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Neurocysticercosis Presenting as Migraine in the United States

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Data Interpretation D
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Patient: **Male, 52-year-old**
Final Diagnosis: **Neurocysticercosis**
Symptoms: **Headache**
Clinical Procedure: **—**
Specialty: **Neurology**

Objective: **Rare disease**

Background: Cysticercosis is a condition caused by infection with the larval form of *Taenia solium*, a pork tapeworm that uses pigs as an intermediate host. Humans become infected when they ingest water or food contaminated with tapeworm cysts. Cysticercosis is increasing in frequency in developed countries due to increased access to travel. Neurocysticercosis occurs when *Taenia solium* cysts embed within the nervous system. The clinical presentation of neurocysticercosis ranges from asymptomatic to life-threatening, largely depending on the brain parenchymal involvement. The diagnosis is typically made with a combination of clinical evaluation, serology, and neuroimaging. Treatment for parenchymal neurocysticercosis may involve anthelmintic agents, symptomatic agents, surgery, or a combination of methods.

Case Report: A 52-year-old man with a medical history of migraine headaches, complicated type 2 diabetes mellitus, and obesity presented with a 4-month change in his migraines becoming severe, worse over his occiput bilaterally, and unresponsive to abortive therapy. His exposure history was unremarkable except for a habit of eating undercooked bacon, by which he would have developed neurocysticercosis via autoinfection. Neuroimaging and serology confirmed a diagnosis of neurocysticercosis and he was treated accordingly with antiparasitic and anti-inflammatory medications.

Conclusions: This presentation is nonspecific and can easily be overlooked, especially if there is an underlying known neurological condition such as migraine. This case illustrates that neurocysticercosis should be considered when an existing neuropathological condition displays a change in presentation or requires a change in therapeutic management, even without obvious risk factors.

Keywords: **Epilepsy, Generalized • Migraine with Aura • Neurocysticercosis**

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/943133>

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Background

Cysticercosis is a condition caused by infection from the larval form of *Taenia solium* [1]. *Taenia solium* is a pork tapeworm that uses pigs as an intermediate host [2]. Humans are an accidental intermediate host and become infected when they ingest cysts in infected pork or feces [2,3]. This infection is endemic in the developing world due to poor sanitary conditions contributing to the transmission of the disease, but it is increasingly becoming diagnosed in developed countries due to increased access to travel and immigration in modern times [3].

Neurocysticercosis is a condition caused by *Taenia solium* when cysts embed within the nervous system [3]. These parasites may be located anywhere from brain parenchyma to the ventricular system and spinal cord and have wide variability in symptoms at presentation, depending on which structures and tissues they have infected [2,3]. Often, patients present initially with seizures and undergo evaluation with neuroimaging, which is a major factor in diagnosis due to its characteristic lesions. Neurocysticercosis is the most common nervous system helminthic infection and is a major cause of epilepsy worldwide [3].

Here, we present the case of a 52-year-old man from the United States with medical history of chronic migraines and no recent travel or farm exposures, who suffered from neurocysticercosis from undercooked bacon that presented as a change in his usual migraines.

Case Report

Here, we present the case of a 52-year-old man with past medical history of migraine headaches, type 2 diabetes mellitus complicated with peripheral neuropathy, hyperlipidemia, and obesity, who presented as an outpatient after a change in his usual migraines over the previous 4 months. The patient stated his migraines now occurred almost weekly, were severe, were worse over his occiput bilaterally, and no longer were responsive to abortive therapy. The patient denied any new focal neurological deficits, changes in migrainous aura, seizures, numbness, weakness, facial asymmetry, dysarthria, or dysphagia. The patient denied recent travel to high-risk areas, with his only notable travel history being attendance on a cruise to the Bahamas 2 years prior. He denied food insecurity and lived at home with his wife and cat in a modern home. On further questioning, the patient denied eating raw or street food but admitted to a habit of eating lightly cooked, non-crispy bacon for most of his life.

His vitals at the time of presentation were unremarkable and his neurological exam was nonfocal. Due to the concerning and persistent change in his migraines, we performed a CT scan, which revealed numerous cystic foci bilaterally within the deep cortical and periventricular white matter parenchyma diffusely throughout each hemisphere, as seen in **Figure 1**. There was no evidence of mass effect or hydrocephalus. These changes were initially regarded as suspicious for congenital neuroglial cysts. The patient was admitted urgently to the hospital for timely neurosurgical consultation. Routine laboratory test results were unremarkable. MRI on admission demonstrated the findings from the CT, but noted possible edema represented by

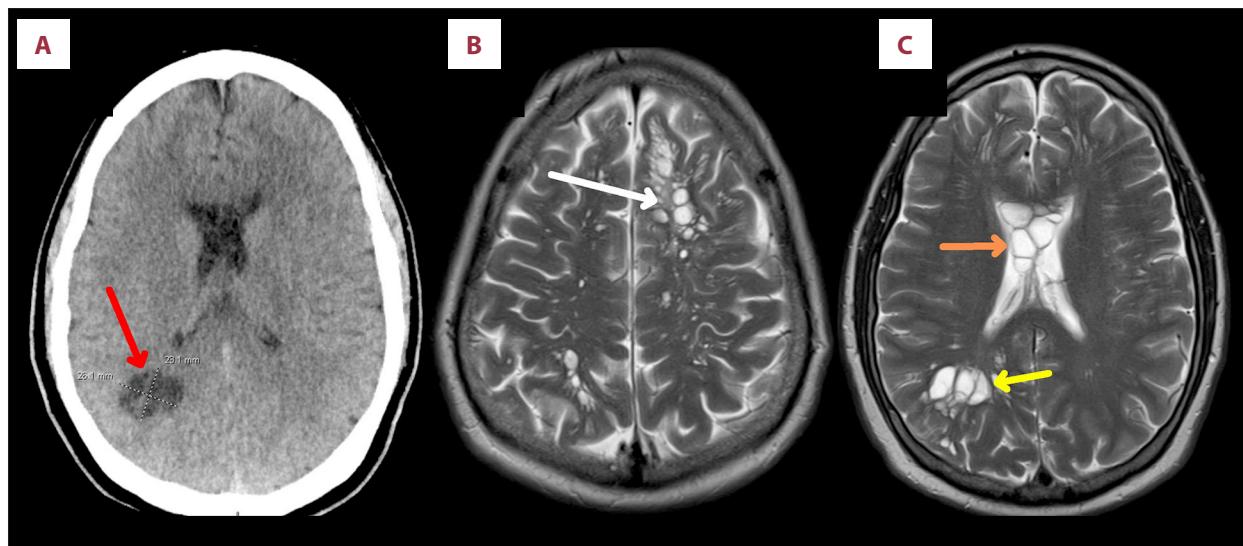


Figure 1. (A) CT head showed a cluster adjacent to the occipital horn of the right lateral ventricle, which measured maximally 2.6×2.3 cm across (red arrow). (B) MRI brain showed multilocular cystic lesions within the frontal and parietal cortices (white arrow). (C) Cystic lesions seen in the corpus callosum (orange arrow) and occipital lobe (yellow arrow).

T2/FLAIR hyperintensity surrounding the cystic foci, increasing the concern for neurocysticercosis. Neurosurgery did not pursue acute neurosurgical intervention and recommended an Infectious Diseases consult to rule out infectious processes.

A thorough infectious disease workup was undertaken. Blood and urine cultures were negative. HIV antibody and RPR were nonreactive. Cryptococcal antigen and *Toxoplasma gondii* IgM and IgG were negative. Cysticercosis IgG Cysts antibody returned positive, confirming the suspicion of neurocysticercosis. He was started on dexamethasone 1 mg 4 times daily for seizure prophylaxis and to reduce cerebral edema and subsequently monitored closely in the ICU. He was given oral albendazole 600 mg twice daily and praziquantel 1800 mg 3 times daily for 14 days total. Dexamethasone was tapered and he was determined to be stable for outpatient follow-up with the Infectious Disease clinic. The patient was successfully treated, with regression of lesions and improvement of headaches.

Discussion

Cysticercosis is a disease that has left a large impact on humanity in history as distant as ancient Greece [6]. To them, the connection between human cysticercosis and pork consumption was plain, and pigs and swine were viewed as impure or unclean throughout the ancient world [6]. Neurocysticercosis is virtually nonexistent in areas of the world that have banned pork consumption, further highlighting the strong link between swine and this disease [7]. Cysticercosis remains prevalent in modern Asia, Latin America, sub-Saharan Africa, and Oceania, particularly in developing countries and areas where pigs are raised as primary food sources [4]. Historically, developed countries have not been major hotbeds for infection due to high scrutiny of food safety and sanitary standards [3]. However, increased rates of immigration to developed countries from endemic countries have led to a significant increase in prevalence in countries such as the United States [8]. Early cases were initially puzzling, such as when members of an Orthodox Jewish community in New York became infected from their houseworkers, who themselves were *Taenia* carriers, distancing the disease from direct exposures to swine [6]. Similarly, our patient lived in the United States and had no recent travel to endemic countries or contact with pigs.

Our patient's lifelong preference for soft bacon may have led to instances of undercooked bacon consumption, but this would have caused him to develop taeniasis, an intestinal tapeworm, and not cysticercosis [1]. Taeniasis occurs when consuming undercooked pork and the larval cysts embedded within, while cysticercosis is contracted when humans ingest eggs found in the feces of other humans with taeniasis [1]. It can only be speculated, but given our patient's predilection

for undercooked pork and benign exposure history, we favor that his cysticercosis was transmitted via autoinfection after improper handwashing after he had contracted taeniasis himself from his eating habits.

Cysticercosis has an extremely variable presentation, with symptoms dependent on the tissues that *Taenia solium* has infected, with neurocysticercosis being diagnosed when tissues in the central nervous system are affected [3]. This variability in symptomatology extends to the severity of these symptoms as well, ranging from completely asymptomatic to acutely life-threatening [2,3]. The most classic presentation is seizure, which is seen in up to 80% of cases [3]. Neurocysticercosis also classically presents with focal neurological deficits, intracranial hypertension, or cognitive decline [4]. Due to high variability in presentation, it is important to consider that neurocysticercosis can present as a benign and isolated neurological symptom, such as the headache seen in our patient. Migraines, particularly those that are chronic and stable, do not warrant neuroimaging [5]. However, acute and persistent changes in migraine frequency or character should raise concern for a new pathology, as in our patient. Clinicians should retain a high index of suspicion and obtain thorough histories in patients with changes in migraine pattern, as etiologies that are considered unlikely may become more probable if there are high-risk features such as travel to endemic countries or occupational exposures.

The treatment of neurocysticercosis is controversial. Antiparasitic drugs such as praziquantel or albendazole have sufficient activity against *Taenia solium*, but there is concern that most of the inflammation occurs when the cysts are killed, giving some clinicians pause when considering treatment [1]. After a risk-benefit discussion, our patient ultimately decided to pursue definitive treatment with albendazole.

Conclusions

There are 3 learning points from this case report. First, neurocysticercosis is a parasitic infection of the brain, most commonly acquired through travel to developing countries in which cysticercosis and taeniasis are endemic. It is very rare for patients to contract neurocysticercosis outside of classic exposures or travel, and such cases in the United States were thought to be nonexistent. Undercooked pork consumption is a theoretical risk factor for neurocysticercosis via autoinoculation, as we suspected in this case. It is historically very unusual to encounter infected pork in the United States, and our case may have public health implications. Second, neurocysticercosis is protean in its manifestations, depending on the level of disease penetrance into the brain parenchyma, which can complicate diagnosis. The great variability of symptoms that

neurocysticercosis can present with should not be understated, and although it is a leading cause of epilepsy worldwide, it can present with more subtlety. Third, it is important to keep neurocysticercosis and other etiologies of central nervous pathology on the differential diagnosis when evaluating acute changes in migraines, even when there are no classic risk factors.

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