The Journal of Emergency Medicine, Vol. ■, No. ■, pp. 1–4, 2018

0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2018.02.048

Clinical Communications: Adult

ARTERY OF PERCHERON STROKE

Matthew L. Wong, MD, MPH and Jonathan A. Edlow, MD

Department of Emergency Medicine, Beth Israel Deaconess Medical Center, and Harvard Medical School, Boston, Massachusetts
Reprint Address: Matthew L. Wong, MD, MPH, Department of Emergency Medicine, Beth Israel Deaconess Medical Center, Rosenberg Building, 2nd Floor, 1 Deaconess Road, Boston, MA 02446

☐ **Abstract—Background: Coma is not a common symptom of stroke. Case Report: We present a patient with a stroke to the artery of Percheron, which infarcted the bilateral paramedian thalami and resulted in coma. Why Should an Emergency Physician Be Aware of This?: Identifying strokes in comatose patients is important for therapeutic management. The bilateral thalami are involved in maintaining consciousness. The artery of Percheron is an anatomic variant in the posterior circulation whereby the bilateral paramedian thalami are perfused by it solely. This is an atypical stroke syndrome that emergency physicians need to be able to identify.** © **2018 Elsevier Inc. All rights reserved.**

☐ **Keywords—artery of Percheron; coma; stroke; reticular activating system**

INTRODUCTION

Although decreased level of consciousness is usually considered a "nonfocal" neurological finding, strokes can cause acute coma. Arousal has a structural and physiological basis in the brain. The reticular activating system (RAS), which involves the pons, midbrain, bilateral thalami, and bilateral hemispheric projections, is responsible for consciousness. Strokes, toxins, and metabolic derangements that affect those areas may result in decreased level of consciousness (1).

The artery of Percheron is an anatomical variant of the posterior circulation in which the paramedian portions of the bilateral thalami are supplied by one end-artery. Thromboembolism of the artery of Percheron results in acute ischemic stroke that causes decreased levels of consciousness, ocular motor dysfunction, and memory and behavioral disturbances. Early identification of these patients is important and may facilitate earlier stroke interventions.

CASE PRESENTATION

A 93-year-old woman was transferred from an outside facility for an acute change in mental status. Emergency Medical Services was initially called to the patient's home when she abruptly lost consciousness in the presence of family and was persistently difficult to arouse. The family did not notice any rhythmic movements. Finger-stick glucose was normal. Her medications included lisinopril, metoprolol succinate, glipizide, metformin, levothyroxine, mirtazapine, and melatonin; she was not on any anticoagulants.

At the hospital, the following tests were normal: noncontrast head computed tomography (CT), complete blood count, a comprehensive metabolic panel, electrocardiogram, and urinalysis. Drug screening showed no acetaminophen, salicylate, or ethanol in her blood. The

None of the authors have any financial interests to declare, though Dr. Edlow sometimes reviews medical malpractice claims, for both plaintiffs and defendants, and sometimes the cases are about stroke.

RECEIVED: 8 February 2018;
ACCEPTED: 28 February 2018

family reported a past history of atrial fibrillation, hypertension, diabetes, and hypothyroidism.

The patient was transferred to our institution 4.5 h after she was last seen normal. On arrival her capillary blood sugar was 117 mg/dL. Vital signs were normal except for mild hypertension (141/95 mm Hg). Her electrocardiogram demonstrated atrial fibrillation without evidence of ischemia. On examination, the patient was responsive only to painful stimuli. Her Glasgow Coma Scale score was 7 (E1, V1, M5). She was mute and had no facial asymmetry. There was no evidence of head trauma. Her pupils were unreactive (the right was 4 mm and the left was 2 mm). Her extremities were mostly flaccid but she had some intermittent seemingly purposeful movements of the right arm, right leg, and the left leg, but she did not move her left arm at all. The rest of her physical examination was unremarkable. Though limited by global impairment, her National Institutes of Health stroke scale was estimated to be 19.

She was intubated and a Code Stroke was initiated. The noncontrast head CT scan did not demonstrate any evidence of ischemia, hemorrhage, or mass effect. CT angiogram of the neck demonstrated patent arteries without stenosis, dissection, occlusion, or aneurysm. CT angiogram of the head demonstrated patent cerebral vessels without evidence of stenosis, dissection, occlusion, or aneurysm. A CT perfusion study showed symmetrical cerebral blood flow and volume. Repeated extensive laboratory testing was normal, including serum and urine toxicology screens. She was admitted to the intensive care unit by the Neurology service.

The patient remained stable overnight. The next day, a noncontrast magnetic resonance image of the brain showed areas of restricted diffusion and hyperintense signal on fluid-attenuated inversion recovery sequences in the bilateral paramedian thalami, as well as the left occipital lobe, consistent with acute infarction (Figure 1). Continuous electroencephalographic monitoring demonstrated encephalopathy without seizure activity. Transthoracic echocardiogram demonstrated normal systolic function, and no patent foramen ovale. Based on the magnetic resonance image, the Stroke Neurology team concluded that the patient suffered an embolic stroke involving the artery of Percheron.

DISCUSSION

Coma, defined by a decreased level of alertness and response to external stimuli, is not commonly caused by acute ischemic stroke. Likewise, acute ischemic stroke does not often present with coma. However, emergency physicians need to be aware that a stroke involving the RAS or its connections can cause abrupt onset of coma (1). The RAS, a group of neurons that is responsible for wakefulness, originates in the pons and extends through the midbrain, then up into the bilateral thalami, and then it projects further cephalad into the bilateral cortical hemispheres. Damage to the RAS can result in coma (1).

The arterial blood supply for these structures originates from the bilateral vertebral arteries in the neck, which run cephalad and then join inside the skull to form the basilar artery. Small perforating branches of the basilar artery supply blood flow to the pons. More cephalad, at the level of the midbrain, the basilar artery bifurcates to form the left and right posterior cerebral arteries (PCAs), which traverse medially to laterally and ultimately supply the occipital lobes (Figure 2) (2).

Small penetrating arteries from the first portion of the PCA supply blood to the thalamus. Normally these are paired arteries, each side supplying the medial thalamus on its respective side. However, in roughly 11% of patients, both thalami will be supplied by a single artery

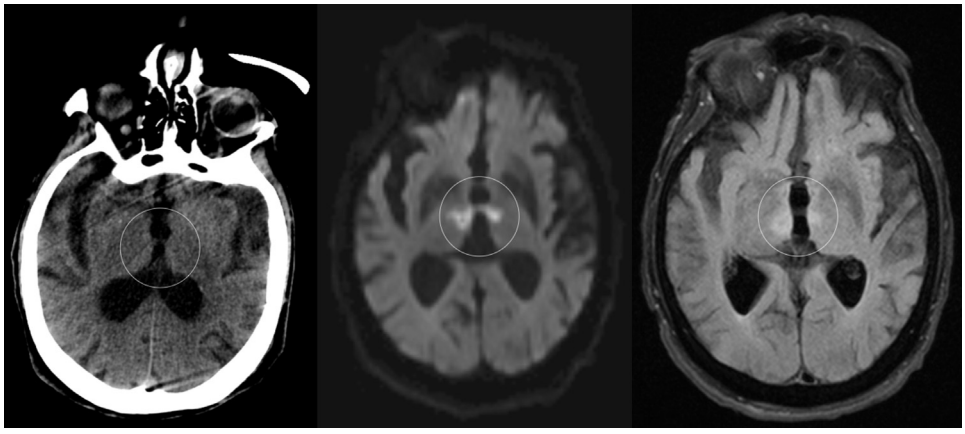

Figure 1. Noncontrast head computed tomography (left) demonstrating no acute thalamic infarct on arrival. Magnetic resonance diffusion weighted imaging (middle) and T2 fluid-attenuated inversion recovery imaging (right) demonstrating signal abnormalities consistent with acute infarct.