Original Article

# Neuropsychological profile in early Parkinson's disease: Comparison between patients with right side onset versus left side onset of motor symptoms

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## Abstract

**Aims:** Though impaired cognition in Parkinson's disease (PD) is well known, data in early PD is sparse. This study was designed to assess the cognitive profile in patients with early PD (motor symptoms <5 years and Hoehn and Yahr stage <2), and to compare the cognitive profile between these patients with right versus left side onset of motor symptoms. **Materials and Methods:** National Institute of National Health and Neurosciences (NIMHANS) neuropsychological battery was used to assess the cognitive profile in 50 patients with early PD and compared with 50 age-, education-, and gender-matched healthy controls. Within the PD group, the cognitive profile was also compared between patients with right side onset motor symptoms (RPD) versus those with left side onset (LPD). The neuropsychological tests assessed the executive functions, memory, attention, visuospatial functions, and psychomotor speed. **Results:** Among the 50 patients, 25 each were RPD and LPD. The two subgroups were matched for age, gender, education, age at disease onset, disease duration, and degree of motor disability. There was no significant difference between the groups on Hoehn and Yahr staging or Unified Parkinson Disease Rating Scale (UPDRS) motor score. Patients with early PD performed significantly worse in the tasks involving memory, executive functions, and attention compared to controls. However, there was no difference in the cognitive profile between RPD and LPD subgroups. **Conclusions:** Patients with early PD have cognitive dysfunction with predominant involvement of frontal and temporal lobes. Side of onset of motor symptoms probably does not have significant role in future development or profile of cognitive dysfunction in PD.

## Key Words

Cognition, executive functions, laterality of motor symptoms, left side onse, memory, parkinson's disease, right side onset

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## Introduction

James Parkinson had mentioned that the intellect is preserved in Parkinson's disease (PD); however, it is now clear that cognitive impairment is common even in early PD and affects a variety of cognitive functions.[1] Several studies have shown that cognitive dysfunction is most prominent in the domains of executive function and memory.[2] However, involvement of visuospatial domain and attention has also been implicated.[3,4] These inconsistencies may be due to varying neuropsychological tests by different authors along with usage of different cut off points for the functional impairment.

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Asymmetry of motor symptoms is considered a crucial criterion for PD and has been used to differentiate idiopathic PD from other parkinsonian syndromes. This asymmetry is considered to be due to asymmetric degeneration of dopaminergic neurons in one of the hemispheres.[5] Various studies have compared the cognitive profile of PD patients with right side onset of motor symptoms (RPD) versus those with left side onset of motor symptoms (LPD). The results are contradictory, with some studies finding no difference in the cognitive performance between the two groups, while others have reported that RPD perform poorly on verbally mediated tasks, whereas LPD perform worse on visuospatial tasks.[6,7] However most of these studies have been carried out on patients with long standing PD and very few studies have addressed this issue at the early stage.[8,9]

The present study was conducted to determine the cognitive profile of patients with early PD and to assess the

effect of laterality of onset of motor symptoms with the cognition.

## Materials and Methods

### Study design

This was a prospective case control study to determine the cognitive profile of patients with early PD defined as duration of motor symptoms of <5 years and Hoehn and Yahr stage of ≤2. The study was approved by the Institute's Ethics Committee and all the participants gave written informed consent before undergoing clinical evaluation.

### Clinical evaluation

All patients were recruited from the outpatient clinic or inpatient of the Department of Neurology, National Institute of National Health and Neurosciences (NIMHANS), a tertiary care teaching hospital in Bangalore, India from October 2011 to December 2012.

The inclusion criteria for recruitment of the subjects were:

1. Diagnosis of PD as per UK Parkinson's Disease Society Brain Bank Diagnostic Criteria (UK-PDS-DC),
2. Disease duration <5 years and Hoehn and Yahr stage ≤2,
3. No history of cognitive decline and a Mini mental status examination score (MMSE) of >24,
4. Age <65 years, and
5. Formal education of >7 years.

Healthy controls were age, education, and gender-matched with MMSE >24. Most of these subjects were relatives of the patients admitted in the hospital.

All the patients were evaluated with detailed clinical history and examination. The Unified Parkinson's Disease Rating Scale, part III (UPDRS-III) was used to evaluate motor disability.[10] Handedness was assessed using Revised Edinburgh Handedness Inventory.[11] Patients were divided into RPD and LPD based on the initial onset of motor symptoms. In addition, Hamilton's depression and anxiety scales were applied on the patients to assess depression and anxiety.[12,13]

### Neuropsychological evaluation

The patients were evaluated with a comprehensive neuropsychological battery developed and standardized for older adults at NIMHANS that included evaluation of verbal episodic memory (story immediate and delayed recall, word trial immediate and delayed recall, and digit span backwards), visual memory (Corsis block test and stick construction immediate and delayed recall), visuospatial abilities (stick construction), executive functions (verbal fluency, category fluency, go-no-go test), attention (digit span forwards, corsis block forwards, picture cancellation task), and psychomotor speed by noting down the time taken in object cancellation task. This is a short, easy to use, and culturally appropriate tool for Indian older adults.[14]

Performance on each task was interpreted as abnormal based on the standard deviation (SD) as compared to controls. Mean minus 1.5 SD was taken as normal and patients' mean score falling below 1.5 SD of the healthy normal controls was considered as abnormal. Patients were also divided based upon their scores into more than 2 SD and 2-3 SD.

### Statistical analysis

Data was analyzed using Statistical Package for Social Sciences (SPSS) software version 16 and a *P* - value of <0.05 was taken as significant. Data was expressed using descriptive statistics such as for continuous variables, mean and standard deviation; and for categorical variables, frequency and percentage. The normality of the distribution was assessed by the skewness of the values. For analysis of continuous variables, nonparametric test (Mann-Whitney test) was employed. The analysis of categorical variables was done by Fischer's exact test as the assumption of chi-square test failed.

## Results

Fifty patients (25 RPD and 25 LPD) along with 50 age, education, and gender-matched controls were recruited for this study. The demographic and clinical profile of the two subgroups of PD (RPD and LPD) patients are given in Table 1. The two subgroups were comparable with regard to age, gender, education, age at disease onset, disease duration, and degree of motor disability. UPDRS-III was assessed in the medication ON state in all the patients and they were divided as tremor and rigidity predominant as done in earlier study.[1]

On comparison with controls, the patients showed significantly impaired performances on the tasks of memory (story immediate and delayed recall, construction immediate and delayed recall, and word third trial and delayed recall), executive functions (verbal fluency), and attention and psychomotor speed (picture cancellation task). On further analysis of RPD and LPD versus controls it was found that

**Table 1: Demographic profile of the patients with Parkinson's disease (PD)**

| Variable | Right side onset (*n* = 25) | Left side onset (*n* = 25) | *P* - value |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | |
| Age (years) | 52.24±7.96 | 48.44±8.07 | 0.10 |
| Women: Men | 5:20 | 8: 17 | 0.52 |
| Years of education | 12.32±2.90 | 12.52±3.22 | 0.82 |
| Age of onset of PD (years) | 49.72± 8.40 | 46.04±8.22 | 0.12 |
| Duration of PD (months) | 27.72±15.3 | 30.96±17.8 | 0.49 |
| Tremor predominant | 19 | 16 | |
| Rigidity predominant | 4 | 9 | |
| Mix tremor and rigidity | 2 | 0 | |
| History of RBD | 2 | 3 | |
| LED (mg/day) | 483±172.5 | 463±249.3 | 0.74 |
| MMSE | 28.68±1.40 | 28.68±1.60 | 1.0 |
| Hoehn and Yahr stage | 1.5±0.30 | 1.6±3.8 | 0.89 |
| HAM-D score | 11.2±5.5 | 12.6±5.30 | 0.63 |
| HAM-A score | 8.88±3.5 | 9.40±4.00 | 0.36 |
| UPDRS-III | 15.12±7.10 | 20.56±12.5 | 0.06 |

RBD = Random eye motionbehaviour disorder, LED = Levodopa equivalent dose, MMSE: Minimental status examination score, HAM-D = Hamilton depression, HAM-A = Hamilton anxiety, UPDRS-III = Unified parkinson disease rating scale, part III

the deficits in both the groups were similar as compared to controls. Table 2 gives comparison of patients with controls.

However, there were no differences in any of the neuropsychological tasks between RPD and LPD groups. With regards to the medications that could interfere in cognition testing, there were 13 (52%) patients on trihexphenidyl in the right side onset group as compared to 11 (44%) in the left sided onset group (*P* - value = 0.68). The mean dose in right sided onset group was 4.1 ± 1.5 mg/day and the left sided onset group was 4.4 ± 1.9 mg/day (*P* - value = 0.66). Table 3 shows the comparative analysis of cognition in patients with RPD and LPD.

## Discussion

The present study assessed the cognitive profile of patients with early PD and the effect of laterality of initial motor symptoms on cognition. Our study found significant impairment in the tasks involving both verbal and visual learning and memory (story immediate and delayed recall, stick construction immediate and delayed recall, word list third trial and delayed recall). This was also reported earlier Weintraub *et al*.[1] This finding suggests that hippocampal pathology, which in fact is common in PD may also contribute to memory impairment in a subgroup of patients with PD.[15,16] The above findings are also supported by a recent study by Yu *et al*., who also reported impairment of both verbal and nonverbal memory in their cohort of 94 PD patients.[8] Thus, our study supports the concept put forward by the above mentioned study that in PD hippocampal pathology may also contribute to memory impairment apart from frontal type of memory impairment.

Executive function has been often reported as the most common neuropsychological dysfunction in patients with PD.[2] The impairment of executive functions suggest the early involvement of frontosubcortical.[17] In our study we found a significant difference in the verbal fluency task between patients and controls which suggests involvement of the left prefrontal cortex and right cerebellum.[18] Abnormalities of verbal fluency test is well described in PD and several studies have reported fluency deficits as predictors of future dementia.[19] There is clearly a degree of overlap between phonemic and category fluency tasks with both relying on frontally based executive strategies. Difference in relative performance of the two tests may be useful in terms of neuroanatomical localization, given that category fluency is more commonly impaired in Alzheimer's disease and semantic dementia, while phonemic fluency impairment indicates subcortical dementias.[20] In our study we found a significant difference only in phonemic fluency task and not on the category fluency task, thus indicating involvement of frontal subcortical component of cognition.

Visuospatial impairment has been described in patients with PD, even when tests reveal few motor component involvement.[3,21] It has been suggested that such visuospatial impairment may be at least partly attributable to frontal-executive dysfunction in addition to deficit in parietal cortex. These clinical observations provide a strong support for the hypothesis that the dementia of PD has a posterior cortical

**Table 2: Domain-wise distribution of cognitive functions between patients and controls**

| Domain | Patients (*n* = 50) | Controls (*n* = 50) | *P* - value |
|---|---|---|---|
| Memory | | | |
| Story IR | 9.38±3.03 | 13.44±1.31 | <0.001 |
| Story DR | 8.46±3.28 | 12.80±1.66 | <0.001 |
| Word list 1 | 4.56±1.19 | 4.52±0.97 | 0.54 |
| Word list 2 | 5.90±1.14 | 6.38±1.21 | 0.09 |
| Word list 3 | 6.68±1.46 | 7.62±1.42 | <0.001 |
| Word list TR | 17.20±3.31 | 18.50±3.24 | 0.09 |
| Word list DR | 6.60±1.27 | 7.28±1.24 | <0.001 |
| Design construction IR | 17.98±4.83 | 20.68±2.38 | <0.001 |
| Design construction DR | 12.42±4.95 | 16.20±2.72 | <0.001 |
| Executive functions | | | |
| Digit span B | 4.84±1.50 | 4.54±0.64 | 0.74 |
| Corsi span B | 4.66±0.98 | 4.42±0.67 | 0.14 |
| Category fluency | 10.52±3.62 | 10.92±2.15 | 0.30 |
| Phonemic fluency | 6.80±3.68 | 9.18±2.67 | <0.001 |
| Go-no-go 1 | 5.80±0.49 | 5.74±0.48 | 0.36 |
| Go-no-go 2 | 14.50±0.81 | 14.59±0.61 | 0.78 |
| Visuospatial functions | | | |
| Design construction | 24.12±0.89 | 23.86±0.90 | 0.12 |
| Attention | | | |
| Picture cancellation | 202.44±43.39 | 136.86±9.57 | <0.001 |
| Digit span forwards | 5.70±1.52 | 5.42±0.83 | 0.64 |
| Corsi span forwards | 5.28±1.07 | 5.32±0.68 | 0.67 |

IR = Immediate recall, DR = Delayed recall, TR = third trial recall

**Table 3: Comparison of cognitive profile in patients with right side versus left side onset symptoms in Parkinson's disease (PD) patients**

| Domain | RPD (*n* = 25) | LPD (*n* = 25) | *P* - value |
|---|---|---|---|
| Memory | | | |
| Story IR | | | |
| Story DR | 8.24±3.12 | 8.68±3.48 | 0.73 |
| Word list 1 | 4.68±1.03 | 4.44±1.35 | 0.44 |
| Word list 2 | 5.88±0.97 | 5.92±1.32 | 0.82 |
| Word list 3 | 6.80±1.22 | 6.56±1.68 | 0.40 |
| Word list TR | 17.56±2.70 | 16.84±3.84 | 0.40 |
| Word list DR | 6.56±0.82 | 6.64±1.63 | 0.75 |
| Design construction IR | 17.12±3.96 | 18.84±5.52 | 0.06 |
| Design construction DR | 12.28±4.21 | 12.56±5.68 | 0.62 |
| Executive functions | | | |
| Digit span B | 4.80±1.08 | 4.88±1.85 | 0.41 |
| Corsi span B | 4.68±0.80 | 4.64±1.15 | 0.87 |
| Category fluency | 10.40±3.22 | 10.64±4.05 | 0.95 |
| Phonemic fluency | 6.52±2.66 | 7.08±4.51 | 0.73 |
| Go-no-go 1 | 5.84±0.47 | 5.76±0.52 | 0.46 |
| Go-no-go 2 | 14.56±0.91 | 14.46±0.71 | 0.27 |
| Visuospatial functions | | | |
| Design construction | 24.16±0.89 | 24.08±0.90 | 0.68 |
| Attention | | | |
| Picture cancellation | 200.4±37.16 | 204.4±49.54 | 0.62 |
| Digit span forwards | 5.48±1.19 | 5.92±1.80 | 0.55 |
| Corsi span forwards | 5.20±0.86 | 5.36±1.25 | 0.79 |

RPD = PD patients with right side onset of symptoms, LPD = PD patients with left side onset of symptoms, IR = Immediate recall, DR = Delayed recall, TR = Third trial recall

basis. Although Lewy body deposition may be the most likely etiological factor, the extent to which subcortical dopaminergic and cholinergic systems influence these areas remain uncertain given that these cortical areas receive innervation from both the systems.[22] A possible explanation why our study failed to show any significant difference in the visuospatial domains may be due to the fact that we used only one test in our battery to assess the visuospatial functions, while most other studies have included more than one test to assess the same.

Dysfunction in attention is also closely related to the impairment of executive functions reported in these patients. Several studies have shown that dysfunction in attention appears most often in connection with complex tasks requiring shifting and/or sustained attention, as well as mental calculations that require sustained mental tracking.[4] The underlying pathological substrate of this dysfunction is not yet clear, but metabolic activity and cholinergic deficits in the thalamus have been suggested as possible mechanisms.[23,24] In our study; digit span forwards, Corsi's span forwards, and picture cancellation task were used to assess attention. While there was no significant difference in the former two tasks, the later task of symbol cancellation showed a significant impairment in the performance of patients compared to controls, suggesting impairment of attention. Moreover, since the task required to cancel the objects with maximal speed, an additional impairment of psychomotor speed in these patients could be responsible.

We did not find any difference in the cognitive domains between the patients with RPD and LPD, similar to that recently reported by Erro *et al*.[9] There are contradictory reports on memory functions in relation to side of initial onset of motor symptoms in PD. Clair *et al*., reported absence of any difference between the two groups in visuospatial memory, while Huber *et al*., reported disadvantage for LPD patients on visuospatial memory.[25,26]

With regard to visuospatial functions, some previous studies have shown that visuospatial functions are more affected in patients with LPD compared to those with RPD, while another study did not find any difference between the RPD and LPD groups.[9,27]

With respect to executive functions, most of the previous studies have used Control Oral Word Association Test (COWAT) and did not find any difference between RPD and LPD groups.[25] On the contrary, two studies yielded opposite results with Cubo *et al*., reporting lower performance in RPD patients, while Tomer *et al*., reporting that patients with RPD generated more words compared to patients with a LPD.[28,29]

For evaluation of attention, most studies used the Wechsler Adult Intelligence Scale-Revised (WAIS-R) Digit Span to assess complex attention and working memory. Huber *et al*., reported poorer verbal span for RPD patients; whereas, Tomer *et al*., reported a disadvantage for patients in the LPD group.[26,29]

Similar findings were obtained in studies using the Visual Span test of the WAIS-R. While one study reported poorer performance in RPD patients, another study did not find any difference between the groups.[26,30] The discrepancies in the above studies may be due to different neuropsychological tests applied as well as due to inclusion of patients with varying duration of PD. Our study and the study by Erro *et al*., did not find any difference between the RPD versus LPD, which may be due to the fact that both studies used early PD as the inclusion criteria.[9]

We found that neuropsychological tests can detect impairment of memory, executive functions, and attention in the early stages of PD. Though fronto-subcortical involvement is known to be the main contributor of cognitive dysfunctions in PD, the involvement of medial temporal lobe may be equally important. Neuropsychological testing detects mild cognitive changes in early PD, which is important clinically for the comprehensive management of the patients. In addition, the neuropsychological assessment can provide insight to the patients and family members for planning their future needs.

Finally, our study failed to support the concept that the side of onset of motor symptoms influence cognition in early PD. These can be attributed to deficiency of tests or due to compensatory mechanisms and neural plasticity in the existing networks that neutralized any effect of pathology with respect to the laterality of symptoms on cognition. However, longitudinal studies are needed on larger cohorts to ascertain the relationship between the initial laterality of motor symptoms and cognitive function.

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