In endemic regions, NCC is a common parasitic disease that can manifest numerous clinical signs and symptoms. The most common presentation is seizure secondary to parenchymal cysts. Cisternal involvement is less common and is usually accompanied by parenchymal cysts and possibly intraventricular cysts. It is assumed that cisternal cysts came to reach this final anatomical location by passing through the ventricular system. Although cisternal cysts can be solitary, they much more commonly occur as a localized conglomeration of cysts as a diffuse infestation of the subarachnoid spaces. Patients with cisternal NCC involvement usually present with hydrocephalus either caused by the shear number of cysts occluding CSF movement or, more commonly, a chronic arachnoiditis associated with cyst degeneration. Rarely, the local mass resulting from a localized pocket of subarachnoid cysts can cause neurological symptoms due to compression of the brainstem, cranial nerves, or the ventricular pathways. Like the other forms of NCC, brain compression caused by the mass effect of the cysts can be aggravated by an inflammatory arachnoiditis. It is thought that the latter can lead to adjacent brain ischemic conditions caused by a microvasculitis. As with other focal entities, the neurological presentation will vary based on the cyst’s neuroanatomical location. Cysts present in the basilar cisterns have been reported to cause hemifacial spasm, trigeminal neuralgia, and brainstem compression. The management of cisternal disease varies depending on clinical experience and institutional bias, because the literature provides only anecdotal reports. In most cases, either the extent of involvement or the advanced-stage disease precludes surgical intervention. Previously open surgical attempts to remove NCC cysts from the subarachnoid spaces were reported to be futile and often dangerous. Resection of cysts densely adherent to the brainstem or cranial nerves was not possible without causing significant neural damage. Overall, the morbidity rate associated with extraparenchymal cysticercosis is greater than that for parenchymal disease.

Endoscopic removal of cisternal neurocysticercal cysts

Technical note

TOORAJ GRAVORI, M.D., THOMAS STEINEKE, M.D., PH.D., AND MARVIN BERGSNEIDER, M.D.

Harbor-UCLA Medical Center and Division of Neurosurgery, University of California, Los Angeles, California

The authors report the use of neuroendoscopy for the treatment of cisternal neurocysticercosis. The utilization of the endoscope is meant to provide a safe and minimally invasive procedure for the removal of symptomatic cysts, minimizing morbidity and mortality relating to the natural history of the disease, as well as possibly avoiding a more extensive standard open craniotomy.

A retrospective review of three cases of cisternal cysticercosis was performed. The presenting signs and symptoms, neuroimaging findings, endoscopic treatment, and outcomes are presented. The technical aspect of the endoscopic resection is described, with visual support provided by intraoperative video. Cerebrospinal fluid shunt placement was avoided in one patient in whom complete resection of the cyst was required. In one patient there were extensive interhemispheric and presmesencephalic cisternal cysts, some of which could not be removed because of their multiplicity and extensive adhesions. Despite the intraoperative rupture of cysts, there were no cases of arachnoiditis or ventriculitis postoperatively.

The minimally invasive endoscopic resection of cisternal cysticercosis is possible and effective in selected patients. Although the most appropriate treatment of cisternal cysticercosis remains a controversial issue, endoscopic resection should be considered as a primary treatment in symptomatic patients in cases that are amenable to endoscopic intervention.

KEY WORDS • cysticercosis • subarachnoid space • neurocysticercosis • endoscopy • minimally invasive

In endemic regions, NCC is a common parasitic disease that can manifest numerous clinical signs and symptoms. The most common presentation is seizure secondary to parenchymal cysts. Cisternal involvement is less common and is usually accompanied by parenchymal cysts and possibly intraventricular cysts. It is assumed that cisternal cysts came to reach this final anatomical location by passing through the ventricular system. Although cisternal cysts can be solitary, they much more commonly occur as a localized conglomeration of cysts as a diffuse infestation of the subarachnoid spaces. Patients with cisternal NCC involvement usually present with hydrocephalus, either caused by the shear number of cysts occluding CSF movement or, more commonly, a chronic arachnoiditis associated with cyst degeneration. Rarely, the local mass resulting from a localized pocket of subarachnoid cysts can cause neurological symptoms due to compression of the brainstem, cranial nerves, or the ventricular pathways. Like the other forms of NCC, brain compression caused by the mass effect of the cysts can be aggravated by an inflammatory arachnoiditis. It is thought that the latter can lead to adjacent brain ischemic conditions caused by a microvasculitis. As with other focal entities, the neurological presentation will vary based on the cyst’s neuroanatomical location. Cysts present in the basilar cisterns have been reported to cause hemifacial spasm, trigeminal neuralgia, and brainstem compression. The management of cisternal disease varies depending on clinical experience and institutional bias, because the literature provides only anecdotal reports. In most cases, either the extent of involvement or the advanced-stage disease precludes surgical intervention. Previously open surgical attempts to remove NCC cysts from the subarachnoid spaces were reported to be futile and often dangerous. Resection of cysts densely adherent to the brainstem or cranial nerves was not possible without causing significant neural damage. Overall, the morbidity rate associated with extraparenchymal cysticercosis is greater than that for parenchymal disease.
In this report, we describe the successful endoscopic management of three patients with cisternal (subarachnoid) NCC. Present in each case was a localized accumulation of cysts in a cisternal space adjacent to the ventricular system. The principal goal for the treatment was to remove safely the cisternal lesions, thereby reducing the morbidity associated with cystic mass effect, degeneration, inflammation, and expansion. A secondary goal was to reduce the need for procedures involving placement of a CSF shunt by decreasing the obstruction to CSF. This was achieved by combining cyst resection with direct treatment of the hydrocephalus—that is, a third ventriculostomy and/or septum pellucidotomy.

CLINICAL MATERIAL AND METHODS
Presentation and Preoperative Management

We reviewed the medical records of three cases in which patients with symptomatic cisternal cysticercosis underwent endoscopic surgery, performed by the senior author (M.B.) at Harbor–UCLA Medical Center between January 1997 and December 2001. The patients were adults who had emigrated from Mexico to the Los Angeles area. In general, all three patients presented with progressive symptoms relating to mass effect and hydrocephalus. Each patient underwent placement of a ventriculostomy drain on admission for the treatment of hydrocephalus. Each patient underwent placement of a VP shunt; neurologic intact & symptom resolved. In one patient, who was returned to the operating room after resolution of meningi-
tis, a CSF shunt was placed after he could not be weaned from external drainage. Based on the findings of CSF within the fourth ventricle and cisterna magna, a CSF shunt was placed in the other patient (Case 2).

**DISCUSSION**

In this report we present a minimally invasive approach to the extirpation of cisternal cysticercosis cysts. In two of our cases, the neurological effects (obstructive hydrocephalus and optic apparatus compression) of the localized mass lesion were alleviated by removal of the cysts. In the third case it was demonstrated that the quadrigeminal cistern could safely be accessed endoscopically. The purpose of this report was not to promote the removal of all cisternal NCC cysts but rather to illustrate that in selected cases this technique was a good alternative or supplement to standard treatment. In our three cases, treatment of the cisternal cysts by standard (nonendoscopic) open microsurgical techniques would have required a craniotomy and either a larger corticectomy or some degree of brain retraction.

Some practitioners have advocated a conservative approach to the management of cisternal cysticercosis. They have contended that the natural history of cysticercosis is benign and that most cysts simply die and “burn-out” within 10 or 20 years. It has been argued that only the associated hydrocephalus needs to be treated because the disease is self limited. This argument arises from the observation that parenchymal cysts often become calcified and die after a period of time. There are, however, numerous accounts of the much higher rates of mortality and morbidity resulting from cisternal cysts because of their propensity to cause direct mass effect on the midbrain and brainstem, as well as inflammation leading to arachnoiditis, cranial neuropathies, and vasculitis.

Cisternal lesions have been treated medically by administering antiparasitic agents albendazole and praziquantel. Authors of case reports have suggested that these agents prevent further expansion of the cystic lesions and in some cases reduce the size of the subarachnoid cysts. Relapses, however, frequently occur. The efficacy of albendazole and praziquantel is debatable. A number of authors have asserted the benefit of medical treatment; however, no large scale prospective studies have been conducted. In a review of the randomized controlled trials published since 1966, Salinas, et al., concluded that there is insufficient evidence that medical therapy for NCC provides any clinical benefit. Furthermore, it is thought that CSF concentrations of albendazole and praziquantel are lower than those in the parenchyma, making medical management even more unlikely to treat cisternal NCC adequately.

Several authors have reported the extirpation of these cysts by open craniotomy or aspiration via CT-guided stereotactic puncture. The very nature of the disease, however, makes traditional surgical procedures inherently risky. These are deep-seeded lesions that may be difficult to access. Skull base approaches may be required to reach and excise the lesions.

**Fig. 1.** Case 1. Axial T1-weighted MR images (left and center) demonstrating nonenhancing interhemispheric and left premesencephalic cysts. Midsagittal T1-weighted MR image demonstrating the extent of the NCC cyst.

**Fig. 2.** Case 2. Postventriculostomy CT scans revealing a right quadrigeminal cyst that extends into the atrium of the right lateral ventricle.
A relative contraindication to resection (including that assisted by endoscopy) of cisternal cysts is the presence of significant inflammation, which is typically demonstrated by extensive enhancement of the cisterns and cysts on MR images. In these cases, the arachnoid and cyst walls are often opaque and indistinguishable, making exploration and removal of the cysts difficult and hazardous. If surgical exploration is considered, a low threshold for converting from the excision to placement of a shunt must be maintained. The preoperative use of steroid therapy must also be considered and may prove beneficial.

Not all cisternal cysts are amenable to endoscopic excision. In each of our cases there were cysts that could be approached via a transventricular route (and associated hydrocephalus). Cysts in the cerebellopontine angle or anterior to the brainstem are likely poor candidates for any surgical approach.

CONCLUSIONS

In this report we demonstrate that removal of cisternal lesions can be both safe and feasible when combining a minimally invasive keyhole procedure with endoscopy. Patients with symptomatic cisternal lesions caused by mass effect (namely, obstructive hydrocephalus or brainstem compression–induced focal deficits) should be considered for this treatment if circumstances permit. Future studies need to be undertaken to assess the efficacy of antiparasitic medications combined with minimally invasive resection for the treatment of cisternal lesions. Furthermore, as experience with endoscopic procedures increases in combination with future technical advances, expiration of nonsymptomatic lesions may be recommended to prevent the late-onset complications of inflammation, arachnoiditis, vasculitis, and ischemia. In applying minimally invasive techniques and endoscopically assisted surgery to the treatment of cisternal NCC, the goal is twofold: 1) to benefit from cyst expiration and 2) to minimize and ultimately eliminate the risks associated with open procedures.

References


Endoscopy for cisternal cysticercal cysts


Manuscript received May 6, 2002. Accepted in final form May 17, 2002.
Address reprint requests to: Marvin Bergsneider, M.D., 100 University of California, Los Angeles Medical Plaza, Suite 219, Los Angeles, California 90024. email: mbergsneider@mednet.ucla.edu.