Case Report of a 52 Year Old Hypertensive with Cerebral Toxoplasmosis as a Stroke Mimic

Type of Article: Case Report

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ABSTRACT

BACKGROUND

The diagnosis of acute ischaemic stroke is sometimes straightforward. The sudden onset of a focal neurologic deficit in a recognizable vascular distribution with a common presentation - such as hemiparesis, facial weakness and aphasia - identifies a common syndrome of acute stroke.[1] But differential diagnostic problems remain because there are some non-vascular disorders which "mimic" stroke. Such mimics have clinical pictures that appear identical to stroke.[1] However, standard acute neuroimaging with non-contrast CT scanning will discover some of these mass lesions mimicking stroke. We therefore present a brief case report on cerebral toxoplasmosis as a stroke mimic

METHODS

The case records of a patient who presented with cerebral toxoplasmosis mimicking a stroke and a review of the literature using google, Medline and PubMed search as well as available literature on the subject were utilized.

RESULTS

We present a 52 year banker, a known hypertensive of 2 years duration who claims good adherence to medications. He presented to us 16 days after he a history of inability to move the left side of his body and other neurologic features suggestive of a right hemispheric ischaemic stroke. Brain CT showed features of cerebral toxoplasmosis and he was found to be seropositive HIV 1.The patient was placed on pyrimethamine, sulphadiazine, dexamethasone, antihypertensives and HAART. He made remarkable improvement on discharge and follow up.

CONCLUSION

It is important for physicians to have a high index of suspicion for stroke mimics and ensure that radiologic brain imaging is done in all suspected stroke patients in order to ensure appropriate diagnosis and treatment as in this index case.

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INTRODUCTION

Stroke mimic is the term employed for manifestations of nonvascular disease processes when a stroke-like clinical picture is produced. The presentation resembles or may even be indistinguishable from an ischaemic stroke syndrome [1].The mimics include both processes occurring within the CNS and systemic events. Distinguishing these non cerebrovascular stroke mimics from strokes is increasingly important in this era of interventional stroke therapies with potential adverse effects. Stroke mimics may be discovered at different points in clinical investigation; obviously a listing of alternative diagnoses or mimics based on the initial working diagnosis of stroke after history and physical examination only will differ from final diagnoses when the mimic is discovered after extensive neuroimaging and laboratory work. That many clinical conditions could simulate stroke has been known for years, but studies

done with the advent of neuroimaging perhaps allowed the first estimate of the frequency of stroke mimics. One early study of patients admitted with an initial diagnosis of cerebrovascular disease found that 30% had unsuspected intracranial lesions; in all fairness, this included patients with slower progression of neurological impairment ("generalized cerebrovascular disease" was the terminology) as well as more acute presentations[2]. A prospective study [3] published 20 years ago reviewed over 800 consecutive patients admitted to a stroke unit from the emergency department of a Canadian hospital. They found the initial diagnosis of stroke incorrect in 13% of patients. The most common misdiagnosis resulted from unwitnessed or unrecognized seizures with the postictal state being misdiagnosed as stroke in 5% of the study group. Most of these patients had postictal confusion or stupor but transient focal neurological signs were observed in about half of the patients including hemiparesis (Todd's paralysis), monoparesis, abnormalities of extraocular movements, or hemisensory deficits. Confounding the issue of seizure and postictal state was the fact that almost one-third of the patients in the study with seizures had experienced a previous stroke which made the clinical exclusion of a new stroke difficult at initial evaluation. One patient with presumed postictal confusion and mild hemiparesis was discovered to have non-convulsive 3 cycle/sec spike and wave activity; the confusion and hemiparesis rapidly resolved after intravenous diazepam administration. They also found a small number of patients in their study group admitted with a diagnosis of acute stroke that were eventually shown to have CNS tumors; 1% of the group was discovered to have a neoplasm with primary CNS and metastatic tumors represented in roughly equal numbers. Hemiparesis was of more gradual onset in all of the tumor patients; one-third of the tumor patients presented with seizures. There was also a small miscellaneous group of patients with a misdiagnosis of stroke that included patients with radial-nerve palsy, vertigo, encephalitis, hepatic encephalopathy, and other medical conditions including cardiac failure. One patient with abrupt hemiparesis was a young woman that later developed a relapsing and remitting pattern of weakness typical of multiple sclerosis.

In a more recent study in our environment, Onwuekwe et al [4] examined the brain scan of a total of 2207 patients over the period with 142 (6.4%) being referred with a prior diagnosis of stroke. There were more males 102(71.8%) than females 40(28.2%). The mean age of the patients was 54.4 years. Computerized tomography scan confirmed the clinical diagnosis of stroke in 93 patients (65.5%) while 33 patients (23.2%) had stroke mimics. A negative scan was found in 16 patients (11.3%). The clinical accuracy for stroke diagnosis was thus 65.5% while diagnostic errors were made in 34.5% of the patients especially those at extremes of age. Cerebral toxoplasmosis was not found in either of the studies which warranted the case report.

With the current scourge of HIV/AIDS and the battle against other non-communicable diseases; it is expected that neurological presentations of stroke and stroke mimics from HIV/AIDS related CNS diseases and NCDs like hypertension and diabetes will be on the increase in Nigeria. There will also be more patients who will have stroke risk factors like hypertension and diabetes coexisting with undiagnosed HIV which will increase the probability of stroke misdiagnosis. This case report intends to increase the awareness of practitioners to the above clinical scenario with a view of increasing the index of suspicion and ensuring correct diagnosis and management of cases.

CASE REPORT

IA a 52 year old married male Christian with tertiary level of education presented with sudden onset inability to move the left side of the body 16days prior to presentation at our centre. The patient was a known hypertensive of 2 years duration who claimed adherence to medication. He developed weakness which

was said to have started on the left leg and progressively worsened to involve the left arm on the same day while waking up from sleep. There was associated deviation of the mouth to the right side and slurred speech. He had headache which he could not localize but described it as throbbing and not worse at any time of the day. There was no history suggestive of raised intracranial pressure or a meningitic process. He was not diabetic and denied history of multiple sexual partners. He had no previous history of blood transfusions, jaundice or indiscriminate use of injectables. He also did not have TIA's or stroke in the past. The patient had no cardiac or urinary symptoms at the time he presented.

He was then taken to a peripheral clinic where he was given some medications and intravenous drugs before he was subsequently referred to the university of Port Harcourt teaching hospital (UPTH).He had no family history of Hypertension, DM, asthma or Sickle cell disease. Patient's lifestyle was sedentary and he consumed moderate salt. However, he took alcohol occasionally but did not use tobacco products in any form.

Examination at the time of presentation revealed a middle aged man, Conscious but drowsy with a left sided facial nerve palsy of the UMN type, tone was normal on the right side but reduced on the left. Power was grade 3/5 on the left upper and lower limbs and grade 4/5on the right. The deep tendon reflexes were brisk globally. Plantar response was flexor bilaterally. Blood Pressure 160/100 mmHg. Due to sudden onset of the symptoms, a clinical impression motor neurological deficits due to a cerebrovascular accident was made and an urgent CT brain was requested.

Brain CT which was done a week after the event showed (figure 1 – 4). Inhomogenously enhancing isodense areas in the right parietal lobe with effacement of the right cerebral sulci, gyri, lateral ventricles and fissures; note significant hypodense area surrounding the lesion and within the ipsilateral cerebral white matter.The interhemispheric fissure is bowed at the level of the lesion. The right lateral ventricle is effaced while the left is mildly dilated. A diagnosis of cerebral toxopasmosis was made.

Patient had pyrimethamine and clindamycin and was placed on HAART. He commenced physiotherapy and discharged home with remarkable improvement as power was grade 4 on the left side on discharge he was followed up at the outpatient clinic and has continued to make sustained improvement.

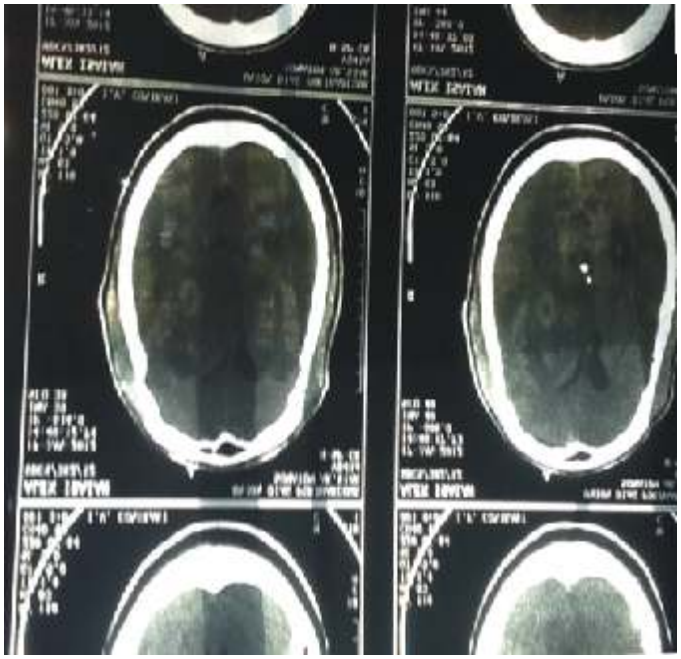
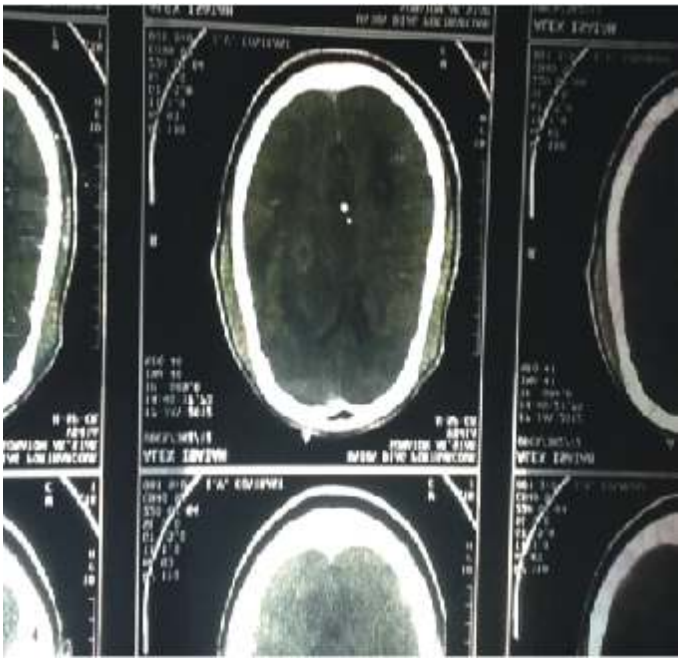


Figure 1



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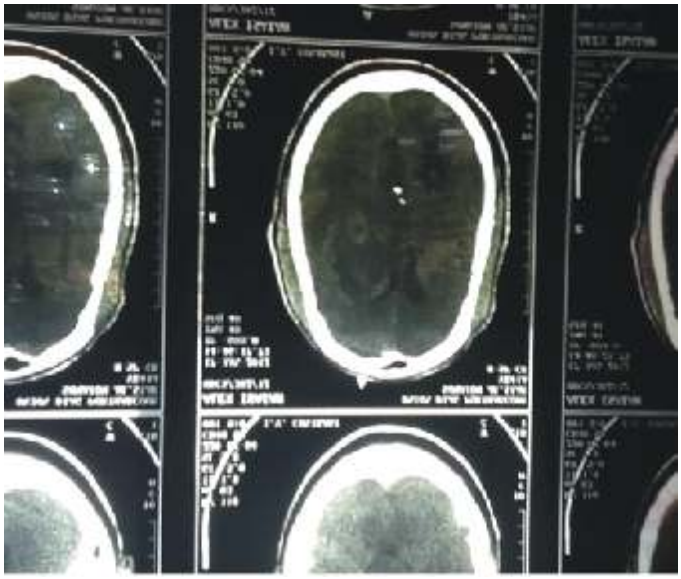

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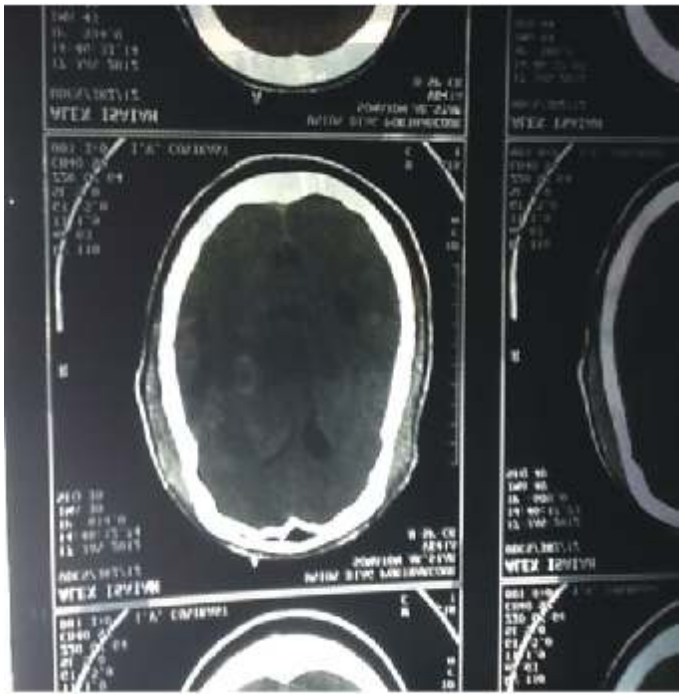

Figure 4

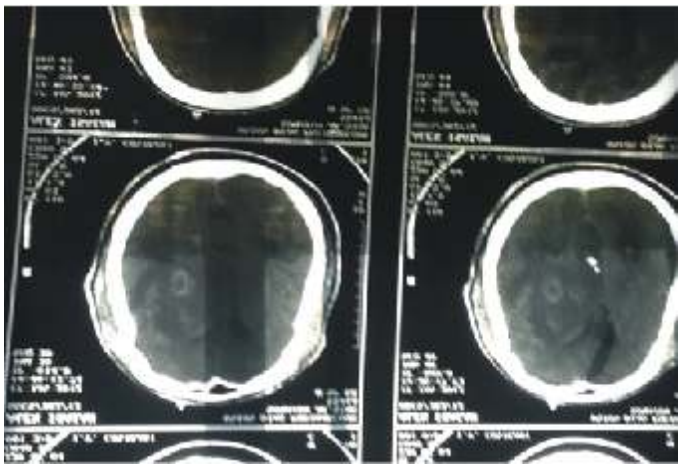

Figure 5

DISCUSSION

Cerebral toxoplasmosis is a stroke mimic. It is one of the most common central nervous system opportunistic infections in HIV-infected individuals and also the most common cause of neurological deficits in patients with acquired immunodeficiency syndrome (AIDS)[5].

Toxoplasmosis is a zoonotic parasitic infection caused by T gondii, an intracellular protozoan estimated to have infected about a third of the world's population. It is transmitted to humans primarily by ingestion of cysts in undercooked pork or lamb or contaminated vegetables or through direct contact with cat faeces. Immunocompetent persons with an acute infection usually develop asymptomatic lifelong latent infection [5]. However, in a setting of immunosuppression, as in human immunodeficiency virus (HIV) infection, there is an increased risk of reactivation of latent infection in various organs, especially the brain. In this setting, toxoplasmosis manifests as a stroke mimic.

Stroke and HIV Infection are both common medical problems in the day to day clinical practice [5]. Even though data from developed countries confirm HIV infection as a risk for stroke the exact mechanism is still not clear. Little data exist on the magnitude of HIV among patients with stroke in Nigeria with most reports being hospital based as shown by Onwuchekwaet al[6] who reported a HIV prevalence of 7.4% among 611 patients with stroke.

Our patient had a clinical event which was consistent with a right hemispheric ischaemic stroke but had CT lesions consistent with cerebral toxoplasmosis. He had risk factors for development of stroke (age, hypertension, sedentary lifestyle) and was seropositive for HIV.

The diagnosis of toxoplasmosis as a stroke mimic can be challenging because clinical presentation of patients with such stroke mimics can confound emergency care

physicians and lead to significant morbidity and mortality for the patient, if unrecognized [7].It is important to note that not all patients presenting as 'stroke patients' have cerebrovascular disease, as some conditions mimic stroke clinically. It is therefore important to reach the correct diagnosis early so as to offer an early medical treatment and better prognosis, especially in theelderly [7]. In order to achieve this, a protocol[8] of proper neurological examination and investigations, of most importance CT scan of the brain (without use of contrast material) should be immediately performed to avoid the significant degree of misdiagnosis of stroke which has been documented outside and even in our own environment though not on cerebral toxoplasmosis.

There seems to be paucity of information regarding infective causes such as toxoplasmosis being stroke mimics. Onwuekweet al [4] in their study of CT images of stroke patients in Nigeria found intracranial neoplasm, subdural haematoma and hydrocephalus as stroke mimics. There was no mention of infective causes which is the reason for this report.

In Singapore, there was a case report of methotrexate induced leucoencephalopathymimicking stroke [9].The patient had clinical and radiologic evidence of stroke which made the case interesting. However the patient recovered spontaneously on discontinuation of methotrexate. She had no radiological evidence of stroke.

Our patient recovered on treatment with pyrimethamine and clindamycin and was commenced on HAART.

CONCLUSION

Despite a high level of accuracy of clinician assessment for stroke, this case report shows misdiagnosis indicating that emphasis needs to be laid on Neuroimagingin stroke evaluation and diagnosis in order to avoid a stroke misdiagnosis.

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