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MR Imaging in Cysticercotic Encephalitis

Oscar H. Del Brutto,¹ Marco A. Zenteno, Perla Salgado, and Julio Sotelo

Cysticercotic encephalitis is a severe form of neurocysticercosis in which the brain parenchyma is harmed by the host immune response to massive cysticerci infestation [1]. Patients with this disorder usually present abnormalities of consciousness associated with intracranial hypertension, visual changes, and generalized seizures. Because these are non-specific clinical manifestations, the diagnosis largely depends on demonstration of characteristic lesions by CT. CT findings include diffuse brain edema, small lateral ventricles without midline shift, and multiple round and small areas of abnormal enhancement after administration of contrast material [1].

MR has been of great value in the diagnosis of neurocysticercosis [2–6]; it is superior to CT in recognition of edema around cysticerci and internal changes indicative of cyst death [2]. In cysticercotic encephalitis, most lesions represent dying parasites surrounded by severe edema [1]; therefore, MR should be an ideal study for these cases. In this report, we evaluate the usefulness and features of MR in two patients with cysticercotic encephalitis.

Methods

Two patients with cysticercotic encephalitis were studied with MR operating at a field strength of 0.5 T*. Images were acquired on a 256 × 256 matrix with 1.1-mm pixel size. The slice thickness was 8 mm with 1.6-mm interslice gaps. Both T1-weighted images (560/26 [TR/TE]) and T2-weighted images (2000/50) were obtained. Findings obtained with MR were compared with those obtained with CT†.

Case Reports

Case 1

A 25-year-old man was admitted 48 hr after the onset of headache, vomiting, and lethargy. Neurologic examination showed bilateral papilledema and generalized hyperreflexia. CSF analysis disclosed 20 mononuclear cells per mm³, with normal glucose and protein levels; enzyme-linked immunosorbent assay (ELISA) and complement-fixation test for cysticercosis were positive. CT scan showed diffuse brain edema without midline shift and scarce areas of abnormal ringlike enhancement after administration of contrast material (Figs. 1A and 1B). MR showed more than 200 small areas of decreased signal intensity in the T1-weighted images, predominantly located in

subcortical areas (Fig. 1C). In the T2-weighted sequences, several lesions were surrounded by focal areas of increased signal intensity representing edema (Fig. 1D). The patient was treated with dexamethasone and mannitol with clinical improvement and was discharged asymptomatic after 15 days.

Case 2

A 3-year-old girl with a 9-month history of generalized seizures was evaluated because of recent onset of decrease of visual acuity and loss of previously acquired developmental skills. There was no family history of neurologic disease. Head circumference was 51 cm. The patient was alert but showed marked irritability. She did not show visual follow of colorful objects and did not attempt to grasp them. Pupillary response and fundoscopic examination were normal. Motor testing showed generalized spasticity with hyperreflexia and bilateral Babinski sign. Lumbar puncture yielded a clear CSF with normal content of cells and proteins, but positive immunologic reactions to cysticercosis (ELISA and complement-fixation test). CT scan showed symmetric decrease in attenuation of subcortical white matter and multiple small areas of abnormal enhancement after administration of contrast materials (Figs. 2A and 2B). MR showed periventricular hyperintensity and about 250 small areas of abnormal signal intensity scattered in cerebral hemispheres and brainstem; these lesions were more easily identified in the T2-weighted sequences with signal properties paralleling CSF (Figs. 2C–2F). The patient was treated with dexamethasone and phenytoin. Neurologic examination 2 weeks later revealed normal visual function. After 4 weeks, she walked without assistance and spoke normally.

Discussion

Modern neuroimaging techniques have greatly improved diagnostic accuracy for neurocysticercosis because they provide objective evidence on location of cysticerci and degree of inflammatory response to the parasite [2, 7, 8]. Severity of this inflammatory response is highly variable since it is related to the host state of responsiveness to cysticerci infestation [9, 10]. In many cases, it develops slowly and often fails to eliminate the parasites. In other patients, however, cysticerci are acutely rejected after entering the brain parenchyma; in these patients the infection is overcome, but the brain usually becomes damaged as a result of the induced immune reac-

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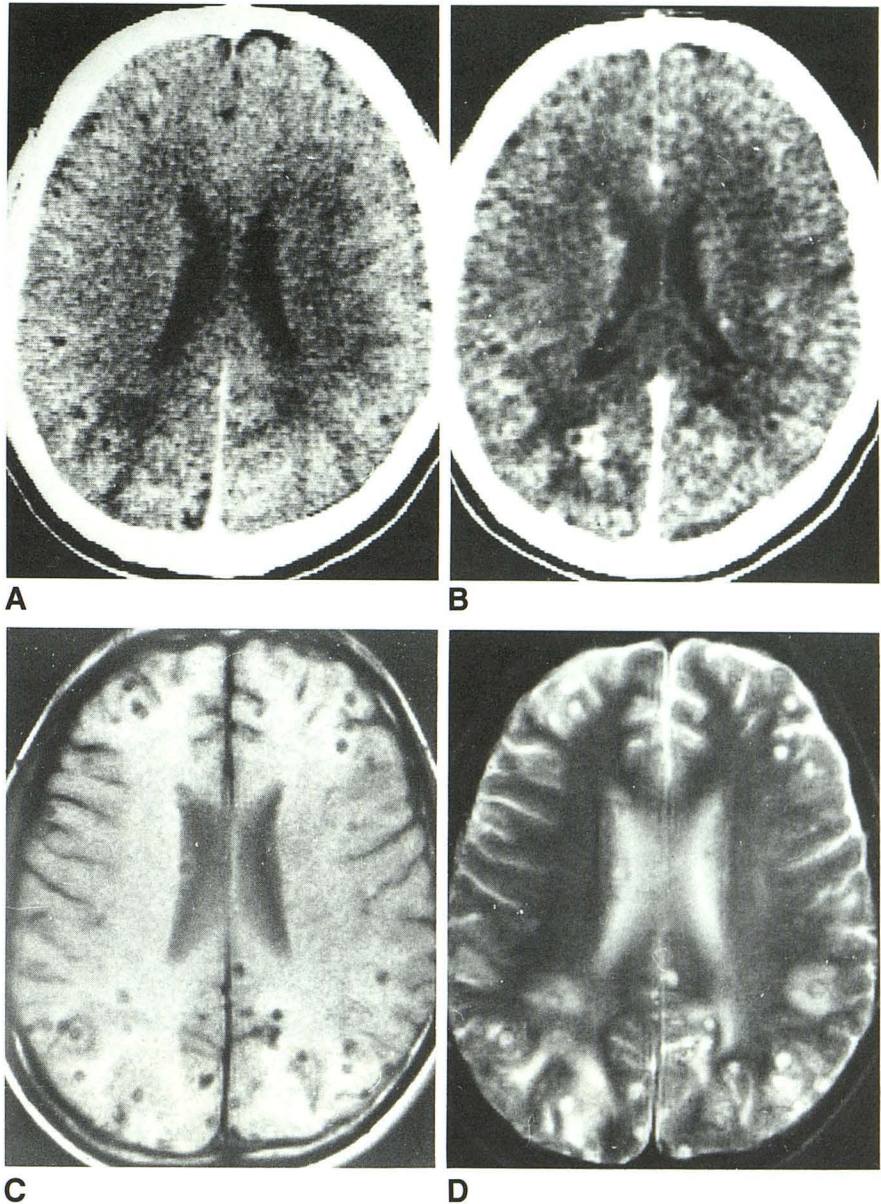
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Fig. 1.—Case 1.

A and B, CT scans. Noncontrast CT scan (A) shows diffuse brain edema and small lateral ventricles and some small cysts in subcortical area. Contrast CT scan (B) shows scarce areas of abnormal enhancement.

C and D, MR images. Both T1-weighted (C) and T2-weighted (D) images show multiple cystic lesions throughout brain parenchyma with signal-intensity properties paralleling CSF.



tion, with a spectrum of clinical manifestations and characteristic CT findings recognized as cysticercotic encephalitis.

Cases reported here had the characteristic multifocal involvement of brain parenchyma previously described in cysticercotic encephalitis [1]. In both cases, MR was more sensitive than CT in detecting small parenchymal cysticerci. In patient 1, cystic lesions had signal-intensity properties paralleling CSF in both T1- and T2-weighted sequences. In patient 2, most cysts were isointense with brain parenchyma in the T1-weighted images and were more easily seen in the T2-weighted images, in which they had relaxation times similar to CSF. This difference probably is related to a greater protein content in the cystic fluid of cysticerci in patient 2.

Lack of scolex in most lesions is a feature of cysticercotic encephalitis. As described by Suss et al. [2], MR is highly sensitive for detecting the scolex of cysticerci producing a characteristic "hole-with-dot" imaging. However, when dead

parasites begin to degenerate, as in cysticercotic encephalitis, proteins from the scolex combine with the vesicular fluid; because the larva is then almost isointense with the vesicular fluid, it is not disclosed by MR. Moreover, the whole cyst may become isointense with brain parenchyma in the T1-weighted images; these cysts are discernible only on T2-weighted images. Knowledge of these features is important for accurate classification of the disease before planning a therapeutic approach. As we have pointed out previously, patients with cysticercotic encephalitis should not receive anticysticercus drugs during the acute phase of the disease since these drugs may exacerbate the inflammatory response [11]. Steroids and/or osmotic diuretics are advised as the first therapeutic measures to reduce the severe brain edema that accompanies this disorder; subsequent follow-up will permit the physician to decide on the use of anticysticercus drugs, especially if a further neuroimaging evaluation shows persist-

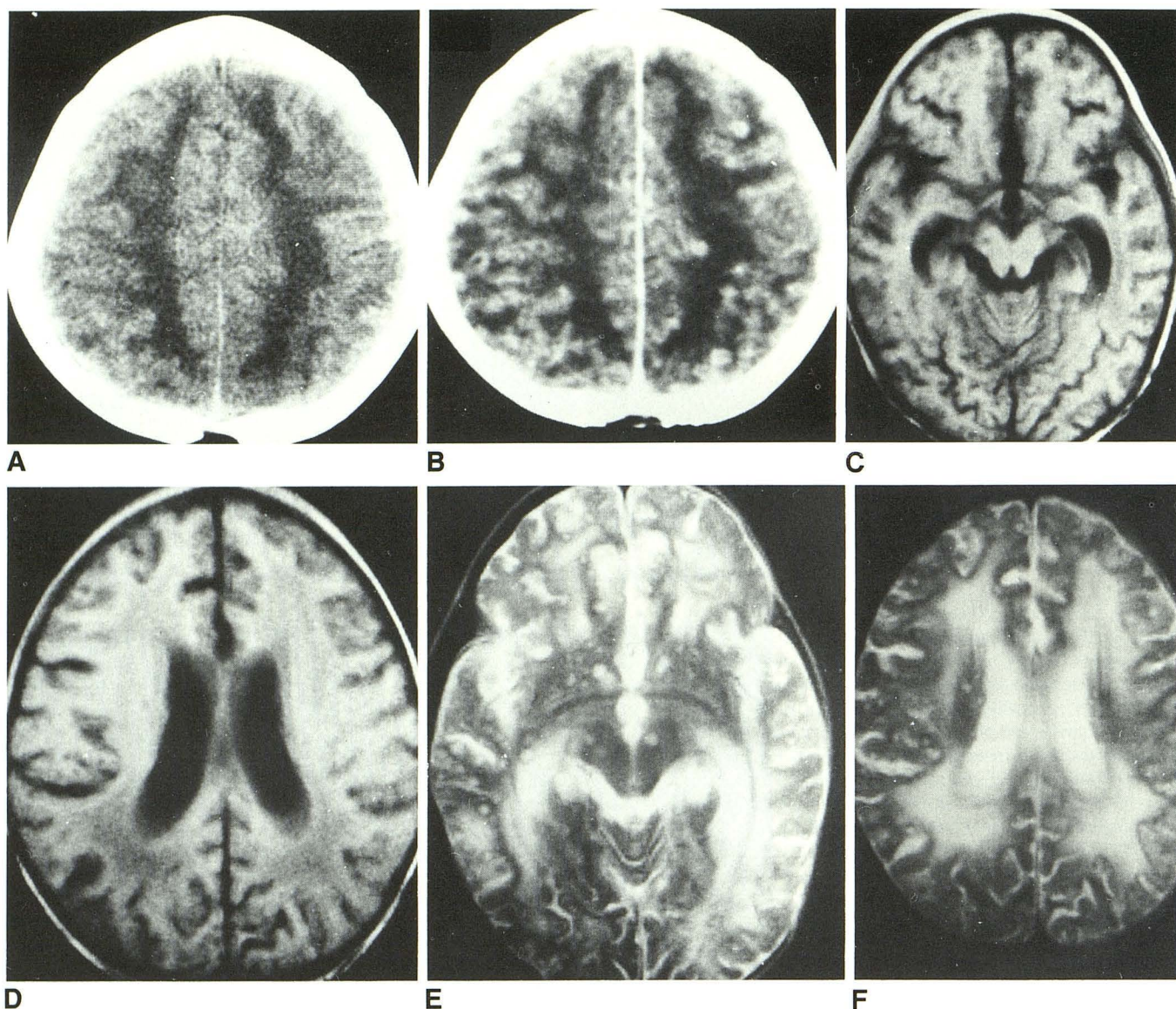


Fig. 2.—Case 2.

A and B, CT scans. Noncontrast (A) and contrast (B) CT scans show diffuse low attenuation of subcortical white matter and multiple small nodular areas of abnormal enhancement in brain parenchyma.

C and D, T1-weighted MR images show scarce cystic lesions within cerebral cortex and brainstem (C) with associated periventricular hyperintensity (D).

E and F, T2-weighted MR images show multiple cystic lesions throughout brain parenchyma with signal-intensity properties paralleling CSF (E) and severe periventricular hyperintensity (F).

ence of lesions after the clinical signs of acute inflammation have disappeared.

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