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Visual Diagnosis in Emergency Medicine

ACUTE BASILAR ARTERY THROMBOSIS

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CASE REPORT

A 42-year-old man developed acute altered mental status while on a transatlantic international flight to the United States. He was last witnessed to be behaving normally, while filling out his immigration form, approximately 30 to 45 minutes before landing. He was drowsy and very difficult to arouse by friends when departing the plane. There was no reported seizure activity witnessed. His friends denied suspicions of internal drug smuggling.

Upon landing, Emergency Medical Services' (EMS) initial assessment revealed pulse 84 beats/min, blood pressure 132/82 mm Hg, respiratory rate 20 breaths/min, pulse oximetry 99% room air, fingerstick blood glucose 114 mg/dL. Additional history was limited by a significant language barrier. EMS administered naloxone with no clinical improvement and the patient was transported to our emergency department (ED).

Physical examination revealed an ill-appearing, somnolent, diaphoretic male with occasional snoring respirations. No external signs of trauma or intraoral injury. Pupils were equal, approximately 4 mm, briskly reactive. Extraocular movements were significant for an involuntary disconjugate gaze. Regular rate and rhythm with no appreciable heart murmur on cardiovascular

examination. Neurologic examination was significant for a mild right ptosis and right hemiparesis with episodic decerebrate posturing. He was disoriented on arrival with severe dysarthria but appeared to follow simple



Figure 1. Magnetic resonance angiography of Circle of Willis. Arrow indicates basilar artery - absent enhancement; left vertebral artery - diminished enhancement; left superior cerebellar artery - absent enhancement.

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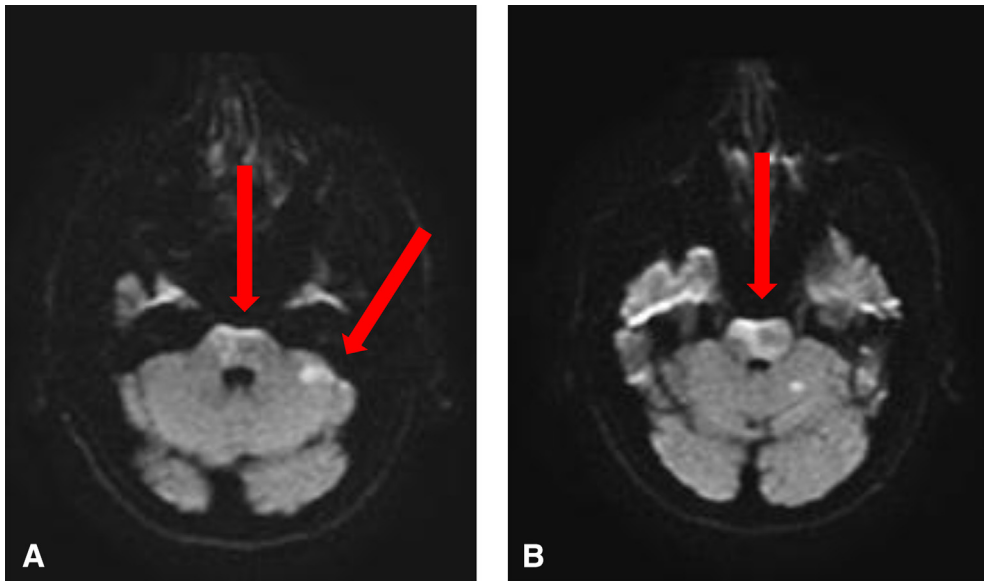


Figure 2. Magnetic resonance diffusion weight imaging. Arrows indicate restricted diffusion in pons (A and B) and left cerebellum (A).

commands (e.g., opening mouth, moving left side hand/leg) despite the language barrier. His initial Glasgow Coma Scale score was 12 and initial National Institutes of Health Stroke Scale score was 12.

A Stroke Alert was activated. His level of consciousness deteriorated and emergent intubation was performed. Laboratory tests including an arterial blood gas, complete blood count, basic metabolic panel, prothrombin time/partial thromboplastin time/International Normalized Ratio, cardiac enzymes, ethanol level, and urine toxicology were normal. In addition, the patient had a normal noncontrast head computed tomography. Magnetic resonance imaging/magnetic resonance angiography imaging revealed a brainstem infarct due to an acute basilar artery thrombosis with extension into the left vertebral artery and left superior cerebellar artery (red arrows, [Figures 1 and 2](#)). Patient received thrombolysis with i.v. alteplase approximately 2 hours after witnessed symptom onset. Arterial recanalization by emergent neuroendovascular mechanical thrombectomy was successfully performed.

DISCUSSION

Acute basilar artery occlusions are rare, accounting for approximately 1% of strokes (1). The basilar artery is formed by the convergence of the right and left vertebral arteries at the pontomedullary junction. It forms an anastomosis with the carotid vessels to form the Circle of Willis, which supplies the brainstem. Basilar artery

thrombosis occurs secondary to severe atherosclerotic vascular stenosis, thromboembolic occlusions, or vertebral artery dissections.

The most common clinical manifestations of basilar artery occlusions are reduced level of consciousness, hemiparesis or quadriparesis, gaze abnormalities, and cranial nerve palsies (2). Severe disruption in the basilar artery's blood supply to the reticular activating system can induce a catastrophic locked-in syndrome with quadriplegia and aphasia.

Acute interventions for basilar artery occlusions include intravenous thrombolysis, intraarterial thrombolysis, endovascular stenting, or mechanical thrombectomy. Emergent arterial recanalization is associated with improved functional outcomes and reduced mortality (3).

Further inpatient workup on this patient revealed a patent foramen ovale (PFO) with atrial septal aneurysm on cardiac echo. The patient underwent closure of the PFO. PFOs have a high incidence in cryptogenic cerebrovascular accident. The patient did remarkably well and was discharged on hospital day 12 with no significant neurologic deficit.

REFERENCES

1. Mattle HP, Arnold M, Lindsberg PJ, Schonewille WJ, Schroth G. Basilar artery occlusion. *Lancet Neurol* 2011;10:1002–14.
2. Baird TA, Muir KW, Bone I. Basilar artery occlusion. *Neurocrit Care* 2004;1:319–29.
3. Lindsberg PJ, Mattle HP. Therapy of basilar artery occlusion. *Stroke* 2006;37:922–8.