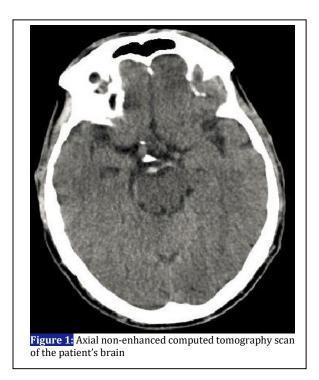
A 69-year-old Man with Sudden Loss of Consciousness, Non-reactive Pupils, and a Bilateral Positive Babinski Sign

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Published online: 2018-04-30



KEY QUESTIONS:

- What are the pathologic findings in figure 1?
- What is the importance of these findings?
- What other diagnostic modalities can be used for a definite diagnosis?

LEARNING POINTS:

Pathologic findings

In figure 1, the basilar artery appears homogenously hyperdense in comparison with the adjacent left middle cerebral artery (MCA), using brain parenchyma as a reference point; thus indicating a hyperdense basilar artery sign (HBAS) (Figure 2, white arrow). Also, the right superior cerebellar artery and left posterior cerebral artery appear to be hyperdense (Figure 2, the red and green arrows, respectively). This is called a hyperdense artery sign (HAS). The artery becomes hyperdense because the intra-arterial clotted blood has a higher Hounsfield unit (80 HU) than the non-clotted flowing blood (40 HU) and thus appears white on non-contrast computed tomography (CT) scan. Pathologically, high hematocrit levels and calcium deposits in the vessel wall (due to arteriosclerotic disease) can result in an incorrect diagnosis of HAS. Sometimes, infections or tumors can make the brain parenchyma surrounding the vessel hypodense, which can give the false impression of a hyperdense vessel (1). To avoid misdiagnosis, these considerations are useful:

- Ensure the attenuation value (Hounsfield units) of all intracranial arteries and veins are nearly the same when there are high hematocrit levels.
- Calcifications are usually located at the periphery of the vessels.
- In contrast to atheromatous calcifications, a hyperdensity thought to be a HAS is reversible (2).
- Koo *et al.* defined hyperdensity as an absolute density of > 43 HU and a MCA ratio (the ratio of the dense MCA to the contralateral MCA) of > 1.2. He showed that using a combination of these two parameters had 100% specificity for the HAS for acute ischemic stroke cases (3).

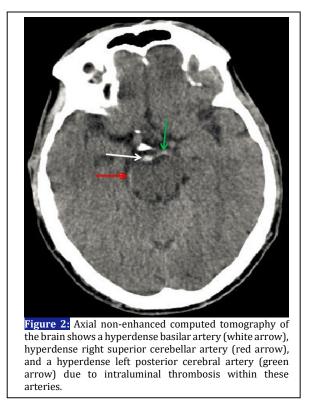
Importance

Acute basilar artery occlusion (BAO) is a rare catastrophic form of stroke, roughly causing around 1% of all strokes (4). BAO occurs usually due to an embolus with a cardiac or large vessel origin, or the formation of a local atherosclerotic thrombosis. In this patient, BAO occurred soon after myocardial infarction.

Depending on the location and extent of occlusion and on the degree of collateral flow, BAO has quite variable clinical and imaging manifestations. The clinical presentation of BAO ranges from nonspecific symptoms such as headache, neck pain, or vertigo to severe disabilities such as decreased consciousness, hemiplegia or quadriplegia, extensor plantar sign, dysarthria, dysphagia, aphagia, ataxia, nuclear oculomotor nerve palsy, bilateral ptosis, anisocoria, non-reactive pupils,

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disorientation, confusion, and/or memory disturbances (5). Thus, BAO should be considered in every patient with decreased consciousness and signs of brain stem dysfunction until proven otherwise.

HBAS is one of the earliest signs of posterior circulation infarcts on non-contrast CT images and is similar to the hyperdense middle cerebral artery sign in anterior circulation infarcts that is sometimes the only finding in an acute presentation. The HBAS may occur before the development of parenchymal hypodensity.

A 2015 systematic review and meta-analysis found only a 52% sensitivity but a 95% specificity of HAS for arterial obstruction on angiography. The researchers concluded that in the setting of acute ischemic stroke, HAS is associated with a high likelihood of arterial obstruction, but its absence only means a 50/50 chance of normal arterial patency. Thin-slice volumetric CT improves the sensitivity of HAS detection (6).

The functional outcomes of BAO have been improved due to developments in intravenous thrombolysis, intra-arterial thrombolysis, endovascular interventions, or mechanical thrombectomy, and clearly rapid diagnosis and early treatment are essential. Any favorable outcomes become impossible even after revascularization in cases of late diagnosis (after 24 hr) of HAS due to BAO (7).

Other diagnostic modalities

• Multimodal CT scans

A non-contrast CT shows ischemic brain parenchyma as a hypo-attenuation area. The sensitivity of a non-contrast CT to show acute posterior circulation infarcts is low. Contrast agents can significantly increase the sensitivity and specificity of CT. On contrast CT images, BAO can be seen as a filling defect within the vessel. On CT angiography, the gold standard, the missing part of the basilar artery can easily be seen. CT perfusion imaging is another diagnostic modality that can roughly distinguish irreversibly damaged areas from reversible ones.

• Multimodal magnetic resonance (MR) techniques

Diffusion-weighted MRI and apparent diffusion coefficient images can show ischemic areas much sooner than spin-echo and fluid attenuation inversion recovery (FLAIR) images. Capillary blood flow can be assessed on perfusion-weighted images. The mismatch between areas with reduced perfusion and diffusion restriction provide information about the penumbra, severely hypoperfused but salvageable tissue. On T2-weighted spin-echo and FLAIR images, infarct areas depict hyper-intense signals. Using MR angiography, the occluded artery can be visualized as having a missing piece due to loss of the blood flow signal.

• Transcranial Doppler and color-coded duplex sonography

On transcranial Doppler and color-coded duplex sonography, an absence of a signal in the basilar artery and the presence of abnormal waveforms in the vertebral arteries and collateral flow are suggestive of BAO. Ultrasound cannot be used to rule out BAO with certainty because of its sensitivity (8).

• Digital subtraction angiography

Digital subtraction angiography is another gold standard imaging method for BAO. However, this modality nowadays has been mostly replaced by the other non-invasive imaging techniques previously discussed.

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