

Case Report

Neurocysticercosis in a Nicaraguan Woman: A Case Report and Disease Overview

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ABSTRACT

Neurocysticercosis is a common parasitic disease resulting from infection with *Taenia solium*. This disease is relatively rare in the United States but is very common in developing nations. Although many patients remain asymptomatic, common life-threatening clinical features of the disease are seizures and hydrocephalus. The case presented is of a Nicaraguan woman previously diagnosed with neurocysticercosis but complaining of new symptoms resulting from her shunt failure in 2012. Physicians should be aware of this rare disease as it is becoming increasingly more prevalent in developed nations such as the United States.

Key words: Neurology, travel medicine, infectious diseases.

INTRODUCTION

Neurocysticercosis (NCC) is the most common parasitic infection of the nervous system and the main cause of adult-onset epilepsy in developing countries. Cantey et al.^[1] reports that NCC accounts for 29% of persons with epilepsy, but could increase towards 50% in disease-endemic countries. Up to 2% of emergency department visits in the United States for seizures are due to NCC.^[1] If left untreated, NCC has a mortality rate of over 50%.^[2] It has been believed that cases of NCC in the U.S. are mainly due to already infected immigrants from Latin America. But, case reports have shown that some cases of NCC are acquired in the U.S. and others involve U.S. travelers who recently visited disease-endemic countries.^[2] In the U.S., NCC is most commonly found in impoverished households and

homes of underrepresented minorities.^[3] Estimated incidence ranges from 0.2-0.6 cases per 100,000 general population and 1.5-5.8 cases per 100,000 Hispanics.^[4] NCC is caused by the larval stage of the *Taenia solium* (pork tapeworm) parasite. The infection results from ingesting *Taenia solium* eggs, usually by eating contaminated food that was prepared by someone with taeniasis or by eating undercooked pork.

Clinical Features: Symptoms of NCC are not only dependent on the number, size, location, and stage of development of the cysticerci, but also the body's immune response to the parasite. Many patients with NCC remain asymptomatic and unaware of their condition, but some patients with more severe forms of the disease present with potentially fatal symptoms. The most common symptom of

NCC is seizures, predominantly partial seizures, which occur in 70-90% of patients suffering from NCC. [5] As the cysticerci calcify, seizures tend to become less frequent although anti-seizure medications are typically continued. The cysticerci can also cause irritation of the ependyma, leading to an inflammatory reaction, resulting in obstructed CSF flow and hydrocephalus. Intracranial hypertension due to hydrocephalus can be life threatening but is typically only present in 10-20% of NCC cases. [5] Involvement of the spinal cord is rare in NCC but occurs in 1-5% of cases. [6] Spinal NCC can be classified as either intramedullary or extramedullary, the latter being further broken into intradural and extradural categories. Most spinal forms involve the subarachnoid spaces, resulting from direct CSF dissemination. [7] Callacondo et al found that the majority of recovered spinal cysticerci had racemose morphology, which is mostly found in basal subarachnoid forms of NCC. Spinal cysts were more commonly found in lumbosacral and cervical regions, followed by the thoracic region. [7]

Diagnosis and Imaging: NCC is commonly diagnosed with a combination of tests including CT and MRI of the brain, CSF analysis, and antibody detection. [5] Even in early infection, a cyst can be visualized on a CT or MRI. However, cysts located in the ventricles or subarachnoid spaces are not visible on CT and may result in false findings on a MRI. [5] Both the CT and MRI have their own advantages; MRI is better for diagnosis and CT is better for detection of any calcifications. CSF analysis is indicated for the majority of patients presenting with new onset seizures or neurological deficits, when imaging does not result in a diagnosis. However, CSF analysis is contraindicated in patients with edema, brain structure displacement, or hydrocephalus. The most common CSF findings include mononuclear pleocytosis, eosinophils, and the presence of

antibodies. [5] It is important to note that eosinophils are also elevated in neurosyphilis and tuberculosis of the CNS, which have similar presentations to NCC.

Differential Diagnoses: Due to the variability in the presentation of NCC, there are many other differentials that must be ruled out. Some to take into consideration are brain abscesses, brainstem gliomas, CNS tumors, CNS toxoplasmosis, CNS cryptococcosis, and tuberculosis of the CNS.

Treatment: As one could imagine, with the large variability in presentation, treatment of NCC varies greatly. Treatments include cysticidal agents, corticosteroids, antiepileptic drugs, and surgical measures to remove the cyst or to insert a shunt for hydrocephalus. [6] Two common cysticidal agents are Praziquantel and Albendazole, which are used in combination with a steroid or immunosuppressants to reduce the inflammation. Praziquantel acts by paralyzing the parasite, whereas albendazole causes energy depletion of the parasite. [6] In the cases of subarachnoid and intraventricular cysts, these drugs should only be used after placement of a shunt due to the risk of increased intracranial pressure. Antiepileptic drugs are used in cases of seizures with calcification. Surgical measures are indicated in patients with mass effect, obstructive hydrocephalus, and uncertain diagnosis. [6] Placement of an intraventricular shunt is a common surgical measure used for NCC patients but has high malfunction rates of 30-70% and must be closely monitored. [6] Follow-up imaging is recommended 2-3 months following treatment.

REPORT OF CASE

Presentation and History: A 48-year-old woman with a history of neurocysticercosis was seen at a clinic site in Chacraseca, Nicaragua in June 2015 for back pain, burning on the entire left side of her body, and headaches. In 2012, the

patient's ventricular shunt stopped working and she was in a coma for four days. The malfunctioned shunt was drained and the patient's current symptoms began after this incident. She was placed on Pregabalin daily for neuropathic pain, Floravital for an iron supplement, and Tebokan for the symptoms due to vascular injury of her brain.

The patient was originally diagnosed with neurocysticercosis after one year of headaches, seizures, nausea, and vomiting. Once admitted to the hospital in Leon, Nicaragua, her symptoms had progressed to hydrocephalus and the inability to speak. A CT scan was performed and she was referred to a larger hospital in Managua, Nicaragua for immediate surgery. The patient was in the hospital for two months post surgery with noticeable vision changes after treatment. The patient's sister was later diagnosed with neurocysticercosis, and went through the same surgery and course of treatment.

Physical Examination: Upon examination, the patient was alert, oriented, and slightly distressed. Pressure on the shunt resulted in slow, but adequate drainage. Her pupils were equal, round, and reactive to light and accommodation with the extra ocular muscles intact. There was no erythema or discharge in her ears with the tympanic membrane intact. Her throat lacked erythema or exudate and the tongue protruded straight. There was some nuchal rigidity with very limited range of motion. The cardiovascular exam was normal with regular rate and rhythm and no murmurs, gallops, or rubs. Her lungs were clear bilaterally with no wheezing. All cranial nerves were intact. Throughout the entire left side of the body, there was weakness, limited mobility, and edema. The patient also showed lack of coordination and balance. Her reflexes were graded as a 1 out of 5 on the left and 2 out of 5 on the right with noticeable atrophy of her left thenar eminence.

Treatment: The patient was taught a lymphatic drainage technique to help with

the left-sided edema. She was given over-the-counter pain medication to take as needed. She was also advised to have her shunt checked in two months.

DISCUSSION

Our patient presented with classic symptoms of NCC including headaches, seizures, nausea, and vomiting, but due to the lack of access to medical care in Nicaragua or lack of knowledge of her life-threatening condition, her disease had time to progress to a more dangerous stage. With earlier intervention, the surgical measures taken in our case could have been prevented. Prevention of disease progression is one of the pitfalls of medical care in developing nations. Although our patient was previously diagnosed with neurocysticercosis, her onset of new symptoms could be due to the failure of her shunt and resulting coma.

Even though NCC is more commonly found in developing countries due to the lack of regulations for the production and preparation of pork, it is becoming increasingly more prevalent in developed parts of the world, including the United States. This increase in prevalence of NCC in the United States is in response to U.S. residents traveling to disease-endemic regions, the impoverished conditions of some American households, and, mostly, the exponential increase in immigration to the U.S. of tapeworm carriers. Although rare, it is imperative that physicians are educated on the presentation of NCC so patients can be diagnosed early and NCC can be differentiated from the many similar diseases of the CNS. Furthermore, physicians should take detailed histories of their patients, including travel histories, to aid in diagnosing NCC.

CONCLUSION

Even as a relatively rare disease, neurocysticercosis can have fatal implications. Physicians should remain aware of the clinical presentations of NCC

in immigrants and individuals who have a travel history to or from an underdeveloped country where *Taenia solium* is prevalent. Clinical features may take months to years to present so it is important to always take a thorough travel history and be cognizant of these foreign diseases. Proper treatment plan and early detection can easily improve the outcome of these patients.

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