PARASITIC INFECTIONS OF THE NERVOUS SYSTEM, WITH AN UPDATE ON FREE-LIVING AMOEBIC INFECTIONS

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Summary

Although the central nervous system (CNS) of humans is not a frequent site of primary infection by parasites, many different protozoa and helminths can affect the brain and spinal cord. This article first briefly reviews these different parasites and their principal clinical manifestations when the CNS is involved. The second part of this article deals at greater length with the amoebae that are known to cause primary infections of the central nervous system, and the disease entities associated with these infections.

Parasitic infections of the CNS: causative agents and clinical presentations

The protozoan infections that effect the nervous system include those caused by amoebae, flagellates and coccidia (Table 1), while a range of nematodes, cestodes and trematodes are also known to cause neurological disorders (Table 2).

Table 1. Protozoan parasites that cause diseases of the nervous system

Parasite species	Clinical condition
Amoebae	
Entamoeba histolytica	Amoebic brain abscess
Naegleria fowleri	Primary amoebic meningo-encephalitis
Acanthamoeba spp	Granulomatous amoebic encephalitis, keratitis
Balamuthia mandrillaris	Granulomatous amoebic encephalitis
Flagellates	
Trypanosoma bruceirhodesiense and *T.* b. gambiense	Encephalitis (in Human African Trypanosomiasis or sleeping sickness)
Trypanosoma cruzi	Encephalitis
Coccidia	
Toxoplasma gondii	Encephalitis
Plasmodium falciparum	Cerebral malaria

Table 2. Helminth parasites that cause diseases of the nervous system

Parasite species	Clinical condition
Nematodes	
Parastrongylus (Angiostrongylus) cantonensis	Eosinophilic meningo-encephalitis
Strongyloides stercoralis	Meningitis in disseminated strongyloidiasis
Toxocara spp	Visceral larva migrans
Gnathostoma spinigerum	Eosinophilic meningo-encephalitis
Trichinella spiralis	Epilepsy or encephalitis
Onchocerca volvulus	Encephalopathy
Loa loa	Encephalopathy
Cestodes	
Taenia solium	Neurocysticercosis gives rise to epilepsy, hydrocephalus or organic dementia
Echinococcus granulosus	Hydatid cyst of the brain or spinal cord
Echinococcus multilocularis	Alveolar echinococcosis
Spirometra	Cerebral sparganosis, seizures
Trematodes	
Schistosoma japonicum	Epilepsy
Schistosoma mansoni	Transverse myelitis, spinal cord granulomas and anterior spinal arteritis
Paragonimus westermani	Seizures

Clinical presentations

The clinical manifestations may be of relatively acute onset or more frequently, insidious and slow. On a global scale, the best-known of the parasitic infections that present acutely is cerebral malaria, a complication of *P. falciparum* infections. Human African Trypanosomiasis (or sleeping sickness), caused by *T. brucei rhodesiense* and *T. brucei gambiense*, also causes encephalopathy within a few weeks of disease onset in the former and in the chronic stages of the disease in the latter. In American trypanosomiasis caused by *T. cruzi*, nervous system involvement could occur in the acute stage.

There are several other, less well-known parasitic infections that cause **encephalopathy.** Toxoplasmosis (primary or re-activatedinfection) is well known to cause neurological disease in immuno-compromised patients, particularly those with organ transplants and HIV / AIDS. Cases of encephalopathy temporally related to mass treatment with ivermectin for onchocerciasis have been reported; most of these cases recovered without serious consequence (Boussinesq et al 2003). Fatalities usually occur in those who are co-infected with *Loa loa* and have a high microfilaraemia. A similar fatal encephalopathy has been described in patients infected with *Loa loa* who have been treated with DEC (Kamgno et al 2008).

Among the organisms that cause **meningo-encephalitis** are the pathogenic free-living amoeba, Naegleria fowleri and the helminths which cause eosinophilic meningitis. **Angiostrongylus cantonensis** is the most common cause of eosinophilic meningitis worldwide. The rat is the definitive host of the parasite and humans are infected by ingesting third-stage larvae, which develop in a molluscan intermediate host, usually slugs or snails (Bunnag, 1999).

Though neurological manifestations of infection with *Toxocara larvae* (toxocariasis, also known as visceral larva migrans) are rare, it is an important differential diagnosis of various neurological disorders. Manifestations of involvement of the central nervous system include dementia, meningo-encephalitis, myelitis, cerebral vasculitis, epilepsy or optic neuritis (Finsterer & Auer, 2007). A review of the literature from the early 50's to the present date found 29 cases of brain involvement in toxocariasis (Moreira-Silva et al, 2004). Of these 28 cases, 20 reported different clinical and laboratory manifestations of eosinophilic meningitis, encephalitis, myelitis or radiculopathy.

The presence of encysted larval stages in the brain may also cause patients to present with seuures such as in neurocysticercosis and in trichinellosis. Neurocysticercosis, infection of the brain parenchyma with the larval stage of *Taenia solium*, is a common cause of focal and generalised seizures of late onset, but it less commonly presents as headache, Parkinsonism, or other neurologic abnormalities. Heavy cyst burden in neural tissue can cause encephalopathy with fever, headache, nausea and vomiting, altered mental status, and seizures (Kraft, 2007). Cerebral sparganosis presenting as seizures caused by sparganum, the larval stages of the cestode *Spirometra spp.* is of importance as surgical removal of the larva results in a complete cure (Alibhoy et al, 2007).

Central nervous system involvement may occur in chronic schistosomiasis caused by any schistosome species, but especially by *S. japonicum*. The usual presentation is focal or generalized tonic-clonic seizures and focal deficits. Transverse myelitis is the most common neurologic manifestation of *S. mansoni* infection, which rarely affects the brain. The possible mechanism of central nervous system involvement is the embolization of eggs or ectopic migration of adult worms (Ross et al, 2002).

Among the other trematodes, *Paragonimus* westermani causing paragonimiasisis a major cause of neurological disease in the Far East, due to cyst formation in ectopic sites in the brain and presents as intracranial space occupying lesions.

Chronic helminth infections such as hydatidosis, may also present with the features of a slow-growing, space-occupying lesion in the nervous system.

In the current era of international travel, changing lifestyles, eclectic food habits and climate change due to global warming, it is particularly important to be aware of the range of parasitic infections that could present with neurological disease. It is also worthwhile remembering that although very few of the parasites that are natural parasites of humans have a predilection for the CNS, in many zoonotic infections, helminth larvae migrate to brain tissue and persist there.

Pathogenic free-living amoebae: the parasites

The pathogenic free-living amoebae are rare causes of human disease. *Acanthamoeba* meningitis and *Naegleria* primary meningo-encephalitis were first reported in 1965 and 1966 in southern Australia and Florida (Intalapaporn et al, 2004). There are now four genera of these amoebae, which have been implicated as aetiological agents of human disease. These are

- *Naegleria fowleri*
- Acanthamoebaspp
- *Balamuthia mandrillaris*
- *Sappinia diploidea*

Together, they are known to cause three distinct clinical syndromes

- Primary amoebic meningo-encephalitis (PAM)
- Granulomatous amoebic encephalitis(GAE)
- Amoebic keratitis (AK)

While *N. fowleri* causes PAM, *Acanthamoeba spp* are known to cause both GAE and AK. B. *mandrillaris* and *S. diploidea* also cause GAE.

Naegleria spp. are primarily soil amoebae that are washed off into surface waters and as such are found worldwide in a wide variety of warm stagnant or slow flowing surface water bodies. Recently these amoebae have also been detected in ground water such as wells (Blair et al, 2008). In Sri Lanka, ***Naegleria spp.*** have been isolated from river water in the Kandy area and irrigation tanks in the Dry Zone (Wijesundera et al 1996, Gunarathna, 2009). Of the 47 species identified on molecular basis to date, only ***N. fowleri*** is known as a human pathogen while two other species ***N. italica*** and *N. australiensis* are now recognized as potential pathogens (De Jonckeer, 2002). The *Naegleria* isolated in Sri Lankan waters is yet to be speciated.

N. fowleri is thermophilic, growing at temperatures upto 45°C. The trophozoite stages thrive in large bodies of warm water such as found in heated swimming pools and thermal springs. Chlorination does not affect its growth. It is a facultative pathogen capable of living many generations without infecting a host. A wide range of animals in addition to humans can be hosts to these amoebae.

The life cycle includes three morphological forms: the amoebic trophozoite, which is the feeding, growing, multiplying form; the flagellate trophozoite, which is rapidly motile; and the dormant cyst stage. The amoebic trophozoite, which is about 10 - 30 µm in diameter, has a single nucleus with a large nucleolus. It is usually found on surfaces of vegetation and mud; it feeds on bacteria. The flagellate form, which typically has 2 flagellae and a single nucleus, is found in the surface layers of warm bodies of water. The cyst is spherical with a smooth, single wall; it is about 10 µm in diameter and is also found in the same locations as the amoebic trophozoite. Human infection usually occurs while bathing or swimming, when the amoebae in the infected waters enter through the olfactory epithelium in the nose. They penetrate the epithelium, pass along the olfactory nerve branches in the cribriform plate to enter the meninges and multiply in the perivascular spaces. The amoebic trophozoites are found in the brain tissue and CSF. The flagellate forms are also occasionally seen in the CSF. When amoebae are present in the CSF, transformation into the flagellate form may be precipitated and visualised by the addition of distilled water to a wet smear.

The symptoms and signs, and changes in the cerebro-spinal fluid are like those of purulent bacterial meningitis, but there is no response to anti-bacterials. Infection is almost invariably fatal.

Like *N. **fowleri,*** the ***Acanthamoeba spp*** that infect humans are also found worldwide, feeding on bacteria. However, they are not necessarily associated with warm fresh water, and can also multiply in brackish conditions (Warhurst, 2008). *Acanthamoeba spp* also have trophozoite and cyst forms, but the trophozoite has only the amoeba stage. It is large (20 - 40 µm in diameter), with a single nucleus and its surface is covered with tiny projections that look like spines (acanthopodia). It is sluggishly motile. The cysts have a polyhedral shape and a double wall. They are about 15 µm in diameter. The widespread, ubiquitous presence of these organisms means that humans are frequently exposed to them, but the parasite usually finds it difficult to colonise humans. Organisms may enter through the nasal passages into the lower respiratory tract, through ulcerated or broken skin or the eye. Infections usually occur in the patients who are immuno-compromised or debilitated in some other way. They are of a chronic type, with a marked granulomatous reaction.

Balamuthia mandrillaris is a leptomyxid free-living amoeba, first described as causing disease in 1990. This species is also found worldwide, in soil and water, but the particular ecological niche occupied by the parasite is as yet unknown. It was first isolated from the brain of a mandrill baboon at the San Diego Wild Animal Park. Several reported cases of ***B. mandrillaris*** had initially been diagnosed as being due to ***Acanfhamoeba.*** Following the development of an immunofluorescence assay, a number of human cases of ***B. mandrillaris*** meningoencephalitis were diagnosed retrospectively (Intalapaporn et al, 2004).

As with other amoebae, B. ***mandrillaris*** has two morphological forms: the trophozoite and the cyst. Trophozoites, which range in size from 12 to 60 µm, are uninucleated with a large, densely staining nucleolus. There may be two or thee nucleoli in some trophozoites. Cysts may be more readily visualised with either GMS or periodic acid-Schiff stains. They range in size from 6 to 30 µm and appear to be double-walled and three-walled by light and electron microscopy, respectively. Immunofluorescence studies are required for differentiating between *B. mandrillaris* and *Acanthamoeba.*

The parasites may enter the human host through the nasal tissue and thence via the lower respiratory tract and circulation or through ulcerated or broken skin. Both forms may be found in the brain tissue of infected individuals.

A single case of amoebic encephalitis thought to be due to ***Sappinia diploidea,*** a soil-living amoeba, was reported in 2001 (Gelman et al, 2001). More recent work using newly developed real-time polymerase chain reaction assays, has suggested that the organism is most likely to be S. ***pedata.*** This amoeba had previously been found only in environmental sources, such as soil and tree bark (Qvarnstrom et al, 2009).

The clinical conditions

Primary amoebic meningo-encephalitis (PAM) caused by N. *fowleri* has a worldwide distribution.

However, disease is rare and to date only 200 cases have been documented. The majority of cases are from developed countries, probably due to availability of reference diagnostic facilities. A few cases have been

reported in India and Thailand (Shenoy et al, 2002). Although the free-living organism been found in Sri Lanka, no cases of PAM have been reported in Sri Lanka to date.

Classically, the disease occurs in healthy young adults or teenagers with a history of swimming in the type of water-body known to be favoured by N. *fowleri* It appears likely that although many people are exposed to infection, for unknown reasons, only a few actually develop disease. Invasion of the brain through the olfactory bulb and cribriform plate results in a rapidly progressive, purulent haemorrhagic necrosis of the frontal cortex, starting with the olfactory bulbs. The pathology is very similar to that of purulent bacterial meningitis. The incubation period in PAM ranges from a few days to about 2 weeks. Patients may complain of distortions in taste and smell at the onset of the illness. They soon develop clinical features of acute meningitis, with fever, headache, vomiting and neck stiffness. Illness rapidly progresses to deep coma, and convulsions may occur. Patients usually die within a week of onset of symptoms.

The CSF usually has a high white cell count, predominantly with polymorphs. It also has a high protein content and low glucose levels. Trophozoites are not easily seen in a Gram stain; they are better visualized with iron haematoxylin stain. A wet mount of CSF may show motile trophozoites. Definitive diagnosis requires direct demonstration of organism. Formation of the rapidly motile biflagellate form may be precipitated by addition of a drop of distilled water to the CSF; this may be observed in an unstained, wet mount, under a regular microscope.

Although the clinical features and pathological changes resemble those of purulent bacterial meningitis, PAM does not usually respond to anti-bacterial agents. However, systemic and intrathecal amphotericin B have been used in patients who have survived. Adjunctive therapies with rifampicin, miconazole, sulfisoxazole have also been tried, but the benefit remains unknown. Most cases described the in literature to date, have been fatal.

Granulomatous Amoebic Encephalitis has also been reported worldwide. One hundred and fifty-six human cases of GAE have been reported from 1956 through 1997 at the Centers for Disease Control and Prevention (Atlanta, GA) 63 of them caused by *B. mandrillaris* (Intalapaporn, 2004). Unlike PAM, GAE due to *Acanthamoeba* is a disease of immuno-suppressed or otherwise debilitated patients. *Balamuthia* can also cause GAE in patients who are otherwise healthy. The mode of acquisition of infection is not always clear. Acanthamoebae may be present as a commensal in upper respiratory tract of normal individuals.

When Acanthamoeba *spp* or B. *mandrillaris* enters a human host, the primary foci are usually in lungs and skin. The organisms spread haematogenously from there to CNS. The brain involvement is frequently patchy: thalamus, diencephalon, brain stem and posterior fossa. The chronic inflammatory, granulomatous response is characterised by multi-nucleated giant cells. Both trophozoites and cysts present in lesions.

The onset of changes in mental status is usually insidious, with focal neurological deficits, seizures, fever, headache and visual abnormalities. The duration of illness varies, but is usually about a month. Skin lesions may be seen at the original site of infection.

Multiple, non-enhancing lesions may be seen on CT. Lumbar puncture is contra-indicated; in any case, amoebae are not found in CSF. Diagnosis is usually made at autopsy, and brain sections will show granulomatous lesions with amoebae and cysts. Concurrent skin lesions may be biopsied to make an indirect diagnosis. The differentiation between *Acanthamoeba* and *Balamuthia* in tissue sections requires immunohistochemistry.

As with PAM, GAE is also nearly uniformly fatal. Diamidines such as pentamidine, propamidine have shown the greatest in *vitro* activity. Azole antifungals and some aminoglycosides such as neomycin and paromomycin have also been tried, but with little success.

Amoebic keratitis caused by infection with *Acanthamoeba spp* is usually associated with minor trauma to the eye or use of contact lenses. Especial risk factors include soaking contact lenses in homemade saline solutions, swimming with contact lenses, and use of extended wear contact lenses. The organism has been isolated from a Sri Lankan patient with central corneal ulceration (Wijesundera et al, 2001).

The initial clinical features of amoebic keratitis include the sensation of a foreign body in eye, tearing, photophobia and ocular pain. In full-blown infections, iritis, dendriform keratitis, hypopyon, increased intra-ocular pressure, and anterior nodular scleritis may occur. Laboratory diagnosis may be made by examining corneal scrapings. Wet mounts can show motile trophozoites and cysts, while fixed slides can be stained with Giemsa or periodic Acid Schiff reagent or calcofluor white to visualize the amoebae. If the patient uses contact lenses, the contact lens and storage system should also be examined for the presence of amoebae. Acanthamoeba keratitis is curable if detected early. The affected areas of cornea should be debrided. Specific treatment with topical propamidine, together with neosporin and miconazole is required for about 1 month.

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ACINETOBACTER *BAUMANNII* – A SUCCESS STORY OF A NOSOCOMIAL PATHOGEN

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The most important representative of the genus *Acinetobacter*, *Acinetobacter baumannii*, has emerged as one of the most troublesome pathogen for healthcare institutions globally. It has a remarkable ability to up-regulate or acquire resistant determinants. *Acinetobacter baumannii* resistant to all known antibiotics have now been reported, signrfying a sentinel event which need prompt action by the international healthcare community. Acting in synergy with this emerging resistant profile is the uncanny ability of *A.* baumannii to survive for prolonged periods throughout hospital environment, thus potentiating its ability for nosocomial spread (Peleg AY et al).

Acinetobacter spp have been implicated in variety of nosocomial infections, including bacteraemia, urinary tract infections, nosocomial meningitis. However, its predominant role is as agent of nosocomial pneumonia, particularly ventilator associated pneumonia in patients confined to hospital intensive care units (ICU). In more recent times, infection involving central nervous system, skin and soft tissue, and bone have emerged as highly problematic for certain institutions.

Microbiology

The genus *Acinetobacter*, as currently defined, comprises gram-negative, strictly aerobic, non fermenting, non fastidious, non motile, catalase positive, oxidase negative bacteria with a DNA G+C content of 39% to 47%. Acinefobacter species of human origin grow well on solid media that are routinely used in clinical microbiology laboratories. These organisms form