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Brief Report

Memory Consolidation and ARIA in Individuals Receiving Anti-amyloid Monoclonal Antibodies

Marc W. Haut [a,b,c,*], Camila Vieira Ligo Teixeira [a], Patrick D. Worhunsky [a], Rashi I. Mehta [a,d], Joseph E. Malone [a,c], Melanie Ward [a,c], Cierra M. Keith [a,b], Holly E. Phelps [a,b], Stephanie Pockl [a,e], Nafiisah Rajabalee [a,e], Khalid Sharif [a,b], Gary Marano [a,d], Pierre-Francois D'Haese [a,d], Ali R. Rezai [a,f]

[a] Rockefeller Neuroscience Institute West Virginia University, Morgantown, West Virginia, USA
[b] Department of Behavioral Medicine and Psychiatry, Morgantown, USA
[c] Department of Neurology, Morgantown, USA
[d] Department of Neuroradiology, Morgantown, USA
[e] Department of Medicine, Morgantown, USA
[f] Department of Neurosurgery, Morgantown, USA

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ABSTRACT

Amyloid related imaging abnormalities (ARIA) are the most significant risk associated with the use of anti-amyloid monoclonal antibodies (MAB) for Alzheimer's disease (AD). Currently, the presence of the APOE ε4 allele is the best predictor for the development of ARIA. However, the degree of baseline memory impairment has not been fully explored as a risk factor for ARIA. Here, we examined MAB outcomes in a memory clinic population and compared patients with AD who developed ARIA to a case-matched group who did not develop ARIA. Participants who developed ARIA had greater numbers of recall intrusions and false positives, both markers for memory consolidation, at baseline than those who did not develop ARIA. We also observed greater baseline hippocampal and supplementary motor cortical atrophy with ARIA. These differences remained when controlling for the APOE ε4 allele and the presence of pretreatment microhemorrhages. Further investigation of memory impairment and associated brain atrophy is warranted to understand ARIA risk and MAB outcomes in AD.

1. Introduction

The Food and Drug Administration (FDA) approval of aducanumab, an anti-amyloid monoclonal antibody, in June of 2021 opened a new era in the treatment of Alzheimer disease (AD). Aducanumab was approved using an accelerated approval based on biological evidence of amyloid-β plaque load reduction in the brain, despite a negative recommendation by an expert advisory panel and conflicting phase III clinical outcome data [1]. The roll out of aducanumab was met with controversy and limited adoption by major medical centers in the face of non-coverage by the Center for Medicare and Medicaid services. However, the subsequent clinical trial data and then FDA approvals of lecanemab [2] and donanemab [3] have both been met with wide adoption. Given the demand for these immunotherapies, understanding their risk-benefit profiles is mandatory.

The most significant risk associated with monoclonal antibodies is amyloid related imaging abnormalities (ARIA). These can take the form of ARIA-H, microhemorrhages and/or superficial siderosis or ARIA-E, edema and/or sulcal effusions [4]. The strongest risk factor for the development of ARIA identified to date is the presence of the APOE ε4 allele [5]. The presence of cerebral microhemorrhages predating treatment and extent of comorbid vascular disease have also been identified as additional risk factors for the development of ARIA [5]. Further stratification of risk associated with ARIA is imperative given the growing use of these agents [6].

Appropriate use recommendations for initiating treatment with MAB [7–9] require that patients be mildly affected, with mild cognitive impairment (MCI) or mild dementia, and with a positive amyloid PET or CSF analysis for amyloid-β (Abeta) and phosphorylated tau (p-tau). Within this mild range of disease progression, there are variations in the

* Corresponding author at: Rockefeller Neuroscience Institute, West Virginia University 33 Medical Center Drive, Morgantown, WV 26505.
E-mail address: mhaut@hsc.wvu.edu (M.W. Haut).

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degree of cognitive impairment. Memory consolidation dysfunction is the prototypical symptom associated with early AD [10]. However, we are not aware of any studies to date that have specifically examined the degree of memory consolidation impairment in individuals receiving MAB as a potential risk indicator for developing ARIA.

We examined the baseline (i.e., pre-MAB treatment) memory performance of individuals who received monoclonal antibodies over the last four years at our multidisciplinary clinic. We focused on measures of memory consolidation: percentage of information retained over time, recognition discriminability, recall intrusions, and false positive errors on recognition. We compared patients who developed ARIA to an age and sex case-matched control group (2:1 ratio). We hypothesized that patients who developed ARIA would demonstrate worse memory consolidation at baseline. We planned to examine differences in brain volume in brain regions associated with the specific measure(s) of memory impairment. As our sample size is limited, we planned a hypothesis-driven approach focusing on brain regions associated with aspects of memory consolidation rather than a whole brain search. Specifically, our previous work [11] identified that the best predictor for percentage of information retained was the entorhinal cortex, while recognition discriminability was associated with an interaction between the entorhinal and the superior frontal cortex. The number of intrusion errors during free recall was best predicted by an interaction between the hippocampus and the supplementary motor cortex, and the best predictor of false positive errors was the interaction between the entorhinal cortex and middle frontal gyrus. Dependent upon any observed differences in memory performance, we planned to examine those previously identified brain regions.

2. Methods

2.1. Participants

We included patients seen in our center and treated with MAB between August of 2021 through September of 2025. All participants received a diagnosis of either MCI or mild dementia due to Alzheimer's disease using established criteria [12],[13],. Final diagnosis and treatment planning were made by consensus of attending neurologists, neuropsychologists, geriatric psychiatrists, neuroradiologists, and geriatricians. All patients were determined to be amyloid positive by CSF analysis using Mayo clinic send out or ^{18F}Florobetaben PET. Cutoffs for treatment included CSF ptau/Abeta ratio over 0.028 or an Amyloid PET whole brain SUVr of at least 1.20 for those over age 65 or at least 1.18 for those aged 65 or younger (i.e., centiloid levels of 38– 40) [14],[15],. Participants provided written informed consent for the use of their clinical data for research purposes.

2.2. Treatment

Treatment followed the appropriate use recommendations for each of the three monoclonal antibodies utilized in our center over the last 4 years: aducanumab [7], lecanemab [8] and donanemab [9]. Occurrences of ARIA were diagnosed by a board certified neuroradiologist. Patients receiving MAB without evidence of ARIA were case matched on age and sex with a 2:1 ratio (control to ARIA) if they had completed at least 6 months of treatment.

2.3. Memory performance

Patients included in the analyses completed a baseline evaluation that included the California Verbal Learning Test – II short form [16]. We used the following measures previously studied as indicators of memory consolidation: percentage retained after a delay, recognition discriminability, number of free recall intrusion errors, and number of false positive errors on recognition [16],[17],. Additional baseline characteristics included the Mini Mental State Examination [18], the Functional Activities Questionnaire [19], the Neuropsychiatric Inventory – Questionnaire [20], and the Wide Range Achievement Test [21].

2.4. Imaging acquisition and processing

All patients completed MRI sequences on either a Siemens or GE 3T system that included a 3D high resolution T1, T2, and T2 FLAIR with $1 \times 1 \times 1$ mm resolution as well as a thin slice (2 mm slice thickness) blood product sensitive GRE imaging sequence. We utilized the T1 and T2 FLAIR with details previously described [11],[17],. We used spatially localized atlas network tiles [22] to measure brain volumes previously associated with each measure of memory consolidation. Volumetric measurements were converted to percentage of total intracranial volume. For interaction terms, the two brain volumes were multiplied with each other. White matter hyperintensity volume (WMHv) was calculated with the Lesion Segmentation Toolbox using the lesion prediction algorithm to calculate total volume in ml [23].

2.5. Statistical analysis

We utilized Chi-square to test for differences between groups for categorical variables (sex, APOE status), and ANOVA for continuous variables. The Benjamini-Hochberg procedure was used to correct for false discovery rate (FDR).

3. Results

3.1. Prevalence of ARIA and Group Characteristics

Of 246 patients receiving MAB, 39 (15.9 %) individuals developed ARIA. Of those 39, there were six cases (15.4 %) of ARIA-H, 21 cases (53.8 %) of ARIA-E, and 12 cases (30.8 %) who developed ARIA-E and ARIA-H together. Five patients (12.8 %) developed severe radiographic ARIA, two of whom were treated with a brief course of steroids. No participant had severe clinical symptoms of ARIA, which is in alignment with the low occurrence of such events as reported by initial clinical trials [7,8]. Of 207 individuals receiving MAB who had not developed ARIA, we case matched 78 individuals based on age and sex as a comparison group. In the non-ARIA group, we only included those on treatment for at least six months. Table 1 demonstrates the groups were highly similar except, as expected, a higher rate of the presence of the APOE ε4 allele in the ARIA group. In addition, the group that developed ARIA were more likely to have baseline microhemorrhages prior to the initiation of treatment. With respect to treatment exposure, most patients in both groups received lecanemab only, while smaller subsets received aducanumab or donanemab, including treatment transitions between drugs and modified dosing for donanemab [24], as detailed in Table 2.

3.2. Memory performance

Baseline memory consolidation performance comparison between the groups revealed a significantly greater number of intrusion errors on free recall and a greater number of false positive errors on recognition in the ARIA group (Table 1). While percentage of information retained over time and recognition discriminability were different, that difference did not survive FDR statistical correction.

3.3. Brain morphometry

Based on our prior work [11], we then investigated group differences in the volume of the hippocampus, supplementary motor cortex and the interaction between these two regions (i.e., regions associated with intrusions), and the entorhinal cortex, middle frontal gyrus and the interaction between the two regions (i.e., regions associated with false

Table 1
Comparison of demographic, cognitive, and brain measures between groups.

Variable	No ARIA N = 78	ARIA N = 39	F/Chi-Square	P value
Age	65.8(8.2)	65.4(8.3)	<1	0.819
Sex (M/F)	30/48	15/24	-	-
Education	14.8(2.6)	15.1(2.5)	<1	0.577
WRATSS	100.5(13.9)	101.7(10.3)	<1	0.635
APOE4 (N/Y)	38/40	9/30	8.7	0.003*
Microhemorrhages (Y/N)	6/72	11/28	8.8	0.003*
A PET WB SUVr	1.48 (0.25)	1.57 (0.26)	2.5	0.118
Ptau/Abeta42	.043(0.04)	.055(0.05)	< 1	0.408
MMSE	26.7(2.8)	26.6(2.6)	<1	0.904
FAQ	5.5(5.5)	5.6(5.4)	<1	0.883
NPI-Total	3.1(2.2)	3.0(2.0)	<1	0.847
CVLT %R	63.3(34.9)	47.9(41.4)	4.5/	0.036
CVLT Discrimination	2.4(0.9)	2.0(1.0)	4.7/	0.032
CVLT False Positives	2.1(2.3)	3.9(3.4)	11.5/	<0.001*
CVLT Intrusions	2.3(2.2)	4.5(4.4)	14.0/	<0.001*
WMHv	2.6(2.9)	2.4(2.5)	<1	0.786
Hippocampal vol	0.0027 (0.0003)	0.0025 (0.0003)	8.6	0.004*
SMC vol	0.0038 (0.0004)	0.0036 (0.0006)	4.7	0.032
Hippocampal x SMC	1.01×10^{-5}	9.18×10^{-6}	9.5	0.002*
Entorhinal cortex vol	0.0014 (0.0002)	0.0013 (0.0002)	1.7	0.191
MFG vol	0.0143 (0.001)	0.0138 (0.0002)	2.2	0.144
Entorhinal x MFG	1.19×10^{-5}	1.18×10^{-5}	2.7	0.106

Age and education in years, WRATSS = Wide Range Achievement Test – 4 Reading subtest Standard Score, MMSE = Mini Mental State Examination, FAQ = Functional Activities Questionnaire, NPI-Q = Neuropsychiatric Inventory Questionnaire, WMHv = white matter hyperintensity volume, CVLT % R = California Verbal Learning Test percent retained—percent of words recalled from trial 4 at the long delay, CVLT Discrimination = California Verbal Learning Test index of discrimination between hits and false positives on recognition, CVLT False Positives = California Verbal Learning Test number of false positives on recognition, CVLT Intrusions = California Verbal Learning Test number of intrusions on recall, SMC = supplementary motor cortex, MFG = middle frontal gyrus.
* = $p < .05$ FDR – critical value = 0.012.

Table 2
Monoclonal antibody treatment pathways in patients with and without ARIA.

Treatment Pathway	ARIA (n = 39)	No ARIA (n = 78)
Aducanumab only	4	2
Aducanumab → Lecanemab	total: - 2 ARIA on aducanumab - 2 ARIA after switching to lecanemab	12
Aducanumab → Donanemab	0	3 (2 with modified dosing)
Lecanemab only	28	54
Donanemab only	3 (2 with modified dosing)	3 (1 with modified dosing)
Lecanemab → Donanemab	0	2 (both modified dosing)

positives). Results are presented in Table 1. The volume of the hippocampus was significantly smaller in the ARIA group. The supplementary motor cortex was smaller in the ARIA group, but this difference was not statistically significant after FDR correction. The interaction of the hippocampal and supplementary motor cortex volumes was significant, with the ARIA group showing significantly lower volume (see Table 1 and Fig. 1). The entorhinal cortex, middle frontal gyrus and the interaction between these regions were not statistically different between groups.

3.4. Post-hoc Analysis

Given the group differences in APOE4 status and the potential influence of baseline cerebral microhemorrhage burden on cognitive performance and brain structural measures, we repeated the analyses for the statistically significant between-group differences in memory and brain volume shown in Table 1, covarying for APOE status and baseline presence of microhemorrhages. The effects on false positives, intrusions, and the interaction between hippocampal volume and supplementary motor cortex volume remained significant ($F = 8.2$, $p = .005$; $F = 6.2$, $p = .015$; and $F = 5.1$, $p = .026$, respectively), with an FDR-corrected critical value of 0.037.

4. Discussion

The aim of this study was to determine if memory consolidation at clinical presentation was different in patients who later developed ARIA while undergoing anti-amyloid monoclonal antibody therapy. We observed a significantly greater number of free recall intrusions in the group that went on to develop ARIA. The specific intrusion error type in memory recall has long been associated with AD [25]. Others have reported a difference in intrusion errors in amyloid positive and negative individuals [26],[27],. In addition to more intrusion errors, we observed a higher number of false positive errors in the ARIA group. We have previously reported that there are a greater number of false positive errors in amyloid positive AD relative to controls, but also a higher number than in behavioral variant frontotemporal dementia [28]. To our knowledge, this is the first report of baseline differences in memory performance in patients who develop ARIA while on anti-amyloid MABs. Group differences in intrusion and false positive errors occurred despite similar performances on other baseline variables including the MMSE and FAQ. Differences in intrusions and false positive errors both survived after controlling for the large effects of APOE ε4 carrier status and baseline microhemorrhages. We should point out that baseline WMHv and amyloid burden were not different between the groups. While APOE ε4 and baseline microhemorrhages remain very important factors in understanding the risks of ARIA, we report that degree of consolidation-based memory impairment at baseline may represent an additional risk factor.

In addition to greater impairment in memory performance, we observed a difference in baseline atrophy in the brain regions previously associated with intrusion errors [11]. Specifically, we observed smaller volume in the hippocampus and the interaction between the hippocampus and the supplementary motor cortex in the ARIA group. While this difference is consistent with our prior finding of this interaction and its relationship to intrusions, reasons why greater atrophy occurs alongside more intrusions in cases of ARIA remain to be determined. There are prior reports of an association between hippocampal atrophy and ARIA [29], but not in combination with atrophy of the frontal cortex. This finding needs to be replicated and interpreted with caution, as we did not observe greater atrophy in the entorhinal cortex, middle frontal gyrus, or the interaction between them alongside the greater number of recognition false positives in the ARIA group as predicted.

The mechanism of action for differences in baseline memory consolidation errors is not clear. We do not believe that baseline memory consolidation is specifically linked to the development of ARIA. Rather, memory consolidation errors are a likely surrogate to other processes associated with the development of ARIA. One possible mechanism is the relationship between memory consolidation errors and the hippocampus. It may be that individuals with greater baseline memory impairment and hippocampal atrophy share vascular risk factors that also predispose to ARIA. The hippocampus is particularly susceptible to vascular-mediated injury, through varied mechanisms, and vascular disease is also a known risk factor for ARIA. Patients with AD and co-occurring cerebrovascular disease often demonstrate greater degrees of hippocampal atrophy. As shown in a detailed structural equation

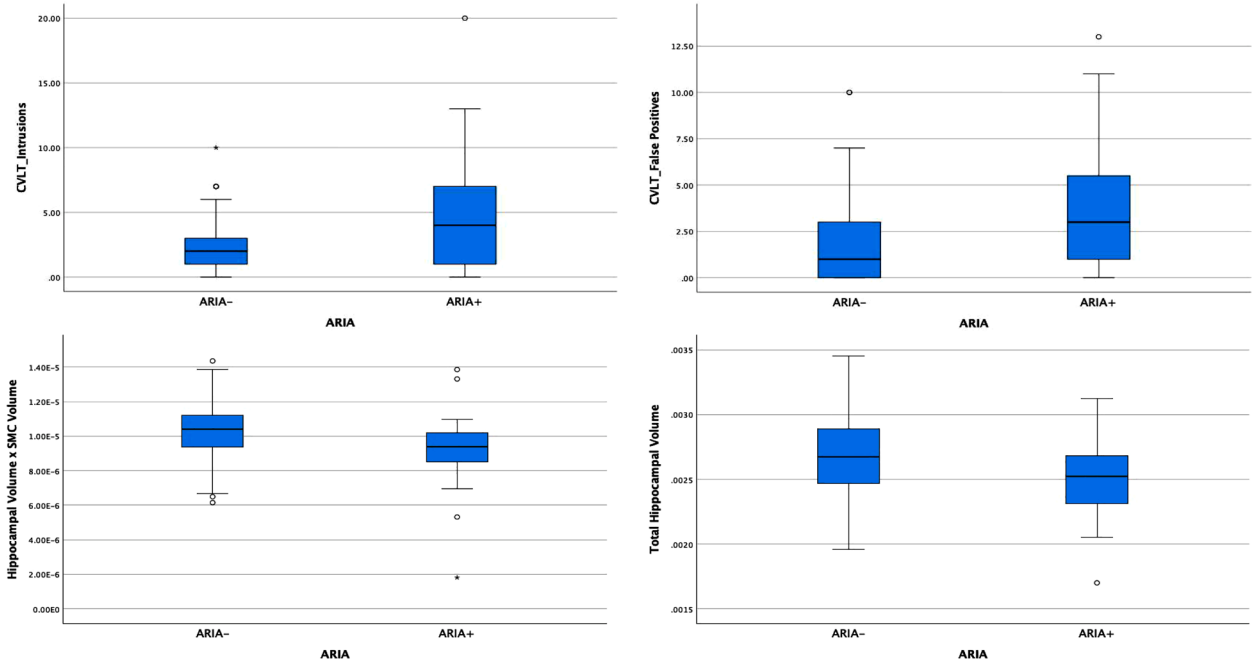

Fig. 1. Differences in memory consolidation and brain volume measures between ARIA and no ARIA groups. CVLT = California Verbal Learning Test; CVLT_False Positives = number of false positives on recognition; CVLT_Intrusions = number of intrusions on recall; SMC = supplementary motor cortex volume; Hippocampal volume = total hippocampal volume/total intracranial volume; Hippocampal volume x SMC volume = (hippocampus volume x supplementary motor cortex volume)/total intracranial volume.

modeling investigation with multiple mediation analyses by Tang et al. [30], vascular factors influence cognitive performance through hippocampal atrophy. In this study, hippocampal atrophy was found to serve as the strongest mediator of vascular effects on cognition, followed by cerebral atrophy and APOE genotype. Thus, hippocampal atrophy may be a primary mediating mechanism for indirect effects of cerebrovascular factors on cognition in AD and as a risk for ARIA.

While this is a unique and potentially important finding, there are limitations. Our sample has limited racial diversity, but a richness in terms of being an understudied rural Appalachian population. In addition, our sample was well-educated overall, and extending these findings to lower education samples will be important. Replication with larger and more diverse samples will be crucial. Given our sample size to date, we did not examine the possible role of different types and severity of ARIA on differences in memory consolidation. A larger sample will also allow for examination of other patterns of atrophy in different brain regions associated with AD. There has previously been a small association between ARIA and hippocampal volume [28]. Though we did not observe differences in the entorhinal volume, middle frontal gyrus, or the interaction between them, it is possible that we were insufficiently powered to observe these effects. Although all matched participants had completed a minimum of 6 months of treatment, after which the rates of ARIA drop, it is possible some in that group will go on to develop ARIA. In addition, baseline microhemorrhages clouds the picture on emerging ARIA after treatment being exclusively related to the antibodies themselves as some level of ARIA may have developed independent of treatment. As this is clinical data without a control group, we cannot address this question with the current design.

In conclusion, in this real-world sample of patients receiving monoclonal antibodies, we observed a baseline difference in two measures of memory consolidation when comparing groups based on ARIA status. Patients who developed ARIA presented with more free recall intrusions and false positives on recognition. Increased baseline atrophy of the hippocampus, supplementary motor cortex, and the interaction between the two brain regions was also observed in those who developed ARIA. The interaction remained significant after controlling for APOE4 carrier status and pretreatment microhemorrhages, further suggesting the importance of additional investigation.

Data statement

Data will be made available upon reasonable request from qualified investigators

Declaration of the use of generative AI and AI-assisted technologies in scientific writing and in figures, images and artwork

Generative AI was not used in the design, analysis or writing of this work

Ethical statement

All participants signed an informed consent form as approved by the IRB at West Virginia University

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CRediT authorship contribution statement

Marc W. Haut: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Camila Vieira Ligo Teixeira:** Writing – review & editing, Writing – original draft, Investigation, Formal analysis. **Patrick D. Worhunsky:** Writing – review & editing, Methodology. **Rashi I. Mehta:** Writing – review & editing, Investigation. **Joseph E. Malone:** Investigation. **Melanie Ward:** Investigation. **Cierra M. Keith:** Writing – review & editing, Investigation. **Holly E. Phelps:** Writing – review & editing, Investigation. **Stephanie Pockl:** Investigation. **Nafiisah Rajabalee:** Writing – review & editing, Investigation. **Khalid Sharif:** Investigation.

Gary Marano: Investigation. **Pierre-Francois D'Haese:** Writing – review & editing, Software, Data curation. **Ali R. Rezai:** Writing – review & editing, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

[1] Budd Haeberlein S, Aisen PS, Barkhof F, et al. Two randomized phase 3 studies of aducanumab in early Alzheimer's disease. J Prev Alzheimers Dis 2022;9(2):197–210.
[2] Van Dyck CH, Swanson CJ, Aisen, et al. Lecanemab in early Alzheimer's disease. N Engl J Med 2023;388(1):9–21.
[3] Sims JR, Zimmer JA, Evans CD, et al. Donanemab in early symptomatic Alzheimer disease: the TRAILBLAZER-ALZ 2 randomized clinical trial. J Am Med Assoc 2023;330(6):512–27.
[4] Sperling RA, et al. Amyloid-related imaging abnormalities in amyloid-modifying therapeutic trials: recommendations from the Alzheimer's Association Research Roundtable Workgroup. Alzheimers Dement 2011;7:367–85.
[5] Hampel H, Elhage A, Cho M, Apostolova LG, Nicoll JA, Atri A. Amyloid-related imaging abnormalities (ARIA): radiological, biological and clinical characteristics. Brain 2023;146(11):4414–24.
[6] Greenberg SM, Bax F, van Veluw SJ. Amyloid-related imaging abnormalities: manifestations, metrics and mechanisms. Nat Rev Neurol 2025;21(4):193–203.
[7] Cummings J, Rabinovici GD, Atri A, et al. Aducanumab: appropriate use recommendations update. J Prev Alzheimers Dis 2022;9(2):221–30.
[8] Cummings J, Apostolova L, Rabinovici GD, et al. Lecanemab: appropriate use recommendations. J Prev Alzheimers Dis 2023;10(3):362–77.
[9] Rabinovici GD, Selkoe DJ, Schindler SE, et al. Donanemab: appropriate use recommendations. J Prev Alzheimers Dis 2025;12(5):100150.
[10] Small BJ, Fratiglioni L, Viitanen M, Winblad B, Bäckman L. The course of cognitive impairment in preclinical Alzheimer disease: three-and 6-year follow-up of a population-based sample. Arch Neurol 2000;57(6):839–44.
[11] Keith CM, Haut MW, Wilhelmsen K, et al. Frontal and temporal lobe correlates of verbal learning and memory in aMCI and suspected Alzheimer's disease dementia. Aging Neuropsychol Cogn 2023;30(6):923–39.
[12] Jack Jr CR, Bennett DA, Blennow K, et al. NIA-AA research framework: toward a biological definition of Alzheimer's disease. Alzheimers Dement 2018;14(4):535–62.
[13] Jack Jr CR, Andrews JS, Beach TG, et al. Revised criteria for diagnosis and staging of Alzheimer's disease: Alzheimer's Association Workgroup. Alzheimers Dement 2024;20(8):5143–69.
[14] Touroutoglou A, Katsumi Y, Brickhouse M, et al. The sporadic early-onset Alzheimer's disease signature of atrophy: preliminary findings from the longitudinal early-onset Alzheimer's disease study (LEADS) cohort. Alzheimers Dement 2023;19:S74–88.
[15] Villeneuve S, Rabinovici GD, Cohn-Sheehy BI, et al. Existing Pittsburgh Compound-B positron emission tomography thresholds are too high: statistical and pathological evaluation. Brain 2015;138(7):2020–33.
[16] Delis D, Kramer J, Kaplan E, et al. *California verbal learning test. Adult version. Manual 2nd ed.*. San Antonio, TX: The Psychological Corporation; 2000.
[17] Keith CM, Haut MW, Vieira Ligo Teixeira C, et al. Memory consolidation, temporal and parietal atrophy, and metabolism in amyloid-β positive and negative mild cognitive impairment. J Alzheimer's Dis 2024;102(3):778–91.
[18] Folstein MF, Folstein SE, McHugh PR. Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975;12:189–98.
[19] Pfeffer RI, Kurosaki TT, Harrah Jr CH, Chance JM, Filos S. Measurement of functional activities in older adults in the community. J Gerontol 1982;37:323–9.
[20] Kaufer DI, Cummings JL, Ketchel P, et al. Validation of the NPI-Q, a brief clinical form of the neuropsychiatric inventory. J Neuropsychiatry Clin Neurosci 2000;12:233–9.
[21] Wilkinson GS, Robertson GJ. WRAT 4: wide range achievement test. Lutz, FL: Psychological Assessment Resources; 2006.
[22] Huo Y, Xu Z, Xiong Y, Aboud K, et al. 3D whole brain segmentation using spatially localized atlas network tiles. Neuroimage 2019;194:105–19.
[23] Schmidt P, Gaser C, Arsic M, et al. An automated tool for detection of FLAIR-hyperintense white-matter lesions in multiple sclerosis. Neuroimage 2012;59(4):3774–83.
[24] Wang H, Serap Monkul Nery E, Ardayfio P, et al. Modified titration of donanemab reduces ARIA risk and maintains amyloid reduction. Alzheimer's & Dementia 2025;21(4):e70062.
[25] Bondi MW, Monsch AU, Galasko D, Butters N, Salmon DP, Delis DC. Preclinical cognitive markers of dementia of the Alzheimer type. Neuropsychol 1994;8(3):374.
[26] Curiel RE, Vaillancourt D, Ortega A, et al. Semantic intrusion errors differentiate between amnestic MCI who are plasma p-tau217+ from p-tau217-after adjusting for initial learning strength. Front Neurol 2025;16:1613694.
[27] Loewenstein DA, Curiel RE, DeKosky S, et al. Utilizing semantic intrusions to identify amyloid positivity in mild cognitive impairment. Neurol 2018;91(10):e976–84.
[28] Keith CM, Haut MW, D'Haese PF, et al. More similar than different: memory, executive functions, cortical thickness, and glucose metabolism in biomarker-positive Alzheimer's disease and behavioral variant frontotemporal dementia. Alzheimers Dis Rep 2024;8(1):57–73.
[29] Salloway S, Wojtowicz J, Voyle N, et al. Amyloid-related imaging abnormalities (ARIA) in clinical trials of gantenerumab in early Alzheimer disease. JAMA Neurol 2025;82(1):19. -.
[30] Tang C, Ding Y, Yang J, He D. Cerebrovascular disease in Alzheimer's disease: brain structure as a critical mediator of cognitive decline. J Prev Alzheimers Dis 2025;12(8):100209.