Parasitic Central Nervous System Infections: Echinococcus and Schistosoma

Kiarash Shahlaie, M.D.,* Mark W. Hawk, M.D.,* Brian R. Hu, B.A.,† J.H. Theis, DVM, PhD,‡ Kee D. Kim, M.D.*

*Department of Neurological Surgery; †Department of Medical Microbiology, University of California, Davis Medical Center, Davis, CA

Central nervous system (CNS) manifestations of Echinococcus and Schistosoma infections occur throughout the world, with incidence increasing in developed regions. A detailed literature review generated a current summary on epidemiology, parasitology, pathology, clinical manifestations, imaging studies, diagnosis, and treatment of neuroechinococcosis and neuroschistosomiasis. Recent advancements have been made in diagnosis, treatment, and prevention of these parasitic CNS infections. Ongoing advancements in neuroimaging and diagnostic studies, as well as efforts to better understand the parasite genome and host–parasite relationships, will likely continue to improve patient management.

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Parasitic infections of the central nervous system (CNS) are most common in developing countries, although prevalence has increased in more developed regions because of immigration patterns. In the Western Hemisphere, most parasitic infections are due to Taenia, Toxoplasma, Naegleria, Trypanosoma, Echinococcus, and Schistosoma. The tapeworm Echinococcus, for example, can infect the CNS, resulting in large cysts that manifest as focal neurological deficits or seizures.†
Trematode (fluke) infections by species of Schistosoma can also involve the CNS, resulting in necrotizing eosinophilic granulomas in the brain that may cause seizures, hydrocephalus, or other diffuse or focal deficits. A review of these 2 helminthic infections—rare but important neurological diseases—provides valuable insight into their diagnosis and management.

**Echinococcosis**
The first detailed report of echinococcosis came from Hippocrates in the fourth century B.C., who described a ruptured hepatic cyst caused by larval forms of Echinococcus. The Greek translation of hydatid disease, in fact, is “watery cyst.” In the 1600s, Hartmannus, Redi, and Tyssen more completely described the disease's etiology, and in 1808, Rudolphi first used the term “echinococcosis.”

Cystic echinococcosis (CE), caused by E. granulosus, was the first disease described, whereas alveolar echinococcosis (AE), caused by E. multilocularis, was not correctly diagnosed until 1855. In the early 1900s, Dévé first described features of this parasite's life cycle in detail.

In the remainder of the 20th century, a better understanding of epidemiology, clinical presentation, and diagnosis was gained. Polycystic echinococcosis (PE), caused by E. oligarthrus and E. vogeli, was subsequently studied. Important contributions to surgical management of hydatid cysts were made by Dowling and others, emphasizing the importance of preventing cyst rupture during excision. Although significant advancements have been made in imaging techniques and treatment, the natural history and optimal treatment of echinococcosis have yet to be clearly defined.

**Epidemiology**
E. granulosus is endemic in South America, Australia, the Mediterranean, the Middle East, and parts of the United States and Canada. CE is particularly common in Turkey, where a study reports 2000 to 3000 cases annually. In North America, endemic regions include the north central United States, Alaska, and central Canada; endemic foci also occur within California and the lower Mississippi Valley. CE is the most prevalent form of E. granulosus, particularly in cattle-raising rural areas due to its domestic life cycle that includes dogs and livestock.

E. multilocularis is endemic in parts of the United States, central Europe, China, Russia, and Turkey. The North American regions most affected by E. multilocularis are similar to areas affected by E. granulosus and include central Canada, the north central United States, and Alaska. This species has a sylvatic cycle involving wild animals like foxes, voles, and lemmings—a reason E. multilocularis is less frequently associated with human disease.

E. vogeli and E. oligarthrus are endemic in Central and South America. PE in humans is less prevalent than other echinococcal infections, with approximately 100 cases reported since 1979. Infections with E. oligarthrus are even less common, accounting for only 3 of these cases.

**Parasitology and Pathology**
The life cycle of E. granulosus involves the dog as the most common definitive host and sheep as intermediate hosts (Figure 1). Humans become infected as incidental intermediate hosts during the larval stage by ingesting eggs from proglottides in the feces of dogs carrying the adult tapeworm. Hands-to-dirt-to-mouth or contamination by the host dog licking a person's face are common means of transmission.

Ingested eggs liberate an embryo that can translocate through the intestinal mucosa and migrate to the liver by way of the portal system. E. multilocularis is frequently acquired by humans through ingestion of unwashed wild berries contaminated with proglottides from foxes defecating in or around bushes. E. vogeli has the bushdog or domestic dog as its definitive hosts, and the paca acting as the intermediate host. E. oligarthrus has various wildcats as its definitive hosts and the agouti as its intermediate host.

Hepatic lesions occur in 60% of cases involving E. granulosus and almost all cases involving E. multilocularis. Pulmonary lesions are seen in 10% to 40% of cases involving E. granulosus but are less common in infections associated with E. multilocularis. Other sites of involvement such as the pericardium, kidneys, skeletal system, or CNS are described but are less common.

PE from E. vogeli involves mainly the liver, lungs, and some abdominal organs. Rare cases of E. oligarthrus have involved the orbit or the heart. CNS involvement is generally infrequent, occurring in approximately 5% of cases involving E. granulosus and 1% of cases involving E. multilocularis. Bone involvement is similarly rare, occurring in 0.5% to 2% of cases of hydatid disease (approximately 50% involve the spine).
Parasitic Central Nervous System Infections continued

Clinical Manifestations
Infections from Echinococcus are usually asymptomatic, and its slow growth rate can result in latent periods between transmission and clinical presentation of up to 20 years. In fact, it is not uncommon for routine imaging studies to incidentally discover Echinococcus lesions. Symptoms from echinococcosis are typically secondary to compressive effects on adjacent structures; therefore, CNS lesions often produce symptoms much earlier than infections elsewhere, although these lesions sometimes achieve a significant size before becoming symptomatic. Bacterial superinfection resulting in abscess formation inside the cyst or immune reactions to cyst leakage can also result in clinical symptoms.

Intracerebral hydatid cysts account for approximately 2% of space-occupying intracranial lesions, even in countries where Echinococcus is endemic. Cerebral E. granulosus infection often involves children under age 15. These patients typically present with headaches, nausea, vomiting, papilledema, and optic atrophy, signs and symptoms consistent with increased intracranial pressure. Intracerebral hydatid cysts account for approximately 2% of space-occupying intracranial lesions, even in countries where Echinococcus is endemic. These patients typically present with headaches, nausea, vomiting, papilledema, and optic atrophy, signs and symptoms consistent with increased intracranial pressure.

Macrocranium and local cranial bulging may result from rapid growth of the cyst, which may expand as much as 5 cm per year. Seizures occur less frequently (as compared to neurocysticercosis) and depend on the location of the cyst. Most cases of intracerebral cysts due to E. multilocularis, on the other hand, involve adults. Symptoms of increased intracranial pressure tend to occur late in the disease process and, unlike children, adults more commonly present with focal neurological deficits and hemiparesis. Seizures are similarly infrequent in adults, but are commonly seen postoperatively. Spinal involvement may present as a radiculopathy, myelopathy, or a combined radiculomyelopathy.

Imaging Studies
Computed tomography (CT) of patients with cerebral hydatid disease typically reveals a spherical intraparenchymal lesion in the distribution of the middle cerebral artery (Figure 2). Cysts tend to be smooth, homogeneous, thin-walled lesions with well-defined borders. Lesions are frequently larger than 2 to 3 cm in diameter, in contrast to smaller

Figure 1. Life cycle of Echinococcus granulosus. Figure provided courtesy of JH Theis, DVM, PhD.
lesions typically found in neurocysticercosis.25,27

Cyst walls are typically iso- to hyper-dense relative to the brain parenchyma, and cyst contents have similar density to the cerebrospinal fluid (CSF). Surrounding edema is usually minimal and enhancement is uncommon.28 Cysts tend to lie close to the calvarium and, as a result, thinning of the inner table of the skull may be apparent.28 Calcifications of the cyst margin or contents are rare, except in multilocular cysts that may result from previously ruptured unilocular cysts.29-32

Multilocular cysts may be polygonal in shape and are more likely to enhance with contrast. Lesions in CE consist of an inner germinal layer and an outer laminated layer surrounded by a fibrous capsule formed by the host. Internal budding often forms daughter cysts, scolecids, and brood capsules.20

Cysts associated with E. multilocularis are less characteristic and may be semi-solid, with areas of necrosis, surrounding edema, and contrast enhancement. Scattered calcifications are more commonly seen. The cysts in AE grow via progressive external budding of the germinal membrane.33

On magnetic resonance imaging (MRI) of cerebral hydatid disease, both the cyst contents and cyst capsule are hypointense relative to brain parenchyma on T1-weighted sequences.34 Like CT, MRI enhancement is uncommon. T2-weighted images reveal hyperintense cyst contents surrounded by a hypointense capsule.28

Both CT and MRI are used to diagnose CE. Although CT is superior in detecting cyst calcifications, MRI is superior in identifying the cyst capsule, surrounding edema, and the anatomical relationships between the lesion and other structures.20 It is a more useful imaging modality for preoperative planning and is the study of choice of most practitioners.

More recently, magnetic resonance spectroscopy (MRS) has been suggested as a diagnostic aid to help differentiate hydatid cysts from other cystic lesions such as bacterial abscesses, neoplasms, and arachnoid cysts.35 The combination of pyruvate, alanine, and acetate peaks may be a distinctive feature of hydatid cysts. Because MRS may also differentiate live cysts from degenerating or dying cysts, it may have a future role in monitoring medical therapy.35

Spinal echinococcosis most commonly affects the thoracic and lumbar spine and may involve the bony elements, paraspinal tissues, the extra or intradural spaces, or the spinal cord itself. When located in the vertebral body, cysts tend to be multilocular and infiltrative, growing along intratrabecular spaces.18,36 They may progress to involve posterior elements such as the ribs or pelvis. Multiple adjacent vertebrae may be involved or non-diseased segments may be interposed between diseased segments.

Radiographs and CT images may demonstrate vertebral body destruction or involvement of posterior elements, contiguous ribs, or paravertebral soft tissues with preservation of intravertebral disc spaces.18 In spinal echinococcosis, CT reveals a spinal cystic mass with fluid density similar to CSF, much like cerebral echinococcosis. When the disease perforates through the cortex and periosteum, it may enter the extradural space. Although rare, intramedullary or intradural extramedullary disease has been reported.26

**Diagnosis**

Peripheral blood smears, eosinophilia, and CSF assessment are neither sensitive nor specific for echinococcosis, similar to neurocysticercosis. Serological tests can include enzyme-linked immunosorbent assay (ELISA), immunofluorescence, indirect hemagglutination, and complement fixation.

The ELISA test has been associated with false positives in hydatid disease, and false negative serological results are not uncommon in hydatid disease involving only the lung, brain, or eye.5,19,37 The immunodiffusion test involves electrophoretic separation of antigens in hydatid fluid. Reaction of these antigens with the patient’s serum is sensitive and highly specific if a precipitin line develops at “arc5” on the gel.38 As with neurocysticercosis, diagnosis is made using a

**Figure 2.** Noncontrast axial head CT demonstrating left hemisphere parenchymal hydatid cysts. CT, Computed tomography. Radiographic figure provided courtesy of KD Kim, MD, J Boggan, MD, and J Brunberg, MD.
combination of clinical, laboratory, and imaging data.²⁹

Treatment

Surgery has been the mainstay of treatment for years, although medical therapy has played an increasingly important role recently (Figure 3).³ Mebendazole, a benzimidazole, was the first drug to be extensively tested.¹⁰,¹²,²³,³⁹-⁴¹ However, poor absorption and severe side effects complicate its administration. Treatment is required at high doses for months to years and its efficacy is unclear.

Albendazole, also a benzimidazole, has been shown to be effective in the treatment of hydatid disease, although its efficacy against cerebral and spinal disease remains unclear.³⁹ Nevertheless, concomitant surgical treatment and anthelmintic therapy is recommended for most cases.⁸ Anthelmintic therapy is also advocated when cyst rupture is involved.⁸

Surgery is considered first-line therapy for cerebral hydatid disease.⁸ The primary goal is complete removal of the entire cyst without rupture. Several surgical techniques have been described to accomplish this goal.⁶ Dowling’s technique of saline irrigation with a silicone catheter between the cyst and brain interface is helpful to flush out the cyst.

In the event of cyst rupture during surgery, however, cyst aspiration, cyst wall extirpation, and extensive hypertonic irrigation of the surgical field has become an accepted routine. If its location poses a high risk of rupture, such as in the orbit, sella turcica, or deep gray matter, the cyst is deliberately punctured and aspirated. In general, percutaneous aspiration of the suspected hydatid cyst should be avoided as liberation of hydatid fluid can lead to anaphylactic shock, and spilled scoleces can lead to secondary hydatidosis.³,¹¹

Previously ruptured cysts, either spontaneously or as the result of trauma or iatrogenic causes, present additional hazards.⁸ In these instances, a trial of medical therapy may be considered. Gamma knife radiosurgery, combined with anthelmintic therapy, has been reported as a potential alternative to an operation.⁸

Surgical treatment for spinal disease is generally challenging. Multiple lesions are frequently present and intraoperative rupture is common. In one study, intraoperative rupture occurred in 44% of cases, typically during laminectomy.⁸ As a result, patients with spinal involvement generally have a poorer prognosis and a higher propensity for recurrence.⁴² Although each case must be individualized, a combination of medical and surgical therapy may be the optimal treatment of spinal echinococcosis.

Schistosomiasis

Schistosomiasis, also known as Bilharzia, is a parasitic disease caused by trematode blood flukes. Schistosomiasis can be traced as far back as ancient Egypt and China. The ancient Greek historian Herodotus described menstruation of men in Egypt consistent with hematuria in a Schistosoma haematobium infection. Performing an autopsy in Egypt, Dr. Theodore Bilharz was the first to describe schistosomiasis in humans, and his name became linked to the disease. Of the 6 species of Schistosoma that give rise to disease in humans, Schistosoma mansoni, S. haematobium, S. japonicum and S. mekongi are the most important ones.

Epidemiology

Approximately 200 million humans are infected with a species of Schistosoma worldwide, resulting in an annual mortality of 200,000. In tropic and subtropic regions, schistosomiasis ranks second to malaria as a significant public health concern. Although less significant in the United States, it is estimated that over 400,000 people are infected with a schistosomal species.⁴³

S. mansoni is the most prevalent and widespread of the group, infecting over 110 million people worldwide. It is endemic in parts of South America, the Caribbean, Africa, the Near East, and Saudi Arabia. S. haematobium infects approximately 90 million people and is endemic in Africa and the Middle East. S. japonicum occurs primarily in China and the Philippines, having been eradicated from Japan in 1996.⁴⁴ S. mekongi is endemic in Southeast Asia and gets its name from its boundaries along the Mekong River. S. japonicum and S. mekongi together infect approximately 2 million people.⁴⁵

Over the last several decades, the prevalence of schistosomiasis has increased. This has been attributed to continued migration of people from endemic to nonendemic regions and to the building of dams and irrigation systems that set up habitats for the parasite, such as the Three Gorges Dam in China.⁴⁶ The majority of cases involve young males who work in rural regions and have a higher likelihood of exposure to infected water.⁴⁵,⁴⁷
Parasitology and Pathology

Humans are the definitive hosts of Schistosoma and certain species of aquatic snails are intermediate hosts (Figure 4). In this organism’s life cycle, eggs are released into fresh water from human feces or urine. The individual eggs then hatch to become miracidia that infect snails. Within the snail, the miracidia become sporocysts that give rise to cercariae that are released into fresh water.

Cercariae then penetrate human skin to reach the liver, where they mature into young flukes. The flukes (male and female) migrate as a pair down the portal system. S. mansoni, S. japonicum, and S. mekongi migrate to the mesenteric veins and S. haematobium migrates to the pelvic veins. Thus, S. mansoni, S. japonicum, and S. mekongi cause intestinal and hepatosplenic disease, and S. haematobium causes urinary tract disease.

CNS involvement is generally rare and its mechanism has been a topic of debate. Some authors suggest that eggs embolize from a hepatic source to the brain by cardiac or pulmonary arteriovenous shunts, or by retrograde venous flow from the vertebral venous plexus.

The majority of spinal schistosomiasis involving the lower thoracic and lumbosacral spine is likely due to the venous plexus connecting intra-abdominal veins with those of the lower spine. Alternatively, the adult worms themselves may migrate through the vertebral venous plexus to reside within cerebral or spinal veins. Direct deposition of eggs, called oviposition, may then occur.

The incidence of brain or spinal cord involvement varies among Schistosoma species. S. japonicum usually affects the brain and only rarely affects the spinal cord. S. mansoni and S. haematobium, on the other hand, tend to involve the spinal cord more frequently than the brain, resulting in myeloradiculopathy. There has been recent evidence of S. mekongi infecting the brain. These distributions may be secondary to differences in egg morphology among the species. The eggs of S. japonicum and S. mekongi, for example, are small and round and may travel by hematogenous routes directly to the brain. Alternatively, the eggs of S. mansoni and S. haematobium are large and have protruding spines that may hinder their translocation to distant sites.

Although clinically apparent cerebral disease is rare, autopsies of patients with schistosomiasis have revealed a significant number of ova deposited in the brain. An autopsy study of 162 cases of known systemic schistosomiasis from S. mansoni found eggs in 26% of the brains studied whereas only 3 cases (1.9%) had known symptomatic CNS involvement. Lack of clinical manifestations may be due to an intermittent showering of the brain by a small
number of ova causing little or no inflammatory response.50,52

**Clinical Manifestations**

Most schistosomal infections are asymptomatic, with severe symptoms occurring in only 5% to 10% of cases.49 Schistosomiasis typically presents in 3 distinct stages. Initially, the patient develops dermatitis after the skin is penetrated by the cercariae. A variable latent stage follows that may last many months before disease onset.

The acute stage, Katayama fever, is characterized by fever, myalgia, and abdominal complaints with diarrhea and dysentery. Rare CNS involvement (neuroschistosomiasis) occurs during this stage, resulting in encephalopathy or encephalitis.57 A chronic stage then follows, characterized by a granulomatous reaction to eggs in the tissues and humoral antibody production.52

Neurological signs may result from acute hepatic encephalopathy or from mass lesions due to eggs lodged in brain capillaries. Symptomatic lesions typically result from eggs gaining access from the portal venous system and vena cava to the spinal cord and cerebral veins through retrograde flow into the Batson vertebral epidural venous plexus.54

Cerebral schistosomiasis typically manifests as 1 of 3 distinct pathological lesions: focal (most common), general, and mixed types.2 Signs and symptoms may include headache, fever, nuchal rigidity, incontinence, personality change, cranial nerve palsies, papilledema, pyramidal tract signs, seizures, and even coma.58

When the spinal canal is involved, lesions typically occur in the lower thoracic or lumbar regions, involving the conus medullaris or cauda equina.49 Spinal symptoms may occur as early as 38 days after infection or could take years to present.59

Spinal involvement generally results in 4 possible presentations.45

The first is an acute transverse myelitis, characterized by acute onset of myelopathy with paresis and bowel or bladder involvement. This differs from other causes of acute transverse myelitis by a tendency to involve the conus rather than the thoracic cord. On pathological examination, multiple small intramedullary granulomas are often seen.

The second presentation is subacute and associated with large intra- or extra-axial granulomatous mass lesions. The third presentation is radicular in nature, usually involving the lower lumbarosacral nerve roots. This commonly results in an asymmetric sensorimotor radiculopathy; granulomas are found along the affected nerve roots. The last form presents as vasculitis—patients may develop an anterior cord syndrome secondary to anterior spinal artery vasculitis.

**Imaging Studies**

On head CT or brain MRI studies, homogeneous or heterogeneous nodular parenchymal lesions with parenchymal lucencies are often seen. Contrast enhancement of the lesion or its periphery may be present.52 Surrounding areas of high T2 signal consistent with perilesional edema may be present. As a result, these lesions may sometimes resemble brain tumors and are typically differentiated with a thorough patient history.52 Although rare, subarachnoid hemorrhage has also been reported.28

Spinal cord lesions commonly present as a filling defect on myelograms with “trifid edges” and intramedullary swelling.47 MRI may reveal nodular lesions within the conus or along the cauda equina nerve roots. Intramedullary swelling may be seen on T1- and T2-weighted sequences (Figure 5).

**Diagnosis**

Characteristic schistosomal eggs may be identified in the stool or urine of infected individuals. A peripheral eosinophilia is often present, but this is neither sensitive nor specific. Tests like ELISA and immunoblot that detect antibodies against Schistosoma are highly sensitive, but have limited value as cross-reaction to other helminthic infections results in low specificity.

Examination of CSF in the acute phase may show high opening pressure, lymphocytic pleocytosis, eosinophilia, elevated protein, and normal glucose levels.2 Electroencephalogram, CT, MRI, and liver, bladder, or rectal biopsy may help support the diagnosis.2,47 As with other parasitic diseases, definitive diagnosis depends on epidemiological information, such as history of travel or residence in an endemic area.

Praziquantel is safe and effective against schistosomiasis and results in an 80% cure rate following 1 treatment course.
tissue from continuing to act as foreign bodies to promote granulomatous reactions. As there is no specific treatment to eliminate existing ova, therapy focuses on preventing the deposition of additional eggs into tissues. Slow resolution of the granulomas over time may result in remission of signs and symptoms.61-63

Cure rates of 75% at 5 to 10 years after treatment with praziquantel have been reported in patients with cerebral disease due to S. japonicum.64 Likewise, praziquantel has been shown to be effective against schistosomal myelopathy.52,65,66 Oxamniquine is an alternative to praziquantel for the treatment of S. mansoni infections; neuropsychiatric side effects from the drug, however, have been reported. Other treatment options with mixed efficacies include artemisinin derivatives, metrifonate, and myrrh.67

Concomitant corticosteroid therapy has been advocated by some authors, particularly in patients with significant mass effect due to granulomatous inflammation and edema.68 Although there is some evidence that corticosteroids may reduce ova deposition by adult worms,69 its use remains controversial and awaits further study.

Some authors have reported praziquantel-resistant cases of schistosomiasis, a trend that is of particular concern as treatment has heavily depended on this drug.70,71 These reports, as well as the difficulties of delivering praziquantel to some endemic areas, have focused interest in ongoing efforts to develop an effective vaccine.72

Decompressive laminectomy is reserved for patients who develop acute neurological changes with evidence of spinal cord or nerve root compression or for patients who fail medical therapy. Laminectomy and biopsy may also serve as an alternative to liver, rectal, or bladder biopsy.47

**Conclusion**

Echinococcosis and schistosomiasis have a worldwide distribution affecting millions of individuals in many countries, including the United States. CNS manifestations often result in management of these challenging cases by neurologists and neurosurgeons. Surgical intervention and medical management remain the mainstay of treatment for neurochirinoccosis and neuroschistosomiasis, respectively, but a multimodal and multidisciplinary approach is often needed to optimize outcome. Ongoing efforts to better understand the epidemiology, pathophysiology, and natural course of these infections will likely improve diagnosis, treatment, and prevention.

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Main Points
- Echinococcus and Schistosoma parasitic infections of the central nervous system (CNS) are most common in develop-
ing countries, although incidence is increasing in more developed regions, including parts of the United States and 
Canada. In tropical and subtropical regions, schistosomiasis ranks second to malaria as a public health concern.
- Infections from Echinococcus are usually asymptomatic, and its slow growth rate can result in latent periods between 
transmission and clinical presentation of up to 20 years. CNS lesions often produce symptoms much earlier than 
infections elsewhere, although they sometimes achieve a significant size before becoming symptomatic.
- Cerebral schistosomiasis manifests as 1 of 3 distinct pathological lesions: focal, general, and mixed types. Signs and 
symptoms may include headache, fever, nuchal rigidity, incontinence, personality change, cranial nerve palsies, 
papilledema, pyramidal tract signs, seizures, and even coma.
- Diagnosis for echinococcosis is made using a combination of clinical, laboratory, and imaging data. Although com-
puted tomography (CT) is superior in detecting cyst calcifications, magnetic resonance imaging (MRI) excels in 
identifying the cyst capsule, surrounding edema, and the anatomical relationships between the lesion and other structures.
- In head CT or brain MRI studies of schistosomiasis, homogeneous or heterogeneous nodular parenchymal lesions 
with parenchymal lucencies often appear that may resemble brain tumors. A thorough patient history can typically 
differentiate them.
- The preferred treatment for cerebral hydatid disease is surgery to completely remove the entire cyst without rupture. 
Albendazole is also advocated, although concomitant surgical treatment and anthelmintic therapy is recommended 
for most cases.
- Schistosomiasis treatment is heavily dependent on praziquantel, a safe and effective drug that delivers an 80% cure 
rate following 1 treatment course. Some cases of praziquantel-resistant schistosomiasis have been reported. Con-
comitant corticosteroid therapy also has been advocated, but it remains controversial and awaits further study.