Conclusion

These 2 case reports emphasize the difficulties of the neuro-radiological diagnosis and of the decision-making for the management of hemorrhagic dissection of the ACA. The second patient was surgically treated but, unfortunately, the wrapping of the dissected arterial wall could not prevent the recurrence of the hemorrhage. On the other hand, the follow-up showed that the dissection tends to heal spontaneously with restoration of a normal arterial lumen and that the conservative treatment was effective with a good outcome in both cases. The literature review (table 1) and our own experience seem to suggest that this therapeutic option could be safely proposed for the management of hemorrhagic dissections of the ACA.

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Increased Oxygen Extraction Demonstrated on Gradient Echo (T2*) Imaging in a Patient with Acute Ischaemic Stroke

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Magnetic resonance (MR) imaging is sensitive to the oxidative state of haemoglobin, as oxy- and deoxyhaemoglobin have different magnetic properties – the blood oxygen level dependent (BOLD) effect. Indeed, functional MR imaging techniques are sufficiently sensitive to the BOLD effect that they can be used to assess cerebrovascular reactivity with a simple breath-holding test or CO₂ challenge [1].

Fig. 1. MR imaging in a patient at 2¼ h after a severe left hemisphere stroke. **a** DWI shows a small hyperintense lesion in the deep left hemisphere white matter (arrow). **b** T2*-weighted image (BOLD) shows a large hypointense area in the left parietal cortex and white matter in the left middle cerebral artery territory suggesting increased oxygen extraction (arrows). **c** Single image from the pre-contrast phase of the PWI sequence showing a subtle low signal area similar to but less obvious than that in **b** (arrows). **d** Non-contrast CT brain scan obtained 10 min after the MR shows hypoattenuation in the left basal ganglia and white matter and some sulcal effacement.

Therefore, in acute ischaemic stroke, an increase in the oxygen extraction fraction (OEF) in ischaemic but still viable tissue [2] could be visible on sequences sensitive to the BOLD effect, such as T2*. A negative BOLD signal was seen in an experimental model at 1.5 T when CBF fell acutely by at least 70% from normal [3]. The only clinical report of this is in a retrospective study of averaged base images from dynamic susceptibility contrast (DSC) perfusion-weighted imaging (PWI), i.e. immediately prior to the contrast bolus arrival, in 5/6 patients imaged within four hours of acute ischaemic stroke distal to an internal carotid artery (ICA) occlusion [4]. However, while it was possible to see a diffuse ill-defined negative BOLD signal in the hemisphere above the ICA occlusion, these images were of insufficient quality to demonstrate a clearly-demarcated negative BOLD signal lesion. There have been no reports of negative BOLD signal lesions on formal T2*-weighted images, i.e. of a quality sufficient to demonstrate the extent of a lesion, in acute stroke. We observed a negative BOLD effect on T2*-weighted MR imaging in the following case.

An 88-year-old woman was admitted to hospital 1 h 55 min after a severe stroke (National Institute of Health Stroke Scale Score (NIHSS) = 27). The patient had ischaemic heart disease but no history of hypertension or respiratory disease. She received supplemental oxygen because of the severe stroke despite oxygen saturation of 98%.

On MR diffusion-weighted imaging (DWI) at 2 h 45 min after stroke (spin-echo echo planar diffusion tensor MRI, $b = 0$ and 1,000 s/mm², 6 non-collinear diffusion gradient directions, 15 axial slices, 5 mm thickness, 1.0 mm slice gap, field-of-view 240 × 240 mm, matrix 256 × 256, TR 10, TE 98.8 ms), there was a small hyperintense area in the left cerebral hemisphere deep white matter (fig. 1a). No acute abnormality was visible on T2-weighted fast spin echo imaging (not shown). On T2* imaging (TR 620, TE 15, GR 20), there was reduced signal in the deep and superficial parts of the left parietal lobe (fig. 1b), considerably larger than the DWI hyperintensity. PWI with standard DSC acquisition technique was severely degraded by movement (not shown), but the images from the PWI sequence prior to contrast bolus arrival (fig. 1c) showed a subtle low signal area, similar in extent but much less obvious than that on T2* imaging. MR angiography was not performed as the patient was too restless, but a flow void was present in the left middle cerebral artery main stem on T2 and T2* imaging, with no increased signal to indicate acute thrombus. A CT scan performed 10 min after MR showed effacement of the left parietal cortical sulci, hypoattenuation in the left basal ganglia and parietal white matter (fig. 1d), and no hyperattenuated artery.

The patient's oxygen saturation (transdermal finger probe) fell from 97% at the start of scanning to 94% when the scan was

stopped, despite supplemental oxygen at 4 l/min. The patient deteriorated and died rapidly from the severe stroke before follow-up imaging or carotid ultrasound could be performed.

The negative BOLD signal lesion should suggest tissue where there is increased oxygen extraction, so it is surprising that the BOLD effect has not been seen more widely in acute ischaemic stroke. A blood flow reduction of >70% (from 58 to 17 ml/100 mg/min) was required to produce measurable BOLD effects (shortening of T2) in well-oxygenated rats, although this did not produce any change on diffusion-weighted imaging [3]. Such flow reductions occur commonly in acute ischaemic stroke. We unfortunately did not obtain reliable PWI or any MRA data in this patient, although there was no evidence on axial imaging of an acutely thrombosed left MCA.

The other notable feature in our patient was the low oxygen saturation while in the scanner, which might have influenced the haemoglobin desaturation in the area of the stroke lesion, thus increasing the BOLD effect. However, this patient was imaged very early after the stroke, perhaps simply at the right moment to see increased oxygen extraction. The relatively small DWI lesion, in contrast to the high NIHSS score, would suggest that the lesion was imaged early in its evolution, but is also consistent with experimental data showing blood flow reduction affecting the BOLD signal before any DWI abnormality is visible [3].

Should there be increased oxygen extraction in the DWI lesion itself? Experimental models [5] and human studies [6–8] show that the DWI-abnormal lesions include viable tissue with areas of increased oxygen extraction [7].

We perform T2*-weighted imaging routinely for our acute stroke assessment to exclude haemorrhage, but have not observed the BOLD effect so convincingly in other patients. Although limited T2*-weighted images are commonly acquired as base images during DSC PWI (fig. 1c) in stroke imaging centres, they might not be reviewed, and it is not known how often formal T2*-weighted imaging is used. We routinely administer oxygen to severe strokes while in the MR scanner, thus usually maintaining reasonable oxygen saturation, unlike in this patient. In the 5/6 patients described previously, the oxygen saturation was not mentioned [4], and there are no experimental data on how oxygen

saturation might influence the perfusion change required to produce a BOLD signal [3].

We conclude that T2*-weighted images may have a role in determining 'tissue at risk' and should perhaps be examined more systematically, as seeing a negative BOLD signal lesion could add to acute stroke assessment.

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