

INTRODUCTION and OBJECTIVE

Structural connectivity differences between motor subtypes of Parkinson's disease (PD) are still unknown. The aim of this study was to evaluate structural changes of motor network in tremor-dominant and non-tremor phenotypes of Parkinson's disease, using diffusion magnetic resonance-based structural network analysis.

MATERIALS and METHODS

A total of sixty-three Parkinson's disease patients (35 patients with tremor-dominant PD [TD-PD] and 28 patients with non-tremor PD [NT-PD]) and 30 controls were involved in this study.

Diffusion magnetic resonance probabilistic tractography was used to measure the connectivity indices of each tract and the efficiency of each node, namely cerebellum, basal ganglia, thalamus and motor cortices.

RESULTS

- NT-PD patients showed structural connectivity alterations in the cortico-basal ganglia motor circuits with nigro-pallidal and striato-frontal involvement, whereas the same motor connections seemed to be relatively spared in the TD-PD.
- Similarly to the structural connectivity changes, nodal efficiency values were markedly increased in NT-PD patients relative to TD-PD patients and controls, at the level of the globus pallidus, putamen, and supplementary motor area.
- The severity of rigidity-bradykinesia was related with lower connectivity indices of striato-frontal tract, and with higher nodal efficiency values of the putamen in NT-PD patients.
- The severity of tremor was related with higher connectivity indices of nigro-pallidal tract, and with lower nodal efficiency values of the globus pallidus in TD-PD patients





These findings suggest that:

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- the NT-PD patients show alterations of the structural connectivity in the cortico-basal ganglia motor circuits, whereas the same motor connections seem to be relatively spared in TD-PD patients;
- striato-frontal involvement plays a central role in the emergence of bradykinesia and rigidity;
- the relative integrity of pallidal connectivity may contribute to the tremorgenesis in patients with TD-PD, by an imbalance in favor of the dopaminergic excitatory influence.

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