Frontal lobe and cerebellar choline signals in alcoholic patients increase with abstinence

G. Ende[1], S. Walter[1], H. Welzel[1], W. Weber-Fahr[1], A. Heinz[1], K. Mann[1]

[1]Central Institute of Mental Health, Mannheim, Germany

We performed a longitudinal ^{1}H multislice MRSI study of the frontal lobe and cerebellum in alcoholic patients and healthy controls. CSF-corrected metabolite values are reported. We found decreased signals of choline containing compounds in the cerebellum of alcoholics compared to controls and increasing signals of choline containing compounds in frontal lobe white matter and the cingulate gyrus of abstinent patients. No changes in NAA could be detected, neither in the longitudinal design nor in comparison with healthy controls.

Introduction

Structural brain damage as well as its partial reversibility in abstinence are well documented in chronic alcohol abuse. There is also evidence for brain metabolic abnormalities in this condition, there are reports of partially reversible decreases of NAA and choline containing compounds (1,2). This study focuses on metabolic alterations of the cerebellum and the frontal cortex in alcoholics at the beginning of withdrawal and after three months of abstinence in comparison with matched healthy controls detected by use of a proton multislice spectroscopic imaging sequence (MSSI). These investigations are part of a longitudinal study of metabolic changes and their time course in alcoholics before and during withdrawal supported by the Deutsche Forschungsgemeinschaft (DFG).

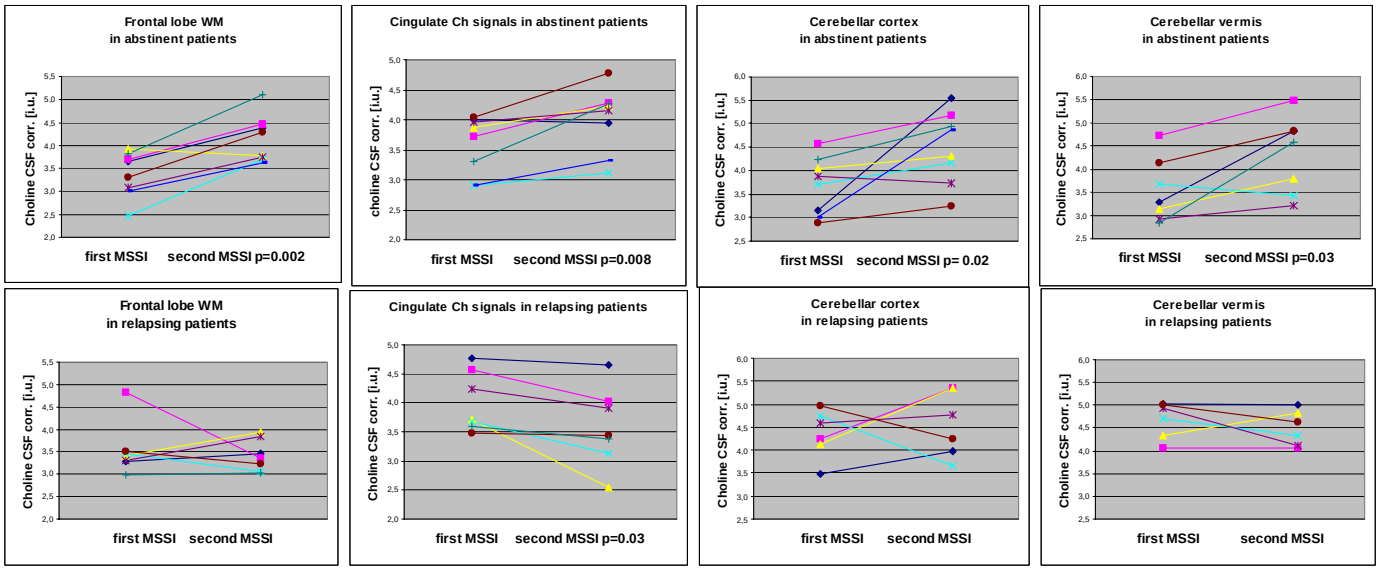
Subjects and Methods

All ^{1}H MSSI studies were performed on a 1.5 T Siemens Vision MRI/MRS system. Two axial planes of ^{1}H MSSI data were collected using a single spin-echo multislice sequence (TE 135/TR 1500), a 36x36 matrix size (nominal resolution of 7.5 mm in-plane), with a slice thickness of 15 mm and a non-selective inversion pulse for lipid suppression. The first ^{1}H MRSI plane was positioned through the cerebellum and pons using anatomical landmarks for reproducible slice positioning and the second slice superior to the lateral ventricles including frontal lobe gray and white matter and anterior cingulate gyrus. Data from two time points of fifteen patients and eight controls have been evaluated so far. Eight of these patients had been completely abstinent whereas seven patients continued to drink alcohol or had a relapse. Voxels were selected from the cerebellar vermis, the dentate nucleus, the cerebellar cortex, frontal lobe white and gray matter, as well as the anterior cingulate gyrus. With use of an automated image coregistration and segmentation program (3) all MRSI voxels were corrected for the CSF content as well as the individual point spread function. For evaluation of the repeated study of the same subject both data files were visualized at the same time for selection of voxels from identical positions.

Results

At the beginning of alcohol withdrawal we could not corroborate previously reported decreased NAA signals in alcoholic patients. We found a significant reduction of the choline signal in the cerebellar cortex, cerebellar vermis and dentate nucleus region in the patients compared to controls. A similar trend but no significance was found for frontal lobe Ch signals.

Identical voxel positions were chosen from the second MSSI data sets. In a paired t-test the NAA signals in patients and controls were again found unchanged. The Ch signal from the cerebellar cortex, cerebellar vermis, frontal lobe white matter and from the cingulate gyrus in the abstinent patient group increased significantly whereas it was unchanged in healthy controls. Furthermore, in relapsing patients the Ch signals in the cingulate gyrus was significantly decreased but unchanged in the other regions.



Discussion

The absence of NAA changes implies that there is no measurable neuronal loss or dysfunction in alcoholics. Neverthelss, other studies report a (reversible) NAA decrease in alcoholics (1,2). Our results of reversible choline signal changes support the hypotheses of an altered cerebral metabolism of lipids in membranes or myelin in these patients. We will further investigate the correlation between the Ch signal and reversible atrophy in abstinent in alcoholics.

References

1. Seitz D, Widmann U, Seeger U, et al. [1999] Clin Exp. Res., 23:158-163
2. Schweinsburg BC, Taylor MJ, Alhassoon OM, et al. [2001] Alcohol Clin Exp Res 25:924-934
3. Weber-Fahr W, Ende G, Braus DF, et al. [2002] Neuroimage 16:49-60