Structural brain damage in Lewy body dementia: a multimodal MRI study

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INTRODUCTION

- Lewy body dementia (LBD) is a heterogeneous condition that encompasses tandem features of fluctuating cognitive decline, parkinsonism and visual hallucinations.
- The pathophysiological mechanisms underpinning the differing clinical features of LBD are still unknown.

The aim of our study is to investigate the pattern of cortical thickness and infratentorial grey matter (GM) atrophy, white matter (WM) vascular hyperintensities and WM microstructural damage in patients with LBD.

METHODS

24 LBD patients (mean age: 72 ± 5.7 years, 7 females) and 20 age-matched healthy controls (HC) underwent clinical and neuropsychological evaluations, and brain structural and diffusion tensor (DT) MRI.

- Cortical thickness (CT) measures and volumes of deep grey matter structures were obtained.
- WM hyperintensities (WMH) were manually assessed on T2-weighted images and lesion probability maps were obtained.
- Microstructural damage of normal appearing WM was assessed using tract-based spatial statistics (TBSS).

RESULTS

- LBD patients were in a moderate stage of dementia (MMSE 17.5 ± 4.7, CDR: 1.2 ± 0.5) with a multidomain cognitive impairment.
- In LBD patients relative to controls, cortical thickness analysis revealed significant foci of cortical damage in left postcentral gyrus, left superior parietal gyrus and bilateral precentral gyrus, and voxel-based morphometry showed a significant atrophy of thalamus bilaterally (Figure 1).
- LBD patients had higher WMH load compared to HC. Among LBD patients, WMH were mainly distributed in the occipital and posterior periventricular regions, (Figure 2).
- In the normal appearing WM, TBSS revealed a distributed and symmetric pattern of fractional anisotropy decrease and mean diffusivity increase in the main supratentorial and infratentorial WM tracts in LBD compared to HC, (Figure 3).

CONCLUSIONS

In LBD, both GM and WM were significantly involved.

Cortical and infratentorial GM damage appeared restricted to specific areas, while WM degeneration was diffuse and apparently unrelated to WMH due to the concomitant cerebrovascular disease.

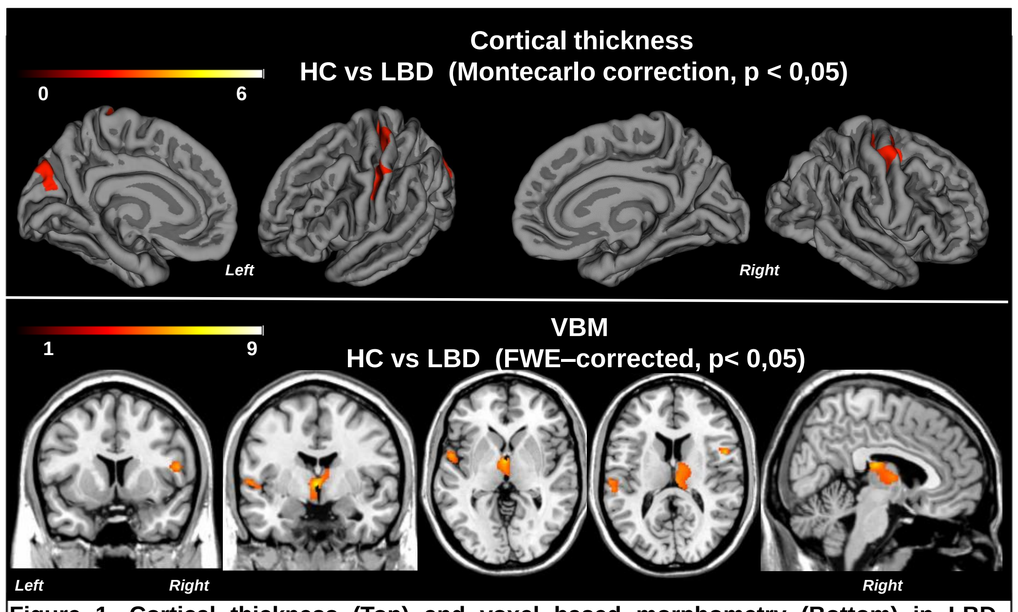

Figure 1. Cortical thickness (Top) and voxel based morphometry (Bottom) in LBD patients relative to healthy controls (HC).
All the results are corrected for age. VBM images are shown in radiological convention (right is right). Color bars represent t-values.

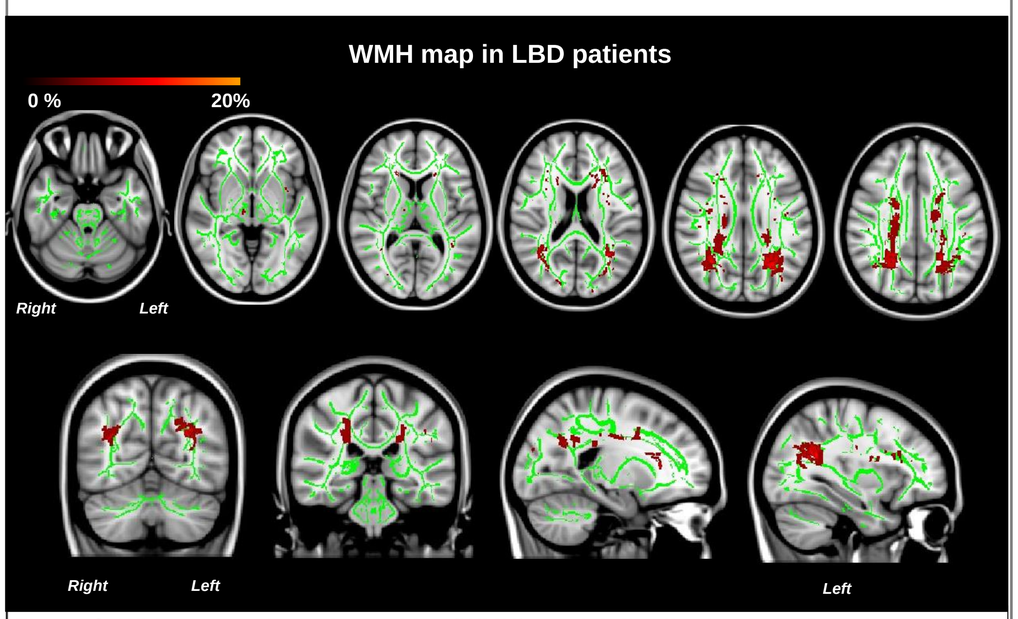

Figure 2. White matter hyperintensities (WMH) spatial distribution in LBD patients.
The color scale (from 0% to 20%) represents the minimum to maximum probability of a lesion occurring in a particular spatial location. Results are shown in radiological convention (right is left).

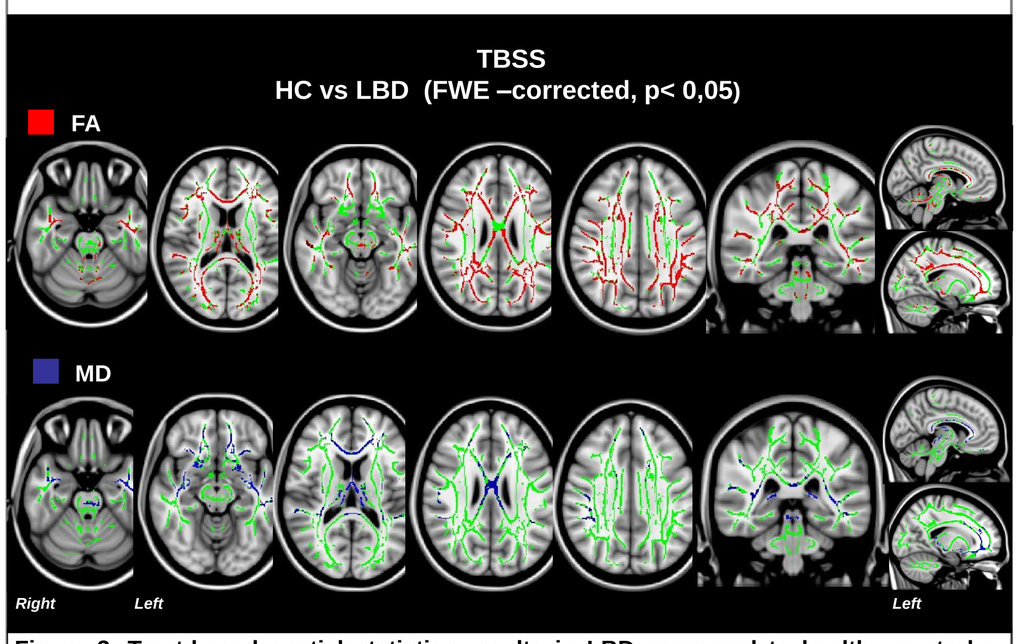

Figure 3. Tract-based spatial statistics results in LBD compared to healthy controls.
Top: decreased fractional anisotropy (FA) in LBD patients compared with HC is shown in red. Bottom: increased mean diffusivity (MD) in LBD patients compared with HC is shown in blue. All the results are corrected for age and shown in neurological convention (right is left).

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