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The Acute Encephalitic Phase of Neurocysticercosis: Computed Tomographic Manifestations

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Brain infestation by *Cysticercus cellulosae*, the larval form of *Taenia solium*, is a common disorder in Mexico and other Latin American countries, Asia, Africa, and some Eastern European countries. Recent immigration has caused an increase in the number of cases of neurocysticercosis in the United States. This work describes the acute encephalitic form of neurocysticercosis in 26 cases. The clinicopathologic and neuroradiologic manifestations are discussed with particular emphasis on the use of computed tomography as a main diagnosis modality which demonstrates multiple diffuse (85%) or localized (15%) enhancing nodules associated with severe edema.

Cysticercosis of the central nervous system in Mexico is found in 2.3%–6.3% of all autopsies [1]. From previous clinical reports, cysticercosis has been classified into types according to the areas of involvement: meningobasal (39%), mixed (23%), parenchymal (20%), intraventricular (17%), and spinal (1%) [2–8]. However, it is our impression from computed tomography (CT) that the parenchymal type of cysticercosis is the most common.

The histologic description of the granulomatous response to neurocysticercosis in the acute encephalitic phase, which is more common in children, has been described [1, 9, 10]. The main feature of the complex picture is severe cerebral edema which often leads to severe cerebral damage [11, 12].

Before the advent of CT, patients with suspected neurocysticercosis were subjected to ventriculography, pneumoencephalography, and angiography [9, 13–16], methods that did not disclose all the features of this entity. In most instances, CT is now capable of identifying this process quite early and completely.

Materials and Methods

Twenty-six cases were studied with CT using the EMI CT 1005 unit with the following factors: 120 kV, 33 mA, and 60 sec scanning time with a 160 × 160 matrix. The age range of the patients was 4–66 years with a predominance in the first and second decades (table 1). It was more common in males than in females: 15 men (57.7%) to 11 women (42.3%).

Observations

The initial clinical manifestations in our patients were focal neurologic deficit in 23, seizure disorder in 21, increased intracranial pressure in 16, ophthalmologic deficit in seven, and psychiatric disorders in four. Later in the course of the disease, the symptoms for the most part were related to various degrees of diffuse increased intracranial pressure [9, 17]. Symptoms are more severe and common in children and adolescents. The disease follows a rapid course that may end in death in a high percentage of patients if adequate and appropriate treatment is not given. There seems to be about 10% mortality in the acute phase

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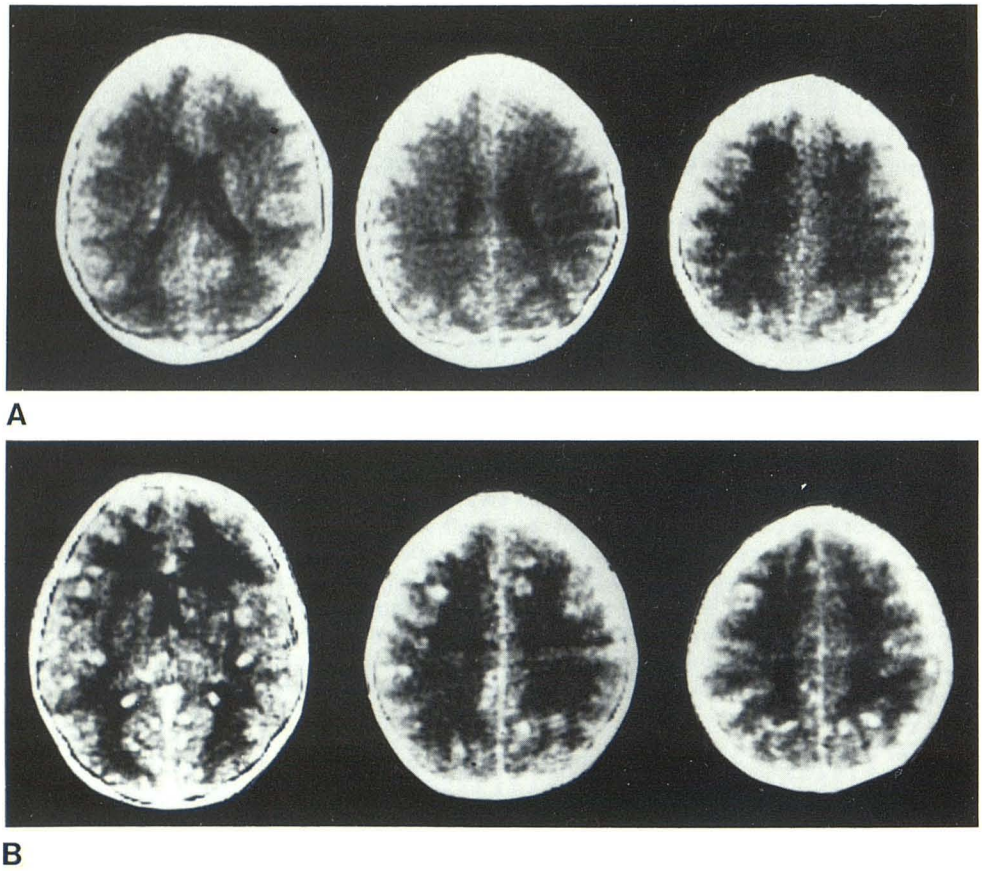


Fig. 1.—10-year-old girl with seizure disorder and severe increased intracranial pressure. **A**, Nonenhanced scan. Diffuse and symmetrical edema in white matter. **B**, Enhanced scan. Multiple small enhancing lesions in both hemispheres.

TABLE 1: **Age of the Patients by Decades**

Age (years)	No. Patients	(%)
0–10	7	(27.0)
11–20	10	(38.5)
21–30	4	(15.4)
31–40	2	(7.7)
41–50	1	(3.8)
51–60	1	(3.8)
61–70	1	(3.8)
Total	26	(100.0)

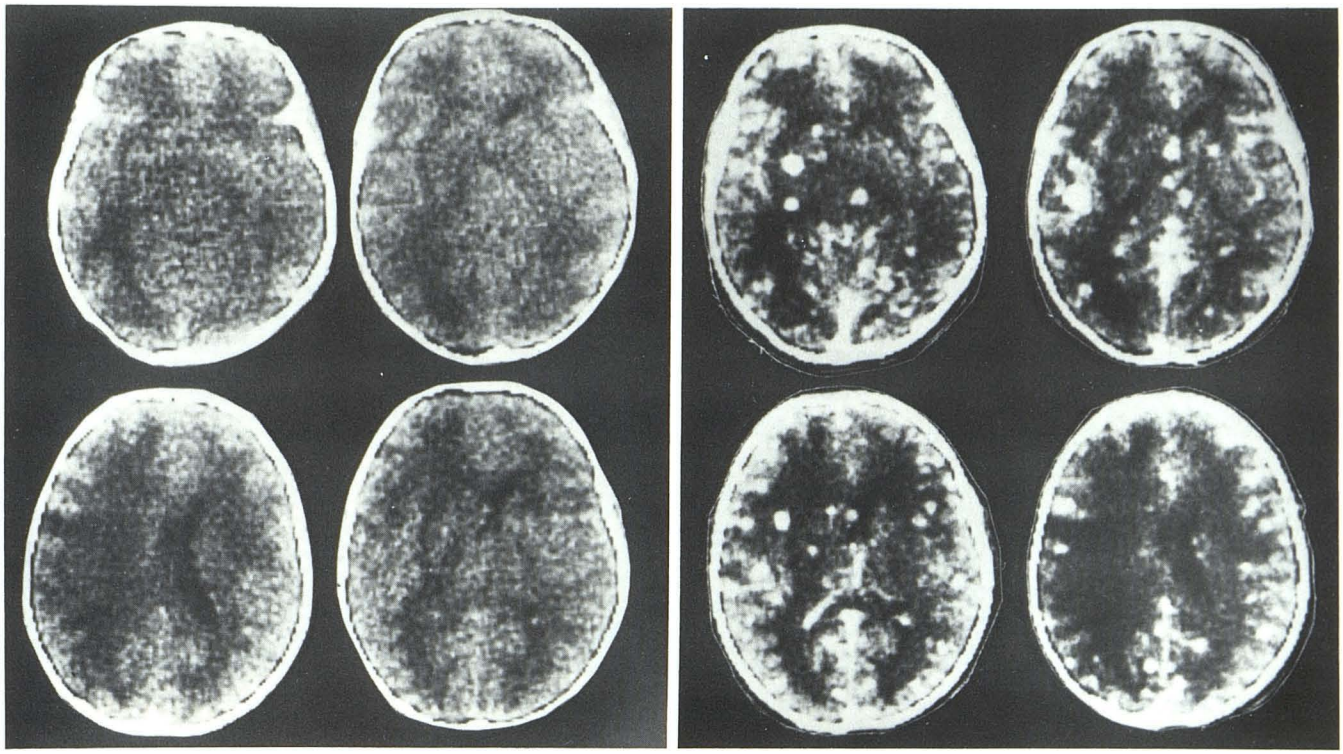
despite appropriate treatment.

The most important cerebrospinal fluid findings were pleocytosis with eosinophilia, increase in protein concentration, decrease in glucose, and positive spinal fluid complement fixation test using Nieto's technique [18].

Computed tomography has allowed the detection of the encephalitic phase of parenchymal cysticercosis, which in our present experience represents 65% of all intracranial cysticercosis. Computed tomography with infusion of iodinated contrast medium demonstrated enhancement of the lesions in 100% of the cases; they were manifested by dense nodular and anular images. This abnormal enhancement persisted throughout the acute stage, and it varied in intensity and duration with each individual. The lesions disappeared progressively along with the clinical improvement. Even when the lesions disappear, CT demonstrates that the edema may persist for several months, even though the patient may be asymptomatic.

Two forms of cerebral cysticercosis in the acute encephalitic phase have been observed: multiple diffuse nodular lesions (85%) (figs. 1 and 2) and localized lesions (15%) (fig. 3). In both types, the lesions vary in size and are commonly located in the cerebral cortex. The lesions are almost invariably associated with severe edema. On the CT scan without intravenous infusion of iodinated contrast material, the edema appeared as low-density areas with irregular contours. These usually were located in the periventricular white matter and in the centrum semiovale, although similar changes were found to a lesser degree in the cortical gray matter and in the basal ganglia.

Of our 26 cases, three could not be followed as they did not return for further examinations; three others died during their hospitalization. The cause of death was related to herniation of the cerebellar tonsils due to severe increased intracranial pressure. A biopsy of one of these cases during a subtemporal decompressive craniectomy revealed granulomatous response to cysticercosis and diffuse meningeal involvement as was demonstrated on CT (fig. 4). In an autopsied case there was diffuse edema and multiple *Cysticercus* cysts with surrounding inflammatory infiltrates. There was also increased vascularity and capillary endothelial proliferation adjacent to the cysts (figs. 5 and 6), which



A B

Fig. 2.—12-year-old boy with left hemiparesis, decreased visual acuity, and signs of severe increased intracranial pressure. **A**, Nonenhanced scan. Diffuse edema, more severe in right hemisphere, with past effect on lateral ventricles. **B**, Enhanced scan. Multiple enhancing lesions in both hemispheres.

could well explain the computed tomographic findings.

The acute encephalitic process resolves differently with each individual. We have observed that in general it lasts 2–6 months and its evolution depends on three main factors: number of lesions present, general health and condition of the patient, and immunologic factors that are not yet well understood.

In two children, skull radiographs demonstrated typical signs of increased intracranial pressure. Carotid angiography performed in seven patients demonstrated irregularities of the lumen of the arteries due to inflammatory changes in the vessel walls. These changes, however, were considered to be nonspecific because they were similar to those seen in other types of vasculitis. There was no demonstrated localized mass effect.

As there is no specific medical treatment for cysticercosis, the only alternative is a symptomatic treatment for the convulsive crisis and cerebral edema (glycerol, acetoazolamide, and steroids) [19–22]. Our patients were treated with dexamethasone (4 mg, three times daily). In the 20 cases that we were able to follow, the acute edema disappeared in 3–6 weeks. There were three deaths despite treatment.

Of the patients who survived the acute encephalitic phase of the disease, 90% were followed clinically and with CT at various intervals up to 3 months, revealing disappearance of the cerebral edema and a return of the ventricular system to normal. The disappearance of the edema correlated well with the clinical improvement. At the end of the 3 month period, the enhancing ringlike lesions were no longer demonstrated in the postcontrast studies.

The CT follow-up on 20 patients performed at intervals up to a year or so after the onset of the illness demonstrated small calcifications in 80%, which can now be detected as early as 8 months after the acute stage (fig. 7). Only 30% of those calcifications were detectable on plain films of the skull. Four of those patients developed slight hydrocephalus as the only demonstrable sequela on CT.

Discussion

The clinicoradiologic manifestations of the encephalitic phase of cysticercosis correlate well with the pathologic findings. Histologically, the granulomatous response of cerebral tissue to the parasite varies according to the different biologic stages of the *Cysticercus*: In the vesicular stage, the inflammatory reaction is well localized to the adjacent nervous tissue and there is only a small ring of edema and glial proliferation. At the end of this stage, when the edema and glial proliferation have increased and a connective tissue capsule begins to form, the capillary network around the parasite begins to proliferate. These phenomena are highly similar to those seen in other types of granulomatous reaction. As the parasite ages, a local immunologic reaction

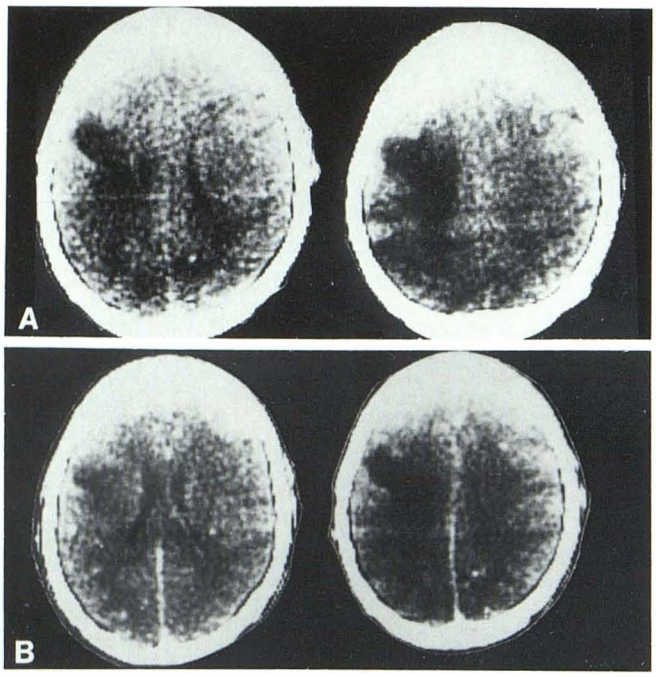


Fig. 3.—18-year-old woman with seizure disorders and left hemiparesis. **A**, Nonenhanced scan. Localized edema in right parietal area. **B**, Enhanced scan. Several enhanced lesions of different sizes mainly localized to right parietal area.

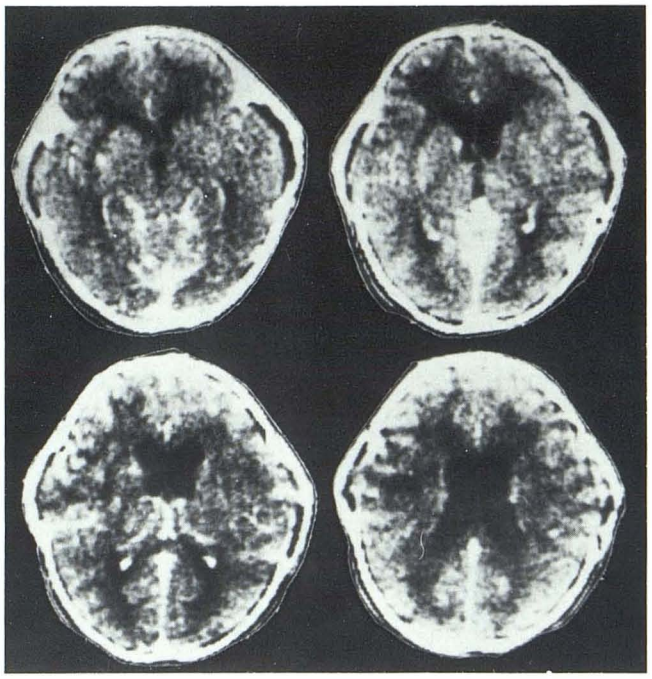


Fig. 4.—15-year-old girl after subtemporal craniectomies for intractable increased intracranial pressure. Enhanced scan. Moderate degree of symmetrical ventricular dilatation, diffuse edema, enhancing leptomeninges, and a few enhancing lesions.

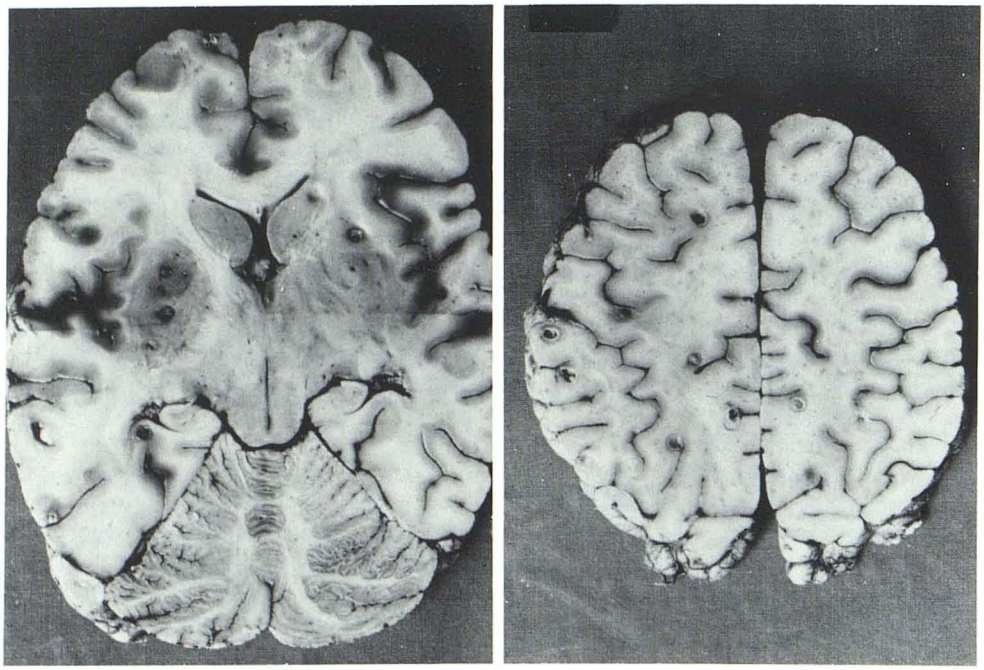


Fig. 5.—13-year-old girl. Diffuse symmetrical edema, compression of ventricular system and multiple small cysticerci. Patient died from severe increased intracranial pressure. **A**, Section at level of ventricular system and basal ganglia. **B**, Section at level of centrum semiovale.

is produced and the inflammatory response begins to change; when the colloidal stage of the vesicular form develops, the inflammatory reaction reaches its peak and from then on there is a steady decline to the granular and nodular calcified stages [9].

Occasionally other intracranial pathologic processes may present CT images similar to this form of cysticercosis, such as tuberculosis when manifested by multiple small tuberculomas, nocardiosis among the mycoses, and with less frequency, multiple small abscesses and metastic processes.

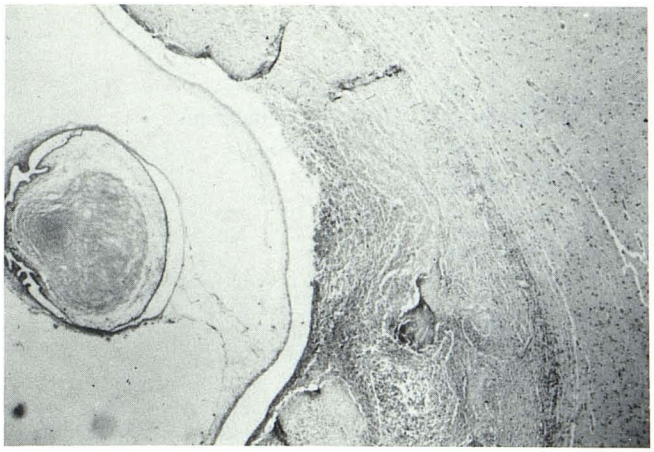


Fig. 6.—Parenchymatous cysticercus. Parasite appears at left; part of rostellum is surrounded by membranous structure that continues with main part of cyst proper joined by thin strand of membrane. Adjacent to cyst, there is strong inflammatory reaction diffusely spread throughout dense connective tissue capsule, but mainly in its outer layers. Nervous parenchyma (far right) shows diffuse gliosis and myelin pallor due to edema. H and E, ×250. (Courtesy of Dr. A. Escobar, Mexico.)

These entities should be kept in mind in the different diagnosis.

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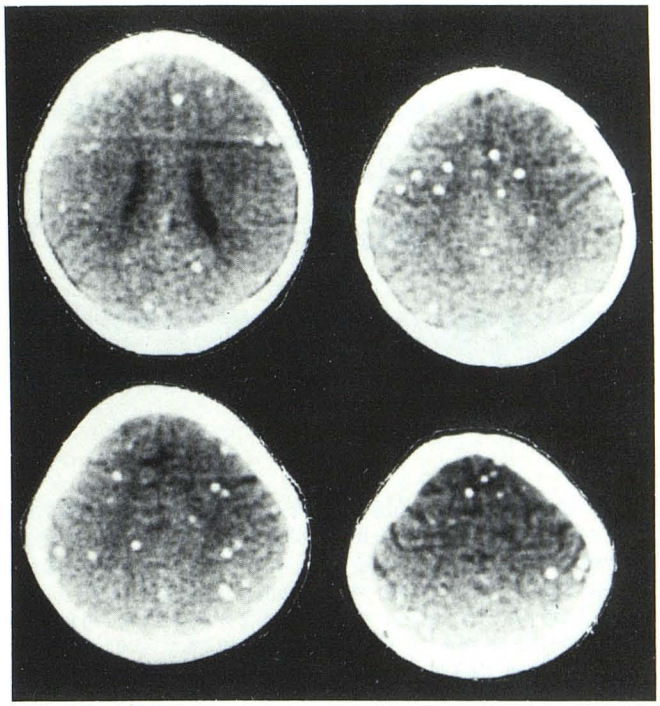

Fig. 7.—14-year-old asymptomatic boy, 2 years after encephalitic phase of neurocysticercosis. Nonenhanced scan. Multiple small intraparenchymal calcifications.

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