suggestive of ataxic hemiparesis. In addition, MRI sequences clearly showed that the small deep lesion was restricted to the posterior limb of the internal capsule without involvement of the thalamus. Based on previous observations regarding ataxic hemiparesis and thalamic ataxia together with the 2 previously reported cases of isolated hemiataxia after capsular lacunar infarct, we strongly believe that in our patient, hemiataxia was more likely due to a lesion of the cerebellar pathways, either the ascending dentatorubrothalamocortical tract or the descending corticopontocerebellar pathway at the level of the posterior limb of the internal capsule. Functional MRI, PET or SPECT would have been of great interest in this patient to demonstrate CCD and thus assess our hypothesis. Such investigations were, unfortunately, not performed.

To conclude, as previously suggested by Luijckx et al. [1,2], as well as discussed in anatomical studies [21–23], the case reported here provides further clinical evidence of the anatomically segregated passage of the cerebellar fibres through the posterior part of the posterior limb of the internal capsule. Interruption of the cerebellar connections in the internal capsule may therefore cause isolated hemiataxia of the cerebellar type as observed in our patient. The determination of whether the descending or ascending pathway is severed and responsible for the cerebellar symptoms appears difficult. However MR diffusion tensor imaging may provide the answer to this question by studying the development of a descending or ascending wallerian degeneration in a given patient with an infarction in the posterior limb of the internal capsule.

References

Viktoria Kapina, MD
Department of Neurology
HUG, University Hospital and Medical School of Geneva
Rue Michelli-du-Crest 24
CH–1211 Geneva 14 (Switzerland)
Tel. +41 79 538 4644, Fax +41 22 751 2301
E-Mail viktoria.kapina@hcuge.ch

Cerebrovasc Dis 2008;25:596–598
DOI: 10.1159/000134377

Floating Basilar Artery: An Interesting Clinical Dilemma
Jamie J. Van Gompel a, C.C. Lin a, Irene Meissner a
Departments of aNeurology and bNeurosurgery, Mayo Clinic, Rochester, Minn., USA

Case Report
Our patient was a 79-year-old right-handed female. She presented with an event of speech and language disturbance, with right-sided weakness and numbness. Computed Tomography (CT) was performed in preparation for possible tissue plasminogen activator (tPA) administration but the event resolved completely within an hour. While being interviewed after admission, the patient had a second spell. She abruptly developed dysarthria, which progressed with right hemiplegia. At 10 min, her right-side function began to return and the event had resolved in 30 min. Magnetic resonant angiography (MRA) revealed minimal backfiling into the basilar artery apex (fig. 1B). Digital subtraction angiography (DSA) did not visualize the basilar artery (fig. 1A).
The following morning, the patient developed internuclear ophthalmoplegia, and left-sided weakness that persisted. A computer tomographic angiography (CTA) confirmed an isolated perfused midbasilar segment through collateral flow with complete proximal and distal occlusion (fig. 1C–E), which we referred to as a ‘floating basilar’ artery. A repeat MRI revealed restricted diffusion in the right dorsolateral midbrain. The patient was discharged to a rehabilitation facility on warfarin to an INR goal of 2.5–3.5.

Discussion

To our knowledge, this is first reported case of coexisting tandem proximal and distal basilar artery occlusion (BAO) [1–7]. However, we suspect a ‘floating basilar’ segment is more common. Further, this case illustrates that CTA may be the imaging modality of choice in posterior circulation disease, here identifying an isolated perfused midbasilar segment. Finally, most studies concerning BAO were published before tPA and this case illustrates the clinical dilemma one faces with chronic BAO in the setting of acute deterioration.

It has become increasingly evident that CTA may offer a better evaluation of the posterior circulation than DSA in the case of basilar occlusive disease [2]. Here, CTA demonstrated the presence of the proximal and distal BAO, as well as persistent perfusion of the mid-portion basilar (fig. 1C), which is not seen on other imaging modalities. The value of CTA seems to be mostly in evaluating vessel patency in modalities that are flow dependent, where critical stenosis or occlusion may produce false negative results with DSA or MRA [2]. The finding of persistent flow in the posterior circulation is of clinical importance as it may dictate the duration and intensity of anticoagulation and the feasibility of neurosurgical revascularization.

The temporal profile of BAO may significantly affect clinical presentation and prognosis. Acute BAO is considered generally incompatible with independent living and atherosclerotic occlusion fares better than embolic occlusion. The literature suggests that patients with more protracted occlusion development have a better prognosis and clinical outcome, due to development of collateral flow [3, 8]. Although our patient’s presentation technically met criteria for emergent tPA administration, this therapy was unlikely to have provided clinical benefit.

In conclusion, chronic BAO is a difficult clinical dilemma, such as in this case. However, if one suspects posterior circulation disease, one may consider CTA as an important adjunct in imaging. Overall, there is much to be learned about this disease and our case highlights the need for further investigation.
References


Jamie J. Van Gompel, MD
St. Mary Hospital, Mayo Clinic
1216 2nd St. SW Joseph 1-229
Rochester, MN 55902 (USA)
Tel. +1 507 284 2511, Fax +1 507 255 7988
E-Mail Vangompel.Jamie@Mayo.edu