Acute basilar artery occlusion: an easily missed uncommon but devastating emergency

WP Chu *, WC Wong, Bill A Lo, KK Lai

Hong Kong Med J 2015;21:375.e1-2
DOI: 10.12809/hkmj154530

A 55-year-old man was admitted in August 2014 as an emergency with sudden onset of vertigo, dizziness, and left-sided weakness. Initial Glasgow Coma Scale score was 15/15. Physical examination revealed left hemiparesis and an upgoing left plantar response. Both pupils were reactive with the left one slightly smaller than the right. Muscle power was grade 4 over 5 for the left upper and lower limbs. Urgent computed tomography (CT) examination of the brain revealed a hyperdense basilar artery, which was initially unnoticed (Fig 1). Subsequently, the patient's level of consciousness rapidly decreased and intubation was required. Urgent magnetic resonance angiography (MRA) identified loss of flow-related signals along the basilar artery (Fig 2). Diffusion-weighted imaging (DWI) found restricted diffusion at the pons and bilateral cerebellar hemispheres (Fig 3). The brainstem appeared normal on the T2-weighted images and there was loss of flow void at the basilar artery (Fig 4). These neuroimaging findings were consistent with acute occlusion of the basilar artery, cytotoxic oedema at the brainstem and bilateral cerebellum. The patient died a week later despite intensive medical intervention.

Acute basilar occlusion is a true neurological emergency. Early diagnosis and treatment are essential to prevent brainstem infarct and death. It is uncommon and accounts for 1% of all strokes.[1] Nonetheless, when present, a hyperdense basilar artery is evident on non-contrast CT images in approximately 65% of patients[2] and enables the diagnosis to be confirmed. Hyperdensity at the occluded basilar artery is due to an intraluminal blood clot and is analogous to the 'hyperdense middle cerebral artery sign' of acute thromboembolism of middle cerebral artery.

A very high index of suspicion is required because CT findings can be subtle. Diagnosis requires careful scrutiny of the basilar artery and the posterior circulation. Hyperdense basilar artery may be the only sign before development of an established infarct. Pitfalls to diagnosis include vascular wall calcification secondary to atherosclerosis, partial volume averaging, haematocrit elevation, and vessel dilation. Meticulous evaluation of the CT images of thin collimation and narrowed window, careful comparison of the density of the basilar artery with other intracranial vessels and previous CT images, if available, will be helpful. A blood clot within the basilar artery will present as a hyperdense intraluminal filling defect. Vascular calcification may present as rim or curvilinear peripheral hyperdensity. In patients with hemo-concentration, there should be generalised increased attenuations of the intracerebral vasculature instead of focal abnormality. Both magnetic resonance imaging (MRI) and MRA can demonstrate the extent of vascular occlusion and the secondary changes including cytotoxic oedema

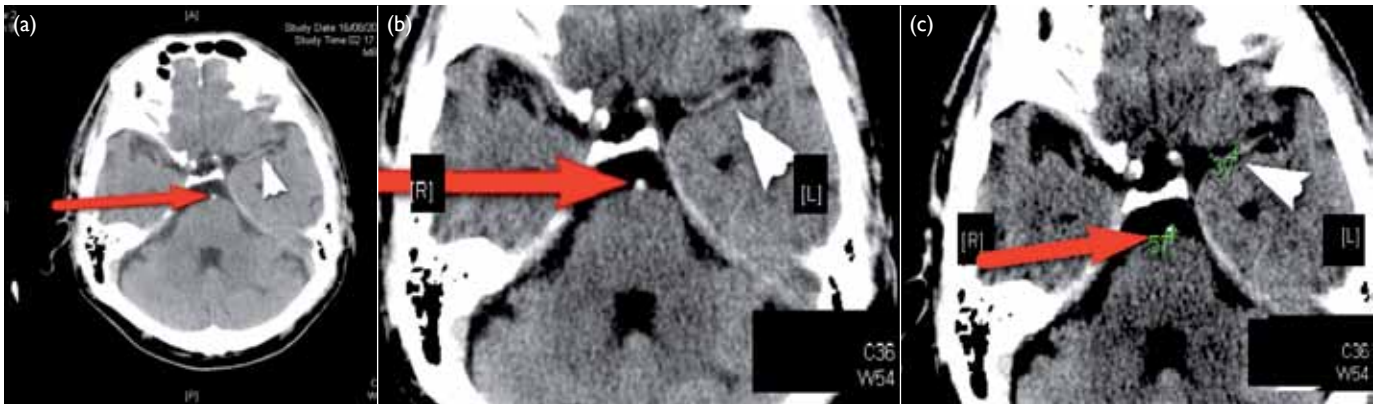

FIG 1. (a) An axial non-contrast computed tomography (CT) brain with usual viewing window (width 90; centre 30) shows hyperdense basilar artery (red arrow) and normal attenuation of the M1 segment of the left middle cerebral artery (MCA; white arrowhead). (b) The same CT image is magnified with its window adjusted (width 54; centre 36). Hyperdense basilar artery (red arrow) is better delineated with greater contrast with the left MCA (white arrowhead). (c) A magnified non-contrast CT brain with narrowed window shows the Hounsfield unit of the basilar artery measured 57 (red arrow) while that of the left middle cerebral artery measured 37 (white arrowhead)

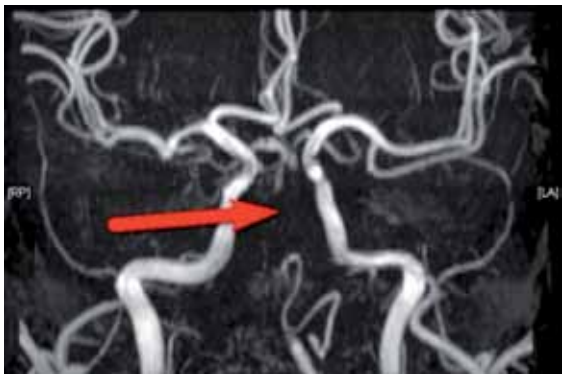

FIG 2. Time-of-flight magnetic resonance angiography shows absence of flow-related signal along the basilar artery (red arrow), highly suggestive of occlusion

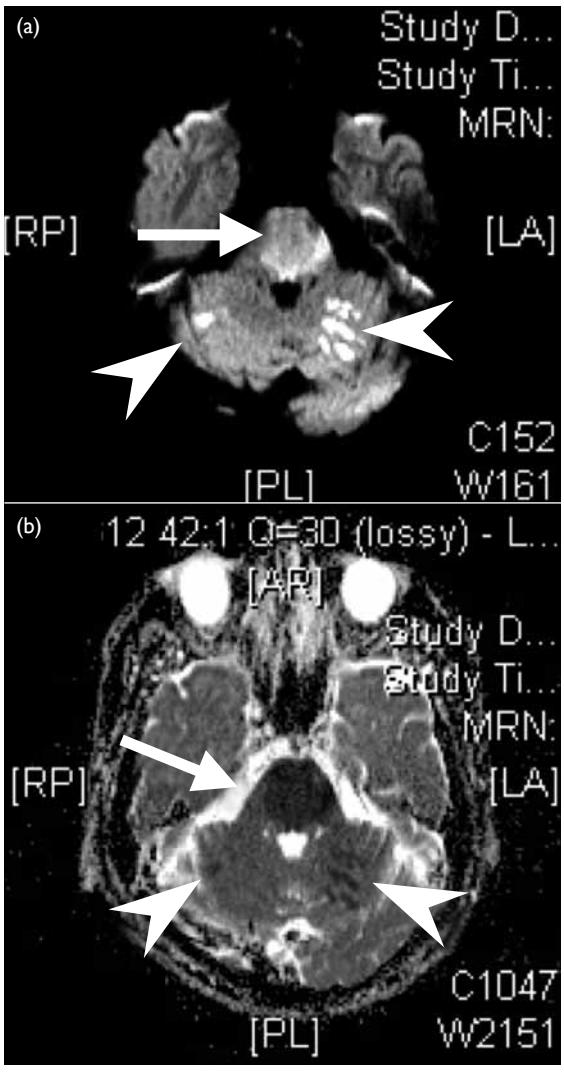

FIG 3. (a) Diffusion-weighted imaging (b value=1000 s/mm^2) shows hyperintense lesions at the pons (arrow) and bilateral cerebellum (arrowheads). (b) The apparent diffusion coefficient map shows low signals at the corresponding sites. Together with the clinical course and the magnetic resonance angiography findings, the overall picture is consistent with acute infarcts at the posterior circulation

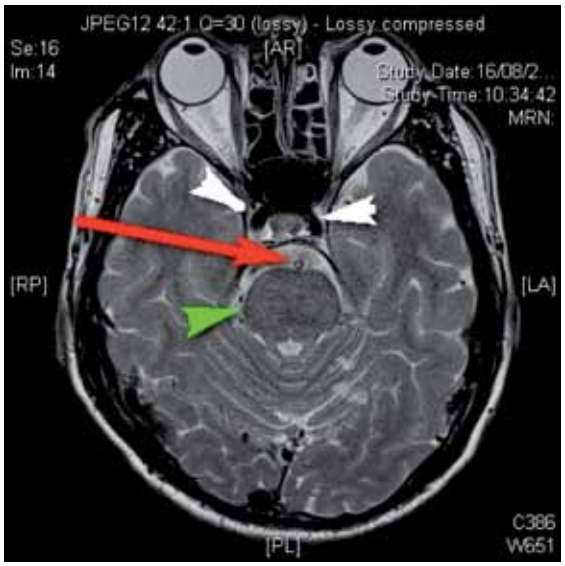

FIG 4. An axial T2-weighted turbo spin echo image of the brainstem shows loss of flow void at the basilar artery (red arrow)

Note the normal flow void (dark) bilateral cavernous portion of the internal carotid arteries (white arrowheads). No abnormal signal was detected at the pons (green arrowhead)

for patients with diagnostic uncertainty. Limited sequences, including time-of-flight MRA and DWI, may be performed within 15 minutes. Of note, DWI is the most sensitive MRI technique to detect cytotoxic oedema before radiological changes are evident on other MRI sequences. Close collaboration between the neurologists, the neuro-interventional radiologists, and neurosurgeons is essential for the management of such patients. Treatment options include intravenous thrombolysis, catheter-directed intra-arterial thrombolysis, and endovascular mechanical thrombectomy. The best approach, however, needs to be defined by future large-scale studies.

WP Chu *, FRCR, FHKAM (Radiology)
WC Wong, FRCR, FHKAM (Radiology)
BA Lo, FRCR, FHKAM (Radiology)
KK Lai, FRCR, FHKAM (Radiology)

Department of Radiology, Tseung Kwan O Hospital, Tseung Kwan O, Hong Kong

* Corresponding author: drvictorchu@yahoo.com

References

1. Goldmakher GV, Camargo EC, Furie KL, et al. Hyperdense basilar artery sign on unenhanced CT predicts thrombus and outcome in acute posterior circulation stroke. Stroke 2009;40:134-9.
2. Mattle HP, Arnold M, Lindsberg PJ, Schonewille WJ, Schroth G. Basilar artery occlusion. Lancet Neurol 2011;10:1002-14.