

An Overlooked Presentation of Alzheimer’s Disease: Posterior Cortical Atrophy

P1-001

Maryam Mohamed Seddik
MPharm Student, University of Hertfordshire (GAF Campus)

PCA is an underrecognized visual-predominant Alzheimer’s syndrome, where early visuospatial deficits often precede memory impairment, contributing to delayed diagnosis.

INTRODUCTION	METHODS	RESULTS	CONCLUSIONS	REFERENCES																									
Posterior Cortical Atrophy (PCA) is a rare, progressive neurodegenerative syndrome primarily affecting the occipital and parietal lobes. Although usually underpinned by Alzheimer’s disease (AD) pathology, PCA presents with visuospatial and perceptual deficits rather than the early memory loss typically seen in AD; this atypical presentation often leads to delayed or incorrect diagnosis. This review summarizes recent literature on PCA’s clinical profile, imaging characteristics, and main symptoms.	A literature search was conducted using PubMed, NIH, and Google Scholar for studies from 2015–2025. Keywords included “posterior cortical atrophy,” “atypical Alzheimer’s,” and “visual variant AD.” Studies were included if they involved human participants diagnosed with PCA or visual variant AD and reported clinical and/or neuroimaging findings. Case reports involving fewer than three participants, non-English publications, and animal studies were excluded in order to focus on human diagnostic presentation and to improve interpretability of findings. Extracted data included author, publication year, sample size, mean or range of participant age, symptoms, neuroimaging findings and stage of diagnosis. Data was summarized using Microsoft Excel.	Table 1. Key clinical and neuroimaging characteristics of included PCA studies (2017–2024). <table><tr><th>Study Type</th><th>Sample Size (N)</th><th>Mean Age / Range</th><th>Imaging Findings</th><th>Stage of diagnosis</th></tr><tr><td>Intl cohort + IPD meta-analysis⁴</td><td>1092</td><td>59.4 yrs (onset)</td><td>Amyloid-PET 94%+, Tau-PET 97%+</td><td>Symptomatic (diagnosed at first presentation)</td></tr><tr><td>Retrospective registry³</td><td>38</td><td>67.8 yrs (presentation)</td><td>Posterior atrophy on MRI; FDG-PET occipitoparietal hypometabolism</td><td>Mild–moderate symptomatic</td></tr><tr><td>Longitudinal cohort²</td><td>117</td><td>—</td><td>Early occipital/parietal atrophy; longitudinal MRI</td><td>Symptomatic (mild-to-moderate dementia stage)</td></tr><tr><td>Prospective longitudinal¹</td><td>12</td><td>56 yrs median (48–63)</td><td>FDG ↓ parieto-occipital; PIB diffuse cortical amyloid</td><td>Symptomatic at diagnosis</td></tr></table> Four studies (n = 1,259) met inclusion criteria. Across studies, PCA showed early-onset visuospatial impairment with consistent occipital-parietal atrophy and FDG-PET hypometabolism, while memory and language remained relatively preserved in early stages.	Study Type	Sample Size (N)	Mean Age / Range	Imaging Findings	Stage of diagnosis	Intl cohort + IPD meta-analysis ⁴	1092	59.4 yrs (onset)	Amyloid-PET 94%+, Tau-PET 97%+	Symptomatic (diagnosed at first presentation)	Retrospective registry ³	38	67.8 yrs (presentation)	Posterior atrophy on MRI; FDG-PET occipitoparietal hypometabolism	Mild–moderate symptomatic	Longitudinal cohort ²	117	—	Early occipital/parietal atrophy; longitudinal MRI	Symptomatic (mild-to-moderate dementia stage)	Prospective longitudinal ¹	12	56 yrs median (48–63)	FDG ↓ parieto-occipital; PIB diffuse cortical amyloid	Symptomatic at diagnosis	A rare and underdiagnosed form of Alzheimer's disease, PCA mostly manifests visually and frequently causes a delayed diagnosis. Improved awareness and use of neuroimaging, alongside multidisciplinary collaboration between neurologists, ophthalmologists, and general practitioners, can support earlier detection. Timely diagnosis enables better symptom management, rehabilitation, and individualized care while patients still retain insight, and may also reduce caregiver burden and healthcare costs. Further research is needed to clarify disease mechanisms and develop targeted, stage-specific treatments.	1. Panegyres PK, Goh J, McCarthy M, Campbell AI. The Nature and Natural History of Posterior Cortical Atrophy Syndrome. <i>Alzheimer Disease & Associated Disorders</i>. 2017 Oct;31(4):295–306. 2. Firth NC, Primativo S, Marinescu RV, Shakespeare TJ, Suarez-Gonzalez A, Lehmann M, et al. Longitudinal neuroanatomical and cognitive progression of posterior cortical atrophy. <i>Brain</i>. 2019 Jun 20;142(7):2082–95. 3. Chapleau M, Renaud La Joie, Yong K, Agosta F, Isabel Elaine Allen, Apostolova L, et al. Demographic, clinical, biomarker, and neuropathological correlates of posterior cortical atrophy: an international cohort study and individual participant data meta-analysis. <i>The Lancet Neurology</i>. 2024 Feb 1;23(2):168–77. 4. Olds JJ, Hills WL, Warner J, Falardeau J, Alasantro LH, Moster ML, et al. Posterior Cortical Atrophy: Characteristics From a Clinical Data Registry. <i>Frontiers in Neurology</i>. 2020 Jun 3;11.
Study Type	Sample Size (N)	Mean Age / Range	Imaging Findings	Stage of diagnosis																									
Intl cohort + IPD meta-analysis ⁴	1092	59.4 yrs (onset)	Amyloid-PET 94%+, Tau-PET 97%+	Symptomatic (diagnosed at first presentation)																									
Retrospective registry ³	38	67.8 yrs (presentation)	Posterior atrophy on MRI; FDG-PET occipitoparietal hypometabolism	Mild–moderate symptomatic																									
Longitudinal cohort ²	117	—	Early occipital/parietal atrophy; longitudinal MRI	Symptomatic (mild-to-moderate dementia stage)																									
Prospective longitudinal ¹	12	56 yrs median (48–63)	FDG ↓ parieto-occipital; PIB diffuse cortical amyloid	Symptomatic at diagnosis																									
				CONTACT  Maryam Seddik, MPharm Student at UH-GAF maryam.seddik@gaf.ac www.linkedin.com/in/maryam-seddik-649552375 																									