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Benson's Disease or Posterior Cortical Atrophy, Revisited

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Abstract

Background: D. Frank Benson and colleagues first described the clinical and neuropathological features of posterior cortical atrophy (PCA) from patients in the UCLA Neurobehavior Program.

Objective: We reviewed the Program's subsequent clinical experience with PCA, and its potential for clarifying this relatively rare syndrome in comparison to the accumulated literature on PCA.

Methods: Using the original criteria derived from this clinic, 65 patients with neuroimaging-supported PCA were diagnosed between 1995 and 2020.

Results: On presentation, most had visual localization complaints and related visuospatial symptoms, but nearly half had memory complaints followed by symptoms of depression. Neurobehavioral testing showed predominant difficulty with visuospatial constructions, Gerstmann's syndrome, and Balint's syndrome, but also impaired memory and naming. On retrospective application of the current Consensus Criteria for PCA, 59 (91%) met PCA criteria with a modification allowing for "significantly greater visuospatial over memory and naming deficits." There were 37 deaths (56.9%) with the median overall survival of 10.3 years (95% CI: 9.6–13.6 years), consistent with a slow neurodegenerative disorder in most patients.

Conclusion: Together, these findings recommend modifying the PCA criteria for "relatively spared" memory, language, and behavior to include secondary memory and naming difficulty and depression, with increased emphasis on the presence of Gerstmann's and Balint's syndromes.

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Keywords

Alzheimer's disease; balint's syndrome; gerstmann's syndrome; posterior cortical atrophy

INTRODUCTION

In 1988, D. FrankBenson and colleagues described five unusual patients with progressive visual difficulties from brain disease that they termed "posterior cortical atrophy" (PCA) [1]. All had environmental disorientation, elements of both Gerstmann's syndrome and Balint's syndrome, and difficulty reading. Their course was progressive and appeared most consistent with a neurodegenerative dementia. In 1994, Benson's group followed-up with an autopsy report showing Alzheimer neuropathology in one of the original patients, plus an additional rapidly progressive case who proved to have prion disease [2]. Subsequent autopsy series confirmed a predominance of a visual phenotype of Alzheimer's disease (AD) [3,4], although other disorders were also present on pathology, such as dementia with Lewy bodies (DLB), corticobasal syndrome (CBS) with corticobasal ganglionic degeneration (CBGD), and the Heidenham variant of Creutzfeldt-Jakob disease (CJD) [3, 5].

Posterior cortical atrophy (PCA) remains an incompletely understood syndrome characterized by early visually-related symptoms resulting from posterior cortical dysfunction. Investigators have proposed clinical Consensus Criteria for the diagnosis of PCA and have reported AD with relative hippocampal sparing as the most common neuropathology [6]. When AD is the cause, there is posterior cortical tau pathology with involvement of the frontoparietal network [7]. PCA particularly occurs as an early-onset (< 65 years of age) AD phenotype, where it has a prevalence of 13% or more [8]. Clinicians often misdiagnose patients with PCA as having primary visual disorders or even psychiatric conditions. Even when they recognize PCA, clinicians consider AD as the probable cause without excluding rarer causes such as DLB, CBGD, or CJD. The resulting delays in diagnosis may deprive those affected with PCA of opportunities to receive proper therapy or to participate in clinical trials.

Since Frank Benson's 1994, autopsy report, there have been many patients characterized in his Neurobehavior Program. This review describes the spectrum of initial presentations of PCA in this program over a 25-year period (1995–2020). The presentations and examination of these patients focused on Benson's original description and the criteria derived from this clinic [9]. This study compares these patients with a review of the accumulated literature on PCA since its original description. It further retrospectively applies current Consensus Criteria [6], and reports data on date of death and survival from symptom onset.

METHODS

A review was performed for all patients diagnosed with PCA. This study was based on initial clinical presentation in the UCLA Neurobehavior Clinic. All patients initially presented because of a progressive, visually-related disturbance, often after having been cleared of a primary visual problem by an ophthalmologist or an optometrist. The patients were diagnosed with PCA by a behavioral neurologist on initial evaluation, which included

both a Neurobehavioral Status Examination (NBSE) and a review of brain imaging available as part of their initial clinical diagnostic evaluation at the first or second clinic visit. All had brain imaging showing predominant posterior cortical atrophy (computerized tomography [CT] or magnetic resonance imaging [MRI]) and/or posterior cortical dysfunction (single photon emission tomography [SPECT] or positron emission tomography [PET]), either bilateral or predominately unilateral, with relative sparing of other cortical areas. As a referral clinic drawing from a wide geographical area, most of these patients were initially seen for diagnostic consultation and subsequently returned to their primary neurologist or physician. Mortality data on these individuals was collected through chart review, and publicly available online obituaries and databases. This study was approved by a UCLA institutional review board.

A total of 65 patients met our program's criteria for PCA [9]. These included five core diagnostic features (all must be present) of (A) insidious onset and gradual progression; (B) presentation with visual complaints with intact primary visual functions (operationalized as visual acuity); (C) evidence of predominant complex visual disorder on examination; (D) proportionally less impaired deficits in memory and verbal fluency; and (E) relatively preserved insight. Supportive features included presenile onset, alexia, elements of Gerstmann's syndrome, ideomotor apraxia, normal physical examination, neuropsychology with predominant visuospatial/perceptual deficits, and brain imaging with posterior cortical abnormality and relatively spared frontal and mesiotemporal regions. In order to increase certainty of our diagnosis, given the limits on long-term follow-up in this mainly referral population, we included only patients who had confirmatory neuroimaging changes on presentation.

Procedures

In addition to a detailed history of visual and other complaints, the PCA patients underwent an extended NBSE of visual and other systems on initial presentation to clinic. Visual system testing began with an acuity check and visual fields by confrontation. Complex visual changes were evaluated with 2-D and 3-D constructions ("shape copy") and a clock drawing task. Testing for simultanagnosia (an element of Balint's syndrome, along with optic ataxia and oculomotor apraxia) involved visual search of a complex picture (e.g., identification of 10 key items from the Cookie Theft Picture from the Boston Diagnostic Aphasia Examination [10]). Testing for optic ataxia involved visually-guided reaching into the peripheral fields (with eyes focused on examiner's nose), and testing for oculomotor apraxia involved visually-guided eye movements into each visual quadrant. Visual object agnosia was tested with identification by use, function, or associated features of items missed on the naming task. Patients were asked to identify black and white facial images of famous politicians and entertainers, and to demonstrate putting on a garment with sleeve inside out. This was followed by gross naming of colors and perception of degraded or overlapping figures.

Additional testing on the intake NBSE screened for other potential deficits. Patients were asked to read six written words and write a sentence to dictation. Additional elements of Gerstmann's syndrome including testing for finger agnosia (identifying fingers) and right-

left confusion (identifying on self and on the examiner). Ideomotor praxis testing consisted of responses to verbal commands including transitive and intransitive actions of both upper limbs. Normal consisted of a perfect score on these screening tests, and any error was marked as abnormal.

The patients underwent non-visual neuropsychological measures based on the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) plus digit spans [11]. They included the Mini-Mental Status Examination (MMSE) as a general cognitive measure [12]. Attention was assessed with verbal digit span forward and verbal digit span backwards. Language tests included confrontational naming on the short 15-item version of the Boston Naming Test ("mini-BNT") with conceptual cuing as necessary to overcome any perceptual difficulty (reading and writing were tested with visual system testing). Memory assessment involved a 10-item word list (15-minute delayed recall), and a true-false memory recognition test.

Neuroimaging over the 25 years ranged from CT to fluorodeoxy-glucose (FDG) PET. A few patients in later years had amyloid-PET. One early patient had only a CT scan; otherwise, all others had an MRI scan often with functional imaging. In earlier years, functional imaging consisted primarily of a SPECT scan, with gradual transition over time to FDG-PET. Since these were primarily referral patients, most initial imaging was done at local or regional centers, in which case copies of the scans were obtained and reread at UCLA.

Data analysis

Descriptive statistics and frequencies were used to summarize the continuous and categorical variables, respectively, for the following: demographic and clinical characteristics, self-reported visual and non-visual symptoms on presentation, and non-visual neuropsychological measures. When not documented in all 65 patients, specific examination findings on the NBSE were reported and plotted as the percentage of abnormal results among the number of patients reported symptoms and performed examinations. Median overall survival (with 95% confidence interval) from symptom onset was estimated with the use of the Kaplan-Meier method. For the analysis of overall survival, data for patients were censored at the date the patient was last known to be alive. When death year was the only available date (i.e., missing month and day), July first of the death year was used.

RESULTS

Sample characteristics

The PCA patients had a mean age of symptom onset of 57.9 years (SD = 6.06) and mean duration of symptoms at presentation to our institution of 3.96 years (SD = 2.41) (see Table 1). More than half of them (52.3%) were women. Most were Caucasian; there were two Latinos (Mexican and Cuban), one African American, and one Asian (Chinese). The patients were mostly college educated with a wide range of professional/occupational backgrounds including two architects, two pilots, and one artist. On presentation, 25 (38%) had been seen by an ophthalmologist and at least 9 (14%) had undergone cataract surgery with persisting symptoms. Also on presentation, 37 (56.9%) were on an acetylcholinesterase inhibitor medication and 14 (21.5%) on memantine.

Presenting visual symptoms

The most common presenting visual complaints were progressive trouble finding or localizing items in their visual surroundings followed by difficulties navigating or orienting in their environment (see Table 2). Finding items requiring scanning was particularly difficult in a crowded visual field of view. Often items were quickly lost if put down, dropped, or even if just stationary in front of them. Patients would get lost in their homes or in familiar surroundings as well as in public places (e.g., theaters) or public bathrooms. Several described this environmental difficulty as problems gauging depth, distances, or orientation (e.g., orienting to a car or to an escalator). Many patients specifically complained of visuospatial impairments in driving (especially staying in lane, seeing traffic cones or signs, judging distances and lights), dressing (correctly donning clothes or shoes), eating (orienting to forks, avoiding spills or knocking things over), and “seeing” the television or computer screen. There were many spatiomotor complaints, which could not be distinguished from ideomotor apraxia (putting keys into keyholes, buckling seatbelts, using remote controls, inserting credit cards, hanging clothes, using tools/instruments/appliances). Some patients had unique visual complaints, grouped as disturbances related to reflections or transparencies (glass doors, reflection from puddles or head lights), visual motion (objects move or their motion is “broken”), color (distinguishing blue from black, red as orange, decreased depth of colors), visual or attentional hemi-field difficulty (two right, two left), face recognition, object recognition, visual hallucinations (two saw people, one “cars float above the road”), and visualization (dreams, projects). Finally, paradoxically, three patients retained the ability to play tennis or basketball, and another could see small objects in the distance while missing large items in front of her.

Presenting non-visual symptoms

Many complained of memory difficulties ($n = 32$; 49%), and although some of these complaints related to visually misplacing items or other visual difficulties, there was clear evidence of episodic verbal memory difficulty on testing in most patients (see Table 3). Likewise, many complained of reading difficulties ($n = 29$; 44.6%), and although some of these complaints related to visually localization, it was difficult to exclude primary lexical impairment. Nevertheless, the most common reading complaint was finding the next line on the page, reading cursive or handwriting, and letters “jumping around” or missing (see Table 3). Visuospatial deficits also played a role in their difficulties writing/drawing and calculating/measuring (especially checkbooks, deciphering coins/money, measuring, reading clocks/dials/watches). Eleven of the 65 patients endorsed initial word finding difficulty along with their visuospatial impairments. Finally, 25 (38.5%) PCA patients endorsed the presence of depression when specifically queried regarding the presence of symptoms of depression on review of systems.

Examination findings

The NBSE tasks with the highest percentage of documented testing abnormalities were constructional tasks of shape copy and clock drawing followed by Gerstmann’s syndrome tasks of calculations and writing (see Fig. 1). Other common posterior cortical (occipital or parietal) abnormalities included the remaining elements of Gerstmann’s syndrome and

Balint's syndrome, as well as difficulties with face recognition, reading, dressing, a visual hemi-field, and upper extremity spatiomotor actions. When considering at least two component signs, elements of Gerstmann's syndrome were present in 48 (73.9%) patients, absent in 10 (15.4%), and unclear or indeterminate in 7 (10.8%). When considering at least two component signs, elements of Balint's syndrome were present in 33 (50.8%) patients, absent in 18 (27.7%), and unclear or indeterminate in 14 (21.5%).

On presentation, the non-visual neuropsychological measures revealed further impairments (see Table 4). The mean MMSE score at time of diagnosis was 20.9 ± 5.69 indicating mild-moderate impairment. Attention was mostly within normal limits on forward or reverse digit spans, although nearly 20% had difficulty performing the reverse digit span. Memory was tested with an auditory verbal learning test with most showing impairment on both delayed recall and recognition testing [11]. Finally, many patients had low category (animals/minute) fluency and confrontational naming on the mini-BNT [11].

Biomarkers and neuroimaging

Seven patients had spinal fluid testing for AD biomarkers (amyloid-β, total tau, phospho-tau) with 6 showing results consistent with AD. Most patients (n =58) had an MRI of the brain, but only 13 (22%) of those MRIs had at least mild atrophy that was predominant in the posterior cortex, per clinical neuroradiological interpretations and reports. Of the 7 patients without MRIs, all had CT scans; 6 of these patients had an FDG-PET with focal parieto-occipital hypometabolism (the last patient with only a CT showed greater posterior atrophy on the images). In total, of 15 patients who had a SPECT scan, 12 (80%) showed focal parieto-occipital hypoperfusion consistent with PCA, and of 24 patients who had an FDG-PET scan, 21 (88%) showed focal parietooccipital hypometabolism consistent with PCA. Two patients had amyloid PET imaging, and both showed diffuse amyloid deposition.

The 2017 Consensus Criteria of PCA were retrospectively applied to these patients' presentations [6]. These Criteria specify "relatively spared antegrade memory function" and "relatively spared speech and nonvisual language functions" [6]. If interpreted as normal memory and language on presentation, no patient met Consensus Criteria. If interpreted as allowing for some degree of memory or language difficulty, albeit not as severe as the visuospatial difficulties, 59 (90.8%) met criteria for PCA. The patients who did not meet Consensus Criteria using this modification had significant memory problems equal to their visuospatial difficulties.

Follow-up and mortality

Analysis of 27 patients who received two or more follow-up visits (ranging 6 months to 10 years) in the clinic revealed that four had developed either CBS (2) or DLB (2). Among the total of 65 PCA patients, including those followed elsewhere, we were able to ascertain death in 37 (56.9%). For those 37, the Kaplan-Meier estimated overall survival at 7.5 years from symptom onset was 84.5% (95% CI: 75.2%, 95.1%); and the median overall survival was 10.3 years (95% CI: 9.6, 13.6 years) (see Fig. 2).

DISCUSSION

PCA is predominantly a visuospatial disorder affecting spatial localization and the performance of tasks dependent on visuospatial functioning. The patients' presentations also included memory difficulty in half the patients and the endorsement of depression in nearly 40%. Testing revealed elements of Gerstmann's syndrome and Balint's syndrome in the majority of patients. On retrospective application of the current Consensus Criteria, 59 (91%) could meet criteria if one allowed the presence of some memory and naming difficulty and depression. Finally, this study found a mean duration from symptom onset to death of 10.3 years, indicating that most patients with PCA have a slow neurodegenerative disorder like AD [13].

In these patients, the visual symptoms and complaints on presentation were primarily spatial rather than perceptual, and affected visuospatial activities such as driving, dressing, eating, reading in finding the next line of print, and even neglect and spatiomotor actions. In sum, PCA was primarily a dorsal (occipital-parietal) visual stream disorder [14–17]. With the possible exception of face recognition difficulty (which may also be affected by spatial disturbances), there were relatively few patients with evidence of ventral (occipital-temporal) visual stream involvement [15]. Other investigators have analyzed latent atrophy factors and report mixed clinical profiles that include ventral atrophy [18]. Similarly, "basic" visual impairments such as form detection and discrimination may occur in PCA [19], but these tasks may also involve spatial processing and do not necessary imply a "primary visual variant" [6]. In fact, the literature has been confusing because of a lack of uniformity in testing, the great overlap in clinical tests, and the lack of dissection of the affected underlying visual mechanisms.

In this study, beyond shape copy (complex figure) and clock drawing, the neurocognitive tasks that were most useful for diagnosing PCA were calculations and writing, both suggesting Gerstmann's syndrome. The majority of PCA patients had components of Gerstmann's syndrome and of Balint's syndrome, indicating involvement of the left inferior parietal and bilateral occipital-parietal regions, respectively [20]. Asymmetric right parietal PCA may also present as predominately visuospatial AD [21]. The acalculia in PCA has extended to measurement knowledge such as the meaning of "grams," "inches," or their relative magnitudes [22], and investigators have suggested that prominence of acalculia and Gerstmann's syndrome, with additional ideomotor apraxia, indicates a "PCA2" [23]. Primary visuospatial impairments may also confound the diagnosis of ideomotor apraxia because of the use of pooled imitation of meaningless as well as meaningful gestures [20].

Although earlier presentations might have shown intact memory, by about 4 years into their course, most of the patients with PCA in this study experience verbal memory difficulty, as well as some naming difficulty and frequent symptoms of depression. Memory problems in PCA can have a number of causes. First, there are reports of early verbal delayed recall deficits in PCA [24–26]. Although the patients in this study do not improve with recognition, some, but not all, investigations have shown improvement with cued recall, retrieval cues, or recognition scores, often in association with normal hippocampal volumes [24, 26–29]. In fact, PCA may spare, or selectively distort, hippocampal memory structures

without significant atrophy until late in the course [30–32]. A decrease in delayed recall in PCA could be due to their parietal cortical damage, possibly from executive effects on encoding and retrieval [26, 33]. Second, PCA patients have difficulty with visuospatial working memory from involvement of the precuneus in spatial attention or from disruption of the dorsal attention network [20, 24, 34]. Third, PCA patients have autobiographical memory impairments, such as spatially fragmented scene reconstruction, which appears correlated with gray matter intensity in left angular gyrus, right hippocampus, and right precuneus [35]. The right precuneus in particular contributes to memory via the retrieval of visual images with spatial and perceptual detail [36]. In addition to memory difficulty, some PCA patients have naming problems consistent with the known overlap of PCA with the logopenic progressive aphasia variant of AD [17, 37], and PCA patients are prone to depression consistent with other variants of AD [38].

About three quarters of patients with PCA have a posterior neocortical, relative hippocampal sparing form of AD [3, 4, 6], leaving another quarter with other causes, such as CBGD and DLB. Motor signs are common in PCA and may suggest either CBGD with early asymmetric motor abnormalities, particularly early apraxia [3, 39, 40]; whereas, DLB may be more symmetrical and suggested by the presence of visual hallucinations [38, 41]. Both PCA and DLB have lateral association occipital cortex hypometabolism, but it is more asymmetric with less overall primary visual hypometabolism in PCA [41–43]. Finally, a rapid clinical course may suggest prion disease or CJD; however, prion disease can be genetic and slow, lasting over a decade, such as from an insertional mutation of the prion protein gene [2, 44, 45].

Neuroimaging generally shows early parietal-occipital changes with sparing of primary visual cortex (“occipital tunnel sign”) [43]. In this study, which was limited to participants with neuroimaging support for PCA, the presenting MRIs had poor sensitivity for PCA (23%) compared to the SPECT (86%) or FDG-PET (91%) scans. Other studies indicate that neuroimaging in PCA, when compared to controls, shows greater gray matter atrophy in the occipital-temporal-parietal regions with dependent white matter alterations of the occipital cortex, corpus callosum, and related tracts and connections [46–48]. When the imaging connectome is analyzed, patients with PCA have diffuse functional connectome alterations with breakdown in posterior brain nodes and decreased connectivity in the visuospatial network [49, 50], but preserved to heightened connectivity in the salience and default mode networks [50–52]. As in a number of patients in this review, amyloid PET is diffusely positive in most PCA-AD patients and more extensive than the reduced metabolism in parietal-occipital regions on FDG-PET [53]. Yet, tau-PET studies of PCA indicate that the posterior brain regions are uniquely vulnerable to tau deposition, which corresponds to their visual dysfunction [54–57]. Although PCA is most commonly an early-onset variant of AD, it does not have a more rapid course as compared to AD in general [13]. On neuroimaging and subsequent pathology, the progression of PCA proceeds from posterior neocortical changes to involve temporal and frontal lobes [53, 58, 59], but in contrast to typical AD, hippocampal, entorhinal and frontal regions undergo a lower rate of change [60].

This study has limitations expected from a retrospective clinical review spanning 25 years. First, it is dependent on the availability and completeness of clinical data, which may be

missing or incomplete. We partially compensate for this limitation by reporting the percentage of positive tests for those that are clearly documented as actually tested or done. Second, many of the results represent clinical neurocognitive examination findings based on the NBSE. Despite the clinical nature of this testing, there was remarkable uniformity in this examination over the 25 years. Third, there was a lack of follow-up for most patients. Most had only an assessment for a subspecialty diagnosis and returned to their referring physicians over a scattered geographical area. Hence, this report focused on their initial presentation. Finally, the neuroimaging consisted of different types of scans from different centers. As a rule, we obtained copies of outside scans for rereading and review.

In conclusion, although we have much to learn, we have clarified a great deal about "Benson's disease" over the last 25 years. The diagnosis of PCA can focus on impairments in daily visuospatial activities such as findings things, navigating, dressing, eating, along with assessment of simple calculation and measurement knowledge. These symptoms reflect the salience of Gerstmann's syndrome and of Balint's syndrome in PCA. Revised diagnostic criteria may allow memory and language changes and the presence of depression, albeit not as prominent as the visuospatial deficits. The neurological examination evaluates for evidence of parkinsonism or movement disorders, and optimal neuroimaging includes FDG-PET because of its value in disclosing posterior cortical dysfunction. Tau-PET may prove particularly useful for differentiating PCA due to AD form PCA due to other causes. When the course deviates from a slow trajectory, prion disease is an important consideration. There is no effective medication for PCA, and acetylcholinesterase inhibitors have not proven clearly helpful [61]; however, treatments may profitably target attention and executive functions [62, 63]. Future prospective studies can develop specific testing that particularly analyzes the underlying visuospatial and calculation processes, investigate neurocognitive profiles that may be predictive of neuropathology, and focus treatment trials on the unique neuropathological aspects of PCA.

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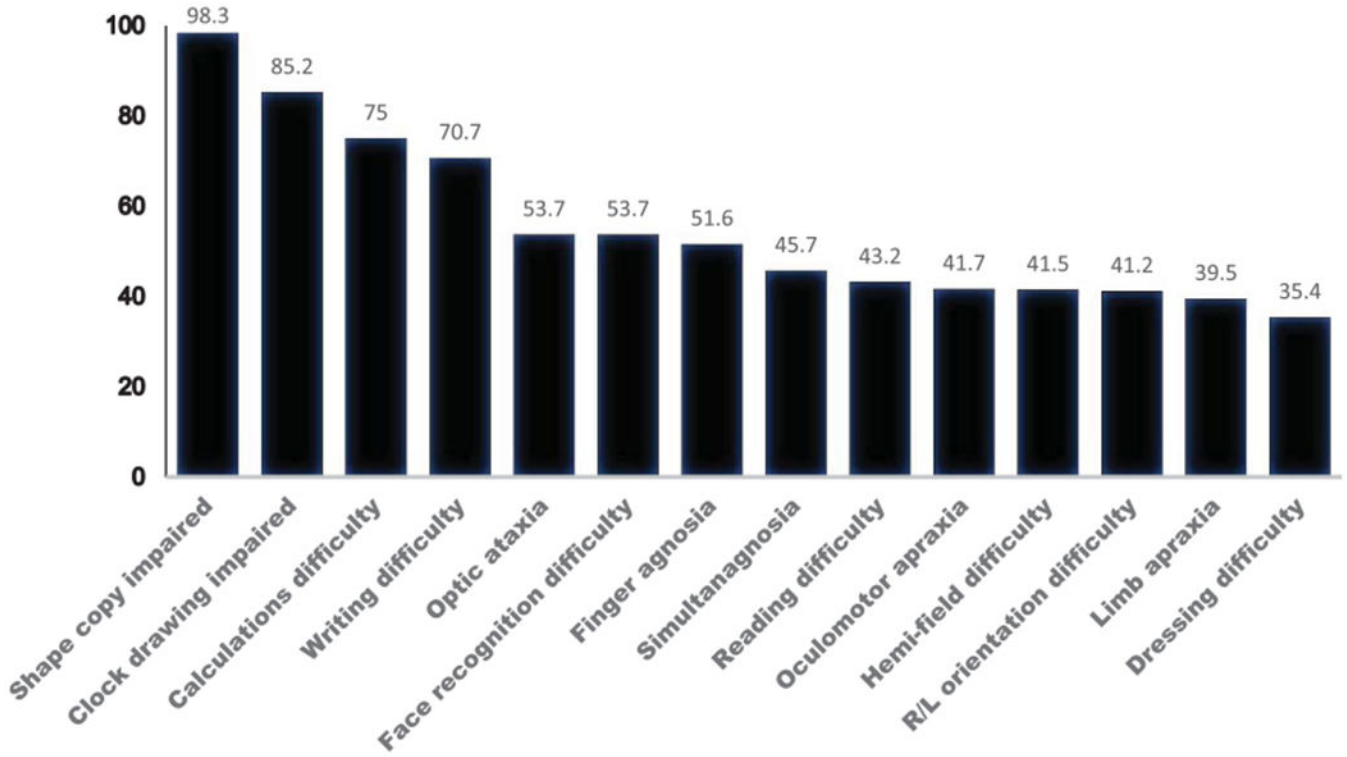

Fig. 1.

Percent of Abnormal Posterior Cortical (Occipital or Parietal) Examination Findings on Presentation. When not documented in all 65 patients, specific examination findings on the NBSE were reported and plotted as the percentage of abnormal results among the number of patients reported symptoms and performed examinations.

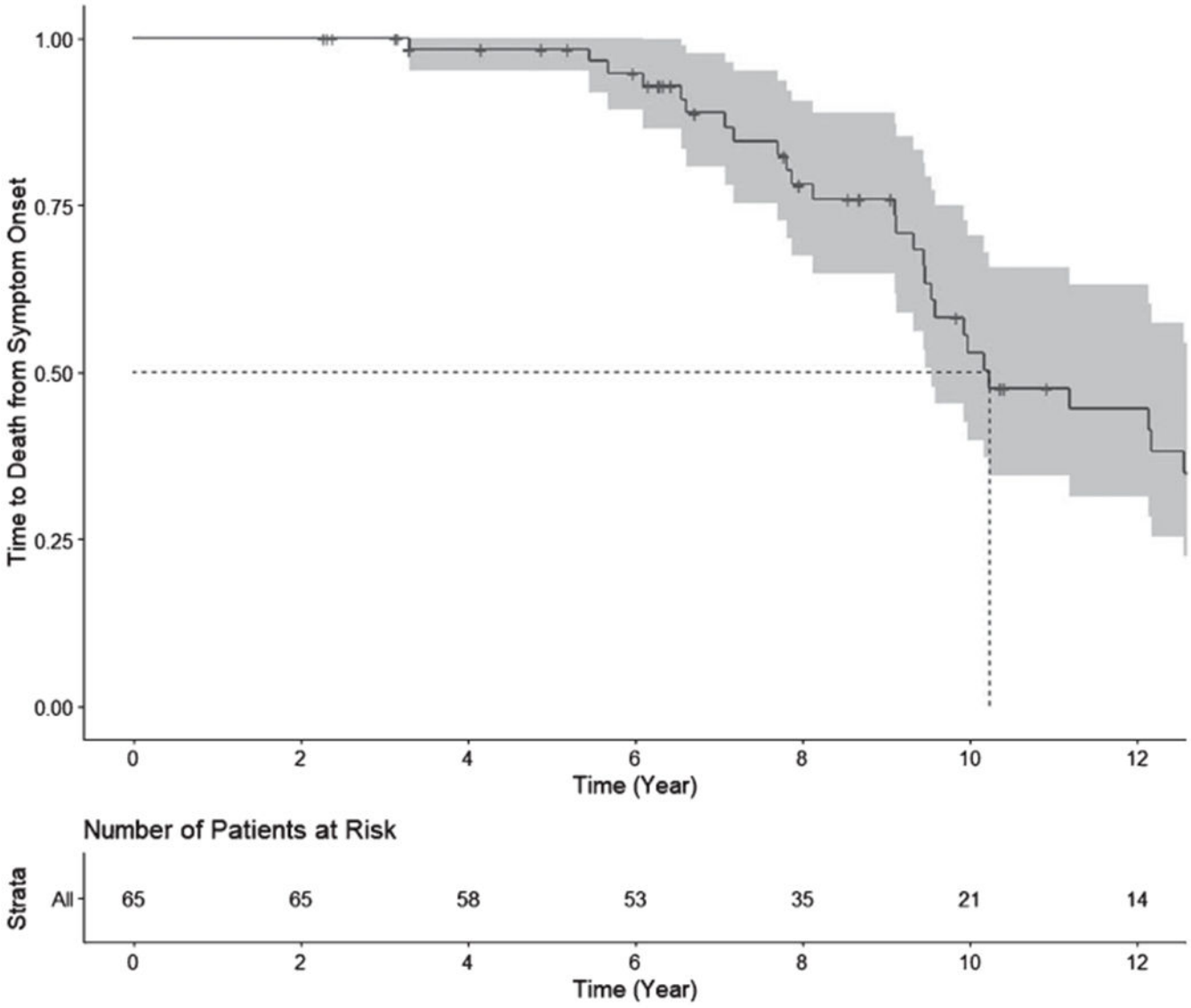

Fig. 2.

Kaplan-Meier Overall Survival Curve with 95% Confidence Interval – Time to Death from Symptom Onset.

Table 1:

Demographic and Clinical Characteristics of 65 Patient with Posterior Cortical Atrophy

	Mean ± SD or N (%)
Age at diagnosis (y)	61.9 ± 6.11
Age at symptoms onset (y)	57.9 ± 6.06
Duration of symptoms (y)	3.96 ± 2.41
Female	34 (52.3%)
Education (y) *	15.3 ± 2.77
Right Handedness	60 (92.3%)
Family history of dementia	18 (28%)

* 4 patients with missing responses.

Table 2:

Visual Symptom on Presentation Reported among 65 Patients with Posterior Cortical Atrophy

Presenting Visual Symptoms	N (%)
Finding/locating items in surroundings	45 (69.2%)
Navigating/orienting in environment	27 (41.5%)
Visuospatial difficulty driving	22 (33.8%)
Visuospatial difficulty dressing	22 (33.8%)
Spatiomotor difficulty with hands and/or apraxia*	15 (23.1%)
Visuospatial difficulty eating	13 (20.0%)
Visuospatial difficulty "seeing" screens/monitors	8 (12.3%)
Disturbed reflections/transparencies	7 (10.8%)
Disturbed effect of visual movement	7 (10.8%)
Color disturbances	6 (9.2%)
Hemi-field difficulty	4 (6.2%)
Face recognition difficulty	3 (4.6%)
Object recognition difficulty	3 (4.6%)
Visual hallucinations	3 (4.6%)
Visualization difficulty (dreams, projects)	2 (3.1%)

*Overlap in symptoms as described by patients and in distinguishing ideomotor apraxia from primary visuospatial difficulty.

Table 3:

Non-Visual Symptoms on Presentation Reported among 65 Patients with Posterior Cortical Atrophy

Presenting Non-Visual Symptoms	N (%)
Memory difficulty	32 (49.2%)
Reading *	29 (44.6%)
Writing/drawing *	13 (20.0%)
Calculating *	12 (18.5%)
Word finding difficulty	11 (16.9%)
Depression	25 (38.5%)

* Overlap of visuospatial dysfunction with disturbances of reading, calculations, and writing.

Table 4:

Non-Visual Neuropsychological Measures (Based on Consortium to Establish a Registry for Alzheimer's Disease [CERAD] plus Digit Spans) for patients with Posterior Cortical Atrophy

Measures	N	Mean ± SD	% below cutoff
Mini-Mental State Examination	58	20.9±5.69 *	77.6%
Forward digit span	59	5.68±1.15	13.6%
Reverse digit span	46	3.04±1.25	19.6%
Category (animals)/minute	47	11.6±5.30	27.7%
Mini-Boston Naming Test	49	11.4±3.68 *	49.0%
Verbal Learning: Delayed recall	40	2.59±2.78	62.5%
Recognition (Positives)	37	6.76±2.77 *	59.5%

* Mean less than cut-off scores for age; Welsh et al., 1994 [11].