Case Report: Neurocysticercosis in the Infant of a Pregnant Mother with a Tapeworm

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Abstract. Taeniasis occurs after ingestion of undercooked pork infected with cysticerci. Most Taenia solium infections are mild; proglottids are rarely noticed in the feces. Cysticercosis develops with ingestion of eggs from a tapeworm carrier. Cysticercosis affects ~50 million people worldwide, and is seen mostly in Central and South America, sub-Saharan Africa, India, and Asia. We present a case of an 18-month-old child living in New York, who presented with seizures caused by neurocysticercosis. A family study found a 22-year-old mother, 7 months pregnant, positive for T. solium, which presented a management dilemma.

INTRODUCTION

Cysticercosis results from infection with the larval stage of the pork tapeworm Taenia solium. The normal intermediate host is the pig, infected through ingestion of T. solium eggs in the stool of a human tapeworm carrier. The tapeworm is acquired through consumption of undercooked pork meat containing the larvae or cysticerci. Humans can be accidentally infected with cysticercosis when they ingest eggs excreted by the tapeworm carrier directly or in contaminated food. Although cysticercosis can occur anywhere in the body and the infection is often asymptomatic, the most common clinical manifestation is a seizure disorder resulting from central nervous system (CNS) cysticerci. We report the case of a child with neurocysticercosis and the subsequent study of his family.

CASE REPORT

An 18-month-old child presented to the hospital with new onset of generalized seizures. He had never lived outside the United States and was previously healthy. Computerized tomography and magnetic resonance imaging (MRI) of the head showed cystic lesions with surrounding edema (Figure 1). Serologic testing for cysticercosis, including Western blot, was positive. The child’s seizure disorder was controlled with oxcarbazepine 75 mg orally every 12 hours, and he was subsequently treated with dexamethasone 0.5 mg orally every 12 hours for 2 days and albendazole 85 mg orally every 12 hours (15 mg/kg/day) for 28 days.

A family study was carried out to identify the source of the child’s infection. The child’s mother was an asymptomatic, apparently healthy 22-year-old woman, who had moved to the United States from El Salvador in 2006. She was 7 months pregnant (G2P1) at the time of the study. The mother and child lived with the child’s father, uncle, and aunt; all of the adults were from El Salvador. Stool specimens from all four adults were examined for ova and parasites, and serum specimens were tested at the CDC by enzyme-linked immuno-electrotransfer blot (EITB) assay for cysticercosis. The mother’s stool was found to contain large numbers of Taenia eggs (Figure 2A). Her stool specimens were also positive for ova of Hymenolepis nana, Entamoeba dispar, Endolimax nana, Entamoeba hartmanii, and Blastocystis hominis. The child’s father’s stool contained E. dispar, B. hominis, and E. nana eggs. The uncle’s stool contained H. nana eggs and that of the aunt contained hookworm eggs. No other tapeworm carrier was detected. Specimens from the child’s mother and father were positive by EITB; neither had symptoms suggestive of neurocysticercosis.

The mother was treated with niclosamide and a mild cathartic. Proglottids were recovered from the stool (Figure 2B). Referrals were provided to the aunt and uncle for treatment of their parasitic infections. Strict hygiene was emphasized in the family. Examination of the mother’s stool 3 months after niclosamide treatment was negative for ova and parasites.

DISCUSSION

This case provides a graphic illustration of the life cycle of T. solium. The source of infection for cysticercosis patients is often a family member, and tapeworm carriers may autoinfect themselves. Although seizures are the most frequent presenting complaint for cysticercosis, symptoms are varied and depend on the anatomic location, size, and stage of the larval cysts. Cysticercosis is often asymptomatic until larval degeneration occurs, provoking an inflammatory response. The time from infection to symptoms can vary from months to many years. Acute management of neurocysticercosis focuses on the control of seizures, CNS inflammation, or other neurologic symptoms. Treatment with anthelmintics can precipitate or increase inflammation and cerebral edema and should be deferred until these conditions have been controlled. Albendazole treatment of patients with viable CNS cysts was shown to significantly decrease generalized seizures over 30 months of follow-up. Reference diagnostic testing and advice on management of cysticercosis and tapeworm are available from the Division of Parasitic Diseases, CDC (770-488-7775 or parasites@cdc.gov).

The finding of tapeworm infection in the child’s mother was not unexpected, but her pregnancy complicated the choice of management. We opted to treat the mother’s tapeworm prepartum, in part to decrease the risk of exposure of the neonate to infected maternal stool at the time of birth. In this case, the non-absorbable drug niclosamide was chosen over praziquantel; available data are insufficient to judge...
the safety of praziquantel in pregnancy. Simultaneous gastrointestinal purge increases the likelihood of recovering the tapeworm and facilitates species identification. In this case, a mild cathartic was chosen, because strong cathartics are not recommended in pregnancy. The mother and father of the child had no symptoms suggestive of neurocysticercosis and were not further evaluated or treated. Positive serology can occur in patients without neurocysticercosis from exposure to *T. solium* eggs without established infection or from cysticerci in skeletal muscle or other locations. Albendazole is contraindicated in pregnancy; anthelmintic treatment of cysticercosis in a pregnant woman should therefore be deferred until after delivery.

Neurocysticercosis is the most frequent cause of epilepsy worldwide. The highest prevalence occurs in developing countries where inadequate sanitation and traditional animal husbandry practices promote access by pigs to human feces. In the United States, *T. solium* is reportable only in a few states, and few systematic epidemiologic data are available. However, facility-based data from California suggest that the frequency of cysticercosis has increased substantially over time, as the state’s immigrant population has grown. In one southern California hospital, 10% of neurology and neurosurgical admissions were attributed to cysticercosis, whereas a study of emergency departments in 11 different states showed neurocysticercosis in 0.5% to 9.9% of seizure patients, with cases diagnosed in nine sites. A small population-based study conducted in El Paso and across the US–Mexico border in Ciudad Juárez showed significantly higher tapeworm prevalence in the US city, possibly because of greater awareness and frequent anthelmintic use in the population living in Mexico.

As our case shows, individuals who have never lived outside the United States can become infected with cysticercosis through contact with a food handler harboring an imported *T. solium* tapeworm. A cluster of cases in an Orthodox Jewish community in Brooklyn in the early 1990s provided a striking example of local, apparently anomalous transmission; the source of the infections was thought to be Latin American housekeepers who had lived in the households of the cysticercosis patients. A recommendation was made to screen immigrants who apply for positions as food handlers or house-
CRYPTICERCOSIS IN NEW YORK

keepers. However, aside from the potential social and legal complexities, systematic screening is technically challenging because microscopic stool examination has poor sensitivity, and the more sensitive coproantigen test is not available as a routine assay. Our cases underscore the need to include neurocysticercosis in the differential diagnosis of new onset seizures in the United States and to seek the infection source when such patients are found.

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