Effects of type II diabetes on cerebral vasoregulation using Continuous Arterial Spin Labeling MRI at 3 Tesla

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Introduction

Cerebral vasoregulation reflects the ability of cerebral microvasculature to adapt to metabolic demands and systemic blood pressure changes and to maintain steady cerebral perfusion. Continuous Arterial Spin Labeling (CASL) perfusion magnetic resonance imaging (MRI) has already proven its ability to measure cerebrovascular reserve[1]. The goal of this study was to determine the effects of type II diabetes (DM) on cerebral vasoregulation using CASL MRI at 3 Tesla.

Materials and method

Twelve healthy subjects (8 men, 4 women, mean age ± SD: 59.5 ± 10.2 years) and twelve subjects with type II DM for more than a year (5 men, 7 women, 58.6 ± 5.2 years) were studied. Controls were not treated for any systemic disease; DM subjects with strokes, cardiac, renal or carotid disease were excluded. MR imaging was performed using a whole-body 3 T MRI scanner (GE Signa Vhi) with quadrature head coil. All subjects had routine T_1-weighted spin echo (T_E/T_R = 3.3/8.1 ms, 24 cm × 19 cm FOV, 256 × 192 matrix size, 3 mm slice thickness), fluid-attenuation inversion recovery (FLAIR) (T_E/T_R = 161/11002 ms, 24 cm × 24 cm FOV, 256 × 160 matrix size, 5 mm slice thickness) and MR Angiography (MRA) (T_E/T_R = 3.9/38 ms, 20 cm × 18 cm FOV, 384 × 224 matrix size, 2 mm slice thickness). Tagged and control images were collected over 5 minutes during: baseline (B_1), CO_2 rebreathing (RB) of air and 5% CO_2, hyperventilation (HV) and a second baseline (B_2). End-tidal CO_2 ($EtCO_2$) was continuously monitored and averaged over 15-second intervals for all conditions. CASL MRI[2,3] was performed with an echo planar imaging sequence (T_E = 31 ms, 24 cm × 24 cm FOV, 64 × 64 matrix size, 5 mm slice thickness). Images were obtained every 8 seconds and averaged for each condition. A perfusion map was then reconstructed for all conditions. A region of interest (ROI) corresponding to the whole brain was selected on each slice on which extracerebral tissue and ventricular areas were carefully extracted. Perfusion (ml/100 g/min) was computed on each ROI. Perfusion differences between B_1 and HV (B_1-HV) and between RB and HV (RB-HV) were also computed. Cerebrovascular reserve (CVR) was computed as the percent of blood flow augmentation during RB compared to blood flow reduction during HV. The percent of $EtCO_2$ change between RB and HV ($\%CO_2$) was also computed. Vascular reactivity (VR) was derived from perfusion maps as percent of flow augmentation during RB compared to flow reduction during HP divided by percent CO_2 change. Two-way ANOVA (group × condition) was used for statistical comparisons.

Results

Figure 1 presents 2 CASL slices at the level of the ventricles for a healthy control and a DM subject. CASL revealed flow augmentation during RB and reduction during HV; RB-HV perfusion difference was blunted in the DM subject compared to the control. Table 1 presents the whole brain perfusion values for the control and DM group, perfusion differences (B_1-HV and RB-HV), CVR, $\%CO_2$ and VR that were averaged by group for all conditions. Absolute perfusion values were significantly different between conditions for both groups. Perfusion differences between control and DM groups did not reach significance. Blood flow reduction (resp. augmentation) between B_1 and HV (resp. RB and HV) was significantly reduced in DM compared to the control group ($p < 0.04$). CVR was reduced in DM compared to the control group ($p < 0.01$), however $\%CO_2$ difference was borderline ($p = 0.055$). VR was not different between groups. Visual inspection of T_1 and FLAIR images revealed mild-to-moderate white matter abnormalities; MRA evaluations were normal for all subjects.

Discussion - Conclusion

Preliminary data suggested that cerebrovascular reserve (perfusion difference between vasoconstriction to vasodilation) is reduced in DM. Age, DM duration and T_1- and T_2-weighted white matter abnormalities may affect small vessel reactivity and contribute to inter-subject variability.

References

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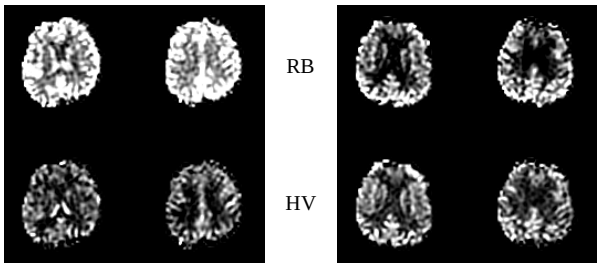

Figure 1: CASL slices for a control (A) and DM subject (B) during CO_2 rebreathing (RB) and hyperventilation (HV).

		Control	DM
Perfusion ml/100 g/min	$Baseline_1$	42.6 ± 13.9	38.7 ± 12.1
	CO_2 rebreathing	52.4 ± 15.9	45.3 ± 16.5
	Hyperventilation	26.5 ± 12.4	29.2 ± 11.2
	$Baseline_2$	39.6 ± 12.8	38.5 ± 12.5
	$Baseline_1$-Hyperventilation	16.1 ± 8.6	9.1 ± 5.3*
	CO_2 rebreathing-Hyperventilation	25.9 ± 9.3	16.1 ± 10.5*
%	CVR	109.8 ± 40.5	62.3 ± 43.1*
	$\%CO_2$	96.6 ± 42.8	66.7 ± 28.1
	VR	1.25 ± 0.54	1.16 ± 0.88

Table 1: Mean values and SD of whole brain perfusion during baseline (B_1), CO_2 rebreathing (RB), hyperventilation (HV), second baseline (B_2), and differences between B_1 and HV (B_1-HV) and between RB and HV (RB-HV), CVR, $\%CO_2$ and VR (*: $p < 0.05$).

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