Angiostrongylus cantonensis

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Life cycle and transmission

In 1938, *Angiostrongylus cantonensis* was discovered in rat lungs by Chen in Canton, China. The first human case description dates from 1945. Recently, the taxonomical position of the worm as changed and *A. cantonensis* has been transferred to the genus *Parastrongylus*, but in this text we will continue to use the generic name *Angiostrongylus*. Infection with *A. cantonensis* is the most common aetiology of eosinophilic meningitis. Angiostrongyliasis occurs primarily in Southeast Asia, throughout the Pacific Basin, including Hawaii, Fiji, Indonesia, Philippines, Japan, mainland China, Taiwan and Papua New Guinea, but also in several Caribbean nations (Bahamas, Cuba, Puerto Rico, Dominican Republic and Jamaica). Occasionally small outbreaks occur.

Final hosts

A wide variety of rodents are final hosts, primarily in the genera *Rattus* and *Bandicota*. Eggs laid by the female worm hatch in branches of the pulmonary arteries. After hatching, first-stage larvae enter the alveoli, migrate up the trachea, are swallowed and reach the alimentary tract. Subsequently, they are excreted in the faeces of the animal. When a snail consumes these droppings, infection of the mollusk will ensue. Within about two weeks, infective third-stage larva will appear. When ingested by a rodent, these L3 larvae migrate to the brain via the blood circulation and develop into fourth-stage larvae and then young adults within the next 4 weeks. They migrate to the subarachnoid space, enter the venous plexus, and are carried to their final destination, the pulmonary arteries.

Humans and rats become infected through eating raw slugs or snails, soiled lettuce contaminated with mollusks, eating a carrier (“paratenic”) host, such as infected planarians, land crabs or freshwater shrimps. Certain freshwater as well as marine fish can become infected. Inside man, the neurotropic third-stage larvae pass from the intestinal tract to the meninges. They die 1-2 weeks after arriving in the human brain. Adult worms do not occur in humans.

Clinical aspects

Angiostrongyliasis (infection with *A. cantonensis*, the rat lungworm) has an incubation period of 2-35 days. Symptoms are due to migration of the larvae in the brain and the inflammatory reaction which occurs. The disease presents with acute moderate to severe headache (100%). Besides the headache,

patients can complain of eyeball pain. Visual problems can occur, due to involvement of one or more cranial nerves (diplopia, acute strabismus, gaze palsy) or due to migration of the larva into the eye, which can lead to retinal detachment and blindness. Nuchal rigidity occurs in about 66% of patients and Brudzinski's sign is present in ± 66%. Facial nerve paralysis, transient ataxia, delirium, seizures, cognitive dysfunction, hyperesthesia in various dermatomes and paraesthesia of arms and legs, trunk or face may occur and some symptoms may persist for months, although chronic disease is rare. Vomiting and nausea are self-limited and stop after a few days. Fever occurs in less than 50% of patients. The disease tends to be more serious in children. The disease is self-limiting. Most symptoms disappear spontaneously within 4 weeks of onset (range 2-8 weeks). Mortality is less than 1%.

Diagnosis

Eosinophilia of peripheral blood or CSF is not always present on initial laboratory testing. Pleocytosis may be absent early in the course of infection. Larvae are rarely detected in the CSF. The CSF can be clear or cloudy, but does not contain blood (except in case of a traumatic tap). The absence of focal lesions on CT or MRI-scanning of the brain distinguishes *A. cantonensis* infections from most other helminthic infections of the brain. Immunodiagnosis (ELISA, Western Blot) is possible in some centers. There is a poor correlation between the serological results of serum and CSF. Since in most cases, larvae will not be recovered in the cerebrospinal fluid and an autopsy will not be performed (the infection is not lethal in general), the diagnosis will be a tentative one, relying on the history, positive serology and exclusion of other causes.

Treatment

Analgesics are usually needed. Steroids (e.g. prednisolone 60 mg/day x 2 weeks or dexamethasone) shorten the duration of the headache. When performing a spinal tap, the opening pressure is increased in about 60% of patients. Repeated spinal taps to reduce the intracranial pressure are sometimes performed. Antihelminthics are thought by some not to be effective and considered to worsen the symptoms, probably because of the inflammatory reaction to antigens released by dying worms. Some clinicians use mebendazole or albendazole, but controlled studies are lacking.

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