

# New findings, imaging may aid diagnosis of concomitant AD in patients with Parkinson's disease dementia

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Dementia is a frequent complication of Parkinson's disease (PD), but it is clinically impossible to distinguish PD dementia (PDD), which develops from the progression of the Lewy body pathology that underlies PD, from PD with coexistent Alzheimer's disease (PDAD). Both have similar characteristics. A team of scientists has found that PDAD patients have much denser accumulations of amyloid plaques in the striatal area of the brain than PDD patients. The results suggest that recently developed imaging techniques may be able to identify striatal amyloid plaques in the living brain and could be useful for distinguishing PDD from PDAD. Their results are published in the April issue of the *Journal of Parkinson's Disease*.

"We sought to determine if the presence, density, or type of striatal plaques were predictive of the presence of a clinicopathological diagnosis of Alzheimer's disease in subjects with PD and dementia," say lead investigators Thomas G. Beach, MD, PhD, of Banner Sun Health Research Institute, and Charles H. Adler, MD, PhD, of the Mayo Clinic. Dr. Brittany Dugger, first author, notes the ability to determine the cause of dementia in [patients](#) with PD is a crucial objective if effective treatments are to be developed.

Researchers performed autopsies on the brains of [elderly subjects](#) who had volunteered to be part of the Arizona Parkinson's Disease Consortium and Banner Sun Health Research Institute [Brain](#) and Body

Donation Program, a longitudinal clinicopathological study of normal aging, dementia, and parkinsonism. They evaluated the brains of patients with a diagnosis of PD without [dementia](#) (PDND), PDD without pathological AD, and PDAD. For comparative purposes, subjects from a previously published study of patients with AD without PD (AD), as well as non-demented normal [control subjects](#) without parkinsonism (NC), were also included. Amyloid plaque densities were graded at several sites in the brain and scored by the researchers as none, sparse, moderate, or frequent. Scores were derived by considering all types of plaques together – cored, neuritic, and diffuse – as well as separately for cored and neuritic plaques, without diffuse plaques.

Investigators found that the AD and PDAD cases had significantly higher cerebral cortex total and neuritic [plaque](#) density scores when compared to PDD, PDND, and NC. In patients with PD, the presence of any type of striatal plaques predicted the clinicopathological diagnosis of AD with 80% sensitivity and 80% specificity. In comparison, the presence of cerebral cortex plaques was 100% sensitive but only 48% specific when PDND, PDAD, and PDD were included; and only 55% specific when only the PDAD and PDD groups were included.

"The results suggest that, with the use of amyloid imaging, the presence of striatal plaques could help clinically distinguish PDD from PDAD," notes Dr. Beach. "Large antemortem-postmortem correlative studies are needed to determine whether a positive striatal amyloid imaging signal would be a sensitive and specific marker of concurrent PD and AD and their clinical severities."

**More information:** "Presence of Striatal Amyloid Plaques in Parkinson's Disease Dementia Predicts Concomitant Alzheimer's Disease: Usefulness for Amyloid Imaging," by B.N. Dugger, G.E. Serrano, L.I. Sue, et al. *Journal of Parkinson's Disease*, 2(2012) 57-65. [DOI: 10.3233/JPD-2012-11073](https://doi.org/10.3233/JPD-2012-11073)

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