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Neurocysticercosis

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Neurocysticercosis (NCC) is one of the most common parasitic infections of the central nervous system in humans and is the most serious clinical manifestation of taeniasis. NCC is caused by the ingestion of the larval form of the pork tapeworm, Taenia solium. This organism also known as the “ pork tapeworm” is one of the eight cestode species that can infect humans. The parasite has a chain of intermediate hosts in low income developing countries where pigs are commonly raised, including the pig farms of South America, South Africa, and parts of Africa and Asia (Nwakudu, & Ekechukwu, 2013). The parasite Taenia solium encysts in the brain and can express a broad range of symptoms including seizures, headache, hydrocephalus, encephalitis, stroke and mental health and cognitive disorders. NCC is a leading cause of neurological disease in low and middle-income countries with 25%-30% affected individuals losing evidence or the parasitic infection. This infection process is usually diagnosed due to the defense of new seizure disorder in an individual (O’Neal & Flecker, 2015).

Over the past two decades, medical advancements such as diagnostic testing, anti-parasitic drugs, anti-inflammatory agents, and neurosurgical procedures have improved the prognosis of patients infected with Taenia solium. However, despite the improvements in treatment options and new methods to diagnose and manage this parasitic infection, many are being infected at the same rate or higher. Aggressive management of this infection is essential to control the epidemic, extrapulmonary neurocysticercosis mainly due to gastrointestinal hyperinfection, and the development of new treatment options available in endemic regions (Garcia, Nuño, & Bill Brant, 2014).

An example of the life cycle of the parasite begins when the human host excretes the parasitic egg through feces. The eggs are then consumed by an omnivore such as a pig. The eggs hatch into larval form in the alimentary tract of the pig and begin to invade the tissue. The larvae called cysticerci are ingested by humans after the consumption of undercooked meat. The lifecycle of the parasite is then completed after the encysted larva is found in the stomach by gastric, small and large baits (Nwakudu & Ekechukwu, 2013). Once the tapeworm eggs are freed from its protective environment, it is able to freely penetrate the wall of the gut and travel into systemic circulation. The parasites are then transferred to the brain while traveling the venous system and may invade multiple areas of the host including the bowel walls, heart, skeletal muscles, eyes and other critical areas such as the central nervous system including the brain and spinal cord. The larvae can invade areas of the brain such as the ventricles and subarachnoid space, lying dormant in an unencapsulated form within the host for years (Nwakudu & Ekechukwu, 2013).

Two major pathways leading to the disease transmission of NCC exist: 1) The infection of humans via contaminated environmental media or food 2) The infestation of swine caused by the direct ingestion of human faeces, contaminated soil, food or water containing the parasitic embryos. Autointe transmission of the eggs by the tapeworm-carrying host, via fecal- oral transmission can also be a potential exposure pathway (Ekanem, Ramar, Garcia & Slaoui, 2016).

Below: Lifecycle of pork tapeworm, Taenia solium. Copyright 2014 by CDC.

Case Study: Neurocysticercosis in Wisconsin

Neurocysticercosis occurs when the encysted larval forms of Taenia solium invade the central nervous system of a human host (Enander et al., 2010). The pathology also known as the “pork tapeworm” is one of the eight cestode species that can infect humans. An example of the life cycle of the parasite begins when the human host excretes the parasitic egg through feces. The eggs are then consumed by an omnivore such as a pig. The eggs hatch into larval form in the alimentary tract of the pig and begin to invade the tissue. The larvae called cysticerci are ingested by humans after the consumption of undercooked meat. The lifecycle of the parasite is then completed after the encysted larva is found in the stomach by gastric, small and large baits (Nwakudu & Ekechukwu, 2013). Once the tapeworm eggs are freed from its protective environment, it is able to freely penetrate the wall of the gut and travel into systemic circulation. The parasites are then transferred to the brain while traveling the venous system and may invade multiple areas of the host including the bowel walls, heart, skeletal muscles, eyes and other critical areas such as the central nervous system including the brain and spinal cord. The larvae can invade areas of the brain such as the ventricles and subarachnoid space, lying dormant in an unencapsulated form within the host for years (Nwakudu & Ekechukwu, 2013).

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Case Study: Neurocysticercosis in Wisconsin

Reported cases of neurocysticercosis are increasing in the United States, especially among immigrants who previously lived in endemic areas and individuals living in rural areas. Here are three separate cases that presented to and were evaluated at the same institution in Wisconsin. (Naddaf, Slaoui, & Stafstrom, 2014)

Patient 1

A 25 year old woman who presented with these focal seizures. She was a new immigrant from the Philippines 2 months prior to symptom presentation. There were no focal neurological deficits on examination. An MRI showed a fdom ring enhancing lesion located in the right frontal lobe. An Electrophysiology (EEG) and serum and cerebrospinal fluid cysticerci IgG were negative. Diagnosis of neurocysticercosis (NCC) was suspected. The patient was treated with phenytoin for 12 weeks, followed by albendazole for 15 days. For seizure prophylaxis she was started on levetiracetam. Two years later patient did exhibit right lip twitching after a minor stress. Two years prior she was started on levetiracetam. She was suspected for NCC being, low suspicion of tumors two brain biopsies were performed, one normal brain tissue and second revealed a microabscess with lymphocytes and eosinophils, with NCC being suspected. Patient was started on prednisone and a 30 day regimen of albendazole. Prescribed gabapentin continued, and Albendazole was started. Patient was controlled by lamotrigine 4 years after initial presentation. Follow up showed decreased size of the brain lesions (Nadaf, Slaoui, & Stafstrom, 2014).

Patient 2

A 32 year old woman complaining of left area dysesthesias and numbness, with radiation into left neck and face. Symptoms also included difficulty speaking, hearing, and doul headaches for the past year. She had admitted to traveling to Mexico several times a year. Brain MRI showed a single 9 x 12mm ring enhancing lesion in the right parietal lobe. Cysticercus seric IgG was 0.01 and considered positive. The patient was started on steroids and a 10-day course of albendazole. Levetracetam was prescribed for seizure prophylaxis. Patient reports being healthy and doing well 8 years after initial presentation (Nadaf, Slaoui, & Stafstrom, 2014).