

# Diagnosis of intraventricular cysticercosis by magnetic resonance imaging: Improved detection with three-dimensional spoiled gradient recalled echo sequences

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## SUMMARY

Neurocysticercosis (NCC) is caused when the cysticercus larvae of *Taenia solium* infect the central nervous system. The larvae usually land in the parenchymal tissue, but quite rarely can lodge in the ventricles and cisterns of the brain. Unlike parenchymal NCC, it is not easy to demonstrate the cysticercus cysts within the cerebrospinal fluid spaces. Computed tomography and even conventional MR sequences can fail to detect such cysts. However, obtaining three-dimensional spoiled gradient recalled echo imaging sequences can help in improving the detection of intraventricular NCC, as is borne out by a case described in the present report. The use of such special MR sequences is therefore strongly advocated in suspected cases of intraventricular NCC.

**Key words:** *intraventricular cysticercosis; magnetic resonance imaging; neurocysticercosis.*

## INTRODUCTION

Neurocysticercosis (NCC) is an endemic neuroparasitosis in the underdeveloped tropical and subtropical regions of the world. The incidence is increasing in the developed countries because of immigration of infected individuals and tourism in endemic areas.<sup>1–4</sup> The current situation demands that radiologists all over the world should familiarize themselves with the imaging characteristics of NCC.

Neurocysticercosis is secondary to the infestation of the central nervous system by the *Taenia solium* larvae. Man can be a definitive as well as an intermediate host. The larvae enter the circulation through the gastric wall and involve many organs including the nervous system. Neurocysticercosis is commonly intraparenchymal and uncommonly intraventricular or intracisternal.<sup>5</sup> Although parenchymal cysticerci can be readily identified on CT and MRI, the lesions within cerebrospinal fluid (CSF) spaces are difficult to identify.<sup>6</sup> We describe a rare case of fourth ventricular NCC, which could be diagnosed only on obtaining special MR sequences.

## CASE REPORT

A man aged 19 years, previously in good health, presented with complaints of episodic headache and vomiting for the last few months. The headache was non-throbbing in character and would last for a few hours at a time. Past history revealed that at the age of 13 years, he had reported with similar complaints at the paediatric emergency facility of another hospital. Computed tomography scan of the brain done at that time was unremarkable. The boy eventually recovered within 3 weeks and kept well until recently when he suffered from recurrence of symptoms.

Neurological examination revealed normal higher functions, normal cranial nerves and no sensorimotor deficit. Routine blood parameters were within normal limits. Non-contrast CT of the brain revealed subtle dilatation of the fourth ventricle with very mild prominence of the ventricular system upstream. Contrast-enhanced CT did not provide any additional information. Magnetic resonance imaging of the brain using T1-weighted and fast fluid attenuated inversion recovery (FLAIR)

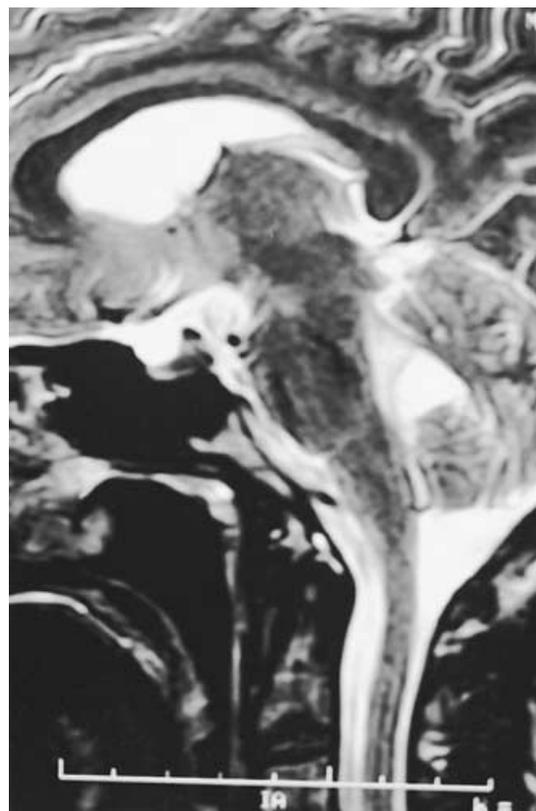
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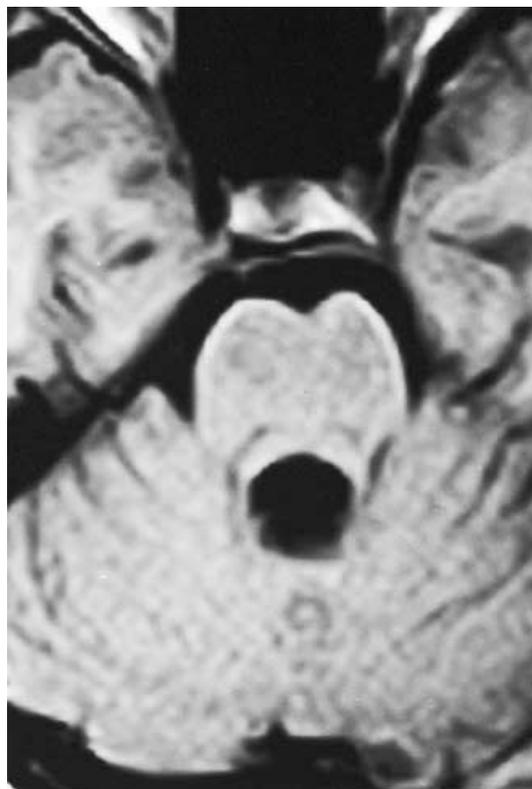
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imaging sequences, was by and large unremarkable except for a prominent fourth ventricle. (Fig. 1). T2-weighted scans showed high-intensity intraventricular cystic fluid in the fourth ventricle, without any demonstrable cyst wall. (Fig. 2). In order to clarify the abnormality more clearly, a three-dimensional (3-D) spoiled gradient recalled echo (SPGR) sagittal sequence was obtained which revealed a small round cyst within the fourth ventricle having a well-defined wall and exhibiting iso-intense signals to the brain parenchyma. The cyst wall, cystic fluid and the central nidus representing the scolex were clearly discernible (Fig. 3). Based on these imaging characteristics, a confident diagnosis of fourth ventricular NCC was made. There was no evidence of any associated parenchymal or sub-arachnoid cysticercosis. Gadolinium (Gd)-enhanced MR did not show any evidence of ependymitis or arachnoiditis.

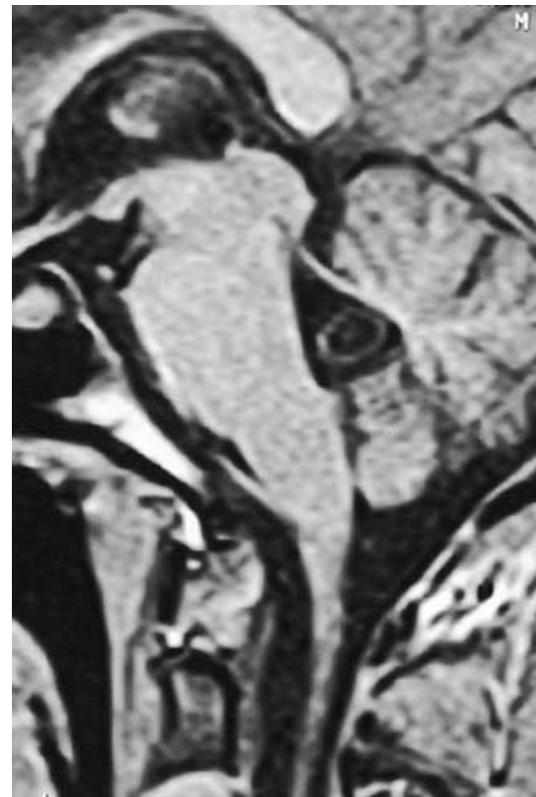
The patient underwent suboccipital craniotomy and the floor of the fourth ventricle was exposed. A rounded pearly white cyst was seen blocking the outlet of the fourth ventricle. The cyst was lifted up and removed by gentle flushing with saline. Histological examination confirmed it to be a cysticercus cyst. Postoperatively, the patient was put on a course of albendazole. He had an uneventful recovery with complete resolution of the symptoms.



**Fig. 2.** T2-weighted sagittal section revealing a high-intensity cystic fluid in the fourth ventricle.



**Fig. 1.** T1-weighted axial section showing a prominent fourth ventricle.



**Fig. 3.** Three-dimensional spoiled gradient recalled echo sagittal section showing fourth ventricular cysticercus cyst with clear definition of the cyst wall, cystic fluid and the central nidus.

## DISCUSSION

Few neurological conditions are as confusing as NCC. This is partly because there are no characteristic clinical features that would clinch the diagnosis and partly because the symptomatology of NCC resembles a multitude of diverse neurological entities. Several factors, such as the immunological response of the host and the number, size and location of oncospheres, contribute to the clinical picture of NCC.<sup>7</sup> Clinically, the condition could present in a variety of forms with convulsive, psychiatric or intracranial hypertensive manifestations.<sup>5</sup> The syndrome of intracranial hypertension occurring with intraventricular NCC comprises violent headache, vomiting and visual disturbances. It is generally observed when CSF circulation is interrupted, as can occur when a cysticercus cyst occludes the foramen of Monroe, the third ventricle, the aqueduct or the fourth ventricle. The symptoms can disappear if the cysticercus moves away from the obstructing site.<sup>8</sup>

The diagnosis of intraventricular cysticercosis has traditionally been made by such invasive procedures as ventriculography, CT ventriculography and pneumoencephalography.<sup>9</sup> With the introduction of MRI, it became possible to image intraventricular cystic lesions non-invasively.<sup>10,11</sup> However, despite the improved depiction of tissue contrast and the multiplanar imaging capabilities of MRI, intraventricular NCC can be missed on routine MR sequences.<sup>11</sup> In our patient, the diagnosis of intraventricular NCC could not be made on T1-/T2-weighted or on fast FLAIR imaging sequences. The lesion could be depicted clearly only on obtaining 3-D SPGR imaging sequences, which distinctly revealed the cyst wall, cystic fluid and the central scolex. In this sequence, a complex radio-frequency pulse is applied to each repetition time to eliminate any residual transverse magnetization or T2 effects. This technique thus helps to increase tissue differentiation and the signal-to-noise ratio.

If vesicular breakdown occurs within CSF spaces, an intense inflammatory reaction can ensue leading to ependymitis or arachnoiditis with secondary hydrocephalus. This can manifest as striking enhancement of the leptomeninges or ventricular ependyma on Gd-enhanced MRI.<sup>12</sup> The concurrent presence of ependymitis or arachnoiditis determines whether surgical removal of the cyst or placement of a shunt is indicated

in the first instance. In the index case, contrast-enhanced MRI did not reveal any evidence of arachnoid or ependymal inflammation. Hence, our patient was subjected to direct removal of the cyst.

To conclude, routine MR sequences might be less sensitive in detecting intraventricular NCC. The role of contrast-enhanced MRI is restricted to the demonstration of any associated arachnoiditis or ependymitis. Nevertheless, to improve the accuracy of MR examination in suspected cases of intraventricular NCC, we strongly recommend that in addition to routine imaging sequences and contrast-enhanced MRI, 3-D SPGR imaging sequences should also be obtained.

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