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Neurocysticercosis and Psycho-Social Trauma

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Authors' contributions

This work was carried out in collaboration between all authors. Author MGV designed the study. Author LJM reviewed the psycho-social history and edited the paper. All authors treated the patient in the hospital and author four and five reviewed the literature on neurocysticercosis and organized neuroimaging results. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

Aims: This paper describes a patient with neurocysticercosis who presented with psychotic features that related to his experiences of civil unrest in Rwanda.
Study Design: A review of the literature on neurocysticercosis is described and the history of genocide in Rwanda in 1994 is summarised. The case of an African refugee to Australia is presented.
Place and Duration of Study: Cairns Base Hospital between 13 June 2011 until 21 July 2011 and then follow up in the community until the end of October 2011.
Methodology: The patient was admitted through the Emergency Department where he was assessed and treated in a medical ward with the involvement of the Psychiatric Liaison Team and Infectious Disease Team. CT and MRI of the brain confirmed the diagnosis of neurocysticercosis. Medical treatment was administered for neurocysticercosis and a subsequent epileptic seizure. Psychiatric treatment and community psychiatric and medical follow-up were undertaken.
Results: Medical treatment of neurocysticercosis was successful but the patient subsequently developed a seizure disorder that was treated effectively. The patient's psychotic disorder was treated with medication and supportive psychotherapy. Unresolved grief related to psycho-social trauma was addressed on an on-going basis during his community treatment.
Conclusions: Neurocysticercosis is disorder that should be considered in immigrants from

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countries that have endemic neurocysticercosis. A small minority of patients present with psychosis (14%). This patient presented with psychosis that reflected his psycho-social trauma.

Keywords: Neurocysticercosis; psychosis; seizures; trauma.

1. INTRODUCTION

Neurocysticercosis (NCYST) is the most common parasitic infection of the brain caused by infestation with Taenia Solium, the pork tapeworm [1]. It is endemic in Central and South America, Asia (particularly India) and Africa [2]. An estimated 50 million people have NCYST [3] which is the leading cause of acquired epilepsy in the world [4]. NCYST is rare in Australia and New Zealand. In 1994 in New South Wales four cases of NCYST were identified over a ten year period and all four patients were immigrants [5]. In the United States the incidence of NCYST is the highest among men (58.4%) and most frequent between the ages of 16 and 60 years (86.9%) with a mean age of 34.5 years [6]. NCYST caused an estimated 50,000 deaths a year in the USA in 2000 [7].

There has been an increasing prevalence of NCYST in recent years in Australia. There are 39 published cases of NCYST and of these 33 patients were reported in the last two decades. The rise in NCYST in Australia is related both to more Australians travelling overseas and more immigrants developing the disease after migrating to Australia [8].

Patients with NCYST are usually asymptomatic for many years and then they present with non-specific neurological symptoms including headaches, nausea, vomiting, ataxia, confusion, meningism and seizures. Seizures are the most common manifestation and occur in 75% of patients. It may take 30 years after the initial infection for seizures to develop [2,9,13]. Hydrocephalus and increased intracranial pressure, seen in approximately 25% of cases, may cause nausea, vomiting and papiloedema [2,9,10]. Psychiatric manifestations of NCYST are found in 65% of outpatients with 52% being depressed and 14% developing psychosis [11].

The mortality rate is as high as 2.2% [6] to 4% [1,13]. Factors contributing to death include hydrocephalus, cerebral oedema, cerebral compression and seizures [12]. There are a few case reports of sudden death attributed to NCYST [13]. Possible explanations for sudden death include cardiac dysfunction from seizure-related arrhythmia/asystole or fatal anoxia due to acute pulmonary oedema. Complications of NCYST include cognitive decline with lifelong disability [14].

The life cycle of the pork tapeworm involves (Fig. 1) humans as definite hosts and pigs as intermediate hosts. Pigs ingest contaminated food or water that contains eggs from human faeces. Ova develop into cysticerci in pig muscles. Human become infected by eating raw/undercooked pork. The solex attaches to the intestinal wall where a tapeworm grows that releases 300,000-700,000 eggs/day into the intestine. These eggs are spread by human faeces into the environment and the cycle continues when pigs ingest the eggs. When the eggs are ingested by humans, they develop into larvae, penetrate the intestinal wall, and disseminate through the vascular system mainly to the brain, eye, heart and muscles. Cysticerci develop in the central nervous system in 60% of cases. [15].

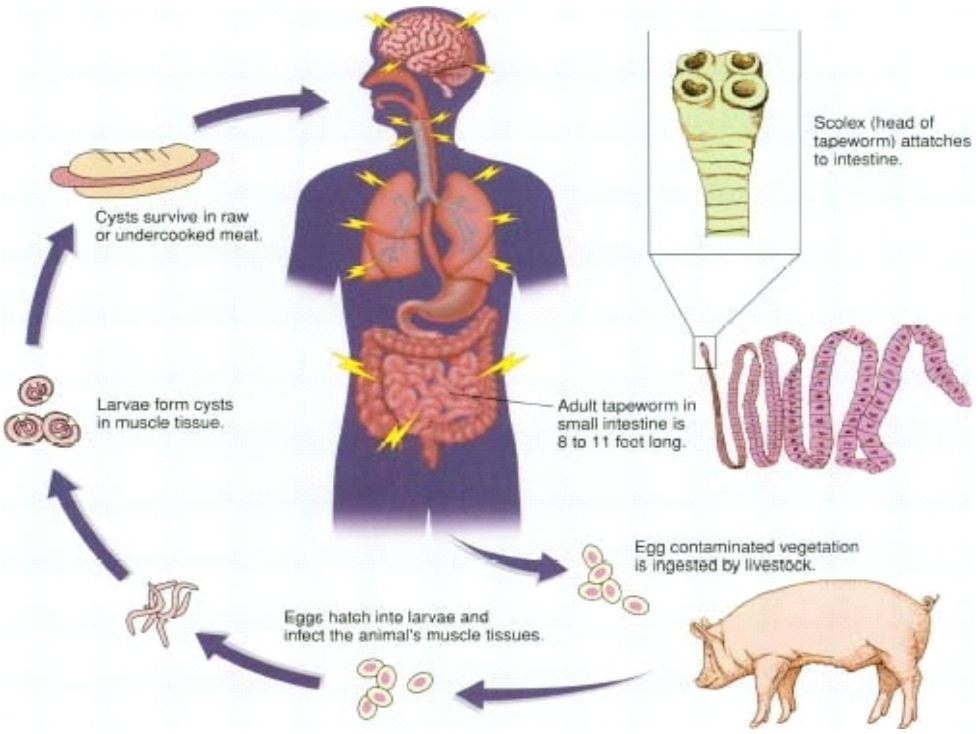

Fig. 1. Life cycle of the pork tapeworm [15]

NCYST is diagnosed by taking a proper history including immigration or travel from endemic areas, food consumption and employment history (meat handlers). Clinical examination needs to include a neurological assessment.

Neuroimaging includes Computed Tomography (CT) of the brain and Magnetic Resonance Imaging (MRI) of the brain. CT is considered a better modality for detecting calcification, while MRI is more sensitive in indentifying intraventricular and subarachnoid disease [10, 13]. Radiological studies of NCYST show cyst structures, enhancing lesions, peri-lesional oedema, parenchymal calcifications and hydrocephalus.

Treatment of NCYST consists of antiepileptic drugs, corticosteroids, and selected surgical procedures aimed at controlling seizures, inflammatory responses and intracranial hypertension. Anti-parasitic medications are generally effective; however, there are rare treatment related deaths (1-4%) in cases with hydrocephalus, intracranial hypertension, and massive parasitic infestations [1,13]. The placement of a ventriculoperitoneal shunt is indicated in cases of intracranial hypertension before the use of antiparasitic drugs. Surgical removal maybe indicated for large or intraventricular cysts [1,10,13].

The prognosis of NCYST is multifactorial and ultimately depends on the host immune response, disease duration and parasite location, load, size and stage [2,13]. Intraventricular and basal subarachnoid NCYST have a worse prognosis due to the development of hydrocephalus [10,13].

2. PRESENTATION OF CASE

AG is a 35 year old Rwandan refugee who is single, lives with a Rwandan friend, works part time as a landscaper and has partial government benefits. He came to Australia one year before he became unwell.

AG was brought into the emergency department by the police on an Emergency Examination Order following concerns from his flatmate about AG's odd behaviour. He was agitated and refused to cooperate. Collateral information was obtained from his flatmate who had known AG for nine months. His flatmate said AG appeared confused for the last two days and although he seemed well that morning, in the afternoon he began screaming and was not making sense. AG did not have any psychiatric, substance abuse or forensic history.

On initial mental state examination AG appeared suspicious, guarded, angry and agitated. His mood was dysphoric with reactive affect. He had persecutory delusions regarding his flatmate. He thought that his flatmate was planning to kill him because his flatmate belonged to the Tutsi tribe in Rwanda. He was oriented to place and person but not to time. He was not cooperative with further cognitive testing. There were no other significant findings except for poor insight and judgment and anger about being in the hospital.

His initial differential diagnosis included First Episode Psychosis, Psychotic Disorder Due to General Medical Condition including malaria, syphilis, HIV or other infectious causes and Delirium.

AG became very agitated after his friend left and required intramuscular Droperidol and Midazolam. He was detained under duty of care. Subsequent CT scan showed multiple cysts in the brain and he was admitted under the medical team with the involvement of the psychiatric consultation liaison team. A more comprehensive history was obtained with the help of an interpreter.

AG reported a two week history of psychotic symptoms. He heard his flatmate's voice saying that he was to blame for the Rwandan genocide and his flatmate's sister was sending messages through the television saying he was responsible for the genocide. He thought his flatmate's sister and another friend had been sleeping on the ward because he heard them making derogatory comments about him to the nurses. He believed the Rwandan government was planning to kill him because he had been working as a spy. He thought all his money had been stolen from his bank and he wanted to change his account numbers so that, "the Rwandan government wouldn't have access to it". He denied suicidal and homicidal ideation as well as symptoms of mood disorder.

He had been hospitalized in Tanzanian for two days for treatment of malaria.
His mother was an alcoholic and there was no family history of suicide or mental illness.

AG was the first born of eight children with two step-brothers and five step-sisters. His mother was Tutsi and his father was Hutu. His parents divorced immediately after his birth. His mother remarried to a Tutsi man who beat AG regularly, made him work hard, and constantly humiliated him. AG was close to his biological father until he was seven years old when his father was beaten to death for being Hutu. AG said he loved his mother, but that she never wanted him around. His mother was verbally abusive when drunk.

His step-father paid for the schooling of the other seven children, but not for AG. AG made money by carrying well water to peoples' homes. He left home at the age of 15, because he found it intolerable. When the genocide in Rwanda escalated his two close friends hid him in Zaire. After the genocide AG returned to find his country ravaged. He managed to start a barber shop and then a photography business. He saved enough money to buy land and build a small house.

During this time, he struggled because the people around him bullied him. He tried to re-connect with his family, but they would never visit him. He was forced to sell his business and house because he received death threats and believed he was being bewitched. He moved first to neighbouring Burundi, then to Tanzania where he spent three years in refugee camps and then finally to Australia in 2010.

Mental state examination after beginning treatment for NCYST showed persisting persecutory delusions as well as auditory hallucinations. His Mini Mental State Examination was 29/30. He was able to understand that he had cysts in his brain but did not understand they were causing his psychotic symptoms.

All medical screening investigations were negative.

CT scan pre and post contrast showed numerous scattered lesions throughout both cerebellar hemispheres. On the pre-contrast many of them were hyperdense and showed no significant vasogenic oedema and no mass effect. Some of them were cystic with an internal blip of hyperdense material within. These cystic lesions showed either subtle or no surrounding enhancement.

Neurocysticercosis was confirmed by brain MRI (Figs. 2,3,4 non-contrast and 5 with constrast). MRI showed the following lesions: one in the right temporal lobe, two in the posterior left temporal lobe, four in the left frontal lobe with one in the inferior frontal lobe showing surrounding oedema, one in the right cerebellum, one in the left cerebellum, two in the right frontal lobe and one in the left posterior parietal/occipital lobe. The radiologist (SS) said the left inferior frontal lesion with oedema was active and could have been causally related to the psychosis.

2.1 MRI Scan before Treatment

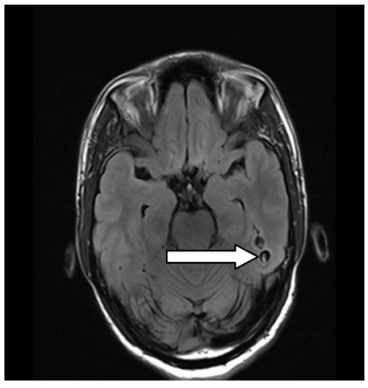

Fig. 2. MRI-FLAIR axial non-contrast

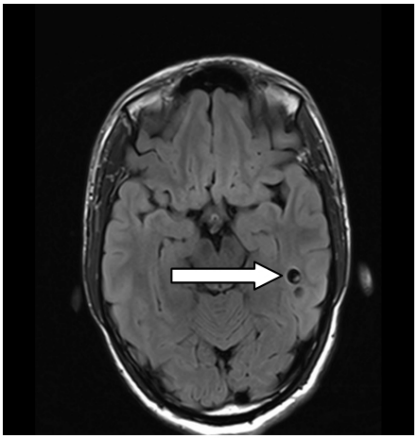

Fig. 3. MRI-FLAIR axial non-contrast

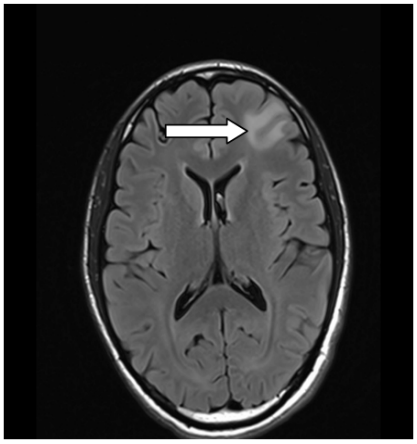

Fig. 4. MRI-FLAIR axial non-contrast showing oedema

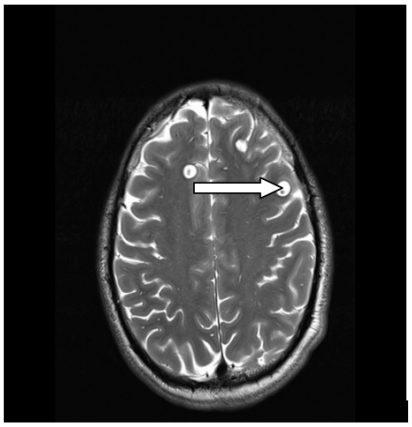

Fig. 5. MRI-T2 weighted with contrast

A CT scan after one month of treatment (Fig. 6) showed a dramatic response with no residual oedema in the brain. The previously well demonstrated cystic lesions were either seen as very tiny residual hypodense tissue or foci of calcification.

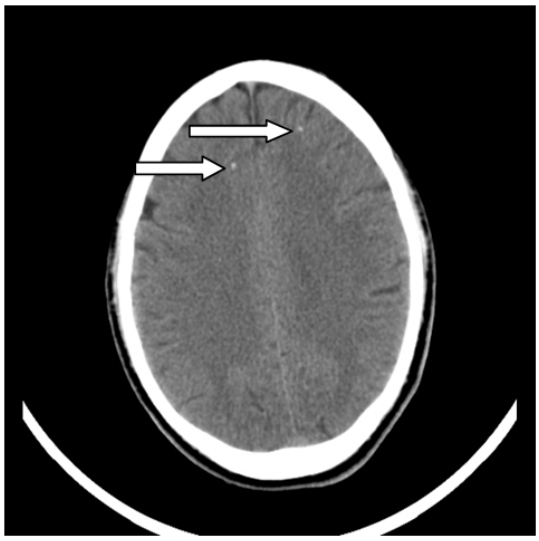

Fig. 6. CT scan 1 month after treatment

AG was placed under an Involuntary Treatment Order (ITO) because of the potential risk of aggression towards his flat mate and his impaired insight and judgment. On a thorough psychiatric assessment the diagnosis of PTSD was confirmed as well as Psychotic Disorder due to General Medical Condition (NCYST) with the differential diagnosis of Psychotic Disorder due to Severe PTSD. He was treated with Haloperidol 2.5 mg nocte, Oxazepam 7.5 mg twice daily and Dexamethasone 4 mg three times/day to reduce cerebral oedema. After four days of steroids AG was given Albendazole 400 mg BD for 7 days. On day 14 of the admission, he was medically stable but he was still psychotic so he was transferred to the mental health unit. Haloperidol was increased to 5mg nocte, his mental state gradually improved and his ITO was revoked. AG was sent on weekend leave with a view to discharge him, however, during the weekend leave, he was brought to the emergency department following a witnessed prolonged seizure. He was re-admitted to a medical team, started on Phenytoin 100 mg BD and discharged a week later with case management by the community mental health service (CMHS).

He was followed by the CMHS for four months. His case manager was a social worker who provided supportive and modified trauma psychotherapy with the assistance of an interpreter for three months to help him deal with the loss, grief and trauma related to his traumatic experience. AG was seen by a psychiatrist with regular reviews of his mental state and also by an infectious disease physician. Haloperidol was gradually reduced and stopped after four weeks and he remained free of psychotic symptoms. His anti-epileptic medication was continued.

3. DISCUSSION

NCYST usually presents with seizures, meningitis and raised intracranial pressure, however, NCYST can masquerade as a glioma, another space occupying lesion, or aseptic meningitis. NCYST should be considered in migrants with seizures and obscure neurological syndromes [5]. Approximately 14% of cases of NCYST present with psychosis [11].

In our case the primary presentation was psychotic symptoms that related to AG's experience of the genocide in Rwanda. Rwanda is a country in central, east Africa inhabited by 85% Hutu and 14% Tutsi tribes people. The imposition of the Catholic religion and an apartheid system fuelled an intense racial hatred that erupted into a civil war in 1994 when a million Tutsi people were killed in a matter of 100 days. [16].

The fact that AG presented with psychosis and suffered from NCYST is the most startling discovery in his treatment. His diagnosis was Psychosis NOS due to a General Medical Condition (NYCST). The differential diagnosis includes Psychosis NOS due to Severe PTSD. He suffered from PTSD for most of his life but he did not have psychotic symptoms until he presented with active NCYST. We hypothesise that AG's abrupt onset of psychosis in the context of longstanding PTSD was due to the highly active cyst with prominent oedema in the left inferior frontal lobe. All his psychotic symptoms (persecutory delusions, delusions of reference and auditory hallucinations) were related to his traumatic experiences. There is no way to prove a causal relationship between neurocysticercosis and his psychotic symptoms or their association with his personal and social trauma. All we can say is that neurocysticercosis may have precipitated a psychosis and that a trauma related psychosis needs to be considered in the differential diagnosis. It is precisely because he may have been diagnosed with Psychosis NOS associated with PTSD that his psycho-social history is relevant. From the moment he was born as both Tutsi and Hutu AG suffered persecution from his own family and society. He was forced to hide and then was driven out of his country and other countries where he sought refuge. His belief that he was a spy may have come from his own confusion about his identity as well as living a secret life. His fears that his friends were going to kill him were related to his experiences of persecution in his own country.

4. CONCLUSION

This paper describes the medical and psychiatric problems of a man from Rwanda who brought with him neurocysticercosis and the emotional scars of a terrible genocide that destroyed his country. Despite his extraordinary resilience and efforts to repatriate to several countries soon after his arrival in Australia he suffered an acute psychotic episode and neurocysticercosis was diagnosed on routine CT and MRI. The symptoms of his psychosis revolved around the psycho-social trauma caused by the genocide in Rwanda. The misfortune of suffering from neurocysticercosis caused him to develop a seizure disorder but also brought him into contact with mental health professionals who were able to help him process and integrate his dissociated traumatic experiences. His neurocysticercosis, seizure disorder and psychosis were all effectively treated. His grief, loss and trauma are issues he will continue to work through in his community psychiatric care.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal. Consent from the patient was obtained using the SDI Patient Consent Form 1.0. See attached.

ETHICAL APPROVAL

Ethical approval was not required.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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