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Visual Diagnosis Pearl

Chronic Neurocysticercosis in a Patient Presenting with Chest Pain

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A 64-year-old woman presented to the emergency department (ED) with chest, left arm and jaw pain in the setting of severely elevated blood pressure. Her previous medical history was significant for hypothyroidism, scleroderma on immunosuppression, interstitial lung disease, obstructive sleep apnea, and prior treated osteomyelitis. She reported having nausea, intermittent headaches, and fatigue prior to the onset of her chest discomfort. In the ED, her heart rate was 96 beats per minute and regular, and blood pressure was elevated to 220/100 mmHg. She did not have elevated jugular venous pressure (JVP) but did have scattered wet crackles over bilateral lower lung fields. The rest of her physical examination, including neurologic exam was unremarkable. Her laboratory tests revealed normal troponins and an unremarkable 12-lead electrocardiogram (ECG). Chest radiograph and beta-natriuretic peptide (BNP) suggested mild pulmonary edema. She was admitted for a hypertensive urgency and acute coronary syndrome rule-out and treated with antihypertensives and diuretics. A non-contrast head CT was obtained given her headache and revealed no acute infarct or intracranial hemorrhage. Mild nonspecific increased T2 signal was noted within the periventricular and deep white matter. A single tiny punctate calcification in the left high posterior frontal subcortical white matter without surrounding edema or enhancement was noted. Her presenting symptoms resolved, though she continued to have mild headaches. On further questioning, she described a one-year history of nearly daily headache located on the top of her head, feeling like weight, debilitating in nature, and associated with photophobia, nausea, and eye pain with lacrimation. She had been taking acetaminophen and naproxen almost daily for symptom control. Magnetic resonance imaging (MRI) of her brain with gadolinium was performed for further characterization. The study confirmed no acute findings, with no acute infarct or intracranial hemorrhage and confirmed a single tiny punctate calcification left high posterior frontal

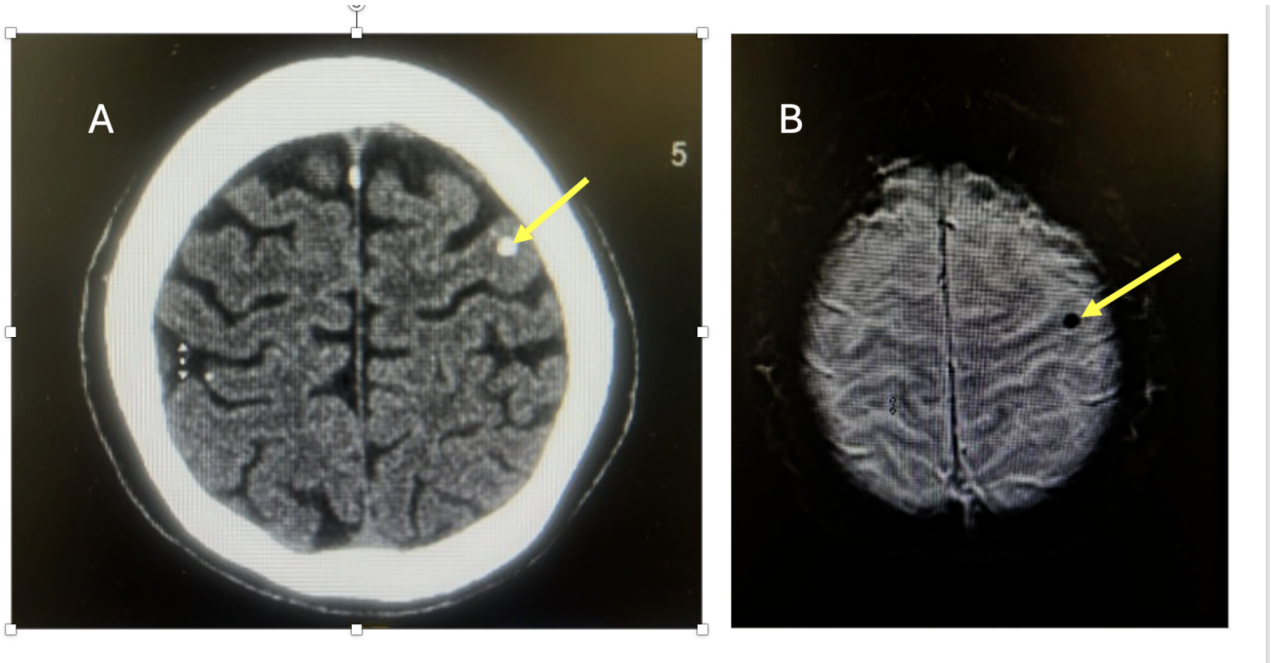

Figure 1. A: Non-contrast head CT demonstrated no acute infarct or intracranial hemorrhage. Mild nonspecific increased T2 signal was noted within the periventricular and deep white matter. A single tiny punctate calcification in the left high posterior frontal subcortical white matter without surrounding edema or enhancement seen. This is possibly sequela of previous cysticercosis (yellow arrow). B: MRI brain with gadolinium confirmed a single tiny punctate calcification in the left high posterior frontal subcortical white matter without surrounding edema or enhancement (yellow arrow). Possibly sequela from previous cysticercosis.

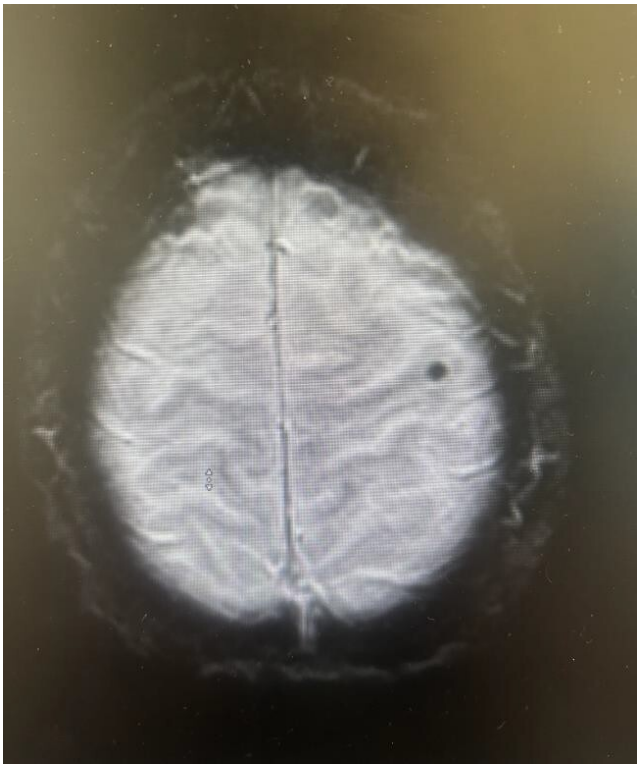

Image 2. MRI W/ Contrast once again demonstrates a single tiny punctate calcification left high posterior frontal subcortical white matter without surrounding edema or enhancement

subcortical white matter without surrounding edema or enhancement. Likely sequela from previous cysticercosis.

DISCUSSION

Our case demonstrates an incidental finding of chronic inactive neurocysticercosis in a patient presenting with unrelated symptoms. The imaging findings are characteristic of calcified parenchymal neurocysticercosis, representing the end stage of parasitic infection with *Taenia solium* larvae. The single small calcification without surrounding edema or enhancement indicates an inactive, calcified granuloma that likely formed years to decades after initial infection. Neurocysticercosis is acquired through ingestion of *Taenia solium* eggs, typically from contaminated food or water or from contact with a tapeworm carrier. There is typically a latent period of months to years between infection and onset of symptoms. Exposure history should include queries about access to safe water and improved sanitation throughout life, contact with tapeworm carriers, and contact with pork-raising areas, which may have occurred years prior to symptom onset.

Computed tomography (CT) scans are more sensitive than MRI for detecting calcified lesions.[1] Typical calcifications are usually small (1-4 mm), dense, and round, though they can be large and irregular. The number of calcifications varies from one to hundreds,[2] but most patients have 1-2 calcifications. Calcified lesions may not be diagnostic in isolation but in endemic areas are highly suggestive of neurocysticercosis. Calcifications represent sequelae of previous infections and do not contain viable parasites. While historically considered "inactive" lesions, calcified granulomas can be foci of seizure activity.[1] Approximately 30-50% of patients with calcified neurocysticercosis develop transient episodes of perilesional edema around calcified foci, which are associated with seizure activation.[1] This perilesional edema is likely due to an immune-mediated inflammatory response to antigens present in the calcified granulomata. The edema typically regresses without treatment in 3-6 weeks, though repeated episodes can occur over decades.[3]

In this patient, the calcification was asymptomatic without perilesional edema or enhancement. Her chronic headaches were more consistent with a primary headache disorder rather than neurocysticercosis-related symptoms. The headache characteristics such as daily occurrence, sensation of weight on top of head, photophobia, nausea, and associated eye pain with lacrimation and suggested possible tension-type headaches or a trigeminal autonomic cephalalgia.

The extent of evaluation required for patients with calcified parenchymal neurocysticercosis depends on symptoms. Asymptomatic subjects without evidence of viable cysts do not require additional evaluation.[3] Patients with seizures should be studied with MRI to exclude coexisting viable cysticerci, especially in the posterior fossa.[3] Patients with symptoms of increased intracranial pressure should get imaging to exclude subarachnoid or ventricular disease. For this patient, no specific treatment for the calcified neurocysticercosis was indicated. The focus appropriately shifted to management of her chronic headaches with outpatient neurology follow-up and her cardiovascular evaluation for chest pain and hypertensive urgency.

REFERENCES

1. Garcia HH, Nash TE, Del Brutto OH. Clinical Symptoms, Diagnosis, and Treatment of Neurocysticercosis. *The Lancet Neurology*. 2014;13(12):1202-1215. doi:10.1016/S1474-4422(14)70094-8

2. Bustos JA, Arroyo G, Gilman RH, et al. Frequency and Determinant Factors for Calcification in Neurocysticercosis. *Clin Infect Dis*. 2021;73(9):e2592-e2600. doi:10.1093/cid/ciaa784. PMID:32556276

3. White AC Jr, Coyle CM, Rajshekhar V, et al. Diagnosis and Treatment of Neurocysticercosis: 2017 Clinical Practice Guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). *Clin Infect Dis*. 2018;66(8):e49-e75. doi:10.1093/cid/cix1084. PMID:29481580