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Clinical correlates of mental dysfunction in Parkinson's disease without dementia

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Background

Parkinson's disease (PD) comprises a range of motor and cognitive disturbances, a portion of which respond little, if at all, to levodopa treatment and are therefore considered to be due to nondopaminergic lesions [1,2]. More severe cognitive impairments and higher risk for developing dementia have been associated with the clinical manifestations of postural instability and gait disorder which are characterized by refractoriness to levodopa [2,3].

Materials and methods

A consecutive series of non-demented PD patients referred to our tertiary care centre received a comprehensive neurological and neuropsychological evaluation in their "on" state. Strict inclusion criteria were applied in order to avoid the influence on cognitive outcomes of factors other than the clinical status of patients. The Unified Parkinson's Disease Rating Scale (UPDRS) motor score was divided into 2 subscores that represented predominantly dopaminergic (subscore A: tremor, rigidity, bradykinesia and facial expression) and nondopaminergic (subscore B: speech and axial impairment) deficiency. Overall cognitive status was rated with the Mini-Mental State Examination (MMSE) and the presence of depressive symptoms with the Beck Depression Inventory - Fast screen. A neuropsychological test battery was further administered to each patient to assess cognitive domains known to be affected even in the early stages of PD, which included the Rey Auditory Verbal Learning Test (RAVLT) (verbal memory), the Trail Making Test (TMT) part B and the Stroop Neuropsychological Screening Test (executive functions), the Line Orientation Test (visuospatial perception), as well as the TMT part A and the Symbol Digit Modalities Test (psychomotor speed and attention). In addition, the intersecting pentagon copying item within the MMSE was graded using a 0-2 rating scale (visuoconstructional ability). Stepwise multiple regression analyses were performed in order to model the effect of clinical parameters on cognitive measures. The predictor variables included age, age at onset of disease, years of education, depression score, daily dose of levodopa, subscore A and subscore B.

Results

A total of 42 patients (22 males, 20 females) with a mean age of 64.9 ± 10.1 years, age at onset of PD of 58.2 ± 11.1 years, PD duration of 6.6 ± 5.3 years and education of 9.2 ± 4.2 years were included. The best predictors of neuropsychological performances were age, age at onset of symptoms and years of education. Subscore A accounted only for an additional 6.9% (p=0.038) of the variation of the RAVLT trial 3 and subscore B for an additional 5.5% (p=0.043) of the variation of the TMT part B, while both subscores were excluded in models of all other measures.

Conclusions

Cognitive decline in PD patients might be owing to the simultaneous effect of age-related and disease-associated neuropathology. Development of impaired postural reflexes and gait difficulties in patients does not appear to be closely related to cognitive dysfunction, at least prior to dementia.

References

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