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Neurologic Parasitic Infections in Immigrants and Travelers

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Abstract

Infectious diseases are increasingly common in modern clinical practice and the contemporary neurologist must be aware of the clinical manifestations, potential complications, and management of common travel-related infections. The authors provide an approach to patients who present with neurologic symptoms, with a history of travel to or residence in tropical and developing countries. Although many other infections are important in this demographic, they focus on three parasitic infections that neurologists may encounter: cerebral malaria, neuroschistosomiasis, and neurocysticercosis. The epidemiology, pathophysiology, clinical manifestations, diagnostic evaluation, and treatment are discussed for each infection.

Keywords

Neuroinfectious diseases; neuroschistosomiasis; cerebral malaria; neurocysticercosis

The emigration of individuals from developing nations and the increasing popularity of travel to regions of the world with endemic “rare” infectious diseases have blurred the once well-demarcated geographic boundaries of disease. Outsourcing of jobs from developed to developing countries and economic globalization has had a modest impact on the socioeconomic status of individuals residing in developing countries. Unfortunately, such change, in the face of a substantial burden of disease, has not led to a significant impact on the morbidity and mortality associated with tropical infections.¹ Individuals with infectious diseases are increasingly common in modern clinical practice, and the neurologist must be aware of the clinical manifestations, potential complications, and appropriate management of common travel-related infections. Neurologic sequelae of many tropical infections include behavioral changes, meningoencephalitis, seizure activity, and paralysis. Any of these symptoms in an individual who has lived or traveled to a region with endemic infections that could affect the nervous system should be questioned regarding contact with sources of infection. Questions should include a detailed travel history, history of fever, exposures (including sexual encounters, history of freshwater swimming, ingestion of contaminated food, insect or animal bites), vaccination history, use of prophylactic medications, and immune status.² Infections based on travel location or area of residence are shown in Fig. 1.² In this article, we focus on three important parasitic infections encountered

in the tropics and developing countries: cerebral malaria, neuroschistosomiasis, and neurocysticercosis. The epidemiology, pathophysiology, clinical manifestations, diagnostic evaluation, and treatment of these infections are discussed.

CEREBRAL MALARIA

Epidemiology

Despite considerable effort, malaria remains one of the most devastating infectious diseases in developing countries.³ In 2008, there were an estimated 243 million cases of malaria worldwide, causing 863,000 deaths. Malaria is most prevalent in Africa, Central and South America, and South-East Asia.⁴ Human infection is caused by four *Plasmodium* species: *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*. *Plasmodium falciparum* is the most common cause of infection and is responsible for 80% of all cases of malaria and 90% of deaths from malaria.⁵ Of the 125 million people from nonendemic countries who travel through malaria-endemic countries each year, 10 to 30,000 will contract malaria.^{6,7}

Cerebral malaria is the most severe complication of malaria and a major cause of acute nontraumatic encephalopathy in tropical countries.⁸ Cerebral malaria is most often caused by *P. falciparum*, which accounts for 80% of fatal cases.⁹ Coinfection with malaria and human immunodeficiency virus (HIV) impacts both the clinical course of malaria and HIV. HIV-infected individuals with malaria have a higher prevalence of fever, parasitemia, and severe or complicated malaria.¹⁰

Life Cycle

The plasmodium species is first ingested by the *Anopheles* mosquito when it feeds on an infected human carrier. Sporozoites are stored in the salivary glands of the mosquito until they feed on a human host. When an infected mosquito takes a blood meal, sporozoites in the mosquito's saliva enter the bloodstream and migrate to the liver. Within minutes of being introduced into the human host, the sporozoites infect hepatocytes, and multiply asexually and asymptotically for several days. Within days, the organisms differentiate to yield thousands of merozoites, which then invade circulating erythrocytes. After 48 hours, the erythrocyte ruptures and merozoites are released, which then invade additional erythrocytes. Some merozoites develop into sexual forms of the parasite, known as gametocytes, which circulate in the bloodstream. This produces an exponential expansion of the infection in the human host that is responsible for the symptoms of the illness.⁸

Pathophysiology of Cerebral Malaria

Sequestration of pRBCs is the primary cause of the neurologic manifestations of cerebral malaria, including subsequent increased intracranial pressure. Sequestration begins with cytoadherence of parasitized RBCs (pRBCs) to the vascular endothelium through an interaction between plasmodium-derived proteins, such as *P. falciparum* erythrocyte membrane protein 1 (PfEMP1), on the surface of pRBCs and ligands on endothelial cells.¹¹ Decreased blood flow associated with cytoadherence eventually leads to microcirculatory obstruction.⁹ Also key to the reduced cerebral blood flow is decreased flexibility of pRBCs and eventual RBC rupture with further obstruction of the microcirculation by fragments of destroyed RBCs.⁹ Parasite-induced sequestration of pRBCs and nonparasitized RBCs may also induce increased cerebral blood volume and concomitant increased intracranial pressure. Increased blood-brain barrier permeability is believed to be a less-important factor.¹²

Clinical Manifestations

Cerebral malaria associated with *P. falciparum* parasitemia is defined as an unarousable coma not attributable to other causes, such as convulsions, postictal confusion, hypoglycemia, or meningitis.¹³ Cerebral malaria most often occurs in pregnant women, children, the non-immune population, and immunosuppressed individuals.¹⁴ The clinical presentation of cerebral malaria differs between children and adults.⁹ Initial symptoms are non-specific in both children and adults, and include intermittent fever with chills, headache, nausea, vomiting, abdominal pain, and myalgias.¹⁵ Children often rapidly develop coma, seizure activity, brainstem dysfunction, and malarial retinopathy.⁹ Seizures are a presenting symptom of cerebral malaria in over 80% of African children.^{16,17} Brainstem dysfunction occurs in over 30% of children with cerebral malaria.^{18,19} Nystagmus, ocular bobbing, and sixth nerve palsies are occasionally present. In adults, cerebral malaria typically presents as a diffuse encephalopathy, in which focal neurologic signs are relatively uncommon.^{9,20} Malarial retinopathy, a finding often seen in patients with cerebral malaria, is characterized by retinal whitening, vessel changes, retinal hemorrhages, and papilledema reflecting changes in blood flow within the brain, and are therefore of pathophysiologic, diagnostic, and prognostic significance (Fig. 2).^{21,22} A prospective autopsy-based study of children dying from cerebral malaria in Malawi found that malarial retinopathy was more specific than any other clinical or laboratory feature in distinguishing malarial from nonmalarial coma.²¹ Long-term neurocognitive deficits, including cognitive difficulties, language, and behavioral problems are increasingly recognized in young children who have had malaria.²³

Diagnostic Studies

Cerebral malaria should be considered in any patient with a febrile illness and impaired consciousness that has resided in or traveled to a region with endemic *P. falciparum*. Examination of thick and thin blood smears with Giemsa staining, when performed by a skilled technician, has a high sensitivity and specificity for detecting parasitemia and identifying malarial species; unfortunately, in areas where malaria is not endemic, technicians with such skills are often nonexistent and the sensitivity and specificity thus drop markedly. Serologic tests are available, but false-positive tests are common.¹⁵ A lumbar puncture should be performed to rule out other causes of meningoencephalitis unless coma or obtundation is present, in which case it should be avoided, as increased intracerebral pressure associated with cerebral malaria could result in herniation and death. Direct ophthalmoscopy should be used to assess for malarial retinopathy. Computed tomography (CT) may demonstrate cerebral edema during the advanced stages of malaria (Fig. 3).²⁴ Magnetic resonance imaging (MRI) often reveals brain enlargement without edema, and transtentorial herniation is a common finding on CT or MRI in fatal cases of cerebral malaria.²⁵

Treatment

Early parenteral antimalarial treatment initiated immediately after cerebral malaria is confirmed, or when highly suspected, is the most important intervention for preventing dire outcomes of cerebral malaria. Choice of treatment should be guided by the regional prevalence of *Plasmodium* species, by where the infection was acquired, and by severity of disease. In areas where chloroquine-resistant *P. falciparum* (CRPF) is common, Southeast Asia, the Amazon region of South America, and certain areas of sub-Saharan Africa, quinine with doxycycline, mefloquine, halofantrine, or artemisinin derivatives should be administered.²⁶ Intravenous quinine, in combination with doxycycline, is the mainstay of antimalarial treatment in these regions.^{27,28} Artemisinin (qinghaosu) derivatives have also been used in the treatment of severe malaria.²⁹ The addition of steroids provides no benefit and may be associated with worse outcome.³⁰ Ancillary treatments include antiepileptic medications and hyperosmolar therapies. Treatment of elevated intracranial pressure with

mannitol has been associated with improved outcome.³¹ Adjunctive treatment with exchange transfusion for severe falciparum malaria is of uncertain benefit.²⁸ Chelating agents, such as deferoxamine and pentoxifylline, have not produced significant benefit in the treatment of severe malaria.³² Due to the complicated nature of treating malaria, local treatment guidelines should be consulted.

NEUROSCHISTOSOMIASIS

Schistosomiasis is a parasitic disease caused by the blood fluke (digenetic trematode) *Schistosoma*. Five species of *Schistosoma* are known to infect humans: *S. mansoni*, *S. mekongi*, *S. japonicum*, *S. intercalatum*, and *S. haematobium*. Most granulomas develop in the intestine, the liver (*S. mansoni* and *S. japonicum*), or genitourinary tract (*S. haematobium*).³³ Cerebral involvement is caused primarily by *S. japonicum*, whereas *S. mansoni* and *S. haematobium* typically cause spinal cord infection.³⁴ Schistosomiasis is endemic to South America, sub-Saharan Africa, Asia, the Middle East, and the Caribbean Islands.³⁵ Over 200 million individuals are infected worldwide each year; immuno-compromised males are at highest risk of acquiring infection.³⁶ Neuroschistosomiasis occurs in a small percentage of patients with schistosomiasis, although this is likely underreported due to the small number of published case reports. In one of the first population-based studies of neuroschistosomiasis during the Philippines campaign of World War II, neurologic manifestations were observed in 2% of American soldiers infected with *S. japonicum*.³⁴ More recently, in a study in sub-Saharan Africa, 1 to 5% of nontraumatic spinal cord injuries were attributed to spinal schistosomiasis, and 4.3% of adults hospitalized in China with schistosomiasis had central nervous system (CNS) involvement.^{37,38}

Life Cycle

Human infection occurs after direct contact with fresh water harboring the larval forms of the parasite. After the larvae penetrate human skin, they transform into the migrating schistosomulum stage. Schistosomulum travel to the lungs and then the liver sinusoids. Parasites reach maturity in 6 to 8 weeks in the liver, at which time they begin to produce eggs. Many of these eggs become trapped in the mesenteric veins or lodge in the liver. Some eggs penetrate the walls of blood vessels, travel through the intestinal wall, and are excreted in feces. The life cycle is complete when the eggs hatch, releasing miracidia that then infect freshwater snails.³⁹

Clinical Manifestations

Neurologic manifestations of schistosomiasis occur after adult worms migrate to the brain or spinal cord. Neurologic involvement can present at any time during infection, but most often occurs months later.^{40,41} Rarely, neurologic symptoms are present within the first several days of the infection, as part of the Katayama syndrome, which causes urticarial rash, myalgias, and bloody diarrhea.⁴² More typically, patients present later with acute or subacute spinal cord involvement, with or without polyradiculitis, including cauda equina syndrome.⁴³ Characteristic features of spinal cord involvement include leg or radicular pain as well as bladder involvement.⁴⁴ Cerebral *S. japonicum* infection is due to deposition of *Schistosoma* eggs in the brain. Supratentorial lesions tend to cause seizures, whereas cerebellar schistosomiasis may cause mass effect with increased intracranial pressure.⁴⁵

Pathophysiology

It has been postulated that ova are carried to the spinal cord through arterial or retrograde venous blood flow via the valveless perivertebral Batson plexus, as evidenced by the predilection for involvement of the thoracolumbar and conus medullaris regions. There has also been some evidence for direct ova deposition without hepatic involvement.^{46,47}

Damage to the spinal cord is produced by the space-occupying effect of the granuloma and the immune reaction of the host against parasite antigens.^{48,49} Granulomas affecting the spinal roots produce congestion and edema. Various mechanisms through which *Schistosoma* eggs enter the brain have been proposed. Some experts hypothesize that eggs migrate through the circulation and settle in intracranial venous sinuses where they form foci of infection. Others theorize that eggs enter the portal venous system and are carried to the brain by the systemic circulation via portosystemic ramus anastomoses.⁵⁰

Diagnosis

Microscopic identification of eggs in brain tissue or cerebrospinal fluid (CSF) provides definitive diagnosis, but is rarely obtained. Detection of *Schistosoma* species by enzyme-linked immunosorbent assay (ELISA) in CSF can confirm the diagnosis of neuroschistosomiasis and is available in most countries with well-developed health care systems, but may be difficult to obtain in developing-world settings.⁵¹ Eosinophilia may be present in CSF. Positive ELISA of the stool or urine can provide additional diagnostic accuracy. Neuroimaging is an important diagnostic tool in neuroschistosomiasis. In cases of spinal cord involvement, lesions are most often present in the lower thoracic and lumbosacral cord. MRI typically reveals swelling of the spinal cord with an ill-defined hyperintensity on T2-weighted imaging and iso- or hypointensity on T1-weighted imaging. Postcontrast imaging reveals solitary or multiple irregularly enhancing patches (Fig. 4). When infection is chronic, medullary atrophy can be seen, with diffuse hyperintensity on T2-weighted imaging and variable degrees of medullary cone expansion.⁵² With cerebral involvement, CT demonstrates a hyperintense area surrounded by a hypointense region of edema. Multifocal lesions are uncommon. A linear enhancement pattern, surrounded by multiple nodular tree-like punctate foci, has been suggested to be pathognomonic of cerebral schistosomiasis (Fig. 5).⁵³

Treatment

Praziquantel is the most effective treatment of neuro-schistosomiasis.⁵⁴ An initial dose of 40 to 60 mg/kg is recommended. Because praziquantel is ovistatic and has little effect on eggs or immature worms, a second dose is required 4 to 6 weeks after initial treatment. Initiation of antiparasitic treatment within 30 days of symptom onset has been associated with a better outcome of neuroschistosomiasis.⁵⁵ For neurologic involvement, especially spinal cord, corticosteroids should be given concomitantly to counter immune overactivation and to decrease edema from the venous congestion caused by the eggs.^{56–60} Surgery is reserved for lesions causing mass effect, but is often performed as a diagnostic procedure, most often when a neoplasm is suspected, resulting in the diagnosis of neuroschistosomiasis.

NEUROCYSTICERCOSIS

Epidemiology

Cysticercosis is a parasitic infection that results from ingestion of eggs from the adult tapeworm *Taenia solium* (*T. solium*).⁶¹ Neurocysticercosis (NCC) occurs when cysticercoids migrate to the CNS. The disease affects over 50 million individuals worldwide, and has been estimated to cause at least 50,000 deaths each year. It is one of the most common causes of epilepsy in the developing world.^{61–63} *T. solium* infection is endemic throughout the world. In Latin America, seroprevalence rates range between 5 and 11%.⁶⁴ Neurocysticercosis is diagnosed with increasing frequency in high-income countries because of travel and increased immigration from endemic regions.^{65,66} Interestingly, in immigrants to the United States as well as in certain endemic areas (India), NCC more typically presents as a solitary intraparenchymal cyst, as opposed to the multiple cysts seen more often in endemic Latin American countries.^{67,68}

Life Cycle and Pathophysiology

Two types of infection from *T. solium* occur in humans: (1) taeniasis, an intestinal tapeworm infection caused by eating undercooked pork containing cysticerci (larva); and (2) cysticercosis, an infection caused by fecal-oral transmission of *T. solium* eggs excreted in the feces of a human with taeniasis.⁶⁹ A pig becomes infected by ingestion of parasite eggs, which then develop into cysticerci within pig muscles and organs. Humans, the definitive host, develop taeniasis by eating pork containing *T. solium* cysticerci; these cysticerci develop into adult worms within the human intestine, and produce eggs that are shed in feces. Cysticercosis occurs when humans ingest food contaminated with *T. solium* eggs or more rarely, via internal or external autoinfection. After ingestion of eggs, the embryos develop, hatch, and are liberated into the intestines.⁷⁰ Oncospheres cross the intestinal wall, enter the bloodstream, and are carried into the tissues of the host where they develop into larval cysts. In most circumstances, these cysts are rapidly destroyed by the host's immune system, except for those located in immunologically privileged sites, such as within the eye or nervous system.⁷¹ In the CNS, cysticerci go through a sequential evolution. Initially, they become established as viable cysts. The cysts remain in this stage protected by the blood-brain barrier and cyst-associated immune-evasion mechanisms for months or years; usually, the host's immune system eventually detects and destroys the cysts via cell-based immune response.^{72–74} Cyst contents gradually become dense and eventually calcified. The classic view of the evolution is that all cysticercoids evolve through vesicular, colloidal, granular-nodular, and calcified stages and that the duration of the process takes several years.⁷²

Clinical Manifestations

Symptoms and signs of NCC typically occur years after initial infection. Clinical manifestations vary according to the location, number, size, and stage of the parasites.^{75,76} Neurologic symptoms of NCC may be acute, chronic, or relapsing. Intraparenchymal cysts, which are the most common form of NCC, typically have a more benign course, whereas extraparenchymal locations (subarachnoid, ventricular, and cisternal) carry a more serious prognosis through focal mass effect or reactive edema to the cyst, which often give rise to symptoms associated with elevated intracranial pressure or hydrocephalus.⁷⁷ Fifteen percent of individuals with NCC have cysts in the ventricular system, the majority located in the fourth ventricle.⁷⁶

Seizures are the most common clinical manifestation of intraparenchymal and extraparenchymal cysts,⁷⁸ occurring in up to 70% of patients.⁷⁹ Cysts that are actively degenerating (colloidal and granular nodular cysts) are the most epileptogenic. Neurocysticercosis may also mimic stroke, tumor, or intracerebral hemorrhage.⁸⁰ When located in the subarachnoid space, cysts can give rise to focal mass effect or to an inflammatory response manifested as basilar meningitis.^{81–84} Communicating hydrocephalus and signs of elevated intracranial pressure may follow. An uncommon but serious form of infection is racemose NCC. This is a cluster of interconnected grape-like cysts without identifiable scolices located in the subarachnoid space, most often around the brainstem and frequently causing hydrocephalus. Another serious form of NCC that occurs more commonly in young women is encephalitis, which is often associated with severe cerebral edema. Spinal NCC is rare, with spinal cord involvement reported to occur in only ~1 to 5% of patients with NCC.^{85–87}

Diagnostic Studies

Any patient with new-onset epilepsy who has resided in a region with endemic *T. solium* infection should be evaluated for NCC. Definitive diagnosis of NCC can be made if there is (1) histopathologic evidence of NCC; or (2) a scolex within a cystic lesion visualized by CT or MRI; or (3) lesion(s) suggestive of NCC by neuroimaging with serologic evidence of *T.*

solium infection by serum enzyme-linked immunoelectrotransfer blot (EITB) or CSF ELISA (although CSF ELISA is less sensitive than serum EITB and therefore rarely necessary).^{15,88} One caveat for interpretation of serum EITB is that sensitivity drops substantially in cases with a solitary lesion or only calcified lesions.⁸⁹ Peripheral blood smear, eosinophilia, and CSF assessment is neither sensitive nor specific for *T. solium* infection.⁹⁰ Examination of the stool for ova and parasites may be performed in patients with NCC to evaluate for intestinal taeniasis.^{91,92} Close contacts should undergo serologic testing for *T. solium* infection and neuroimaging when serology is positive and neurologic symptoms are present, especially if they report seizures or headache. Imaging typically reveals single or multiple cysts with variable calcification, cyst wall enhancement, or surrounding edema. MRI findings of NCC correspond to the pathologic stage and location of the infection (Fig. 6): Vesicular lesions appear as a hypodense/hypointense collection without edema or enhancement; degenerating (colloidal and granular nodular) lesions demonstrate first rim- and then heterogeneous-enhancement with surrounding inflammation/edema; calcified cysts appear hyperdense on CT and may have increased signal on MRI susceptibility sequences, usually without enhancement or surrounding edema, although this can sometimes occur with a final stage of breakdown.^{93–95} Compared with CT, MRI is more sensitive for detection of the intraventricular (Fig. 7) and racemose forms of NCC, the scolex of the parasite, and additional cysts.

Treatment

Treatment recommendations depend upon the number, size, and location of cysts. Controversy exists regarding when and how long to treat NCC. Experts agree that most symptomatic NCC occurs during the inflammatory response induced by death of the cyst. If neuroimaging reveals only calcified cysts indicative of inactive infection, treatment with anthelmintics is not necessary. Lesions that are ring-enhancing represent dying parasites and expert consensus has not yet determined whether anthelmintic or corticosteroid treatment is beneficial. Homogeneously enhancing or hypodense lesions suggest active infection and should be treated with albendazole or praziquantel.⁹⁶ A typical regimen consists of 15 mg/kg/day of albendazole administered in two divided doses for 10 to 15 days. Clinical trials report more-favorable outcomes with albendazole, especially when subarachnoid cysts are present.⁹⁷ Pre-treatment with prednisone or dexamethasone for 2 to 4 days should be considered, especially for patients with a large number of cysts to reduce development of perilesional edema associated with anthelmintic treatment. Expert consensus has not been reached regarding the duration of anthelmintic treatment for large subarachnoid cysts, arachnoiditis, and cysticercotic encephalitis. Some experts believe repeated therapy over months to years is necessary. Regardless, concomitant steroids are recommended (e.g., prednisone 1 mg/kg) during treatment with albendazole and should be tapered off after anthelmintic treatment. Treatment of viable cysts has been associated with long-term improvement of seizures, and steroids are likely beneficial for solitary degenerating lesions as well.⁹⁸ Multiple studies have shown a decrease in the number and size of intraparenchymal lesions in patients treated with steroids.⁹⁹ However, the dosing, timing, and duration of steroids are not well-defined. Neurosurgical intervention should be considered for hydrocephalus, accessible racemose cysts in the basal cisterns, or a cyst in an intraventricular location.¹⁰⁰

CONCLUSION

With the increase in travel to developing countries and the dramatic rise in emigration from these regions, there is a high likelihood of encountering patients with tropical infections in our medical practice. For the neurologist confronted with an immigrant or returning traveler with neurologic symptoms, the challenge of diagnosing and treating a potential CNS

infection can be daunting. Obtaining a detailed travel and exposure history as well performing a thorough neurologic examination is essential. The neurologist should be able to formulate an initial list of potential etiologies and perform appropriate diagnostic testing, including laboratory studies and neuroimaging, to aid in the diagnosis. Experts in travel medicine are available in most major medical centers or via international groups, such as the Neuroinfectious Disease Section of the American Academy of Neurology, and should be consulted for assistance with diagnosis and treatment.

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World Map of Infectious Organisms

Figure 1.
World map of infectious organisms.

Figure 2. Malarial retinopathy. White-centered retinal hemorrhage (A) and orange vessels in a Malawian child with cerebral malaria. Macula retinal whitening (B) around the foveola (central dark disc) in a child with cerebral malaria. Cotton wool spots are also visible superotemporal to the optic disc. Vessel changes (C) in a Malawian child with cerebral malaria—from red to pale orange. Vessel changes (D) in a Malawian child with cerebral malaria—from red to white. (With permission from Idro R, Jenkins NE, Newton CR. Pathogenesis, clinical features, and neurological outcome of cerebral malaria. *Lancet Neurol* 2005;4:827–840. Photographs courtesy of Dr. Nicholas Bearre, Malari-Liverpool Wellcome Trust Clinical Research Program College of Medicine, Malawi.)

Figure 3.

A 31-year-old female patient with contrast-enhanced axial computed tomography scan showing effacement of cerebral cortical sulci and compression of the body of the ventricles, suggestive of generalized cerebral edema. (Reprinted with permission from Patanker TF, Karnad DR, Shetty PG, et al. Adult cerebral malaria: prognostic importance of imaging findings and correlation with postmortem findings. *Radiology* 2002;224:811–816.)

Figure 4.

A 30-year-old Brazilian-American man who presented with lower extremity pain and urinary retention found to have spinal schistosomiasis: Sagittal magnetic resonance images (MRIs) of the lumbar spine reveal (A) expansive intraspinal lesion (T1-weighted precontrast images); (B) enhancement of the inferior spinal cord, conus, and cauda equina nerve roots (postcontrast T-1 weighted images); and (C) hyperintensity in the cord that extends rostrally and caudally, reflecting secondary venous congestion or infarction (T2-weighted images). (With permission from Cho TA, Venna N. Neurologic infections. Hosp Physician Neurology Board Review Manual 2008;12(5):1–16. Copyright 2011 by Turner White Communications, Inc., Wayne, PA. All rights reserved.)

Figure 5.

A 67-year-old woman who presented with headache and seizures. (A, B) Axial contrast-enhanced magnetic resonance images show a large confluent enhancing mass (arrows) in the left frontal lobe and multiple small nodules in the periphery (arrowheads). (Reprinted with permission from Liu HC, Lim TC, Feng X, et al. MRI in cerebral schistosomiasis: characteristic nodular enhancement in 33 patients. *American Journal of Roentgenography* 2008;191:586.)

Figure 6.

A 40-year-old man from El Salvador presents with headache, nausea, and generalized seizure. (A) Fluid-attenuated inversion-recovery (FLAIR) and (B) T1 postcontrast magnetic resonance imaging (MRI) sequences demonstrating multifocal rim-enhancing cysts with mild surrounding edema in a degenerating neurocysticercosis lesion. A 44-year-old woman from Colombia presents with chronic left upper extremity tingling and pain. (C) Hyperintensity on non-contrast computed tomography, (D) hypointensity on FLAIR MRI, and (E) increased MRI susceptibility effect corresponding to calcified neurocysticercosis.

Figure 7.

A 33-year-old woman originally from El Salvador who presented with several weeks of headaches and double vision and decreased level of consciousness. (A) T1 axial and (B) T1 sagittal brain magnetic resonance imaging revealed a thin-walled cystic lesion in the fourth ventricle with an eccentric enhancing nodule suggestive of neurocysticercosis. (C) Expansion of the intraventricular cyst deforms the walls of the fourth ventricle, producing subependymal venous congestion with resulting signal abnormality noted on fluid attenuated inversion-recovery (FLAIR) images.