Influence of a severe internal carotid artery stenosis on diffusion and perfusion values in acute stroke patients

Philipp Kaesemann[1], Götz Thomalla[2], Bastian Cheng[2], Andras Treszl[3], Jens Fiehler[4], and Nils Daniel Forkert[5]

[1]*Department of Computational Neuroscience, University Medical Center Hamburg-Eppendorf, Hamburg, Germany,* [2]*Department of Neurology, University Medical Center Hamburg-Eppendorf, Germany,* [3]*Department of Medical Biometrics and Epidemiology, University Medical Center Hamburg-Eppendorf, Germany,* [4]*Department of Diagnostic and Interventional Neuroradiology, University Medical Center Hamburg-Eppendorf, Germany,* [5]*Department of Radiology and Hotchkiss Brain Institute, University of Calgary, Calgary, Alberta, Canada*

Purpose: Acute stroke patients with an artery occlusion and coexisting stenosis may be affected by a more severe hypoperfusion situation compared to patients with the same artery occlusion without coexisting stenosis, which may result in a faster stroke progression and also to a worse clinical outcome. On the other hand, a chronic carotid stenosis may also evoke the development of an improved collateral circulation as a compensatory mechanism to overcome the long-lasting perfusion impairment, which may be even beneficial to endure the reduced perfusion situation in case of an acute ischemic stroke. Moreover, a chronic carotid stenosis may also lead to an ischemic preconditioning of the brain tissue with a protective effect as already reported for cardial[1] and cerebral tissue[2]**.** The objective of this study was to compare lesion volumes as well as diffusion and perfusion parameters in patients with an acute ischemic stroke resulting from middle cerebral artery (MCA) occlusion with or without a coexisting internal carotid artery (ICA) stenosis.

Material and Methods: MRI datasets, including diffusion-weighted (DWI) and perfusion-weighted MRI (PWI), from 32 patients with MCA occlusion with or without additional extracranial ICA stenosis imaged within 4.5 hours of symptom onset and prior to treatment were available and analyzed in this study. The ICA stenosis was diagnosed by ultrasound imaging within 5 days after stroke symptom onset according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria[3] applying a threshold > 70%, which is commonly assumed to represent a high-grade stenosis. Both patient groups consisted of 16 patients. The DWI datasets were acquired using TR = 3500 ms, TE = 89 ms, flip angle = 90°, diffusion weightings of 0 and 1000 s/mm², image in-plane resolution = 0.94 mm², and 24 slices of 5 mm thickness. The PWI datasets were acquired with TR = 2000 ms, TE = 45 ms, flip angle = 90°, image in-plane resolution = 0.94 mm², and 24 slices of 5 mm thickness.
Apparent diffusion coefficient (ADC) parameter maps were calculated based on the DWI datasets and used for a semi-automatic segmentation of the acute diffusion lesion using a volume growing approach with an upper threshold of 550 x 10^9 mm²/s and interactively defined seed points. Absolute perfusion maps of cerebral blood volume (CBV), cerebral blood flow (CBF), mean transit time (MTT) and Tmax were calculated using the PWI datasets by deconvolution of the concentration time curve for each voxel with the arterial input function, which was selected from the contralateral MCA, using a block-circulant singular value decomposition and a truncation threshold of 20%. After rigid registration of the perfusion parameter maps to the corresponding ADC dataset, tissue-at-risk (TAR) volumes were generated by subtracting the diffusion lesion from the hypoperfusion lesion defined by Tmax >6s (see Fig. 1). Furthermore, both lesion segmentations were used to extract median ADC and perfusion parameter values separately for the diffusion lesion and tissue-at-risk. Median instead of mean values were extracted to account for the non-normal distribution of the parameter values within the two lesions. A student's t-test was used to test for significant differences of the diffusion lesion and TAR volume between the two groups while a two-way MANOVA was used to compare the diffusion and perfusion parameters for region (DWI lesion vs. tissue-at-risk) and group (M1 vs. M1+ICA) within one model.

Results: No significant differences were found between the groups regarding the diffusion lesion and tissue-at-risk volumes (p = 0.381 / p = 0.724). The statistical analysis of the diffusion and perfusion parameters revealed that the CBV is the only parameter with a significant difference (p=0.009) between patients with and without a coexisting stenosis. In contrast to this finding, all other diffusion and perfusion parameters did not reach statistical significance. More precisely, higher CBV values were found in the diffusion lesion (2.1 ml/100g vs. 2.7 ml/100g) as well as in the tissue-at-risk (3.5 ml/100g vs. 4.2 ml/100g) for the patient group with a coexisting ICA stenosis.

Discussion and Conclusion: The results of this study suggest that an ICA stenosis in acute stroke patients only leads to a significant alteration of the cerebral blood volume while no significant differences were found regarding the lesion volumes or other diffusion and perfusion parameters tested. The elevated CBV situation in acute stroke patients with a coexisting stenosis may be an indicator for an improved collateral circulation or ischemic preconditioning in patients with a pre-existing proximal stenosis. In summary, all typically applied thresholds, except those based on the CBV parameter, for perfusion-based stroke analysis and quantification appear to be also valid in case a coexisting ICA stenosis,

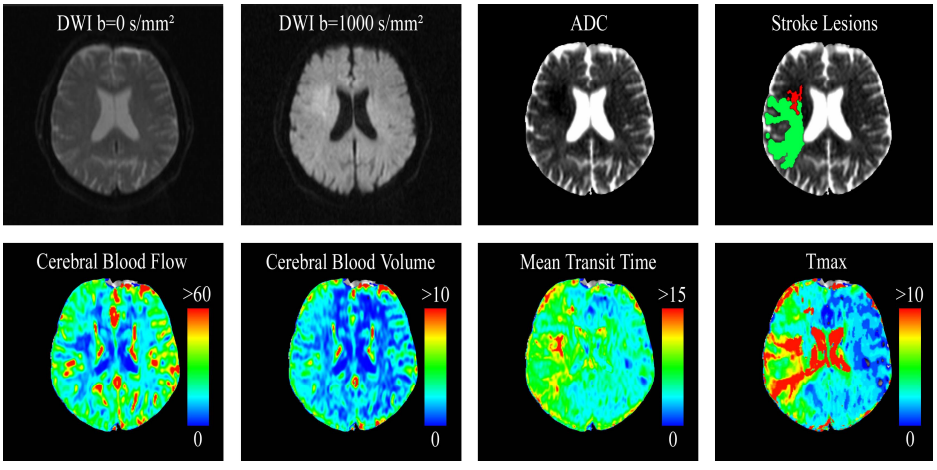

Fig. 1: Selected slice from a DWI dataset and corresponding ADC parameter dataset used for definition of the diffusion lesion (red lesion) as well as perfusion parameter maps CBF (in ml/100g/s), CBV (in ml/100g), MTT (in s), and Tmax (in s). Tmax >6s is also used for tissue-at-risk definition (green

References:

(1) Schmidt MR, et al. Am J Physiol Heart Circ Physiol 2007; 292(4):H1883-90.
(2) Wegener S, et al. Stroke 2004; 35(3):616-21.
(3) Collaborators NASCET. N Engl J Med 1991; 325(7):445-53.