Brain Abscess and Cortical Blindness Due to Neurocysticercosis in an Adolescent Patient

*Corresponding Author(s): Nnennaya U Opara*
Charleston Area Medical Center. Health Education and Research Institute, 3200 MacCorkle avenue SE, Charleston, WV, 25304, USA.
Tel: 304-388-9920; Email: Nnennaya.Opara@camc.org

Received: Dec 29, 2021
Accepted: Jan 12, 2022
Published Online: Jan 14, 2022
Journal: Neurology and Neurological Sciences: Open Access
Publisher: MedDocs Publishers LLC
Online edition: http://meddocsonline.org/
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**Keywords**: Brain abscess; Neurocysticercosis; Cortical blindness; Hemiparesis; Vomiting; fever.

**Abstract**

**Background**: Neurocysticercosis is generally known as a common cause of most recent-onset seizures in both adults and children in Tropical areas especially where there is no other suggestion of an underlying neurological disorder. However, very rare cases of bilateral cortical blindness caused by helminths in children has been reported. It is still unclear whether healthy adolescents with no pre-existing health problem, could be vulnerable to developing such sequelae due to Neurocysticercosis.

**Case Report**: We report the case of a 14-year-old African boy from Nigeria with bilateral cortical blindness caused by Neurocysticercosis with Taenia Solium. According to the boy’s mother, symptoms began with headaches, vomiting, fatigue, visual loss, and fever (100F). Clinical investigations led to a diagnosis of cortical blindness and encephalitis due to NCC. Appropriate treatment was administered which resulted in resolution of most symptoms but permanent blindness.

**Conclusions**: After review of 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 history of travel to endemic region should prompt a workup for NCC, and immediate treatment. Localization of cysticerci can occur in any part of the brain, and so with cysts invasion and damage to the occipital cortex could cause bilateral vision loss.

**Background**

Humans, contract cysticercosis through Feco-oral route with T. Solium eggs from tapeworm contaminated surfaces or carriers [1]. Following 2-3 months after infestation, the ingested eggs could be found in other organs such as heart, lungs, liver, and abdominal cavity, but rarely found in spinal cord. Lack of specificity of neurological symptoms of Neurocysticercosis, makes it difficult to diagnose the disease based off clinical findings alone [2].

The term Neurocysticercosis, is described as a condition, which develops when metacestodes of Taenia Solium, spreads through the blood stream and is seeded in the brain. The presence of the eggs causes local swellings in the affected part of the brain, which could manifest with different neurological symptoms/deficits. Neurocysticercosis is prevalent in Africa and raises concern for public health and economic livelihood causing morbidity and mortality in these regions [3]. Climate can also be a contributory factor to the parasite transmissions in Africa. Africa’s climate zones a divided into: Arid (North of
Senegal, Mali, Burkina Faso, and Niger). Semi-arid zone (south-
ern SeneGambia, Guinea-Bissau, Togo, Benin, and Nigeria). The 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 cys-
ticercosis throughout these areas, thus, there is a high need for
more studies centered in these regions on Neurocysticercosis
high prevalence rates [3].

In this report, we present a case of bilateral cortical blind-
ness caused by Neurocysticercosis in a 14-year-old African male
from Nigeria. Patient presented to our clinic, complaining of
headaches, fever 102 Fahrenheit, loss of vision in both eyes,
hemiparesis, nausea & vomiting, confusion, and neck stiffness.
A thorough neurological examination with CSF analysis and
imaging studies was conducted, which yielded a diagnosis of
cortical blindness and encephalitis due to Neurocysticercosis.
Appropriate treatment was administered to patient, and there
was resolution of symptoms with some residual complication-
visual deficits in both eyes.

**Case presentation**

We present a case of a 14-year-old African male from Ni-
geria who presented to the hospital with symptoms of throb-
ing headaches, vomiting, fever 102 F, slurred speech, seizures,
hemiparesis, bilateral loss of vision, suprapubic pain, and confu-
sion. Symptoms began 3 days prior to the hospital visit. Patient’s
parents thought the symptoms were of malaria origin and man-
aged that with antimalarial drugs (hydroxychloroquine) and Ty-
lenol for the pains. However, when patient’s symptoms failed to
improve, and patient complained of not being able to see, he
was brought to the hospital for treatment.

Neurological examination upon arrival revealed bilateral loss
of vision, hemiplegia of the left part of both upper and lower
extremities with loss of sensation. Patient also complained of
suprapubic discomfort with urinary retention. His immuniza-
tions were all up to date. There was no family history of any
neurological diseases.

During physical examination, he was febrile (104F), confused,
and oriented only to name. Patient had an episode of seizure
which was relieved with phenytoin. BMI was normal (21kg/m²).
Ophthalmological examination showed normal pupillary light
reflexes and with no obvious cause for his vision loss.

Several diagnostic tests were done which included Blood
culture, urinalysis, and cerebrospinal fluid analysis were per-
formed serology (enzyme-linked immunoelectrotransfer blot
EITB) of blood for cysticercosis antibodies to glycoprotein an-
tigens was positive suggestive of cysticercosis. Head CT scan
showed soft tissue local inflammation caused by cyst degenera-
tion (Figure 1) and presence of scolexes in the cerebral cortex
and multiple cysticercus granuloma in the occipital lobe (Figure
2). Brain MRA report also shows a wedge-shaped T1-Weighted
hypointense and T2-Weighted hyperintense lesion in the body
of the right caudate nucleus. There was a restricted diffusion
of contrast on DWI. The lesion measured 2.3cm in diameter
suggestive of an acute/subacute infarct. Multiple T1W hypoin-
tense, with T2W and FLAIR hyperintense areas were seen in the
subcortical regions of both temporal and parietal lobes (Figure
3). There was no restriction of contrast diffusion on DWI1, sug-
gestive of white matter changes. A final diagnosis of cysticercal
encephalitis with cortical blindness was reached.

Management of patient began with bladder catheterization,
which yielded 200ml of cloudy urine and relieved the suprapu-
bic discomfort. Urinalysis showed bacteriuria, and IV antibiotics
with Levofoxacin 250mg every 24 hours for 3 days was started.
Slow IV infusion of 15mg/kg infusion of phenytoin was also
given to control seizures. Glucocorticoid (IV methylprednisol-
one 20mg every 6 hours) was administered to lower pressure
in the brain. Anthelmintic drug (oral Albendazole 400mg BID)
was also added to the treatment regimen. Patient symptoms
improved over the course of five days of in-hospital stay. A pre-
scription of oral Albendazole with same dosage was given to pa-
tient for additional 10-day course upon discharge from hospital.

A repeat brain CT with contrast was done 3 months following
discharge from hospital, which showed a complete resolution
of the lesions (Figure 4). There was also no further recurrence
of symptoms at 1-year follow-up visit, and no neurological ab-
normality was seen during clinical examination of patient ex-
cept for blindness in both eyes.
Neurocysticercosis is a major cause of morbidity and mortality in people of all ages [4]. Since it is seen as a disease of the poor socio-economic status, the approach towards its control has remain controversial. Neurocysticercosis is known to be the most common cause of adult-acquired epilepsy world-wide and causes 30% of epilepsy cases in most endemic areas of Africa, Asia, and Latin America, particularly the regions where people live in proximity with pigs [5]. However, due to tourism, it is becoming increasingly prevalent in some developed countries. A criterion for Neurocysticercosis diagnosis based on clinical, neuroimaging, serological, histopathological, and epidemiological approaches have been suggested by Del Brutto et al., [6]. These criteria define the diagnosis of Neurocysticercosis based on positive neuroimaging findings, resolution of cystic lesion after anthelminthic therapy, history of travel to disease-endemic areas, and clinical symptoms as basis for diagnosis of NCC in our patient. Hemiparesis in our patient could be due to extra parenchymal NCC vasculitis caused by inflammatory occlusion of the arteries at the brain stem due to arachnoiditis.

Visual loss in Neurocysticercosis (NCC) can be multifactorial. A study conducted by Chang et al [7] analyzed 23 patients with loss of vision from NCC. Approximately, 50% were due to optic neuropathy from papilledema. The remainder were caused by chiasmal damage and retrochiasmal lesions [8]. However, the etiology of cortical blindness due to cysticercosis is still poorly understood. In our patient, we hypothesize that the cause of his vision loss is either due to parenchymal cysts invasion of the arteries of the base of the brain resulting in vasculitis of the occipital branch of cerebral artery or compression of the posterior cerebral artery by large cysts. It is important to note that cysts can spread to every part of the brain, and local immune reaction to the presence of these cysts, stage of cysts, location, and sizes, presents with different neurological symptoms. Additionally, there could be a possibility that he must have acquired T. Solium infestation years prior to symptoms manifestation. Several literatures have described unusual clinical manifestations of NCC-like extrapyramidal symptoms such as hemiballismus [9], dorsal midbrain syndrome [10], and nominal aphasia. Therefore, more research studies focused on newer and effective treatment strategies for the management of patients with NCC cortical blindness is strongly needed.

Conclusions

Neurocysticercosis (NCC) can produce complex neurological symptoms, and in most cases, run 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 travelled to or lived in an endemic region. Prompt treatment is very imperative to prevent permanent neurological damage.

References