Parenchymal Neurocysticercosis as One of the Most Neglected Tropical Diseases: A Case Report

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Neurocysticercosis is a parasitic nervous system disease and one of the most neglected tropical diseases that emerges as the most common cause of epilepsy and acquired seizures in developing countries. A person may be infected following ingesting foods contaminated with Taenia solium eggs. This article discusses the case of parenchymal neurocysticercosis in a 25-year-old male with a chief complaint of focal-to-bilateral seizures. The seizure happened after eating and lasted for five minutes. The patient had left unilateral hemiparesis, which lasted for around 24 hours following the seizure. The patient had five focal seizures in the last year but has never sought medical treatment. The patient had a habit of consuming raw pork. CT scan showed an oval lesion with heterogeneous density in the right centrum semiovale with minimal perifocal edema. The patient was given albendazole, phenytoin, pantoprazole, dexamethasone, and folic acid. After treatment, the patient's condition improved and MRI results showed no abnormality. In this case we report a parenchymal neurocysticercosis with atypical phenomenon, such as focal neurological deficit due to space occupying lesions with improvement in symptoms and neuroimaging results. This offers up new paths for research into the causes of focal neurological deficit other than infarction and hemorrhage.

Introduction

Neurocysticercosis is a parasitic disease that affects the nervous system. It is caused by a parasitic cyst/larvae infection of the Taenia solium tapeworm.[1] Taenia solium infection spreads by fecal-oral route, mostly through the consumption of raw pork in countries across the world with insufficient food safety and sanitary regulations.[2[

Neurocysticercosis is endemic in the majority of low-income countries, such as in Latin America, Asia, and sub-Saharan Africa and contributes to 30% of epilepsy cases in endemic areas.[3] Indonesia is one of the countries in Asia where neurocysticercosis is endemic, particularly in Papua and Bali.[4] Unfortunately, its incidence is unknown due to neuroimaging that are not available to all in endemic countries.[5] This has led in an underestimation of the global incidence and burden of neurocysticercosis, making it one of the most serious neglected tropical diseases.[6 [

Case Presentation

A 25-year-old male came into the emergency room with a chief complaint of focal-to-bilateral seizures before their ER visit. The seizure happened in the following sequence: eyes glance to the left, head turns to the left, left arm jerking, then whole body rigidity with eyes squinting upwards. Before onset, the patient felt sudden dizziness, stiffness, and weakness. The seizure happened after eating and lasted for five minutes. Consciousness was maintained in all steps before rigidity, nausea (+), vomiting (+), headache (+), and there is a laceration on the left shoulder due to a fall. The patient had left unilateral hemiparesis, which lasted for around 24 hours following the seizure. Two weeks prior, the patient complained of sudden neck stiffness that disappeared on its own, lasting only about five minutes. The patient had five focal seizures in the last year but has never sought medical treatment. The patient had a habit of consuming raw pork. There was no significant family history. His vital signs and physical examination were within normal limits. Laboratory results showed an increase in liver enzymes (ALT and AST.(

Head CT-scan for this patient were conducted to confirm the diagnosis. Head CT-scan showed an oval lesion with heterogeneous density measuring +/- 0.7 x 0.6 x 0.7 cm in the right centrum semiovale with a central dot inside and minimal perifocal edema. Ring contrast enhancement is observed when contrast is administered.

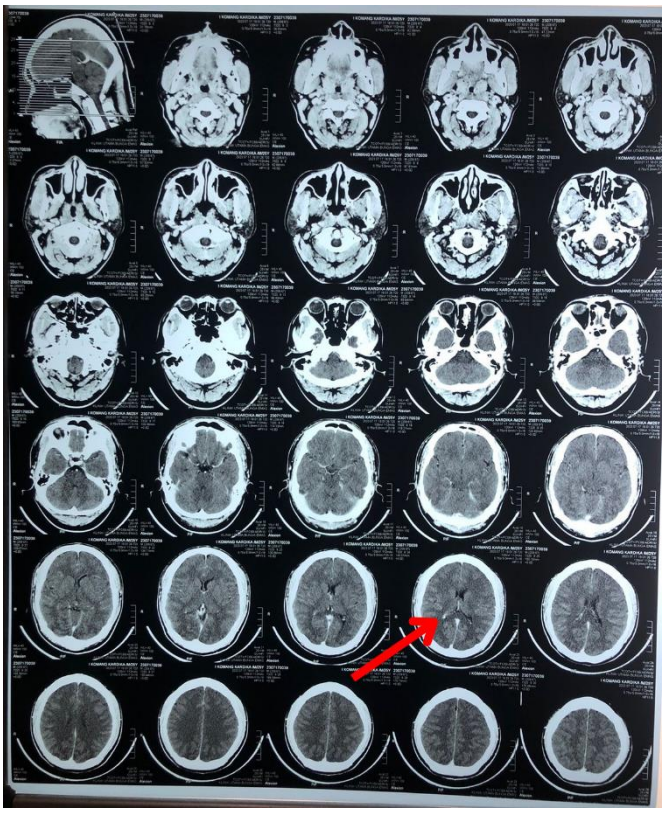

Figure 1. Head CT-scan

Based on clinical symptoms of seizures and CT scan results of the brain parenchyma, the patient was diagnosed had parenchymal neurocysticercosis with symptomatic epilepsy. The patient was given albendazole, phenytoin, pantoprazole, dexamethasone, and folic acid for 1 month. After treatment, the patient's condition improved, MRI results showed no abnormality, and the symptoms of seizures were no longer present.

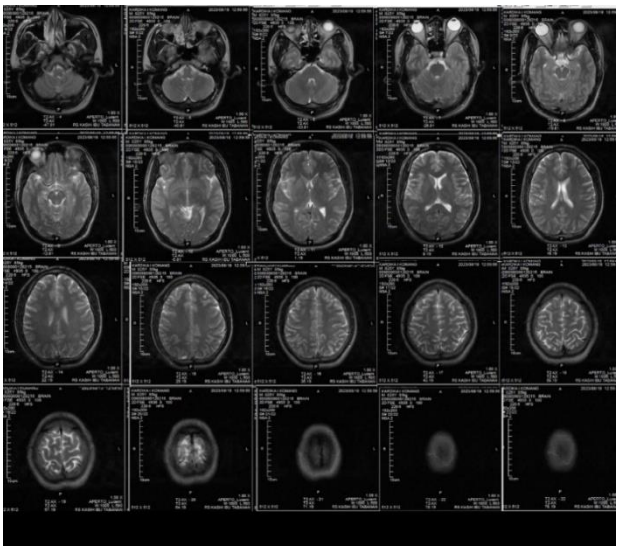

Figure 2. Head MRI Post Treatment

Discussion

Neurocysticercosis is one of the World Health Organization's 17 most neglected tropical diseases, and it is expected to be eradicated by 2030.[7] It is caused by ingesting food contaminated with embryonated eggs of Taenia solium. This disease has been related to insufficient waste handling and free-range pigs.[3 [

Taenia solium is a human tapeworm that can cause two clinical presentations: taeniasis if adult worms found in the small intestine and cysticercosis if cyst/larval stage found in the tissue.[6] Pigs and humans are hosts in the natural life cycle of Taenia solium. Pigs can ingest human feces carrying embryonated eggs (or gravid proglottids) and hence become intermediate hosts in countries with poor sanitation. The embryonated eggs will then hatch, pass through the intestinal wall, enter the circulatory system, migrate to numerous tissues, such as the striatal muscle, and develop cysticerci in this tissue.[8] The cysticerci will hatch in the small intestine when humans consume contaminated undercooked pork meat. The tapeworm then attaches itself to the intestinal wall using its scolex, a jaw-like structure containing suckers and hooks. This is called taeniasis.[9] Humans are also able to become intermediate hosts by consumption of foods or drinking water infected with Taenia solium eggs or by human-to-human transmission. The same situation as in the case of pork will occur, with the striatal muscle, subcutaneous tissues, central nervous system, and eyes being the most afflicted organs. The condition is known as neurocysticercosis when it occurs in the central nervous system.[3] We suspect that the patient we reported experienced neurocysticercosis through eating undercooked pork that contained Taenia solium eggs .

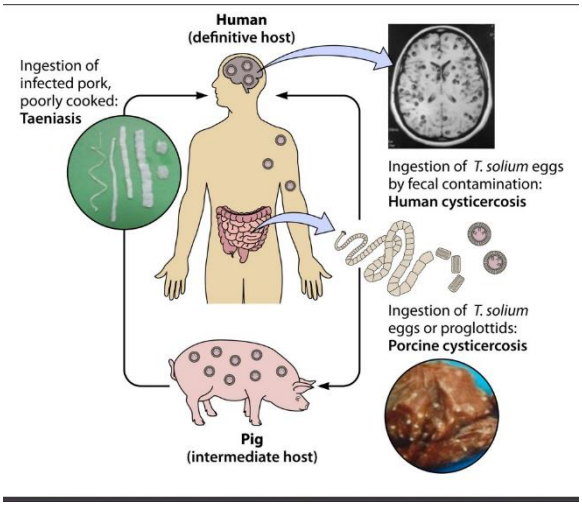

Figure 3. Life cycle of *Taenia solium* [8]

Neurocysticercosis is classified into two types based on the parasite's location: parenchymal (in the brain and medulla tissues) and extraparenchymal (primarily in the intraventricular and subarachnoid spaces, in between the leptomeninges, arachnoids, and pia mater, where cerebrospinal fluid circulates). The clinical, therapeutic, and prognostic consequences of neurocysticercosis in the parenchymal and extraparenchymal types are completely different.[10], [11] We discovered a parenchymal neurocysticercosis in this patient.

There are four stages of parenchymal neurocysticercosis, and the neuroimaging results varies depending on the stage. In the vesicular stage, the cyst measures 4-5 mm, is filled with clear fluid, has a thin, semi-transparent wall, an eccentric opaque scolex, and usually does not display symptoms. The cyst begins to deteriorate in the colloid stage, causing an inflammatory reaction that culminates in the replacement of clear cyst fluid with gelatinous material due to hyaline degeneration in the larvae. In the granular nodular stage, the cyst wall is replaced by lymphoid nodules and necrosis with the scolex transforming into mineralized granules, edema decreases, and ring enhancement persists in CT scan. In the nodular calcification stage, CT scan reveals granular tissue that has converted into calcified nodules without contrast enhancement.[12] In this case, CT scan showed an oval lesion with heterogeneous density in the right centrum semiovale with minimal perifocal edema and ring contrast enhancement indicating the patient was in the granular nodular stage of parenchymal neurocysticercosis.

Clinical signs of neurocysticercosis range from asymptomatic to severe critical symptoms that are related with larval size, quantity, stage, location, and host immune reaction.[13] In this case, the cyst is located in the white matter (in the right centrum semiovale), that generates adult-onset epilepsy.[6] Headache, which is the second most frequent symptom after epilepsy and focal neurological deficit (unilateral hemiparesis), which is a rare symptom of neurocysticercosis (only 5-15% of cases) are also experienced by this patient.[13[

Seizures are the major or perhaps the sole symptom of parenchymal neurocysticercosis, occurring in 80% of symptomatic patients.[14] Neurocysticercosis is the predominant cause of acquired epilepsy in the majority of the developing world, and it has been regarded as on of the primary disease causing the excess percentage of epilepsy recorded from these areas.[15] According to research, all stages of parenchymal neurocysticercosis are connected with reactive seizure via different mechanisms. At the vesicular stage (viable cysts), it develops as a result of inflammation or compressive effects on the brain parenchyma. Reactive seizure may develop in the colloidal and granular stages as a result of the inflammatory process associated with the host immune system's attack on the parasites. However, at the nodular calcified stage, gliosis occurs around deceased parasites, and late exposure of remaining antigenic material to the brain parenchyma may result in a persistent epileptogenic lesion.[16] Recurrent inflammatory episodes and consequent reactive seizure activity may culminate in

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persistent local or distant epileptogenic lesions, resulting in acquired epilepsy.[17[

Parenchymal neurocysticercosis is associated with the focal neurological deficit such as pure motor hemiparesis (PMH).[18] PMH can be caused by lacunar infarction or space occupying lesions.[19] In regard to our case, unilateral hemiparesis that occur in this patient was not caused by lacunar infarction based on neuroimaging results. A CT scan of the brain revealed no indication of recent infarction or bleeding. An oval lesion in right centrum semiovale with minimal perifocal edema were the results in CT scan of brain. As this is not owing to a lacunar infarction, the hemiparesis in our case is most likely attributable to the compressive impact of oval lesion with minimal perifocal edema on the right centrum semiovale.

Most focal neurological deficit cases caused by space occupying lesion are irreversible.[20] However, after dexamethasone, phenytoin, and albendazole treatment, the patient's clinical manifestations in this case improved significantly during hospital stay. The symptoms of recurrent seizures and focal neurological deficit were gone after one month of medication, and the cyst was no longer visible on MRI findings .

Dexamethasone which is a steroid is used to control local inflammatory effects (cerebral edema) and minimize the worsening of neurological symptoms due to parasite death.[21] Antihelmentic drugs that usually used for neurocysticercosis are praziquantel and albendazole. However, in our case, we used albendazole since it is more effective than praziquantel in eliminating cysts and causing overall clinical improvement, and it also has good pharmacological interactions with dexamethasone. Dexamethasone increases plasma level of albendazole by decreasing the clearance of albendazole sulfoxide, the active metabolite of albendazole.[22] Antiepileptic drugs are frequently utilized since seizures are the most prevalent manifestation of neurocysticercosis. Phenytoin and carbamazepine are the first line therapy for control secondary seizures due to neurocysticercosis.[23[

Conclusion

Neurocysticercosis is one of the most neglected tropical diseases. It caused by infection of Taenia solium via fecal-oral route. In our case, the patient experienced neurocysticercosis through eating undercooked pork that contain Taenia solium eggs. Neurocysticercosis is classified into two types based on the parasite's location, such as parenchymal and extraparenchymal with different clinical manifestation, therapeutic, and prognosis. We report a parenchymal neurocysticercosis with atypical phenomenon, such as focal neurological deficit (unilateral hemiparesis). Although in general focal neurological deficits due to space occupying lesions are irreversible, in this case we report a neurocysticercosis patient with improvement in symptoms and neuroimaging results. This offers up new paths for research into the causes of focal neurological deficit other than infarction and hemorrhage.

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Author contribution: We declare that all listed authors have made substantial contributions to all of the following three parts of the manuscript:

-Research design, or acquisition, analysis or interpretation of data؛

-drafting the paper or revising it critically؛

-approving the submitted version.

We also declare that no-one who qualifies for authorship has been excluded from the list of authors.

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Ethical considerations: This case report is based solely on anonymized laboratory data without involving direct patient interaction or intervention. No identifiable patient information is included, ensuring compliance with ethical guidelines and does not require formal ethical clearance as it involves retrospective data analysis.

Highlights:

- A report of parenchymal neurocysticercosis with atypical phenomenon, such as focal neurological deficit due to space occupying lesions.

- Neurocysticercosis is one of the most neglected tropical diseases that caused by infection of *Taenia solium* via fecal-oral route.

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