1H METABOLITE LEVEL CHANGES IN CERTAIN BRAIN STRUCTURES DURING MILD TRAUMATIC BRAIN INJURY

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Introduction

Mild traumatic brain injury (mTBI) is the most common neurotraumatologic disorder (70-90 %) with rarely any pathologic manifestation in magnetic resonance imaging (MRI)¹. However, diffuse changes of brain metabolites like N-acetylaspartate (NAA), creatine (Cr) and ratios of NAA to Cr and choline (Cho) were found in mTBI patients^{2,3}. The aim of our study was to correlate these changes in certain parts of the brain to patients' clinical cognitive scores and possible state of unconsciousness.

Patients and Methods

Twenty-one mTBI patients and the same number of sex- and age-matched healthy volunteers participated in our study. All volunteers and patients (on day 2 to 3 after their injury) underwent a neuropsychological examination and a 1.5 Tesla MR imaging and spectroscopy session. Standard clinical MRI protocol (to exclude patients with MRI-visible pathology) was followed by 2D MR spectroscopic imaging in axial cross-section of the cortex (11x11x20 mm voxels, TE/TR=135/1500 ms) and three single-voxel spectroscopy (SVS) measurements located in the frontal grey and white matter bilaterally, and in the upper part of the brain stem (5 to 12 ml voxels, TE/TR=135/4000 ms). Data were evaluated using LCModel, in case of SVS with water scaling.

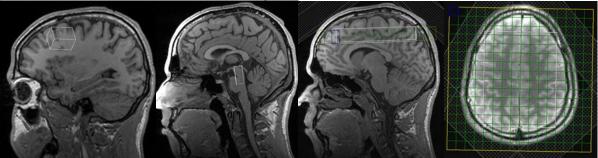


Fig.1: Spatial localization of all the volumes of interest in our MR spectroscopy study (from left to right): frontolateral SVS voxel, brain stem SVS voxel, CSI grid superposed in a saggital; and in an axial MR image.

Results

SVS examinations have shown significantly lower NAA in mTBI patients compared to healthy volunteers in their left ($p_{wilcoxon}=0.030$) and right ($p_w=0.008$) frontal lobes. In the brain stem voxel, this decrease was only significant for the subgroup of 7 patients with unconsciousness ($p_w=0.018$).

NAA and NAA/Cr ratios in the frontal lobe SVS of the patients were significantly positively correlated to a few of the clinical cognitive performance scores (Wechsler memory scale, Stroop and other tests, p ranging from 0.013 to 0.05).

CSI measurements have revealed no changes of patients' metabolites in the semioval center (for NAA/Cr, $p_W=0.93$ left and 0.68 right), but more significant NAA/Cr decresase in the frontal transitional grey-white matter area ($p_W=0.05$ left and 0.08 right). In volunteers and patients, CSI revealed focal areas of increased lactate concentration in the posterior area of the CSI slice, with patients probably having lower lac/NAA in these ($p_W=0.05$).

Discussion

We confirm decrease in NAA in the frontal lobes of the mTBI patients and in the upper brain stem of those with reported unconsciousness. Our data prove a positive correlation of NAA signal to certain clinical scores of the patients. To our best knowledge, this is the first work to show this. Decreased NAA supports the hypothesis of neuronal injury resulting from numerous neurodegenerative cascades after mTBI. We believe that this study contributes to a better definition of its pathomechanisms in the brain space and the role of metabolites involved.

References

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