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MIDC METRO INFECTIOUS DISEASE CONSULTANTS

The Evolution of a Ring-Enhancing Lesion

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Abstract: We report a case of probable neurocysticercosis in a 30-year-old non-Hispanic Caucasian patient without international travel history who presented with breakthrough seizures and a ring-enhancing lesion with perilesional edema. He first developed seizures two years prior, and his workup then demonstrated a single ring-enhancing lesion that was initially attributed to head trauma. On his presentation for breakthrough seizures, his ring-enhancing lesion demonstrated new perilesional edema, and on repeat imaging one month later, near resolution of the cystic lesion. His serum and cerebrospinal fluid serologies for *Taenia solium* were negative. Of note, the patient reports a friend who developed seizures at around the same time. While this patients demographics, lack of travel history, and negative serologies are not typical of neurocysticercosis in the United States, the characteristic evolution of his cystic lesion from the vesicular to the granular stage on MRI with late-onset perilesional edema and a classical presentation of seizures is most consistent with neurocysticercosis. Considering the absence of other epidemiological risk factors and the occurrence of seizures in close contact during the same timeframe, we hypothesize that the patient was exposed to *T. solium* through the consumption of contaminated food in the United States.

Keywords: neurocysticercosis; ring-enhancing; seizure; *T. solium*

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Case Presentation

A 30-year-old male with seizure disorder presented for two breakthrough seizures in 2023. His seizure disorder was diagnosed in 2021 when he was started on levetiracetam. He had magnetic resonance imaging (MRI) brain scans performed by his neurologist in 2021 (Figure 1) and 2022 (Figure 2), which

demonstrated a single ring-enhancing lesion that was attributed to head trauma during his seizure in 2021. An intracystic enhancement was noted in the 2022 MRI that was possibly consistent with a scolex but, per the patient, his neurologist had ruled out neurocysticercosis (NCC) due to a lack of epidemiological risk factors.

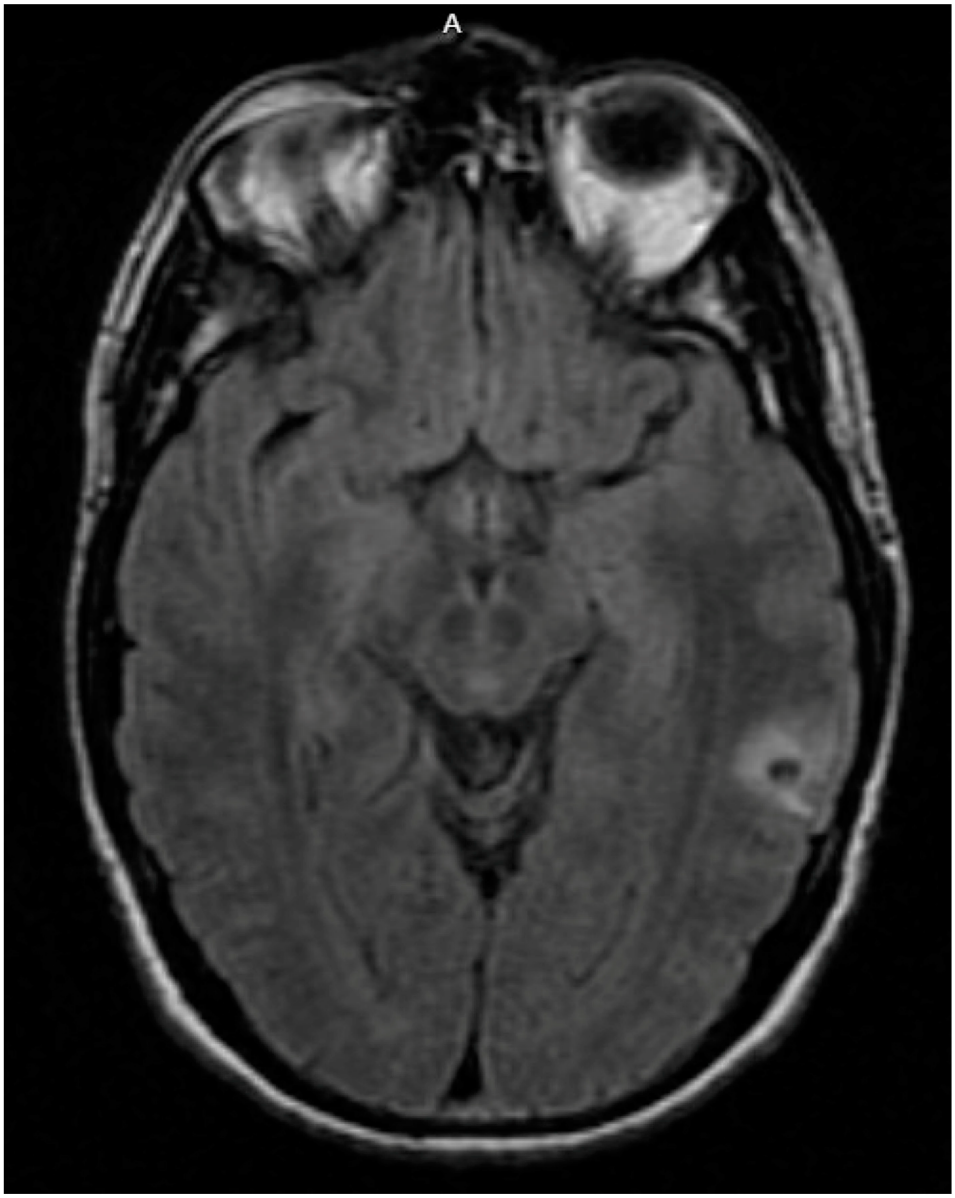

Figure 1: MRI brain, T2–fluid-attenuated inversion recovery (FLAIR), 2021. A 4 mm ring-enhancing lesion with halo of surrounding edema in the left posterior temporal–parietal lobe. In the clinical context of head trauma, imaging correlates with a small subacute parenchymal hemorrhage that may be post-traumatic in origin or could be related to an underlying small cavernoma.

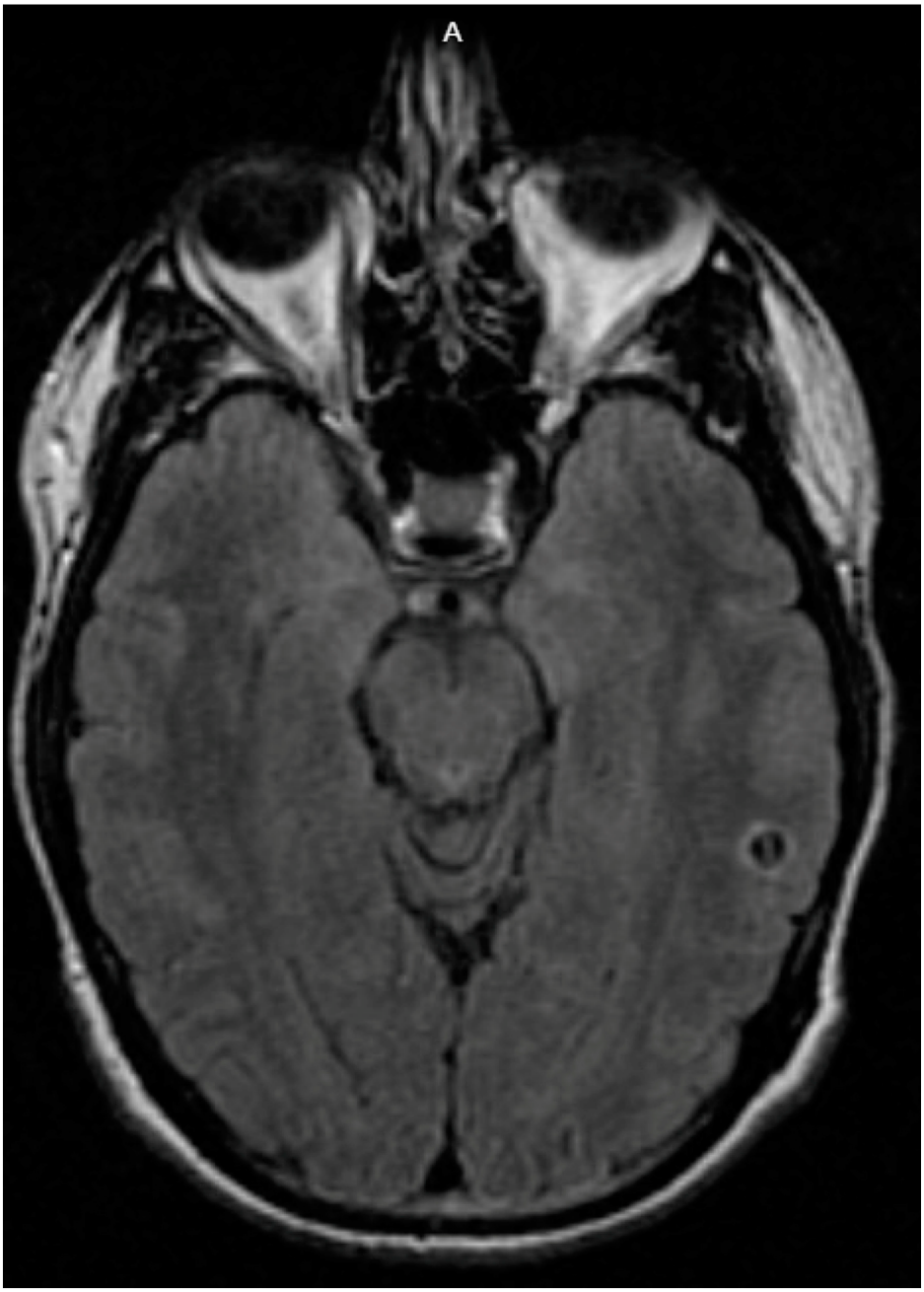

Figure 2: MRI brain, T2–FLAIR, 2022. An 8 mm ring-enhancing lesion with a central focus of signal likely due to a scolex with very minimal enhancement of the cyst wall and no associated edema, consistent with the vesicular stage of neurocysticercosis. The lesion was initially attributed to head trauma on interpretation in 2022.

On admission in the spring of 2023, the patient reported a mild headache after his two breakthrough seizures. He denied recent illness, fever, cough, rash, visual disturbances, gait disturbances, or weight loss. The patient is a non-Hispanic Caucasian male born and raised on the East Coast of the United States. He had been working as a middle school teacher for several years. He had no history of overseas travel except for a trip to Puerto Rico in 2022. He had no consistent exposure to live animals. Of note, the patient reported living in San Francisco from 2018 to 2019. He had a

friend whom he often dined out with who also developed new seizures in 2021; however, we were unable to contact his friend for further information. On admission, the patient was afebrile without neurological deficits or neck stiffness. His laboratory results demonstrated elevated creatine kinase and therapeutic levetiracetam levels. An MRI of the brain during his admission in 2023 revealed an 8 mm ring-enhancing lesion with regional vasogenic edema (Figure 3). Given the degenerating cyst with significant edema, the decision was made to withhold antiparasitic treatment with close outpatient follow-up. The neurology team increased twice-daily levetiracetam dosage from 750 mg to 1500 mg, which appropriately suppressed further seizures. No corticosteroids were used to control intracranial edema.

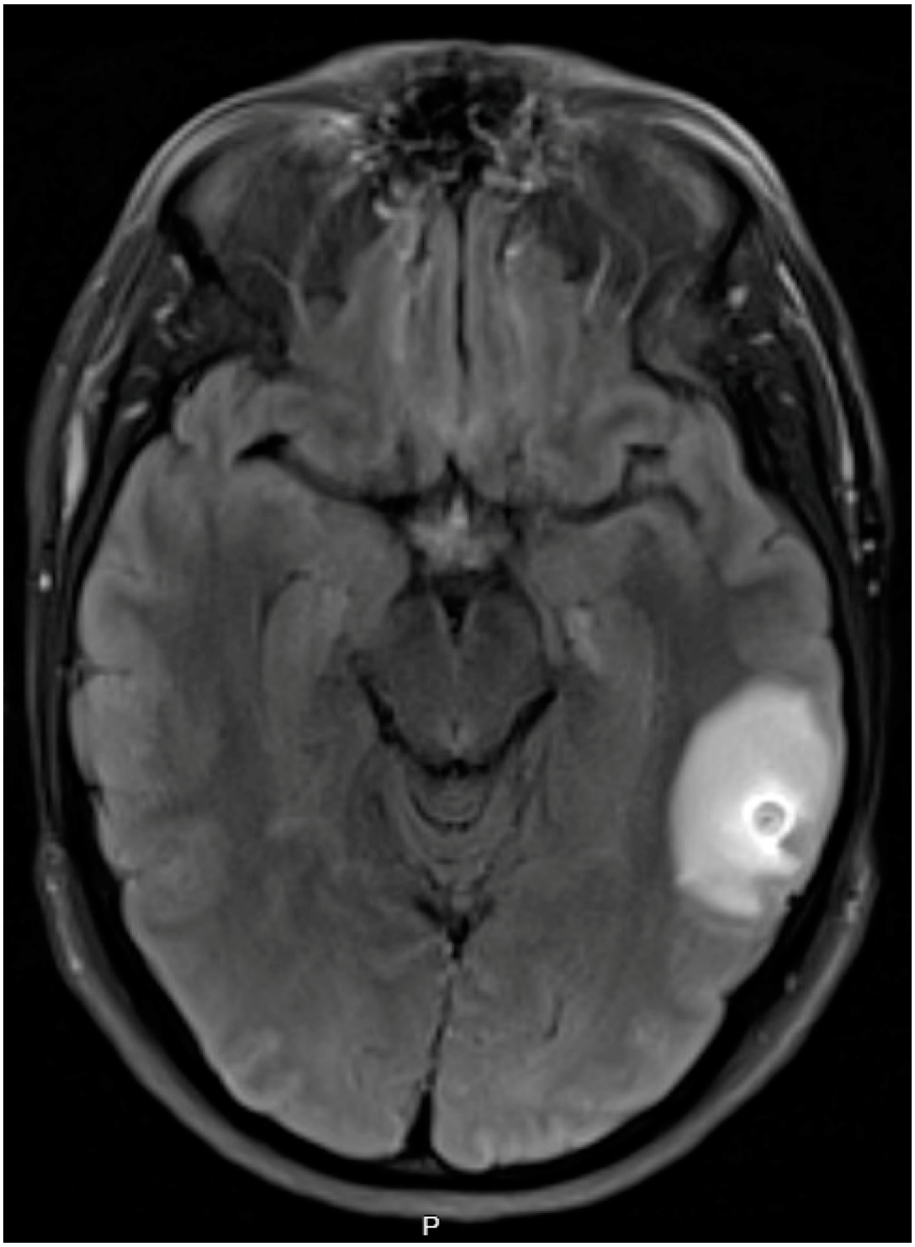

Figure 3: MRI brain, T2–FLAIR, on admission 2023. An 8 mm ring-enhancing lesion within the left temporal–occipital region with regional vasogenic edema, consistent with the colloidal phase of neurocysticercosis.

A repeat MRI one month later demonstrated that the cyst had decreased in size to 4.5 mm with total resolution of vasogenic edema, consistent with the granular stage of NCC (Figure 4), and demonstrated near-resolution on repeat MRI three months later (Figure 5). Serum cysticercosis IgG enzyme-linked immunosorbent assay (ELISA), serum and CSF cysticercosis enzyme-linked immunoelectrotransfer blot (EGIB) were negative. Tuberculosis interferon-gamma release assay, cryptococcal antigen, CSF studies including culture and meningitis panel, syphilis screen, *Toxoplasma gondii* antibodies, *Trypanosoma cruzi* antibodies and flow cytometry were negative as well.

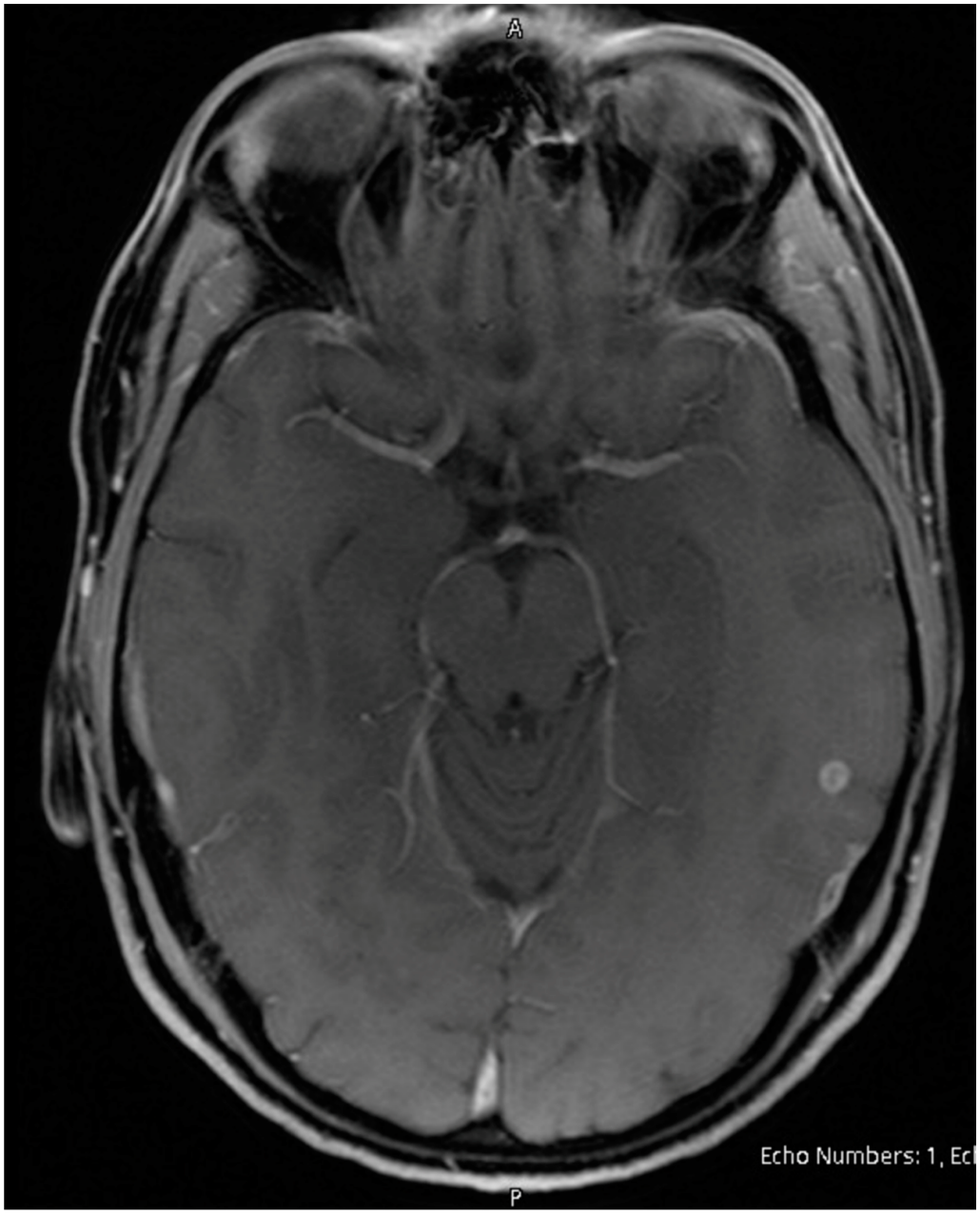

Figure 4: MRI brain, T1-weighted, one month after 2023 admission. The presumed cysticercoma in the left temporal lobe decreased in size along with a marked decrease in surrounding edema, consistent with the granular stage of neurocysticercosis.

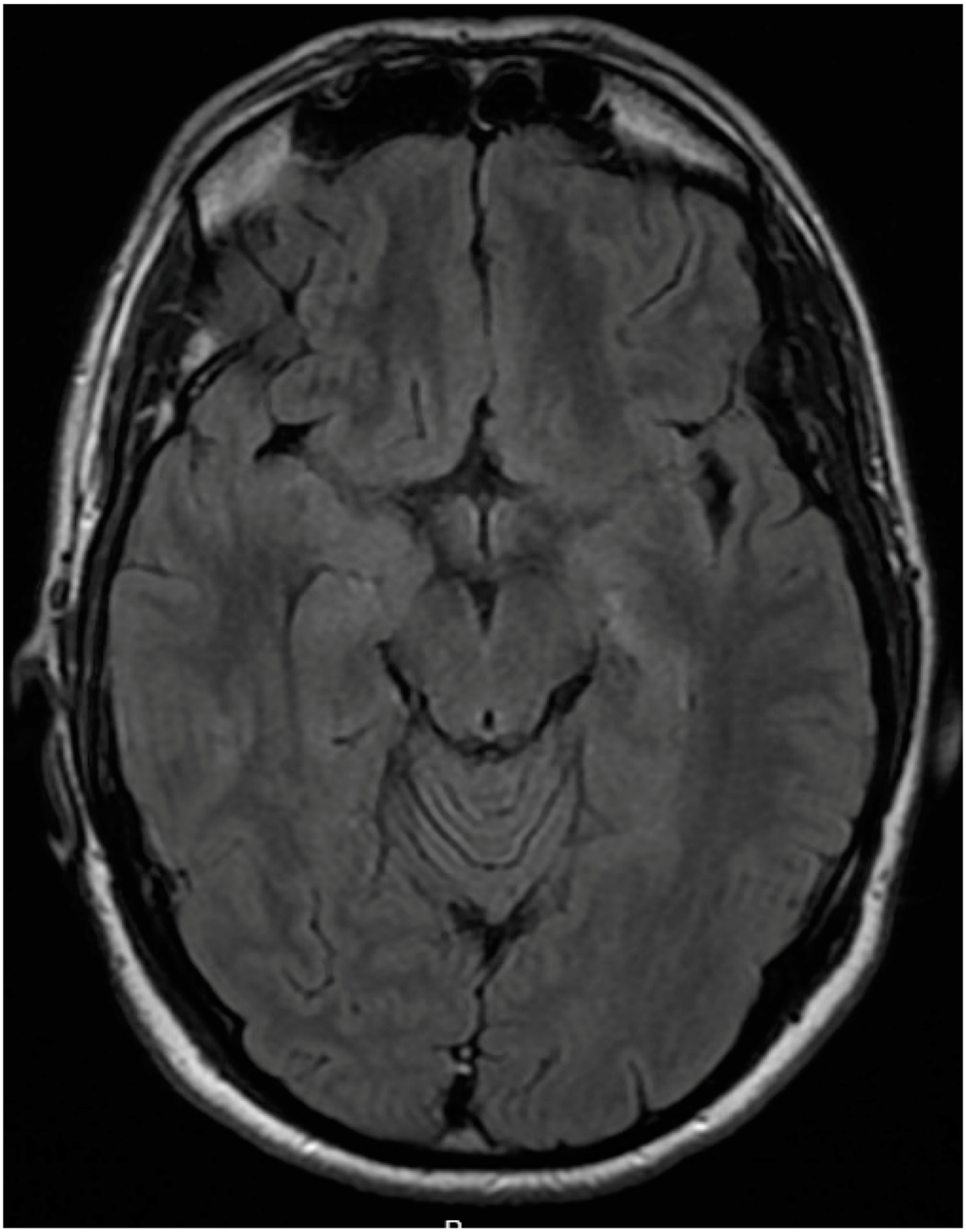

Figure 5: MRI brain, T2-FLAIR, three months after 2023 admission. Consistent with near-resolution of the cystic lesion.

Discussion

Neurocysticercosis is the most common cause of acquired epilepsy worldwide, and is endemic to countries in Africa, Asia, and Latin America [1]. NCC in the United States affects mainly young adult Hispanic immigrants in the Southwest, with approximate estimates of 0.2 to 0.6 cases per 100,000 individuals in the general population and 1.5 to 5.8 cases per 100,000 among Hispanic people [2]. The primary route of transmission of NCC is through the ingestion of *T. solium* eggs, which are present in the feces of individuals infected with the adult tapeworm. Cases of transmission have mostly been documented in the setting of travel to an endemic area with contaminated food or water, but have also been documented in the setting of a tapeworm carrier present in the household [2], including housekeepers who may work but not reside in the household [3], and theoretically, in the setting of imported contaminated produce [4]. There was a recent case report of another patient in the United States who developed NCC without travel history or other identified risk factors [5]. The fact that a person who is a close contact of our patient developed a seizure disorder within the same period could signify a potential epidemiological link. Since their primary shared activity was dining out together, it is plausible that they may have been exposed to *T. solium* through contaminated food in San Francisco. However, our inability to obtain further information from the friend of our patient restricts our ability to draw definitive conclusions.

While neither the patients demographics, exposure history, nor travel history are typical of NCC, the characteristic evolution of his cystic lesion from the vesicular to the granular stage to near-resolution on MRI, and a classic presentation of seizures, is most consistent with NCC. The intracystic lesion detected in the 2022 imaging possibly represents a scolex, which would fulfill an absolute criterion for NCC diagnosis [6]. The patients negative serologies do not definitively rule out NCC; although the sensitivities of EGIB and ELISA tests are approximately 98% when there are more than 2 ring-enhancing lesions, the detection rate for patients with a single lesion or those with nonviable infections is closer to 28% [7,8]. In cases of both single and multiple lesions, antibody detection has been shown to be more sensitive in serum compared to cerebrospinal fluid [7].

NCC is divided into four main neuroradiological stages: vesicular, colloidal, granular, and calcified [9]. Per the Infectious Diseases Society of America (IDSA) guidelines, antiparasitic agents are not recommended for calcified lesions, but they are strongly recommended for viable early-stage NCC. The evidence for treating small solitary nonviable NCC lesions is evolving. Without antiparasitic treatment, approximately 30% to 40% of single NCC granulomas will radiographically clear within a year, while approximately 50% will become calcified lesions [10,11]. In patients with significant cerebral edema, the cystericidal activity of albendazole may worsen edema and increase seizure risk [12]. In patients without significant edema, the IDSA endorses weak to moderate evidence for antiparasitic treatment for patients with nonviable, noncalcified single enhancing lesions [10]. A 2021 Cochrane review cites moderate evidence for antiparasitic treatment compared to placebo for single nonviable, noncalcified intra-parenchymal cysts for seizure reduction (RR 0.61; 95% CI, 0.4 to 0.91; 5 trials, 396 participants), and increased radiological clearance of lesions (RR 1.22; 95% CI, 1.07 to 1.39; 13 trials, 1324 participants) [11]. Overall, there is increasing evidence towards the treatment of nonviable, noncalcified NCC lesions. For our patient, the decision was made to hold antiparasitic treatment given the already significant vasogenic edema triggering breakthrough seizures.

Conclusion

This is a case of probable neurocysticercosis in a non-Hispanic male born and raised in the United States, with no history of international travel, who presented with seizures and a workup that demonstrated an evolving ring-enhancing lesion. Despite negative *T. solium* serologies, the clinical presentation of seizures and the radiographic progression of the ring-enhancing lesion from the vesicular to granular stage are most consistent with neurocysticercosis.

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Informed Consent Statement: We have received patient consent to include their medical information in a clinical case report.

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Conflicts of Interest: No conflicts of interest to disclose.

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