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Intermittent enhancement in chronic nodular calcified neurocysticercosis

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Neurocysticercosis (NCC) is the most common cause of new onset seizures and epilepsy in the developing and tropical world. There has been a marked increase in domestic cases of diseases traditionally associated with developing countries, and now NCC should be high on a radiologist's differential diagnosis list for a patient with seizures. Radiologic findings of NCC correlate with the parasite's life cycle within the host. The calcified granulomas signify the final stage (nodular calcified) as nonenhancing punctate calcifications on imaging and are traditionally known to remain withsout enhancement. Here we describe a unique case of intermittent enhancement of a cortical calcified nodule secondary to NCC that was followed for over 10 years. Radiologists must challenge the traditionally accepted progression of imaging findings and accept that calcified nodules of chronic NCC may intermittently enhance.

Introduction

The list of etiologies for new-onset seizures is relatively extensive. Neurocysticercosis (NCC) is at the top of the differential diagnosis list of new-onset seizures and epilepsy in the developing and tropical world (1). Owing to the diverse nature of new citizens and visitors in the USA, there has been a marked increase in domestic cases of diseases traditionally associated with developing countries. As such, NCC should be a serious consideration on a radiologist's and clinician's diagnostic differential list for a patient with seizures. A byproduct of this increased prevalence is better understanding of the pathological process and imaging characteristics of this disease.

We describe a unique case of intermittent enhancement of a cortical calcified nodule secondary to NCC. Once thought to remain without enhancement after reaching the fourth stage in the life cycle, the interval imaging of the presented patient's calcified nodule offers a challenge to the traditionally accepted progression of imaging findings.

Case report

A 21-year-old Hispanic male (with a past medical history only significant for a congenital solitary kidney) presented to our institution's emergency room (ER) in the early morning with altered mental status. It should be noted that at baseline, the patient was a normally functioning young male with no mental deficits or delays. A family member of the patient reported that at approximately 4:30 am, the patient was "snoring or making strange noises" and was nonresponsive. During the initial ER evaluation, the patient responded to his name and was able to enunciate complete words and several words together but appeared extremely confused and unable to completely follow commands.

A nonenhanced computed tomographic (CT) scan of the brain (Fig 1A) revealed a coarse calcification in the right frontal lobe with associated edema. Multiple other coarse calcifications were noted in the superficial cortical areas as well as deep in the basal ganglia without surrounding edema (not shown). A subsequent brain magnetic resonance imaging (MRI) scan (Figs 1B and 1C) demonstrated T2/FLAIR hyperintensity surrounding a coarse calcification in the superficial right frontal lobe corresponding to the same lesion identified on CT, which revealed ringlike enhancement following contrast administration. EEG did not reveal any global or focal epileptic activity upon initial

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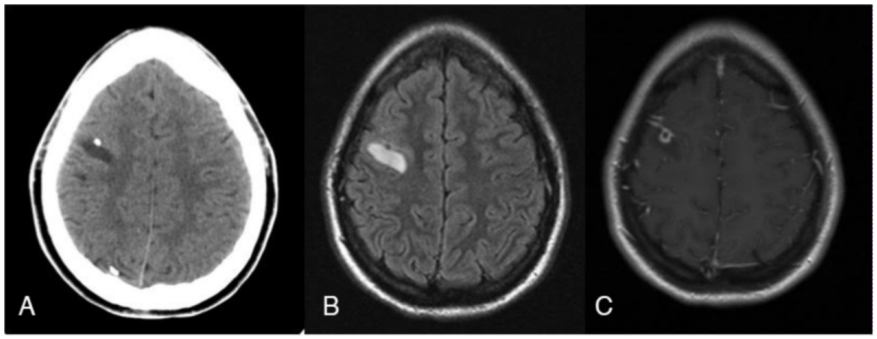

Figure 1. Initial presentation following seizure. Axial brain NECT of the brain (A) with a superficial coarse calcification in the right frontal lobe. FLAIR sequence axial brain MRI (B) shows hyperintensity surrounding the same coarse calcification in the superficial right frontal lobe, which revealed ringlike enhancement following contrast administration (C).

hospital evaluation. Given the clinical history and diagnostic imaging findings, a diagnosis of chronic nodular calcified neurocysticercosis complicated by seizure was made.

The patient was referred to a neurologist and prescribed a daily regimen of carbamazepine. Multiple routine outpatient followup imaging exams assessed the enhancing lesion. The first 4-month followup MRI showed minimal residual perilesional edema, and the enhancement was completely resolved (Fig. 2). The next MRI at the one-year anniversary of the sentinel seizure revealed complete resolution.

Approximately 3 years later, a routine MRI revealed T2/FLAIR hyperintensity surrounding the same coarse calcification in the superficial right frontal lobe as was seen on initial presentation. A followup CT demonstrated stability of the existing calcifications. All were unchanged, and no new calcifications were noted. In addition, CT also demonstrated ringlike enhancement following contrast administration. Three-month and 15-month followup exams showed that the edema and enhancement had resolved. However, the next MRI performed again revealed T2/FLAIR hyperintensity and ring enhancement surrounding the same coarse calcification in the superficial right frontal lobe.

Following another three years and 6 negative MRI exams, the patient was finally taken off of carbamazepine. His neurologist stopped following the enhancing lesion as well. The patient returned to the ER after another 3 years. This was his first seizure in more than 10 years since his initial presentation. CT again revealed a stable right frontal lobe calcification (Fig. 3A), and MRI again demonstrated T2/FLAIR hyperintensity surrounding the same calcification, associated with ringlike enhancement following contrast administration (Fig 3B).

Discussion

Taenia solium is classified as a zoonotic cestode with two hosts. Although humans are the definitive host of the disease after oral ingestion of its eggs, pigs are the intermediate host carrying T. solium in the larval stages everywhere in its body (2). Human ingestion of the eggs occurs via consumption of pork or raw vegetables irrigated by unclean water. The T. solium eggs pass into the gastric cavity and intestines, and the infective oncospheres they contain enter the vascular compartment (and eventually tissues) to mature into maximally sized cysts in about 3 months (2). Upon entering tissue, the cystic lesions can remain viable for years and immunomodulate in order to cloak themselves from detection by the host. Neurocysticercosis refers to the spread of cysts into the tissue of the brain. It is then further classified into parenchymal and extraparenchymal NCC.

Traditionally, the radiographic findings of NCC correlate with the life cycle within the host and are traditionally

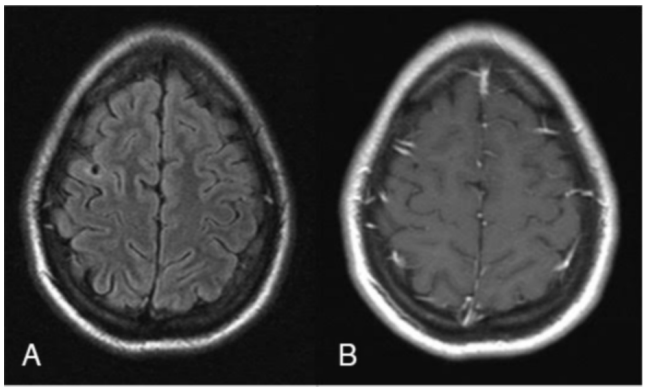

Figure 2. Routine followup exam. FLAIR sequence axial brain MRI (A) 4 months later shows minimal hyperintensity and complete resolution of enhancement on postcontrast T1 sequences (B).

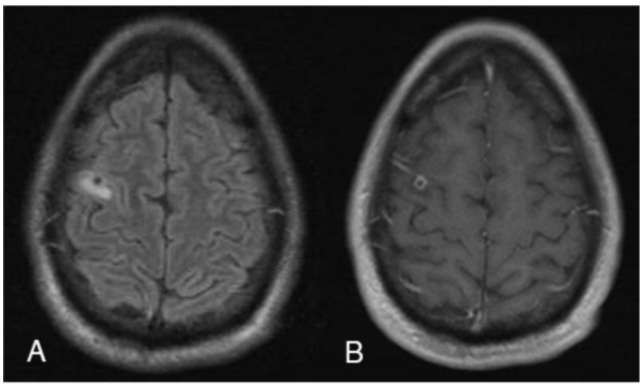

Figure 3. First seizure in 10 years. FLAIR sequence axial brain MRI (A) shows hyperintensity surrounding a stable right frontal lobe calcification, which revealed ringlike enhancement following contrast administration (B).

described first as "vesicular," then "colloid vesicular," then "granular nodular," and finally "nodular calcified" (3, 4). The initial vesicular stage enhances minimally and, although it represents a viable larval cyst, there is little host response to the increased enhancement. On imaging, a peripheral nodule within the cyst represents the larval scolex, and this becomes less distinct as the cyst deteriorates into the colloid vesicular stage.

During the colloidal stage, the larval cyst begins to leak fluid into the surrounding parenchyma and elicits an immunologic response, which enhances strongly on CT and MRI. As the fluid continues to leak and the cyst involutes, it takes on the form of a nodule (granular nodular stage), and continues to enhance strongly until it eventually becomes a calcified granuloma. The calcified granulomas eventually move to their final stage as nonenhancing punctate calcifications, and are classically known to remain without enhancement (3, 5).

Anecdotal reports have surfaced recently that challenge the currently held view about enhancement characteristics. Several case series have described perilesional edema and/or ring enhancement in calcified NCC lesions as well. These reports are partially limited by lack of long-term followup and potential inclusion of incompletely involuted granular nodular cysticerci. The long followup period and negative serology definitively places our patient's lesion in the stage 4 category. Following an exhaustive review of the literature, we believe that this is the first reported case to demonstrate fluctuating ring enhancement in a single calcified granuloma in a patient with chronic NCC followed for over 10 years.

There are competing views as to whether the perilesional edema and/or ring enhancement are associated with seizures or not. Nash et al. reported that patients with enhancing calcified lesions were more likely to show perilesional edema and that perilesional edema was associated with seizures and neurological morbidity (6). However, Gupta et al. stated that enhancement is related to perilesional inflammation and is not associated with increased seizure activity (7). Explanations for persistent edema and/or enhancement are even less clear; however, most analyses point to an inflammatory response directed against sequestered antigens (5).

In our patient, ring enhancement and perilesional edema, which always occurred in tandem, were present at the time of seizure activity and frequently when the patient was asymptomatic. However, our patient was on antiepileptic medications for the majority of the followup period. He experienced a recurrent seizure approximately 3 years after cessation of therapy. Our case is limited, however, as no imaging was obtained to demonstrate the intermittent nature of enhancement while the patient was off of medication and seizure-free.

Our case, in conjunction with prior reports, effectively concludes that chronic calcified granulomatous NCC lesions may in fact enhance and can wax and wane. Current radiological literature must reflect the possibility for perilesional edema and/or enhancement during the final stage of NCC (nodular calcified) and not mistake enhancing calcified nodules for earlier stages, as there may be important management changes. Furthermore, more studies are needed to elucidate the cause of persistent enhancement in NCC's final stage as well as the clinical significance of the reemergence of perilesional edema and enhancement in the calcified granuloma.

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