Cortical Blindness due to neurocysticercosis in an adolescent patient

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Case Report

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Abstract

BACKGROUND: Neurocysticercosis (NCC) is a common cause of recent-onset seizures in both adults and children in tropical areas, especially when there is no other suggestion of an underlying neurological disorder. In addition, there have been reports of very rare cases of bilateral cortical blindness caused by helminths in children. It is still unclear whether healthy adolescents with no pre-existing health problems could be vulnerable to developing such sequelae due to NCC.

CASE REPORT: We report a case of a 14-year-old African boy from Nigeria with bilateral cortical blindness caused by NCC due to *Taenia solium*. According to the boy's mother, symptoms began with headaches, vomiting, fatigue, visual loss, and fever (40.0 °C). Clinical investigations led to a diagnosis of cortical blindness and encephalitis due to NCC. Appropriate treatment was administered, and it resulted in the resolution of most symptoms, though the patient remained permanently blind.

CONCLUSIONS: After reviewing the literature and experience learned from our patient case presentation, we suggest that early neurological evaluation and serology tests for NCC in patients with seizures of unknown origin and with a history of travel to endemic regions should prompt a workup for NCC and immediate treatment. Localization of cysticerci can occur in any part of the brain; therefore, cyst invasion and damage of the occipital cortex can cause bilateral vision loss.

Background

Humans contract cysticercosis through the feco-oral route by ingesting *T. solium* eggs from tapeworm-contaminated surfaces or carriers [1]. Two to three months after infestation, the ingested eggs can be found in organs such as the heart, lungs, liver, and abdominal cavity, but rarely in the spinal cord. Since the neurological symptoms of neurocysticercosis (NCC) are nonspecific, it is difficult to diagnose the disease based on clinical findings alone [2].

NCC develops when metacestodes of *Taenia solium* spread through the bloodstream and are seeded in the brain. The presence of eggs causes localized edema in the affected part of the brain, which can manifest as different neurological symptoms/deficits. NCC is prevalent in Africa; it raises concerns about public health and economic livelihoods as it causes morbidity and mortality on the continent [3]. The climate also contributes to parasite transmission in Africa. Africa's climate zones are divided into the arid zone (north of Senegal, Mali, Burkina Faso, and Niger), semi-arid zone (Southern Senegambia, Guinea-Bissau, Togo, Benin, and Nigeria), and sub-humid zone (north of Ghana, Cote d'Ivoire, Sierra Leone, Benin, and central parts of Nigeria). These regions have a high level of heterogeneity, which influences the transmission of cysticercosis; thus, there is a high need for more studies on the high prevalence rates of NCC in these regions [3].

In this report, we present a case of bilateral cortical blindness due to NCC in a 14-year-old African boy from Nigeria. The patient presented to our clinic with complaints of headaches, fever (40.0°C), bilateral vision loss, hemiparesis, nausea and vomiting, confusion, and neck stiffness. A thorough neurological

examination with CSF analysis and imaging studies was conducted, and a diagnosis of cortical blindness and encephalitis due to NCC was made. The appropriate treatment was administered to the patient, and there was resolution of symptoms, with a residual complication—visual deficits in both eyes.

Case Presentation

We present a case of a 14-year-old African boy from Nigeria who presented to the hospital with symptoms of throbbing headaches, vomiting, fever (40.0°C), slurred speech, seizures, hemiparesis, bilateral vision loss, suprapubic pain, and confusion. The symptoms had began 3 days before the hospital visit. The patient's parents thought the symptoms were of malaria and started him on an antimalarial drug (hydroxychloroquine) and Tylenol for the pain. However, when the patient's symptoms failed to improve and he complained of not being able to see, he was brought to the hospital for treatment.

Neurological examination upon arrival revealed bilateral vision loss and hemiplegia in the left part of both upper and lower extremities, with loss of sensation. The patient also complained of suprapubic discomfort and had urinary retention which raised a suspicion of a possible urinary tract infection. His immunizations were all up to date, and he had no family history of neurological diseases.

During the physical examination, he was febrile (40.0°C), confused, and oriented only to his name. He had a seizure episode that was relieved with phenytoin. His body mass index was normal (21 kg/m²). Ophthalmological examination revealed normal pupillary light reflexes and no obvious cause of vision loss.

Several diagnostic tests were performed, including blood culture, urinalysis, and cerebrospinal fluid analysis [Table-1]. Serology (enzyme-linked immunoelectrotransfer blot) of blood for cysticercosis antibodies to glycoprotein antigens was positive, suggesting cysticercosis. Head computed tomography (CT) scan showed local soft tissue inflammation due to cyst degeneration [Fig. 1], scolexes in the cerebral cortex, and multiple cysticercus granulomas in the cerebral cortex (Fig. 2). Brain magnetic resonance angiography (MRA) showed a wedge-shaped T1-Weighted hypointense and T2-Weighted hyperintense lesion in the body of the right caudate nucleus. Diffusion of contrast was restricted on diffusion-weighted imaging. The lesion measured 2.3 cm in diameter and was suggestive of an acute/subacute infarct. Multiple T1W hypointense areas with T2W and FLAIR hyperintense areas were seen in the subcortical regions of both the temporal and parietal lobes (Fig. 3). There was no restriction of contrast diffusion on DWI, suggesting white matter changes. A final diagnosis of cysticercal encephalitis with cortical blindness was made.

Management of the patient began with bladder catheterization, which drained 200 ml of cloudy urine and relieved the suprapubic discomfort. Urinalysis showed bacteriuria, and IV levofloxacin 250 mg was administered every 24 h for 3 days. A slow IV infusion of 15 mg/kg phenytoin was also administered to control the seizures. A glucocorticoid (IV methylprednisolone 20 mg every 6 h) was administered at a

lower pressure in the brain. An antihelminth drug (oral albendazole 400 mg BID) was also added to the treatment regimen. The patient's symptoms improved over the course of five days of in-hospital stay. A prescription of oral albendazole at the same dosage was administered to the patient for 10 additional days upon discharge from the hospital.

A repeat brain CT with contrast was performed 3 months after discharge from the hospital, and it showed complete resolution of the lesions [Fig-4]. There was also no further recurrence of the symptoms at the 1-year follow-up visit, and no neurological abnormality was noted during clinical examination of the patient, except for bilateral blindness.

Discussion

Neurocysticercosis (NCC) is a major cause of morbidity and mortality in people of all ages [4]. Since it is seen as a disease of those with a poor socioeconomic status, the approach towards its control has remained controversial. NCC is known to be the most common cause of adult-acquired epilepsy worldwide, and it causes 30% of the epilepsy cases in most endemic areas of Africa, Asia, and Latin America, particularly in regions where people live in proximity to pigs [5]. However, tourism is becoming increasingly prevalent in developed countries. Criteria for the diagnosis of NCC based on clinical, neuroimaging, serological, histopathological, and epidemiological approaches have been suggested by Del Brutto et al. [6]. These criteria are used to diagnose NCC based on positive neuroimaging findings, resolution of the cystic lesions after antihelminth therapy, history of travel to disease-endemic areas, and clinical symptoms, and were the basis for the diagnosis of NCC in our patient. Hemiparesis in our patient could be due to extraparenchymal NCC vasculitis caused by inflammatory occlusion of the arteries at the brain stem due to arachnoiditis.

Visual loss in NCC can be multifactorial. A study conducted by Chang et al. [7] analyzed 23 patients with vision loss due to NCC. Approximately 50% of the cases were due to optic neuropathy caused by papilledema. The remaining lesions were caused by chiasmal and retrochiasmal lesions [8]. The etiology of cortical blindness due to cysticercosis remains poorly understood. In our patient, we hypothesized that the cause of vision loss was either parenchymal cyst invasion of the arteries of the base of the brain, resulting in vasculitis of the occipital branch of the cerebral artery, or compression of the posterior cerebral artery by large cysts resulting in infarction and subsequent blindness. It is important to note that cysts can spread to every part of the brain, and due to local immune reactions to the presence of these cysts, their stage, location, and size, NCC can present with different neurological symptoms. It is possible that our patient acquired *T. solium* infestation several years prior to symptom manifestation. Several studies have described the unusual clinical manifestations of NCC-like extrapyramidal symptoms, such as hemiballismus [9], dorsal midbrain syndrome [10], and nominal aphasia. Therefore, more research focusing on the newer and more effective treatment strategies for the management of patients with NCC cortical blindness is strongly needed.

Conclusions

NCC can produce complex neurological symptoms, and, in most cases, have an undulant course. It is a treatable and preventable disease. The possibility of this infectious disease should always be considered during the diagnostic work-up of any patient with symptoms suggestive of neurological, personality, or cognitive disorders, particularly among patients who have traveled to or lived in an endemic region. Prompt treatment is imperative for preventing permanent neurological damage.

Declarations

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The authors declare no competing interest. The legal guardian of the patient gave full consent/approval for the publication of this case report.

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Table

Variables	patient	normal
Cell count	30 cells (eosinophils)	< 5
Glucose	55mg/dl	50-80 mg/dl
Protein	70mg/dl	15-60mg/dl
Opening pressure	100mmH ₂ O	90-180mmH ₂ O

Table-1: CSF analysis showing moderate pleocytosis with eosinophilia, and slight increase in protein with high concentration of gammaglobulin.

Figures

Figure 1

Head computed tomography (CT) scan showing local inflammatory soft tissue reaction to cystic invasion in the posterior cerebral cortex.

Figure 2

Head CT scan showing multiple scolexes (red arrows) in the cerebral cortex.

Figure 3

Head CT scan showing Multiple T1W hypointense, with T2W and FLAIR

hyperintense, areas in the posterior and parietal cortices, indicative of infarction due to stroke from cysticercal invasion of the vascular bed.

Figure 4

Head CT scan taken three months after discharge from hospital showing complete resolution of the cysts.