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Neuroparasitic Infections: Nematodes

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Abstract

Globalization has produced an increase in the number of people at risk for contracting parasitic infection. Central nervous system infection by nematodal parasites can be devastating. Early recognition and treatment of infection can significantly decrease morbidity of the parasitic infection, as well as the risk of secondary superinfection. The clinical presentation, diagnosis, and treatment for five of the more common nematodal infections of the nervous system—*Angiostrongylus* spp., *Baylisascaris procyonis*, *Gnathostoma spinigerum*, *Strongyloides stercoralis*, and *Toxocara* spp.—is reviewed.

Objectives—On completion of this article, the reader should be able to summarize the clinical presentation, diagnosis, and treatment of the common nematodal infections of the nervous system.

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Keywords

Parasite; nervous system; nematode

Nematodes, commonly known as “roundworms” because of their round cross section, comprise the second largest phylum in the animal kingdom. Nematodes can live freely but many parasitize humans, most often as accidental hosts. With increasing globalization and exotic travel, parasitic infection of the central nervous system (CNS), once considered a “tropical” infection, is becoming increasingly more prevalent in all parts of the world. In addition, immunosuppression increases susceptibility to opportunistic parasitic infection. Although infected individuals may remain asymptomatic for many years, a higher parasite burden is correlated with greater morbidity and mortality. The epidemiology, pathophysiology, clinical presentation, and recommended diagnostic evaluation and treatment for selected nematode infections are reviewed in this article (Table 1).

ANGIOSTRONGYLUS (ALICATA'S DISEASE)

Epidemiology

The earliest reported infection with *Angiostrongylus cantonensis* occurred in the rat population of Canton, China in 1933 and went virtually unnoticed until the first human case was reported in Taiwan in 1945.¹ Human infection is caused by ingestion of infected aquatic or terrestrial snails (usually *Lymnaea catascopium*), of slime produced by an infected snail or slug, or of certain shrimp and freshwater fish.² Infection with *Angiostrongylus* spp. is often asymptomatic and remains undetected for years, so recall of dietary history is often problematic. Abdominal disease due to *A. costaricensis* infection was recently reported in the Caribbean and Central America, but concomitant involvement of the CNS was not noted.^{3,4}

Pathophysiology

Mature *Angiostrongylus* worms reside in the pulmonary arteries of rodents and are thus commonly called “rat lung worms.”⁵ After entry into a host rat, adult female parasites lay eggs in the pulmonary vasculature. The eggs then hatch into young larvae that migrate to the pharynx, where they are swallowed and eventually excreted in the feces. Freshwater scavengers, such as shrimp, snails, crabs, and some fish, are invaded by the larvae and harbor the developing larvae. Mucus produced by infected snails also can be infective. Once ingested by a human host, the larvae migrate to the lungs or brain and die. *A. costaricensis*, unlike *A. cantonensis*, usually remains in the intestines. Tissue damage is common during migration, in part due to the host's inflammatory response to proteins secreted by the worm. Host immune status does not appear to affect disease severity.⁶

Clinical Findings

Symptoms typically develop after an incubation period of up to 2 weeks. Although systemic infection is rare, acute abdominal discomfort similar to appendicitis has been reported.⁷ Most infections produce neurological symptoms, most often a persistent headache. Additional symptoms are common and depend upon the extent of invasion and host inflammatory response.² There may be involvement of the cranial nerves, spinal cord, or the eye.⁸ Ocular involvement can occur with or without invasion of the CNS.⁹ Intraocular invasion can affect vision through destruction of tissue during migration or by causing retinal detachment. Although most infections present acutely, a chronic pain syndrome due to *Angiostrongylus* infection has also been reported.¹⁰ In addition, one-third of patients will develop hyperesthesia involving a limb or the trunk.¹¹

Diagnosis

Angiostrongylus spp. is one of several parasites that causes eosinophilic meningitis.¹² Cerebrospinal fluid (CSF) pleocytosis is common, with pronounced eosinophilia, increased protein concentration, and elevated opening pressure.¹³ Spinal fluid eosinophilia is usually present 2 to 4 weeks after symptoms develop, then wanes, returns again between weeks 6 and 8, then declines toward the end of the third month.¹¹ Definitive diagnosis is achieved by detecting larvae in biopsy tissue, or, more rarely, in the CSF. The diagnosis is more often based on clinical findings and exposure history. Detection of anti-*A. cantonensis* antibodies is both sensitive and specific, with sensitivity higher in CSF than in serum.^{14,15}

Neuroimaging

Computed tomography (CT) imaging may reveal hyper-intensities in the basal ganglia or contrast enhancement of the meninges.¹⁶ T1-weighted magnetic resonance imaging (MRI) postcontrast administration often demonstrates leptomeningeal enhancement and thickening, increased signal in the basal ganglia, as well as small hemorrhages seen with gradient imaging

(Fig. 1).¹⁶ Chronic infection often produces a granulomatous lesion that can be mistaken for tuberculosis.

Treatment

Treatment is supportive, with most infections being self-limited. Steroids and antiparasitic medications are ineffective. Older studies recommended periodic drainage of CSF to remove the nematode and any eggs that might be present; however, this therapy is no longer widely practiced.¹⁷ Recovery is usually complete, with children faring slightly better than adults.

BAYLISASCARIS

Epidemiology

The raccoon roundworm, *Baylisacaris procyonis*, is endemic in raccoon populations of North America and present in up to 80% of raccoons in the bicoastal and Midwestern United States.¹⁸ In a recent study of California communities reporting high densities of raccoons, 28 to 49% of surveyed properties had raccoon scat containing *B. procyonis* eggs.¹⁹ Only mild intestinal infection occurs in the raccoon, but parasites can reside within the small bowel of the raccoon for many years. Female adult procyonids produce millions of eggs per day, which are shed with the feces. These eggs are very resilient and can remain viable in the environment for years.²⁰ Ingestion of eggs by species other than the raccoon results in extraintestinal migration of the larvae, with 5 to 7% of migration leading to the brain, causing “neural larval migrans.”²¹ Children with pica, developmental delay, or exposure to raccoons are at highest risk for contracting *Baylisacaris* infection and resultant CNS infection.²² Severity of human disease is directly proportional to the number of larvae migrating to the brain, thus to the number of eggs ingested.²³

Pathophysiology

B. procyonis eggs are ingested by humans in the adult form. Larvae are infrequent in human infection, and unlike other parasites (such as *Toxocara*), the parasite grows as it migrates from the gastrointestinal tract to the CNS, with the adult size reaching up to 2 mm in length.²⁴ During migration to the CNS, neurotoxins are secreted by the developing procyonid and contribute to the formation of eosinophilic granulomas.

Clinical Findings

B. procyonis infection has been associated with eosinophilic meningoencephalitis, cardiac pseudotumor, and retinitis.²⁵ Children may develop retinitis, with or without concomitant encephalopathy.²⁶ Although adults rarely develop clinical signs of infection, mild cases of ocular larva migrans have been reported.²⁷ Children occasionally have a slowly progressive disease course, and the presence of a profound developmental delay without explanation should raise concern for this infection. Most infections are fatal or neurologically devastating.

Diagnosis

Definitive diagnosis is obtained through identification of *B. procyonis* larvae in a tissue sample.²⁸ CSF and peripheral eosinophilia are sometimes present but nonspecific. Stool examination is not helpful because eggs and larvae are not shed in stool. Anti-*B. procyonis* antibodies in CSF and serum can be detected via enzyme-linked immunosorbent assay or indirect fluorescent antibody, but neither test is available commercially.¹⁸ Both tests are specific for procyonids and do not cross-react with other ascarids (e.g., *Toxocara*).

Neuroimaging

Although often initially normal, with progression of infection the MRI eventually reveals deep white matter abnormalities and global atrophy (Fig. 2).²⁹ Head CT is usually normal.

Treatment

Unfortunately, *Baylisascaris* infection does not respond to treatment with anthelmintics. Early diagnosis is uncommon and when infection does become clinically evident, the extent of neurological damage is usually severe and, for the most part, irreversible. Ivermectin can reduce CNS eosinophilia but does not alter disease course.³⁰ Anti-inflammatory medications may be useful early in the course of disease but have not improved outcome. As treatment is largely ineffective, prevention is the best approach to reducing *Baylisascaris* infection.³⁰ Raccoons should be discouraged from nesting or eating pet food around children's play areas or residential backyards. Toys exposed to animal excrement should be destroyed because *Baylisascaris* is resistant to formalin and other typical decontamination efforts.³¹

GNATHOSTOMIASIS

Epidemiology

The definitive hosts for *Gnathostoma* spp. infection include dogs, cats, lions, leopards, minks, and raccoons.³² Travel through or residence in areas of endemic *G. spinigerum* infection, such as Southeast Asia and South America, increases the risk of contracting infection. Certain regional specialties are more likely to contain the parasite, including dishes containing raw fish (ceviche) in Latin America, sashimi in Japan, or fresh water eel (sumfak) in Thailand.^{33,34} Food handlers in endemic regions are at especially high risk of contracting infection through skin penetration. The parasite is not killed by soaking in lime, even after 5 days. Effective methods for eradication of larva include boiling in water for 5 minutes and soaking in vinegar for at least 6 hours or in soy sauce for 12 hours.

Pathophysiology

Adult worms reside within the gastric wall of the definitive host and discharge large quantities of eggs into the stomach. Eggs are excreted with the feces and, if exposed to water, hatch within 7 days. The gnathostome larva matures within the intermediate host, a tiny crustacean of the genus *Cyclops*. *Cyclops* is then ingested by a variety of waterborne organisms, including fish, crawfish, and eels.³⁵ Hearty gnathostomes can encyst within the host until conditions are appropriate for migration or reproduction. Human infection occurs by ingestion of infected water or animals or by introduction of the parasite through skin wounds. Adult gnathostomes reach 2 to 3 cm in length within definitive hosts but are often much smaller in accidental hosts, such as humans. In humans, larval forms do not migrate to the gastric wall as they do in other mammals but continue migrating through subcutaneous and visceral structures. The parasite can also enter the human host through skin wounds produced by rodents, fish, or cats. Once in subcutaneous tissue, the parasite usually migrates to the liver but can migrate to other areas.

Clinical Findings

Initial symptoms depend upon the mode of infection. If the organism was ingested, mild gastrointestinal distress usually occurs.³⁶ When skin penetration is the cause of infection, a migrating cutaneous swelling will develop.³⁷ Clinical symptoms are produced by parasite migration, host inflammatory response, and proteolytic and hemolytic toxins secreted by the gnathostome.³⁸ The classic manifestation of CNS gnathostomiasis is radiculomyelitis, presenting as severe radicular pain and paresthesia.³⁹ The pain usually lasts from 1 to 5 days and likely represents the time it takes for the organism to migrate from the nerve root to the CNS. Cranial nerves can also be affected but usually only later in the course of disease after

the spinal cord has been traversed.³⁹ Meningitis is uncommon, especially during early infection, but can occur in patients who are less severely affected or who have rapid rates of organism migration. Subarachnoid hemorrhage occurs in up to 25% of infected patients, and in Thailand, some experts suggest that almost one-quarter of hemorrhagic strokes may be caused by gnathostomiasis infection.⁴⁰⁻⁴²

Diagnosis

CSF examination often reveals a mild eosinophilic pleocytosis with xanthochromia or elevated red blood cell count.⁴² History of migratory subcutaneous swellings in a patient with eosinophilic meningoencephalitis or hemorrhagic stroke should suggest the diagnosis of gnathostomiasis. Extraction of the worm from infected tissue can provide the definitive diagnosis and treatment.²⁸ Serological tests, such as immunoblot detection of the 24-kDa band, are both sensitive and specific.⁴³

Neuroimaging

Neuroimaging often reveals intracranial hemorrhage or resultant obstructive hydrocephalus.^{40,41} Scattered foci of hyperintensity on T2 MRI are also described, usually bilaterally (Fig. 3). Contrast-enhanced imaging usually demonstrates meningeal involvement.

Treatment

Mebendazole and ivermectin are equally effective treatments for gnathostomiasis.⁴⁴ Gnathostomiasis can persist for up to a decade and may require multiple treatments to achieve total eradication.^{36,45} Surgical resection should be performed if an organism is identified. Adjunctive steroids have been useful in some cases.⁴¹ An increase in serum antibodies suggests treatment failure.

STRONGYLOIDES

Epidemiology

Strongyloidiasis is a human intestinal infection most often caused by *Strongyloides stercoralis*. Other subspecies, such as *S. fulleborni*, although pathogenic in primates, typically cause only minor infections in humans. Historically, strongyloidiasis had been confined to tropical and subtropical regions, but infection is increasingly common in Europe and the United States.⁴⁶ In the United States, cases are encountered primarily in tertiary medical centers, especially in the Mid-Atlantic region. In addition, with the advent of HIV infection and more frequent use of immunosuppressant medications, more people in the developed and developing world are at risk for contracting this infection and for developing hyperinfection.^{47,48}

Pathophysiology

Strongyloides spp. are capable of living as parasites or free-living organisms. There are three developmental stages: filariform (infective), rhabditiform, and adult. Rhabditoid larvae can become filariform or differentiate into male or female and maintain the rhabditiform cycle indefinitely. Strongyloidiasis is most prevalent in areas of poor sanitation and is usually acquired through contact with the parasite in contaminated water or by direct penetration of the skin by the filariform larvae. In addition, host autoinfection can occur when the parasite completes its life cycle within the host. After entering a human host, the parasite enters the venous circulation, migrates through the lung alveoli, and eventually burrows into the small intestine, where it can reside for up to 50 years.⁴⁹ From this site, worms can be released into the stool or develop into the filariform state and reinfect the host. Infection also facilitates coinfection with other agents, sometimes resulting in overwhelming bacteremia with dissemination to the CNS and other organs.⁴⁷ Disseminated *S. stercoralis* infection is more

likely to occur in the immunocompromised host.⁵⁰ Massive worm burden, known as hyperinfection syndrome, may occur when the usual parasitic life cycle is accelerated.⁵¹ Hyperinfection is usually limited to the gastrointestinal tract or lungs and is rare in the CNS.

Clinical Findings

Infection may persist for many decades without producing symptoms. Acute disease is limited to the gastrointestinal tract and lungs. Patients often develop wheezing, diarrhea, and postprandial abdominal pain.⁵² Transient low-grade fever is common. Chronic disease develops over days to weeks and usually includes a dermatological manifestation called *larva currens*.⁵³ The perianal region is the initial site of involvement, but most patients do not notice this early manifestation. The larvae migrate at a rate of up to 5 cm/d and travel subcutaneously or internally to other organs. Disseminated disease can produce infection in other organ systems, including the CNS.^{46,54} Alteration in mental status and meningismus are the most common manifestations of CNS involvement, but penetration of vessel walls can produce mycotic aneurysm and intracranial hemorrhage, even vasculitis.⁵⁵ If bacterial hyperinfection develops, brain abscess, caused by *Escherichia coli* in ~30% of cases, may produce focal neurological symptoms.⁵⁶

Diagnosis

Serum eosinophilia is common during primary infection but wanes with dissemination of infection.²⁸ Diagnosis can be confirmed by identification of *Strongyloides* spp. rhabditiform larvae in stool, serum, CSF, or peritoneal fluid. Larvae do not appear in the stool until approximately 1 month after initial infection. In patients with disseminated infection, larvae may also appear in the sputum. Larvae can be detected in duodenal secretions with the Entero-test (Hedero, Palo Alto, CA); a weighted gelatin capsule is swallowed, the gelatin dissolves allowing the string to pass into the duodenum and 4 hours later the string is removed and examined for larvae. Due to the low sensitivity of direct identification tests, testing of serial samples is recommended, especially for stool specimens. If strongyloidiasis is suspected but not detected by direct identification tests, antibody detection testing should be performed.^{57, 58} Unfortunately, antibody detection tests cannot distinguish between past or present infection, can be negative in patients with disseminated infection, and may cross-react with other helminthic and filarial infections. Of the available antibody tests, enzyme immunoassay has the highest sensitivity (~90%).⁵⁸

Neuroimaging

In patients with chronic infection, neuroimaging is often nonspecific, but atrophy may be prominent (Fig. 4). Additional abnormalities include abscess formation or mycotic aneurysms, either of which may occur along any vascular distribution but usually spare the extracranial vascular system.⁵⁹

Treatment

Ivermectin is the treatment of choice, but thiabendazole, albendazole, and mebendazole are also effective.⁶⁰ Steroids should not be used during acute infection, as they may promote dissemination. Disseminated disease carries a mortality rate of almost 80%, so early detection and treatment is imperative.^{47,61}

TOXOCARA

Epidemiology

Toxocariasis is endemic in all parts of the world.⁶² Most human *Toxocara* spp. infections are caused by *T. canis*, but *T. cati*, and *T. leonina* infections also occur. Recent studies suggest that

living in a rural area, ownership of dogs, and dementia are associated with a higher risk for CNS *T. canis* infection.⁶³ Children who eat earth (geophagia, pica) are also at higher risk of becoming infected. An infected dog or cat can excrete up to one million eggs each day, and eggs can survive in the environment for many years.⁶⁴ Some experts have disputed the role of dogs as vectors of transmission, noting that up to half of patients do not own a pet and cannot recall any close animal contact.⁶³ In northern industrialized countries, seroprevalence of this infection is 5% in urban adults and up to 40% in children and rural farmers. In the West Indies and Bali, seroprevalence rates approach 80%.^{65,66}

Pathophysiology

Introduction into a human host is accidental and occurs by ingestion, most often on contaminated hands.⁶⁷ Once in the human gastrointestinal tract, eggs remain in the small bowel for a short period, hatch into second-stage larvae, and then migrate to the liver. Larvae then enter the portal circulation and migrate through small-caliber vessels to the viscera, producing a mild inflammatory response along the migratory path.²⁸ Chronic inflammation can eventually induce granuloma formation. Migration to the brain is uncommon but usually produces a more dramatic inflammatory response than migration through the periphery.⁶⁸

Clinical Findings

Toxocariasis is almost always a benign self-limited disease, but ocular or cerebral involvement can cause significant morbidity, and infection in the elderly can be fatal.⁶⁹ Symptoms depend on the disease burden and system infected, but weakness, lethargy, fever, and headache are common.⁶⁸ Visceral larva migrans occurs mainly in children with disseminated infection and can produce granulomas in the liver, lungs, kidneys, heart, muscle, brain, or eye.⁷⁰ Ocular involvement, known as ocular larva migrans, can produce symptoms of optic neuritis or blindness and occurs when the parasite migrates to the optic nerve head.⁷¹ Infection can also produce ocular findings similar to retinoblastoma; as retinoblastoma is treated by enucleation, toxocariasis should be excluded as an etiology before such treatment is considered.⁷² Although the majority of human *Toxocara* spp. infections are asymptomatic, subtle cognitive symptoms may not be appreciated or attributed to other behavioral conditions.⁷³ Unlike other human nematode infections, cognition is affected during almost all chronic *Toxocara* spp. infections and can range from hyperactive behavior in children to dementia in elderly adults.⁷³

Diagnosis

Toxocara spp. larvae are only rarely identified in clinical specimens. Because the parasite enters the human host as a mature adult, eggs are not isolated in stool.²⁸ Detection of eggs from other organisms, such as *Ascaris* and *Trichuris*, suggests exposure to fecal material where *Toxocara* may also reside.⁷⁴ Serum and CSF eosinophilia is a frequent finding, but treatment can reverse this abnormality, and chronic infections may have a blunted eosinophilic response.^{67,68} Antibody testing for second-stage *T. canis* excretory-secretory larval antigens (TESAg) is sensitive and specific for visceral larva migrans provided the serum is pretreated to remove cross-reacting antibodies to organisms such as *Ascaris suum*.⁷⁵ TES-Ag testing can be performed on blood or CSF samples.

Neuroimaging

Contrast-enhanced head CT often demonstrates vasogenic edema and a heterogeneous enhancement pattern resembling malignant gliomas (Fig. 5; personal communication, Dr. Nezhik Oktar). MRI imaging can reveal subcortical and white matter disease, resembling small vessel vasculitis. Imaging abnormalities suggestive of small infarctions on FLAIR and T2 sequences often demonstrate microhemorrhages on gradient-echo sequences.⁷⁶

Treatment

Although diethylcarbamazine is the treatment of choice, mebendazole and albendazole are also effective against toxocariasis.⁷⁷ Suggested length of treatment is 3 to 4 weeks. In patients with ocular involvement, steroids should be administered and an ophthalmologist consulted.⁷⁸

CONCLUSION

Nematodal infection of the CNS includes a large and diverse variety of parasites. Although many of the published cases are from tropical and subtropical countries, the incidence of many parasitic infections is increasing throughout the world, due to a combination of increased global travel and immunosuppression. In addition, some nematodal infections, such as *Baylisacaris procyonis*, have caused infections mainly within the United States. The indolent course of many of the nematodal infections make identification difficult, but prompt recognition and diagnosis of some of these infections can prevent additional morbidity and mortality.

Treatment for nematodal infection varies according to the organism (Table 2) but is often limited to management of symptoms. Once a nematodal infection has been identified, a tropical medicine expert should be consulted to assist with determining the appropriate treatment, as treatment regimens do change and newer investigative medications may be available. Several online references are useful for obtaining assistance with diagnosis and expert consultation:

- The World Health Organization Fact Sheet, available at <http://www.who.int/inf-fs/en/index.html>
- Centers for Disease Control, Division of Parasitic Diseases, available at <http://www.dpd.cdc.gov/dpdx/>
- The Medical Letter, available at <http://www.medletter.com/index.html>

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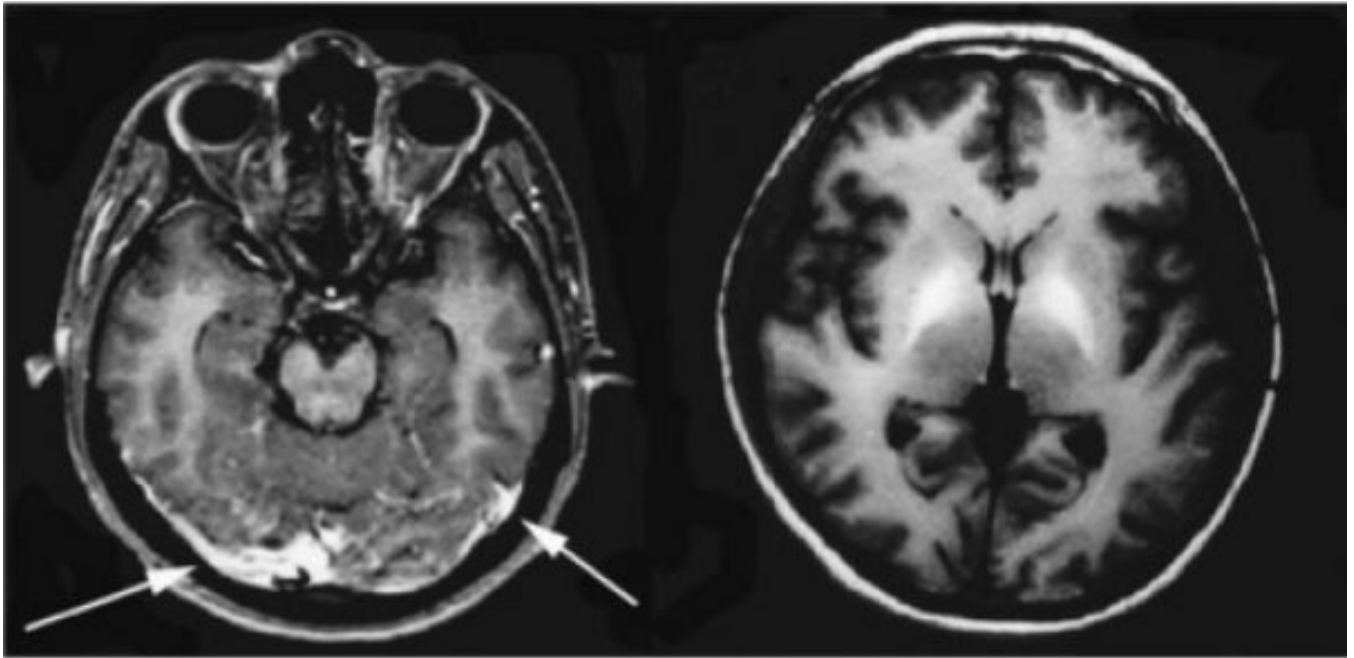


Figure 1. Patient with *Angiostrongylus* infection. Axial T1 contrast-enhanced images demonstrate meningeal enhancement (left, arrows) and markedly increased signal intensity within the globus pallidus (right). (Reprinted with permission from Tsai HC, Liu YC, Kunin CM, et al. Eosinophilic meningitis caused by *Angiostrongylus cantonensis* associated with eating raw snails: correlation of brain magnetic resonance imaging scans with clinical findings. *Am J Trop Med Hyg* 2003;68:281–285.)

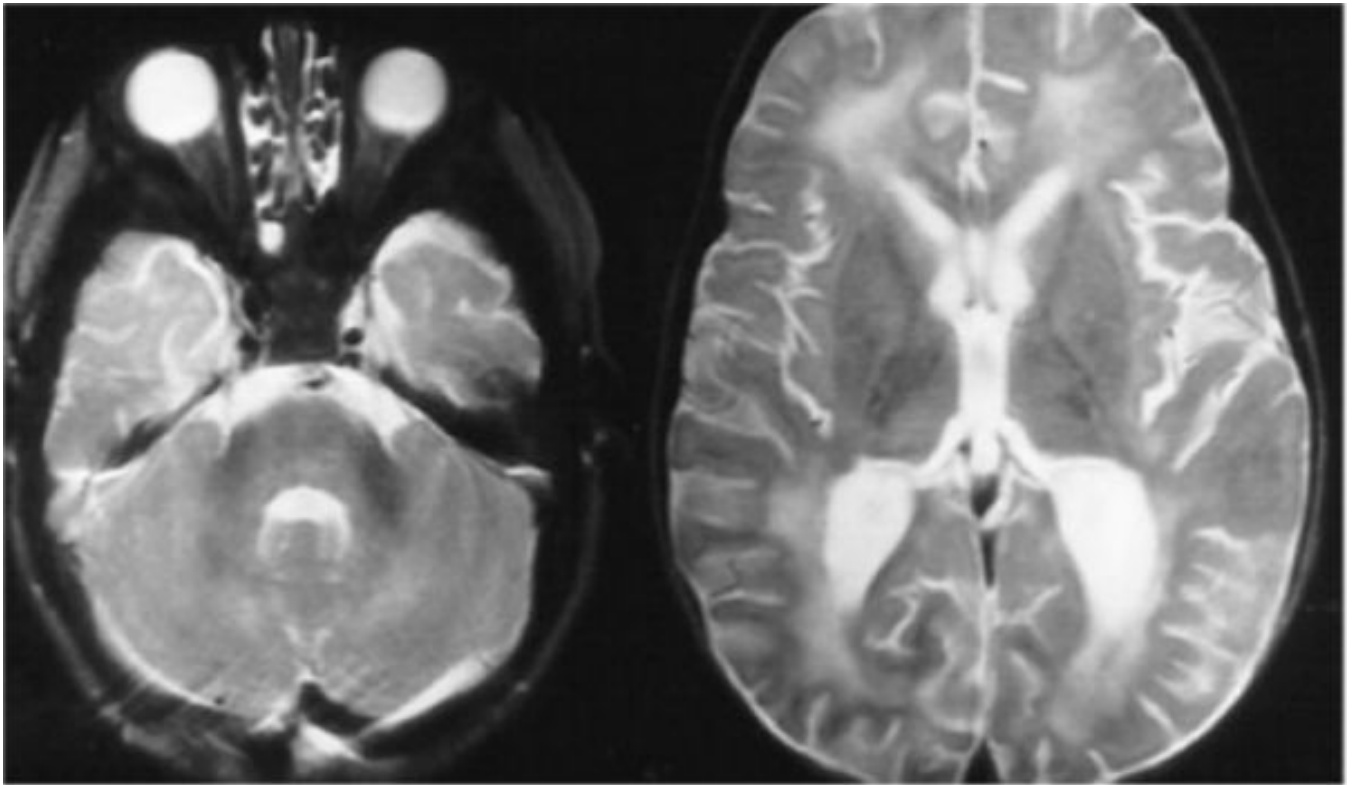


Figure 2. Thirteen month-old patient with *Baylisascaris* infection. Bilateral, patchy T2 hyperintensity is seen predominantly in the white matter, including the periventricular regions and corpus medullaris of the cerebellum. (Images courtesy of Dr. Howard Rowley.)

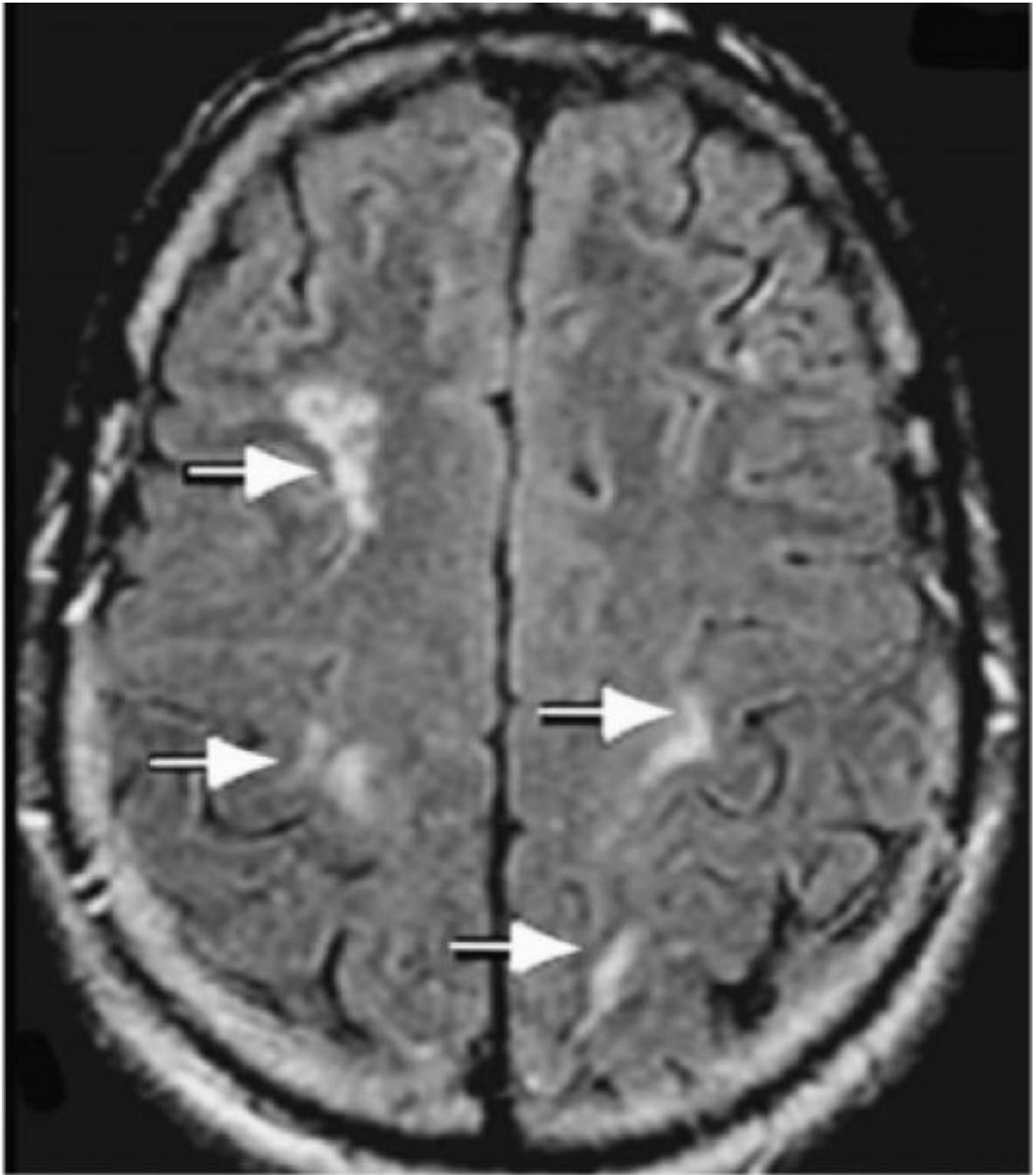


Figure 3. Patient with *Gnathostoma* infection. Axial FLAIR MRI of the brain showing bilateral clusters of small rounded hyperintensities in deep white matter (arrows). (Reprinted with permission from Hughes AJ, Biggs BA. Parasitic worms of the central nervous system: an Australian perspective. Intern Med J 2002;32:541–553.)

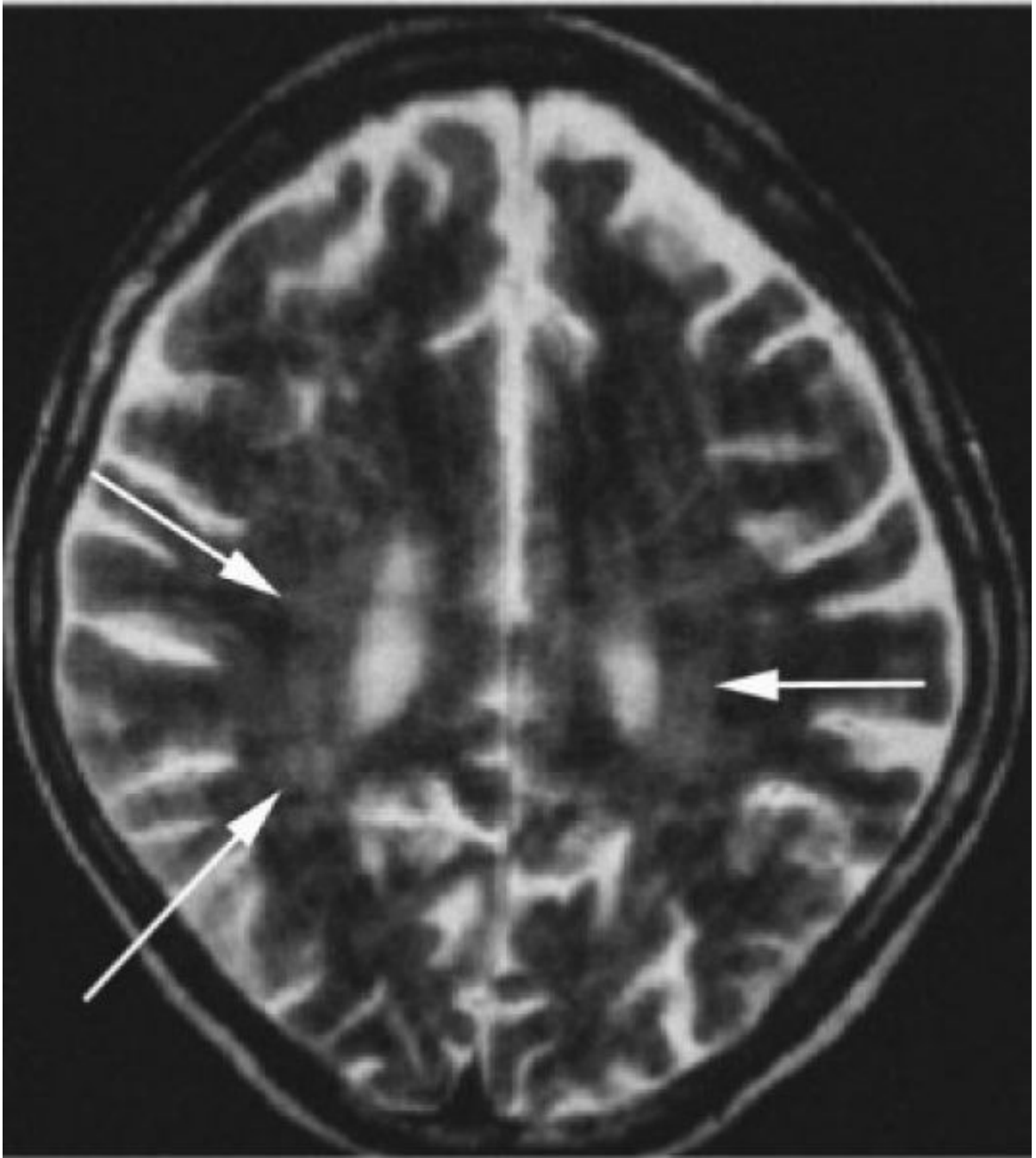


Figure 4. Thirty-five-year-old immunosuppressed patient with *Strongyloides* infection. Axial T2-weighted images reveal global atrophy and patchy periventricular white matter hyperintensities (arrows). (Reprinted with permission from Kothary NN, Muskie JM, Mathur SC. *Strongyloides stercoralis* hyperinfection. Radio-graphics 1999;19:1077–1081.)

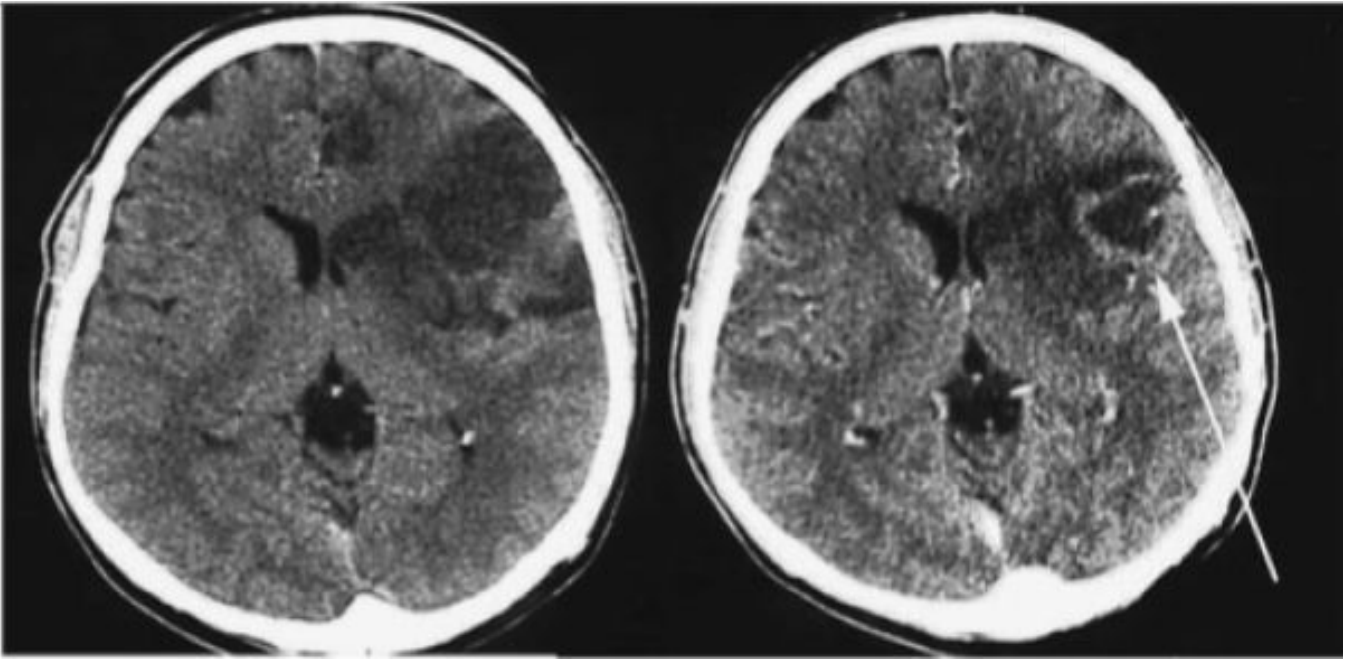


Figure 5. Patient with *Toxocara* infection. Noncontrast CT imaging demonstrates left frontal hypodensity consistent with edema (left), and contrast-enhanced imaging reveals a ring enhancing lesion (arrow on right). (Reprinted with permission from Oktar N, Barçin E, Kazandi A, Korkmaz M. Cerebral *Toxocara* mimicking a malignant glioma. *Norol Bil D* 2002;19:#12.)

Table 1
Imaging Findings of Selected Nematode Infections of the CNS

Parasite	Type of Lesion	Location	Other Features
Angiostrongylus	High T1 signal in basal ganglia and/or cerebral peduncles	White matter, deep gray matter of cortex	Meningeal enhancement, edema; CT may show ring-enhancing lesions
Baylisascaris	Global atrophy, patchy regions of T2 hyperintensity	White matter of cerebral cortex and cerebellum	Ventriculomegaly, more common in children than adults
Gnathostomiasis	Small clusters of rounded hyperintensities on FLAIR and T2	Near gray-white junction of cerebral cortex	Usually bilateral; CT may show intracerebral hemorrhage
Strongyloides	Global atrophy, periventricular white matter hyperintensities on T2 and FLAIR	White matter, spinal cord	Abscess may occur, usually unilateral; meningeal enhancement may be seen especially in spinal lesions; can superinfect tumors and other lesions
Toxocara	Single or multiple lesions, which are low-signal on T1, hyperintense on T2-weighted MR images, and homogeneously enhancing	Gray and white matter, cortical and subcortical; may occur in the spinal cord	Hypodense lesions on CT with variable enhancement patterns, may be mistaken for gliomas

For additional information and references, please refer to text.

Table 2

Treatment of Selected Nematode Infections of the CNS

Parasite	Medication	Dosage	Precautions	Potential Side Effects
Angiostrongyliasis	Symptomatic care			
Baylisascariasis	Symptomatic care			
Gnathostomiasis	Albendazole	400 mg PO bid for 21 days; steroid use controversial	Use of concurrent steroids or praziquantel may cause toxicity	Abdominal pain, jaundice, alopecia
Strongyloidiasis	Ivermectin; steroids may cause dissemination	200 mcg/kg/d PO for 2 days; may repeat in 14 d	Avoid use in first term of pregnancy	Mild; generally very well tolerated
Toxocariasis	Mebendazole	25 mg/kg/d PO single dose for 4 weeks	Caution in patients on anticonvulsants or medications metabolized by the p450 system	Jaundice, abdominal pain, headaches, alopecia

For treatment options and further information, please consult The Medical Letter or contact the CDC.