Influence of Callosal Microstructural Compromise on Interhemispheric Speed of Processing in Mild Traumatic Brain Injury

Tuesday 3:10-3:20 PM | SSJ18-02 | Room: S406B

PURPOSE

The corpus callosum (CC) is at specific risk in Mild Traumatic Brain Injury (MTBI) and critical for interhemispheric communication. Here we test the hypothesis that microstructural compromise as measured by diffusion MRI affects performance on a novel interhemispheric speed of processing task (IHSPT).

METHOD AND MATERIALS

The study is approved by the institutional review board. 36 MTBI subjects (11 male, 25 female; mean age 36 years) within 4 weeks of injury and 27 controls were included (12 male, 15 female; mean age 37 years). IHSPT measures latency over 80 trials between visual word stimulus presentation to the right vs left visual hemifield. Patients with positive IHSPT values were included (indicating probable left language dominance, necessitating information crossing the CC). Diffusion MRI was performed on 3T (Skyra, Siemens) with 5 b-values (up to 2.5ms/m² with 60 directions). Diffusion metrics of fractional anisotropy, diffusivity and kurtosis (mean, radial and axial; MD, RD, AD, MK, RK, AK) were calculated as well as compartment-specific white matter microstructure metrics, including axonal water fraction (f), a measure of axon density, intra-axonal diffusion (Daxon), reflective of axonal integrity, and extracellular diffusion along and perpendicular to the axis of the axon (Depar and Dperp), sensitive to glial and inflammatory changes, and changes in myelination, respectively. Region-of-interest analysis was done using freesurfer segmentation of the CC. Relationship between IHSPT performance and diffusion measures was assessed using Pearson's partial correlation in both MTBI and control groups.

RESULTS

In controls, we found correlations between IHSPT and several diffusion measures all localizing to the splenium (MD, RD, AK, and Deperp; p<0.05), lost in MTBI subjects. MTBI subjects, on the other hand, showed significant correlations between IHSPT and kurtosis diffusion measures in the genu of the CC (MK, AK, and RK) (Table 1).

CONCLUSION

In MTBI subjects, we find a relationship between CC body microstructural complexity and IHSPT not seen in controls. Furthermore, the normal relationships seen in controls between tissue microstructure and interhemispheric processing are lost after MTBI.

CLINICAL RELEVANCE/APPLICATION

Understanding how white matter injury affects cognitive performance is the critical next step for better assessing MTBI patients. Here we show altered relationships between CC microstructure and specific IHSPT between MTBI patients and controls.