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Neuroparasitology and Tropical Neurology: Chapter 24 ...
Ocular Baylisascaris procyonis infection results from ingestion of infective eggs of B. procyonis, the raccoon ascarid. Herpes simplex virus type 2 (HSV-2) infection of the retina is the result of either primary infection or reactivated disease. Herein, we report a case of a 12-year-old female resident of the Bronx in New York City, who presented with pan-uveitis and vision loss.

Human African trypanosomiasis or sleeping sickness is a neglected tropical disease that affects populations in sub-Saharan Africa. The disease is caused by infection with the gambiense and rhodesiense subspecies of the extracellular parasite Trypanosoma brucei, and is transmitted to humans by bites of infected tsetse flies. The disease evolves in two stages, the hemolymphatic and meningoencephalitic stages, the latter being defined by central nervous system infection after trypanosomal traversal of the blood–brain barrier. African trypanosomiasis, which leads to severe neuroinflammation, is fatal without treatment, but the available drugs are toxic and complicated to administer. The choice of medication is determined by the infecting parasite subspecies and disease stage. Clinical features include a constellation of nonspecific symptoms and signs with evolving neurological and psychiatric alterations and characteristic sleep–wake disturbances. Because of the clinical profile variability and insidiously progressive central nervous system involvement, disease staging is currently based on cerebrospinal fluid examination, which is usually performed after the finding of trypanosomes in blood or other body fluids. No vaccine being available, control of human African trypanosomiasis relies on diagnosis and treatment of infected patients, assisted by vector control. Better diagnostic tools and safer, easy to use drugs are needed to facilitate elimination of the disease.

Neglected tropical diseases are a group of mostly infectious diseases that thrive among poor populations in tropical countries. A significant proportion of the conditions affecting the neurological system in such countries can be attributed to neglected tropical diseases of helminth, protozoan, bacterial, or viral origin. The neurological burden of neglected tropical diseases has not been thoroughly investigated yet, but is expected to be significant; its full appreciation, estimation, and recognition present significant challenges, as shown by the case of the "silent epidemic" of epilepsy. While tropical infections involving the nervous system are today largely preventable or treatable, as vaccines or chemotherapeutic agents are available to kill or neutralize the responsible agents, associated morbidity – when established – cannot be cured. In resource-poor settings it is likely that many infections will not be treated and will therefore progress into their advanced and severe stages, thus being increasingly associated with irreversible morbidity; this is also the case for neurological morbidity, which often entails permanent disability. Public health should aim at reducing the burden of tropical neurological diseases through interventions addressing the infection, the associated morbidity, and the disability deriving from it.

The nature of many parasitic infections of the central nervous system (CNS) requires immunodiagnosis to confirm presumptive diagnoses. The CNS is the primary site of parasite infection for some parasitic organisms and for others, neurological infection occurs only in immunocompromised hosts. Still other parasites cause ectopic infections of the CNS and occur very rarely. This review concentrates on laboratory diagnosis of diseases that are caused by parasites with a primary predilection for the CNS. Emphasis is placed on laboratory diagnostic methods that are used and suitable for clinical diagnosis, rather than a comprehensive review of all the experimental methods that have been reported in the literature. Immunodiagnosis is not appropriate for the diagnosis of all parasitic infections of the CNS; in those cases, alternative diagnostic methods are presented, but not discussed in detail. In some instances potential new antigens or methods are presented, particularly if adoption of these methods is expected in the near future.

Fascioliasis is a worldwide, zoonotic disease caused by the liver trematodes Fasciola hepatica and Fasciola gigantica. Neurological fascioliasis has been widely reported in all continents, affecting both sexes and all ages. Two types of records related to two physiopathogenic mechanisms may be distinguished: cases in which the neurological symptoms are due to direct effects of a migrating juvenile present in the brain or neighboring organ and with cerebral lesions suggesting migration through the brain; and cases with neurological symptoms due to indirect immuno-allergic and toxic effects at distance from flukes in the liver. Neurological manifestations include minor symptoms, mainly cephalalgias, and major symptoms which are nonspecific, extremely diverse, varying from one patient to another and even within the same patient, and comprising meningeal manifestations and impressive neurological manifestations. The puzzling neurological polymorphism leads to confusion with cerebral tumors, multiple sclerosis, lesions of the brainstem, or cerebro-meningeal hemorrhages. Only blood eosinophilia and information on infection source guide toward correct diagnosis by appropriate coprological and/or serological techniques. Although neurological patients usually recover after fasciolicide treatment or surgical worm extraction, sequelae, which are sometimes important, remain in several patients. The need to include possible neurological complications within the general frame of fascioliasis becomes evident.

Parasitic infections of the central nervous system (CNS) include two broad categories of infectious organisms: single-celled protozoa and multicellular metazoa. The protozoal infections include malaria, American trypanosomiasis, human African trypanosomiasis, toxoplasmosis, amebiasis, microsporidiasis, and leishmaniasis. The metazoal infections are grouped into flatworms, which include trematoda and cestoda, and roundworms or nematoda. Trematoda infections include schistosomiasis and paragonimiasis. Cestoda infections include cysticercosis, coenurosis, hydatidosis, and sparganosis. Nematoda infections include gnathostomiasis, angiostrongyliasis, toxocariasis, strongyloidiasis, filariasis, baylisascariasis, dracunculiasis, micronemiasis, and lagochilascariasis. The most common route of CNS invasion is through the blood. In some cases, the parasite invades the olfactory neuroepithelium in the nasal mucosa and penetrates the brain via the subarachnoid space or reaches the CNS through neural foramina of the skull base around the cranial nerves or vessels. The neuropathological changes vary greatly, depending on the type and size of the parasite, geographical strain variations in parasitic virulence, immune evasion by the parasite, and differences in host immune response. Congestion of the leptomeninges, cerebral edema, hemorrhage, thrombosis, vasculitis, necrosis, calcification, abscesses, meningeal and perivascular polymorphonuclear and mononuclear inflammatory infiltrate, microglial nodules, gliosis, granulomas, and fibrosis can be found affecting isolated or multiple regions of the CNS, or even diffusely spread. Some infections may be present as an expanding mass lesion. The parasites can be identified by conventional histology, immunohistochemistry, in situ hybridization, and PCR.

Parasitic infections of the central nervous system (CNS) have increased over the last couple of decades, partly due to a drop in the living conditions of large populations in the world and the AIDS epidemic. Parasitic infections of the CNS are indolent and often life threatening, hence, an early diagnosis is imperative. While brain biopsy and laboratory analysis remain the gold standard for diagnosis, neuroimaging contributes significantly to diagnosis and follow-up. Imaging can demonstrate the extent of infection and complications and possibly, the type of parasitic infection when characteristic features are evident. The disappearance of the parasite or inflammation, gliosis, and/or calcification suggest a therapeutic response. The initial experience of the CT scan has been greatly enhanced by MRI which is currently the imaging modality of choice. This has been due to the greater tissue contrast resolution of MRI and its ability to detect subtle changes in the tissue parenchyma. Advanced techniques such as diffusion-weighted imaging (DWI), perfusion imaging (PI), MR angiography (MRA), and MR spectroscopy (MRS) have been used to improve the sensitivity for characterizing the type, viability, and burden of the parasites and the host tissue response. Additionally, it is possible to demonstrate the complications of the primary infection and those secondary to treatment, in some cases.

Babesiosis is a worldwide emerging infectious disease caused by intraerythrocytic protozoa that are transmitted by Ixodid ticks, or less commonly through blood transfusion or transplacentally. Although headache and lethargy are common symptoms, babesiosis is uncommonly associated with specific neurological dysfunction in humans. Decreased level of consciousness or coma are rare complications that are associated with severe and often fatal disease but the pathogenesis is unclear.

Cysticercosis, an infection caused by the cystic larvae of the pork tapeworm Taenia solium, is one of the most frequent parasitic infections of the human nervous system (neurocysticercosis). It is endemic in most of Latin America, the sub-Saharan Africa, and vast parts of Asia, including the Indian subcontinent. It has also been increasingly diagnosed in developed countries because of migration of people from endemic zones and exposure in travelers. The life cycle involves the development of the adult tapeworm in the human small intestine (after ingesting infected pork with cysts) and larval infection in pig tissues (after ingesting human stools containing the eggs of the tapeworm). Humans get infected by the fecal-oral route, most often from a direct contact with an asymptomatic Taenia carrier. Most common clinical presentations are seizures (particularly late-onset seizures), chronic headaches, and intracranial hypertension. However, cysticerci can locate anywhere in the human nervous system, thus potentially causing almost any neurological syndrome and making clinical diagnosis a difficult task. Neuroimaging is the main diagnostic tool, and specific serology confirms the diagnosis and helps to define the diagnosis when images are unclear. Factors such as location (extraparenchymal versus intraparenchymal), number, size and evolutive stage of the parasites determine the clinical manifestations, therapeutic approach, and prognosis. Management includes symptomatic drugs (analgesics, antiepileptic drugs, anti-inflammatory agents) and in many cases cysticidal drugs, either albendazole or praziquantel. In recent years, efforts have focused on transmission control and potential elimination in endemic regions.

Schistosomiasis is a parasitic disease caused by blood flukes of the genus Schistosoma. Currently 200 million people worldwide are infected. Neurological manifestations are a result of the inflammatory response of the host to egg deposition in the brain and spinal cord and is usually seen in patients with recent infection with no evidence of systemic illness. Cerebral and cerebellar disease can result in headache, seizure, and increased intracranial pressure. Cerebral schistosomiasis is more common in Schistosoma japonicum, but increasing cases due to Schistosoma mansoni are being reported in the literature. Other complications of cerebral schistosomiasis include delirium, loss of consciousness, visual field impairment, focal motor deficits, and ataxia. Myelopathy is the most common neurological manifestation of Schistosoma mansoni and the conus medullaris and cauda equine are the most common sites of involvement. Severe disease can result in flaccid paraplegia with areflexia, sphincter dysfunction, and sensory disturbance. Early recognition and prompt treatment are essential when physicians are faced with schistosomiasis involving the central nervous system. Schistosomicidal drugs, such as praziquantel, steroids and surgery, are the mainstay of therapy for this severe form of schistosomiasis.

Trichinellosis is a parasitic zoonosis caused by the nematode Trichinella spp. Neurotrichinellosis represents one of the most important complications of severe trichinellosis in humans and is sometimes fatal, especially when Trichinella spiralis is involved. There are numerous mechanisms responsible for the involvement of the nervous system through direct or indirect involvement of the parasite. In the latter, inflammatory cells, especially eosinophils, appear to play a crucial role. Encephalopathy, neuromuscular disturbances, and ocular involvement represent the most frequent presentations of neurotrichinellosis, with the first being the most responsible for fatalities. The diagnosis is based on imaging (CT or MRI), which shows nodular multifocal hypodensities in serologically positive individuals with relevant epidemiological factors (e.g., consumption of raw pork). However, only direct diagnosis by muscle biopsy can give the absolute certainty of infection. Albendazole and mebendazole are the anthelminthic of choice and should be used with corticosteroids to prevent allergic manifestations.