Differences in cortical thickness between patients with Non-epileptic Attack Disorder and healthy controls

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Background

- Non-epileptic Attack Disorder (NEAD) is a functional neurological disorder which superficially resembles epilepsy but in which seizures are not caused by epileptiform discharges in the brain (LaFrance, Reuber & Goldstein, 2013).
- While many experts see NEAD as a multifactorial biopsychosocial condition (Brown & Reuber, 2016) little is known about the neurobiological underpinnings of the disorder.
- To date only two sMRI studies have examined structural differences between NEAD and controls.
- These studies have yielded conflicting results with one reporting predominantly right hemispheric changes in NEAD and the other bilateral changes (Labate et al., 2012; Ristić et al., 2015 respectively).

Methods

- T1 weighted 3T sMRI brain scans of patients with NEAD (n = 17, 15 female, mean age = 33.70, SD = 13.45, range = 16 to 58) and age and gender matched healthy controls (n = 17, 15 female, mean age = 33.58, SD = 11.77) acquired between 2009 and 2013 were reprocessed retrospectively and automatically segmented using FreeSurfer (v. 5.3.0). NEAD group inclusion was based on a confirmed NEAD clinical diagnosis and video-EEG where possible.
- Group differences for cortical thickness (CT), cortical surface area (CSA), cortical folding (CF), and sulcal depth (SD) were examined using the built in GLM FreeSurfer utility (GFD, v. 1.4), controlling for age and intracranial volume. Results were corrected for multiple comparisons using false discovery rate (FDR) at p < 0.01.

Preliminary Results

Brain regions showing significant cortical thickness differences between NEAD patients and healthy matched controls

- Cortical thickness: NEAD > controls - left and right superior parietal and inferior parietal, left paracentral, and right cuneus.
- Cortical thickness: NEAD < controls - left superior frontal, right lateral orbitofrontal, left superior temporal, left middle temporal, right superior temporal, and left insula.

No significant difference for CSA, CF, and SD.

Conclusion

- Consistent with Ristić et al (2015) we also show bilateral structural changes in brain regions associated with regulatory mechanisms, emotion processing, motor function and sensory control.
- However, our results differ from the previous study in terms of the direction of these changes.
- Work is ongoing to increase sample size, assess heterogeneity, medical history, comorbidity, and duration of disorder.

Key take home message

- These results lend support to the presence of underlying structural brain changes in NEAD.
- In NEAD, symptoms may result from pathological brain processes associated with emotional distress.
- Future studies should attempt to correlate clinical features with structural brain changes and to conduct longitudinal research. This is important as it will allow us to better understand the meaning of these changes.

References


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