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MR Imaging of Neurocysticercosis

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Twenty-six patients with neurocysticercosis were studied with MR imaging to correlate their clinical presentation with the location and appearance of their neurocysticercosis lesions. Intraventricular cysts were present in 14 patients (54%), parenchymal cysts were present in 18 (69%), and intraventricular together with parenchymal cysts were present in six (23%). Intraventricular cysts were detected by mass effect, ventricular obstruction, detection of a cyst rim, and/or CSF flow void adjacent to the cyst. The intensity of most intraventricular and parenchymal cysts presumed to be viable was similar to that of CSF on both T1- and T2-weighted sequences. Cysts presumed to be degenerated had increased signal intensity on T1-weighted images, probably resulting from increased protein content. Pericystic high signal intensity surrounding lesions of various ages was seen on both proton-density- and T2-weighted images and represents gliosis, edema, and inflammation. Patients with parenchymal cysts had symptoms of seizures, while those with intraventricular cysts generally had symptoms related to obstructive hydrocephalus. Aqueductal stenosis, seen in 10 patients (38%), was possibly due to ependymal inflammation or adhesions caused by prior ventricular infection by neurocysticercosis. One patient with the racemose form of neurocysticercosis demonstrated abundant cyst wall proliferation resulting in obstructive hydrocephalus. In six patients scanned 1-6 months after oral praziquantel therapy, there was no change in the MR appearance of intraventricular cysts, while some parenchymal cysts showed evidence of degeneration.

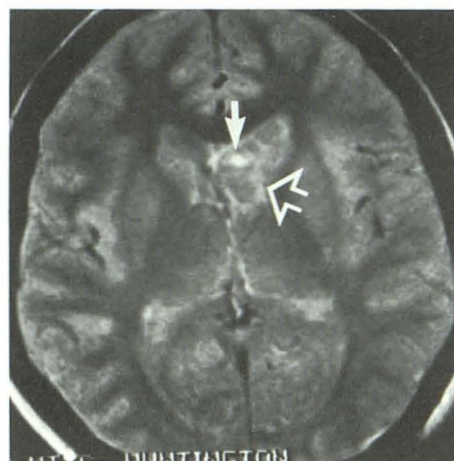
We found MR to be useful in detecting the cysts of neurocysticercosis and the accompanying signs of cyst degeneration and pericystic inflammation. MR was inferior to CT in the detection of parenchymal calcifications.

Neurocysticercosis is an infection of the CNS by the larval stage of the pork tapeworm *Taenia solium*. The disease is prevalent in developing nations, particularly in regions of poor sanitation and hygiene. It is the most common cause of seizures in young adults in endemic areas [1]. With immigration of individuals from endemic areas, cysticercosis is becoming increasingly prevalent in the United States [1].

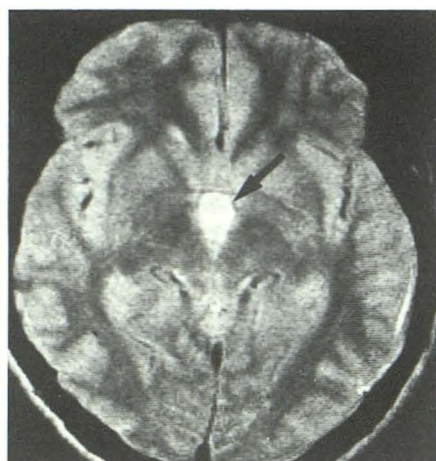
The diagnosis of neurocysticercosis often depends on a combination of clinical symptoms, CSF analysis, and radiologic findings [2]. CSF antibody or antigen titers are useful, but the findings can be nonspecific [2-4]. Radiologic findings are the most sensitive [2]. CT is beneficial in identifying parenchymal calcifications associated with neurocysticercosis; however, intraventricular cysts often are difficult to identify with CT owing to the similarity of densities between cyst fluid and CSF. MR imaging, with its multiplanar imaging capabilities, excellent depiction of tissue contrast, and sensitivity to flow effects, is a powerful technique for the evaluation of neurocysticercosis, particularly in the presence of intraventricular cysts. The present study analyzes the location and MR appearance of neurocysticercosis lesions in 26 patients and correlates these findings with clinical presentation.

TABLE 1: Summary of Findings in Patients with Neurocysticercosis

Variable	Type of Cyst			Total
	Parenchymal	Intraventricular	Mixed Parenchymal/ Intraventricular	
Cyst characteristics				
Total no.	12	9	26	47
Locations of intraventricular				
Third ventricle	–	3	3	6
Fourth ventricle	–	2	2	4
Lateral ventricle	–	3	1	4
Subarachnoid cistern	–	1	4	5
MR intensities				
Isointense relative to CSF	2	7	14/10 ^a	33
Hyperintense relative to CSF	10	2	2/0 ^a	14
Patient characteristics				
Total no.	12	8	6	26
Presenting symptoms				
Seizures	10	0	3	13
Nausea/vomiting and/or headache	2	8	3	13
Patients with aqueductal stenosis	1	6	3	10
Means of diagnosis				
CSF analysis	8	6	4	18
Surgery	4	2	2	8

^a Parenchymal/intraventricular.

1



2

Fig. 1.—Axial MR image (SE 2000/60) through lateral ventricles. Cysticercosis cyst in anterior horn of left lateral ventricle displays rim of hyperintensity (open arrow) caused by ependymal inflammation. Scolex (solid arrow) is well defined as focus of hyperintensity within cyst.

Fig. 2.—Axial section (SE 2000/60) through level of third ventricle. A 1-cm cyst within third ventricle (arrow) displays prominent hyperintensity suggestive of cyst degeneration. Scolex cannot be identified.

Materials and Methods

Between May 1983 and July 1988, 26 patients, 18 males and eight females 7–69 years old, with a diagnosis of neurocysticercosis underwent cranial MR imaging. The clinical records of these patients were reviewed retrospectively to determine the presenting symptoms; that is, seizures vs signs of hydrocephalus (e.g., headache, ataxia, and nausea and vomiting). Diagnoses of neurocysticercosis were corroborated by findings at surgery and/or CSF analysis. All MR studies were performed on a 0.35-T,* 0.5-T† or 1.5-T‡ superconducting MR unit. All patients were initially scanned in the axial plane with dual-echo spin-echo (SE) technique, 2000–3000/30–100/2–4 (TR range/TE range/excitations). Sagittal and coronal SE images (1000/30–80/

2) were obtained in most cases to further characterize lesions observed during the initial axial sequences. Slice thicknesses of either 5 or 10 mm were used. The matrix size was either 128 × 256 or 256 × 256. Each MR study was evaluated for the site(s), number, and signal pattern of intracranial cysts. Cysts within the subarachnoid cisterns were included within the intraventricular group. The cyst rim was assessed for thickness, the presence and extent of pericystic high signal intensity, and alterations in ependymal signal intensity.

The ventricular system was evaluated for evidence of obstruction manifested by general ventricular dilatation (out of proportion with sulcal enlargement), third ventricular enlargement, temporal horn enlargement, and periventricular high signal (on T2-weighted images) suggestive of transependymal CSF flow. Aqueductal stenosis, as evidenced by a diminutive area of aqueductal low signal, was evaluated on both sagittal and axial MR images.

MR results were correlated with brain CT scans and pathologic findings when available. Correlation was made between the clinical presentation of the patients and the MR pattern of neurocysticercosis.

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Six patients received oral praziquantel therapy at a dosage of 60 mg/kg/day for up to 2 weeks and underwent a repeat MR examination 6 weeks to 6 months after the completion of therapy. MR examinations were evaluated for changes in the number and appearance of cysts.

Results

The MR findings of the 26 patients are summarized in Table 1.

Intraventricular Cysts

Fourteen (54%) of the 26 patients in our series had intraventricular cysts. In eight (31%) of the 26 patients, intraventricular cysts were present without parenchymal cysts. In each of these eight (and in a total of 11 of the 14 patients with intraventricular cysts), there was a clinical history of nausea and vomiting or headaches or both. The symptoms were believed to be related to elevated intracranial pressure caused by the cysts. Eight patients required the placement of a ventriculoperitoneal shunt for varying degrees of hydrocephalus. Two patients each had cysts in the fourth, third, and lateral ventricles, while one patient had a cyst in both the third and lateral ventricles. The intraventricular cysts were typically 1–2 cm in diameter. Eleven (79%) of the 14 patients had evidence of a pericystic ependymal inflammatory reaction,

as manifested by a hyperintense rim best seen on T2-weighted images (Fig. 1). The cysts generally had a signal intensity similar to that of CSF on T2-weighted images. Mass effect, visualization of the cyst rim, and CSF flow void adjacent to these cysts aided in their identification. Two of the intraventricular cysts had fluid that on T2-weighted images was hyperintense relative to CSF (Fig. 2).

Six (23%) of the 26 patients had both intraventricular and parenchymal cysts. Three of these six patients presented with nausea, vomiting, and headaches. Four of these six patients had multiple parenchymal cysts; one patient had a total of five. One patient had an unusually large fourth ventricular cyst and three identifiable parenchymal cysts (Fig. 3). Although three of the six patients presented with seizures, cyst degeneration was thought to be present in only two of the three. Prominent pericystic ependymal reaction was seen in two of the three seizure patients.

Parenchymal Cysts

Eighteen (69%) of 26 patients presented with parenchymal cysts that involved the gray/white-matter junction in all but one case. Twelve (46%) of the 26 patients had MR evidence of parenchymal cysts only. Ten patients (83%) in this subgroup of 12 patients had a history of recent seizures. All of these 10 patients had MR findings of pericystic high signal intensity suggesting gliosis and/or edema (Fig. 4). In one

Fig. 3.—Sagittal sections (SE 1000/40) in patient with large fourth ventricular cyst.

A, Scolex (straight solid arrow) is well demonstrated. Dilated cerebral aqueduct, with central flow void (curved arrow), probably is due to to-and-fro CSF flow caused by obstructive effects of subjacent fourth ventricular cyst (open arrow).

B, Section 5.5 mm lateral to A shows superiorly located 1-cm parenchymal cyst with central scolex (arrow). Signal intensity of cyst fluid is similar to that of CSF on this sagittal image. No pericystic edema is present, suggesting cyst viability.

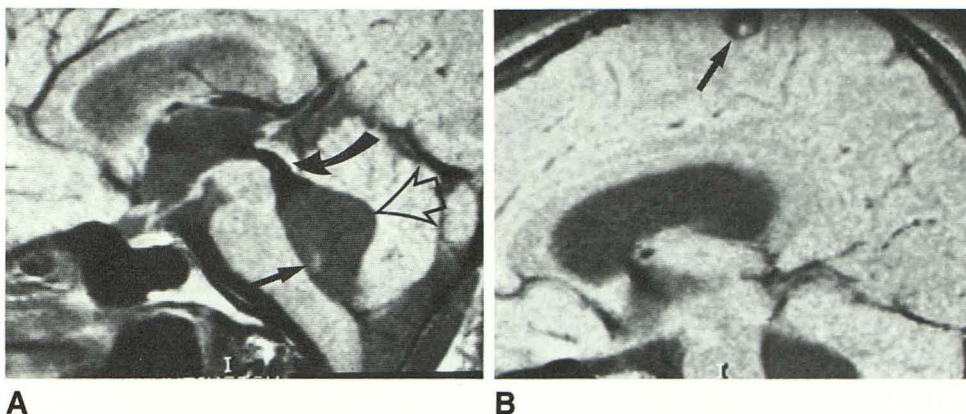
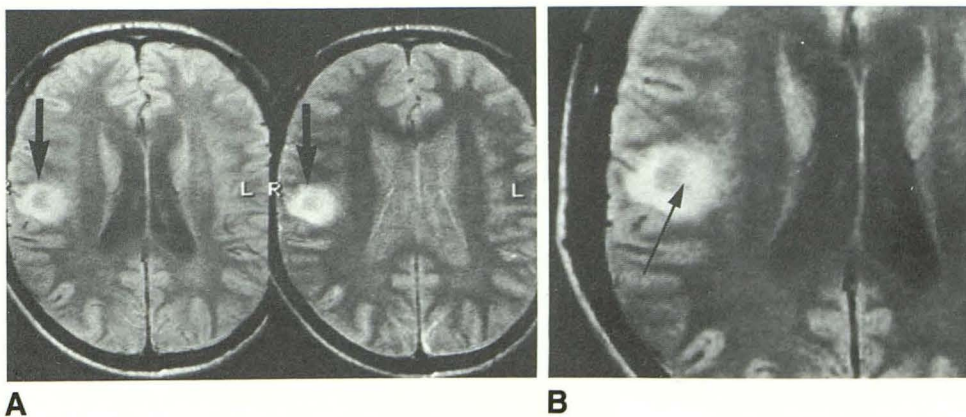


Fig. 4.—Patient with recent onset of seizures.

A and B, Axial MR images, SE 2000/30 (left) and SE 2000/60 (right), show right parietal cystic lesion with pericystic rim of hyperintensity (arrows) suggestive of inflammatory reaction with gliosis and/or edema. Cyst fluid is isointense relative to CSF.

B, Magnified view of first-echo image. What appears to be scolex can be faintly visualized along medial cystic wall (arrow).



case, the extensive pericystic inflammatory reaction simulated a glioma (Fig. 5). Pathologic examination of the surgical specimen from this lesion and another cyst with extensive pericystic high-signal change revealed surrounding gliosis (Fig. 6). One (8%) of these 12 patients with parenchymal cysts presented with only headaches, and another (8%) had unrelated clinical findings in which the neurocysticercosis cyst was found incidentally. One patient in this group who presented with headaches and ataxia was found to have an unusually large 4-cm cyst in the cerebellum that was surgically resected (Fig. 6). Typically, the cysts in these 12 patients were 1–2 cm in diameter. A central or eccentric high-signal focus, suggestive of a scolex, could be identified in only six cysts. Pathologic correlation was available in two patients with this appearance.

In both cases, a scolex was identified (Fig. 7). In one of these cases, the organism appeared viable. However, there was a significant amount of pericystic high signal indicating an inflammatory reaction (Fig. 7).

Aqueductal Stenosis

In 10 (38%) of 26 patients (nine of 14 patients with intraventricular cysts and one of 12 patients with parenchymal cysts only) cerebral aqueductal stenosis was identified that was best appreciated on sagittal images (Fig. 8) as an absence of the normal aqueductal CSF flow void on midline slices. This finding may be associated with ependymal inflammation (granular ependymitis with subependymal gliosis) or

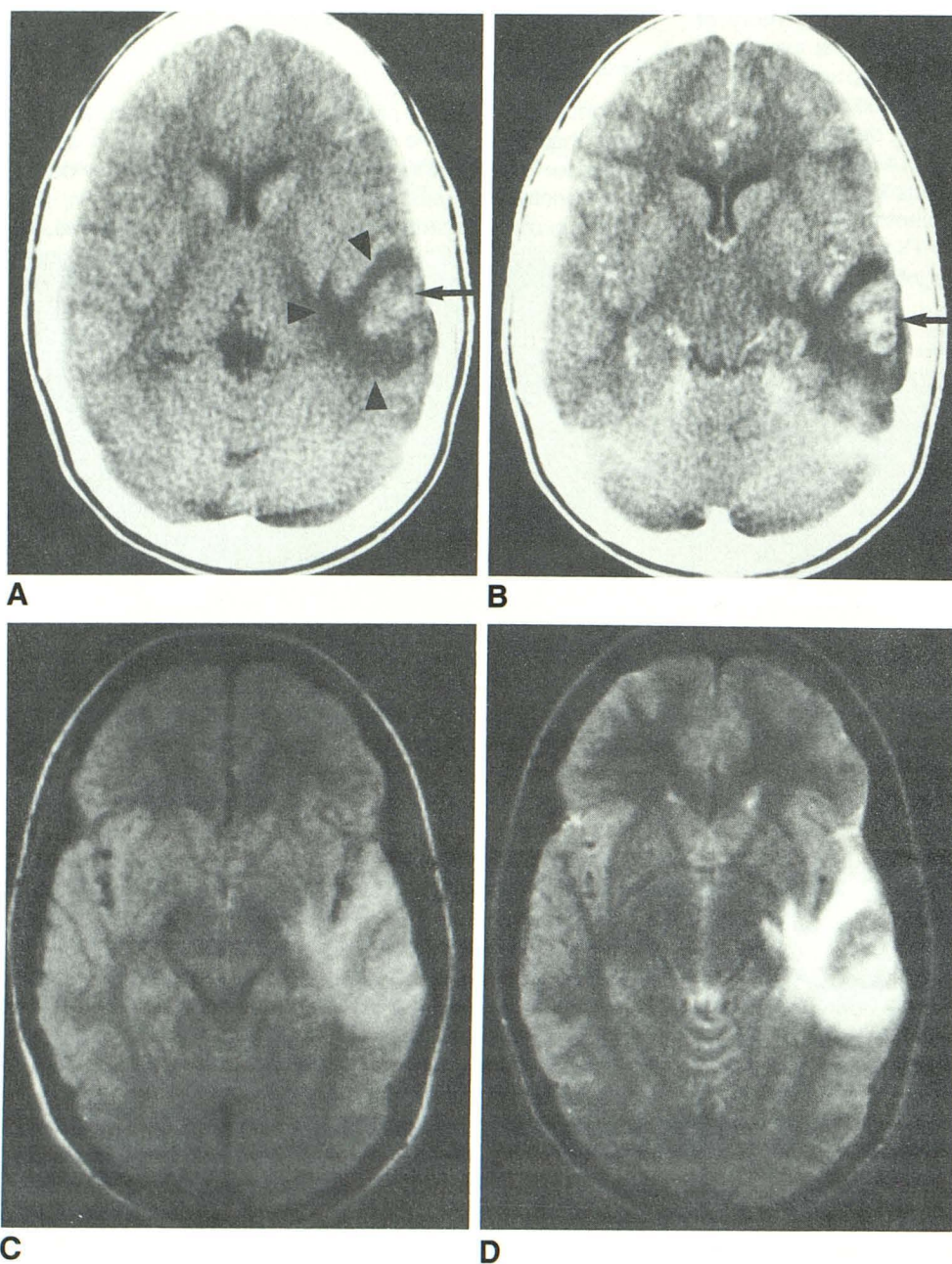


Fig. 5.—CT and MR findings in patient with seizures.

A, Nonenhanced brain CT scan shows left posterior temporal mass (arrow) with surrounding area of low attenuation (arrowheads) consistent with parenchymal edema.

B, Contrast-enhanced CT scan shows enhancement within mass (arrow).

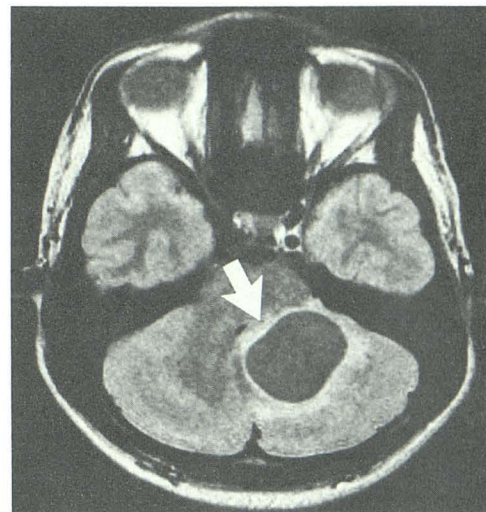
C, First-echo image of axial MR brain study (SE 2450/30) shows large area of increased signal intensity surrounding this same lesion representing edema.

D, Edematous changes appear more intense on second-echo image (SE 2450/100). Preoperative diagnosis was glioma. However, at surgery, a degenerated cysticercosis cyst was found with extensive edema and gliosis surrounding the lesion.

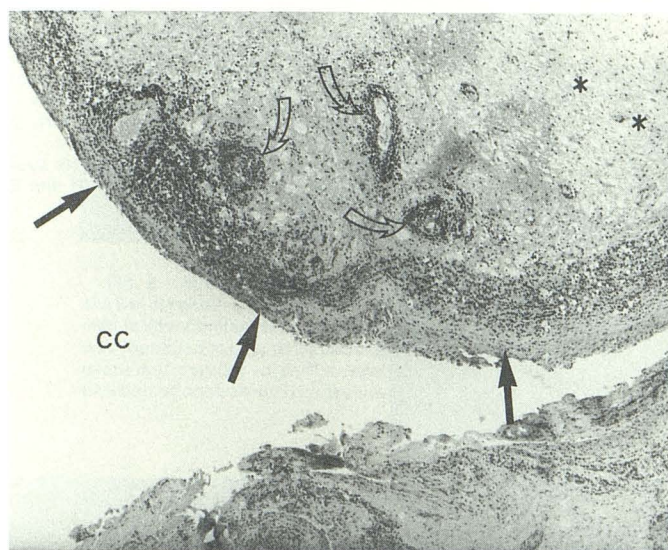
Fig. 6.—A, Axial section (SE 2000/40) shows atypically large 4-cm cerebellar parenchymal cyst. Note pericystic rim of hyperintensity (arrow), indicating inflammatory reaction. Cyst was removed at surgery.

B, Histopathologic specimen from excised tissue shows cyst cavity (CC), host-derived cyst wall and adjacent inflammation (solid arrows), perivascular inflammation (open arrows), as well as inflammatory reaction with gliosis and edema in surrounding cerebellar tissue (asterisks). (H and E, $\times 68$)

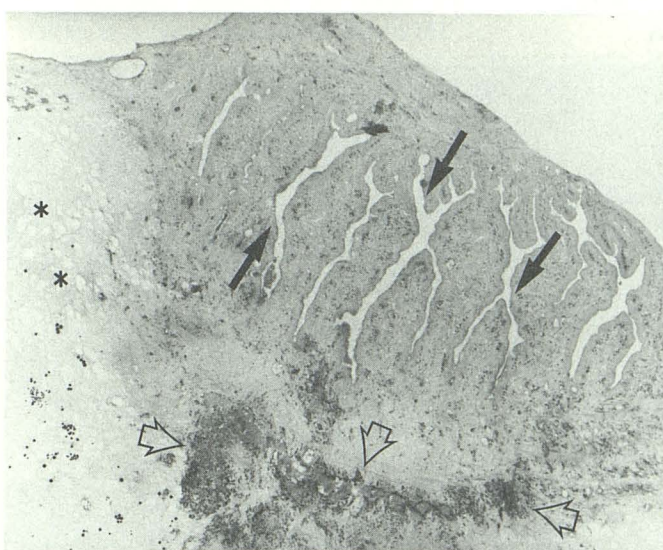
C, Nonviable larva found in same surgical specimen. Note poorly defined internal chitinous wall within parasite (solid arrows). Also seen are adjacent inflammatory infiltrate (open arrows) and cerebellar gliosis and edema (asterisks). (H and E, $\times 134$)



A



B



C

adhesions due to prior ventricular infestation. Seven of the 10 aqueductal stenosis patients were judged to have some degree of hydrocephalus.

One of these 10 patients presented with abundant cyst wall proliferation within the quadrigeminal plate cistern, associated with the racemose form of neurocysticercosis, which resulted in obstructive hydrocephalus and required shunting. The proliferative changes in the cyst wall were well demonstrated on both T2-weighted axial and coronal images (Fig. 9). The cyst fluid intensity was similar to that of CSF on both T1- and T2-weighted images. This patient also had a history of presumed bacterial meningitis that was unresponsive to therapy.

CT Correlation

CT brain scans were obtained (at comparable times) in 10 (38%) of the 26 patients. Seven (70%) of the 10 CT examinations demonstrated parenchymal calcifications (Fig. 10A). MR was less sensitive than CT in demonstrating the size and

number of calcifications. In only three (43%) of these seven MR studies was there evidence of calcifications (small signal-void areas) (Fig. 10B). In seven (70%) of 10, CT demonstrated ventriculomegaly consistent with hydrocephalus; however, CT could not define the cause of hydrocephalus. MR was able to identify the cause of hydrocephalus in six of these seven patients (usually an intraventricular cyst) (Fig. 11). MR was more sensitive than CT in showing the extent of pericystic gliotic/edematous changes (Fig. 7).

Changes After Praziquantel Therapy

Among the seven parenchymal cysts in six patients, there was evidence of degeneration, manifested by increased signal on T1-weighted images, in only three cysts (in two patients) after praziquantel therapy (Fig. 12). One of the seven parenchymal cysts regressed and was no longer visible after therapy. All six patients had intraventricular cysts also. In one of these patients, an intraventricular cyst was removed sur-

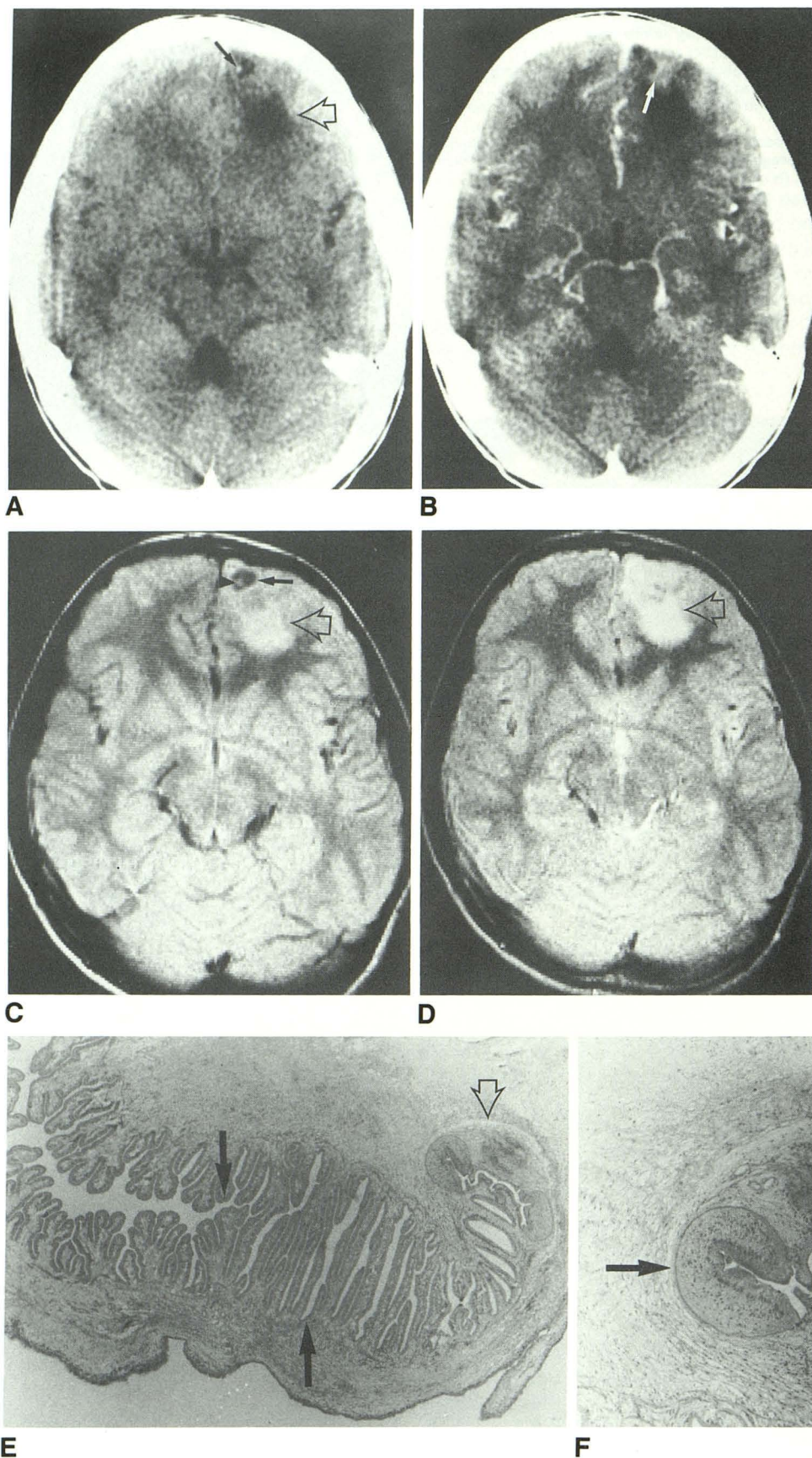


Fig. 7.—A, Nonenhanced axial CT image shows left frontal cysticercosis lesion in patient with seizures. Central calcification (solid arrow) is present within cyst, and there is a small adjacent area of decreased attenuation consistent with parenchymal edema (open arrow).

B, After administration of IV contrast material, CT shows only small region of pericystic enhancement (arrow).

C and D, Proton-density-weighted, SE 2500/30 (C), and T2-weighted, SE 2500/80 (D), axial MR images show left frontal pericystic inflammatory reaction—high-signal-intensity area (open arrows)—with better definition than on CT. These changes were more pronounced on second-echo image (D). Cyst, with fluid isointense relative to CSF (solid arrow), and central scolex (arrowhead) are better seen on first-echo image (C). Calcification was not visible on MR images. Lesion was surgically removed.

E, Histopathologic specimen from excised tissue shows invaginated scolex with well-defined, highly convoluted, and internalized cell membrane (chitinous wall) (solid arrows). This extends to muscular suckers at right of photograph (open arrow). (H and E, $\times 180$)

F, Magnified view of E depicts three muscular suckers (arrows). (H and E, $\times 500$)

gically. No signal or morphologic changes were noted in intraventricular cysts after praziquantel therapy.

Discussion

When the embryonated ova of *T. solium* are ingested, usually as a result of fecal contamination of food or water or autoinfection in tapeworm carriers, gastric acid releases the hexacanth larvae from their encasement, thus creating the

intermediate host state. Once the hexacanth larvae penetrate the intestinal mucosa, they are transported via the lymphatics or venules to the systemic circulation. The blood-borne organisms have a predilection for localization in skin and muscle, where they are asymptomatic, and in the CNS (particularly the cerebral gray matter, periventricular tissues, and leptomeninges), where they may present with a variety of clinical manifestations. The hexacanth larval cells proliferate, eventually producing cysts, typically 1–2 cm in diameter. A small invagination develops along one margin of the cyst and proliferates and differentiates to become the scolex. Cyst fluid is usually clear. Cloudiness of the cyst fluid usually indicates death of the parasite. With death of the parasite, there is resultant cyst degeneration and a striking host inflammatory cell and gliotic reaction. It is probable that during the life span of the cysticercus, there is active suppression of host defenses by factors secreted by the parasite; as the organism loses viability, host immune and inflammatory responses are brought into play, thereby accounting for the marked brain edema about degenerating cysticerci [5].

The diagnosis of neurocysticercosis is confirmed by clinical history, serology and CSF analysis, CT and MR findings, and occasionally, surgical findings. A history of travel to or immigration from an endemic area assists in making the diagnosis. Serology and CSF analysis can be helpful in the case of intraventricular infestation [3, 4, 6]. The CT identification of multiple focal brain parenchymal calcifications [7] and the demonstration of the actual cyst (and the scolex within it) with CT and/or MR [2] are particularly valuable in arriving at a diagnosis. However, in some cases (Fig. 5), the CT and MR findings may still be inconclusive, necessitating surgical resection and histopathologic diagnosis. CT and MR can also

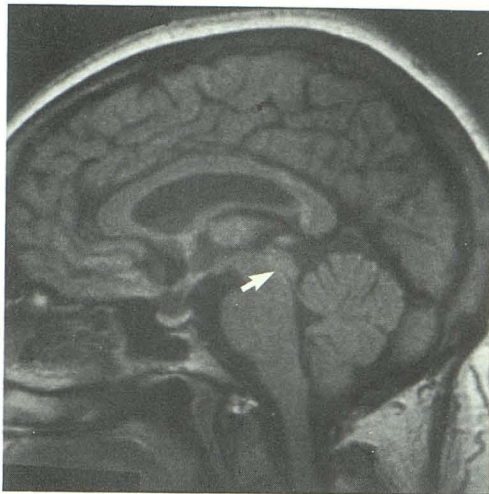


Fig. 8.—Midline sagittal 5-mm section (SE 1000/40) shows cerebral aqueduct stenosis (arrow) in patient with intraventricular infestation. (Aqueductal narrowing was identified in nine of 14 patients with intraventricular cysts and may be related to ependymal inflammatory reaction or adhesions from previous infestation.)

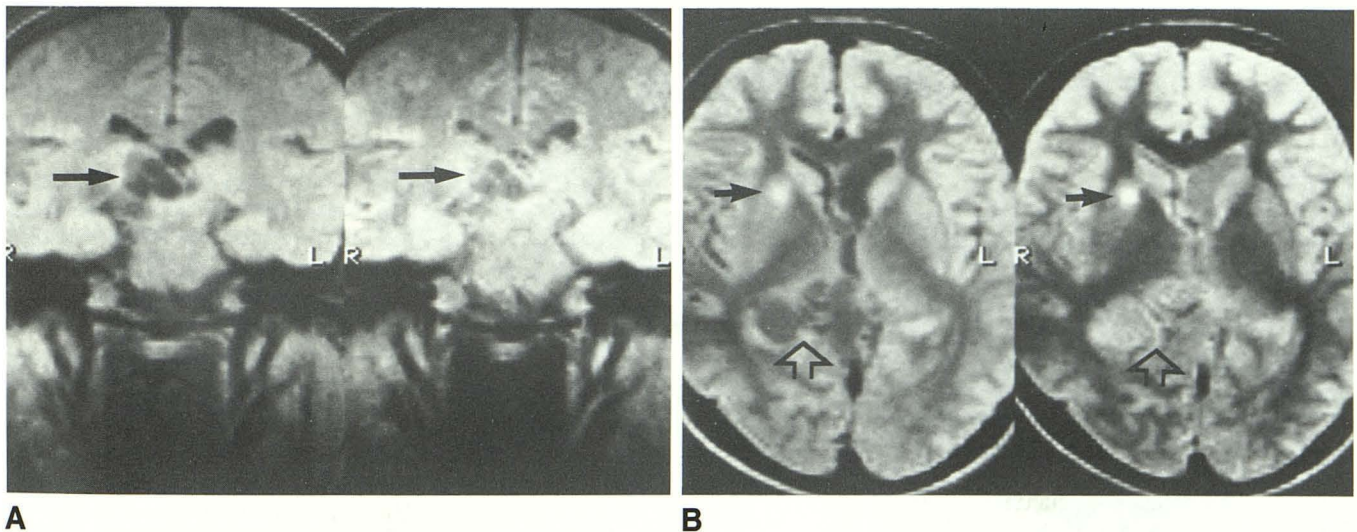


Fig. 9.—A and B, Coronal, SE 1000/28 (left) and SE 1000/56 (right) (A), and axial, SE 2000/28 (left) and SE 2000/56 (right) (B), sections in patient with racemose form of cysticercosis. Multiple cysts are present in ambient cistern on coronal images (A, arrows). Cysts were observed extending from quadrigeminal plate cistern into right retropulvinar cistern (open arrows) on axial images (B). Axial images also demonstrated an additional hyperintense lesion within right globus pallidus (solid arrows). On both echoes, during coronal and axial imaging, cysts remained isointense relative to CSF and produced little evidence of ependymal reaction. Racemose form of neurocysticercosis is believed to be a proliferative reaction of cyst in which no scolex is formed, but cyst infiltrates subarachnoid spaces irregularly and conforms to their peculiar configurations in various locations. The patient received a ventriculoperitoneal shunt to relieve obstructive hydrocephalus.

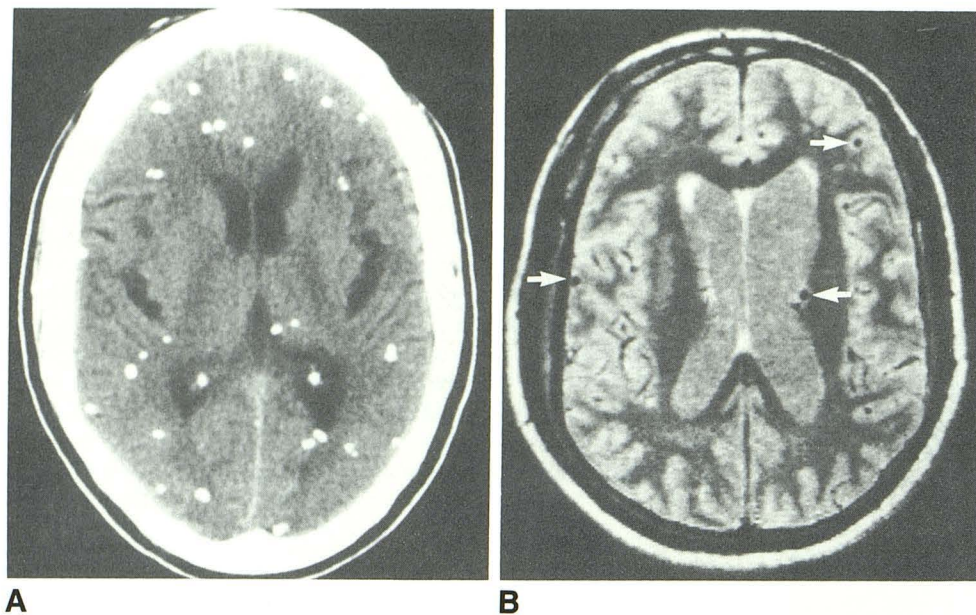
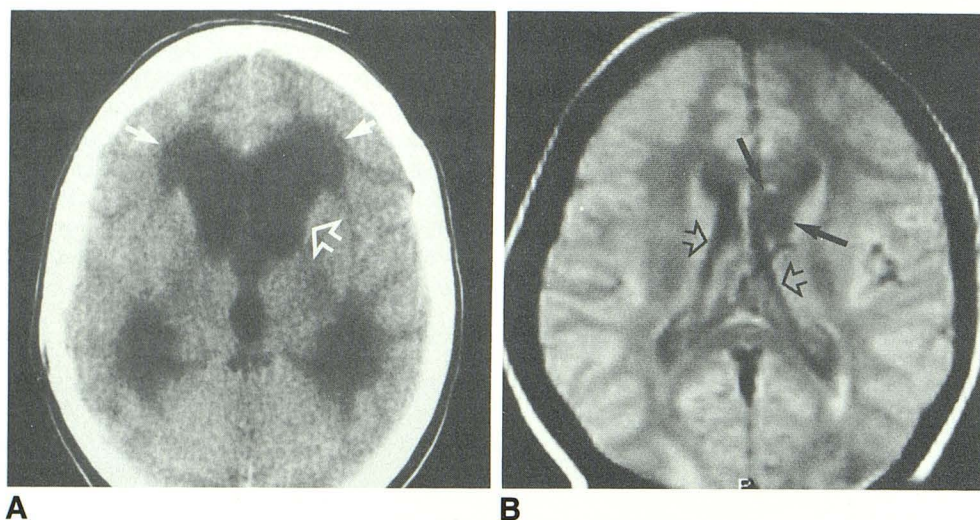


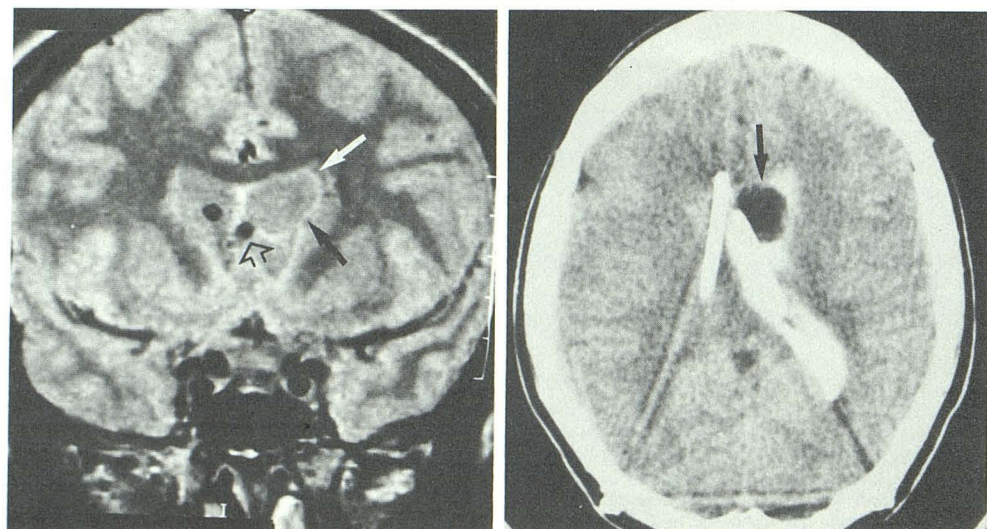
Fig. 10.—A, Nonenhanced brain CT scan reveals numerous small parenchymal calcifications caused by old cysticercosis lesions.

B, Axial MR brain scan (SE 2000/60) displays calcific foci as small signal-void zones (arrows). Parenchymal calcifications of neurocysticercosis are better detected with CT than with SE MR imaging.



A

B



C

D

Fig. 11.—A, Nonenhanced brain CT scan shows third and lateral ventricular dilatation as well as periventricular low attenuation (solid arrows) consistent with hydrocephalus with transependymal CSF flow. Frontal horn dilatation is slightly more prominent on left side (open arrow).

B, Axial first-echo MR brain scan (SE 2000/30) after placement of bilateral ventriculoperitoneal shunt tubes (open arrows). There is left frontal horn intraventricular cysticercosis cyst (solid arrows) with well-defined cyst rim. Ventriculoperitoneal shunt tubes display low signal intensity (open arrows).

C, Coronal MR scan (SE 3000/40) further defines left frontal horn cyst (solid arrows), which displaces left shunt tube medially and inferiorly (open arrow).

D, Contrast ventriculography performed during follow-up brain CT scan confirms presence of cyst (arrow).

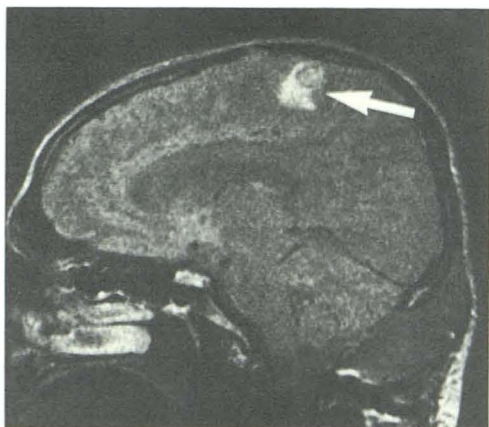


Fig. 12.—Sagittal section (SE 1000/80) of patient in Fig. 3 after surgical resection of fourth ventricular cyst and subsequent oral praziquantel therapy. There is now evidence of pericystic edema, consistent with adjacent inflammatory reaction, surrounding superiorly located parenchymal cyst (arrow).

be of assistance in the follow-up evaluation of patients. Intraventricular cysts were encountered in 14 (54%) of the 26 patients. The cyst fluid was generally isointense relative to CSF; however, in two cases, slightly increased cyst fluid intensity (relative to CSF) was present, which may have been related to cyst degeneration or lack of flow effects within the cyst fluid or both. Mass effect, a cyst rim, adjacent CSF flow void, and ventricular outflow obstruction were all beneficial in confirming the presence of intraventricular cysts. Eleven (79%) of the 14 patients with intraventricular cysts showed evidence of pericystic ependymal reaction. This finding may be a sign of initial cyst degeneration or may be the result of cystic attachment to the ependyma, eliciting a granulomatous response [8]. Nine (90%) of the 10 patients with aqueductal stenosis had intraventricular cysts. The aqueductal stenosis may have been the result of prior intraventricular cyst degeneration resulting in ependymal scarring and adhesions [9]. The direct sagittal imaging capabilities of MR were vital in diagnosing aqueductal stenosis. Eleven patients (79%) with intraventricular cysts had symptoms of frequent headaches, nausea, and vomiting, which may have been related to increased intracranial pressure. Intraventricular cysts may remain clinically silent until they degenerate, inciting an ependymal reaction, or until they cause obstructive hydrocephalus [9–12].

One patient (Fig. 9) presented with the racemose form of neurocysticercosis. Racemose cysts usually lack a scolex and are typically found in the cisterns around the rostral brainstem or the sylvian fissure [1]. They may produce obstructive hydrocephalus [1]. The degenerating cyst may incite an excessive leptomeningeal fibroblastic and inflammatory reaction, causing extensive cyst wall proliferation. In our patient, the proliferative changes in the cyst wall were well demonstrated by MR. The patient also had a history of meningitis. The racemose form of neurocysticercosis is known to be associated with chronic meningitis [9].

Parenchymal cysts were present in 18 (69%) of the 26 patients and were noted to involve the gray/white-matter

junction almost invariably. No cases of meningeal neurocysticercosis were encountered in our series. Typically, the parenchymal cysts that were considered viable were isointense relative to CSF on T1- and T2-weighted images. It is known that with cyst degeneration, cyst fluid becomes more proteinaceous and gelatinous [9]. This was manifested on MR images as increased signal intensity of the cyst fluid on T1- and proton-density-weighted sequences, probably due to T1-shortening effects of increased protein solutes and surface-layer effects of water molecules interacting with macromolecules within the cyst fluid [13].

Viable parenchymal larvae survive for approximately 5 years and then die for unknown reasons [14]. Upon the death of the parasites, a more pronounced host immune response is initiated with inflammation and edema. This inflammatory reaction often causes seizures [5, 9]. Correlation with pathologic specimens in two patients with marked pericystic inflammatory reaction (evidenced by increased signal intensity) demonstrated gliosis and edema (Figs. 5 and 6). Secondary astrocytic gliosis of variable intensity has been described as the most common pathologic change in the parenchyma immediately surrounding the cyst [9]. Because marked pericystic high signal was seen in one patient with a histopathologically demonstrated cysticercus (Fig. 7), the presence of pericystic hyperintensity may not always be a reliable indicator of the stage of cyst degeneration. Most of the parenchymal cysts encountered were 1–2 cm in diameter. However, one patient was found to have a cerebellar cyst 4 cm in diameter with a rim of pericystic edema (Fig. 6).

Although the advantages of MR in neurocysticercosis were obvious—that is, better tissue characterization, multiplanar imaging capabilities, etc.—SE MR imaging was insensitive to parenchymal calcifications that were well demonstrated on CT (Fig. 10) [7]. Gradient-echo MR imaging may display greater sensitivity to the presence of parenchymal calcifications. However, when compared with CT, MR better demonstrated the extent of pericystic inflammation (Fig. 7).

Praziquantel is a heterocyclic pyrazinoisoquinoline oral anthelmintic agent that is most effective against the *Schistosoma* species [15]. In some cases, it has been shown to be effective in the treatment of cysticercosis and typically is administered with a total daily oral dosage of 60 mg/kg for 4 days to 2 weeks [16–18]. Praziquantel acts by increasing the permeability of the organism's cell membranes to calcium ions, causing massive muscle contraction and paralysis followed by disintegration. Dexamethasone is often given in combination with praziquantel to reduce the inflammatory reaction (with accompanying edema) arising from larval degeneration, which can itself result in additional clinical symptoms, such as seizures or elevated intracranial pressure [12, 16].

One difficulty with praziquantel therapy is that patients with neurocysticercosis, especially those with parenchymal cysts, often do not present until cyst degeneration has already begun. The viable larvae tend not to cause appreciable symptoms, thus making evaluation of drug effectiveness difficult [9].

In the present series of six patients who received praziquantel therapy, two of the patients with parenchymal cysts demonstrated either a reduced number of detectable cysts or

evidence of the development of pericystic edema and cyst degeneration on follow-up MR studies (Fig. 12). None of the intraventricular cysts showed appreciable changes on repeat MR examinations performed 6 weeks to 6 months after a full course of therapy. These preliminary findings suggest that the CSF concentration of praziquantel may not have been sufficiently high to effect the destruction of viable larvae, and that a longer period of treatment may be required for the eradication of intraventricular cysts.

We conclude that MR is useful in evaluating neurocysticercosis, especially in the case of intraventricular cysts, not only because of MR's ability to actually detect cysts, but also because of its ability to detect signs of cyst degeneration and pericystic inflammation. Cerebral aqueductal stenosis due to periaqueductal ependymal inflammation was well demonstrated with MR. However, MR was insensitive to parenchymal calcifications that are typically seen well with CT. In our series of neurocysticercosis patients, treatment with praziquantel did not appear to appreciably affect intraventricular cysts.

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REFERENCES

- McCormick GF, Zee CS, Heiden J. Cysticercosis cerebri: review of 127 cases. *Arch Neurol* 1982;39:534-539
- Suss RA, Maravilla KR, Thompson J. MR imaging of intracranial cysticercosis: comparison with CT and anatomopathologic features. *AJNR* 1986;7:235-242
- Rydzewski AK, Chrischold ES, Kagan IG. Comparison of serologic tests for human cysticercosis by indirect hemagglutination, indirect immunofluorescent antibody, and agar gel precipitin test. *J Parasitol* 1975;61:154-155
- Miller B, Goldberg MA, Heiner D, Myers A, Goldberg A. A new immunological test for CNS cysticercosis. *Neurology* 1984;34:695-697
- Rickard MD, Williams JF. Hydatidosis/cysticercosis: immune mechanisms and immunization against infection. *Adv Parasitol* 1982;21:229-296
- Flisser A, Larralde C. Cysticercosis. In: Walls KW, Schantz PM, eds. *Immunodiagnosis of parasitic diseases*, vol. 1. *Helminthic disease*. New York: Academic Press, 1986:109-161
- Bentson JR, Wilson GH, Helmer E, Winter J. Computed tomography in intracranial cysticercosis. *J Comput Assist Tomogr* 1977;1:464-471
- Scaravilli F. Parasitic and fungal infections of the nervous system. In: Adams JH, Corsellis JA, Duchen LW, eds. *Greenfield's neuropathology*, 4th ed. New York: Wiley, 1984:315-317
- Escobar A. The pathology of neurocysticercosis. In: Palacios E, Rodriguez-Carbajal J, Taveras JM, eds. *Cysticercosis of the central nervous system*. Springfield, IL: Thomas, 1983:27-54
- Marquez-Monter H. Cysticercosis. In: Marcial-Rojas, ed. *Pathology of protozoal and helminthic diseases with clinical correlation*. Baltimore: Williams & Wilkins, 1971:592-617
- Shanley JD, Jordan MC. Clinical aspects of CNS cysticercosis. *Arch Intern Med* 1980;140:1309-1313
- Apuzzo MLJ, Dopkin WR, Zee C-S, Chan JC, Giannotta SL, Weiss MH. Surgical considerations in treatment of intraventricular cysticercosis. An analysis of 45 cases. *J Neurosurg* 1984;60:400-407
- Fullerton GD. Physiologic basis of magnetic relaxation. In: Stark DD, Bradley WG Jr, eds. *Magnetic resonance imaging*. St. Louis: Mosby, 1988:36-55
- Handler LC, Mervis B. Cerebral cysticercosis with reference to the natural history of parenchymal lesions. *AJNR* 1983;4:709-712
- Praziquantel—a new antiparasitic drug. *Med Lett Drugs Ther* 1982;24:108-109
- Spina-Franca A, Nobrega JPS, Livramento JA, Machado LR. Administration of praziquantel in neurocysticercosis. *Trop Med Parasitol* 1982;33:1-4
- Sotelo J, Escobedo F, Rodriguez-Carbajal J, Torres B, Rubio-Donnadieu F. Therapy of parenchymal brain cysticercosis with praziquantel. *N Engl J Med* 1984;310:1001-1007
- deGhetaldi LD, Norman RM, Douville AW. Cerebral cysticercosis treated biphasically with dexamethasone and praziquantel. *Ann Intern Med* 1983;99:179-181