Bilateral Hypoperfusion and Normal Diffusion MR Images in a Case of Acute Embolic Stroke

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A 56-year-old man with a history of paroxysmal atrial fibrillation was referred for sudden mutism and right hemiplegia. Improvement of the motor deficit occurred during transportation. Brain MRI 100 min after onset showed hyperintense middle cerebral artery (MCA) branches bilaterally on FLAIR sequence, and T₂* hypointensities in the M1 segment of the left MCA and in a right insular branch corresponding to fresh thrombi. MR angiography revealed a left carotid and M1 occlusion. No parenchymal

bleeding was seen, apart from a left parietal microbleed. DWI was normal while apparent diffusion coefficient (ADC) maps revealed a small region of ADC restriction (20%) in the left deep MCA territory. Bolus tracking perfusion-weighted images (PWI) showed bilateral large abnormally perfused areas: (1) in the left MCA territory (mean transit time = 133%, cerebral blood flow = 56% of normal-appearing right frontal area), corroborating the neurological deficit, and (2) in the right temporo-occipital region (mean transit time = 126%, cerebral blood flow = 54%) (fig. 1).

After MRI completion, global aphasia persisted (National Institute of Health Stroke Scale score = 5) and the heart rate was irregular. The laboratory tests were normal. Based on the expected language handicap in case of infarct completion and on the presence of at-risk tissue in the contralateral hemisphere, intravenous thrombolysis (6 mg of rt-PA bolus followed by infusion of 52 mg of rt-PA in 1 h) was initiated 160 min after onset. Follow-up MRI showed recanalization of the left MCA and bilateral regression of early vessel signs on FLAIR. The carotid occlusion persisted. A left deep MCA infarct and a right small insular infarct were visible, without cerebral bleeding. The language deficit progressive-

Fig. 1. a Before thrombolysis: 100 min after stroke onset, bilateral hyperintense vessel signs in distal branches of the MCA (light arrows) without parenchymal signal changes on FLAIR and DWI. Focal ADC decrease in the deep MCA territory (black arrow). Decreased regional cerebral blood flow (rCBF) and increased

mean transit time (MTT) on PWI with left carotid and proximal MCA occlusion on MR angiography. **b** After thrombolysis: on the 24-hour MR follow-up, recanalization of the left MCA with a deep left MCA infarct and a right insular infarct.

ly improved with a modified Rankin score of 1, one month after hospital discharge.

This case highlights the fact that DWI can be normal in acute ischemic stroke even 2 h after stroke onset. ADC maps should then be examined to search for subtle ADC decrease. The decision to treat was taken after weighing the potential benefit of thrombolysis on bilateral penumbras against the risk of intracranial hemorrhage in a patient with rapid early neurological improvement. Up to one third of the patients eligible for intravenous thrombolysis, with early neurological improvement, experience severe subsequent deterioration if not treated [1–3]. This suggests that intravenous thrombolysis should not necessarily be withheld solely because of regressing neurological symptoms. The decision to treat was favored by the persistent disabling language deficit, and by the MR pattern: lack of DWI signal changes (DWI-clinical mismatch) [4] and bilateral perfusion defect (bilateral PWI-DWI mismatch). These suggest that, 2 h after onset, most of the ischemic parenchyma represented penumbras. This case also illustrates the usefulness of multimodal MRI to detect distal embolic thrombi. Bilateral PWI defects were easy to identify, whereas the subtle brightness of right MCA branches on FLAIR and T_2^* signal drop in distal arteries can be overlooked, especially when clinical data do not point to the contralateral hemisphere.

References

- 1 Baumann CR, Baumgartner RW, Gandjour J, von Budingen HC, Siegel AM, Georgiadis D: Good outcomes in ischemic stroke patients treated with intravenous thrombolysis despite regressing neurological symptoms. *Stroke* 2006;37:1332–1333.
- 2 Cocho D, Belvis R, Marti-Fabregas J, Molina-Porcel L, Diaz-Manera J, Aleu A, Pagonabarraga J, Garcia-Bargo D, Mauri A, Marti-Vilalta JL: Reasons for exclusion from thrombolytic therapy following acute ischemic stroke. *Neurology* 2005;64:719–720.
- 3 Smith EE, Abdullah AR, Petkovska I, Rosenthal E, Koroshetz WJ, Schwamm LH: Poor outcomes in patients who do not receive intravenous tissue plasminogen activator because of mild or improving ischemic stroke. *Stroke* 2005;36:2497–2499.
- 4 Lansberg MG, Thijs VN, Hamilton S, Schlaug G, Bammer R, Kemp S, Albers GW: Evaluation of the clinical-diffusion and perfusion-diffusion mismatch models in DEFUSE. *Stroke* 2007;38:1826–1830.

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