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. Acute basilar artery is evident on non-contrast CT images in approximately 65% of patients 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.
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.

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