

Taenia solium neurocysticercosis

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Taenia solium neurocysticercosis (NCC) is an infection of the human central nervous system (CNS) caused by the hematogenous spread of the encysted intermediate larval (metacestode) stage of the porcine tapeworm, *Taenia solium* (1, 2, 3, 8, 10, 13). It is one of the most common parasitic diseases and the single most common cause of epileptic seizure disorders both in the developing world and increasingly in the United States due to immigration from endemic areas (1, 2, 3, 5, 7, 8, 9, 10). Local acquisition of the disease is now occurring in the United States, with approximately 1,000 new cases of neurocysticercosis (NCC) diagnosed per year (8, 10). Conservative figures estimate 50,000 deaths every year due to neurocysticercosis (NCC), with no less than 50 million people infected worldwide with intestinal taeniasis caused by *Taenia solium* (3, 8).

Taenia solium is a two-host cestode and is classified as follows: domain Eukarya, kingdom Animalia, phylum Platyhelminthes (flatworms), class Cestoidea (tapeworms), order Cyclophyllidea, family Taenidae, genus *Taenia*, and species *solium* (1, 8). The adult tapeworms are flat, sectioned worms with 3 anatomic regions (8, 10). Adults may measure up to 10-30 feet in length and live for up to 25 years, producing tens of millions of infective eggs over their lifetime (1, 4, 5, 6, 7, 8). The surface of the worm is covered by microvilli (microtriches). These microtriches allow for maximal absorption of nutrients from the host intestine (8). The scolex evaginates and anchors the worm to an intestinal villus (mucosa) of the human definitive host by muscular suckers and protienaceous hooks (8, 9, 10). No other final hosts are known in nature (1, 2, 4, 7). This form of attachment does not damage the host. The neck of the scolex is metabolically active, and contains stem cells that section the body of the worm. The body, or strobila, consists of a variable number of sections, called proglottids (8). Proglottids are approximately 1 cm wide, 1-2 cm long, and 2-3 mm thick (2). Each proglottid functions independently with respect to reproduction, digestion, excretion,

and locomotion (8). Proglottids have their own sex organs (monoecious) and can become gravid by copulating with other proglottids of the same or another worm (8, 10). The gravid proglottids at the terminal end are compacted with eggs that are the source of the larval stage infection, or cysticercosis (1). As new proglottids develop from the base of the scolex, at the proximal end of the strobila, older ones mature, moving caudally to the distal end of the worm for detachment (2, 5, 7, 8). As they near the end of the strobila, they may rupture, releasing the infectious eggs, or they may break off, remaining intact in the feces. The eggs and/ or proglottids are expelled through the feces of the tapeworm carrier intermittently and may contaminate food sources in areas where sanitation is relatively poor (2, 4, 8). Each proglottid released contains thousands of eggs (50-60,000), which are fully embryonated, infective, and resistant to adverse conditions (6, 7, 9, 10). The free proglottids have the ability to locomote independently for many feet due to their own extensive orthogonal musculature system, locating to a more suitable environment for infecting subsequent hosts (8). The eggs have the ability to survive for months to years in the environment (6). Autoinfection may occur when proglottids move retrograde rather than caudally by reverse peristalsis (6, 8, 10). Hand to mouth autoinfection or close contact with food preparation by an infected carrier occur in the majority of documented cases (2, 8, 10).

Cysticercosis develops when humans or pigs ingest taenia contaminated food and acquire the larval infection within their bodies. Pigs, the obligate intermediate hosts, become infected by ingesting feces containing eggs or proglottids (1, 2, 5, 12). Once inside the pig's intestinal tract, the action of bile and pancreatic enzymes cause the eggs to lose their coats, enabling them to cross the intestinal wall, enter the blood stream, and infect the tissue (7). When the adult cestode infests the human intestine by ingesting undercooked infected pork, intestinal taeniasis develops (2, 4, 8). Human cysticercosis develops when a human host ingests taenia

infected eggs by fecal contamination, replacing the pig as the intermediate host (1, 2, 5). Humans are host specific for the adult tapeworm (cestode) and the only source of cysticercosis for pigs and other humans, whereas both humans and pigs may act as intermediate hosts for the embryo or larval form (4, 5, 7). While taeniasis is usually asymptomatic, the host becomes a new and continuous source of infective eggs (4, 8). Once ingested, the mechanism is the same as that of the pig; the taenia eggs cross the digestive tract, enter the circulatory system, and are carried to subcutaneous tissue, muscle, viscera, and the central nervous system (CNS) through the bloodstream (4, 6, 7). The parasites become established in the tissues as larval cysts, reaching maturity within 3 months (1). The disease can occur anywhere in the human body, but becomes symptomatic in the nervous system, as neurocysticercosis (NCC) or the eye (1, 5). The infection ranges from a single lesion to several hundreds, and range in size from a few millimeters to several centimeters. These viable cysts are 10 to 20 mm in diameter, are thin walled sacks filled with clear cyst fluid, and modulate the immune system of the host in order to evade imminent destruction by it. Larger cysts, measuring more than 50 mm in diameter are occasionally found (1).

Neurocysticercosis (NCC) is a common pleomorphic disease that may affect both males and females from birth to advanced age; however, the incidence of occurrence peaks in middle-aged adults (6, 7, 11). Manifestations vary with number, size, and topography of the lesion and intensity of the host's immune response to the parasite (11).

Neurocysticercosis (NCC) occurs when the cysts penetrate the blood-brain barrier into the central nervous system (CNS). Symptoms of the disease can occur within months or years after initial infection. There are four stages in the pathology of neurocysticercosis (NCC): vesicular, colloidal, granular nodular, and nodular calcified (6). The most common clinical manifestations of neurocysticercosis (NCC) include seizures, headache, nausea or vomiting,

and focal neurological deficits (altered mental status, visual changes, or dizziness) that are dependent on the number, location, size, and viability or stage of degenerative cysts (1, 2, 3, 5, 6, 7, 9, 10). Pathophysiology of neurocysticercosis (NCC) is related to brain dysfunction. The characteristic dysfunction occurs by two mechanisms: (a) mass effect, and (b) inflammation. Mass effect results from the presence of space-occupying lesions within the brain (10). Inflammation is due to the human immune system's response to the dying (degenerating) cysts, as viable larvae do not induce an inflammatory response, thereby causing no signs of illness (8, 9, 10). Viable parasites avoid immune system detection by disguising their surface structures with molecules derived from the host and secreting other immunomodulatory enzymes (8, 10, 13). Degenerating cysts lose these capabilities and release antigens that are detected by the immune system, causing inflammation (13). Epilepsy occurs in 50-90% of neurocysticercosis (NCC) patients (7, 9, 11). Seizures may be focal, focal with secondary generalization, or generalized (2). Generalized seizures are associated with multiple lesions, whereas single lesions may present as partial seizures (7, 11). The seizures are controlled and resolve as the inflammation subsides. Headaches may be hemicranial or bilateral and may be misdiagnosed as uncomplicated migraine or tension headaches (2). Neurocysticercosis (NCC) also may be associated with chronic depression and psychosis (2, 11).

Treatment measures include antiparasitic drugs, surgery, and symptomatic medication (1). Therapy must be individualized per patient according to the advancement of the disease and the location of any lesions (6). Treatment of tapeworm carriers is critical to interrupt the transmission of cysticercosis. *Taenia solium* can be cured with a single 2 gram dose of niclosamide or 5 mg/kg of praziquantel (PZQ). Niclosamide is the drug of choice due to its inability to be absorbed into the intestine. This would avoid any adverse neurological

symptoms in patients who also have neurocysticercosis (NCC). Treatment with either drug has more than 95% efficacy (5). Albendazole also displays a high parasitocidal effect (1). Since cestodes produce new proglottids from the neck region, below the scolex, it is imperative that the scolex be expelled. If it is not expelled, it will regenerate into a full tapeworm within a few months. After treatment if the expelled scolex is identified, then its passage confirms cure (5).

Neurocysticercosis (NCC) is a complex infection resulting from an encounter between a highly sophisticated parasite and an elaborate immune response (4). Potential eradication of cysticercosis should be accomplished by removing it from both hosts. Despite the elimination of tapeworm carriers, pig carriers will remain as the reservoir of infection to establish new infection and disease transmission in humans (12). Therefore, vaccination of pigs and immunotherapy are of interest to interrupt the *Taenia solium* parasite's life cycle and potentially eliminate the source of infection for humans (9, 12).

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