

Neurocysticercosis presenting as a 'Stroke Mimic'

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Abstract

A 62 year old Nepalese gentleman presented with left sided weakness and sensory loss. Initial brain CT scanning was suggestive of acute infarction but a subsequent MRI scan showed cysts with oedema. Cysticercosis serology was positive and a diagnosis of neurocysticercosis was made. The patient made almost a complete recovery after treatment with albendazole, praziquantel and steroids. Neurocysticercosis should be considered in the differential diagnosis when patients originating from endemic areas present with focal neurological deficit.

Key Point

- Consider Neurocysticercosis in the differential diagnosis for patients originating from endemic areas, presenting with acute focal neurological deficit

Introduction

A diagnosis of stroke is usually suspected when there is a sudden onset of neurological deficit. The majority of strokes are ischaemic events but intracranial haemorrhage will cause a similar presentation and is an important differential. Presentations of non-vascular pathology with acute neurological signs, giving the initial appearance of stroke, can be termed 'stroke mimics'. The initial management of an ischaemic stroke is time-dependant in the hyperacute setting; intravenous thrombolysis is licenced up to 4.5 hours post onset (with the potential to expand this time window using advanced imaging techniques). Emergency, Acute and Stroke physicians are expected to arrange investigations, including imaging, on an urgent basis for these patients and decide if thrombolysis is clinically indicated. Exclusion of 'stroke mimics' is therefore important.

Case Report

A 62 year old gentleman presented with weakness and numbness in his left leg for 16 hours. The onset was sudden and then symptoms plateaued. On examination there was reduced sensation to sharp and to fine touch distal to his knee and reduced power (MRC 2/5) in the leg. The plantar response was up-going and the patient had a hemiplegic gait.

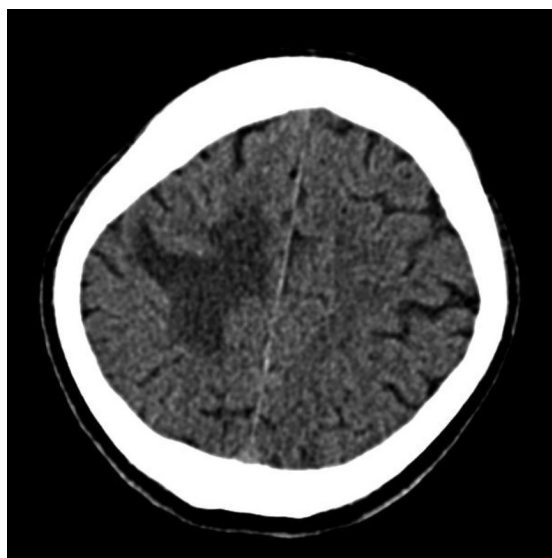


Figure 1. CT scan. An extensive area of low attenuation change high in the right frontal and parietal lobes close to the vertex with some sparing of the overlying cortex. Appearances were attributed to acute ischemic change in the right frontal and parietal lobe.

Funduscopy was normal. Neurological examination was otherwise unremarkable.

The patient had previously been fit and well with no past medical history of note, no regular

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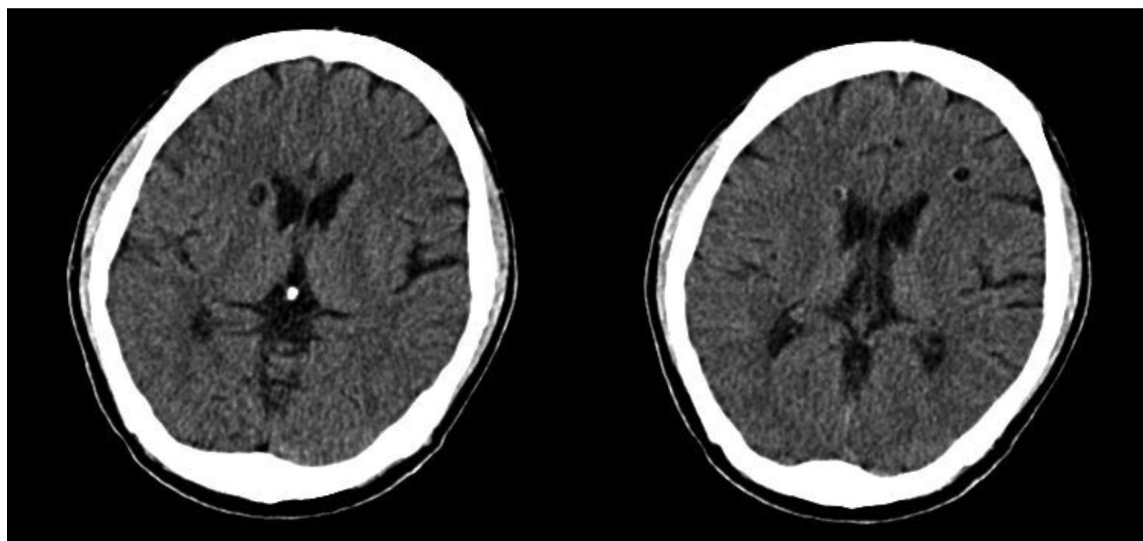


Figure 2. CT scan. Discrete lesions originally reported to represent mature lacunar infarcts, in the right thalamus and left frontal lobe. The appearance of these lesions was thought to be atypical for infarct and prompted further imaging.

medications and no preceding symptoms. The only risk factor for cerebrovascular disease was a history of smoking 2 cigarettes daily for 30 years. There was no history of prior seizures, ischaemic heart disease, peripheral vascular disease, diabetes mellitus, atrial fibrillation or personal or family history of stroke. There were some communication challenges as the patient had limited spoken English. He was native to Nepal and had worked most of his life as a Ghurkha, travelling worldwide. He had come to the UK two years previously.

The initial CT head scan showed an extensive area of low attenuation in the right frontal and parietal lobes, consistent with acute ischaemia (Figure 1). The radiologist reported: “a couple of well-defined, mature lacunar infarcts” in the left frontal lobe and right thalamic head (Figure 2). The

images were reviewed by the Stroke Consultant and felt to be atypical. Following further discussion with a Consultant Radiologist experienced in Neurological imaging, cerebral MRI was requested to further characterise the lesions. The MRI scan revealed discrete lesions with very low density centres, which were reported to be more characteristic of cystic lesions (Figure 3). One lesion in the medial, right parietal lobe had surrounding oedema. There was no oedema around lesions in the left frontal lobe and the head of the right caudate nucleus. There was some ring enhancement of a lesion in the low right parietal lobe. The radiological report postulated that the appearances were consistent with neurocysticercosis.

The patient had not received any initial treatment for ischaemic stroke as the history placed him

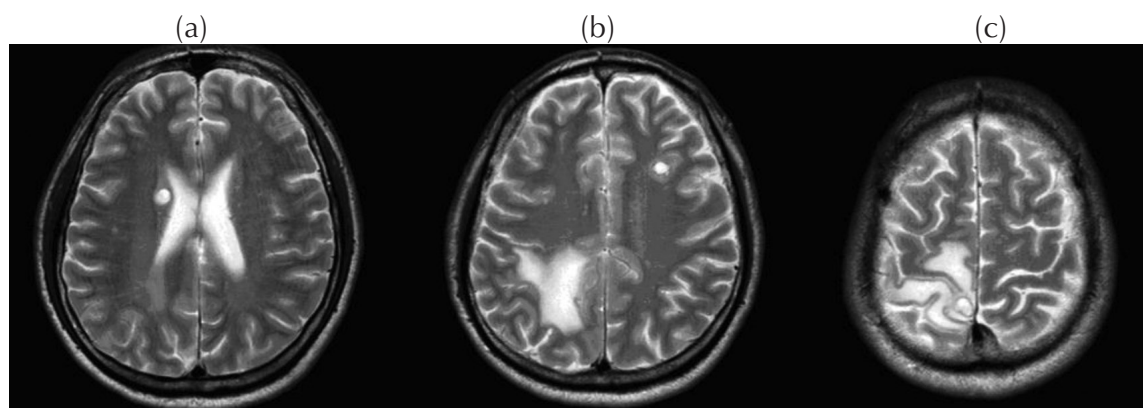


Figure 3. MRI scan, Single Shot TSE images. There is a small cystic lesion in the head of the right caudate nucleus (3a) and a similar small cystic lesion in the left frontal lobe (3b). There is a small enhancing area in the left frontal lobe and a further small lesion showing some ring enhancement in the low right parietal lobe. The largest lesion however, which does also show enhancement, is situated medially in the right parietal lobe with a moderate amount of surrounding cerebral oedema (3c). The lesions have low density centres and appearances were suggestive of neurocysticercosis.

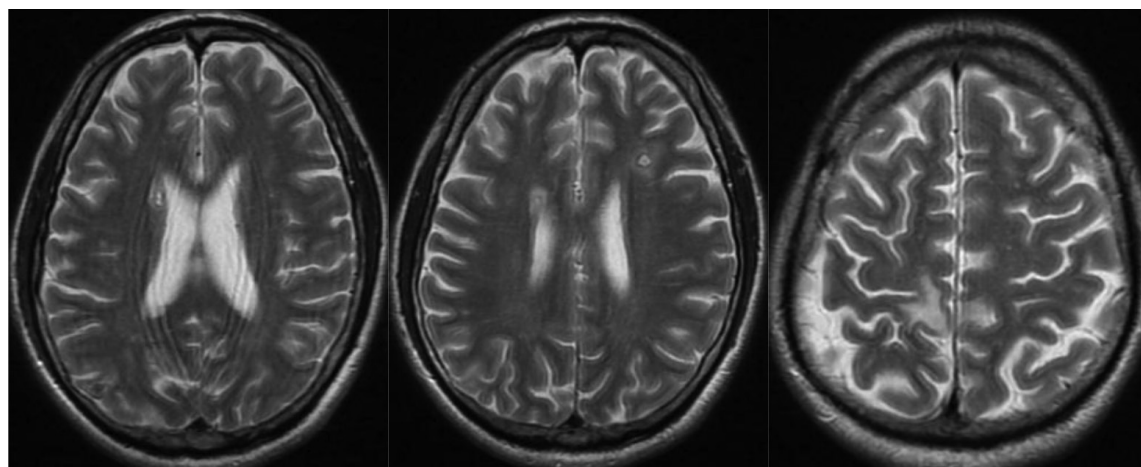
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Figure 4. MRI scan, T2 images. These images show considerable improvement in changes seen in Figure 3.

outside of the licensed time-frame for intravenous thrombolysis.

HIV testing, syphilis serology and Quantiferon TB Gold IT testing were negative. Inflammatory markers, full blood count, renal function, liver function tests and cholesterol were within the normal range. Serum enzyme-linked immunoelectrotransfer blot for the detection of anti-cysticercal antibodies was positive.

Anti-parasitic treatment was started, comprising albendazole 400 mg twice a day plus praziquantel 1000 mg three times a day, along with prednisolone 40mg daily in view of his cerebral oedema. This treatment was continued for 2 weeks. The patient was commenced on the anti-epileptic levetiracetam 250 mg twice a day as a prophylactic measure, as killing live cysts invokes an inflammatory response

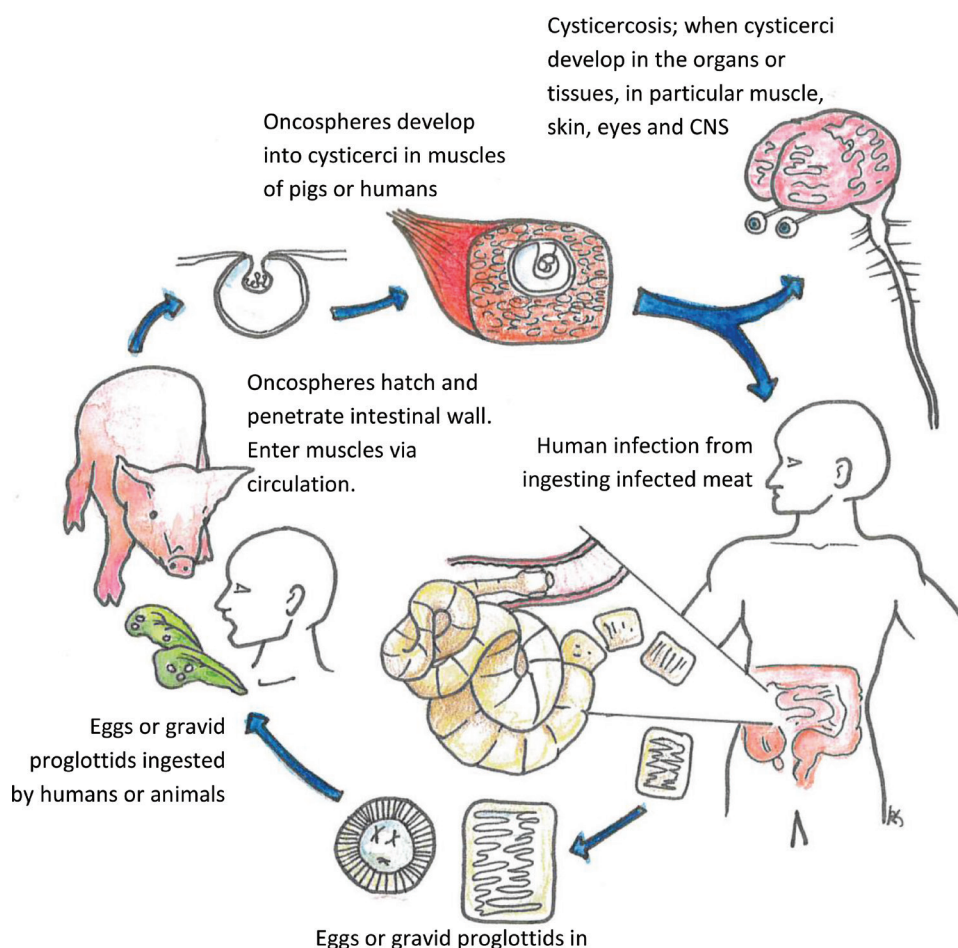


Figure 5. Life cycle of *Taenia solium*.

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Table 1. Diagnostic criteria for neurocysticercosis.¹³

Absolute	Histological demonstration of the parasite from a biopsy of a brain or spinal cord lesion Evidence of cystic lesions showing the scolex on the neuroimaging studies Direct visualisation of subretinal parasites by fundoscopic examination
Major	Evidence of lesions highly suggestive of neurocysticercosis on neuroimaging studies Positive serum immunoblot for the detection of anticysticercal antibodies Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel Spontaneous resolution of small single enhancing lesion
Minor	Evidence of lesions compatible with neurocysticercosis on neuroimaging studies Presence of clinical manifestations suggestive of neurocysticercosis Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens Evidence of cysticercosis outside the central nervous system
Epidemiological	Individuals coming from or living in an area where cysticercosis is endemic History of travel to disease-endemic areas Evidence of household contact with <i>T. Solium</i> infection

that may trigger seizures. His weakness gradually improved.

An MRI scan 2 months after completion of treatment showed significant reduction of cerebral oedema associated with the medial, right parietal lobe lesion and a small reduction in the size of the cyst (Figure 4). The other lesions remained unchanged. By then his motor power had returned to normal with residual sensory loss confined to the sole of his foot. The patient subsequently developed focal seizures affecting the left leg, which were controlled following an increased dose of levetiracetam.

Discussion

The cestode *Taenia solium* (*T. solium*) has a life-cycle involving humans as the definitive host for adult tapeworms (Figure 5). Eggs and gravid proglottids shed in faeces are ingested, and pigs act as the intermediate host. Oncospheres penetrate the intestinal mucosa and settle in tissues to form cysts. These are ingested by humans eating pork. Infection may also arise from eating unwashed vegetable matter contaminated with the sticky proglottids. The presence of tissue cysts in pigs or humans is referred to as cysticercosis. The presence of cysts within the central nervous system is referred to as neurocysticercosis.

NCC is the most common parasitic infection of the central nervous system and also is the most common cause of epilepsy worldwide.¹ NCC can present in a multitude of ways, from Brown-Sequard syndrome² to acute hydrocephalus due to intraventricular NCC,^{3,4} although epilepsy is by far the most common presentation globally. *T. solium* infection is common throughout Asia, Sub-Saharan Africa and Latin America but NCC is rare in the UK, with all cases being acquired outside the country. When it does present in the UK, epilepsy is again the most common manifestation.⁵ NCC itself can cause cerebrovascular events,⁶ but it rarely presents with sudden onset focal neurological deficit as the only sign.^{7–12}

Currently definitive diagnosis of neurocysticercosis is made using a set of objective

diagnostic criteria (Table 1).¹³ These include Absolute, Major, Minor and Epidemiological criteria; a definitive diagnosis is made when one absolute criterion or two major, one minor with one epidemiological criteria. This case fulfils three major criteria (highly suggestive imaging, positive serum antibody testing and radiological response to treatment), one minor (clinical manifestation) and an epidemiological criterion (native to endemic area and travel to endemic area) and so a definitive diagnosis can be made.

Cerebrovascular events are described with NCC¹⁴ and there are thought to be two mechanisms. Firstly leptomeningeal inflammation, caused by antigen release by cysts, leads to local endarteritis and infarction.⁶ Secondly haemorrhage may occur adjacent to cysts, and this may or may not arise from pre-existing aneurysms.¹⁶ It is important to be mindful that even if there is a diagnosis of NCC there may still be superimposed infarction or haemorrhage, which may happen spontaneously or be precipitated by treatment.¹⁵ Since infarction in NCC is usually inflammatory in origin and not thrombo-occlusive, such cases would be unlikely to gain benefit from thrombolytic treatment, and would carry the additional risk of haemorrhage.

NCC can also present with focal neurological manifestations without infarction or haemorrhage.¹⁷ The cysts themselves may cause mass effect giving rise to signs relating to their position, but more widespread clinical features follow spreading tissue oedema as part of the inflammatory response to a leaking, degenerating cyst.

Treatment of NCC usually involves the use of anti-parasitics, anti-convulsants and steroids. The use of anti-parasitic drugs to kill cysticerci (the larval stage of the worm in the form of a cyst) has been controversial. Cysticerci should eventually die without treatment. If cysticerci are already dead and calcified, anti-parasitic treatment is unnecessary. If cysticerci are alive but asymptomatic treatment may lead to injury, inflammation and symptoms.

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A meta-analysis of randomised controlled trials,¹⁸ and a further randomised control trial since then,¹⁹ supports the use of cysticidal therapy in cases of enhancing lesions and showed an association with complete resolution of cystic lesions. The meta-analysis used seizure activity as an outcome, with a reduction in seizures after cysticidal treatment. The majority of patients present with seizures and so are also treated with anti-convulsant medication. Our patient had not had an identifiable seizure at presentation and was given prophylactic anti-convulsants. One case report details using prophylactic anti-convulsant medication in a case of NCC presenting only with focal neurological signs.¹⁰ All cases are treated with corticosteroids to reduce inflammation and cerebral oedema.

In this case the presentation initially appeared consistent with an ischaemic stroke. Had the patient presented within 4.5 hours of onset of symptoms it is a possibility that he may have received thrombolytic treatment with potential adverse outcomes. There are several important radiological nuances to this case that resulted in the correct diagnosis and treatment. Although the CT images had the appearances of infarct, they were not typical of acute infarct. The hypodense areas were more in keeping

with established infarct which did not correlate with the timing of the symptoms. The ‘infarcted’ area only extended up to the cortex and did not involve it, which is not characteristic of thrombo-occlusive ischaemia. Cerebral oedema is also more likely to cross arterial territories and may not demonstrate the predictable pattern of ischaemic stroke.

This patient had been in the UK for two years and would not have acquired the infection in the UK. Many patients develop a high cyst burden before developing symptoms and so infection may be present asymptotically for some time prior to presentation. Migration from endemic to non-endemic areas is increasing and any history of living in an endemic area fulfils an epidemiological criterion for NCC diagnosis. NCC remains a rare stroke mimic in the UK but should be considered amongst migrant patients. Case series from endemic areas have demonstrated that NCC can be a cause of stroke as well as a stroke mimic.²⁰ A thorough travel history along with a careful evaluation of radiological clues will enable clinicians to initiate appropriate investigation and management.

Conflict of interest

Nothing to declare.

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