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IMAGES IN EMERGENCY MEDICINE

Neurology

A 54-year-old with dysarthria

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1 | PATIENT PRESENTATION

A 54-year-old male with a history of intravenous heroin abuse presented to the emergency department for dysarthria. The patient was initially found down with an unknown last known well time. The patient had a blood pressure of 237/101 mmHg. He was alert with a left-sided gaze preference and right-sided Romberg drift. Labs were remarkable for white blood cell count (WBC) 25.7 103/μl and creatine kinase (CK) 5776. Drug screen was positive for opiates. Computed tomography brain and angiography imaging showed bilateral cerebellar hemispheric ischemia, lateral ventricle dilatation, and a right proximal vertebral artery with non-occlusive diminutive distal flow (Figure 1). Several hours later, the patient subsequently experienced a rapid neurological decline from Glasgow Coma Scale (GCS) 11 (E4V2M5) to GCS7 (E2V1M4), prompting intubation for airway protection. Mannitol and 2% hypertonic saline were administered. External ventricular drain was prepared for potential deterioration. Subsequent magnetic resonance imaging showed stable, bilateral subacute cerebellar infarcts with hydrocephalus (Figure 2) and the patient was ultimately extubated and required prolonged rehabilitation.

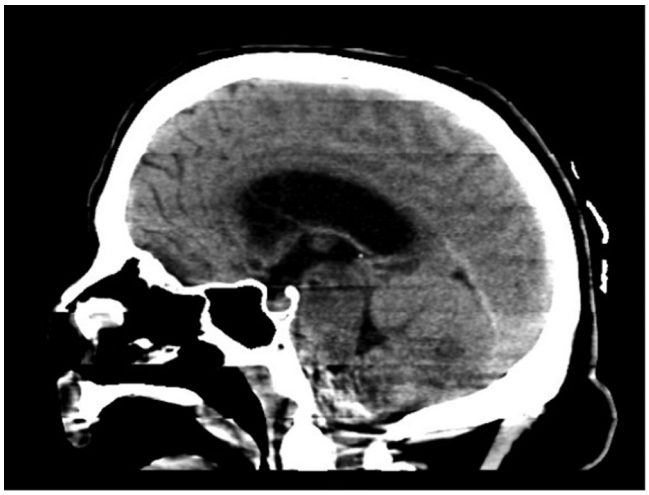

FIGURE 1 Noncontrast computed tomography (CT) brain sagittal view with obliterated fourth ventricle with ventricular dilatation above the tentorium suggestive of obstructive hydrocephalus. Loss of normal gray-white matter differentiation of cerebellum suggestive of diffuse bilateral infarction

2 | DIAGNOSIS: BILATERAL CEREBELLAR STROKE

2.1 | Discussion

Bilateral ischemic cerebellar strokes are rare cerebrovascular accidents (CVAs).[1] Large volume infarcts have a high risk of developing space-occupying brain edema in the compact posterior fossa—risk of rapid, life-threatening herniation through compression of the brain stem and the fourth ventricle with resultant hydrocephalus.[2] Opiate abuse has been described as a rare cause of cerebral ischemia though mechanisms including vasospasm and severe hypoperfusion, rhabdomyolysis via muscle compression, and direct toxicity.[3,4]

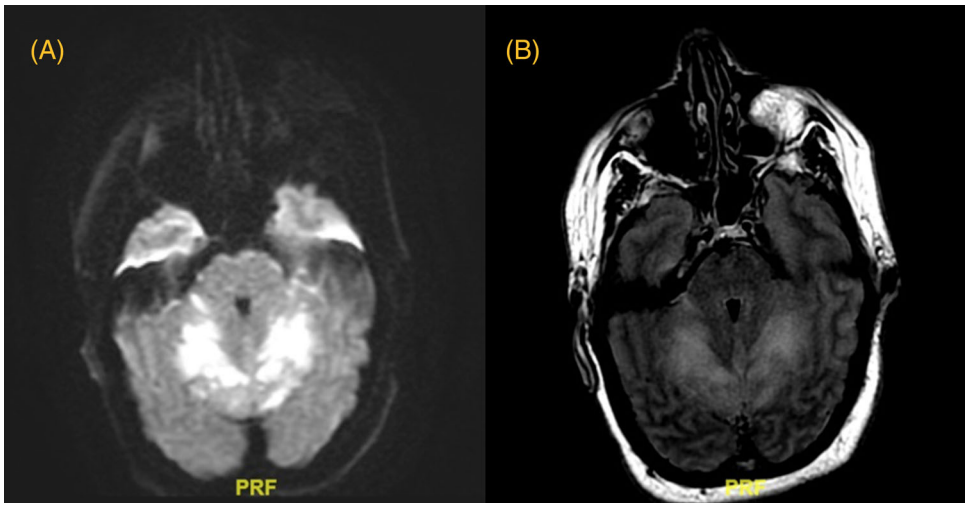

FIGURE 2 Magnetic resonance imaging axial views with large volume bilateral cerebellar infarcts and moderate volume bilateral mesial temporal lobe subacute nonhemorrhagic infarcts. (A) Diffusion weighted imaging. Positive signal suggestive of ischemia. (B) Fluid-attenuated inversion recovery. Positive signal in area of infarct suggestive of a more subacute onset of stroke. Abbreviation: PRF, posterior right foot

In our case, stable repeat imaging and an elevated CK suggested a subacute presentation and prompted holding off suboccipital craniectomy given the high likelihood of being outside of the acute edema window for effective surgical intervention.[5] A positive drug screen with a known history of intravenous drug abuse point to a rare drug-associated ischemia as a potential etiology. Timing of pathology, repeat imaging and clinical assessment, and maintaining a wide differential, including drug-induced ischemia, in relatively young patients without typical CVA risk factors are essential in management of severe cerebellar strokes with unknown onset.

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