

Changes in Brain Energy Metabolism after Carotid Artery Stenting by in vivo ^{31}P NMR Spectroscopy

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Introduction

The aim of this study was to evaluate a potential improvement in brain energy metabolism in vivo after carotid artery stent placement (CAS) in patients with high grade symptomatic ICA stenosis. For this purpose we employed ^{31}P and ^1H magnetic resonance spectroscopic imaging (MRSI).

Methods

Twelve patients with high grade ICA stenosis according to NASCET criteria were examined pre-interventional with a subsequent postinterventional scan within a period of 2-4 days. For comparison, 6 healthy subjects were scanned as a control group. MRS of the brain was performed on a 3T whole body system with a double tuned $^1\text{H}/^{31}\text{P}$ volume head coil. For ^1H MRS, a 1.5 cm axial slice including the basal ganglia was recorded with 2D chemical shift imaging (CSI, circular phase encoding on a 24x24 matrix extrapolated to 32x32, 240 mm² FOV, TR 1500 ms, TE 30 ms). A 3D CSI sequence was used for ^{31}P MRS employing circular phase encoding with a weighted acquisition scheme on a 10x10x8 matrix extrapolated to 16x16x8 resulting in an series of axial slice with nominal 3 cm thickness and 17.5x17.5 mm² in plane resolution (TR 2000 ms, TE 2.3 ms). ^1H spectra were analysed using LCModel, ^{31}P spectra were analysed with jMRUI. The tissue pH was based on a titration curve obtained from a model solution using the chemical shift difference between the inorganic phosphate (PI) and the phosphocreatine (PCr) signal. The signal intensities of PCr from ^{31}P MRS and the sum of creatine and phosphocreatine (tCr) were assumed to serve as an indicator for the status of energy metabolism while N-acetylaspartate (tNAA) may be an indicator of neuronal damage in the area ipsilateral to the stenosis. For the ^{31}P MRSI data eight voxels, 4 from each side for a bilateral comparison, were analyzed in terms of total average of metabolite concentration and tissue pH. Respective brain regions were identified on the ^1H metabolic maps and averaged as well.

Results.

For all metabolite of interest the signal intensity could be assessed with an accuracy better than 15%. Results comparing ipsilateral and contralateral tissue prior and following stent implantations are summarized in the Figure. The last symbol in each panel represents the data from healthy controls where ipsilateral and contralateral hemispheres were averaged. The asterisk marks significant changes compared to control. Prior to stent implantation, the tissue pH of the ipsilateral side was significantly increased. Regarding metabolite concentrations, ipsilateral tNAA is decreased compared to the controls while all other changes did not reach the level of statistical significance ($p < 0.05$).

Discussion

The decrease in tNAA clearly indicates neuronal damage in the hemisphere ipsilateral to stenosis, which is probably the result of a sustained perfusion deficit. This tissue reveals an increased pH, which seems to be reversed upon stent implantation. Such a finding contradicts the hypothesis that hypoperfusion induces acidosis due to increased anaerobic glycolysis. However, the examination was performed with patients at rest and may indicate an overcompensation under this condition. Such a rationale would be consistent with the absence of a significant change in the PCr/tCr ratio.

Conclusion

^{31}P MRSI shows a reversible increase in tissue pH of the ipsilateral hemisphere. This rather reflects an overcompensation in the resting brain than the actual perfusion deficit.

