Parasitic Diseases that Cause Seizures

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The propensity of parasites to invade the brain, cause disease, and give rise to seizures and epilepsy varies greatly. This reflects the wide diversity of parasites that differ in fundamental ways. This manuscript reviews parasites that invade the brain or its vasculature and give rise to seizures.

Parasites are a disparate group of organisms that commonly differ from one another in fundamental ways. They can be broadly classified into single-celled organisms (protozoa) or multicelled helminthes (worms). A limited number of parasites frequently infect the human brain; some cause symptomatic disease regularly while others cause few, if any, symptoms except under certain conditions, such as immunosuppression. Parasites specific to humans can accidentally be located in abnormal or ectopic locations, including the brain or spinal cord. A relatively large number of animal parasites infect humans, sometimes migrating through or lodging in the tissues including the brain. Many animal parasites have been found in the human brain, but these are rare occurrences, considered medical curiosities, and almost never diagnosed short of histological identification. However, a limited number characteristically infect the brain and give rise to recognizable clinical syndromes.

Although infections of individual parasites are unique and reflect their biological diversity, much of the pathology of protozoa is related to their ability to multiply in the human host, sometimes to very large numbers as opposed to infection with helminths. The ability of protozoa to multiply in the host is enhanced by immunosuppression. This explains why almost all of the severe parasitic opportunistic infections in AIDS patients are protozoan parasites. Even though helminths, with a few exceptions, do not multiply in the human host, they have the propensity to cause great harm because they are relatively large, many times macroscopic organisms that cause disease by physical disruption of tissue as they migrate and by engendering an intense commonly eosinophilic inflammatory response. Inflammatory response to ova deposited in tissues is another common pathophysiological mechanism of disease. Some helminth larvae can be very large, causing disease because of their enlarging mass.

Helminths are multicelled, relatively complicated organisms that are classified into nematodes, trematodes, and

cestodes types of worms based upon a number of fundamental basic differences in their biology, structure, organization, life cycle, and biochemistry. There are substantial differences within groups, so the pathophysiology of infection and disease frequently differ. Helminth infections tend to provoke a peripheral eosinophilia as they migrate through the tissues but wane upon sequestration in the organs, including the brain. For the most part, helminths lodged in the brain do not cause a systemic eosinophilia, particularly if infection occurred months to years earlier. If there is an inflammatory response to these parasites, eosinophils are localized to the brain. If the parasite or its antigens have access to the CSF, eosinophils may be detected there.

In the setting of a consistent exposure and clinical context, eosinophilic meningitis suggests a helminth infection of the brain or spinal cord.

Seizures have been described in virtually every parasitic infection involving the human brain. However, with the exception of *Taenia solium* cysticercosis and malaria due to *Plasmodium falciparum*—which are likely responsible for most of the seizures due to parasitic disease worldwide (1)—the presence, proportion, or number of seizures due to parasites are not cataloged in the literature. Therefore, there are no firm data to compare the incidence of seizures in parasites or the numbers of patients affected. A sense of the prevalence of seizures in the various parasitic infections follows and is shown in Table 1, based upon a selected review of the literature. The pathophysiology and mode of infection among the varying parasitic entities frequently differ. A brief description is given for parasites of interest. For a more complete understanding, the reader is referred to chapters in specialized texts (2), reviews (3–8), or original reports.

Selected Organisms that Cause CNS Infection and Disease Protozoa

Malaria caused by *Plasmodium falciparum* is one of the most common causes of CNS infection and acute seizures worldwide (1, 9–11). As the trophozoite stage in the red blood cell matures into schizonts, they sequester and adhere to the small venules of the brain and other organs; this occurs by way of the PfEMP1 family of varying surface proteins produced by

TABLE 1.

	Trivial Name
Protozoa	
*Plasmodium falciparum	malaria
<i>Trypaosoma</i>	African sleeping sickness
<i>Rhodesiense</i>	
<i>T. gambiense</i>	
<i>Naeglaria fowleri</i>	Primary amebic encephalitis
<i>Acanthamoeba</i> sp	Granulomatous amebic encephalitis
<i>Balmuthia madrillilaris</i>	
Toxoplasma gondii	toxoplasmosis
Trypanosoma cruzi	Chagas' disease
<i>Microsporidia</i> spp	microsporidiosis
Nematode	
<i>Gnathostoma</i> sp	Gnathosomiasis
<i>Angiostrongylus cantonensis</i>	Eosinophilic meningitis
Toxocara canis	Visceral larval migrans
Toxocara cati	Toxocara infection
<i>Bayliascaris procyonis</i>	Larval migrans Menino-encephalitis Larval migrans
<i>Strongyloides stercoralis</i>	Disseminated strongyloidiasis
<i>Trichinella</i> sp	Trichinosis
<i>Loa loa</i>	Loiasis and treatment
Trematode	
Paragonimus sp	Paragonimiasis
<i>Alaria americana</i>	Larval migrans
S. mansoni S. hematobium S. japonicum <i>S. mekongi</i> <i>S. intercalatum</i>	Schistosomiasis
Cestode	
Taenia solium	Cysticercosis
<i>Taenia multiceps</i>	Coenurosis
<i>Echinococcus granulosus</i> and sp <i>E. alveolaris</i>	Hydatid disease
Spirometra sp	Sparganosis

the parasite and exported to the surface of the red blood cells (12, 13). The pathophysiology of brain involvement is likely multifactorial but is related to the degree of parasitemia, sequestration of schizonts in the brain venules, and the resulting

vascular and perivascular damage. Suggested mechanisms of injury include anoxia, parasite toxin, vascular leakage, increased intracranial hypertension, metabolic derangements, stroke, and others (14).

In Africa, seizures are a frequent reason for hospitalization (1, 15). In one hospital ward, seizures accounted for 15.8% of the total admissions, and 69% of those were due to *falciparum* malaria (1). Malaria accounted for about 1/3 of all cases of seizures seen in an emergency room setting in Africa (15). The most common etiology was febrile seizures (15).

Seizures are the most common neurological complication of acute *falciparum* malaria, occurring in 25% of cerebral malaria (CM) and 75% without defined CM (1, 16). A history of seizures is found in up to 90% (16, 17) of those with CM; in a large retrospective series of all malaria cases admitted from a highly endemic region in Africa, 37.5% of all children hospitalized with malaria presented with a history of seizures in the setting of acute malaria (9). In this series, occurrence was uncommon before 6 months of age, increased to 48.6% at 27 to 33 months, were more likely with a higher parasitemia, and had a shorter course of disease. Fifty-six percent had more than one seizure, and 22% of those with seizures experienced prolonged seizure episodes lasting 7 to 30 min.

Recurrent seizures during hospitalizations occur in between 24 percent and 62 percent (12) of patients with CM and is one of a number of the predictors of severe sequelae or death (10, 14, 18–20). Development of multiple seizures and status epilepticus is sometimes mentioned as leading to a poor outcome as well.

The presence of seizures in patients without CM brings into question whether seizures in malaria are, in actuality, febrile seizures brought about by fever due to malaria. Although this may be the cause in some cases fever due to malaria, it is not felt to be the case in CM (1, 12, 14). A majority of patients have seizures not characteristic of febrile seizures: They commonly occur in persons who are not febrile, and seizures are commonly recurrent—many times complex and focal in nature.

Seizure activity was closely observed and monitored prospectively using EEG recordings every 12 hr during the course CM by Crawley et al. (18) in 65 of 110 admitted patients. Sixty-eight percent and 42% had a history of seizures and status epilepticus, respectively. During observation, 38% had no seizure activity, while 43% had one or more partial seizures, and 18.4% had generalized seizures. They found that 25% had subtle clinical seizures, as suggested and verified by EEG recordings that would have likely gone unnoticed. They suggested that inability to arouse some of these patients might have been due to status epilepticus. This selected group of patients had a particularly grim outcome, as 11% died and 12% had severe sequelae, including one patient who developed epilepsy at 1 month following discharge.

How many patients develop epilepsy following CM? Van Hensbroek et al. (16) prospectively followed 624 children with CM. Of the 466 out of 490 survivors, 5 had seizures at 1 month and another at 6 months and 18 months, suggesting the development of epilepsy in the short term is infrequent—a result similar to Brewster et al. (10), who found about 1% of CM developed epilepsy. Carter et al. (21) retrospectively compared children with a history of CM with complicated seizures, uncomplicated CM, and a control population. Active epilepsy was noted in 6.4%, 4.6%, and 1.1%, respectively, suggesting CM malaria with complicated seizures predisposes to epilepsy.

The etiology of CM and seizures is beyond the scope of the discussion. However, there are many features of a diffuse encephalopathy with an increase in intracranial pressure in the most severe cases (14, 22) and increase in brain fluid (23), only occasionally with edema and infarcts in many of the most severely affected. EEG studies in patients with partial epilepsy localized activity to the watershed parietal temporal region, which suggests that hypoxia is likely playing a role.

African Trypanosomiasis

The protozoan parasites *Trypanosoma gambiense* and *T. rhodesiense* cause two usually distinct clinically syndromes of African sleeping sickness. *T. rhodesiense*, a normal parasite of urbivores in East Africa, is transmitted by the tsetse fly (24). Visitors to game parks are accidentally infected after being bitten by an infected fly, resulting in an acute febrile illness. CNS involvement commonly occurs within weeks postinfection. In contrast, the infection caused by *T. gambiense* affects predominantly humans; it is spread by tsetse flies and the disease is usually insidious over months to years. While *T. gambiense* causes epidemics that can devastate areas of Africa, *T. rhodesiense* infects humans uncommonly. The late or second stage of disease is characterized by CNS involvement. Although there is occasional mention in texts (25, 26) of seizures in patients. Case reports (27, 28) and large series of *T. gambiense* (24, 29) fail to list seizures as a manifestation. However, treatment with melarsoprol may give rise to neurological syndromes associated with seizures (30). Treated and recovered patients are not reported to have an increased prevalence of seizures or epilepsy.

Toxoplasmosis

Infections due to *Toxoplasma gondii*, a coccidian protozoan parasite, frequently result in an asymptomatic systemic infection in humans that commonly, if not uniformly, involves the brain as well as other tissues. Eye manifestations are common in congenital infections and sometimes in acquired infections. CNS manifestations may occur as a consequence of congenital infection, rarely as encephalitis (31) or mass lesions in immunocompetent individuals (32), but most frequently in AIDS and the immunosuppressed. Patients later develop intensely inflammatory, necrotic lesions, causing diffuse and localized symptoms including headaches, confusion, mass effects, focal deficits, and seizures (33). Congenital involvement may be acute (34) or chronic with seizures occurring in 1.1 to 7.9 percent after 1 to 7 years (35).

Chagas' Disease

Infections with *Trypanosoma cruzi* usually occur after the bite of an infected reduviid bug. Meningoencephalitis occurs rarely in acute disease—usually in children less than 2 years of age—and CNS manifestations are not a regular feature in chronic disease (36). However, patients with chronic long-standing asymptomatic infection may recrudesce and develop meningoencephalitis that is clinically and radiologically indistinguishable from toxoplasmosis as a consequence of immunosuppression or AIDS. Seizures are a common manifestation in this situation (36). *T. cruzi* can also be transmitted by infected blood or organs, causing acute meningoencephalitis in recipients.

Free-Living Amoebae

Acanthamoeba spp., *Naegleria fowleri*, *Balamuthia mandrillaris*, and occasionally other species are a diverse group of free-living amoebas that can rarely cause a severe, commonly fatal multifocal meningoencephalitis (37). They differ in biology, host preference, clinical course, and epidemiology. *Acanthamoeba* spp. results in CNS and systemic infection, mostly in the immunosuppressed and in AIDS (granulomatous amebic encephalitis). The course is semi-acute, lasting weeks, and is commonly associated with seizures. *Balamuthia mandrillaris* affects both immunocompetent and immunosuppressed individuals. Skin infections, commonly involving the face, can predate CNS involvement. Seizures have been described. *N. fowleri* causes the most fulminant infection (primary amebic encephalitis), usually resulting in death in less than a week. Infections occur in immunocompetent individuals when amoebae infect the brain through the cribriform plate in persons exposed to contaminated fresh water, usually while swimming in warm lakes or ponds. Seizures are not usually a clinical feature in this infection. *Entamoeba histolytica*, a common cause of colitis and liver abscess, can rarely metastasize to brain.

Helminth Infections of the Brain

Eosinophilic Meningitis Due to *Angiostrongylus cantonesis*

Infection with this nematode, the rat lungworm, is a common cause of acute eosinophilic meningitis primarily in the Far East, but small epidemics have been reported more recently from the Caribbean and elsewhere. Infections are acquired after ingestion of raw snails, contaminated vegetation, or other animals that have ingested infected snails and thereby acquired infectious larvae. After ingestion, the larvae recapitulate the migration in the rat to the meninges, causing eosinophilic meningitis. Serious manifestations and mortality occur uncommonly but may be seen in heavy infections and when the larvae invade the brain parenchyma. Consequently, seizures are an infrequent manifestation (39).

Schistosomiasis

Schistosomiasis, a major infection of the tropics, is caused by a number of species of intravascular-dwelling trematodes. Even though complications are uncommon, spinal and CNS infections are not unusual at referral centers because of the considerable number of persons infected. Humans are infected after cercariae—released into the water from freshwater snails—burrow through the skin and eventually find their way into the venules of the intestine (*Schistosoma japonicum*, *S. mansoni*) or urinary tract (*S. hematobium*) and deposit ova in the respective organs. Alternatively, some ova are carried by the bloodstream, primarily to the liver and less so to the lungs. Ova incite a granulomatous response that results in most of the disease manifestations. CNS disease occurs as a consequence of a significant inflammatory response to ova abnormally present in the spine or brain through embolization or deposition by ectopic adult worms. Acute encephalitis, usually in the presence of a systemic illness with fever and eosinophilia, occurs in about 2 to 3 percent of cases of acute schistosomiasis (Katayama fever) due to *S. japonicum* (40). Multifocal and nonfocal findings are typical (41), but seizures are uncommonly mentioned. Chronically infected patients occasionally develop tumor-like

granulomatous masses in the brain. Systemic manifestations are lacking, but seizures are a frequent symptom (42). Both *S. mansoni* and *S. hematobium* may occasionally involve the spinal cord in both early and late infections; brain involvement rarely occurs but is a cause of seizures (43).

Paragonimiasis

Cerebral paragonimiasis (44, 45) is caused by a number of species of *Paragonimus* through ectopic migration of worms to the brain or embolization of ova through blood vessels. Among the many species of this trematode that can infect humans, *P. westermani* is most common. Snails release cercariae that then embed into the tissues of crabs or crayfish, where they develop into infectious metacercariae. After ingestion, these hatch and migrate through the bowel wall and make their way to the lung, forming lung cavities. Ectopic migration is not infrequent and, in part, depends on the species. The route of migration to the brain is unsettled, but depending on the reported series, worms are varyingly found in the brain together with eggs, inciting acute or chronic inflammation. In longstanding cases, common in older reports, “soap bubble calcifications” are characteristic of infection. Seizures and epilepsy are present in most patients, usually with accompanying focal deficits.

Visceral Larval Migrants

Ingestion of significant eggs of animal *Ascaris* species—most commonly *Toxocara cati* and *Toxocara canis*—give rise to systemic eosinophilic syndromes caused by larvae migrating through the tissues. Disease is caused by granulomatous inflammatory response to the larvae (46). CNS involvement including death is uncommon but well described. Seizures have been described in from 6 to 51 percent (47). They were recurrent in 9 of 14 patients in one series (549). A number of reports have correlated partial epilepsy with higher prevalence of antibodies to *T. canis*, suggesting prior infection is a cause of epilepsy (50, 51).

Strongyloidiasis

Strongyloides stercoralis is a frequent intestinal infection in the tropics and in immigrants but is also found in the United States, particularly in Appalachia. Although usually asymptomatic and low grade, in the immunosuppressed host (commonly those on corticosteroids) and the malnourished, the larvae produced by the female in the small intestine reinvade in the large intestine (autoinfection) or the perianal skin, often resulting in a massive infection termed “hyperinfection syndrome.” Larvae course through the bloodstream and tissues, causing CNS disease, many times accompanied by secondary bacterial meningitis originating from the denuded bowel wall (52). Seizures are not common. Although eosinophilia is frequent in chronic low-grade infections, it is not reliably found in a majority with hyperinfection syndrome.

Trichinosis

Trichinella spp. cause disease following ingestion of infective larvae present in undercooked infected meat—traditionally pork but also a large number of other mammals. These develop into adult worms in the intestine, produce numerous

larvae offspring that enter the bloodstream and subsequently invade and develop in muscle cells. Symptoms and signs include myositis, carditis, and inflammation of other organs, including the brain, caused by invasion or aborted development. The characteristic syndrome is an acute febrile disease with eosinophilia, diarrhea, edema, facial swelling, and other systemic signs and symptoms. Neurological manifestations are uncommon but, when present, are frequently multifocal and nonspecific. Both pathology and MRI suggest the presence of microinfarcts. Seizures are rarely present as reflected in recent reports (53–55) but are mentioned in the older literature.

Larval Cestodes

The life cycle of cestodes has two morphologically unique stages, usually alternating between two different hosts. The tapeworm resides in the intestine of the definitive host and produces infectious ova; following ingestion, they develop into larvae in the tissues of the intermediate host. All larval infections of humans are enzootic; humans are unintended accidental hosts and varying susceptible to infection and larval development. With the exception of *T. solium*, larval cestode infections of the brain are rare in humans. Most cause symptoms because the cystic larval become large and cause mass effects; there may be some degree of accompanying inflammation.

T. solium Cysticercosis

Neurocysticercosis, infection with the larval form of the tapeworm *T. solium*, usually affects the brain and is a common cause of seizures in endemic regions due to degenerating viable cysts and calcified cysticercal granulomas. Studies in Latin America and India show about 29% of active epilepsy in endemic areas is attributable to this infection (38). Neurocysticercosis is reviewed elsewhere in the present series.

Two species of *Echinococcus* may involve the brain: *E. granulosus* and related species commonly involve the liver and lungs of humans but can be found in almost any tissue or organ, including the brain, and most characteristically as cystic masses (56, 57). The second species, *E. multilocularis*, frequently infects the human liver but grows aberrantly as a membranous proliferating mass, inciting a chronic inflammation. It rarely metastasizes to the brain and commonly appears as a multicystic inflammatory mass (58). The larval infection of *Taenia multiceps* and related tapeworms manifest in humans as cystic masses (coenuri) of the parenchyma or spaces of the brain, usually with accompanying inflammation (59). The pathophysiology is similar to *T. solium* neurocysticercosis. The larval infection of the animal tapeworm genus *Spirometra*, called sparganosis, usually presents as inflammatory parasitic masses that may occasionally migrate within the brain. CT shows varying amounts of hypodensities, nodular enhancing lesions, and punctate calcifications. Infections are mostly chronic, and seizures are particularly common (60).

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