Parasitic infections of the spine: case series and review of the literature

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OBJECTIVE Although parasitic infections are endemic to parts of the developing world and are more common in areas with developing economies and poor sanitary conditions, rare cases may occur in developed regions of the world.

METHODS Articles eligible for the authors' literature review were initially searched using PubMed with the phrases "parasitic infections" and "spine." After the authors developed a list of parasites associated with spinal cord infections from the initial search, they expanded it to include individual diagnoses, using search terms including "neurocysticercosis," "schistosomiasis," "echinococcosis," and "toxoplasmosis."

RESULTS Two recent cases of parasitic spinal infections from the authors' institution are included.

CONCLUSIONS Key findings on imaging modalities, laboratory studies suggestive of parasitic infection, and most importantly a thorough patient history are required to correctly diagnose parasitic spinal infections.

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KEYWORDS parasite; spinal infection; neurocysticercosis; schistosomiasis; echinococcosis; toxoplasmosis

ALTHOUGH parasitic infections are more common worldwide in areas with developing economies and poor sanitary conditions, rare cases may occur in developed regions of the world. There are a number of rare parasitic diseases that may involve the CNS, causing patients to present with common symptoms such as seizures, motor or sensory deficits, and pain. It is imperative that clinicians develop a broad differential diagnosis when evaluating these patients, even when clinical symptoms and workup may direct one toward an inflammatory, neoplastic, or degenerative process. Patient history and demographics are vital to the diagnosis of these diseases.

A number of these parasitic diseases affecting the CNS may involve the spine. Patients may present with typical symptoms such as back pain, numbness, weakness, or bowel/bladder incontinence, leading the clinician to order relevant imaging of the CNS. In cases of parasitic infection, there is seldom a diagnosis made even after imaging identifies the underlying lesion. These lesions can easily be mistaken for other more common surgically treatable

pathologies. Therefore, thorough understanding of the presentation and guidelines for treatment of these rare parasitic infections is necessary, especially as the population of the US diversifies and parasitic infections are identified more often. In this case discussion and review of the literature, we present the most common parasitic spinal infections, their clinical presentation, risk factors, and the most up-to-date management guidelines.

Methods

We reviewed 2 unique cases of parasitic spinal infections at our institution and the relevant imaging. Articles eligible for our literature review were initially searched using PubMed with the phrases "parasitic infections" and "spine." After we developed a list of parasites associated with spinal cord infections from our initial search, we expanded it to include individual diagnoses, using search terms including "neurocysticercosis," "schistosomiasis," "echinococcosis," and "toxoplasmosis." All articles within

ABBREVIATIONS ELISA = enzyme-linked immunosorbent assay; IHA = indirect hemagglutination assay.

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TABLE 1. General characteristics of spinal parasitic infections

Name of Disease	Pathogen	Transmission	Signs & Symptoms	Imaging	Diagnosis	Treatment
Neurocysticercosis	<i>T. solium</i>	Ingestion of <i>T. solium</i> eggs ¹⁷	Brain cysts (4 stages): vesicular, colloidal, nodular/granular, & calcified granulars; ¹³ seizures/epilepsy; headaches; focal neurological deficits ¹⁷	1) Vesicular stage: well-defined scolex; ^{13,17} 2) colloidal stage: ring enhancement, loss of scolex, edema; ^{13,17} 3) nodular/granular stage: decreased enhancement & edema, initiation of calcification, no cystic component; ^{13,17} 4) calcified stage: calcified lesions ⁵⁰	Epidemiological factors, neuroimaging, serological tests, funduscopy, histology ^{15,17}	Antiparasitic therapy (albendazole, praziquantel) & corticosteroids (not recommended in patients w/ calcified lesions); ^{24,58} antiepileptics ⁵⁸
Schistosomiasis	<i>S. mansoni</i> , <i>S. haematobium</i> , <i>S. japonicum</i>	Penetration of skin by schistosomal larvae ¹⁷	Muscle weakness, asymmetrical sensorimotor abnormalities, altered mental status, high eosinophil count, lumbar pain, radiculopathy ^{17,23}	MRI—abnormal T1WI & T2WI signals, heterogeneous pattern of enhancement, spinal cord compression, enlarged spinal cord ^{17,49}	Kato-Katz thick-smear, ELISA, IHA, or immunofluorescence, neuroimaging ⁴⁸	Praziquantel & corticosteroids, artemether (prophylaxis) ⁴⁸
Echinococcosis	<i>E. granulosus</i>	Ingestion of <i>Echinococcus</i> eggs ¹⁷	Long history of back pain, neurological deficits, spinal compression syndrome ^{28,45}	Well-defined multiloculated osteolytic lesion; ⁵⁹ T2WI showing cystic lesions w/ high signal intensity; hypointense lesions on T1WI ⁵⁹	Neurological examination, neuroimaging, serological tests ^{17,28}	Surgery w/ concomitant antiparasitic therapy (albendazole, mebendazole) ⁴⁰
Toxoplasmosis	<i>T. gondii</i>	Ingestion of cysts in undercooked meat or of oocysts in contaminated food & water; spinal toxoplasmosis typically only seen in immunocompromised patients ³¹	Acute-onset paraparesis, sensory & bladder dysfunction, fever ^{3,26}	Enhanced intramedullary lesions ⁴⁷	Serum & CSF cytology & immunological studies, neuroimaging ²⁶	Oral pyrimethamine & sulfadiazine, steroids (requires further investigation) ^{26,47}

T1WI = T1-weighted imaging; T2WI = T2-weighted imaging.

these searches were screened, and we included articles focusing on the parasitic infections specifically affecting the spinal cord and spine. The majority of the studies were case reports (Tables 1–5).

Case Reports

Case 1

This patient was a 49-year-old man with a past medical history of tuberculosis who presented to our institution with the chief complaint of sensory loss in his arms and legs. The patient was originally from Guatemala and had resided in the US for approximately 3 years. On initial neurological examination, he had decreased sensation to light touch in the upper extremities, worse on the right side. His motor function was preserved. He was also found to have marked impairment in proprioception. MRI sequences of the cervical spine demonstrated large, cystic, enhancing lesions, most prominent dorsal to the spinal cord and causing significant compression. The most prominent lesion spanned the posterior fossa through C2, and an additional lesion was causing stenosis at C6–7. Additional imaging demonstrated multiple enhancing lesions

as well as calcified nodules throughout the brain. MRI of the thoracic and lumbar spine demonstrated diffuse meningeal enhancement as well as several more enhancing lesions. The patient was started on albendazole as well as steroids.

Due to his neurological deficit, the patient underwent a suboccipital craniectomy and C1 laminectomy for resection of the intradural extramedullary lesions. Multiple large intradural cysts were encountered and removed. Both imaging and pathology were consistent with neurocysticercosis (Figs. 1 and 2). Postoperatively, the patient did well and continued to demonstrate improvement in his sensory deficits on follow-up. He was continued on albendazole and his steroids were tapered off.

Case 2

This patient was a 38-year-old man who had a 1-year history of low-back pain. He was known to have a pelvic mass of unknown origin, which was being monitored by his primary care provider. He presented to our institution with a 1-week history of bowel incontinence as well as subjective lower-extremity weakness. He denied urinary incontinence. On neurological examination, the patient

TABLE 2. Case reports and larger case series of neurocysticercosis

Case Reports	Pt Age (yrs), Sex	Symptoms	Imaging	Biopsy Findings	Treatment	Improvement of Symptoms?
Sheehan et al., 2002	16, F	Progressive bilat hand pares-thesias, decreased respira-tory rate	MRI showed intraparenchymal lesion, cystic in nature w/ rim enhancement, at C1–2 w/ focal cord enlargement & signs of edematous change	Cyst wall remnants from intramedul-lary cysticercosis, reactive gliosis	Resection, praziqu-antel, & steroids	Yes
Chaurasia et al., 2015	35, M	Back pain, unilat rt lower-ex-tremity weakness, decreased sensation to pain & temp on lt, decreased sensation to position & vibration on rt (clinical Brown-Séquard), uri-nary retention, constipation	MRI showed ring-shaped cysticercosis lesion w/ eccentric dot (scolex of larvae) at T11	No biopsy	Albendazole & pred-nisolone	Yes
Torabi et al., 2004	35, M	Low-back pain; progressive rt leg weakness; decreased sensation to light touch, vibration, & position in rt leg; decreased sensation to temp in lt leg; urinary incontinence	MRI showed abnormal intramedullary enhancement on lt C5 & rt T4, w/ abnormal signal in T5–9, conus medullaris, & thecal sac	No biopsy	Albendazole & dexa-methasone	Yes
Larger Case Series	No. of Pts	Significant Findings				
Colli et al., 2002	12	In 9 of 12 pts cysticercosis was associated w/ hydrocephalus, & each of these pts developed nerve root compression symptoms 7–48 mos later. Prognosis was worse in pts w/ associated arachnoiditis & spinal cord compression.				
Alsina et al., 2002	6	Subarachnoid spinal neurocysticercosis occurred in 5 pts & intramedullary neurocysticercosis occurred in 1 pt. All pts were eventu-ally ambulatory after treatment. Only the pt w/ intramedullary neurocysticercosis was managed w/ medical therapy alone.				
Del Brutto & Garcia, 2013	43	All pts presented w/ some degree of transverse myelopathy. On MRI, the scolex of the parasite was only visualized in 16 pts. Of the 20 pts treated w/ surgery, 12 fully recovered, whereas all 13 medically treated pts fully recovered.				

Pt = patient; temp = temperature.

had intact motor strength in the arms and legs and decreased sensation in the plantar aspect of the right foot. MRI sequences of the patient's lumbar spine demonstrated a complex-appearing polycystic mass extending from the pelvis through two of the sacral neural foramina into the epidural space from the L5–S1 junction down to the bottom of the sacrum. Severe mass effect and obliteration of the foraminal contents on the right side at S1–2 and S2–3 were demonstrated (Fig. 3).

The patient underwent bilateral L5 laminotomies and S1–2 laminectomies for resection of the epidural mass. According to pathological findings, the epidural mass was suggestive of parasitic infection, specifically *Echinococcus*. The patient was started on albendazole and corticosteroids for treatment, and his right lower-extremity pares-thesias and sensation improved dramatically.

Discussion

Neurocysticercosis

Cysticercosis, the most common parasitic infection of the CNS, is caused by *Taenia solium*. The disease occurs secondary to the ingestion of embryonated parasite eggs. Once ingested, the parasite traverses through the small bowel into the bloodstream to reach a variety

of sites, including the skeletal muscles, eyes, and neural structures. This parasite affects approximately 50 million people worldwide and carries a prevalence of 3%–6%.¹⁷ Although the parasite mainly affects endemic regions, it has become more prevalent in the US due to the immigration of patients from highly affected regions.¹⁶ Intracranial involvement is more common with this pathology; spinal cysticercosis has an incidence of only 1.5%–3%.

Spinal neurocysticercosis involving the spinal cord is extremely uncommon—it is reported to be seen in only 1%–6% of patients diagnosed with neurocysticercosis.⁵⁵ Leptomeningeal involvement is relatively more common; it is found approximately 6–8 times more often than the intramedullary form.¹⁷ The intramedullary form occurs secondary to hematogenous spread, whereas the intradural-extramedullary lesions are thought to be “drop lesions” that spread from the intracranial space. Similar to neoplastic lesions, neurocysticercosis lesions may be found in the vertebral bodies, in epidural/subdural/subarachnoid spaces, and within the spinal cord itself (intramedullary). Due to the mass effect and limited space within the canal relative to the intracranial space, spinal cysticercosis may be more likely to result in neurological compromise. Neurological deficits occur secondary to mass effect from the cysts as well as an inflammatory reaction following

TABLE 3. Case reports and larger case series of neuroschistosomiasis

Case Reports	Pt Age (yrs), Sex	Symptoms	Imaging	Biopsy Findings	Treatment	Improvement of Symptoms?
Ueki et al., 1995	34, M	Urinary retention, low-back pain, progressive spastic paraparesis, decreased sensation below T10 level	MRI showed enlarged spinal cord below T9 w/ spotty enhancement	<i>S. mansoni</i> ova w/ granulomatous inflammation	Resection, praziquantel, & dexamethasone	Yes
Palin et al., 2015	20, F	Progressive low-back pain radiating to both legs, lower leg weakness, urinary retention	MRI showed an edematous, expanded conus w/ enhancement from T10–11 to L1–2	Chronic granulomatous inflammation w/ positive <i>Schistosoma</i> serology	Resection, praziquantel, & methylprednisolone, followed by prednisone	Yes
Odeku et al., 1968	13, M	Progressive low-back pain, bilat leg weakness, urinary hesitancy		Schistosomal granuloma w/ <i>S. hematobium</i> ova	Resection, niridazole, promethazine, high fluid intake	Yes
Kamel et al., 2005	62, M	Progressing lower-extremity myelopathy	MRI showed diffuse patchy signal change in lower thoracic cord associated w/ spinal cord swelling	No biopsy; serum schistosomal ELISA was positive	Praziquantel & steroids	Yes
Herskowitz, 1972	29, M	Back pain, leg weakness, difficulty in urination, constipation		Focal areas of necrosis w/ granulomatous reaction enclosing <i>S. mansoni</i> ova	Resection, fuadin, & Decadron	Yes
Larger Case Series	No. of Pts	Significant Findings				
Ferrari et al., 2011 ²²	4	Immune complexes containing soluble egg antigen of <i>S. mansoni</i> were found in the CSF of 4 pts w/ spinal cord schistosomiasis, suggesting an inflammatory disease process.				
Wan et al., 2006	10	10 adult pts from a <i>Schistosoma</i> -endemic area presented w/ progressive lower-extremity weakness, along w/ bowel & bladder dysfunction, & lesions were misdiagnosed as tumor. Pathology later revealed conus medullaris schistosomiasis, a form of ectopic schistosomiasis. Serological testing was positive for <i>Schistosoma</i> IgG in all cases.				
Silva et al., 2004	16	16 adult pts from a <i>Schistosoma</i> -endemic area w/ known schistosomal myeloradiculopathy w/ symptoms including lower-extremity weakness/anesthesia/pain, bladder incontinence, &/or sexual impotence were treated w/ praziquantel, methylprednisolone, & prednisone, & were shown to have significant improvement of neurological symptoms.				
Jiang et al., 2008	4	4 pts w/ acute progression of motor, sensory, & autonomic dysfunctions were found to have spinal cord schistosomiasis secondary to <i>S. japonicum</i> (less frequent). CSF samples from all pts were ELISA positive for <i>S. japonicum</i> . Resection & administration of praziquantel & steroids were both required for adequate treatment. All pts had improvement of symptoms.				

treatment. In addition, patients may present with a variety of symptoms ranging from expected clinical findings concordant with lesion location to those that are less common, such as Brown-Séquard syndrome.¹⁰

Neurocysticercosis typically occurs in 4 stages. The vesicular stage is first, with the presence of a cyst and scolex. The next stage (colloidal) demonstrates ring enhancement and edema. In the third stage (nodular-granular) there is decreased enhancement and edema. During the nodular-granular stage calcification of the lesions begins. The fourth and final stage is called the calcified stage, and it is during this stage that CT/MRI sequences will demonstrate calcification.¹⁷ The best imaging modality is MRI with gadolinium because it will demonstrate mass effect, edema, and enhancement as well as the intensity of the cystic fluid. In addition, high-resolution T2-weighted sequences (3D constructive interference in steady state [3D-CISS]) can demonstrate the cyst and scolex. Subarachnoid cysts can be delineated using MR myelography. In cases of intramedullary involvement, it is extremely difficult

to differentiate neurocysticercosis from other vascular, inflammatory, demyelinating, or neoplastic pathologies without additional information, such as the presence of other lesions in the intracranial space.

Treatment for patients who are asymptomatic typically involves an antiparasitic agent, usually albendazole, combined with an antiinflammatory medication, typically corticosteroids, to reduce inflammation due to larval death.²⁴ Surgical intervention is reserved for patients presenting with mass lesions causing neurological deficits. Spinal lesions such as intramedullary lesions are rarely an indication for surgery. Only those lesions that are accessible by using a low-morbidity approach should undergo resection.

Neuroschistosomiasis

Schistosomiasis is an infection caused by blood-dwelling platyhelminths (flatworms) from the genus *Schistosoma*, which affects more than 230 million people in 74 countries across Africa, Asia, and the Americas.^{9,52} Incidence of this disease is generally found in endemic areas,

TABLE 4. Case reports of toxoplasmosis

Case Reports	Pt Age (yrs), Sex	Symptoms	Imaging	Biopsy Findings	Treatment	Improvement of Symptoms?
Resnick et al., 1995	45, M	Lower-extremity weakness & coordination difficulty, urinary retention	MRI of spine showed long, homogeneously enhancing intramedullary lesion at T4, w/ surrounding edema	Profuse acute & chronic inflammation, <i>Toxoplasma</i> tachyzoites	Anti- <i>Toxoplasma</i> chemotherapy	No
Garcia-Gubern et al., 2010	40, M	Flaccid paralysis of both legs & decreased sensation to pain, touch, temp, proprioception, & vibration	Spinal MRI showed diffuse abnormal hyperintense swelling; brain MRI showed multiple bilat ring-enhancing intraaxial lesions	No biopsy; anti- <i>Toxoplasma</i> IgG immune titer was positive, positive for HIV	Sulfadiazine, pyrimethamine, folinic acid, HAART for HIV, dexamethasone, & methylprednisolone	Yes
García-García et al., 2015	48, M	Dysarthria, urinary retention, rt arm weakness, decreased sensation to temp & pain	T2 MRI of the spine showed diffuse high signal from C4 to T10, w/ enlargement at cervical level; T1 MRI showed a fusiform intramedullary enhancing lesion btwn C5 & C6; brain MRI showed bilat ring-enhancing lesions	Positive for HIV	Antituberculosis drugs, sulfadiazine, pyrimethamine, & dexamethasone	Yes
Kung et al., 2011	34, M	Bilat lower-extremity weakness, sensory level at L4, constipation	Expansile intramedullary enhancing lesion at T11–12	<i>T. gondii</i> cysts	Resection, sulfadiazine, pyrimethamine, dexamethasone, HAART	Yes
Rodríguez et al., 2013	40, M	Lumbar back pain	Expansile medullary enhancing lesion at T10–12	<i>T. gondii</i> tachyzoites	TMP-SMX, clindamycin, steroids (unspecified)	Yes

HAART = highly active antiretroviral therapy; TMP-SMX = trimethoprim-sulfamethoxazole.

but it has also been reported in Western countries due to immigration and tourism. Approximately 20 million people progress to develop severe disease, including infection within the CNS.¹⁷

There are 3 main organisms that are known to infect humans—*Schistosoma japonicum*, *S. mansoni*, and *S. hematobium*. Spinal cord lesions are often caused by infection from *S. mansoni* and *S. hematobium*, whereas *S. japonica* is responsible for most cases of cerebral schistosomiasis.⁵² There have, however, been some cases of *S. japonica* also leading to spinal infections.³³

Initial transmission of these trematodes is from freshwater snails, which act as intermediate hosts and release infective cercaria into the water, which can then penetrate through human skin. Once inside the body, the cercaria transform into schistosomulum and migrate to the lungs via the lymphatic system and blood circulation; there they mature and then enter into portal circulation to carry out the remainder of their life cycle.⁹ Infection of the CNS is believed to be by either distribution of ova through venous shunts or retrograde migration of adult worms from the abdominal veins to the Batson venous plexus.^{9,17,52} The worms and ova travel through the valveless Batson plexus and into the venous system of the spinal cord. When ova are deposited within the spinal cord, there is an inflammatory response from the host, which leads to many of the neurological symptoms associated with this advanced stage of schistosomiasis. In more severe cases, inflammatory processes can lead to space-occupying granulomatous masses and necrosis of CNS tissue. Ferrari et al.

found *S. mansoni* antigen-containing immune complexes within the CSF in all 4 of their patients with known spinal neuroschistosomiasis.²²

Clinically, spinal schistosomiasis tends to present acutely or subacutely and most often involves the lower spinal cord.²³ One of the earliest signs can be low-back pain with radiation down to the lower extremities. Additional associated symptoms include lower-extremity weakness and paresthesias, bladder dysfunction, deep tendon reflex abnormalities, constipation, and sexual impotence.

The disease can present as acute myelopathy, conus medullaris syndrome, or acute/subacute lower-limb myeloradiculopathy.⁹ The medullary form, which involves the spinal cord predominantly, usually has a fast course and leads to severe weakness and a symmetrical distribution of symptoms.²³ Conus medullaris syndrome develops over a slower course, has less severe symptoms, and is often asymmetrical in distribution. The myeloradiculopathy form is the most common presentation.

MRI is the imaging modality of choice to help diagnose spinal cord schistosomiasis. A common finding that can be seen is enlargement of the spinal cord, specifically in the lower spinal cord and conus medullaris region.^{23,43,50,53,56} This is due to intramedullary granuloma formation. Saleem et al. noted moderate expansion of distal spinal cord in all 8 of their patients presenting with spinal cord schistosomiasis.⁵⁰ Silva and colleagues reported this finding in 62.5% of patients.⁵³ Another common finding is thickened cauda equina roots with heterogeneous contrast enhancement.^{2,23}

TABLE 5. Case reports and larger case series of spinal hydatid disease

Case Reports	Pt Age (yrs), Sex	Symptoms	Imaging	Biopsy Findings	Treatment	Improvement of Symptoms?
Ashraf et al., 2013	65, M	Lumbar back pain, incontinence, decreased sensation bilaterally in saddle distribution	Multiple loculated cystic swellings in lt paraspinous area at S2	No biopsy	Preop albendazole, excision, postop albendazole & praziquantel	Yes
Kaen et al., 2009	59, M	Thoracic back pain, bilat lower-extremity weakness, numbness below T6	MRI detected clusters of multiloculated cysts at T6 & at T10–12	No biopsy	Excision, postop albendazole, reop for recurrence of symptoms	No
Kotil et al., 2010	30, F	Lumbar back pain, rt sciatic pain, difficulty ambulating	T1 MRI demonstrated hypointense cystic lesion in L4–5 region; T2 MRI demonstrated hyperintense lesion	No biopsy	Albendazole	Yes
El-On et al., 2003	53, M	Back pain, difficulty ambulating	MRI demonstrated destruction of L4 & cystic lesions in rt iliopsoas muscle	Protoscolices demonstrated microscopically from sample acquired from CT-guided aspiration	Preop albendazole, excision, continued albendazole postop, repeat surgery after neurological deterioration, combination albendazole & praziquantel	No
Larger Case Series	No. of Pts	Significant Findings				
Prabhakar et al., 2005	4	4 pts w/ persistent back pain & paraplegia were found to have spinal hydatid disease. Hematological studies were initially inconclusive, & all pts underwent excision after imaging data suggested hydatid disease. 2 pts required repeat surgery due to symptomatic recurrence.				
Hamdan, 2012	9	9 pts w/ back pain, paraparesis, & varying degrees of urinary incontinence were found to have spinal hydatid disease. 8 of 9 pts had bone involvement, & the pt w/o bone involvement was shown to have a dumbbell cyst & recovered fully w/o recurrence. The other 8 required repeat surgery because of neurological deterioration following initial surgery. All pts received albendazole & praziquantel.				

Imaging findings may give a hint regarding neuroschistosomiasis. However, further studies must be done before the diagnosis can be confirmed. The presence of ova in the stool or urine or of adult worms in a rectal biopsy specimen is reported in 40% of acute neuroschistosomiasis cases.⁹ CSF analysis may show eosinophils, lymphocytic pleocytosis, increased protein concentration, and increased IgG index. The most reliable immunological method for diagnosis is the enzyme-linked immunosorbent assay (ELISA), with 50% sensitivity and 95% specificity. Indirect hemagglutination assay (IHA) tests have sensitivities ranging from 70% to 90%, and the combination of both immunological tests has a sensitivity of 90% and specificity of 93%.⁴⁸

However, the most definite method of diagnosis is tissue biopsy via surgery.⁵⁶ This is an invasive technique but may be necessary because the presence of schistosomiasis infection on noninvasive tests can be coincidental if the patient lives in an endemic area. A tissue biopsy of a granuloma would show schistosome ova surrounded by necrosis, inflammatory reaction, and demyelination.⁹

There are two pharmaceutical treatment options for spinal cord schistosomiasis: schistosomicidal drugs, such as praziquantel, and steroids.^{9,48} Praziquantel is the drug of choice for treating schistosomiasis and works directly

against adult schistosome worms.¹⁸ The cure rate associated with this drug is approximately 60% but can be as high as 85%–90%. Steroids work by reducing the inflammatory process that results from ova invasion within the spinal cord.⁵⁶ In addition, surgical removal of granuloma or decompressive laminectomy may also be warranted for symptomatic relief, especially in cases of severe spinal cord compression.

Toxoplasmosis

Toxoplasmosis is the most common opportunistic CNS infection affecting patients with AIDS. The disease is caused by *Toxoplasma gondii*, which is an obligate intracellular protozoan parasite.⁵² Approximately 500 million people are infected globally, with the highest incidences being in France and Central America and as high as 17%–35% in the US.

The parasite affects two main hosts—cats and humans. It undergoes its sexual cycle within the feline small intestine, and oocysts are then released into water and soil via feces.²⁵ Humans are infected after ingesting oocysts through undercooked meats, contact with cats, or contaminated vegetables. Once within the human intestine, oocysts release sporozoites or bradyzoites into the lumen, where they transform and enter into blood and lymphatic

FIG. 1. Case 1. Neurocysticercosis of the cervical spine demonstrated on MRI sequences. **A:** T2-weighted sagittal image demonstrating cysts in the posterior fossa extending down into the cervical region. **B:** T1-weighted sagittal image with gadolinium showing the cysts that did not enhance. **C:** T2-weighted axial image at C1 demonstrating the cystic lesions dorsal to the spinal cord. **D:** T1-weighted axial image with gadolinium at C1. **E:** T2-weighted axial image at the base of the posterior fossa.

circulation. They can then reach a number of target sites of infection, one of them being the CNS.

Initial infection can often present with mild lymphadenopathy or may also be asymptomatic.³⁷ The infection becomes reactivated in the setting of severe immunosuppression with CD4+ lymphocyte counts less than 200 cells/ml²—hence its strong association with AIDS.^{37,47} Toxoplasmic encephalitis is a well-studied and -observed syndrome in the setting of immunosuppression. However, spinal cord involvement is not as common a presentation. In addition, infection of the spinal cord is seldom seen alone and is often associated with intracranial involvement. The most common finding in spinal cord toxoplasmosis is vacuolar myelopathy.

García-García et al. found 26 cases of HIV/AIDS-related spinal cord toxoplasmosis in their literature review.²⁵ The most common presenting symptoms were extremity

weakness, sensory loss, incontinence, and altered deep tendon reflexes.³⁷ Although spinal cord toxoplasmosis is not a common presentation, it should be suspected in immunodeficient individuals presenting with acute or subacute myelopathy.

Once again, MRI with contrast is the optimal imaging modality for visualizing infectious lesions.²⁵ Lesions will present as hyperintense on T2-weighted or with postcontrast enhancement on T1-weighted sequences. Localized intramedullary ring-enhancing lesions are a common MRI finding associated with toxoplasmosis.²⁶ A normal spinal cord in the presence of abnormal signal can hint at a vacuolar myelopathy, whereas if there is enlargement of the spinal cord, one should consider *Toxoplasma* myelitis.

In addition to MRI, CSF cytology and immunological antibody tests are also valuable diagnostic tools. In fact, they are the gold standard for detecting infectious mi-

FIG. 2. Case 1. Intraoperative images of a cysticercus, which was identified and subsequently removed via a suboccipital surgical approach.

FIG. 3. Case 2. Echinococcosis of the sacral spine demonstrated on MRI sequences. **A:** T1-weighted sagittal image with gadolinium demonstrating cystic lesions at the sacral region that do not enhance and are isointense when compared to the thecal sac. **B:** T2-weighted sagittal image demonstrating cysts that extend into the sacral region. **C:** T2-weighted axial image demonstrating the cystic lesions at S1 causing mass effect on the thecal sac and traversing roots. **D:** T1-weighted axial image with gadolinium demonstrating the cysts that did not enhance. **E:** T2-weighted axial image demonstrating cysts extending into sacral foramina.

croorganisms.²⁶ Analysis of CSF can show a moderately elevated protein level up to 1000 mg/dl, normal glucose, and mild mononuclear pleocytosis.²⁵ Elevated CSF and serum *Toxoplasma* IgG and IgM levels can also help with the diagnosis. Tissue biopsy may show the presence of bradyzoites or tachyzoites. However, tissue biopsy has been associated with significant morbidity and mortality, and therefore noninvasive testing is recommended first.³ Open spinal cord biopsy should only be performed in the setting of acute decline in function or failure to respond to treatments.⁴⁶

There is not much literature describing a treatment regimen specific to spinal cord toxoplasmosis. Therefore, the same treatment used for toxoplasmic encephalitis is used for spinal cord involvement.³ The first-line treatment of choice is a combination of pyrimethamine and sulfadiazine with folinic acid. Trimethoprim-sulfamethoxazole is also an effective therapy option. Steroids have also been used, with success, for treatment of symptoms.^{25,47} There is no well-defined role for surgical intervention in these cases.

Echinococcal Disease

The two most common causative pathogens of echinococcal disease are *Echinococcus granulosus* and *E. multilocularis*. *Echinococcus granulosus*, also known as the dog tapeworm, is transmitted to humans via the fecal-oral route, usually from the ingestion of eggs found in dog feces. This pathogen usually causes infection in the liver in the form of a hydatid cyst and remains a significant health problem in South America, Eastern Europe, Africa, and western China.³² Exposure to sheep is a significant risk

factor, and endemic disease tends to occur in places where dogs, the definitive host, might come into frequent contact with sheep, as seen on farms. In such endemic areas, prevalence can be up to 6%.³⁸ *Echinococcus multilocularis* usually causes alveolar disease and is a significant health concern in Eastern Europe and Central Asia.²⁰ The definitive host for *E. multilocularis* is typically a fox, so infection rates are greatest where there is a high fox population. Although involvement of echinococcal disease in the CNS is rare, the most commonly involved part of the CNS is the thoracic spine.³⁹

If spinal involvement is present, the most likely symptoms are nonspecific and are the result of spinal cord compression causing radiculopathy or myelopathy.¹ However, it is not uncommon for large cysts to remain asymptomatic. It is therefore important for the clinician's differential diagnosis to remain broad when presented with a patient with spinal pathology, because spinal echinococcal disease is potentially curable.

Plain radiographs can visualize cystic lesions in contiguous vertebral bodies, bone lysis, and spondylitis, but follow-up imaging with CT and/or MRI is usually necessary. Ultrasonography may be helpful in detecting abdominal involvement.¹² CT provides better bone resolution and can visualize osteolytic lesions in the vertebral bodies. The lesion does not enhance with intravenous contrast.⁴⁵ MRI is the most sensitive imaging modality to detect spinal hydatid disease, but in the absence of MRI, CT myelography can also demonstrate spinal cord involvement.^{44,45} T1-weighted images usually demonstrate an isointense or hypointense cyst and cystic wall, whereas T2-weighted images demonstrate a hyperintense cyst with

a hypointense cystic wall.⁴⁴ Berk et al. describe the lesion on MRI as a unique sausage-like shape with two dome-shaped ends with no debris in the lumen.⁶ Last, diffusion-weighted imaging can distinguish between spinal hydatid cysts and abscesses because the fluid in abscesses is more viscous, which restricts water movement and yields a hyperintense signal compared to cysts.¹⁹

The differential diagnosis of spinal echinococcal disease is broad and includes spinal tuberculosis (*Mycobacterium tuberculosis* and *Echinococcus* share some endemic areas), malignancy, abscess, and cystic lesions such as spinal arachnoid cysts or spinal aneurysmal bone cysts.^{17,41} Clinical history, imaging studies, and laboratory studies can significantly narrow this differential diagnosis, but only surgical exploration and histopathological examination can provide a definitive diagnosis. Serodiagnostic tests are specific but not sensitive.

Surgery is the treatment of choice for spinal echinococcal disease, although long-term preoperative treatment with an anthelmintic like albendazole may reduce intracystic pressure.^{27,44} The most commonly reported procedure is simple decompression with laminectomy, although the need to perform spinal fusion should always be considered depending on the extent of the lesion. Most of the surgical procedures use a posterior approach, but some studies have reported anterior approaches.⁴⁴ In general, the preference is to remove echinococcal cysts radically because needle aspiration carries a significant risk of cystic rupture. This same principle applies to spinal echinococcal disease, but one case report demonstrated the complete resolution of symptoms in a patient with advanced-stage echinococcosis.⁵⁴ The use of scolical agents intraoperatively to prevent the dissemination of the parasite during surgery has been described in abdominal and pelvic cases of hydatid cyst removal.^{7,8} Their use in spinal cases has not been extensively studied but can theoretically provide a similar protective benefit.

Conclusions

Although parasitic infections of the spine are rare in the developed world, they are worth considering in a differential diagnosis, especially in countries with high rates of immigration and tourism such as the US. Presenting symptoms of parasitic spinal infections are often nonspecific, so their diagnosis can be easily overlooked. Key findings on imaging modalities, laboratory studies suggestive of parasitic infection, and most importantly a thorough patient history are required to correctly diagnose parasitic spinal infections.

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Disclosures

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Author Contributions

Conception and design: Assina. Acquisition of data: Assina. Analysis and interpretation of data: Assina, Majmundar, Patel, Dodson. Drafting the article: all authors. Critically revising the article: all authors. Reviewed submitted version of manuscript: Assina, Majmundar, Patel, Dodson, Goldstein. Approved the final version of the manuscript on behalf of all authors: Assina. Statistical analysis: Goldstein.

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