Longitudinal structural network reorganisation in early relapsing-remitting multiple sclerosis [Abstract]

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Background: Multiple sclerosis (MS) is characterized by relapses and remissions indicating damage and compensatory processes occurring early in the disease. Over time, cortical pathology is highly relevant for disability, while brain networks evolve towards a disconnected organization as the disease progresses. However, it is poorly understood how and when pathology impacts cortical networks and in particular, how the network responds to damage in the very beginning of the disease.
Aim: To address cortical pathology by quantifying structural connectivity patterns over 12 months in patients with early relapsing-remitting MS.
Methods: Here we investigated cortical grey matter networks longitudinally as derived from structural 3 Tesla MRI in 92 patients in the initial phase of the disease (65 female / 27 male; mean age: 32.9 ± 9.9 years; mean disease duration: 12.1 ± 14.5 months) and in 101 healthy controls (59 female / 42 male; mean age: 19.7 ± 0.9 years). Longitudinal brain volume atrophy was analyzed using SIENA and cortical thickness changes were quantified using FreeSurfer. Brain networks were computed based on cortical thickness inter-regional correlations between anatomical regions and fed into graph theoretical analysis. Finally, subgroup analyses were performed between patients with "no evidence of disease activity" (NEDA) during this period and those with disease activity (EDA).
Results: Over one year, increased local cortical connectivity and an emerging modular-constructed network were detected in patients - a pattern reported to be associated with adaptation, efficiency and compensation. These longitudinal dynamics were attested in both patients with NEDA and EDA, indicating continuous cortical reorganisation independent of disease activity. This local and modular cortical reorganisation was not detected in healthy controls over the same period of time and emerged beyond measureable signs of atrophy using established morphometric tools.
Conclusion: Our findings demonstrate that despite initiation of neuroinflammatory damage, substantial structural adaptation processes emerge cortically in the early disease stage. This subtle reorganisation of the cortex architecture is quantifiable by structural MRI in patients with and without disease activity, suggesting a principal response of the network evolving from the onset of this chronic disease.

Disclosure

The authors declare no conflict of interests.

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