Cortical thickness in lupus patients with cognitive impairment

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BACKGROUND

Cognitive dysfunction is a relatively frequent syndrome observed in persons with Systemic Lupus Erythematosus (SLE). Although consensus definitions include cognitive dysfunction as a neuropsychiatric SLE manifestation (NPSLE), there is some controversy about whether this syndrome is directly a result of underlying CNS activity/ damage, or whether cognitive dysfunction derives from non-CNS related factors (e.g., fatigue). Recently, automated segmentation techniques have been used to investigate SLE and show a normal cortical thickness in SLE compared to controls and frontal-parietal reduction pattern in NPSLE. Our aim is to study the correlation between cognitive function and cortical thickness in SLE patients with cognitive impairment but *without* overt neurological syndromes used to give a diagnosis on NPSLE.

METHODS

We evaluated the cortical thickness of 30 controls (21F; age 40+\-12) and 66 SLE (62 F; age 46+\-11). Out of 66 SLE, 55 underwent a comprehensive neuropsychological evaluation performed by trained examiners supervised by a clinical neuropsychologist. Two cognitive indices were derived including 1) a dichotomous cognitive impairment vs intact index and 2) a continuous variable referring to the proportion of measures impaired (of 12 indices).

Imaging acquisition and processing

Axial T1-weighted 3D FSPGR volume imaging (TR/TE/TI 7/2/400, voxel 1mmx1mmx1mm) was acquired on a 3T GE Scanner.

Cortical grey matter segmentation was performed with the Freesurfer image analysis suite http://surfer.nmr.mgh.harvard.edu). With the same tool we performed a parcellation of the cortex into 32 rois for each hemisphere (Desikan-Kyliany Atlas). In each cortical region we obtain the average of the cortical thickness.

Statistical Analysis: We performed a least square test to compare the cortical thickness of SLE versus controls; we used a multivariate regression test to see the correlation between cognitive impairment scales results and thickness values.

RESULTS

Out of 55 SLE patients, 18 (32%) were observed to have cognitive impairment (all female; 47+\-12), compared to 37 (67%) intact patients (35 female; 46+\-12). We did not observe any cortical thickness differences between our patients and controls in all the 68 cortical labels. We observed that compared to cognitively intact patients, cognitively impaired SLE patients differed in cortical thickness in the following regions: posteriorcingulate bilaterally, in the frontal pole left parstriangularis, right isthmuscingulate, lateraloccipital ($p<0.01$) and in the left bankssts and superiopatietal bilaterally ($p<0.05$). In addition, a statistically significant correlation between the proportion of indices impaired and thickness (Fig.1) was observed in the right lateraloccipital label ($p<0.001$); in the left paracentral, left parstriangularis, left posteriorcingulate and right superiorparietal ($p<0.01$); in the left cuneus, left lateraloccipital, right isthmuscingulate, right posteriorcingulate, right precuneus ($p<0.05$).

CONCLUSION

In the absence of other neurological syndromes, cognitive function is associated with cortical thickness alterations in the brain. The precise etiology of these changes is not known, and future investigation of disease manifestations in relation to cortical thickness is warranted. Moreover, although SLE is often considered a subcortical white matter disease, there is increasing attention paid to cortical and subcortical grey matter structures. Our results seem to suggest that these structures may also be vulnerable to the effects of SLE.

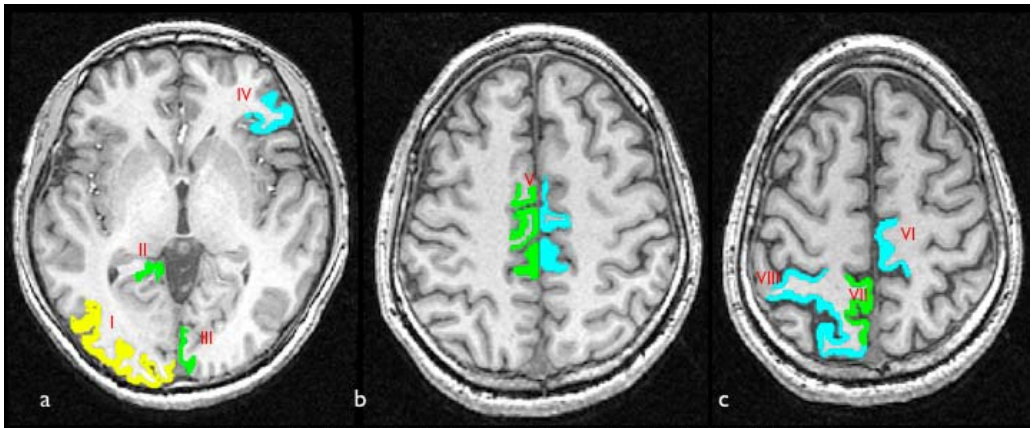

Figure 1 Statistically significant areas differentiated by p values: yellow= $p<0.001$; teal= $p<0.01$; green= $p<0.05$. a) right lateraloccipital (I), right isthmuscingulate (II), left cuneus (III) and left parstriangularis (IV), b) bilaterally posteriorcingulate (V) c) left paracentral (VI), right precuneus (VII) and right superioparietal (VIII)