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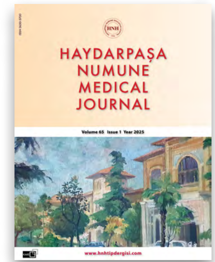

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ORIGINAL ARTICLE

Neuropsychometric Differences in Posterior Cortical Atrophy and Alzheimer's Disease

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Abstract

Introduction: Posterior cortical atrophy (PCA), a rare syndrome also known as the visual variant of Alzheimer's disease, has been the subject of limited neuropsychological research. This study aimed to examine neuropsychometric deterioration patterns in PCA and compare them with those in typical Alzheimer's disease (AD).

Methods: The study included 19 patients diagnosed with AD (n=12) or PCA (n=7) who were treated at a private hospital. Neuropsychometric data assessing cognitive domains including attention, memory, executive functions, visuospatial skills, and language were collected.

Results: Statistical analysis revealed that the PCA group had significantly higher long-term spontaneous recall scores on the Öktem Verbal Memory Processes Test (p=0.021) compared with the AD group. Additionally, Benton Face Recognition Test scores were significantly lower in the PCA group (p=0.032). Although not statistically significant, immediate visual memory scores were lower in the PCA group (p=0.092) compared with AD patients.

Discussion and Conclusion: This study aimed to facilitate differential diagnosis through neuropsychological tests. Although this aim was not fully achieved, the results are valuable. Larger studies with more comprehensive test batteries may provide further insight into differential diagnosis and support cognitive rehabilitation approaches.

Keywords: Alzheimer's disease; neurodegenerative diseases; neuropsychologic tests; posterior cortical atrophy.

Typical Alzheimer's disease (AD) is primarily characterized by an amnestic syndrome associated with hippocampal and temporoparietal atrophy, typically emerging around the age of 65 [1]. The initial pathology in the medial temporal lobes leads to memory impairment and functional deficits in learning new information [2,3]. However, early-stage variant phenotypes of Alzheimer's disease that exhibit focal atrophy can manifest as frontal variants, language variants (left parietal), and visual–perceptual variants (right parietal) [1,4].

Posterior cortical atrophy (PCA), also known as the visual variant of Alzheimer's disease, is a rare syndrome resulting from neurodegeneration in the parietal, occipital, and occipitotemporal cortices [5]. In this syndrome, impairments in the ventral pathway (occipitotemporal pathway) may manifest as components of Gerstmann syndrome (acalculia, finger agnosia, and right-left disorientation), while dysfunctions in the dorsal pathway (occipitoparietal pathway) may lead to components of Balint's syndrome (simultanagnosia, optic ataxia, and oculomotor apraxia)

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[6,7]. Additionally, symptoms such as alexia, visual agnosia, and transcortical sensory aphasia [6], as well as dressing apraxia, ideomotor apraxia, or constructional apraxia, can also be observed [7]. Homonymous hemianopia is an early indicator of PCA. Moreover, structural and functional impairments in the temporoparietal cortex can lead to issues such as a decline in verbal fluency, anomia, and working memory problems [8]. While episodic memory, executive functions, and language skills appear to be preserved in the early stages of PCA, as the disease progresses to dementia, a comprehensive deterioration of nearly all cognitive functions can occur [5].

Posterior cortical atrophy is considered a challenging condition to diagnose. Patients are often examined by several ophthalmology specialists before the neurological nature of the disease is recognized [6]. PCA causes impairments across multiple cognitive domains, although not all of these impairments are observed in every patient. As suggested by studies in the literature, each PCA patient presents with unique features and cognitive deficits. Some of these impairments, such as simultanagnosia, may be overlooked during neuropsychometric assessments unless specifically addressed.

The aim of this study was to compare the neuropsychometric profiles of patients with PCA with those of patients with typical Alzheimer's disease and, through this comparison, to identify characteristic patterns of cognitive impairment in PCA as assessed by neuropsychometric evaluation in dementia clinics.

Materials and Methods

This retrospective study was conducted between 2017 and 2020 at the Neurology Clinic of Medipol University Hospital. Data were collected from patients diagnosed with PCA and AD. Seven patients with PCA and twelve patients with AD were included in the study. Due to the rarity of PCA [9], only 10 cases were collected over a 3-year period, with three suspected cases excluded. The diagnoses were made based on the clinical criteria for Alzheimer's disease revised in 2011 by the NINCDS (National Institute of Neurological and Communicative Disorders and Stroke) and ADRDA (Alzheimer's Disease and Related Disorders Association), as well as the diagnostic criteria for PCA revised by the Posterior Cortical Atrophy Study Group in 2015 [10,11]. Patients diagnosed with other diseases that could cause cognitive impairment and illiterate patients were excluded from the study. Patients with primary eye diseases were excluded from the PCA group. Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki. The study was approved by the Medipol University Non-Interventional Clinical Research Ethics Committee (approval number 10840098-604.01.01-E.20637).

Study Design

Neuropsychometric data routinely administered at the Medipol University Hospital Dementia Clinic were used for both PCA and AD patients. The following tests were administered to assess cognitive domains:

- Attention: Forward and backward digit span tests from the Wechsler Memory Scale–Revised (WMS-R) battery [12].
- Executive Functioning: Category fluency test, phonemic fluency test, fruit-name pairing test [13], proverb interpretation test [14], similarity test [15], Stroop test [16], clock drawing test [17].
- Memory Assessment: Öktem Verbal Memory Test (ÖSBST) [18], WMS-R logical memory test, WMS-R visual reproduction test [12].
- Visual Perceptual Functioning: Benton Face Recognition Test [19].
- Language Skills: Boston Naming Test [20].

Additionally, the Standardized Mini Mental Status Examination (MMSE) [21] and the Clinical Dementia Rating (CDR) scale [22] were also used. The data obtained from these assessments were statistically analyzed.

Statistical Analysis

The data obtained in the present study were analyzed using the SPSS 17 for Windows package program. Patient characteristics were reviewed using descriptive statistics. Discrete variables are presented as count (%) and continuous variables as mean (SD), as appropriate. For comparisons of demographic characteristics and neuropsychological features between patients with PCA and those with AD, the independent-samples Mann–Whitney U test was applied to non-normally distributed continuous variables, and the Pearson chi-square test was applied to categorical variables. The limit for statistical significance was set at $p<0.05$.

Results

The mean age of patients with PCA was 62.42±9.48 years, whereas the mean age of patients with AD was 73.41±8.75 years. Patients with PCA were significantly younger than those with AD. Demographic characteristics and general cognitive features of the participants are shown in Table 1. Apart from age, no significant differences were found

Table 1. Demographic and global cognitive features of patients

	PCA		AD		
	Min-max	Mean±SD	Min-max	Mean±SD	p
Age (year)	55–82	62.42±9.48	61–85	73.41±8.75	0.011*[a]
Education (year)	5–15	7.71±4.11	0–11	4.83±.3.45	0.107[b]
MMSE	13–22	17±0.74	10–22	16.75±4.02	0.276[a]
CDR	1–2	1.4±0.44	1–2	1.33±0.44	0.837[a]
	n	%	n	%	
Gender					
Female	4	57.1	7	58.3	0.96[b]
Male	3	42.9	5	41.7	

[a]: Mann-Whitney test; [b]: Chi-square test. *: p<.05. PCA: Posterior cortical atrophy; AD: Alzheimer disease; SD: Standard deviation; MMSE: Standardize mini mental test; CDR: Clinical dementia rating scale.

Table 2. Neuropsychometric profile in PCA and AD

	PCA		AD		
	Min-max	Mean±SD	Min-max	Mean±SD	p
Attention					
Digit Span-F	3–6	4.57±1.27	3–5	4±0.95	0.284
Digit Span-B	0–6	2.14±2.03	0–4	1.58±1.16	0.548
Memory					
I-Visual memory	0–7	1.57±2.63	0–6	3.25±1.86	0.092
L-Visual memory	0–7	0.85±1.21	0–6	1±2.33	0.383
I-Story memory	0–16	8.42±5.65	0–18	8.35.19	0.899
L-Story memory	0–20	8.28±7.76	0–17	5.83±5.89	0.470
ÖVMPT-I	0–5	2.85±1.95	0–4	1.91±1.50	0.259
ÖVMPT-L	15–126	58.57±37.99	15–68	44.41±14.85	0.612
ÖVMPT-S	0–15	5±5.29	0–4	0.75±1.42	0.021*
ÖVMPT-R	0–13	4.14±4.87	0–10	4.75±3.19	0.491
ÖVMPT-DR	0–15	9.42±5.79	1–12	7.41±3.50	0.330
Executive f.					
Fruit-name fluency	0–9	4.57±3.04	0–9	4.58±2.96	1
Phonemic fluency	0–35	17.85±13.49	0–28	10.75±9.94	0.252
Categorical fluency	3–14	9.57±4.15	4–14	9.83±3.27	0.966
Abstract Thinking	0–4	2±1.52	0–5	2.75±1.42	0.319
Similarity	1–10	5.71±2.87	2–10	5.08±2.15	0.521
Clock drawing	0–3	1.14±1.34	0–4	2.16±1.80	0.175
Stroop-TD	46–243	96.28±72.14	8–286	129.16±67.84	0.271
Stroop error	0–40	13.42±14.85	2–46	15.33±12.61	0.524
Visuoper. skills					
BFRT	25–37	31.57±4.96	30–42	37.08±3.67	0.032*
Language skills					
BNT	11–27	19.57±5.82	9–24	16.33±3.98	0.204

*: p<.05. PCA: Posterior cortical atrophy; AD: Alzheimer disease; SD: Standard deviation; Digit Span-F: Digit span forward; Digit Span-B: Digit span backward; I-Visual memory: Immediate visual memory; L-Visual memory: long-term visual memory; I-Story memory: Immediate story memory; L-Story memory: Long-term story memory; ÖVMPT: Öktem verbal memory process test; I: Immediate; L: Learning; S: Spontaneous; R: Recognition; DR: Delayed recall; executive func.: Executive function; Stroop-TD: Stroop time difference; Visuoper. Skills: Visuoperceptual skills; BFRT: Benton facial recognition test; BNT: Boston naming test; Global Cog. Skill: Global cognitive skills; MMSE: Mini mental state examination; CDR: Clinical dementia rating scale.

between the two groups in terms of other demographic data and general cognitive characteristics (p>0.05). Both groups demonstrated similar cognitive features.

No significant differences were found in attention scores, as measured by the forward and backward digit span tests of the WMS, between patients with PCA and AD (p>0.05). WMS immediate visual memory scores were lower in patients with PCA compared with those with AD, although the difference approached statistical significance (p=0.092). WMS long-term visual memory scores were also relatively lower in patients with PCA, whereas WMS story memory and verbal memory scores were lower in patients with AD. Among these measures, only the verbal memory spontaneous recall score reached statistical significance (p=0.021), while the other parameters did not (p>0.05).

When executive function performance was examined, phonemic fluency scores were higher in the PCA group; however, no statistically significant differences were observed in the other fluency tests (p>0.05). Similarly, no significant differences were found in abstract reasoning or similarity tests. The clock drawing test, which assesses both executive function and visual perception, revealed slightly lower scores in patients with PCA, but this difference was not statistically significant (p>0.05). Likewise, no significant difference was observed in Stroop test scores (p>0.05) (Table 2).

Evaluation of visual–perceptual skills using the Benton Face Recognition Test showed that patients with PCA had significantly lower scores than patients with AD (p=0.032). In the Boston Naming Test, patients with PCA scored higher than those with AD; however, this difference was not statistically significant (p>0.05).

Discussion

In our study comparing the neuropsychological profiles of patients with PCA and AD, patients with PCA had statistically higher spontaneous recall scores in verbal memory than patients with AD, but significantly lower Benton Face Recognition Test scores. Immediate visual memory scores were lower in patients with PCA than in those with AD, although this difference approached statistical significance.

PCA is a dementia subtype that occurs at a younger age than AD [9]. This explains the significant age difference between the two groups and does not pose a disadvantage for the interpretation of the results. Moreover, the similarity of the two groups in terms of education level, MMSE scores, and dementia stage provides a solid baseline for the discussion of the findings.

When visual memory performance, one of the distinctive cognitive impairments in PCA, was compared, patients with PCA were found to have lower immediate visual memory scores than patients with AD, with the difference approaching statistical significance. This finding is consistent with the literature [23–25].

In Alzheimer's disease, the first lesions typically appear in areas associated with memory and learning, such as the hippocampus and the association cortex, and the most common complaint in patients with AD is forgetfulness [26]. Studies comparing the clinical presentations of AD and PCA report relatively small differences; however, memory impairment is generally more pronounced in AD than in PCA. Kas et al. [27] reported that episodic memory is more severely affected in typical AD, and Peng et al. [28] showed that short-term memory and spontaneous retrieval skills are more impaired in patients with AD than in those with PCA in neuroimaging-based studies. In the present study, spontaneous recall was statistically more impaired in patients diagnosed with AD than in those diagnosed with PCA, in line with the literature.

PCA is a neurodegenerative disease characterized clinically by severe visual–perceptual problems. Visual–perceptual disorders arise from dysfunction of the dorsal (occipitoparietal) and ventral (occipitotemporal) visual pathways. These higher-order visual–perceptual deficits represent the core clinical and cognitive features that define the PCA syndrome [29]. In our study, the mean BRFT scores of patients in the PCA group indicated severe impairment relative to age- and education-adjusted norms. When compared with the AD group, patients with PCA demonstrated significantly worse visual–perceptual abilities. Valenti et al. [30], in a neuroimaging study of patients with amyloid deposition in posterior brain regions without dementia, suggested that posterior amyloid accumulation, as observed in PCA, leads to visual–perceptual impairments. They examined the relationship between amyloid pathology and neurocognitive performance and reported impairments in the BRFT in 23% of participants and in the Benton Judgment of Line Orientation Test in 13.6%, findings associated with parietal lobe pathology. Previous studies consistently indicate that patients with PCA exhibit more severe visual–perceptual disturbances than patients with AD, and the results of our study confirm this observation [23,26,31–33].

Studies comparing PCA and AD emphasize that visual–perceptual deficits predominate in PCA, whereas memory impairment is more prominent in AD. The findings of the present study support this distinction, indicating that visual–perceptual dysfunction is a hallmark of PCA, while memory disturbances are more characteristic of AD [23,24].

Identifying the distinctive neuropsychological features of PCA, known as the visual variant of AD, may reduce misdiagnosis and improve recognition of PCA in clinical practice. However, studies on PCA, an atypical form of Alzheimer's disease, remain limited both in the international literature and in our country, which underscores the value of the present study.

Limitation

The small sample size is a limitation of this study. Given the rarity of PCA, this limitation is understandable. Nonetheless, the results support findings from previous studies.

Conclusion

The data from this study suggest that, compared with patients with AD, patients with PCA experience more prominent visual–perceptual deficits and relatively better memory performance. Future studies with larger sample sizes, more comprehensive neuropsychometric test batteries, and advanced imaging techniques may provide further insight into the relationships between cognitive impairments and contribute to improved differential diagnosis and rehabilitation strategies.

Ethics Committee Approval: The study was approved by Medipol University Non-Interventional Clinical Research Ethics Committee (No: 10840098-604.01.01-E.20637, Date: 28.06.2018).

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