 3 shows that 24 patients with seizures had inflammatory single or multiple parenchymatous cysts. No correlation was found between severity of seizure and intensity of infestation. For example, a patient who presented in status epilepticus had a single frontal cyst (figure 3). In the group of 11 patients with intraventricular cysts, seven had hydrocephalus and increased ICP. Two patients with active parenchymatous and subarachnoid cysts had meningitis; the other two patients with inflammatory parenchymatous cysts had seizures.

*Immunodiagnostic methods.* ELISA of serum and EITB of serum and CSF specimens were used to support the diagnosis. Among the 33 patients, the ELISA was positive for 16 (62%): 11 of 11 with intraventricular disease, 4 of 19 with multiple parenchymatous inflammatory and subarachnoid cysts, and 1 of 14 with a single parenchymatous cyst.

EITB was performed on serum and on CSF obtained by lumbar puncture or ventriculostomy. The test was positive for

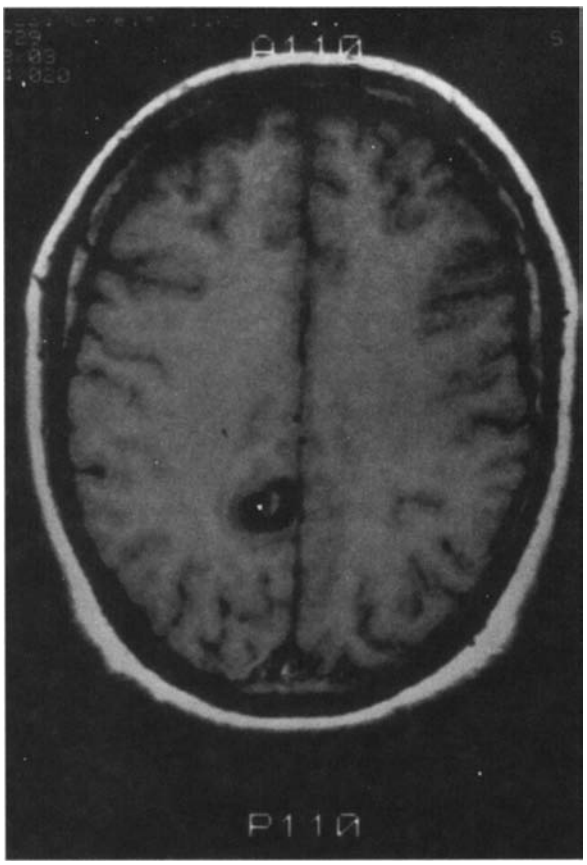

**Figure 1.** Axial $T_1$-weighted MRI shows a right posterior parietal active cyst in a 29-year-old man with tonic clonic generalized seizures. Some edema surrounding the cysts may represent inflammation. The scolex is visible in the cyst as a hyperintense structure.

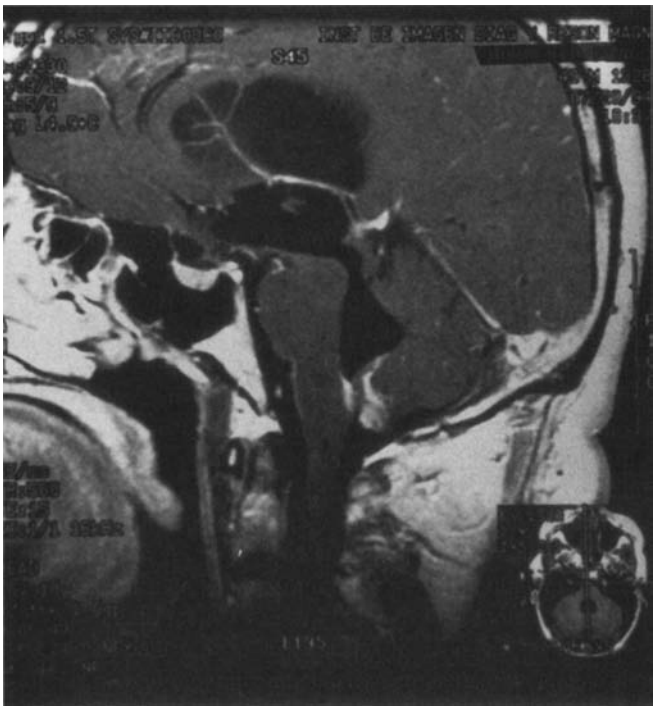

**Figure 2.** Sagittal $T_1$-weighted MRI with gadolinium shows intraventricular and subarachnoid active cysts in a 45-year-old man with increased intracranial pressure, meningeal signs, and hydrocephalus.

12 patients (80%): 4 of 4 with intraventricular cysts, 2 of 2 with subarachnoid cysts and meningitis, 5 of 19 with parenchymatous disease, and 1 of 14 with a single parenchymatous cyst.

Lymphocytic pleocytosis (total cell count, 19–384) was demonstrated for the two patients with meningeal irritation.

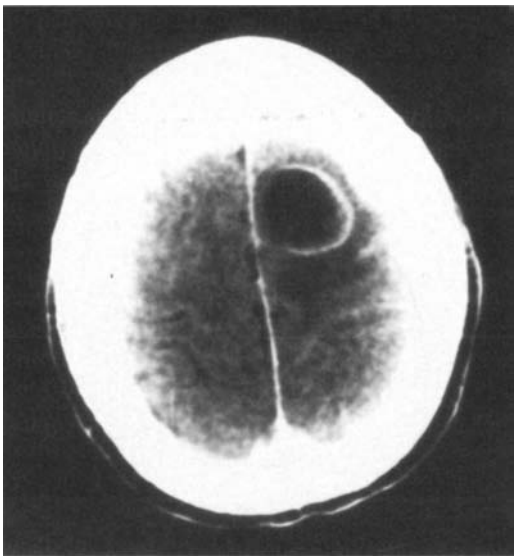

**Figure 3.** Axial CT with intravenous contrast shows a single parenchymatous inflammatory cyst with ring enhancement in a 28-year-old man who presented with tonic clonic generalized seizures.

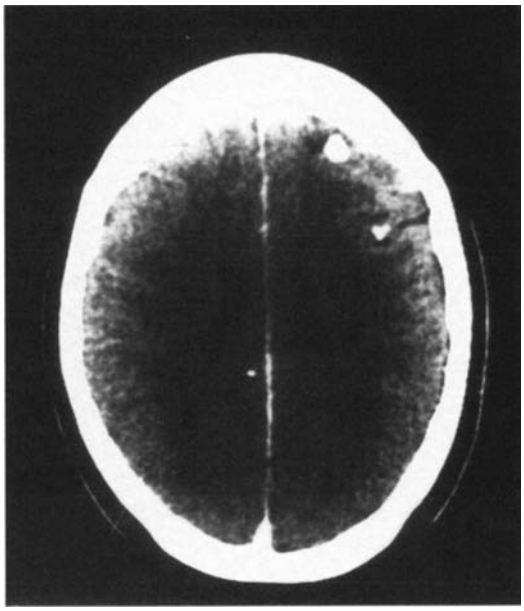

**Figure 4.** Axial CT with intravenous contrast shows multiple inactive parenchymatous calcifications in a 49-year-old woman, 6 months after treatment of cysticercosis with praziquantel.

The eosinophil count ranged from 1 to 41 cells. The CSF findings for the other 13 patients were either normal or not significant, with only slight increases in the protein level and number of cells.

*Tissue diagnosis.* Such diagnostic testing was performed for four patients. Two patients' specimens were obtained from a single lesion; the other two patients' specimens were of intraventricular content obtained through ventriculostomy and from multiple cysts removed from the posterior fossa, respectively.

*EEGs.* EEGs were performed for 32 patients. The EEG did not add to the information obtained in the medical history, physical examination, and neuroimaging. EEGs were normal for 21 patients. EEGs showed abnormalities consisting of postictal diffuse slowing in 5 patients, mild diffuse transient frontal

**Table 2.** Clinical presentation of neurocysticercosis.

| Presentation | No. of patients with indicated condition/total no. of patients (%) |
|---|---|
| Epileptic seizures | |
| Tonic clonic generalized seizures | 21/33 (64) |
| Partial simple seizures | 3/33 (9) |
| Tonic clonic generalized seizures or partial simple seizures | |
| Normal examination findings | 22/33 (67) |
| Mild hemiparesis | 2/33 (6) |
| Increased intracranial pressure, due to | |
| Obstructive hydrocephalus | 7/33 (21) |
| Cerebral edema or meningitis | 2/33 (6) |

**Table 3.** Clinical and neuroimaging correlations in cases of neurocysticercosis.

| No. of patients | Clinical presentation | Neuroimaging findings |
|---|---|---|
| 22 | Epileptic seizures | Inflammatory parenchymatous cysts, single or multiple |
| 2 | Epileptic seizures | Inflammatory parenchymatous and intraventricular cysts |
| 7 | Increased intracranial pressure | Intraventricular cysts with hydrocephalus |
| 2 | Increased intracranial pressure, meningitis, and cerebral edema | Multiple subarachnoid parenchymatous and intraventricular cysts |

slowing in 1 patient with increased ICP and hydrocephalus, focal spikes in 2 patients, and persistent focal slowing in 3 patients.

### Treatment

Six patients with seizures and parenchymatous cysts declined treatment with anthelmintic medications. Of this group, two patients had active cysts that became inflammatory (one in 18 months and the other in 23 months), and four had inflammatory cysts. The inflammatory stage lasted <12 months before the cysts disappeared, leaving a calcified dot. These patients were treated only with antiepileptic drugs and kept under close supervision.

Twenty-seven patients received treatment with praziquantel (50 mg/[kg · d] for 15 days). Steroids were given to all patients during the 15 days of treatment. A CT or MRI was performed every month for 3 months and every 2 months thereafter. Neuroimaging showed progressive disappearance of the parenchymatous cysts after the first month of treatment (figures 8A and 8B). Most parenchymatous cysts disappeared by the third to sixth month following treatment.

None of the intraventricular cysts disappeared with use of praziquantel. For all 11 patients with intraventricular cysts, we waited 3 months to begin treatment with albendazole. These patients received two cycles of treatment with albendazole, 1 month apart. Each cycle consisted of administration of a dose of 15 mg/(kg · d) for 15 days. Intraventricular cysts disappeared with use of albendazole by the third month after treatment in all patients (figure 9A and 9B). Hydrocephalus was present in seven patients; four of these patients required a ventriculoperitoneal shunt. The remaining three patients, who had moderate hydrocephalus and a good clinical condition, were followed closely and did not require surgical intervention.

Among the 24 patients with seizures, 16 were weaned of antiepileptic medication 1 year after they were free of seizures. They have continued to be seizure-free. Eight patients continued to take antiepileptic medication, and they have been seizure-free. Findings of MRI of the brain have remained normal in this group.

## Discussion

The development of an active cysticercus, from the time the eggs are ingested, is completed in ~60–70 days [9]. Cysts can be parenchymatous, subarachnoid, or intraventricular. A viable larva in an active-phase cyst can live up to 7 years [9].

Active cysts usually do not cause clinical symptoms, unless they are located in crucial areas of the brain where they may cause seizures or hydrocephalus. Active intraventricular cysts may persist for years, but they may become symptomatic if they obstruct the CSF flow, with consequent hydrocephalus and increased ICP. The lateral ventricles and the fourth ventricle are the most common locations. Intraventricular cysts (33%) in this series were seen less frequently than parenchymatous cysts (66%).

There is significant morbidity and mortality associated with intraventricular disease [1–4]. According to the current standards, when both hydrocephalus and increased ICP occur, either a ventriculoperitoneal shunt or a temporary ventriculostomy must be performed as a means of controlling the increased ICP [10]. Surgical removal of the cysts should be done if they

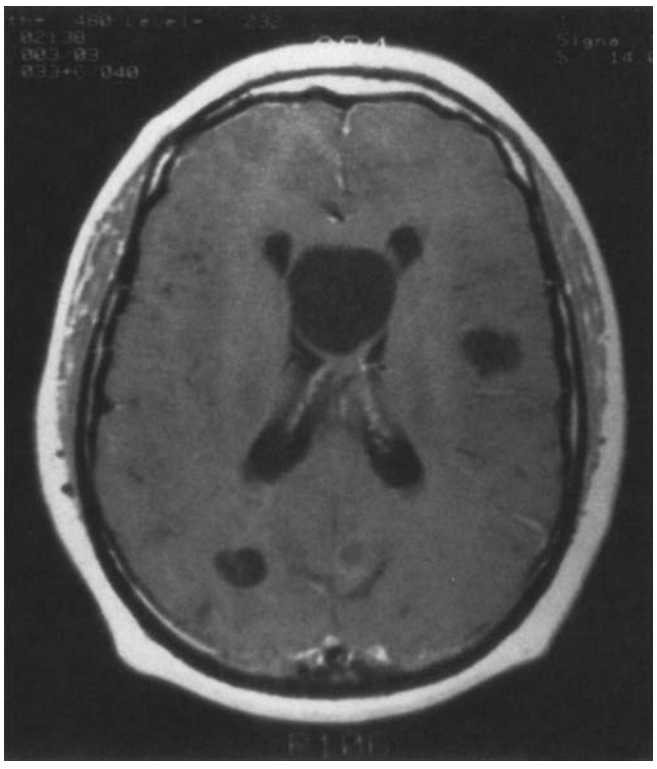

**Figure 5.** Axial $T_1$-weighted MRI with gadolinium shows multiple active inflammatory, parenchymatous, and intraventricular cysts in a 46-year-old man who presented with headaches and tonic clonic generalized seizures.

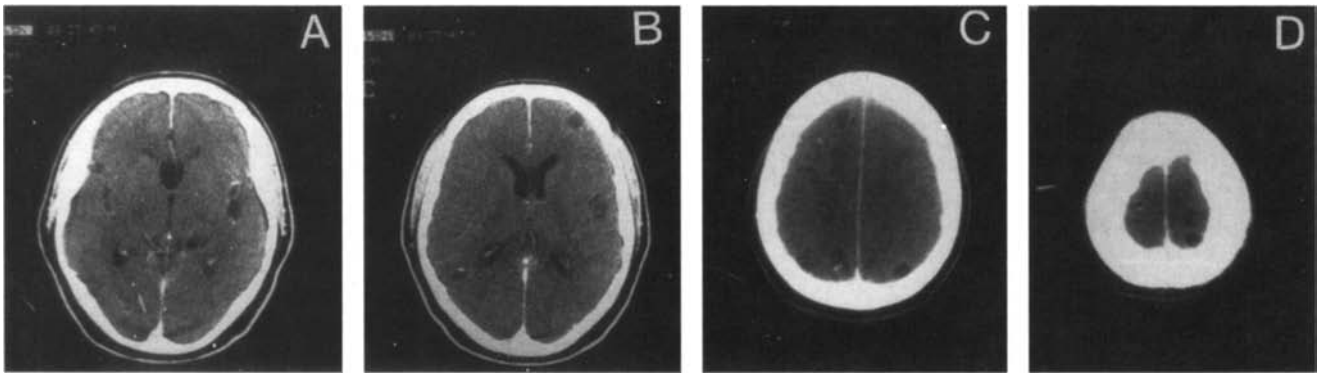


**Figure 6.** Different views of the same CT scan, showing examples of cysts in different stages of evolution. *A,* Active subarachnoid cysts on both Sylvian fissures and an intraventricular cyst at the foramen of Monro. *B,* Left frontal active cyst, right parietal calcification, and active left Sylvian fissure cyst. *C,* Calcified lesions and concurrent active cysts. *D,* Left parietal inflammatory cyst with ring enhancement.

are big, if they are obstructing CSF flow, or if they complicate shunt functioning. In patients with intraventricular cysts without hydrocephalus, or with only slight dilatation of the ventricles, we favor a trial of albendazole therapy and close supervision in case a diversionary procedure is needed.

Some investigators have advocated the use of anthelmintic drugs as well as shunts [1, 6] as a mean of decreasing shunt failures. The use of praziquantel in ventricular disease is controversial, since earlier studies have associated such therapy with a poor outcome [4]. Both failures [4] and successes [1] with praziquantel have been observed in the treatment of intraventricular cysts. Albendazole, a member of the benzimidazole group, may destroy some of the intraventricular cysts as early as 3 weeks after the initiation of therapy, with the cysts totally disappearing within 3–6 months, as seen by neuroimaging [11, 12].

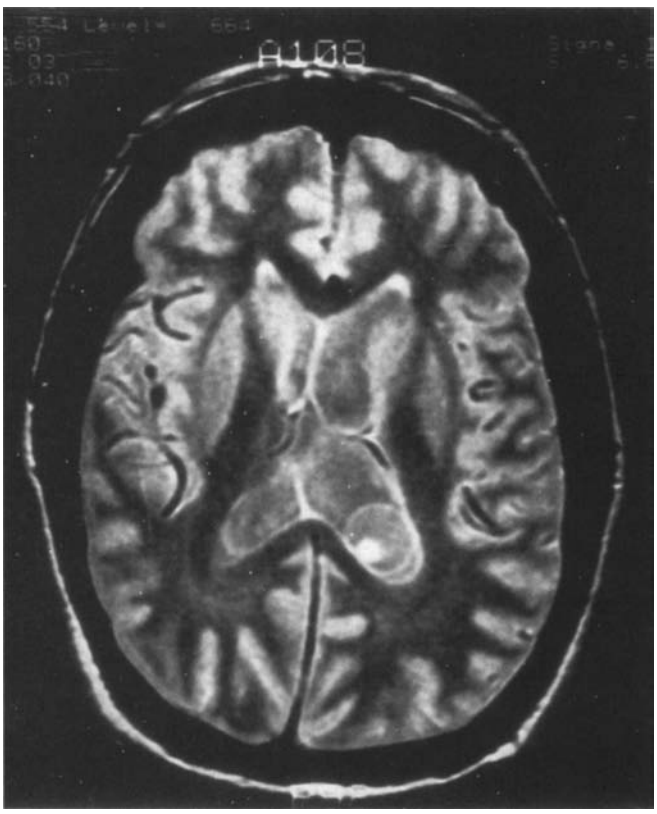

**Figure 7.** Axial proton-density MRI reveals an intraventricular cyst in the occipital horn of the left lateral ventricle of a 32-year-old man who presented with symptoms of increased intracranial pressure and meningeal signs. The scolex is visible in the cyst.

We now use a treatment protocol with albendazole (investigational new drug number 44,491) to treat intraventricular cysts, at a dose of 15 mg/(kg · d) for 15 days. Two courses of medication are given 1 month apart. In our series, all of the intraventricular cysts disappeared after this approach. Certainly, as some authors have mentioned, there is a possibility that this response ascribed to a albendazole could be the reflection of the natural history of the infection.

When the patient is hospitalized with moderate or severe hydrocephalus, a ventriculoperitoneal shunt is placed at the time of admission, in conjunction with albendazole therapy. The most common cause of dysfunction of the ventriculoperitoneal shunt in this series was obstruction of the shunt by either a gelatinous material from cysts or a high CSF protein level. Three of our patients who underwent a shunt procedure for intraventricular cysticercosis required repeated procedures, a fact that may justify surgical removal of the cysts when feasible [1].

Hydrocephalus returned in some patients who had intraventricular cysts, even after the cysts had disappeared. These cases with chronic inflammatory reaction demonstrate that neurocysticercosis is a serious and disabling condition with obscure peculiarities in its natural history. A reinfection can be the cause of this relapse in some cases. However, there may be other causes.

When the cysts die, there is liberation of antigenic substances with local cell reactions that generate an inflammatory response throughout the ventricular system (ventriculitis) [13]. A severe localized reaction fixes the cyst capsule to the ventricular wall or the subarachnoid tissue with strong adhesions and thickening that may produce irreversible blockage of the CSF circulation.

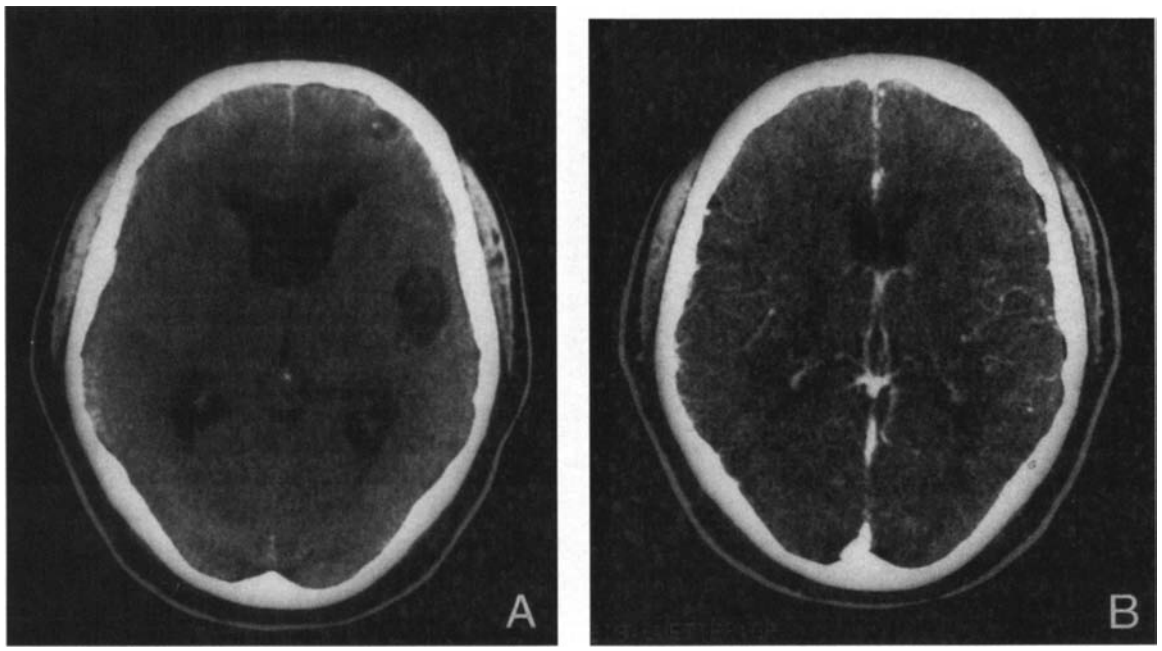


**Figure 8.** *A,* CT scan shows intraventricular and parenchymatous active cysts. *B,* CT scan 11 months later shows resolution of the cyst. The patient was a 46-year-old man who presented with headaches and tonic clonic generalized seizures.

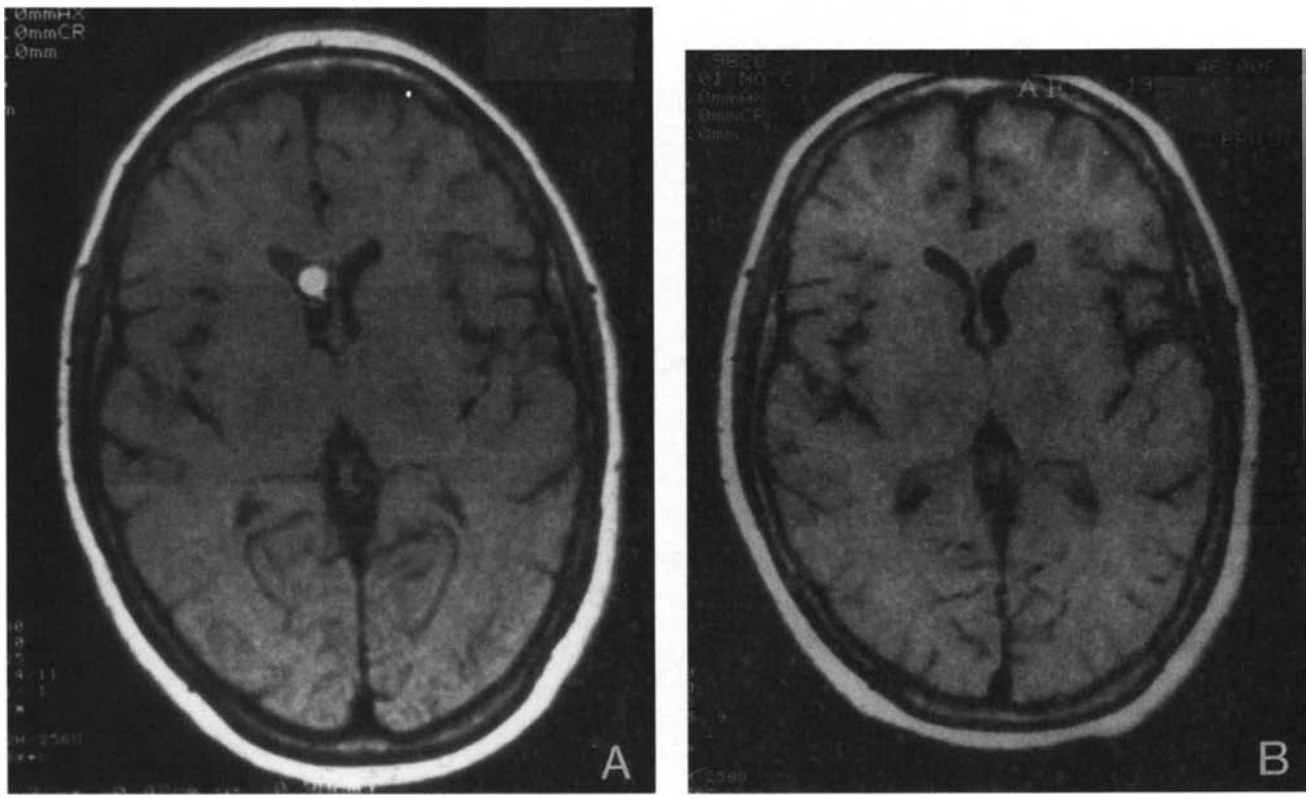


**Figure 9.** *A,* $T_1$-weighted MRI shows an intraventricular cyst in the frontal horn of the right lateral ventricle. *B,* $T_1$-weighted MRI performed 13 months later shows resolution of the intraventricular cyst. The patient was a 53-year-old woman who presented with recurrent tonic clonic generalized seizures.

These changed tissues cannot be surgically removed without damage to the brain tissue [13]. Therefore, the associated hydrocephalus requires a ventriculoperitoneal shunt.

In silent areas of the brain, parenchymatous cysts may go unnoticed during the inflammatory phase, and they eventually calcify. In crucial areas of the brain, parenchymatous inflammatory cysts become symptomatic when the miniature parasite dies, igniting a response from the host, with inflammatory activity and edema [7]. Clinical abnormalities largely result from this host inflammatory response and may include tonic clonic generalized seizures and partial motor seizures [7, 14–16].

In this study, tonic clonic generalized seizures were the most common clinical presentations of parenchymatous neurocysticercosis (64%) (table 2). Most patients who presented with seizures had inflammatory, parenchymatous cysts and significant pericystic edema. This observation is emphasized in other articles [17].

Inflammatory cysts undergo spontaneous resolution into the inactive, calcified stage [18, 19]. However, inflammatory, symptomatic cysts require symptomatic treatment (i.e., with antiepileptic drugs). Patients who present with inflammatory cysts also may have active cysts that may not be seen on neuroimaging. Those active cysts will eventually become inflammatory, and some of them will become symptomatic. This fact provides a rational argument for the use of anthelmintic drugs in patients with inflammatory cysts, in order to accelerate the death of the parasite in the active cysts [4, 10, 16, 20–22].

Subarachnoid cysts in the basilar cisterns can produce hydrocephalus, increased ICP, and meningeal irritation. Subarachnoid cysts are always accompanied by parenchymatous cysts. Recommendations for treatment of subarachnoid cysts include anthelmintic drugs, shunting procedures if there is hydrocephalus, and surgical removal of the cysts [10].

CSF immunodiagnostic methods, particularly immunoblotting, are useful and supportive but have limitations. Some investigators have noted that the EITB assay is highly sensitive in patients with multiple enhancing intracranial lesions but not in those with single or calcified lesions [23]. Antibodies are detectable as frequently in serum as in CSF, regardless of the number or apparent condition of the cysts [23].

Recent data have shown that ELISA does not perform well with this disease and has a high rate of both false-positive and false-negative results [24]. A negative result does not exclude neurocysticercosis; a positive result is not specific for neurocysticercosis, especially in groups with high exposure. Furthermore, they may give false-positive reactions in patients with tuberculosis and parasitic diseases such as echinococcus [25] and *Taenia saginata* infection. The sensitivity of EITB is better than that of ELISA [6]. If ELISA is used alone, it could underestimate seroprevalence [24].

## Conclusions

The data collected here permit us to draw the following conclusions. First, intraventricular neurocysticercosis is a serious and disabling condition with obscure peculiarities in its natural history, grave complications, and a high rate of poor outcomes. Second, we have used albendazole in conjunction with shunt procedures successfully to treat intraventricular cysticercosis. In cases of intraventricular cysts without hydrocephalus or with mild hydrocephalus, and in cases of subarachnoid cysts, several courses of albendazole, close supervision, and follow-up are indicated.

Third, parenchymatous disease, which results largely from the host inflammatory response [7], presents most commonly with epileptic seizures. These cysts disappear spontaneously within 6 months without treatment. Since inflammatory cysts may coexist with active cysts not seen on neuroimaging, it seems reasonable to start treatment with anthelmintic drugs as an attempt to accelerate the death of the parasite in the active cysts. In general, the prognosis for parenchymatous cysts is good [1].

Fourth, neuroimaging is the most important clinical tool for the diagnosis of neurocysticercosis. The finding of cysts in different stages of evolution helps in the diagnosis. The fifth and final point is that sensitivity of serodiagnosis is low in cases with only parenchymatous cysts. For >80% of patients with meningeal irritation and intraventricular cysts, EITB of serum and CSF was positive.

### Acknowledgment

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