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CASE REPORT

Neurocysticercosis-related epilepsy in a South African patient: a case report

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Neurocysticercosis (NCC) is considered a public health problem in various parts of the world and it is one of the preventable causes of epilepsy in developing countries. It is defined as the infection of the central nervous system (CNS) and meninges by the larval stage of *Taenia solium*. This case report discusses a male patient diagnosed with NCC who presented with a single brain lesion, and who subsequently developed seizures.

NCC is not only a significant cause of acquired epilepsy in many developing countries, but it is also of increasing concern in developed countries due to the increased immigration of cysticercus carriers. Although NCC (parasitic infection caused by pork tapeworm) and taeniasis (parasitic infection caused by various tapeworm species) may be encountered worldwide, the prevalence of NCC is highest in specific communities in developing countries. Factors that influence this high prevalence include feeding on pork, poor sanitary conditions, and free-roaming of pigs. NCC remains on the World Health Organization (WHO) list of neglected tropical diseases; endemic primarily in developing countries south of the Sahara, Asia, and Latin America.

Keywords: neurocysticercosis, cysticercus, epilepsy, south of the Sahara, taeniasis, World Health Organization

Introduction

Neurocysticercosis (NCC) is classified as a widespread, commonly occurring helminthic infection of the central nervous system (CNS). It is acquired by ingesting eggs of the pork tapeworm, *Taenia solium*. NCC is also one of the most important causes of secondary epilepsy worldwide.[1]

While the causes of epilepsy vary, infections of the CNS are at the forefront of the aetiologies of epilepsy and/or epileptic seizures in persons in countries south of the Sahara.[2] It is also of concern in developed countries due to the migration of cysticercus carriers.[1,3,7,9]

The life cycle of *Taenia solium* cysticercus and its development in the brain

Two hosts, namely humans and pigs (porcine), are involved in the life cycle of *Taenia solium*. The parasite's life cycle is shown in Figure 1.[4] Pigs are the intermediary hosts, whereas humans act as both definitive and intermediary hosts. Cysticercosis is a zoonotic disease primarily caused by the direct ingestion of *Taenia solium* eggs from carriers via faecal-oral contamination.

Pigs were thought to be solely responsible for the occurrence of human cysticercosis. However, Garcia and Del Brutto report that although pigs are part of the life cycle of the tapeworm (*Taenia solium*, the causal agent of cysticercosis), human and porcine cysticercosis (primarily in muscle and the CNS) is only acquired by ingestion of microscopic tapeworm eggs through faecal-oral contamination.[5]

The larval stages of the parasitic cestode, *Taenia solium*, can infect both humans and pigs causing a condition known as cysticercosis. The root cause of this infection occurs when eggs shed in the faeces by human tapeworm carriers are inadvertently consumed [1]. The eggs are highly contagious and do not need time outside of a host to develop. Ingesting either the eggs or gravid proglottids (loosely defined as numerous developing eggs in various stages, housed within their branched uterus) can lead to infections in both pigs and humans [2], [7].

Typically, humans are exposed to eggs by consuming food and water that has been tainted by faecal matter carrying these eggs or proglottids, as well as via transmission from person to person. Tapeworm carriers may also infect themselves through faecal-oral transmission (e.g. due to improper hand hygiene). Following ingestion of either the eggs or proglottids, oncospheres will hatch within the individual's intestine [3], [8] leading to invasion of the intestinal wall, bloodstream entry, and migration to several tissues and organs where they develop over 60–70 days into cysticerci [4], [9]. Some cysticerci will migrate to the CNS, causing serious sequelae such as NCC.

This differs from other taeniasis, which causes intestinal infection with adult tapeworms. Humans acquire intestinal infections with *Taenia solium* after eating undercooked pork containing cysticerci [5]. Cysts evaginate and use their scolices to affix to the small intestine. Adult tapeworms mature and can remain in the small intestine for years [6].

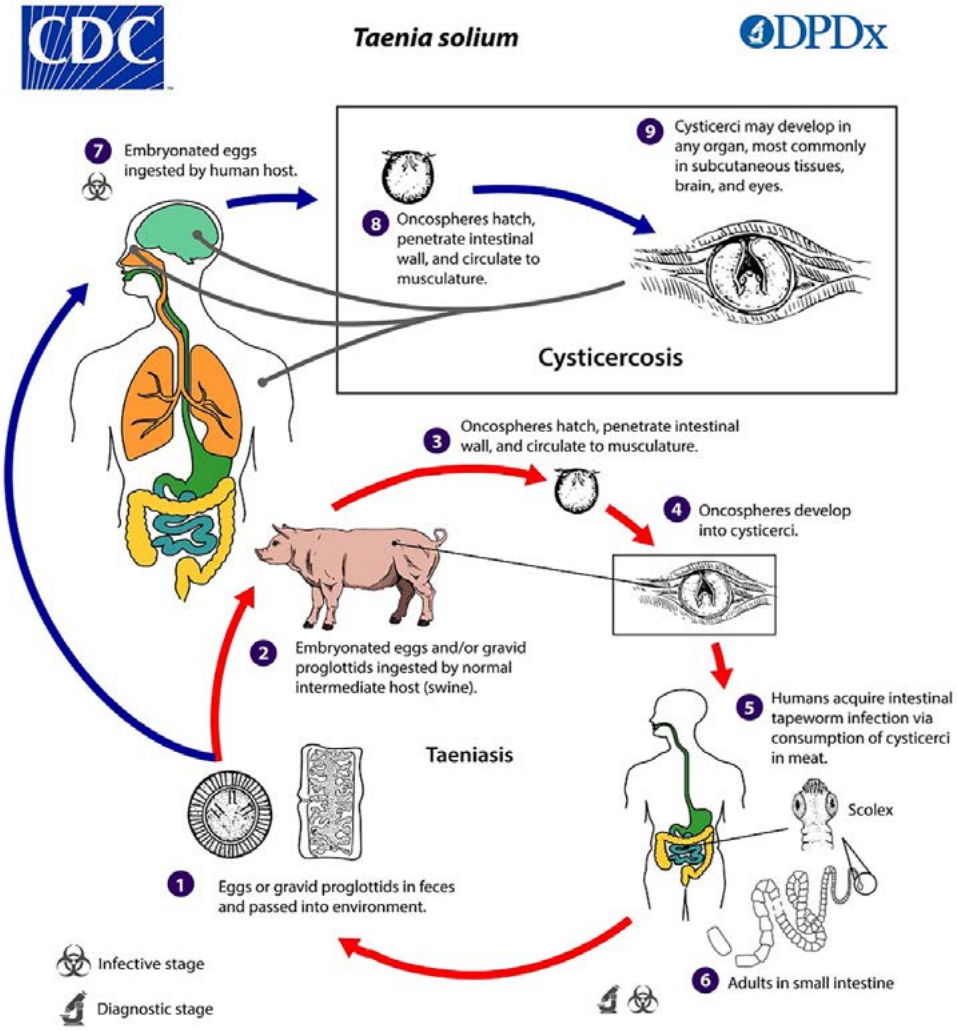

Figure 1: Life cycle of *Taenia solium* cysticerci[4]

Case report

A 72-year-old male presented at his local hospital on 23 June 2022 with a first generalised epileptic seizure. Three months before this episode, he experienced the occasional dizzy spell and had regular headaches with right-eye muscle pulling.

On examination, the patient was clinically afebrile, fully conscious, orientated, and showed no neurological abnormalities. A dietary history of the patient revealed no regular consumption of pork or pork products. On further questioning, the patient could not remember eating any undercooked pork or undercooked pork products. Unfortunately, no long-term dietary history was documented. Therefore, it is plausible that the patient became infected by ingesting *Taenia solium* eggs through faecal-oral transmission, or by possible autoinfection.

Magnetic resonance imaging (MRI), computed tomography (CT) scan, blood samples (haematology and chemistry), a cerebrospinal fluid (CSF) analysis, and an electroencephalogram (EEG) were requested at the time of admission. The CT scan, shown in Figure 2, confirmed a solitary cystic lesion with periventricular and subcortical white matter leukoaraiotic changes in the left parietal region surrounded by microvascular ischaemic, demyelinating changes. The EEG showed no abnormalities. Haematological, blood chemistry tests and CSF analysis were all within normal limits.

The patient was subsequently treated with an anthelmintic, ZENTEL (albendazole) 400 mg, at a dosage of one tablet

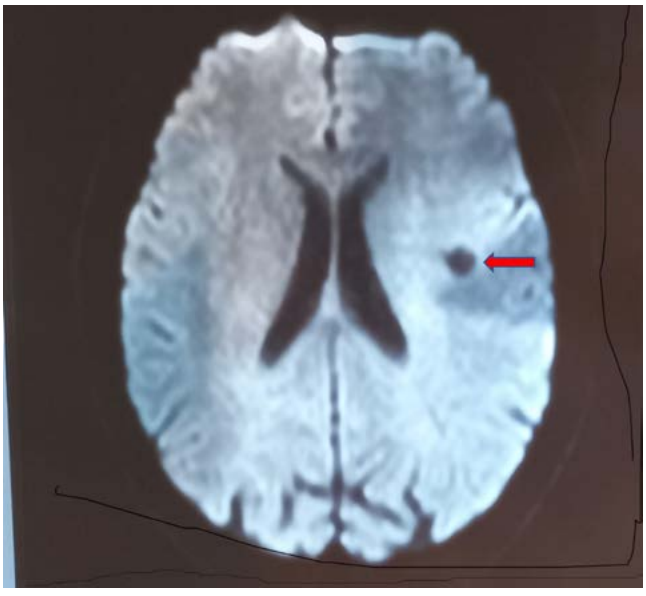

Figure 2: Patient neuroimaging CT scan showing a solitary cystic lesion (arrowed) in the left parietal region; this finding is characteristic of NCC

three times daily for three weeks. The patient also received an anticonvulsant, Epilizine CR 200 mg (sodium valproate), at a dosage of one tablet twice daily.

The patient suffered a second seizure after two months of Epilizine CR 200 mg treatment. This anticonvulsant therapy was adjusted to 400 mg, at a dosage of one tablet twice daily. The patient's treatment was consolidated with Epilizine CR 500 mg, at a dosage of one tablet twice daily.

The patient continued to remain seizure-free following NCC treatment. Regular monitoring every five months is essential to assess a patient's progress. This is necessary to evaluate any recurrence or complications that may occur so the treatment plan can be adjusted accordingly.

Discussion

The clinical picture of NCC is variable; seizures and epilepsy are the most common manifestations, followed by headaches. The diagnosis of NCC is well established and is based on neuroimaging findings as seen in this patient.[6]

It is not fully understood why some patients develop seizures/epilepsy while others do not. Differences across these categories of patients may explain the predisposition to develop epilepsy.[7] However, a higher proportion of patients with inactive NCC present with seizures and/or epilepsy when compared with patients who have active lesions. Furthermore, patients with an increased number of cysts develop more seizures. The patient described had only one calcified cyst in the left parietal region of the brain but still developed seizures. Calcified NCC represents the healed end-stage of the natural evolution of cysticercus cellulosae (the larval form of *Taenia solium*) in the brain parenchyma and is considered to have a causal relationship with epilepsy. Despite this, in a proportion of the population in endemic countries, it may remain asymptomatic and often such lesions are discovered accidentally. Hence, the actual burden of epilepsy is unknown.

From a total of 25 studies across regions south of the Sahara, including two from South Africa, Owolabi et al.[3] concluded that the overall prevalence of NCC in patients with epilepsy was 22%. This figure varied within the territories of regions south of the Sahara. The major risk factors for acquiring NCC are poor environmental sanitation and poor food and drinking water hygiene. NCC may be controlled and prevented by generating awareness of hygienic consumption of food and water and the provision of good sanitation.

Evidence suggests that increased serum levels of matrix metalloproteinase-9 (MMP-9) were significantly associated with seizure recurrence.[8] Additionally, the TLR4 gene abnormalities may trigger inflammation around calcified NCC, leading to an increase in perilesional oedema and provocation of seizures.[8] Our knowledge of NCC has undoubtedly improved over the past two decades. However, these advances in diagnostic and therapeutic approaches by no means represent the final word regarding the disease.[9]

The recommendations detailed in the World Health Organization (WHO) guidelines on the management of *Taenia solium* include some of the following modalities for diagnosing NCC:[10]

- Neuroimaging using either a CT scan or MRI is considered the gold standard for diagnosing NCC.
- Serological testing for detecting parasitic antigens may be helpful in remote and rural settings where access to sophisticated neuroimaging technology is not available.
- Antigen-ELISA testing is also useful to monitor parasite burden in response to antiparasitic treatment.

However, these assays may be difficult, owing to the lack of tests and insufficient sensitivity in patients with solitary or calcified cysticerci.

Concerns remain about the optimal length of therapy with antiepileptic drugs for patients with NCC seizures/epilepsy. For example, patients on prolonged antiepileptic medication usage may have short and long-term side effects. When a patient with seizures/epilepsy is in remission (free of seizures for two years or longer) discontinuation of medication may be justified.[2] Also, albendazole (the main ingredient in ZENTEL) leads to leakage of parasite antigen, resulting in an increase in perilesional oedema, causing neurological symptoms other than those caused by cysts in the brain.

Conclusion

Further research is necessary, especially in developing countries, to better understand NCC and the long-term sequelae of calcified cysticerci in the brain parenchyma, which may be associated with breakthrough seizures. It is important to note that the treatment of NCC depends on the viability of the cyst and its complications. Therefore, treatment must be individualised based on the disease manifestations. Finally, education strategies for the prevention of NCC must be considered in countries south of the Sahara. This approach will, hopefully, reduce the burden of NCC and thereby improve overall public health.

Conflict of interest

None.

Funding source

None.

Ethical approval

Written, informed consent was obtained from the patient for the publication of this case report and the accompanying image. If necessary, a copy of this written consent is available for review from the Editor-in-Chief of the JMLST SA.

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