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Visual Diagnosis Pearl

Incidental Rim-Enhancing Cerebellar Cystic Lesion

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An 89-year-old woman presented to the emergency department after referral by her retinal specialist for acute right eye vision loss of eight days duration. Her previous medical history included paroxysmal atrial fibrillation (on aspirin), hypertension, coronary artery disease, left bundle branch block, large cell lymphoma in remission, and renal oncocytoma status post radiofrequency ablation. She described waking with sudden vision loss, perceiving only a small central light at the end of a dark tunnel. She denied focal neurological symptoms or fever. She did not endorse any current or chronic headaches or photophobia. She reported a recent fall attributed to impaired depth perception She did not endorse fall related head trauma. Her vital signs were notable for irregularly irregular pulse with a rate of 83 beats per minute, a blood pressure of 181/72 mmHg, normal oxygen saturation on room air. Physical examination was significant for an afferent pupillary defect in her right eye. On fundoscopy the right retina was pale and edematous. There were no visible retinal emboli The remainder of her neurological exam was non focal and without cerebellar signs. Based on her history and physical, central retinal artery occlusion (CRAO), with concern for giant cell arteritis (GCA) was the most likely diagnosis. Her labs were significant for a potassium 3.2 mEq/L (N 3.5 to 5.0–5.2 mEq/L), a creatinine 0.9 mg/dL (N 0.6 to 1.1 mg/dL), erythrocyte sedimentation rate (ESR) 62 mm/hr. (N 0–30 mm/hr.), and C -reactive protein (CRP) 36.4 mg/L (<0.3 mg/dL). She was admitted for further CRAO management. Pulse-dose methylprednisolone at 1g IV daily for three days was administered, then transitioned to oral prednisone 60 mg daily. Temporal artery biopsy was performed on hospital day 3. An outpatient carotid Doppler had demonstrated severe atherosclerotic plaque in the left distal common carotid artery. A non-contrast head computed tomogram (CT) demonstrated no acute intracranial hemorrhage or midline shift. A 13 x 9 mm hypoattenuation was identified at the left cerebellar hemisphere. The official read suggested it could represent an age-indeterminate infarct however they could not exclude an underlying solid lesion and recommended magnetic resonance imaging (MRI). CT has limited sensitivity for characterizing posterior fossa lesions due to beam-hardening artifact from the petrous bones, making MRI the standard modality for further evaluation. MRI revealed a cystic lesion with rim enhancement and wall nodularity in the left cerebellar hemisphere measuring 1.9 x 1.4 cm (Figure 1) without restricted diffusion and no significant perilesional edema. A questionable enhancing lesion in the subependymal region of the right lateral ventricle was also identified. Punctate foci of restricted diffusion in the right external capsule and splenium of the corpus callosum with T2/FLAIR hyperintensity were noted, suggestive of acute to subacute infarction—consistent with thromboembolic events in the setting of atrial fibrillation and severe carotid atherosclerosis. MRI of orbits were unremarkable without optic neuritis.

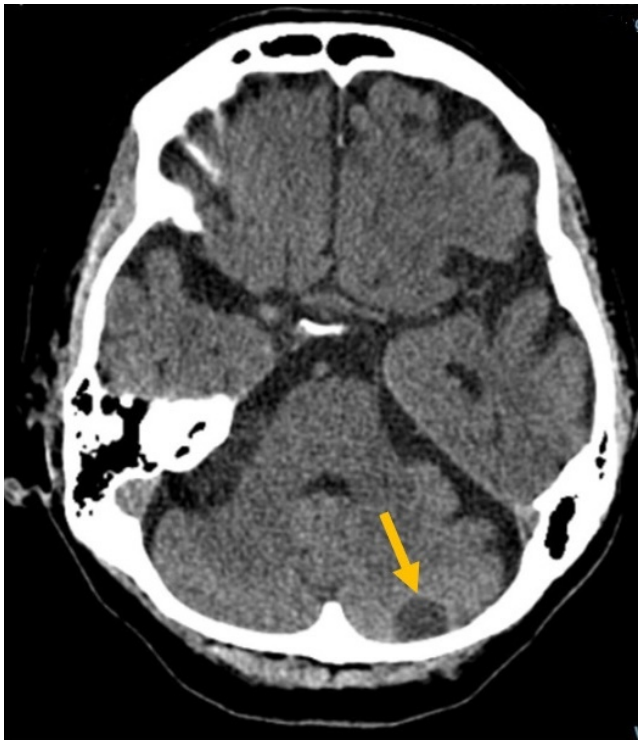

Figure 1. Head CT without contrast with an age-indeterminate infarct (yellow arrow) however cannot exclude underlying solid lesion.

For the cerebellar lesion, Toxoplasma IgG was positive but IgM negative, indicating prior exposure without acute infection. A positron emission tomogram (PET) scan was negative for fluorodeoxyglucose (FDG) uptake, arguing against high-grade neoplasm or active lymphoma recurrence. The patient and her daughter declined neurosurgical biopsy given her age and asymptomatic status. She was discharged on prednisone 60 mg daily pending biopsy results, with anticoagulation (apixaban 5 mg BID) initiated for atrial fibrillation per neurology and cardiology recommendation.

DISCUSSION

The rim-enhancing cystic cerebellar lesion with wall nodularity raised a broad differential in this elderly immunosuppressed patient with prior lymphoma. The absence of restricted diffusion was a key finding, effectively excluding pyogenic abscess, in which all abscess cavities demonstrate hyperintense diffusion weighted image (DWI) signal with low apparent diffusion Coefficient (ADC).

This is a quantitative MRI technique is used to measure the random movement (diffusion) of water molecules within tissue. whereas neoplastic cysts generally show high diffusion and low fractional anisotropy.[1] The lack of significant perilesional edema is atypical for high-grade neoplasms or metastases. Cerebellar hemangioblastoma is the most common primary intra-axial posterior fossa tumor in adults. It classically presents as a cyst with mural nodules. However, ring enhancement of the cyst wall is uncommon 8.7%) compared with metastasis (100%). Features favoring metastasis include multiple mural nodules, absence of vascular flow voids, and mildly enhanced mural nodules, none of which were seen.[2] The patient's age is older than typical hemangioblastoma presentations (median 37 years for sporadic cases). Central nervous system (CNS) lymphoma recurrence was also considered. This usually presents as ring-enhancing lesion in immunosuppressed individuals. The negative FDG-PET significantly reduced this likelihood, as typical CNS lymphoma demonstrates FDG uptake approximately 2.5 times higher than normal gray matter.[3]

Regarding the CRAO, GCA must be ruled out urgently in patients older than 50 years with elevated ESR and CRP. This patient's elevated inflammatory markers appropriately prompted pulse-dose corticosteroids and temporal artery biopsy. The concurrent acute cerebral infarcts underscored the thromboembolic burden in the setting of untreated atrial fibrillation and severe carotid disease and were likely related to the same embolic mechanism as the CRAO.

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