



## Posterior cortical atrophy: Clinical, neuroimaging and neuropsychological features

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### Abstract

Posterior cortical atrophy (PCA) represents a rare variant of degenerative dementia, predominantly marked by visuospatial deficits and cognitive impairment. The diagnostic complexity consist of atypical symptoms at onset and non-specific neuroimaging findings. In this retrospective case study, we investigated clinical, neuropsychological, and imaging aspects in five cases with pure PCA. 754 patients who presented to the dementia outpatient clinic of Çukurova University Faculty of Medicine Neurology Department between 2013-2022 were retrospectively analyzed, and 5 cases diagnosed with PCA were included in the study. Mini-Mental State Examination (MMSE), clock drawing test (CDT), cookie thief test, simultanagnosy tests (Arcimboldo pictures, Navon letters), and imaging findings of the cases were documented. The average age of the subjects was 60.6 years, with an onset typically occurring at a presenile stage. Simultanagnosia was a consistent finding in all patients, with some exhibiting additional visuospatial deficits. Mini-Mental State Examination (MMSE) scores averaged 20.20, and clock drawing test results indicated impairment. Notably, 40% of the patients displayed depressive symptoms, often alongside visual complaints as a common presentation. Crucially, all patients retained speech fluency. Our findings underscore the significance of employing neurocognitive assessments, particularly the clock drawing test, when evaluating patients with complaints related to visuospatial and cognitive functions. Early diagnosis and intervention are pivotal for effectively managing PCA.

**Keywords:** posterior cortical atrophy, dementia, simultanagnosia, early diagnosis, neurocognitive tests

### 1. Introduction

Posterior cortical atrophy (PCA) is a rare degenerative dementia syndrome, typically presenting with signs of cortical visual dysfunction usually followed by memory impairment (1). It has been proposed that PCA could be a subtype of Alzheimer's disease. In addition, the most common pathological findings in PCA are tau neurofibrillary tangles and beta-amyloid neuritic plaques, which are characteristic of Alzheimer's disease (2). However, non-Alzheimer pathologies such as subcortical gliosis, Creutzfeldt-Jakob disease, Lewy body dementia, Pick's disease, and corticobasal degeneration may be observed in these patients (3). Presenile onset, the prominence of visuospatial deficits, preserved verbal fluency, and memory problems being more obscure helps differ from Alzheimer's disease (4-6).

Simultanagnosia, ocular apraxia, agraphia, finger agnosia, acalculia, dressing apraxia, alexia, and prosopagnosia are the main clinical manifestations of the disease (7). However, these symptoms are not routinely assessed in neurology clinics. Brain MRI scans of PCA cases with nonspecific findings are often reported as "normal". Due to atypical symptoms, patients are losing unnecessary time in psychiatry or ophthalmology clinics, being undiagnosed and under unnecessary medications (antidepressants, antipsychotics, etc) (8,9).

This study aims to investigate the basic parameters that

should be considered in outpatient clinics to facilitate early and accurate diagnosis of PCA cases.

### 2. Materials and Methods

A total of 754 cases who applied with symptoms of dementia in the outpatient clinic of the Neurology Department of Çukurova University Faculty of Medicine between 2013 and 2022 were retrospectively examined, and 5 cases identified as pure PCA according to the posterior cortical atrophy diagnostic criteria defined by Crutch et al. in 2017 (10) were included. The education period of the patients was stated in years. Patients with drug use that may affect cognitive functions, lesions that may affect optic nerves and pathways, findings such as vascular lesions, masses in brain MRI examinations, distinct electrolyte abnormality, kidney failure, liver failure, vitamin B12 deficiency, thyroid function disorder, etc., which may cause cognitive impairment were excluded. The neuropsychological evaluations [Mini-Mental State Examination (11), clock drawing test (12), cookie theft (13), Arcimboldo objects (14), Navon letters simulation (15)] and imaging findings of the cases were documented.

#### 2.1. Neurocognitive Tests

Neurocognitive tests were conducted in a quiet room by a neurologist experienced in behavioral neurology, lasting approximately 30 minutes.

**2.2. Mini-Mental State Examination (MMSE)**

Both the 2 different versions according to the education level were performed and patients' orientation, registration memory, attention and calculation, sentence construction, shape copying parameters were assessed.

**2.3. Clock Drawing Test (CDT)**

The patient is asked to draw a circle on a blank piece of paper to form a clock. Afterward, they are asked to place the numbers of the clock within the circle neatly. Once the numbers are placed, they are asked to draw the hands to indicate ten past eleven. The scoring of the test is as follows: (1=No drawing attempt or explanation. 2=The impression gained from the drawing suggests the patient partially understood the explanation, but the figure does not resemble a clock. 3=The placement of the clock dial and numbers is unrelated. The hands are not drawn. 4=There is a more significant disorder in the numbering sequence. The integrity of the clock dial is also disrupted. 5=The clock numbers are drawn intensively in one place or in the opposite direction. 6=The clock numbers are drawn correctly, but inappropriate use of the hands (for example, writing like a digital clock). 7=The clock numbers are drawn correctly, but the distinction between the hour and minute hands cannot be made. 8=The clock numbers are drawn correctly, but the placement of the hands is incorrect. 9=The clock numbers are drawn correctly, the minute hand placement is correct, the hour hand placement is not exact. 10=The clock numbers are drawn correctly, the placement of the hands is correct.)

**2.4. Cookie Theft Test**

The cases are shown a picture related to cookie theft (Fig. 1) and they are asked to describe everything they see in the picture and write as much as they can about what is seen in the picture. They should be encouraged to continue writing for two minutes. Scores range from 0 (no relevant text) to 4 (full explanation in grammatical sentences).

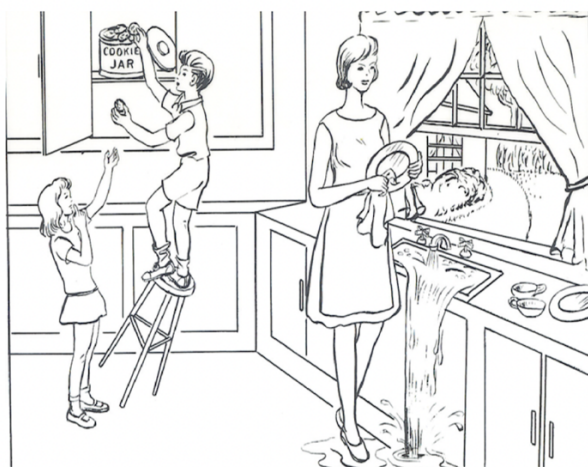


Fig. 1. The Cookie Thief Test

**2.5. Arcimboldo Pictures**

The cases are shown pictures by Italian painter Giuseppe Arcimboldo, who arranged fruits and vegetables in a way that

they resemble human portraits. The cases are expected to both individually express the fruits and vegetables and to see the portrait formed from these (Fig. 2).



Fig. 2. Arcimboldo portraits

**2.6. Navon Letters**

Navon letters are large letters made up of repeating smaller letters of a different kind. The cases are expected to express both these large letters and the other small letters that form the large letters (Fig. 3).

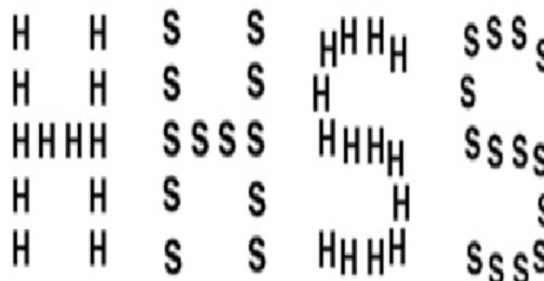


Fig. 3. Navon Letters Simulation

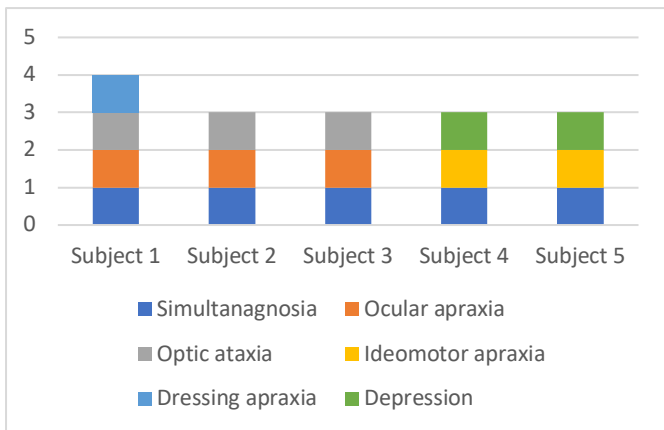
**3. Results**

Out of five cases diagnosed with PCA, three were male patients. The average age of the cases was 60.6±5.07 (55-66), and their educational level was determined as 7.2±5.84 (0-15) years. The onset age of the disease was 58.2±4.20 (53-63), and the duration from the onset of complaints to diagnosis was 2.4 ±1.14 (1-4) years. The speech was fluent in all cases. MMSE was 20.20±1.30 (19-22), and clock drawing test was 1.60±0.89 (0-2). All of the cases had simultanagnosia, three male cases had optic ataxia and ocular apraxia, and two female cases had ideomotor apraxia and depression. In the brain MRI examinations of the cases with parietooccipital atrophy, parietooccipital hypometabolism was observed in brain PET examinations. The detailed demographic, clinical, and imaging findings of all cases are summarized below (Table 1, Fig. 4).

**Table 1.** Clinical and PET/CT features of the subjects

	Subject 1	Subject 2	Subject 3	Subject 4	Subject 5
Age	55	58	66	66	58
Sex	M	M	M	F	F
Disease onset age	53	56	63	62	57
Education (years)	11	15	5	0	5
MMSE	20	19	19	21	22
CDT	2	0	2	2	2
Simultanagnosia	+	+	+	+	+
Ocular apraxia	+	+	+	-	-
Optic ataxia	+	+	+	-	-
Ideomotor apraxia	-	-	-	+	+
Dressing apraxia	+	-	-	-	-
Depression	-	-	-	+	+
<b>Hypoperfusion in PET/CT</b>	Bilateral occipitoparietal	Bilateral posterior parietal	Significant widespread involvement in bilateral occipital lobes	Bilateral occipitoparietal	Bilateral occipitoparietal

MMSE: Mini-Mental State Examination, CDT: Clock Drawing Test PET/CT: Positron emission tomography/computed tomography



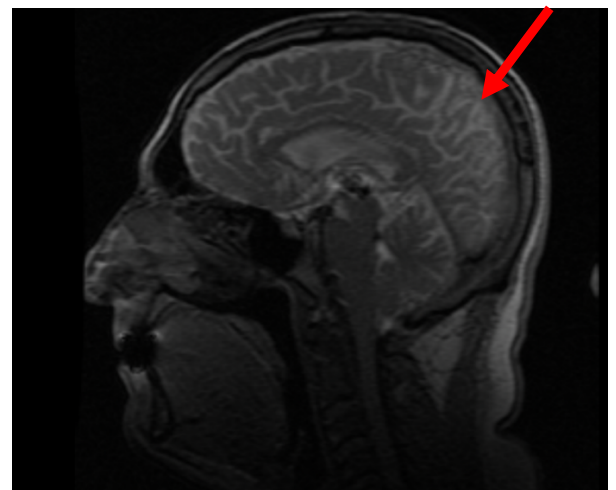
**Fig. 4.** Clinical Characteristics of the Subjects

**3.1. Case 1: A.K.**

The complaints of the 55-year-old male patient, a high school graduate, started as not being able to find the toilet at home and confusing the grocery shop list. He was unable to bath or dress appropriately. Recently, memory loss was added as a symptom with few narratives of being lost on the streets. According to his caregiver, his complaints started two years ago. There were no peculiarities in his personal or family history. In the neurological examination, he had impaired time orientation, ocular ataxia, dressing apraxia, and ocular apraxia. His speech was fluent.

MMSE: 20/30 (counting back, memory, time orientation). The clock drawing test was 2/10. He only read the small letters in the Navon letters. He looked at the Cookie Theft picture but could not interpret it. He only said fruits in Arcimboldo objects.

There was bilateral posterior parietal and hippocampal atrophy in Brain MRI (Fig. 5). Bilateral occipitoparietal hypometabolism was detected in Brain PET (Fig. 6).



**Fig. 5.** Atrophy in posterior regions in brain MRI



**Fig. 6.** Bilateral occipitoparietal hypoperfusion on brain PET

**3.2. Case 2: M.Y.**

A 58-year-old male patient, a university graduate and traffic expert, presented to the Ophthalmology clinic with a complaint of blurred vision. He had an unremarkable ophthalmological

evaluation and therefore was referred to our clinic. In addition, he stated progressive difficulty in reading, since two years. He stated that he had complaints of not being able to read for 2 years and that it was progressively getting worse.

There were no peculiarities in his personal or family history. In the neurological examination, he had ocular ataxia and ocular apraxia. His speech was fluent. MMSE: 19/30 (memory, counting back, and he couldn't copy intersecting pentagons). He only read the small letters in the Navon letters. He looked at the Cookie Theft picture but could not interpret it. He only said fruits in Arcimboldo objects.

There was posterior parietal atrophy in Brain MRI (Fig. 7), and bilateral posterior parietal hypometabolism was observed in Brain PET.

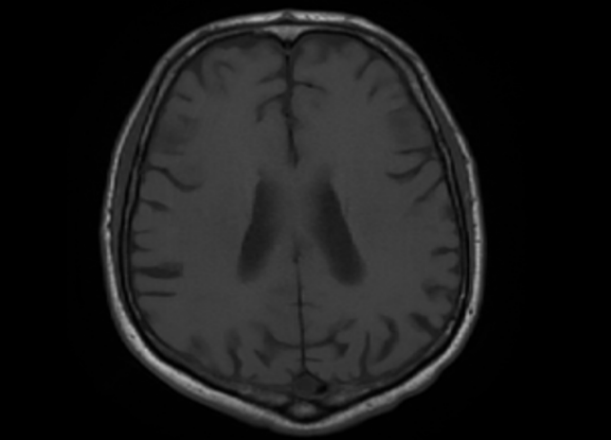


Fig. 7. Posterior parietal atrophy in brain MRI

### 3.3. Case 3: E.Ö.

A 66-year-old male patient, who graduated from primary school, applied to our clinic due to vision loss and perception problems. The patient, who had previously applied to the Ophthalmology clinic, was referred to our clinic when no ophthalmological pathology was found. It was learned that the patient's complaints have been for 3 years. There were no peculiarities in his personal or family history. In his neurological examination, he had ocular ataxia and ocular apraxia, and his visual acuity was natural. His speech was fluent. MMSE: 19/30 (orientation, memory, counting backwards, copying) was determined. The CDT was 2/10. He only read the small letters in the Navon letters. He looked at the Cookie Theft picture but could not interpret it. He only said fruits in Arcimboldo objects. Brain MRI showed biparietooccipital atrophy and partial bilateral hippocampus atrophy (Fig. 8). Brain PET showed widespread hypometabolism more evident in bilateral occipital lobes. (Fig. 9).

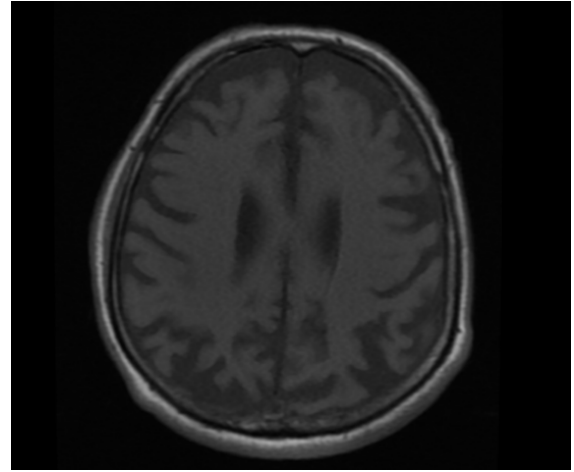


Fig. 8. Biparietooccipital atrophy in brain MRI

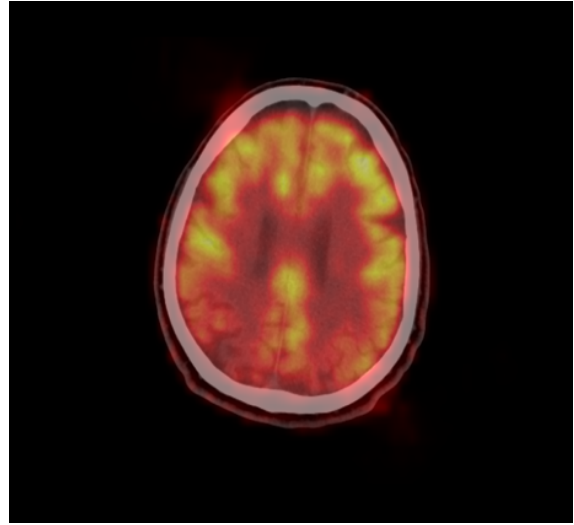


Fig. 9. Widespread hypometabolism more evident in bilateral occipital lobes in brain PET

### 3.4. Case 4: M.Ç.

The complaints of the 66-year-old female patient, who had no education, started two years ago by confusing the places of the rooms. The patient's relatives stated that the patient's appetite had decreased greatly, she did not want to do anything, she did not want to talk to anyone and she slept all the time.

There were no peculiarities in his personal or family history. In her neurological examination, her mood was depressed, her speech was fluent, and bilateral ideomotor apraxia was present. MMSE: 21/30, the clock drawing test was 2/10. She looked at the Cookie Theft picture but could not interpret it. She only read some small letters in the Navon letters. She could only say some fruits in the Arcimboldo objects.

In the brain MRI biparietal atrophy was detected (Fig. 10). In the brain PET, hypometabolism was observed in biparietooccipital (Fig 11).

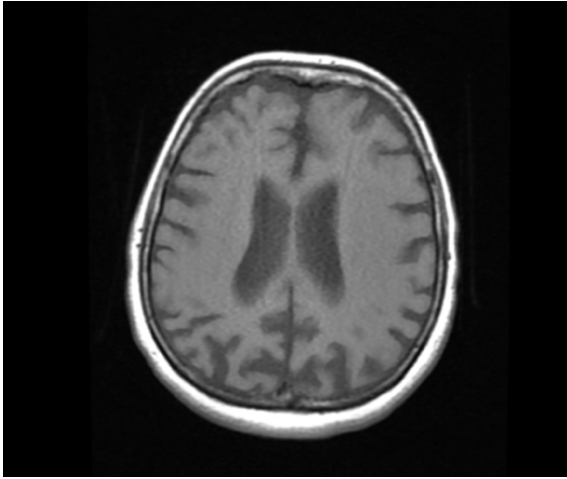


Fig.10. Biparietal atrophy in brain MRI

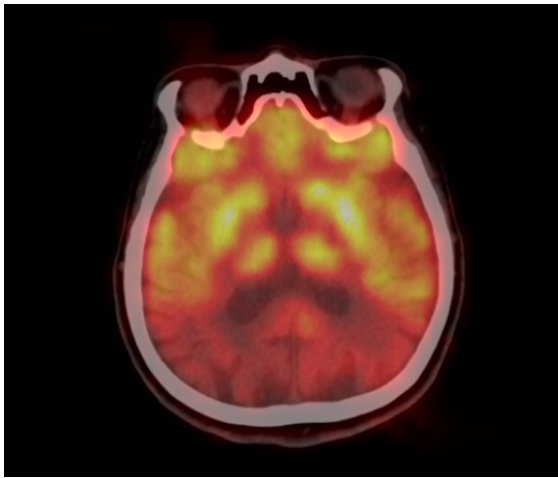


Fig. 11. Biparietooccipital hypometabolism in brain PET

### 3.5. Case 5: E.Y.

The complaints of the 58-year-old female patient, a primary school graduate, consists of not being able to find her belongings and crying for no reason for about 1 year. The patient's relatives stated that the patient, who was previously a very skilled woman in housework, no longer wanted to do any work, constantly blamed herself, and was constantly tired. There were no peculiarities in his personal or family history. In her neurological examination, her mood was depressed, her speech was fluent, alexia, agraphia, ideomotor apraxia was present. MMSE: 22/30 was determined. The clock drawing test was 2/10. She looked at the Cookie Theft picture but could not interpret it. She only read some small letters in the Navon letters. She could only say some fruits in the Arcimboldo objects. In brain MRI, bilateral posterior cortical atrophy was observed. In brain PET, bilateral occipitoparietal hypometabolism was detected.

## 4. Discussion

In this article, five cases recognized as PCA according to the criteria established as a result of the consensus of the PCA study group in 2017, have been described. (10) All of the cases presented with visuospatial complaints.

The most common initial complaints of the cases were reading problems, difficulty seeing the object, impairment in

navigation ability and depression. The most noticeable clinical finding of our patients was simultanagnosia. Speech was fluent in all cases. These findings of our cases are consistent with previously published PCA case series (16,17,18). In the case series of Shebani et al., the average onset age of the disease was 59.2, while in the study by Çolakoğlu et al., it was found to be 55.8. In our study, the onset age of the disease was 58.6, consistent with the literature. Also, the onset age of the disease in our study was significantly lower than Alzheimer's patients, similar to previous studies (19,20).

While a family history is frequently encountered in Alzheimer's disease, no similar history has been reported in any of the PCA series (21). Consistent with the literature, none of the patients in our study had a family history.

Depression may be evident in the early stages of PCA and making cognitive sign recognition ever more difficult. The early appearance of these symptoms is attributed to the relative preservation of insight (22). A study comparing clinical characteristics of PCA and AD patients found greater insight as well as higher levels of depression and antidepressant use in PCA patient and also delay diagnosis (23). Suárez-González et al found depression the most frequently detected neuropsychiatric disorder using the Neuropsychiatric Inventory in PCA patients (24). We detected depression in 40% of our cases, and these patients were referred to us from the psychiatric clinic. In consistency with literature diagnosis of these patients was delayed due to onset with psychiatric symptoms.

Several case series showed tendency for women to be diagnosed with depression at the onset of the disease (25,26). Our findings also were in line with the literature.

Although rarely visual and auditory hallucinations, delusions, disinhibition, euphoria, REM sleep behavior disorder, abnormal motor movements are documented in some patients with PCA, we did not detect these findings in any of our cases. Autopsy findings of all these cases reported in the literature were positive for Lewy body dementia (27).

The average MMSE of the patients in our study was  $20.20 \pm 1.30$  (19-22) and all patients had impaired CDT [ $1.60 \pm 0.89$ (0-2)]. Most common reason for admission were visual symptoms and 40% of our patients had previously been seen by an ophthalmologist. The average MMSE score in PCA in the literature was found to be  $20.9 \pm 5.69$ , the clock drawing test was found to be impaired in 85% of patients. The most common reason for admission were visual symptoms and 38% of the patients was previously seen by an ophthalmologist (28). Our findings were largely in line with the literature.

Neuroimaging studies in PCA have reported extensive hypometabolism of the bilateral posterior cortex, most prominently in the parietooccipital regions and temporooccipital regions (29,30,31). A few studies reported right hemisphere dominance, but it was observed that these

patients presented in the early stages (32,33). In our study, we detected hypometabolism and atrophy in bilateral parieto-occipital areas, which is consistent with the literature.

The most important limitation of our study is that it is retrospective and there is no control or AD group to compare clinical and PET findings. However, the fact that memory problems are at the forefront in AD and visual system complaints are not seen frequently among the initial findings significantly reduces the concerns about the clinical diagnosis of our cases (34). In addition, the inclusion of the occipital region in the brain PET examination in our cases removes us from the possibility of AD. Patients with dementia with Lewy bodies also show occipital hypometabolism (35). However, these patients have extrapyramidal symptoms and visual hallucinations that are not present in our patients. Secondly, the diagnosis of depression was made with the anamnesis taken from the patient and family, and a depression scale was not administered to the patients. However, the patient's relatives complained of depression rather than high cortical dysfunction. The fact that the most prominent finding in two of our cases was depression and that it was not detected at all in our other three cases emphasizes that this finding should be considered in detail in future studies on PCA. Thirdly, our number of patients was small and there were lack of follow-up in the patients for various reasons (application from different geographies, application only for the purpose of a committee report, the nature of the course of the disease, etc.). Therefore, only the first application findings were included in our study.

In conclusion, simultanagnosia was detected in all cases that presented with visuospatial complaints and were diagnosed with PCA in our study, and a significant impairment was observed in the clock drawing test. Performing these tests initially in cases presenting with complaints of impairment in visual perception and navigation ability will allow PCA cases, which have a diagnosis period of 1-4 years, to receive earlier diagnosis and treatment.

#### Conflict of interest

The authors declared no conflict of interest.

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None to declare.

#### Authors' contributions

Concept: Z.S.Ş., A.E., İ.B.G., Design: Z.S.Ş., A.E., İ.B.G., Data Collection or Processing: : Z.S.Ş., A.E., İ.B.G., Analysis or Interpretation: : Z.S.Ş., A.E., İ.B.G., Literature Search: Z.S.Ş., Writing: Z.S.Ş.

#### Ethical Statement

Approval was obtained from the Ethics Committee of the Çukurova University Faculty of Medicine before the study (6.01.2023/35/129).

#### References

1. Cerami C, Crespi C, Della Rosa PA, Dodich A, Marcone A, Magnani G et al. Brain changes within the visuo-spatial attentional network in posterior cortical atrophy. *J Alzheimers Dis.* 2015;43(2):385-95.
2. Torso M, Ahmed S, Butler C, Zamboni G, Jenkinson M, Chance S. Cortical diffusivity investigation in posterior cortical atrophy and typical Alzheimer's disease. *J Neurol.* 2021 Jan;268(1):227-239.
3. Esteves S, Ramirez Romero DA, Torralva T, Martínez Cuitiño M, Herndon S, Couto B et al. Posterior cortical atrophy: a single case cognitive and radiological follow-up. *Neurocase.* 2018 Feb;24(1):16-30.
4. Solyga VM, Western E, Solheim H, Hassel B, Kerty E. Posterior kortikal atrofi [Posterior cortical atrophy]. *Tidsskr Nor Laegeforen.* 2015 Jun 2;135(10):949-52.
5. DE Brito MH, Zampieri PF, Coutinho AM, Brucki SMD. Brain [18F]FDG PET-CT imaging in posterior cortical atrophy. *Arq Neuropsiquiatr.* 2019 Oct 24;77(10):752-753.
6. Whitwell JL, Graff-Radford J, Singh TD, Drubach DA, Senjem ML, Spychalla AJ et al. 18F-FDG PET in Posterior Cortical Atrophy and Dementia with Lewy Bodies. *J Nucl Med.* 2017 Apr;58(4):632-638.
7. Holden SK, Bettcher BM, Pelak VS. Update on posterior cortical atrophy. *Curr Opin Neurol.* 2020 Feb;33(1):68-73.
8. North C, Desai R, Saunders R, Suárez-González A, Bamiou D, Costafreda SG et al. Neuropsychological deficits in Posterior Cortical Atrophy and typical Alzheimer's disease: A meta-analytic review. *Cortex.* 2021 Oct;143:223-236.
9. Neitzel J, Ortner M, Haupt M, Redel P, Grimmer T, Yakushev I et al. Neuro-cognitive mechanisms of simultanagnosia in patients with posterior cortical atrophy. *Brain.* 2016 Dec;139(Pt 12):3267-3280.
10. Crutch SJ, Schott JM, Rabinovici GD, Murray M, Snowden JS, van der Flier WM et al. Alzheimer's Association ISTAART Atypical Alzheimer's Disease and Associated Syndromes Professional Interest Area. Consensus classification of posterior cortical atrophy. *Alzheimers Dement.* 2017 Aug;13(8):870-884.
11. Keskinoglu P, Ucku R, Yener G, Yaka E, Kurt P, Tunca Z. Reliability and validity of revised Turkish version of Mini Mental State Examination (rMMSE-T) in community-dwelling educated and uneducated elderly. *Int J Geriatr Psychiatry.* 2009 Nov;24(11):1242-50.
12. Manos PJ, Wu R. The ten point clock test: a quick screen and grading method for cognitive impairment in medical and surgical patients. *Int J Psychiatry Med.* 1994;24(3):229-44.
13. Nyffeler T, Pflugshaupt T, Hofer H, Baas U, Gutbrod K, von Wartburg R, Hess CW, Müri RM. Oculomotor behaviour in simultanagnosia: a longitudinal case study. *Neuropsychologia.* 2005;43(11):1591-7.
14. Kubon J, Sokolov AN, Popp R, Fallgatter AJ, Pavlova MA. Face Tuning in Depression. *Cereb Cortex.* 2021 Mar 31;31(5):2574-2585.
15. Navon D. Forest before the trees: the precedence of global features in visual perception. *Cogn Psychol* 1977; 9(3):353-83
16. Saleh C, Seidl U, Stegentschik K, Schumacher F, Fehrenbach RA. Posteriore kortikales Atrophie-Plus-Syndrom. Eine Kasuistik [Posterior cortical atrophy-plus syndrome. A case report]. *Fortschr Neurol Psychiatr.* 2020 Aug;88(8):528-531.
17. Mukku SSR, Chintala H, Nagaraj C, Mangalore S, Sivakumar PT, Varghese M. Posterior cortical atrophy variant of Alzheimer's

- dementia-A case report. *Asian J Psychiatr.* 2018 Jun;35:109-112.
18. Giunta M, Libri I, Premi E, Brattini C, Paghera B, Archetti S et al. Clinical and radiological features of posterior cortical atrophy (PCA) in a GRN mutation carrier: a case report. *Eur J Neurol.* 2021 Jan;28(1):344-348.
  19. Shebani Z, Nestor PJ, Pulvermüller F. What's "up"? Impaired Spatial Preposition Processing in Posterior Cortical Atrophy. *Front Hum Neurosci.* 2021 Dec 1;15:731104.
  20. Colakoglu BD, Kurt P, Kaya GC, Yener G. A Distinct dementia syndrome: posterior cortical atrophy: clinical and cognitive assesment, and brain spect findings *Archives of Neuropsychiatry,* 2010;47(3):201-207.
  21. Tang-Wai DF, Graff-Radford NR, Boeve BF, Dickson DW, Parisi JE, Crook R et al. Clinical, genetic, and neuropathologic characteristics of posterior cortical atrophy. *Neurology.* 2004 Oct 12;63(7):1168-74.
  22. Wong B, Lucente DE, MacLean J, Padmanabhan J, Quimby M, Brandt KD et al. Diagnostic evaluation and monitoring of patients with posterior cortical atrophy. *Neurodegener Dis Manag.* 2019 Aug;9(4):217-239.
  23. Mendez MF, Ghajarian M, Perryman KM. Posterior cortical atrophy: clinical characteristics and differences compared to Alzheimer's disease. *Dement Geriatr Cogn Disord.* 2002;14(1):33-40.
  24. Suárez-González A, Crutch SJ, Franco-Macías E, Gil-Néciga E. Neuropsychiatric Symptoms in Posterior Cortical Atrophy and Alzheimer Disease. *J Geriatr Psychiatry Neurol.* 2016 Mar;29(2):65-71.
  25. Aharon-Peretz J, Israel O, Goldsher D, Peretz A. Posterior cortical atrophy variants of Alzheimer's disease. *Dement Geriatr Cogn Disord.* 1999 Nov-Dec;10(6):483-7.
  26. Schmidtke K, Hüll M, Talazko J. Posterior cortical atrophy: variant of Alzheimer's disease? A case series with PET findings. *J Neurol.* 2005 Jan;252(1):27-35.
  27. Josephs KA, Whitwell JL, Boeve BF, Knopman DS, Tang-Wai DF, Drubach DA et al. Visual hallucinations in posterior cortical atrophy. *Arch Neurol.* 2006 Oct;63(10):1427-32.
  28. Yerstein O, Parand L, Liang LJ, Isaac A, Mendez MF. Benson's Disease or Posterior Cortical Atrophy, Revisited. *J Alzheimers Dis.* 2021;82(2):493-502.
  29. Singh TD, Josephs KA, Machulda MM, Drubach DA, Apostolova LG, Lowe VJ, Whitwell JL. Clinical, FDG and amyloid PET imaging in posterior cortical atrophy. *J Neurol.* 2015 Jun;262(6):1483-92
  30. Agosta F, Mandic-Stojmenovic G, Canu E, Stojkovic T, Imperiale F, Caso F, Stefanova E, Copetti M, Kostic VS, Filippi M. Functional and structural brain networks in posterior cortical atrophy: A two-centre multiparametric MRI study. *Neuroimage Clin.* 2018 Jun 12;19:901-910.
  31. Wang XD, Lu H, Shi Z, Cai L, Liu S, Liu S, Han T, Wang Y, Zhou Y, Wang X, Gao S, Ji Y. A Pilot Study on Clinical and Neuroimaging Characteristics of Chinese Posterior Cortical Atrophy: Comparison with Typical Alzheimer's Disease. *PLoS One.* 2015 Aug 12;10(8):e0134956
  32. Nestor PJ, Caine D, Fryer TD, Clarke J, Hodges JR. The topography of metabolic deficits in posterior cortical atrophy (the visual variant of Alzheimer's disease) with FDG-PET. *J Neurol Neurosurg Psychiatry.* 2003 Nov;74(11):1521-9.
  33. Koch G, Stefani A, Panella M, Giordano A, Schillaci O, Marfia GA. Posterior cortical atrophy with unilateral occipito-temporal degeneration. *J Neurol.* 2004 Dec;251(12):1530-1.
  34. Mendez MF, Monserratt LH, Liang LJ, Chavez D, Jimenez EE, Maurer JJ et al. Neuropsychological Similarities and Differences Between Amnesic Alzheimer's Disease and its Non-Amnesic Variants. *J Alzheimers Dis.* 2019;69(3):849-855.
  35. Yousaf T, Dervenoulas G, Valkimadi PE, Politis M. Neuroimaging in Lewy body dementia. *J Neurol.* 2019 Jan;266(1):1-26.