

Cortical Thickness Abnormalities in Non-Comorbid Medication-Naive Patients with Major Depressive Disorder and Patients with Social Anxiety Disorder

Thursday 12:45-1:15 PM | NR394-SD-THB4 | NR Community, Learning Center Station #4

PURPOSE

An overlap of clinical symptoms between major depression disorder (MDD) and social anxiety disorder (SAD) suggests similar brain mechanisms of the two disorders. However, few studies directly compare the brain structure between the two disorders. Aim of this study was to assess cortical thickness alterations between non-comorbid medication-naive MDD patients and SAD patients.

METHOD AND MATERIALS

High resolution T1 weighted images were acquired from 37 non-comorbid MDD patients, 24 non-comorbid SAD patients and 41 healthy controls (HC). Vertex-based analysis of cortical thickness (corrected with clusterwise probability of $p < 0.001$) were performed and groups differences were compared by ANOVA analysis followed by post-hoc analysis.

RESULTS

Both MDD and SAD patients, relative to HC, showed cortical thickening in the bilateral medial prefrontal cortex, posterior dorsolateral prefrontal cortex, insular cortex, left temporal pole, and right superior parietal cortex; cortical thinning in the left lateral OFC and bilateral rostral middle frontal cortex. Besides, MDD patients showed specifically greater thickness in left fusiform, right lateral occipital cortex; thinner thickness in bilateral lingual, and left cuneus; SAD patients showed specifically thinner cortical thickness in the right precentral cortex. Furthermore, there were significant negative correlations between HAMD score and cortical thickness in the left SFC, right caudal MFC and right insula in MDD group.

CONCLUSION

Our results indicated that MDD and SAD share a common pattern of gray matter abnormalities in sacilence network and dorsal attention network. In addition, we found disorder-specific involvement of the visual recognition network in MDD and the fear circuitry in SAD.

CLINICAL RELEVANCE/APPLICATION

These consistent structural differences in the two patient groups may contribute to the broad spectrum of emotional, cognitive and behavioural disturbances observed in MDD patients and SAD patients. These findings provide new evidence of shared and specific neuropathological mechanisms underlying MDD and SAD.