

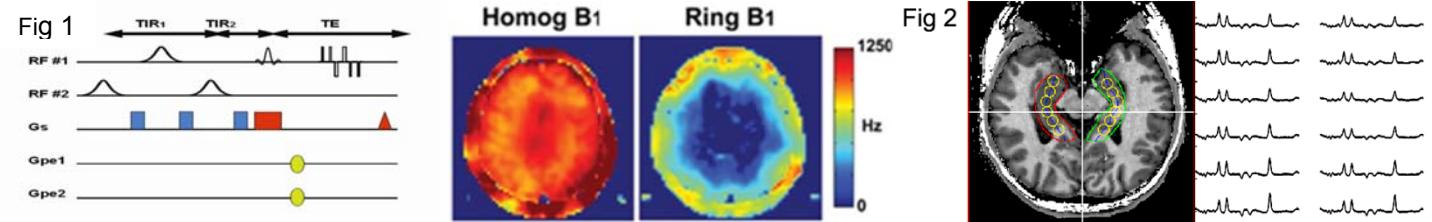
Reduced Hippocampal NAA/Choline in Blast Related Traumatic Brain Injury with Memory Impairment: A 7T MRSI Study

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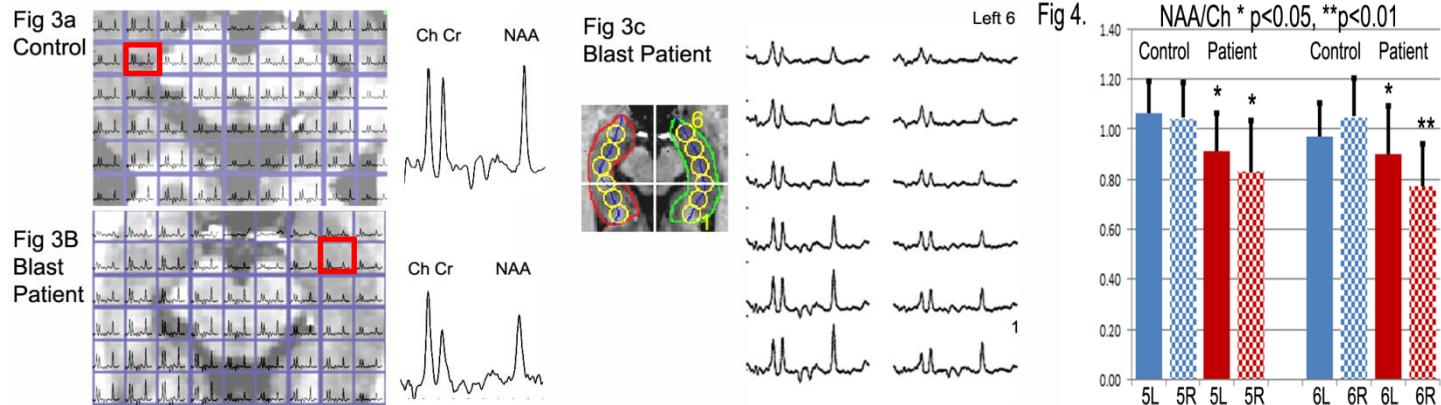
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Introduction: Mild traumatic brain injury due to blast exposure has gained attention for its prevalence in recent conflicts and a lack of positive imaging correlates in conventional MRIs. Despite the absence of conventional MRI findings, these patients express a constellation of ongoing deficits including memory dysfunction, migraines, and a variety of behavioral changes. Previous studies in civilian mild TBI have reported decreases in the NAA/Choline ratio in MRI normal regions. Therefore the goal of this study was to determine if high resolution MRSI studies at 7T of the temporal lobe (TL), and the hippocampus (HC), could detect metabolic alterations in blast exposed patients with memory dysfunction.

Methods: MRSI data at 7T was acquired from 15 healthy control subjects and 8 patients exposed to explosive blasts with self-reported memory dysfunction. The MRSI data (10mm thick slice, 24x24 encodes over a FOV of 192mmx192mm) was acquired with an echo time of 40ms with an 8 element transceiver array. Two distinct RF distributions, a homogeneous distribution for water suppression, excitation and refocusing, and a “ring” distribution for outer volume suppression were used (Fig 1) for localization. HC data was reconstructed using a semi-automated voxel shifting method which uses the anatomical features of the HC to place 6 voxels along the length of the hippocampus (Fig 2 - control subject). Due to the significant heterogeneity in metabolite ratios along the HC formation, patient and control data were compared from equivalent anatomical locations.



Results: Displayed in Fig. 3 are grid reconstructions (and spectra – red boxes) from a region about the hippocampus from a control subject (Fig 3a) and a blast exposed patient with memory impairment (Fig 3b). This patient shows dramatic reductions in NAA/Ch (>2SDs) from both the left and right anterior HC locations in comparison to the control subject (see inset spectra). Fig 3c displays individual spectra reconstructed along the hippocampal formation for the same blast exposed patient. NAA/Ch declines along the posterior-anterior direction of the HC bilaterally. Fig 4 displays pooled data grouped according to controls (blue) and patients (red), side (L-solid/R-check) and hippocampal location (5 & 6, 6 most anterior). Significant declines ($p<0.05$, $p<0.01$) in NAA/Ch were seen in the two most anterior HC loci (5&6) in the patients. A significant decline in NAA/Cr, $p<0.01$, was also seen from the most anterior position of the right HC.



Conclusions: Our data demonstrates significant decrements in NAA/Ch in the anterior HC in patients exposed to explosive blasts with memory impairments, consistent with the anatomical localization of memory (hippocampus and TL). In patients with the largest decrements, declines in NAA/Cr are also seen in the anterior locations of the hippocampus. These studies are amongst the first MRI studies to document the presence of injury in blast exposed patients.