31P and 1H MR spectroscopic studies on changes of cerebral brain metabolism induced by alcoholism and detoxification

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Introduction

Alcohol has various effects on the nervous system, in particular on the neurotransmitter regulation. These effects depend on the duration of excessive alcohol consumption. Changes in the lipid membrane of neuronal cells were reported for chronic high alcohol consumption, while cessation of alcohol consumption causes psychic, cognitive, and vegetative withdrawal syndromes which may be related to dysfunctional release of neurotransmitters. Since MRS is discussed as a surrogate marker for dysfunctional neurotransmitter regulation it has been used in several studies. A vast of publications report ^{1}H-MRS detected metabolic changes (e.g.: Ende et al., Biol. Psych. 2005, Neuroimage 2006, Proceedings ISMRM, 2008; Durazzo et al., Alcohol Clin Exp Res. 2006). Two publications which used ^{31}P-MRS (Estelai et al., Alcohol Clin Exp Res. 2001) report a change in the broad phospholipid hump. Since more specific information about phospholipid metabolism is available with proton decoupled ^{31}P MRS we have used ^{31}P and ^{1}H MRS to study cerebral metabolic changes. To obtain reference data, 12 volunteers were also examined.

Methods

29 patients who underwent a detoxification therapy were included in the study. In addition to several clinical tests which were performed during treatment (e.g. DEMTECT, AWS) the patients received MRS examinations at day 1 and day 7 (final day). ^{31}P and ^{1}H MRS was performed and data were analysed from the areas marked in Figure 1. The protocol included T1-weighted anatomical data (MPRAGE, 1x1x1 mm), 31P-3D-CSI (10x10x8 extrapolated to 20x20x16, FOV 300x300x200 mm) and 2 slices 1H-2D-CSI (16x16 extrapolated to 32x32, FOV 240x240 mm). Spectroscopic data were registered to the anatomy by low resolution anatomical data recorded with same location and angulation as the spectroscopic data. Spectra were analysed with jMRUI for ^{31}P data or LCModel for ^{1}H data. Signal intensities were corrected for T1 and T2 relaxations. Metabolite concentrations were obtained using the phantom replacement method. The partial volume of CSF was taken into account as covariate when comparing data from day 1, day 7, and healthy volunteer (Fig.2). A non-parametric correlation (Spearmans R) of the clinical data and CSF corrected metabolite concentrations was performed to measure effects of alcohol withdrawal.
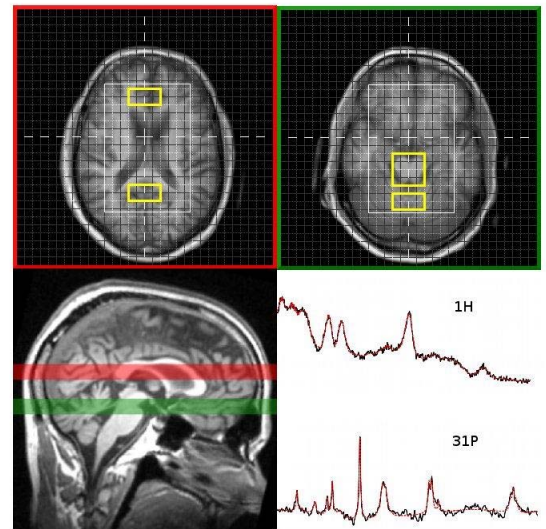

Fig.1: ROIs and typical spectra. The colours mark respective slices.

Results

All areas showed a decreased tNAA and TMA in patients compared to controls corroborating already published data. The TMA decrease is also seen in the ^{31}P data and is paralleled by a decrease in the ethanolamine based membrane compound. Most prominent changes were seen for phosphoethanolmin (PEth) in the frontal region. This region revealed significant correlation between AWS-score and metabolite changes as shown in Table 1 for the neuronal marker NAA, the membrane compound phosphoethanolamine and the high energy metabolite ATP. The negative correlation between NAA and the total score of alcohol withdrawal symptoms indicates that a recovery in NAA, which has also been reported in literature (e.g. Ende et. al, Proceedings ISMRM 2008) is more pronounced in patients with less severe symptoms during withdrawal. The positive correlation for PEth and ATP suggest that patients with higher concentrations of the respective metabolite, i.e. with less severe symptoms regarding metabolite concentrations, suffer more severely from the withdrawal symptoms.

	Day 1		Day 7	
	AWS-T	AWS-P	AWS-T	AWS-P
NAA	0	0	-	0
PEth	+	+	0	0
ATP	+	+	+	+

Table 1: Correlations between Alcohol Withdrawal Score (AWS) and metabolite concentrations. +/- indicate positive/inverse correlations. AWS-P score: psychic syndromes, AWS-T score: sum of psychic and vegetative syndromes.

Discussion

We found significant correlations of the AWS scores with MRS detectable metabolic changes in the frontal brain during alcohol withdrawal. It seems that less severe symptoms favour neuronal recovery. On the other hand, patients with less severe "damage" in membrane and energy metabolism experience more severe symptoms during withdrawal.