When sleep is NOT the best medicine - Percheron variant cerebral infarct presenting as coma and somnolence  
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Introduction
The artery of Percheron (AOP) is an unusual anatomical variation, a single arterial trunk that supplies blood to the paramedian thalamic nuclei and in some instances the midbrain [1].

When a stroke occurs, it affects bilateral thalamic areas, occasionally including the midbrain.

Due to its diverse range of clinical manifestations, diagnosing an AOP stroke is often challenging. Typically, it presents with symptoms including vertical eye palsy, hypersomnolence, and sometimes coma.

Our case involves a 75-year-old man whose initial symptoms were marked by an onset of coma, coupled with oculomotor palsy.

Case
A 75-year-old male with a history of hyperlipidemia and hypertension presented unresponsive with a Glasgow Coma Score of 6.

Pupillary light reflex in both eyes demonstrated minimally reactive, anisocoria with the right being pinpoint while the left pupil being dilated. The patient flexed his arm and extended his leg on painful stimulus.

He had no witnessed seizure activity and displayed no focal neurological deficits. Computed tomography (CT) of the head and CT angiography was unrevealing. Lab work was within normal limits.

EKG was unremarkable. Treatment with naloxone showed no improvement.

The patient was intubated and transferred to the ICU.

Vital signs showed transient intermittent fevers and labs showed mild leukocytosis.

An additional workup was done to rule out other possible missed causes. Lumbar puncture was negative. Electroencephalogram (EEG) revealed no seizure activity.

Decision making
Magnetic resonance imaging (MRI) was done 24 hours after admission and revealed bi-thalamic infarction and partial midbrain involvement [Figure 1]. The patient was started on aspirin and Plavix.

Over several days, the patient began to regain consciousness. It was noted that the patient did have left-sided oculomotor palsy and complained of diplopia.

The patient was extubated on day 6 but required reintubation for an additional 48 hours due to hypersomnolence and progression to coma-like state again.

The patient was re-imaged showing some edema due to the evolution of the stroke for which hypertonic saline was initiated to decrease brain edema.

Two days later the patient improved and became responsive, and he was started on stimulants to maintain a wakeful state which allowed for successful extubation.

Patient was eventually discharged to rehab, with diplopia being a residual deficit.

Conclusion
Bilateral thalamic stroke due to AOP occlusion is a rare type of ischemic stroke and cause of coma. Diagnosis of AOP stroke is often made retrospectively beyond the thrombolysis or endovascular treatment window [2]. In the event a patient presents with unexplained unresponsiveness, with normal CT imaging, a stroke should not be ruled out and the patient should have an urgent MRI scan. Overall, the prognosis is good with low mortality and often reversible coma, necessitating early diagnosis and optimal supportive care [2].

References