

## Review Article

# Anti-Amyloid Monoclonal Antibodies and Amyloid-Related Imaging Abnormalities in Early Alzheimer Disease

Hendriks J<sup>1\*</sup>, Antoniou M<sup>2</sup> and Babic I<sup>3</sup>

<sup>1</sup>Department of Neurology, Erasmus University Rotterdam, Rotterdam, Netherlands

<sup>2</sup>Department of Neuroradiology, National and Kapodistrian University of Athens, Athens, Greece

<sup>3</sup>Department of Neuroimmunology, University of Split, Split, Croatia

**\*Address for Correspondence:** Joost Hendriks, Department of Neurology, Erasmus University Rotterdam, Rotterdam, Netherlands; E-mail: joost.hendriks@erasmusuniversity.edu

**Received:** 11 January 2026; **Accepted:** 23 April 2026; **Published:** 5 August 2026; **DOI:** 10.5281/zenodo.22310607

**Citation of this article:** Hendriks J, Antoniou M and Babic I (2026) Anti-Amyloid Monoclonal Antibodies and Amyloid-Related Imaging Abnormalities in Early Alzheimer Disease. Curr Res Neurol Neurosurg, 9(1): 01-08.

**Copyright:** © 2026 Joost Hendriks, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

### Abstract

**Background:** Lecanemab, donanemab and aducanumab clear cerebral amyloid-beta and modestly slow cognitive decline in early symptomatic Alzheimer disease, but each carries a class-defining safety concern: amyloid-related imaging abnormalities (ARIA).

**Objective:** To review pivotal trial efficacy, ARIA incidence and mechanism, MRI monitoring, and management of anti-amyloid monoclonal antibody therapy.

**Methods:** Narrative review of pivotal randomized trials, imaging-society guidance, pharmacovigilance data, and case reports.

**Findings:** Lecanemab reduced clinical decline by roughly 27% over placebo, with ARIA-E in 12.6% of treated patients; donanemab slowed decline by about 35%, with ARIA-E near 24%. Aducanumab trials gave discordant results. ARIA risk rises several-fold in APOE4 homozygotes, and rare fatal hemorrhages have followed anticoagulation or thrombolysis.

**Conclusions:** Anti-amyloid antibodies offer modest, genuine benefit, but safe use depends on genotype-informed eligibility screening and structured serial MRI monitoring.

**Keywords:** Alzheimer disease; Lecanemab; Donanemab; Aducanumab; Amyloid-related imaging abnormalities

## Abstract

**Background:** Lecanemab, donanemab and aducanumab clear cerebral amyloid-beta and modestly slow cognitive decline in early symptomatic Alzheimer disease, but each carries a class-defining safety concern: amyloid-related imaging abnormalities (ARIA). **Objective:** To review pivotal trial efficacy, ARIA incidence and mechanism, MRI monitoring, and management of anti-amyloid monoclonal antibody therapy. **Methods:** Narrative review of pivotal randomized trials, imaging-society guidance, pharmacovigilance data, and case reports. **Findings:** Lecanemab reduced clinical decline by roughly 27% over placebo, with ARIA-E in 12.6% of treated patients; donanemab slowed decline by about 35%, with ARIA-E near 24%. Aducanumab trials gave discordant results. ARIA risk rises several-fold in APOE4 homozygotes, and rare fatal hemorrhages have followed anticoagulation or thrombolysis. **Conclusions:** Anti-amyloid antibodies offer modest, genuine benefit, but safe use depends on genotype-informed eligibility screening and structured serial MRI monitoring.

**Keywords:** Alzheimer disease; lecanemab; donanemab; aducanumab; amyloid-related imaging abnormalities

## Introduction

Alzheimer disease is defined pathologically by extracellular amyloid-beta plaques and intracellular neurofibrillary tau tangles, and for three decades the "amyloid hypothesis" held that removing amyloid-beta should slow clinical decline. Early attempts to test this hypothesis with antibodies that bind soluble or monomeric amyloid-beta largely failed: solanezumab, targeting soluble amyloid-beta, did not slow decline in mild Alzheimer disease in the EXPEDITION3 trial, reinforcing doubts about the hypothesis itself [1]. The therapeutic picture changed with a newer generation of antibodies engineered to bind aggregated amyloid-beta - protofibrils and fibrillar plaque - which achieve much

greater plaque clearance on amyloid PET than earlier agents.

Aducanumab was the first of this class to show dose-dependent plaque removal in humans, in a small phase 1b study that also first drew attention to a dose-limiting, APOE4-linked imaging finding termed amyloid-related imaging abnormalities [2]. ARIA had in fact been anticipated and formally defined a few years earlier by an Alzheimer's Association Research Roundtable workgroup, which distinguished vasogenic-edema/effusion abnormalities (ARIA-E) from hemosiderin-deposition abnormalities - microhemorrhage and superficial siderosis (ARIA-H) - and proposed severity grading still used today [3,4]. ARIA has since accompanied every plaque-clearing antibody tested in Alzheimer disease, at incidences far exceeding those seen with placebo, and its management has become inseparable from the clinical use of this drug class [5].

This review is a narrative synthesis, not a report of the authors' own cases. It surveys the pivotal efficacy trials of lecanemab, donanemab and aducanumab; the incidence, mechanism and risk factors of ARIA; current MRI monitoring protocols; management of symptomatic disease; real-world safety signals; and patient selection, to give clinicians a single account of benefit and risk in this rapidly evolving field.

The clinical stakes of getting this balance right are considerable. Early symptomatic Alzheimer disease - mild cognitive impairment or mild dementia due to Alzheimer pathology - is precisely the population in which a disease-modifying therapy could plausibly preserve independence for the longest period, yet it is also a population that is often elderly, frequently anticoagulated for atrial fibrillation or venous disease, and heterogeneous in vascular comorbidity. Each of these features intersects directly with ARIA risk, which is why regulatory approval

of this drug class has been accompanied, almost uniquely among neurological therapeutics, by mandatory genotype discussion, structured imaging surveillance, and formal appropriate-use documents rather than a simple label alone.

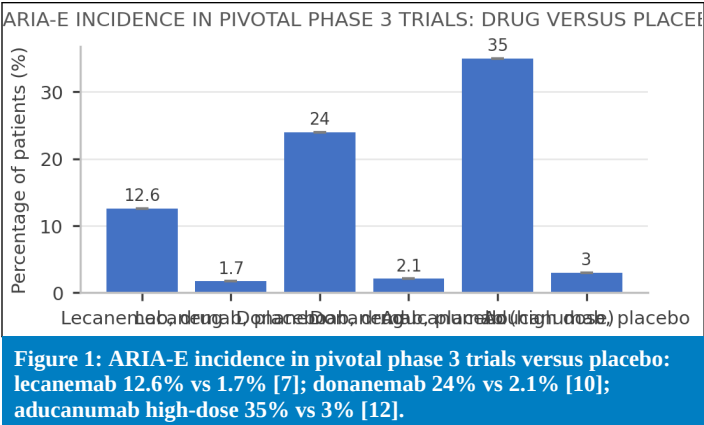
**Trial Evidence for Anti-Amyloid Monoclonal Antibodies**

Lecanemab, an antibody selective for soluble protofibrils, was tested in a phase 2b dose-finding study that established a biomarker- and dose-dependent relationship between amyloid clearance and ARIA-E, particularly in APOE4 carriers [6]. The pivotal phase 3 CLARITY-AD trial randomized 1795 people with early symptomatic Alzheimer disease and confirmed amyloid pathology to lecanemab or placebo for 18 months; the primary endpoint, the Clinical Dementia Rating-Sum of Boxes, declined roughly 27% less with lecanemab than placebo, a statistically significant but clinically modest difference. ARIA-E occurred in 12.6% of lecanemab-treated participants versus 1.7% on placebo, and ARIA-H in 17.3% versus 9.0%; most ARIA was radiographic and asymptomatic, though a minority of cases were symptomatic and severe cases were reported [7].

Donanemab, which targets a pyroglutamate-modified amyloid-beta epitope specific to deposited plaque, first showed amyloid lowering and slowed clinical and tau-PET progression in the phase 2 TRAILBLAZER-ALZ study [8,9]. The phase 3 TRAILBLAZER-ALZ 2 trial, enrolling participants stratified by tau burden, found roughly 35% slowing of decline on the integrated Alzheimer's Disease Rating Scale in the low/medium tau population, alongside ARIA-E in about 24% of treated participants versus 2.1% on placebo, ARIA-H in about 31% versus 14%, and three deaths adjudicated as ARIA-related [10]. A subsequent analysis showed a slower, modified titration schedule preserved amyloid-lowering efficacy while substantially

reducing ARIA-E incidence, informing a revised dosing regimen [11].

| Table 1: Pivotal phase 3 trials of anti-amyloid monoclonal antibodies in early symptomatic Alzheimer disease [7,10,12]. |                         |                                               |                          |                          |
|-------------------------------------------------------------------------------------------------------------------------|-------------------------|-----------------------------------------------|--------------------------|--------------------------|
| Antibody (trial)                                                                                                        | Target                  | Primary endpoint result                       | ARIA-E (drug vs placebo) | ARIA-H (drug vs placebo) |
| Lecanemab (CLARITY-AD)                                                                                                  | Aβ protofibrils         | ~27% slower CDR-SB decline                    | 12.6% vs 1.7%            | 17.3% vs 9.0%            |
| Donanemab (TRAILBLAZER-ALZ 2)                                                                                           | Pyroglutamate-Aβ plaque | ~35% slower iADRS decline                     | 24% vs 2.1%              | 31.4% vs 13.6%           |
| Aducanumab (EMERGE/ENGAGE)                                                                                              | Aggregated Aβ           | Discordant (EMERGE positive, ENGAGE negative) | 35% vs 3% (high dose)    | Not separately reported  |



Aducanumab's two identically designed phase 3 trials, EMERGE and ENGAGE, gave discordant results: EMERGE met its primary endpoint at the high dose while ENGAGE did not, a discrepancy attributed partly to a protocol amendment mid-trial that increased high-dose exposure. ARIA-E occurred in roughly 35% of high-dose participants versus about 3% on placebo [12]. The US Food and Drug Administration granted accelerated approval based on amyloid-lowering as a surrogate endpoint despite the discordant clinical results, a decision that drew sustained criticism from independent reviewers and led to restricted uptake and eventual commercial discontinuation [13].

The aducanumab episode remains instructive precisely because it separated the two questions this review treats together: whether an antibody can lower amyloid burden, which all three agents plainly do, and whether that biological effect reliably translates into a clinical benefit large enough to justify ARIA risk in an individual patient - a judgment that has since driven demand for more granular, genotype-stratified risk-benefit discussion at the point of prescribing.

### Mechanisms and Risk Factors for ARIA

ARIA is thought to arise when antibody binding to vascular and parenchymal amyloid-beta triggers local immune activation, transiently increases blood-brain-barrier permeability, and disrupts perivascular clearance pathways already compromised by coexisting cerebral amyloid angiopathy [14,15]. This mechanistic link to cerebral amyloid angiopathy explains why ARIA-H shares imaging features and risk factors with spontaneous lobar microhemorrhage, and why patients with heavier vascular amyloid burden are more susceptible to both spontaneous and treatment-induced hemorrhagic change [15,5].

The single strongest risk factor identified across trials and meta-analyses is APOE4 genotype, particularly homozygosity, which is associated with several-fold higher ARIA-E incidence than in noncarriers for both lecanemab and donanemab, a pattern confirmed in a pooled meta-analysis of phase 3 anti-amyloid trials [16]. Higher antibody dose, faster titration, and greater baseline amyloid or microhemorrhage burden further increase risk, which is why dose-titration schedules and baseline MRI screening are now integral to treatment protocols rather than optional precautions [11,17].

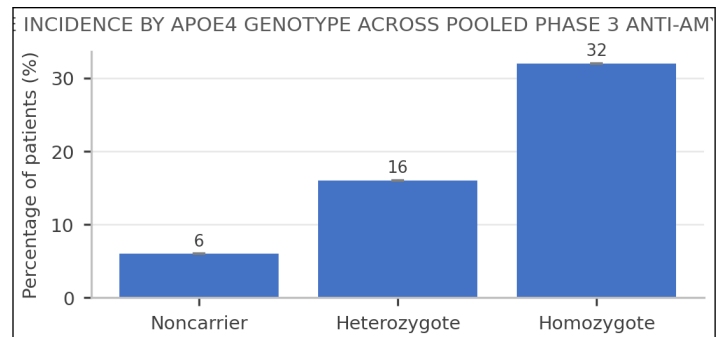


**Figure 2: APOE4 genotype and ARIA-E risk stratification across pooled phase 3 anti-amyloid immunotherapy trials, showing several-fold higher incidence in  $\epsilon 4$  homozygotes than in noncarriers [16].**

Most ARIA-E and ARIA-H events occur early in the treatment course, typically within the first several infusions, then decline in frequency as amyloid burden falls and any residual vascular vulnerability has already been unmasked; recurrent ARIA after dose interruption and rechallenge does occur but appears less severe on average than the index event, informing algorithms that allow cautious re-treatment rather than mandating permanent discontinuation after every episode [5,16]. Whether ARIA reflects a purely adverse immune reaction or, in some patients, an epiphenomenon of the same vascular clearance process that ultimately produces efficacy remains debated, and this uncertainty is one reason management guidance still leans on severity-graded caution rather than a single mechanistic rule [14].

### MRI Monitoring and Diagnostic Approach

Because most ARIA is radiographically detected before symptoms appear, structured surveillance MRI is central to safe use of this drug class. Neuroradiology society guidance recommends baseline MRI before treatment initiation to exclude high burdens of microhemorrhage or siderosis, followed by scheduled surveillance scans timed to early infusions - when ARIA incidence peaks - together with standardized detection and severity-grading terminology for radiology reports [18,17]. Sequence

requirements emphasize fluid-attenuated inversion recovery for ARIA-E and susceptibility-weighted or gradient-echo sequences for ARIA-H, since the two forms of ARIA require different sequences for reliable detection and can coexist [4,19].

Updated practice recommendations refine the timing of surveillance scans around specific infusion numbers for each antibody, provide guidance on when to interrupt or discontinue dosing based on ARIA severity, and address practical issues such as scanner availability and reporting turnaround that determine whether monitoring protocols are followed in real-world practice rather than only in trials [17,19].

**Management of Symptomatic ARIA and Real-World Safety**

Most ARIA is mild, radiographically detected, and resolves with temporary dosing interruption alone; management algorithms in the published appropriate-use recommendations for both lecanemab and donanemab stratify action by ARIA severity grade and symptom status, ranging from continued dosing with enhanced monitoring for mild asymptomatic ARIA-H, through dose interruption for moderate-to-severe or symptomatic ARIA-E, to permanent discontinuation for severe or recurrent events [20,21]. Corticosteroids are commonly used for symptomatic ARIA-E, though evidence for this practice remains largely empirical rather than trial-validated [20].

Real-world experience has surfaced safety signals not fully captured in trial populations. A widely discussed case reported fatal multifocal cerebral hemorrhage in a lecanemab-treated patient who received intravenous thrombolysis for an incident ischemic stroke, illustrating a specific and now widely flagged contraindication: concurrent or recent anticoagulant or thrombolytic therapy substantially raises hemorrhagic risk in ARIA-susceptible patients [22]. A pharmacovigilance

disproportionality analysis of post-marketing adverse event reports corroborated ARIA-E, ARIA-H, cerebral edema and hemorrhage as the dominant safety signals for lecanemab, with most events clustering within the first six weeks of treatment and disproportionately affecting APOE4 carriers, consistent with trial data [23].

| Table 2: Severity-graded management approach to ARIA per published appropriate-use recommendations [20,21]. |                |                                                                   |
|-------------------------------------------------------------------------------------------------------------|----------------|-------------------------------------------------------------------|
| ARIA type/severity                                                                                          | Symptom status | Recommended action                                                |
| ARIA-H, mild                                                                                                | Asymptomatic   | Continue dosing, routine surveillance MRI                         |
| ARIA-E, mild-moderate                                                                                       | Asymptomatic   | Interrupt dosing, repeat MRI before resuming                      |
| ARIA-E or ARIA-H, moderate-severe                                                                           | Symptomatic    | Interrupt dosing, clinical evaluation, corticosteroids considered |
| Recurrent or severe ARIA                                                                                    | Symptomatic    | Permanent discontinuation                                         |

**Patient Selection, Eligibility, and Real-World Considerations**

Eligibility for pivotal trials required biomarker-confirmed amyloid pathology, an early symptomatic stage (mild cognitive impairment or mild dementia), and exclusion of substantial cerebrovascular disease, extensive microhemorrhage, or anticoagulant use - criteria that also inform current appropriate-use recommendations [20,21]. Biomarker confirmation increasingly relies on plasma assays such as phosphorylated tau 217, which discriminates Alzheimer disease from other neurodegenerative disorders with high accuracy and could substantially broaden practical access to eligibility testing beyond amyloid PET or cerebrospinal fluid sampling [24]. Revised diagnostic and staging criteria that formally incorporate biomarker status into Alzheimer disease definitions provide the conceptual scaffold linking diagnosis to treatment eligibility [25].

Translating trial eligibility into clinic populations has proven restrictive: a real-world eligibility study in a French

memory clinic found that only a minority of patients being evaluated for cognitive concerns met pivotal-trial-like eligibility criteria for lecanemab, chiefly because of comorbid vascular imaging findings, anticoagulant use, or disease stage outside the early symptomatic window [26]. This gap between trial and clinical populations means that real-world ARIA rates, and the balance of benefit to risk, may differ from those reported in the pivotal trials, reinforcing the need for continued pharmacovigilance as these agents reach broader practice [23].

## Conclusion

Anti-amyloid monoclonal antibodies have converted decades of amyloid-hypothesis failure into a demonstrable, if modest, clinical effect: lecanemab and donanemab each slow cognitive and functional decline in early symptomatic Alzheimer disease, while aducanumab's discordant trials and subsequent withdrawal illustrate the risk of over-interpreting surrogate biomarker endpoints. Across all three antibodies, amyloid-related imaging abnormalities are the defining safety concern, occurring far more often than with placebo, concentrated in APOE4 carriers and homozygotes, and mechanistically linked to underlying cerebral amyloid angiopathy. Structured baseline and surveillance MRI, genotype-informed risk discussion, avoidance of concurrent anticoagulation or thrombolysis, and severity-graded management algorithms now published as formal appropriate-use recommendations together define safe practice. As eligibility broadens through plasma biomarkers and revised diagnostic criteria, real-world data - already showing that many clinic patients fall outside trial eligibility and that early, APOE4-weighted ARIA signals persist post-marketing - will be essential to confirming that the benefit-risk balance established in pivotal trials holds in everyday neurological practice.

## References

1. Honig, L.S., Vellas, B., Woodward, M., Boada, M., Bullock, R., Borrie, M., Hager, K., Andreasen, N., Scarpini, E., Liu-Seifert, H., Case, M., Dean, R.A., Hake, A., Sundell, K., Hoffmann, V.P., Carlson, C., Khanna, R., Mintun, M., DeMattos, R., Selzler, K.J., Siemers, E. (2018) Trial of Solanezumab for Mild Dementia Due to Alzheimer's Disease. *New England Journal of Medicine*, 378(4): 321-330.
2. Sevigny, J., Chiao, P., Bussière, T., Weinreb, P.H., Williams, L., Maier, M., Dunstan, R., Salloway, S., Chen, T., Ling, Y., O'Gorman, J., Qian, F., Arastu, M., Li, M., Chollate, S., Brennan, M.S., Quintero-Monzon, O., Scannevin, R.H., Arnold, H.M., Engber, T., Rhodes, K., Ferrero, J., Hang, Y., Mikulskis, A., Grimm, J., Hock, C., Nitsch, R.M., Sandrock, A. (2016) The antibody aducanumab reduces A $\beta$  plaques in Alzheimer's disease. *Nature*, 537(7618): 50-56.
3. Sperling, R.A., Jack, C.R., Black, S.E., Frosch, M.P., Greenberg, S.M., Hyman, B.T., Scheltens, P., Carrillo, M.C., Thies, W., Bednar, M.M., Black, R.S., Brashear, H.R., Grundman, M., Siemers, E.R., Feldman, H.H., Schindler, R.J. (2011) Amyloid-related imaging abnormalities in amyloid-modifying therapeutic trials: recommendations from the Alzheimer's Association Research Roundtable Workgroup. *Alzheimer's & Dementia*, 7(4): 367-385.
4. Barakos, J., Sperling, R., Salloway, S., Jack, C., Gass, A., Fiebach, J.B., Tampieri, D., Melançon, D., Miaux, Y., Rippon, G., Black, R., Lu, Y., Brashear, H.R., Arrighi, H.M., Morris, K.A., Grundman, M. (2013) MR Imaging Features of Amyloid-Related Imaging Abnormalities. *American Journal of Neuroradiology*, 34(10): 1958-1965.
5. Hampel, H., Elhage, A., Cho, M., Apostolova, L.G., Nicoll, J.A.R., Atri, A. (2023) Amyloid-related imaging abnormalities (ARIA): radiological, biological and clinical characteristics. *Brain*, 146(11): 4414-4424.
6. Swanson, C.J., Zhang, Y., Dhadda, S., Wang, J., Kaplow, J., Lai, R.Y.K., Lannfelt, L., Bradley, H., Rabe, M., Koyama, A.,

- Reyderman, L., Berry, D.A., Berry, S., Gordon, R., Kramer, L.D., Cummings, J.L. (2021) A randomized, double-blind, phase 2b proof-of-concept clinical trial in early Alzheimer's disease with lecanemab, an anti-A $\beta$  protofibril antibody. *Alzheimer's Research & Therapy*, 13: 80.
7. van Dyck, C.H., Swanson, C.J., Aisen, P., Bateman, R.J., Chen, C., Gee, M., Kanekiyo, M., Li, D., Reyderman, L., Cohen, S., Froelich, L., Katayama, S., Sabbagh, M., Vellas, B., Watson, D., Dhadda, S., Irizarry, M., Kramer, L.D., Iwatsubo, T. (2023) Lecanemab in Early Alzheimer's Disease. *New England Journal of Medicine*, 388(1): 9-21.
  8. Mintun, M.A., Lo, A.C., Duggan Evans, C., Wessels, A.M., Ardayfio, P.A., Andersen, S.W., Shcherbinin, S., Sparks, J., Sims, J.R., Brys, M., Apostolova, L.G., Salloway, S.P., Skovronsky, D.M. (2021) Donanemab in Early Alzheimer's Disease. *New England Journal of Medicine*, 384(18): 1691-1704.
  9. Shcherbinin, S., Evans, C.D., Lu, M., Andersen, S.W., Pontecorvo, M.J., Willis, B.A., Gueorguieva, I., Hauck, P.M., Brooks, D.A., Mintun, M.A., Sims, J.R. (2022) Association of Amyloid Reduction After Donanemab Treatment With Tau Pathology and Clinical Outcomes: The TRAILBLAZER-ALZ Randomized Clinical Trial. *JAMA Neurology*, 79(10): 1015-1024.
  10. Sims, J.R., Zimmer, J.A., Evans, C.D., Lu, M., Ardayfio, P., Sparks, J., Wessels, A.M., Shcherbinin, S., Wang, H., Monkul Nery, E.S., Collins, E.C., Solomon, P., Salloway, S., Apostolova, L.G., Hansson, O., Ritchie, C., Brooks, D.A., Mintun, M., Skovronsky, D.M. (2023) Donanemab in Early Symptomatic Alzheimer Disease: The TRAILBLAZER-ALZ 2 Randomized Clinical Trial. *JAMA*, 330(6): 512-527.
  11. Wang, H., Monkul Nery, E.S., Ardayfio, P., Khanna, R., Otero Svaldi, D., Gueorguieva, I., Shcherbinin, S., Andersen, S.W., Hauck, P.M., Engle, S.E., Brooks, D.A., Collins, E.C., Fox, N.C., Greenberg, S.M., Salloway, S., Mintun, M.A., Sims, J.R. (2025) Modified titration of donanemab reduces ARIA risk and maintains amyloid reduction. *Alzheimer's & Dementia*, 21(4): e70062.
  12. Budd Haeberlein, S., Aisen, P.S., Barkhof, F., Chalkias, S., Chen, T., Cohen, S., Dent, G., Hansson, O., Harrison, K., von Hehn, C., Iwatsubo, T., Mallinckrodt, C., Mummery, C.J., Muralidharan, K.K., Nestorov, I., Nisenbaum, L., Rajagovindan, R., Skordos, L., Tian, Y., van Dyck, C.H., Vellas, B., Wu, S., Zhu, Y., Sandrock, A. (2022) Two Randomized Phase 3 Studies of Aducanumab in Early Alzheimer's Disease. *The Journal of Prevention of Alzheimer's Disease*, 9(2): 197-210.
  13. Alexander, G.C., Knopman, D.S., Emerson, S.S., Ovbiagele, B., Kryscio, R.J., Perlmutter, J.S., Kesselheim, A.S. (2021) Revisiting FDA Approval of Aducanumab. *New England Journal of Medicine*, 385(9): 769-771.
  14. DiFrancesco, J.C., Longoni, M., Piazza, F. (2015) Anti-A $\beta$  Autoantibodies in Amyloid Related Imaging Abnormalities (ARIA): Candidate Biomarker for Immunotherapy in Alzheimer's Disease and Cerebral Amyloid Angiopathy. *Frontiers in Neurology*, 6: 207.
  15. Greenberg, S.M., Bacskai, B.J., Hernandez-Guillamon, M., Pruzin, J., Sperling, R., van Veluw, S.J. (2020) Cerebral amyloid angiopathy and Alzheimer disease - one peptide, two pathways. *Nature Reviews Neurology*, 16(1): 30-42.
  16. Jeong, S.Y., Suh, C.H., Lim, J.S., Shim, W.H., Heo, H., Choi, Y., Kim, H.S., Kim, S.J., Lee, J.H. (2025) Incidence of Amyloid-Related Imaging Abnormalities in Phase III Clinical Trials of Anti-Amyloid- $\beta$  Immunotherapy. *Neurology*, 104(8): e213483.
  17. Cogswell, P.M., Andrews, T.J., Barakos, J.A., Barkhof, F., Bash, S., Chiang, G.C., Franceschi, A.M., Jack, C.R., Pillai, J.J., Poussaint, T.Y., Raji, C.A., Ramanan, V.K., Tanabe, J., Tanenbaum, L., Whitlow, C.T., Yu, F.F., Zaharchuk, G., Zeinab, M., Benzinger, T.S. (2025) Alzheimer Disease Anti-Amyloid Immunotherapies: Imaging Recommendations and Practice Considerations for

- Monitoring of Amyloid-Related Imaging Abnormalities. *American Journal of Neuroradiology*, 46(1): 24-32.
18. Cogswell, P.M., Barakos, J.A., Barkhof, F., Benzinger, T.S., Jack, C.R., Poussaint, T.Y., Raji, C.A., Ramanan, V.K., Whitlow, C.T. (2022) Amyloid-Related Imaging Abnormalities with Emerging Alzheimer Disease Therapeutics: Detection and Reporting Recommendations for Clinical Practice. *American Journal of Neuroradiology*, 43(9): E19-E35.
19. Roytman, M., Mashriqi, F., Al-Tawil, K., Schulz, P.E., Zaharchuk, G., Benzinger, T.L.S., Franceschi, A.M. (2023) Amyloid-Related Imaging Abnormalities: An Update. *American Journal of Roentgenology*, 220(4): 562-574.
20. Cummings, J., Apostolova, L., Rabinovici, G.D., Atri, A., Aisen, P., Greenberg, S., Hendrix, S., Selkoe, D., Weiner, M., Petersen, R.C., Salloway, S. (2023) Lecanemab: Appropriate Use Recommendations. *The Journal of Prevention of Alzheimer's Disease*, 10(3): 362-377.
21. Rabinovici, G.D., Selkoe, D.J., Schindler, S.E., Aisen, P., Apostolova, L.G., Atri, A., Greenberg, S.M., Hendrix, S.B., Petersen, R.C., Weiner, M., Salloway, S., Cummings, J. (2025) Donanemab: Appropriate use recommendations. *The Journal of Prevention of Alzheimer's Disease*, 12(5): 100150.
22. Reish, N.J., Jamshidi, P., Stamm, B., Flanagan, M.E., Sugg, E., Tang, M., Donohue, K.L., McCord, M., Krumpelman, C., Mesulam, M.M., Castellani, R., Chou, S.H.Y. (2023) Multiple Cerebral Hemorrhages in a Patient Receiving Lecanemab and Treated with t-PA for Stroke. *New England Journal of Medicine*, 388(5): 478-479.
23. Yan, L., Zhang, L., Xu, Z., Luo, Z. (2025) A real-world disproportionality analysis of FDA adverse event reporting system (FAERS) events for lecanemab. *Frontiers in Pharmacology*, 16: 1559447.
24. Palmqvist, S., Janelidze, S., Quiroz, Y.T., Zetterberg, H., Lopera, F., Stomrud, E., Su, Y., Chen, Y., Serrano, G.E., Leuzy, A., Mattsson-Carlsson, N., Strandberg, O., Smith, R., Villegas, A., Sepulveda-Falla, D., Chai, X., Proctor, N.K., Beach, T.G., Blennow, K., Dage, J.L., Reiman, E.M., Hansson, O. (2020) Discriminative Accuracy of Plasma Phospho-tau217 for Alzheimer Disease vs Other Neurodegenerative Disorders. *JAMA*, 324(8): 772-781.
25. Hansson, O., Jack, C.R. (2024) A clinical perspective on the revised criteria for diagnosis and staging of Alzheimer's disease. *Nature Aging*, 4(8): 1029-1031.
26. Vrillon, A., Götze, K., Dumurgier, J., Cognat, E., Hourregue, C., Munoz-Musat, E., Decaix, T., Hugon, J., Estrada, J., Sebbagh, M., Bouaziz-Amar, E., Lilamand, M., Paquet, C. (2026) Eligibility for lecanemab treatment in a French memory clinic setting. *Journal of Neurology*, 273(4): 228.