Case Report

Cranial CT Findings in a Nigerian Child with Transient Bilateral Cortical Blindness - A Case Report.

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Abstract

Transient cortical blindness is not an uncommon pathology but bilateral lesions with brain computed tomographic (CT) scan correlate has not been previously documented in the Nigerian population.

A 2-year old Nigerian female child presented 4 days following a fall. She was well after the fall for two days but she subsequently lapsed into unconsciousness with associated convulsions. At presentation she was febrile (Temperature: 38.5°C). Her peripheral blood film showed malaria parasite. There was no evidence of meningitis. Her brain CT revealed hypodensities in the occipital lobes, right temporal lobe and both cerebellar hemispheres. She became conscious one day after admission and following quinine administration. Cortical blindness was observed at this time. Follow up in the clinic showed resolution of the blindness. The probable aetiology and the management of the child are discussed.

Keywords: Bilateral cortical blindness, cerebral malaria, trauma

Introduction

Cortical blindness, otherwise called cortical visual impairment, can be caused by many factors including perinatal ischemic-hypoxic insult, trauma, intracranial infections, and infestations like cerebral malaria and following imaging studies like cerebral angiography and cranial computed tomography (CT)[1-10]. In many of these cases, the lesion is often unilateral and many are reversible. Only few cases have been reported in Nigerian population and these were often part of neurological complications of cerebral malaria[5,7,10]. Another rare cause reported from this environment is pregnancy induced hypertension[11]. To the best of our knowledge this is the first reported case of transient bilateral cortical blindness in Nigeria with CT correlation.

In this article we discuss the aetiology, cranial CT findings and management of a 2-year old Nigerian girl with reversible bilateral cortical blindness.

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Case Summary

A 2-year old girl was referred from a private hospital on account of a 2-day history of seizures. She fell from a height of about 8 meters 4 days prior to presentation. She fell with her occiput hitting a brick pavement. There was no loss of consciousness. She was initially managed conservatively and discharged home but developed generalized tonic-clonic convulsion two days after the fall. She

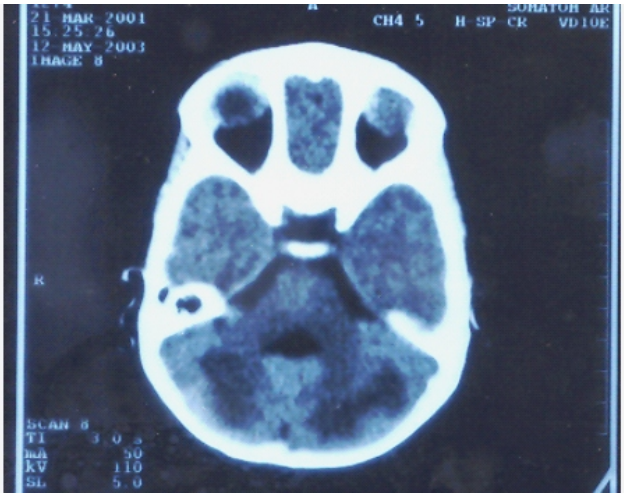


Fig. 1: Non-enhanced cranial computed tomographic scan of the patient showing hypodense lesions in both cerebellar hemispheres.

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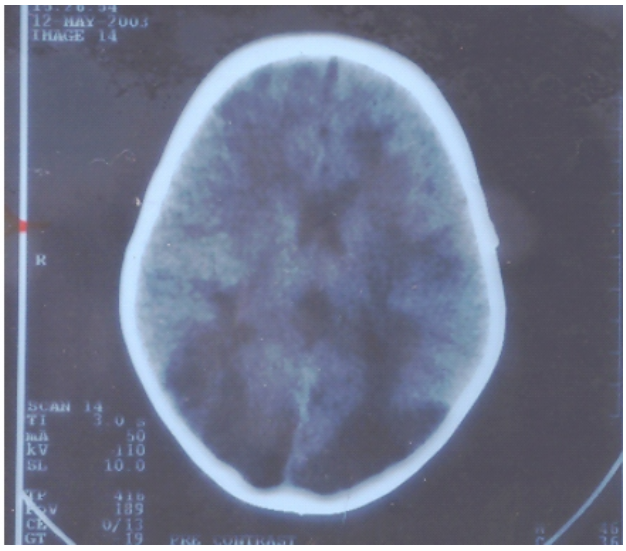


Fig. 2: Non-enhanced cranial computed tomographic scan of the patient showing multiple hypodense lesions worse in the both occipital lobes.

presented at another hospital from where she was referred to our centre. At presentation, she was unconscious but febrile with a temperature of 38.40C. Her pupils were 4mm bilaterally with good reaction to light. She had generalised hypertonia and hyperreflexia. There were no other abnormal findings. An initial diagnosis of traumatic intracranial haemorrhage with early posttraumatic seizure was made. Cerebral malaria was also considered as differential diagnosis. Her peripheral blood film revealed trophozoites of *Plasmodium falciparum*. Other laborarory results including cerebrospinal fluid analysis were within normal limit.. Her cranial CT scan revealed multiple areas of non enhancing hypodensities in the occipital lobes, both cerebellar hemispheres as well as the temporal lobes (figures 1 and 2). The lesions were worse in both occipital lobes. There was no intracranial haemorrhage. She was treated with quinine, antibiotics and dexamethasone. Antibiotics were withdrawn when laboratory investigations did not suggest an infection. She regained consciousness on the second day of admission. At this time it was observed that the patient could not see. Further ophthalmic evaluation showed nil light perception bilaterally with normal size pupil but with sluggish light reaction. The optic disc was normal on fundoscopy. We thus made a diagnosis of bilateral cortical blindness. She was discharged 12 days after admission. She was seen in the clinic 4 weeks after discharge. At this time, the patient had started seeing. She could walk without support and also able to follow fingers. The patient was scheduled for a repeat cranial CT but had been lost to follow up.

Discussion

Bilateral cortical blindness is not a common pathology. The causes of cortical blindness are many, whether it is unilateral or bilateral. Blindness following trauma, malaria fever, postmeningitic hydrocephalus, encephalitis, asphyxia neonatorum had been previously reported in Nigeria before the advent of brain CT[2,5,7,10].

The aetiology in this 2-year female Nigerian is not certain but there are two possibilities. In the first instance, the history of fever, convulsion and malaria parasitaemia would suggest cerebral malaria. The fact that the patient was well neurologically for two days after her fall would support this. Malaria causes multiple necrosis/ infarction of the brain. These lesions can be found in many parts of the brain but they are often very marked in the thalamus and the cerebellum[12]. These pathologic changes are obvious on the brain Ct[12] and magnetic resonance imaging (MRI). The CT findings of hypodense lesions in the occipital lobes and cerebellar hemispheres in our patient will thus support the diagnosis of cerebral malaria. The rapidity of the response to quinine treatment is also in keeping with cerebral malaria. Cortical blindness following cerebral malaria has been reported by many authors from Nigeria[2,5,7,10] but bilaterality with correlated CT changes have not been reported from this environment.

Another probable aetiology in this patient is head injury. The findings of multiple hypodense lesions on the brain CT may suggest multifocal contusions. The patient fell on her occiput and the lesions may suggest direct impact to these regions of

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the brain. However, given the extent of the brain CT changes observed, the patient should have developed neurological changes earlier than two days after the injury if this was the case, though late presentation from expanding oedema is not unlikely. We initially considered bilateral posterior cerebral artery thrombosis with infarction of the occipital lobes. The extent of the areas of hypodensities on the cranial CT and the associated cerebellar hypodense lesions would not support this. Cerebral vessel angiography would have been helpful to ascertain this but currently, this is not readily available in our centre.

Meningitis and extraaxial collection were also considered but results of the various investigations including cerebrospinal fluid analysis and cranial CT did not support these.

Whatever was the cause in this patient, the CT findings of bilateral occipital lobes infarctions would explain the patient's blindness due to damage to the geniculocalcarine fibres in these regions of the brain.

The appropriate management of the problem is not clear cut. The patient had 1 week cause of dexamethasone though the effect of this is not certain. There have been various reports on the spontaneous resolution of cortical blindness irrespective of the cause[10,12-13]. Thus, the patient could still have improved even if the steroid was not used.

What is also not clear is the pathway for vision in the improved cases of traumatic cortical blindness. The partially injured geniculocalcarine tracts may recover their function and this may be responsible for the improved vision. Some other authors have also suggested extra- geniculocalcarine pathways[14]. This may be a form of plasticity. Plasticity is very effective in children and may be the case in this 2-year old child giving the extent of damage on CT and the degree of improvement so far observed.

Cortical blindness is often unilateral but may be bilateral as in this case. Recovery may occur even in bilateral lesions. With the advent of CT scan and other modern imaging modalities diagnosis and prognostication in patients with bilateral cortical blindness may become easier. The clinical and laboratory findings as well as the rapid response to quinine administration would suggest cerebral malaria as the cause in this patient.

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