Prospective Characterization of Cognitive Function in Typical and ‘Brainstem Predominant’ Progressive Supranuclear Palsy Phenotypes

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ABSTRACT

Objective Clinicopathological studies over the last decade have broadened the clinical spectrum of progressive supranuclear palsy (PSP) to include several distinct clinical syndromes. We examined the cognitive profiles of patients with PSP-Richardson's syndrome (PSP-RS) and two atypical ‘brainstem predominant’ PSP phenotypes (PSP-parkinsonism, PSP-P; and PSP-pure akinesia with gait freezing, PSP-PAGF) using a comprehensive neuropsychological battery.

Methods Fourteen patients diagnosed as PSP-RS, three patients with PSP-P and four patients with PSP-PAGF were assessed using a comprehensive battery of neuropsychological tests.

Results The typical PSP-RS subgroup demonstrated greater impairments in processing speed [t(19) = -4.10, p = 0.001 (d = 1.66)] and executive function [t(19) = -2.63, p = 0.02 (d = 1.20)] compared to the ‘brainstem predominant’ PSP phenotype.

Conclusion This is the first prospective study to demonstrate that PSP-RS and ‘brainstem predominant’ PSP phenotypes can be differentiated on cognitive grounds. These differences correspond with variations in pathological profiles reported in the literature.

Key Words Progressive supranuclear palsy; phenotypes; cognition; neuropsychology.
profile, which includes cognitive slowing, deficits in attention, and early and severe frontal executive dysfunction with difficulties in allocating attentional resources, problems with planning, shifting concepts and prominent retrieval-based memory deficits. These cognitive deficits have been linked to subcortical pathology and associated frontal deafferentation as well as damage to cortical frontal regions and underlying white matter tracts.

To date, only two studies have prospectively examined the neuropsychological profiles of atypical PSP phenotypes. Both of these studies compared PSP-P with typical PSP-RS. Although they found no significant differences between the phenotypes in general cognition or executive function, neither study comprehensively examined cognition. This has left unanswered, the question of whether clinicopathological distinctions between these PSP groups extend to differences in cognitive profiles in a prospective cohort. Clarifying this issue may aid earlier and more accurate differentiation of classic and ‘brainstem predominant’ PSP phenotypes.

To this end, this study utilized a comprehensive neuropsychological examination to prospectively investigate the severity of cognitive changes in typical PSP and the ‘brainstem predominant’ PSP phenotypes. Given the greater pathological involvement of the basal ganglia, diencephalon, brainstem, cerebellum and frontal cortical structures in PSP-RS compared to the ‘brainstem predominant’ PSP phenotypes (PSP-P, PSP-PAGF), it was predicted that the PSP-RS group would exhibit more severe deficits in processing speed, attention and executive function than the ‘brainstem predominant’ PSP phenotype group.

MATERIALS & METHODS

Participants

Fourteen patients with PSP-RS, 3 patients with PSP-P and 4 with PSP-PAGF were recruited from a specialist movement disorder clinic at a tertiary referral hospital in Melbourne, Australia. Participants were diagnosed by an experienced movement disorder specialist (D.W.) according to the diagnostic criteria for PSP phenotypes. The inclusion criteria for PSP-RS were early falls, cognitive dysfunction, eye movement abnormalities, and postural instability; PSP-P included asymmetric parkinsonian symptoms (rigidity, bradykinesia and in some cases tremor), non-axial dystonia and modest response to levodopa; and PSP-PAGF included an early history of freezing of gait or hypophonia of gradual onset without limb rigidity and tremor and no sustained response to levodopa, dementia or eye movement abnormalities. Patients with history of major focal neurological or psychiatric disorders (history of stroke, severe depression, or psychosis), major head injury, drug or alcohol abuse, and global cognitive decline (as indicated by the Mini Mental State Examination score < 24) were excluded. No participants had their diagnosis changed during the two-year follow-up period, and in all participants who have died since study commencement (n = 5), post-mortem findings have confirmed the study neurologist’s clinical diagnosis (PSP-RS).

Materials and procedures

Clinical examination

All participants underwent systematic neurological examination that included the Progressive Supranuclear Palsy Rating Scale and the Hoehn and Yahr Stage of Illness Rating Scale (HYRS).

Neuropsychological battery

All participants completed a comprehensive neuropsychological examination that assessed six cognitive domains: processing speed, memory, language, working memory, visuospatial function, and executive function (Table 1 for list of tests).

To minimize the influence of motor, speech and oculomotor dysfunction on test performance, baseline control comparisons, modified scoring and presentation procedures were used (Table 1).

All patients gave written informed consent to the study, which was approved by The Alfred Hospital Human Research Ethics Committee (HREC reference: 66/09).

Statistical analyses

Nonparametric Mann-Whitney tests were used to compare the clinical and demographic characteristics of the groups. Cognitive domain indices were derived by averaging the z-scores of the tests within each cognitive domain by group. For 5 of the 6 indices, the aforementioned assessment modifications were incorporated into the index, allowing between-
group analysis with \( t \)-tests for unpaired samples. The language index required covariation of baseline speed, so analysis of covariance was conducted for this index. Due to the limited sample size, no corrections for multiple comparisons were made.

Standardized scores were calculated for each cognitive test using published normative data. Impairment was classified relative to the normative mean as mild \( \geq 1.5 \) standard deviations (SD), moderate \( \geq 2 \) SD, or severe \( \geq 3 \) SD.23

**RESULTS**

**Demographic and clinical data**

As shown in Table 2, there were no significant differences between the typical and ‘brainstem predominant’ PSP phenotypes on age \( \text{mean (SD) = 69.93 (5.66)} \) vs. \( 67.67 (15.63); p = 0.40 \), education \( \text{mean (SD) = 10.79 (2.69)} \) vs. \( 13.33 (4.16); p = 0.26 \), levodopa medication \( \text{mean (SD) = 6/14 vs. 4/7; p = 0.10; Fisher's test} \) or level of clinical disability \( \text{PSPRS:}\ p = 0.09; \) HYRS: \( p = 0.16 \). Thus, the groups were well matched with respect to the demographic variables and disease severity.

Not surprisingly, the PSP-RS group showed a trend towards shorter disease duration relative to the ‘brainstem predominant’ PSP phenotypes \( 3.7 \) vs. \( 5.8; p = 0.10 \).

**Neuropsychological performance**

As shown in Table 3, there were significant differences between the groups on processing speed, \( t(19) = -4.10, p = 0.001, d = 1.66 \), with the PSP-RS group showing significantly slower general processing speed \( \text{mean = -0.36, SD = 0.89} \) than the ‘brainstem predominant’ PSP group \( \text{mean = 0.76, SD = 0.35} \). Similarly, the PSP-RS group performed significantly worse \( \text{mean = -0.27, SD = 0.65} \) than those in the ‘brainstem predominant’ PSP group \( \text{mean = 0.54, SD = 0.69} \) on the executive function index \( t(19) = -2.63, p = 0.02, d = 1.20 \); the effect size of these differences was large. In contrast, no significant group differences were observed for the memory, \( t(19) = -1.01, p = 0.32, d = 0.47 \), language \( t(19) = 3.83, p = 0.42, d = 0.92 \), working memory \( t(19) = 0.41, p = 0.19, d = 0.62 \), and visuospatial function indices, \( t(19) = -1.13, p = 0.28, d = 0.30 \).

As shown in Table 4, supplementary analyses comparing PSP-P and PSP-PAGF from a clinical perspective revealed mild to moderate impairments on measures of processing speed, verbal memory and fluency for all individuals in the PSP-P group. In contrast, no individuals with PSP-PAGF demon-

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**Table 1.** Neuropsychological tests and corresponding baseline measures

| Tests                      | Baseline (Y/N) | Baseline measures/modifications                                                                 |
|----------------------------|----------------|-----------------------------------------------------------------------------------------------|
| Processing speed           |                |                                                                                               |
| TMT\(^{24}\)               | Y             | Stimuli presented at eye level                                                                 |
| Victoria Stroop\(^{25}\)   | Y             | Stimuli presented at eye level                                                                 |
| Working memory             |                |                                                                                               |
| Digit span\(^{26}\)        | Y             | Baseline for controlling speed of speech                                                       |
| Spatial span\(^{27}\)      | N             |                                                                                               |
| Memory                     |                |                                                                                               |
| RAVLT\(^{27}\)            | N             |                                                                                               |
| VR\(^{28}\)                | Y             | Modified scoring; motor errors performed in the copy score were not deducted from the recall score |
| Executive function         |                |                                                                                               |
| WCST-6\(^{29}\)           | N             |                                                                                               |
| COWAT\(^{30}\) (Phonemic) | Y             | Baseline for controlling speed of speech                                                       |
| Victoria Stroop\(^{30}\)   | Y             | Stimuli presented at eye level                                                                 |
| TOL\(^{31}\)              | Y             | Removed time limit                                                                             |
| Language                   |                |                                                                                               |
| BNT\(^{32}\)              | Y             | Stimuli presented at eye level                                                                 |
| COWAT\(^{30}\) (Semantic) | Y             | Baseline for controlling speed of speech                                                       |
| Visuospatial function      |                |                                                                                               |
| VOSP\(^{33}\)             | Y             | Stimuli presented at eye level                                                                 |

BNT: Boston Naming Test, COWAT: Controlled Oral Word Association Test, RAVLT: Rey Auditory Learning Test, TMT: Trail Making Test, TOL: Tower of London test, VOSP: Visual Object and Spatial Perception Battery, VR: Visual Reproduction, WCST-64: Wisconsin Card Sorting Test-64.

**Table 2.** Demographic and clinical characteristics of the PSP phenotypes

| Demographical and clinical information                  | Typical PSP (PSP-RS) M (SD) Range | ‘Brainstem predominant’ PSP M (SD) Range | \( p \) |
|---------------------------------------------------------|------------------------------------|----------------------------------------|------|
| Age                                                     | 69.93 (5.66) (61–82)               | 67.67 (15.63) (51–82)                  | 0.40 |
| Education                                               | 10.79 (2.69) (8–16)                | 13.33 (4.16) (10–18)                   | 0.26 |
| Disease duration                                        | 3.68 (1.60) (2–7)                  | 5.83 (2.51) (4–9)                      | 0.10 |
| PSPRS                                                   | 35.20 (10.40) (17–50)              | 35.67 (14.47) (19–45)                  | 0.09 |
| HYRS                                                    | 3.06 (0.63) (2–4)                  | 4.00 (1.00) (3–5)                      | 0.16 |
| On levodopa                                             | 6/14                               | 4/7                                    | 0.10 |

Age, education and disease duration are in years. PSP: progressive supranuclear palsy, PSP-RS: PSP-Richardson’s syndrome, M: mean, SD: standard deviation, PSPRS: progressive supranuclear palsy rating scale, HYRS: Hoehn and Yahr Stage of Illness Rating Scale.
strated clinically significant impairment (≤ 1.5 SD) in any cognitive domains.

**DISCUSSION**

This study shows that typical PSP-RS has a cognitive profile that is distinct from the cognitive profile of the ‘brainstem predominant’ PSP phenotypes (PSP-P and PSP-PAGF). Specifically, we found significantly greater executive function deficits and significantly slower speed of information processing in the PSP-RS subgroup compared to the ‘brainstem predominant’ PSP phenotypes.3,4,8 Specifically, the greater executive dysfunction documented in the current PSP-RS cohort corresponds well to reports of greater frontal pathology in this group.5,8 The more pronounced psychomotor slowing also corresponds well to the more severe subcortical pathology in the PSP-RS group.4

Clinically-driven consideration of cognition in the ‘brainstem predominant’ PSP phenotypes indicated that it may be possible to identify discrete cognitive profiles in PSP-P and PSP-PAGF. Specifically, none of the individuals in the PSP-PAGF group demonstrated clear evidence of cognitive impairment on any domain of function, whereas all of the individuals in the PSP-P group showed mild to moderate impairment in processing speed, memory and fluency.

PSP-RS has a shorter naturally occurring disease

**Table 3.** Between group comparisons [means (SD)] of the typical and ‘brainstem predominant’ PSP phenotypes on cognitive indices and the mean performance of the ‘brainstem predominant’ PSP phenotypes relative to age- and education-stratified normative data

| Cognitive domains | Typical PSP (n = 14) | ‘Brainstem predominant’ PSP (n = 7) | T   | P   |
|-------------------|----------------------|-------------------------------------|-----|-----|
| **Executive function** | -0.27 (0.65) | 0.54 (0.69) | -2.63 | 0.02†† |
| **Processing speed** | -0.36 (0.89) | 0.76 (0.35) | -4.10 | 0.001†† |
| **Memory** | -0.22 (0.83) | 0.18 (0.88) | -1.01 | 0.32 |
| **Language** | -0.24 (0.76) | 0.51 (0.86) | 3.83 | 0.42 |
| **Working memory** | -0.34 (0.86) | 0.41 (1.14) | 0.41 | 0.19 |
| **Visuospatial function** | -0.38 (4.5) | 1.13 (1.0) | -1.13 | 0.28 |

Cohen’s d = 1.20
Cohen’s d = 1.66

*Wisconsin Card Sorting Test-64; Controlled Oral Word Association Test (phonemic)*; Victoria Stroop-Color/dot ratio*; Tower of London*; Trail Making Test-Part A*; Victoria Stroop-dot*; Rey Auditory Verbal Learning Test; Visual Reproduction*; Boston Naming Test*; Semantic Fluency*; Digit Span-forward span and backward span difference score*; Spatial span-forward span and backward span difference score; Visual Object and Space Perception Battery*; (** = presentation modification; †† = scoring modification; §§ = removed time limit). †††Indicate statistically significant results; z-scores are relative to the means and standard deviations of the complete sample. PSP: progressive supranuclear palsy, SD: standard deviation.

**Table 4.** Mean performances of the PSP phenotypes relative to age- and education-stratified normative data

| Cognitive domains | Typical PSP-RS (n = 14) | ‘Brainstem-predominant’ PSP | PSP-P (n = 3) | PSP-PAGF (n = 4) |
|-------------------|--------------------------|-----------------------------|---------------|------------------|
| **Executive function** | -1.8 (-2.6, 1.0)* | -1.5 (-1.7, -1.2)* | -0.3 (-1.8, 1.3) |
| **Processing speed** | -4.9 (-10.6, -0.1)* | -2.1 (-2.6, -1.0)* | -0.4 (-2.2, 0.7) |
| **Memory** | -1.7 (-2.3, -1.1) | -1.6 (-1.7, -1.5) | -1.0 (-2.5, 0.7) |
| **Language** | -1.9 (-2.9, -0.6) | -1.5 (-1.9, -0.8) | -0.5 (-1.5, 0.6) |
| **Working memory** | -0.4 (-2.2, 0.6) | -0.2 (-1.7, 0.6) | -1.2 (-1.4, 1.4) |
| **Visuospatial function** | -1.6 (-3.3, -0.1) | -1.1 (-2.1, -0.1) | -0.4 (-1.3, 0.7) |

*Indicates impairment (≥ 1.5 SD from age- and/or education-adjusted normative mean). Mild impairment = 1.5–2.0 SD below the normative mean; moderate impairment = 2.1–3.0 SD below the normative mean; severe impairment ≥ 3.0 SD below the normative mean. PSP: progressive supranuclear palsy, PSP-RS: PSP-Richardson’s syndrome, PSP-P: PSP-parkinsonism, PSP-PAGF: PSP-pure akinesia with gait freezing, SD: standard deviation.
course than the ‘brainstem predominant’ PSP phenotypes, but the groups in this study were well matched in terms of disease severity. It is therefore unsurprising that the PSP-RS group trended ($p = 0.10$) toward having a shorter mean disease duration (3.68 years) than the ‘brainstem predominant’ PSP phenotypes (5.33; 5.50 years). This trend is unlikely to be the cause of the group differences in cognitive ability, as the PSP-RS group performed more poorly on cognitive assessment while being earlier in their disease duration than the ‘brainstem predominant’ PSP phenotypes. Given that the groups were well matched in terms of disease severity, it seems probable that the relatively greater impairment in the PSP-RS group represents a real group difference between these phenotypes.

The relatively small size of the patient sample in the current study and the fact that clinical diagnoses were made in vivo limits the interpretation of the results. However, all of the cases included in the current study have been seen at a tertiary center with a high rate of clinical diagnostic accuracy confirmed by pathological examination. Moreover, current significant findings together with the large effect sizes despite the small size of the PSP groups suggest that the reported group differences in cognition are clinically meaningful. The small sample size may, however, have resulted in insufficient statistical power to identify subter group differences in other cognitive domains. Future work with larger cohorts would be able to address this issue.

The field of PSP research is moving away from considering PSP as a single clinical entity.\textsuperscript{1,3,6,7} This prospective study offers the first clear evidence that typical PSP-RS can be differentiated from two ‘brainstem predominant’ PSP phenotypes (PSP-P and PSP-PAGF) on the basis of cognitive ability. Specifically, individuals with PSP-RS have more impaired executive functioning and processing speed than individuals with the ‘brainstem predominant’ PSP phenotypes. The current findings provide preliminary evidence that PSP-RS and ‘brainstem predominant’ PSP phenotypes may be differentiated by the severity of cognitive deficits. Further studies, with larger sample sizes are clearly needed, but this study suggests that such work may be clinically fruitful.

**Conflicts of Interest**

The authors have no financial conflicts of interest.

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