Neurocysticercosis: a reversible and rare cause of seizure in the developed world

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DESCRIPTION

A 22-year-old man, originally from India, presented to an emergency department in Canada with a new-onset seizure and collapse associated with a few seconds of right eye and tongue deviation. This event occurred following 6 weeks of difficulty concentrating and unilateral left-sided headache. On examination, no focal neurological deficits were found. A non-contrast CT head was requested which showed a left-sided occipital lesion. A follow-up brain MRI revealed the ‘hole-with-dot’ lesion pathognomonic for neurocysticercosis (NCC).

NCC is caused by the tapeworm *Taenia solium* and is the most common parasitic infection of the central nervous system, endemic to many low-income countries worldwide. *T. solium* is transmitted among humans via the faecal–oral route, with pigs acting as an intermediate host. Humans are a definitive host for the intestinal tapeworm which produces eggs containing an infective embryo (oncosphere). This embryo can actively cross the intestinal wall into the blood stream and deposit in various tissues in the body.

NCC is recognised when *T. solium* embryos deposit into the brain. The first of three stages in NCC consists of the ‘colloidal’ stage where larvae exist as cystic vesicles containing a scolex (figure 1A). These cysts can persist for years before degenerating in the ‘nodular’ stage, which in turn can lead to vasogenic oedema. This explains the epileptogenicity of NCC. It then enters the ‘calcified’ stage.

NCC can be diagnosed solely on neuroimaging with visualisation of a scolex within a cystic lesion. This gives the pathognomonic ‘hole-with-dot’ appearance (figure 1B). Other neuroimaging features suggestive of parenchymal NCC include: well-demarcated and round lesions, non-enhancing with intravenous contrast and less than 20 mm in diameter. Serological testing using the enzyme-linked Immunolectrotransfer blot (EITB) assay can support the diagnosis in addition to clinical and radiological findings.

NCC is the primary cause of preventable epilepsy in low-income and middle-income countries with millions estimated to be infected worldwide. NCC is commonly observed in India with single enhancing nodules being more frequently seen in individuals younger than 30 years of age. Late-onset, generalised, tonic-clonic seizures are the most common presentation of NCC with the onset of seizures typically occurring between the ages of 20–49 years. In the USA, 2% of emergency department visits due to seizure were caused by NCC. Although not endemic to developed countries, there is an increasing number of patients with NCC in developed countries due to increasing rates of international travel and immigration. Therefore, when presented with cases of late-onset seizures, NCC should be taken into consideration.
Images in consideration as it can no longer be considered an ‘exotic’ disease in the developed world.

Our patient was started on phenytoin for control of seizures. He was then placed on steroids and initiated on a 14-day course of albendazole, given the paradoxical increased risk of seizure secondary to the start of treatment. He will have a follow-up MRI scan in 6 months to ensure resolution of lesion, at which point the decision for continuation of phenytoin will be made.

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REFERENCES

Learning points
► The “hole-with-dot” sign seen on neuroimaging is pathognomonic for neurocysticercosis and sufficient to diagnose NCC in the absence of further clinical data.
► Neurocysticercosis should be considered in patients who present to emergency with late-onset seizure as incidence of NCC is increasing in the developed world due to increasing rates of immigration and international travel.