

Spinal neurocysticercosis

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Worldwide, cysticercosis is the most common parasitic infection of the central nervous system. In endemic regions, the incidence of neurocysticercosis (NCC) approaches 4% of the general population. The disease is predominantly intracranial, the authors of most series generally report the incidence of spinal NCC as only 1.5 to 3% of all cases. Although spinal NCC is relatively rare, it represents a distinct clinical entity that can have devastating consequences for the patient. Because of the limited size of the spinal canal, the mass effect of these lesions is poorly tolerated. Most spinal NCC occurs in the subarachnoid space where mass effect can cause spinal cord compression, although obstruction of cerebrospinal fluid pathways due to scarring of the subarachnoid space can also cause symptoms.

The authors treated six patients with spinal NCC. In five cases the lesions were located in the subarachnoid space, and in one the lesion was intramedullary. All patients with subarachnoid spinal NCC required excision of the symptomatic lesions; in two cases initial medical therapy had failed. The patient with intramedullary spinal NCC experienced mild symptoms and underwent steroid therapy. All patients experienced variably improved outcomes and were eventually ambulatory.

Medical therapy should be carefully considered in selected patients in whom symptoms are stable and nonprogressive. Surgical intervention is required when severe or progressive deficits occur to prevent permanent injury. In some patients recovery may be limited as a result of inflammatory injury to the spinal cord or arachnoidal adhesions.

KEY WORDS • cysticercosis • neurocysticercosis • spinal cord • spinal lesion • parasitic infection

Worldwide, cysticercosis is the most common parasitic infection affecting the CNS.^{6,7,10,12,13,17-19} Neurocysticercosis typically involves the brain parenchyma, intracranial subarachnoid space, or ventricular system and is often self-limited unless hydrocephalus requires surgical intervention.²⁴ Spinal NCC is rare even in endemic regions and may require more aggressive management because of the natural confines of the spinal canal. The location of the mass lesion, its size, and the inflammatory response generated by cyst breakdown are important factors in the management of spinal NCC.^{2,4,15} We review six cases of spinal NCC in which the patients underwent evaluation and treatment, and we present a review of the literature.

CASE REPORTS

Clinical Data

This report represents a retrospective review of individual patients who were treated at UCLA Medical Cen-

ter and affiliated hospitals (Olive View-UCLA, Harbor-UCLA Medical Center, and Cedars-Sinai Medical Center) between 1992 and 2001 (Table 1). In all patients clinical evaluation for intraspinal mass lesions included MR imaging and/or CT scanning of the spinal column and brain. The diagnosis of NCC was made based on neuroimaging and serological studies in selected cases in which diagnosis remained unclear. All patients initially received dexamethasone therapy and selected patients received praziquantel therapy pre- and postoperatively. Decompressive surgery was performed in patients with progressive or persistent severe neurological deficits. Outcome was documented for as long as each patient was available for follow up, which was highly variable (range 6 months-5 years).

Case 1

This 38-year-old Hispanic man with a history of intracranial NCC and hydrocephalus (previously treated by placement of a VP shunt) was evaluated for progressive lower-extremity weakness, unsteady gait, and difficulty initiating micturition over a 1-month period. Muscle strength was Grade 3-4/5 in lower extremities bilaterally with diminished reflexes. He experienced diminished sensation in his right lower extremity, and sphincter tone was

Abbreviations used in this paper: CNS = central nervous system; CSF = cerebrospinal fluid; CT = computerized tomography; MR = magnetic resonance; NCC = neurocysticercosis; UCLA = University of California, Los Angeles; VP = ventriculoperitoneal.

TABLE 1
Summary of data obtained in six patients with NCC*

Factor	1	2	3	4	5	6
age (yrs), sex	38, M	14, F†	36, M	40, M	28, M	80, M
H/O NCC	yes (VP shunt)	no	yes‡	no	yes‡	no
weakness	LE	UE/LE	UE/LE	no	UE/LE	no
tone	–(LE)	–(UE/LE)	+(UE/LE)	normal	+	+
sensory loss	–rt thigh	T4 level	T4–9 level	none	none	T-5 level
bladder dysfunction	yes (resolved)	yes (resolved)	yes (resolved)	no	no	no
lesion location	L2–3	C5–T1	C-5	FM	C-4	T4–5, T7–9
medical therapy	DXM/PRZ	DXM	DXM	DXM/PRZ	DXM	DXM
EM or IM	EM	EM	EM	EM	IM	EM
excision	complete	incomplete	complete	complete	none	complete
intraop US	yes	no	yes	no	no	no
follow up (yrs)	1§	1	0.5§	2	5	1.5
outcome	RTW	I-amb	I-amb	normal**	normal	improved

* ALB = albendazole; DXM = dexamethasone; EM = extramedullary; FM = foramen magnum; I-amb = independently ambulatory; IM = intramedullary; LE = lower extremities; PRZ = praziquantel; RTW = return to work; UE = upper extremities; US = ultrasonography; + increased; – decreased.

† History of sacral myelomeningocele repaired at birth.

‡ Multiple intracranial parenchymal calcifications.

§ Lost to long-term follow-up.

** Hydrocephalus requiring VP shunt placement.

slightly diminished. An MR imaging study of the lumbar spine (Fig. 1 *upper left* and *right* and *lower left*) revealed two large eccentric cystic lesions at L2–3 with diffuse scarring throughout the entire lumbar subarachnoid space. He was initially treated with dexamethasone therapy and began praziquantel therapy when his paraparesis progressed, requiring lumbar laminectomy and excision of two cystic lesions. Intraoperative ultrasonographic localization was required, as was sharp microsurgical dissection because of the severe densely scarred subarachnoid space. Gross examination of the cystic lesion revealed a thin-walled, friable whitish bladder, and no scolices were identified within the cyst. Histological evaluation showed a translucent cyst with an eosinophilic lining and clear fluid with chronic inflammatory cells consistent with cysticercosis. Postoperative MR imaging (Fig. 1 *lower right*) demonstrated the absence of the lesions but dense arachnoidal scarring remained evident. Initially he experienced minimal sensorimotor improvement but was able to ambulate with assistance. He was transferred to rehabilitation, improved over 2 months, and was eventually able to return to work as a gardener within the year.

Case 2

This 14-year-old Hispanic girl in whom a sacral meningocele had been repaired at birth, was transferred to our institution with a 2-month history of neck and upper back pain, a 2-week history of progressive quadriparesis, and an inability to void for 1 day. Neurological examination revealed diminished tone in all extremities with Grade 3/5 power in all extremities and a T-4 sensory level. An MR imaging study revealed a large cystic intradural, extramedullary mass compressing the posterior aspect of the spinal cord from C-5 to T-1. Initially she received intravenous dexamethasone therapy for 1 week, but she required an emergency C5–T1 laminectomy and excision of a large intradural cystic mass because her neurological symptoms worsened. Complete resection of the lesion was not possible because the cyst wall adhered to the

spinal cord. Histological examination of the cyst showed a thin wall containing thick whitish fluid consistent with cysticercosis. Postoperatively her muscle strength improved rapidly to Grade 4/5 in all four extremities, she was able to ambulate using a walker, and she regained baseline urinary function. She was discharged to a rehabilitation hospital. Six months later she could ambulate independently, and her status was unchanged at 1 year.

Case 3

This 36-year-old Hispanic man, in whom a diagnosis of NCC with intracranial calcifications had been previously established, presented with a 1-week history of rapidly progressive spastic quadriparesis. His motor strength was Grade 3-4/5, and he had a sensory level of T-4 on the right and T-9 on the left. Sphincter function was normal. An MR imaging study demonstrated a cystic mass lesion at C-5 with anterolateral compression of the spinal cord with several adjacent subarachnoid septations. The patient underwent urgent decompressive laminectomy and intradural exploration, during which we found dense arachnoidal scarring that precluded visualization of the cyst and required intraoperative ultrasonographic localization. Sharp dissection of the arachnoid was required for cyst removal, ultimately allowing free flow of CSF in the subarachnoid space.

Strength and sensation immediately improved, but on postoperative Day 3 the patient developed severe bilateral lower-extremity weakness. A CT myelography study demonstrated a subarachnoid block at the level of the laminectomy. Reexploration was undertaken. We performed a limited dissection of the thickened arachnoid and a duraplasty to expand the subdural space. Postoperatively his rehabilitation was slow. At the 6-month follow up he was able to walk independently.

Case 4

This 40-year-old Hispanic man presented with headaches, nausea, vomiting, and fever over several weeks.

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Fig. 1. Case 1. Sagittal T₁- (upper left) and T₂-weighted MR images (upper right) and contrast-enhanced axial T₁-weighted MR image (lower left) demonstrating mass lesion compression of the cauda equina at L2–3 with diffuse scarring throughout the subarachnoid space. Sagittal T₁-weighted MR image (lower right) with contrast demonstrating removal of cyst. Arachnoidal scarring persists.

Neurological examination demonstrated normal status except for nuchal rigidity. Magnetic resonance imaging revealed mild hydrocephalus and a cystic lesion at the posterior aspect of the foramen magnum (Fig. 2). Serological studies indicated a diagnosis of NCC, and the patient underwent praziquantel therapy for his symptoms. He experienced worsening headaches, and repeated MR imaging demonstrated worsening ventriculomegaly. He underwent a suboccipital craniotomy, C-1 laminectomy, and intradural exploration to remove numerous cysticercal cysts that densely packed the cisterna magna. Postoperatively his symptoms improved, but he eventually underwent placement of a VP shunt to treat persistent headaches and hydrocephalus. He received praziquantel but could not return to work because of continued headache. Headache remained at 2 year follow-up examination.

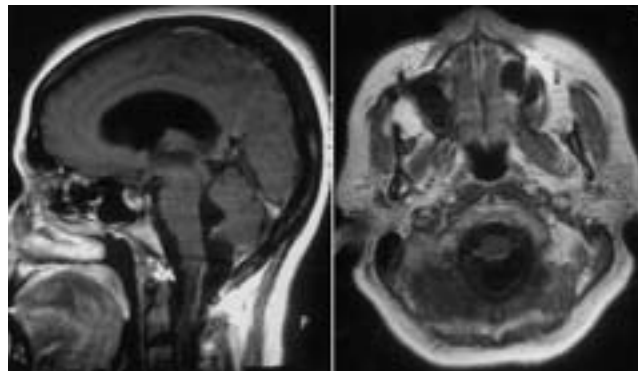


Fig. 2. Case 4. Sagittal (left) and axial (right) T₁-weighted contrast-enhanced MR images demonstrating low-intensity mass occupying the foramen magnum and obstructive hydrocephalus.

Case 5

This 28-year-old Hispanic woman presented with slowly progressive spastic quadriparesis. Her strength (Grade 4/5) was decreased throughout all extremities with increased tone and hyperreflexia. Bladder and bowel function was normal. Magnetic resonance imaging revealed an enhancing intramedullary lesion of the spinal cord at C-1 but no evidence of subarachnoid lesions (Fig. 3). Several intracranial parenchymal calcifications, consistent with NCC, were present. Because of her mild and stable symptoms, she was treated with a course of dexamethasone and closely observed. Her neurological status gradually improved over several months. Without further treatment, she eventually returned to normal activities during the 5-year follow-up period.

Case 6

This 80-year-old Hispanic man presented with a several-month history of upper thoracic back pain that was severely exacerbated by coughing. Subsequently, he developed progressive difficulty with ambulation, but there was no evidence of significant lower-extremity sensory abnormality or bowel/bladder dysfunction. Physical ex-

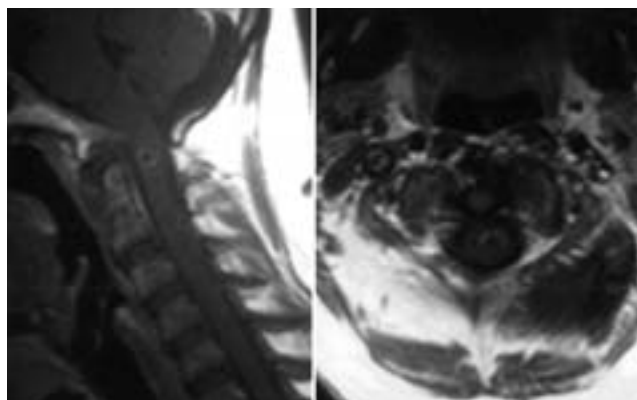


Fig. 3. Case 5. Sagittal (left) and axial (right) T₁-weighted contrast-enhanced MR images revealing an intramedullary lesion at C-1 and no other visible masses.

amination revealed full power in the lower extremities, a T-5 sensory level, and diffuse lower-extremity hyper-reflexia with a spastic gait. An MR imaging study revealed multiple intradural, extramedullary cystic structures displacing the spinal cord anteriorly at the T4-5 and T7-9 levels (Fig. 4 *upper left, upper right, and center*). There was no significant contrast enhancement noted. The patient subsequently underwent T4-5 and T7-9 laminectomies and endoscopically assisted resection of six distinct cystic masses that were easily separated from surrounding arachnoidal adhesions. Pathological evaluation of the cysts showed proteinaceous fluid consistent with spinal cysticercosis. Postoperatively the patient improved clinically. Repeated serial MR imaging revealed successful decompression of the spinal cord, and the patient experienced no recurrence of symptoms during the 18-month follow-up period (Fig. 4 *lower left and lower right*).

DISCUSSION

Cysticercosis infestation of the CNS was first described in humans in 1550 by Paranoli.¹⁶ The parasitic nature of the pork tapeworm, *Taenia soleum*, was recognized in the latter part of the 19th century by Leuckart and Küchenmeister.¹⁶ The life cycle is well known: pigs are the intermediate host and humans are the definitive (or occasionally intermediate) host.^{14,16,22,27} Neurocysticercosis typically results from ingestion of cysticercal eggs in food contaminated by human or porcine feces. Gastric acid releases the larvae from the eggs, which penetrate the intestinal mucosa and gain access to the bloodstream, where most frequently they migrate to muscle, brain, and the eyes.^{14,16} Dissemination in the CNS occurs through small capillaries into the parenchyma or through the choroid plexus into the ventricles, eventually leading to the sub-arachnoid space. The signs and symptoms related to the CNS are usually secondary to mass effect, the inflammatory reaction causing arachnoiditis and basal meningitis, or obstruction of the subarachnoid pathways of the ventricular system.^{1,14,16,18,24}

Cysticercosis is widely endemic in Mexico, Latin America, tropical Africa, India, and Southeast Asia.^{14,16,27} As indicated by autopsy findings, the incidence in these regions can be as high as 4% of the general population;^{10,22} however, in more recent reports from Brazil, as many as 7.3% of hospital admissions were shown to be related to NCC.²⁵ In the United States, the first case of NCC was reported by Walter Dandy in 1927, and there have been more frequent reports of NCC in the Southwestern United States in recent years.^{1,11,22,24,26} McCormick¹¹ reported that only 14 cases of NCC were treated at Los Angeles County Hospital between 1918 and 1965, but that 59 NCC-related admissions were treated between 1978 and 1979. Despite an increasing number of NCC cases overall, the number of spinal NCC cases remains very low.^{13,22,24}

Epidemiology of Spinal Neurocysticercosis

Spinal NCC is rare compared with intracranial NCC involving the brain, basal cisterns, and ventricles. Despite the widespread incidence of NCC in endemic areas, there have been fewer than 200 cases of spinal NCC reported in the world literature.^{2-4,6,9,13} In 1963, Canelas, et al.,³ report-

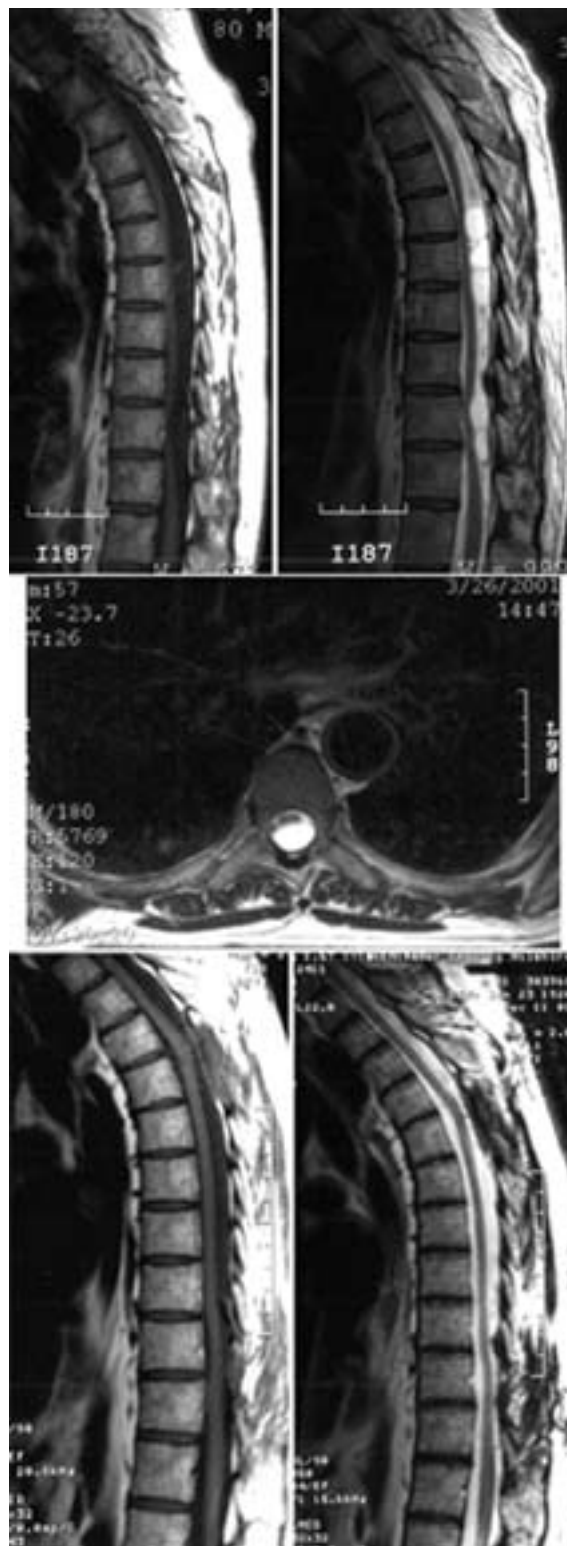


Fig. 4. Sagittal T₁- (*upper left*) and T₂-weighted (*upper right*) MR images demonstrating multiple cystic structures at T4-5 and T8-9 posterior to the spinal cord over multiple thoracic segments. *Center*: Axial T₂-weighted MR revealing a large posterior cyst with compression and anterior displacement of the spinal cord at T4-5. One-year postoperative sagittal T₁-weighted (*lower left*) and T₂-weighted (*lower right*) MR images demonstrating decompression of the spinal cord.

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ed a 2.7% incidence of spinal NCC in 296 cases of NCC. Since that time, others have suggested that the incidence of spinal NCC is as high as 20%; however, an incidence of 1.5 to 3% is most often reported.^{3,4,10,22,23}

Spinal NCC occurs in patients with an established diagnosis of intracranial NCC in approximately 75% of the cases, and isolated cases of spinal NCC are thought to be uncommon.^{4,6,17} The availability as well as improved diagnostic capacity of MR imaging in detecting spinal lesions may eventually indicate that the incidence of spinal NCC is significantly higher than is currently reported, but this has not been established. Some authors have considered the autopsy incidence of spinal NCC to be an underestimate of the true incidence because the spinal canal is not usually examined pathologically and small asymptomatic cysticercal cysts may be easily overlooked.^{3,20}

Subarachnoid and Intramedullary Spinal Neurocysticercosis

Similar to intracranial NCC, spinal NCC lesions can occur in either the subarachnoid space or parenchyma of the spinal cord. Subarachnoid location of spinal NCC occurs most frequently^{4,10,20} in approximately 80% of cases and is thought to result from larval migration through the ventricular system into the spinal subarachnoid space.^{3,20} Intramedullary spinal NCC occurs less frequently in the remaining 20% of cases; however, more cases have recently been reported.^{6,9,13} An intramedullary location is considered to result from direct hemopoietic spread similar to that which occurs in parenchymal intracranial NCC.^{9,20} Extradural spinal NCC is considered exceedingly rare,²⁰ although there have been recent reports of such lesions.¹²

Although there is a high incidence of intracranial NCC lesions, it is unclear why spinal NCC lesions are not found more often. The infrequency of spinal NCC lesions has been considered to be related to CSF reflux at the craniovertebral junction, which propels floating cysts back into the intracranial space rather than the spinal canal.²⁰ Despite these theories, the distribution of NCC occurs equally throughout the spinal canal.⁴ Although cerebral NCC is related to regional blood flow, intramedullary spinal lesions are not clearly related to regional blood flow in the spinal cord. If blood flow was the sole factor in distribution, the expected relative incidence of intramedullary spinal NCC would be 10–15%. The discrepancy between the expected distribution due to blood flow and reported distribution of NCC lesions remains unexplained.

It is important to understand that the location of a subarachnoid spinal cysticercal cyst is not necessarily fixed; migration of the cyst has been demonstrated during myelographic procedures⁷ similar to that within the ventricle.^{1,7,10} Evidence that NCC cyst migration occurs only underscores the importance of timing between neuroimaging and excision; it is necessary to ensure that the lesion will be within the planned surgical field.

Signs and Symptoms

Spinal NCC-related symptoms may depend on several factors including: 1) location (that is, intramedullary compared with subarachnoid); 2) spinal level; 3) lesion size; and 4) the presence (or absence) of inflammation and

arachnoid scarring due to cyst degeneration. The most common clinical signs are myelopathy and progressive weakness, induced by spinal cord and/or cauda equina compression.^{2–4} Small intramedullary lesions often become symptomatic early, whereas extramedullary lesions may become very large and exhibit relatively late and insidious onset of symptoms. This may be particularly true in the lumbar cistern, where these lesions may become quite large before cauda equina symptoms occur, as occurred in our Case 1. The inflammatory process that occurs when the parasite dies may be the most problematic mechanism of symptom generation, whereas live cysts are thought to cause less inflammation and may be more successfully treated by excision.^{8,10,19}

Diagnostic Evaluation

Magnetic resonance imaging is the diagnostic study of choice for evaluating spinal NCC because it provides non-invasive multiplanar images of a large area of the spinal cord, cauda equina, and any potential intraspinal pathological entity. On T₁-weighted MR images the cyst wall but not the cyst itself can be demonstrated because the contents are often isointense to CSF.^{26,28} Magnetic resonance imaging with T₂-weighted sequences is particularly useful in demonstrating these lesions; their signal intensity is increased within the cyst presumably because of a higher protein content. Additionally T₂-weighted MR imaging can also demonstrate pericystic edema in the spinal cord parenchyma.^{10,26,28} Occasionally the actual scolex is visualized on MR images as a mural nodule.^{10,28} Myelography and postmyelography CT can be useful for detecting small subarachnoid spinal NCC lesions;^{4,6,13,17} however, myelography may have limited value when there is arachnoidal scarring and obstruction of CSF pathways. Computerized tomography is better than MR imaging for visualizing calcifications associated with degenerated NCC cysts, but this is much more pertinent to brain parenchymal disease than the spinal form.

Serological studies are highly sensitive and specific for confirming the diagnosis of NCC. Rosas²¹ has reported that in using the enzyme-linked immunosorbent assay, the sensitivity is 87% and the specificity is 97% for CSF, whereas in serological studies the sensitivity is only 50% and the specificity is only 70%.

Management of Spinal Neurocysticercosis

In the treatment of brain and meningeal NCC, the indications and efficacy of medical therapy with praziquantel or albendazole continue to evolve. Some authors have reported improved outcome when the aforementioned medical therapy was compared with steroid therapy alone.²⁵ Parenchymal NCC is considered to be most responsive to pharmacological intervention;⁸ the efficacy remains unclear when treating subarachnoid, cisternal, or intraventricular cysticercosis.^{10,18,19,26} Albendazole may be more effective than praziquantel;^{10,19,25} however, limited study controls and heterogeneity of patient groups make outcomes difficult to interpret. Garg, et al.,⁵ reported two patients with intramedullary NCC in whom improvement was related to albendazole: in one case the patient regained the ability to walk independently and in the other the patient was able to stand independently.

The inflammatory reaction associated with cyst degeneration in the brain may cause increased intracranial pressure due to obstruction of subarachnoid pathways.^{10,25} If medical therapy does indeed cause increased inflammation leading to clinical deterioration as the NCC cysts die, then spinal NCC may require surgical therapy as a first-line treatment because of the confines of the spinal canal. Steroid therapy may ameliorate this by reducing the inflammatory response. Some authors have recommended prescribing medical therapy in all patients with NCC because the infection is systemic with repeated focal manifestations.^{12,13} In patients with localized disease and inflammation-induced symptoms/signs, steroid therapy alone may be adequate treatment, as it was in our Case 5. For these reasons, medical treatment of spinal NCC appears to be a less viable option because patients with these lesions often present with progressive neurological deficits requiring prompt surgical treatment, as was evident in our cases.

Surgical Therapy

Surgical treatment is indicated in cases of spinal NCC in which patients experience severe and progressive neurological dysfunction regardless of whether medical therapy has been attempted. Excision of extramedullary lesions is often difficult because of the arachnoidal scarring secondary to cyst degeneration, but sharp dissection, gentle irrigation, and Valsalva maneuvers may assist in extirpating adherent cysts.^{1,4,17,24} The inflammatory process may be so severe that visualization and localization require ultrasonography to assist in excision,^{4,24} and some cysts cannot be readily or completely resected because of this inflammatory response and their adherence to the spinal cord.^{4,24} Subarachnoid scarring-induced CSF flow obstruction can cause part or all of the symptoms; it must be treated with duraplasty to reestablish CSF flow, which was required in our Case 3. Although resection of intramedullary cysts has the inherent risks associated with surgical treatment of the spinal cord, it is similarly indicated in patients with progressive neurological deterioration. Excision of intramedullary NCC lesions has been described as being possible after myelotomy or requiring microsurgical dissection from the parenchyma prior to removal.¹³

Clinical Outcomes of Spinal Neurocysticercosis

Outcomes in patients with spinal NCC are related to the following factors: 1) location (that is, intramedullary or extramedullary; cauda equina or spinal cord levels); 2) the severity of inflammation (arachnoid scarring or parenchymal injury); and 3) chronicity of symptoms/time to treatment. Patients with intramedullary spinal NCC lesions appear to experience poorer outcomes, which is ascribed to both the intrinsic injury and the inflammatory injuries associated with cyst degeneration.^{2,13,15,20} Acute neurological deterioration secondary to mass effect is often resolved after prompt excision of the lesion, as illustrated by the results obtained in our Cases 1 and 2, but patients with chronic arachnoidal scarring or spinal cord inflammation may suffer poorer outcomes, as was the case in two of our patients (Cases 3 and 4), despite surgical intervention.^{2,4} Patients with NCC-induced extrinsic spinal cord and

cauda equina compression in the absence of severe arachnoidal scarring appear to have better outcomes than those with intramedullary lesions, as illustrated in Case 6. Reporting small series, several authors have indicated that patients who suffer rapid subarachnoid cyst-induced deterioration may improve and return to full activity, but nearly 50% experience some continued or recurrent symptoms attributed to arachnoidal inflammation.^{4,13}

CONCLUSIONS

Spinal NCC is very rare compared with intracranial NCC, which has a relatively high incidence in endemic regions of the world. Spinal NCC should be considered in the differential diagnosis in high-risk populations in which patients present with new symptoms suggestive of a spinal mass lesion. Subarachnoid spinal NCC occurs in 80% of cases, and 20% are intramedullary lesions. Signs and symptoms may include myelopathy, radiculopathy, or cauda equina syndrome, depending on location of the cyst. The inflammatory arachnoiditis resulting from cyst degeneration may severely limit recovery despite successful excision of the lesion. Medical therapy may be considered in patients with stable symptoms but is unlikely to alleviate acute and progressive spinal NCC symptoms, which should be addressed surgically.

Acknowledgment

The authors would like to thank Ms. Samantha Phu for her assistance in preparing this manuscript.

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Manuscript received April 30, 2002.

Accepted in final form May 17, 2002.

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