Review paper

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Comparative Evaluation of Donanemab and Lecanemab in Early Alzheimer's Disease: A Clinical Practice-Oriented Narrative Review

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Keywords

Monoclonal antibodies; amyloid beta-peptides; apolipoprotein E; cognitive dysfunction

Abstract

Aim: The regulatory approval of donanemab (Kisunla) and lecanemab (Leqembi) represents a pivotal moment in Alzheimer's disease therapeutics, offering the first treatments that demonstrate meaningful clinical benefits while targeting underlying amyloid pathology. This narrative review provides a comprehensive clinical comparison of these novel anti-amyloid monoclonal antibodies to guide clinical practice decisions. **Materials and Methods:** This narrative review analyzes available clinical evidence on donanemab and lecanemab, focusing on treatment efficacy, amyloid clearance, safety profiles, and factors relevant to clinical implementation. **Results:** Both treatments demonstrate statistically significant slowing of cognitive decline in early Alzheimer's disease, with donanemab showing 35 % slowing in the low-medium tau population (CDR-SB: -1.86 vs placebo -2.42, $p < 0.001$, TRAILBLAZER-ALZ 2 study, 18 months) and lecanemab demonstrating 27 % slowing on clinical rating scales (CDR-SB: -1.21 vs placebo -1.66, $p < 0.001$, CLARITY-AD study, 18 months). Donanemab achieves greater amyloid clearance (84 % reduction at 18 months) compared with lecanemab (68 % reduction at 18 months), potentially allowing for treatment discontinuation after achieving adequate clearance. However, donanemab carries higher rates of amyloid-related imaging abnormalities (ARIA), with ARIA-E occurring in 24 % versus 12.6 % for lecanemab. Clinical implementation requires careful patient selection based on APOE ε4 status, baseline imaging findings, and tau burden assessment. **Conclusion:** This review presents evidence-ba sed decision algorithms for treatment selection, comprehensive ARIA monitoring protocols, and practical implementation strategies. The choice between these therapies should be individualized considering patient-specific risk factors, infrastructure requirements, and cost-effectiveness considerations. While both represent significant therapeutic advances, their modest clinical benefits and safety considerations necessitate judicious patient selection and specialized monitoring capabilities.

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Introduction

Alzheimer's disease (AD) affects over 6.5 million Americans and represents one of the most pressing public health challenges of our time [1]. The pathological hallmarks include accumulation of amyloid-beta (Aβ) plaques and neurofibrillary tangles composed of hyper-

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phosphorylated tau protein [2]. For decades, therapeutic approaches focused primarily on symptomatic treatment with cholinesterase inhibitors and NMDA receptor antagonants, providing only modest relief without addressing underlying pathology [3].

The therapeutic landscape transformed dramatically with the regulatory approvals of lecanemab (Leqembi) by the U.S. Food and Drug Administration (FDA) on January 6, 2023, under accelerated approval, followed by traditional approval on July 6, 2023, and donanemab (Kisunla) receiving FDA approval on July 2, 2024. These represent the first disease-modifying therapies to demonstrate meaningful clinical benefits while targeting amyloid pathology [4,5]. The European Medicines Agency (EMA) approved lecanemab on November 14, 2024, with restrictions to patients with zero or one APOE ε4 allele [6].

Lecanemab, developed by Eisai and Biogen, is a humanized IgG1 monoclonal antibody that binds with high affinity to soluble Aβ protofibrils [7]. Donanemab, developed by Eli Lilly, specifically targets a modified form of deposited Aβ present only in brain amyloid plaques [8]. While both have demonstrated the ability to clear amyloid plaques and slow cognitive decline, their distinct clinical profiles present different risk-benefit considerations for patients with early AD [9,10].

This narrative review synthesizes current evidence to provide clinicians with practical guidance for treatment selection, patient monitoring, and clinical implementation. Rather than attempting a systematic meta-analysis, this review focuses on translating pivotal trial evidence into actionable clinical recommendations, addressing key implementation challenges, and providing decision-making frameworks for real-world practice.

Methods

Search Strategy and Data Sources

This narrative review was conducted in accordance with established principles for clinically oriented review articles. A comprehensive literature search was performed in PubMed/MEDLINE, Embase, and the Cochrane Library, covering the period from January 2020 to September 2024. The search strategy combined Medical Subject Headings (MeSH) and free-text terms related to donanemab, lecanemab, Alzheimer's disease, anti-amyloid therapy, amyloid-related imaging abnormalities (ARIA), monoclonal antibodies, and cognitive decline. The final search was completed on September 30, 2024, and only publications written in English were included.

Inclusion and Exclusion Criteria

Eligible studies included peer-reviewed publications reporting clinical trial outcomes for donanemab or lecanemab, regulatory documents, and clinical practice guidelines. Studies were required to involve patients with early-stage Alzheimer's disease and to provide safety or efficacy data derived from Phase 2 or Phase 3 trials. Preclinical and animal studies, case reports involving fewer than ten patients, studies focused exclusively on other anti-amyloid agents, and non-English publications were excluded.

Data Extraction and Synthesis

Data extraction focused on key domains relevant to clinical implementation, including regulatory approval status, clinical trial efficacy outcomes, safety profiles, monitoring requirements, and practical considerations for routine use. Given the narrative design of the review, formal methodological quality assessment tools were not applied. Instead, emphasis was placed on pivotal Phase 3 trial data and regulatory agency evaluations as the most clinically relevant sources of evidence.

Clinical Trial Evidence and Comparative Effectiveness

Pivotal Trial Characteristics

The efficacy and safety profiles of donanemab and lecanemab were established through large-scale Phase 3 randomized controlled trials with distinct design features that influence clinical interpretation [4,5]. The TRAILBLAZER-ALZ 2 study evaluated donanemab in 1,736 participants with early symptomatic AD, employing a unique tau stratification strategy that divided participants into low-medium tau (n = 1,182) and high tau (n = 552) groups [5]. The low-medium tau population served as the primary efficacy population, representing patients most likely to benefit from amyloid-targeting therapy.

The CLARITY AD trial assessed lecanemab in 1,795 participants with early AD without tau stratification, providing evidence across a broader population spectrum [4]. Both studies enrolled patients with mild cognitive impairment or mild dementia due to AD with confirmed amyloid pathology, establishing the foundation for their respective clinical indications [11,12].

Important Note: All comparisons presented represent indirect, cross-trial comparisons as no direct head-to-head studies have been conducted between donanemab and lecanemab.

Table 1. presents a direct comparison of key clinical parameters between donanemab and lecanemab to facilitate evidence-based treatment selection. The comparison encompasses mechanisms of action, dosing regimens, efficacy outcomes from pivotal trials, safety profiles including ARIA rates, and economic considerations. Notable differences include donanemab's superior amyloid clearance (84 % vs 68 %) but higher ARIA rates (24 % vs 12.6 % for ARIA-E), and the potential for treatment discontinuation with donanemab versus continuous therapy with lecanemab.

Cognitive and Functional Outcomes

Donanemab demonstrated cognitive benefits, particularly in the tau-stratified population. In the low-medium tau group,

treatment resulted in 35 % slowing of decline on the integrated Alzheimer's Disease Rating Scale (iADRS, 18-month timeframe, mean difference 3.20 points, 95 % CI 1.51-4.88) and 36 % slowing on the Clinical Dementia Rating-Sum of Boxes (CDR-SB) scale (18-month timeframe, mean difference 0.56 points, 95 % CI 0.20-0.92) [TRAILBLAZER-ALZ 2 study] [5]. In the combined population, these benefits were attenuated to 22 % and 2 9 % respectively, highlighting the importance of patient selection based on tau burden.

Lecanemab showed consistent but different magnitude benefits across the overall study population, with 27 % slowing of decline on the CDR-SB scale (18 - month timeframe, mean difference -0.45 points, 95 % CI -0.69 to -0.21) and meaningful improvements on the ADAS-Cog14 scale (18-month timeframe, mean difference -1.44 points, 95 % CI -2.27 to -0.61) [CLARITY-AD study] [4,13]. These within-trial effects reflect different study populations and tau stratification approaches rather than direct superiority comparisons. [14].

Biomarker Outcomes and Amyloid Clearance

Both treatments achieved substantial reductions in brain amyloid burden, though with different kinetics and depth of clearance. Donanemab treatment resulted in 84 % reduction in amyloid plaque burden at 18 months (mean change from baseline -79.2 centiloids), with 80 % of participants in the low-medium tau group achieving amyloid clearance (defined as < 24.1 centiloids) [TRAILBLAZER-ALZ 2] [5]. This level of clearance enables the unique treatment discontinuation strategy employed with donanemab.

Lecanemab demonstrated 68 % reduction in amyloid burden at 18 months with a mean difference of -59.1 centiloids compared with placebo (95 % CI -62.9 to -55.3) [CLARITY-AD] [4]. While numerically different from donanemab, this level of clearance was achieved with greater consistency across the study population and may be sufficient for sustained clinical benefit with continued treatment.

Table 1. Comparative Clinical Profile of Donanemab versus Lecanemab*

Parameter	Donanemab	Lecanemab	Source
Regulatory Status			
FDA Approval	July 2, 2024	January 6, 2023 (accelerated); July 6, 2023 (traditional)	FDA.gov
EMA Status	Under review	November 14, 2024 (restricted to 0-1 APOE ε4)	EMA.europa.eu
Mechanism	Targets deposited Aβ in plaques	Targets soluble Aβ protofibrils	Primary trials
Dosing	Monthly IV infusion	Biweekly IV infusion	Product labeling
Efficacy (18 months)			
CDR-SB improvement	36 % slowing (low-medium tau)	27% slowing (overall population)	TRAILBLAZER-ALZ 2; CLARITY-A
Primary endpoint	iADRS: 3.20 points	CDR-SB: -0.45 points	Primary trials
Amyloid clearance	84 % reduction	68 % reduction	Primary trials
Safety Profile	24.0 %	12.6 %	
ARIA-E rate	31.4 %	16.9 %	Primary trials
ARIA-H rate			Primary trials
APOE ε4 homozygous ARIA-E	40.6 %	Restricted population	TRAILBLAZER-ALZ 2; EMA guidance
Implementation			
Treatment duration	Limited (discontinuation after clearance)	Continuous	Clinical protocols
Annual cost (USD)	$32,000-$39,000	$26,500	Published analyses

*Indirect, cross-trial comparison; no head-to-head data available
Abbreviations: Aβ = amyloid-beta; CDR-SB = Clinical Dementia Rating-Sum of Boxes; iADRS = integrated Alzheimer's Disease Rating Scale; ARIA-E = amyloid-related imaging abnormalities with edema; ARIA-H = amyloid-related imaging abnormalities with hemorrhage; APOE = apolipoprotein E; IV = intravenous; FDA = Food and Drug Administration; EMA = European Medicines Agency.

Safety Profile and Risk Assessment

Amyloid-Related Imaging Abnormalities (ARIA)

ARIA represents the most significant safety concern for both treatments, manifesting as either edema/effusion (ARIA-E) or hemorrhage/hemosiderin deposits (ARIA-H) [15,16]. The incidence and severity patterns differ between the two therapies, influencing clinical decision-making and monitoring requirements.

Treatment with donanemab was associated with a notable incidence of amyloid-related imaging abnormalities. ARIA with edema or effusion (ARIA-E) occurred in 24.0 % of treated participants compared with 2.1 % in the placebo group. ARIA with hemorrhagic manifestations (ARIA-H) was observed in 31.4 % of treated patients versus 13.6 % receiving placebo. Overall, any form of ARIA was reported in 37 % of patients treated with donanemab. Symptomatic ARIA occurred in 6.1 % of participants, and severe ARIA-E requiring hospitalization was documented in 1.6 % of cases [17].

Lecanemab demonstrated a lower overall incidence of ARIA compared with donanemab. ARIA-E occurred in 12.6 % of treated patients versus 1.7 % in the placebo group, while ARIA-H was observed in 16.9 % compared with 8.7 % in placebo. The overall incidence of ARIA was 21.3 %. Symptomatic ARIA was reported in 2.8 % of patients, and severe ARIA-E requiring medical intervention occurred in 0.7 % of cases [4].

ARIA Risk Stratification by APOE ε4 Status

APOE ε4 genotype represents the strongest known predictor of ARIA risk [18,19]. In patients treated with donanemab, ARIA-E occurred in 15.8 % of non-carriers, 20.1 % of heterozygotes, and as many as 40.6 % of homozygotes [17]. In contrast, lecanemab was associated with a lower overall risk of ARIA-E, estimated at approximately 8.9 % among non-carriers and heterozygotes combined [20]. Data for homozygous carriers remain limited, particularly in Europe, due to regulatory restrictions on treatment eligibility.

MRI Monitoring Algorithm

Safe administration of anti-amyloid therapy requires a standardized MRI protocol for detection and monitoring of ARIA. Fluid-attenuated inversion recovery (FLAIR) sequences in axial and sagittal planes are essential for identifying ARIA-E, while T2-weighted gradient-echo (GRE) or susceptibility-weighted imaging (SWI) sequences are necessary for detecting ARIA-H. T1-weighted sequences provide structural assessment and serve as a baseline for comparison.

Management decisions should be guided by the severity of imaging findings. Mild ARIA-E lesions measuring ≤1 cm may allow continuation of therapy with intensified monitoring. Moderate lesions between 1 and 4 cm generally warrant temporary treatment interruption, with resumption considered after radiological resolution. Severe ARIA-E lesions exceeding 4 cm require mandatory treatment suspension and careful reassessment before any rechallenge. The presence of symptomatic ARIA necessitates immediate interruption of therapy regardless of lesion size. Newly detected or progressive ARIA-H should be evaluated individually, with consideration given to permanent discontinuation when clinically appropriate.

Treatment Selection and Clinical Implementation

Patient Selection Framework

Optimal patient selection requires integration of clinical presentation, biomarker confirmation, and neuroimaging findings [21,22]. The most appropriate candidates are individuals with early symptomatic AD, including mild cognitive impairment or mild dementia, with confirmed amyloid pathology demonstrated by PET imaging or cerebrospinal fluid analysis.

When considering donanemab, priority should be given to patients with low to intermediate tau burden, those willing to accept a higher ARIA risk in exchange for potentially greater amyloid clearance, and individuals able to adhere to monthly infusions and intensive MRI surveillance. Patients should also understand the potential for treatment discontinuation once adequate amyloid clearance has been achieved.

Lecanemab may be preferable for patients across a broader range of tau burden who favor a more predictable safety profile and are willing to commit to continuous biweekly treatment. In European clinical practice, APOE ε4 status requires particular attention because of regulatory limitations affecting treatment eligibility.

Figure 1 provides a systematic clinical decision algorithm for early Alzheimer's disease treatment with anti-amyloid therapies.

Infrastructure Requirements

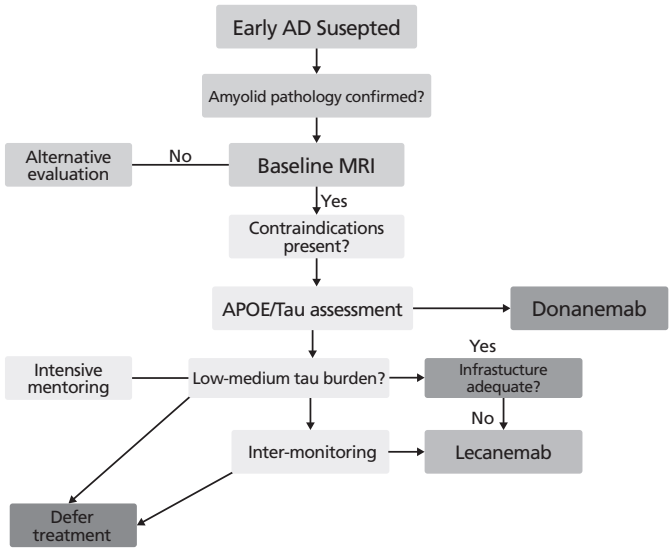
Implementation of anti-amyloid therapies requires substantial healthcare infrastructure. Dedicated infusion facilities staffed by trained personnel must be available, along with protocols for the management of infusion-related reactions and appropriate monitoring equipment. Reliable access to high-quality brain MRI with standardized ARIA protocols is essential, as is the availability of radiologists experienced in ARIA detection and severity grading. Rapid reporting, ideally within 48 hours, is necessary to support timely clinical decision-making.

In addition, diagnostic support should include access to amyloid PET imaging or cerebrospinal fluid analysis, APOE ε4 genotyping, and, where available, emerging blood-based biomarker testing to facilitate patient selection and monitoring.

Limitations

Several limitations should be considered when interpreting this review. The narrative design does not incorporate formal methodological quality assessment and may therefore introduce selection bias. All comparisons between donanemab and lecanemab are indirect and based on cross-trial analyses, as no head-to-head randomized controlled trials have been conducted. Furthermore, the pivotal studies differed in inclusion criteria, tau stratification approaches, and primary endpoints, which limits the validity of direct comparisons.

Figure 1. Clinical Decision Algorithm for Early Alzheimer's Disease Treatment Selection



Author-created decision algorithm based on clinical trial data and regulatory guidance.

This flowchart provides a systematic approach for clinicians to select appropriate anti-amyloid therapy based on patient characteristics, biomarker profiles, and infrastructure considerations. Key decision points include confirmed early AD diagnosis with amyloid pathology, tau burden assessment, APOE ε4 genotype, baseline MRI findings, and patient preferences regarding treatment duration and monitoring intensity.

Real-world evidence remains limited for both therapies, restricting insight into long-term effectiveness and safety in routine clinical practice. The focus on published Phase 3 data may also introduce publication bias and fail to capture rare adverse events. Finally, regulatory differences between jurisdictions, particularly the European restrictions for lecanemab, limit the generalizability of clinical recommendations.

Clinical Practice Recommendations

Treatment Selection

For patients with early Alzheimer's disease and confirmed amyloid pathology, treatment decisions should be individualized based on tau burden, APOE ε4 status, comorbidities, and local infrastructure capabilities. The potential cognitive benefits must be carefully weighed against the risk of ARIA, particularly in APOE ε4 homozygotes or in patients with baseline microhemorrhages. Before initiating therapy, clinicians should verify that the necessary monitoring and emergency management resources are available.

Monitoring Protocols

Baseline evaluation should include a comprehensive medical history, standardized cognitive assessment, brain MRI using an ARIA protocol, APOE ε4 genotyping, and structured patient counseling regarding benefits and risks. During treatment, MRI surveillance should follow a risk-stratified schedule interpreted by experienced radiologists, with clearly defined criteria for

Table 2. ARIA Monitoring Schedule for Donanemab and Lecanemab

Treatment	Time Point	Week	MRI Type	Clinical Rationale	MRI Sequences Required
Donanemab					
	Baseline	0	Brain MRI	Baseline assessment, exclusion criteria	FLAIR, T2*, T1
	Pre-Infusion 5	10	Safety MRI	Early ARIA detection (peak risk period)	FLAIR, T2*, T1
	Pre-Infusion 7	14	Safety MRI	Continued safety monitoring	FLAIR, T2*, T1
	Pre-Infusion 14	26	Safety MRI	Long-term safety assessment	FLAIR, T2*, T1
	Monthly visits	30+	Clinical assessment	Routine clinical monitoring	As clinically indicated
	Symptom-driven	Variable	Symptom MRI	New neurological symptoms	FLAIR, T2*, T1
Lecanemab					
	Baseline	0	Brain MRI	Baseline assessment, exclusion criteria	FLAIR, T2*, T1
	Pre-Infusion 7	14	Safety MRI	Safety monitoring	FLAIR, T2*, T1
	Pre-Infusion 14	26	Safety MRI	Continued safety assessment	FLAIR, T2*, T1
	Biweekly visits	28+	Clinical assessment	Routine clinical monitoring	As clinically indicated
	Symptom-driven	Variable	Symptom MRI	New neurological symptoms	FLAIR, T2*, T1

Abbreviations: ARIA = amyloid-related imaging abnormalities; MRI = magnetic resonance imaging; FLAIR = fluid-attenuated inversion recovery; T2* = T2-star weighted imaging.

treatment interruption or discontinuation. Institutions should also establish standardized protocols for the management of adverse events and for clinical decision-making regarding treatment rechallenge when appropriate [23,24].

Table 2. provides the standardized MRI monitoring protocol essential for safe implementation of anti-amyloid therapies. The schedule reflects risk-stratified monitoring, with more intensive surveillance for donanemab due to its higher ARIA incidence. Critical time points include pre-infusion safety MRIs at weeks 10 and 14, with ongoing clinical monitoring and symptom-driven imaging as indicated. All MRI assessments require standardized sequences (FLAIR, T2*, T1) and trained radiologists for optimal ARIA detection.

Figure 2 illustrates the MRI monitoring schedule for the detection and surveillance of ARIA during treatment with donanemab and lecanemab. The monitoring framework includes several categories of assessment: baseline brain MRI performed prior to treatment initiation to establish reference findings, scheduled safety MRI examinations conducted at predefined intervals for early detection of ARIA, routine clinical visits for ongoing clinical evaluation, and additional symptom-driven MRI scans performed in response to newly emerging neurological symptoms. The monitoring intensity is highest during the initial phase of therapy, as the first six months of treatment represent the period of greatest risk for the development of ARIA.

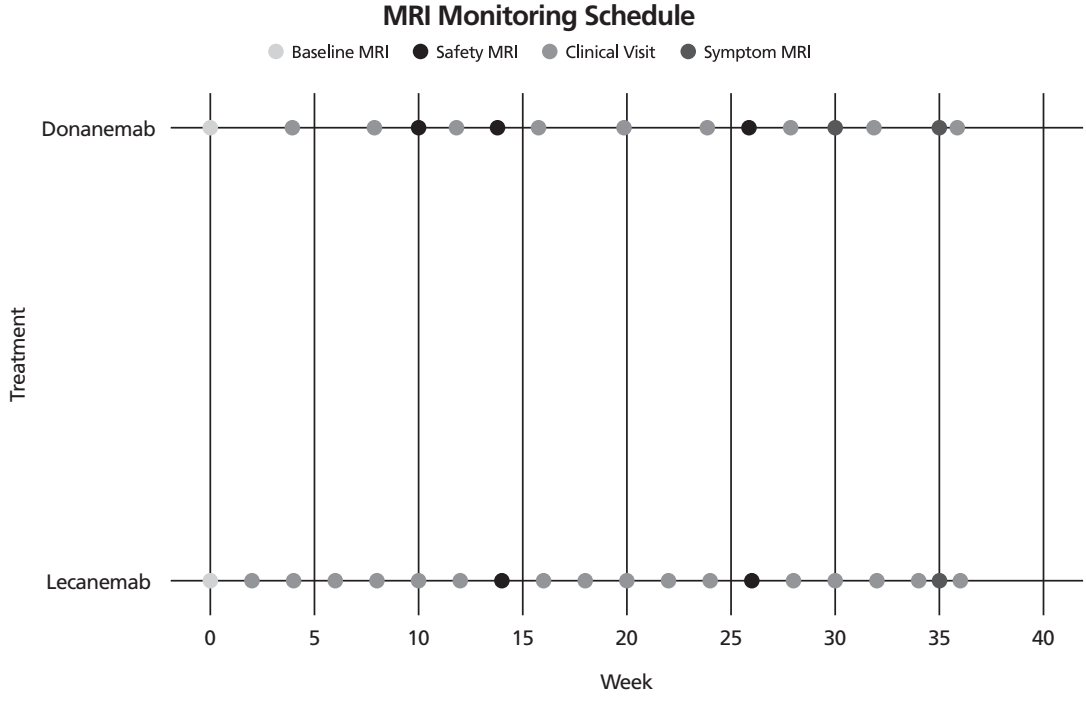
Conclusion

The regulatory approvals of donanemab and lecanemab represent significant milestones in Alzheimer's disease therapeutics, offering the first treatments that meaningfully slow disease progression while targeting underlying pathology. While both demonstrate statistically significant clinical benefits within their respective trials, their implementation requires careful consideration of individual patient factors, safety profiles, and healthcare system capabilities.

Based on within-trial evidence, donanemab may provide greater amyloid clearance, particularly in patients with low-medium tau burden, while offering the potential for treatment discontinuation after achieving amyloid clearance. However, its higher ARIA risk profile necessitates enhanced monitoring and careful patient selection. Lecanemab demonstrates consistent effects across diverse patient populations with a different safety profile, making it potentially more suitable for broader clinical implementation.

The choice between these treatments should be individualized based on patient-specific risk factors, tau burden assessment, APOE ε4 status, and healthcare system capabilities. Both treatments require specialized infrastructure, comprehensive patient education, and

Figure 2. ARIA Surveillance Timeline for Donanemab and Lecanemab Therapy



Author-created decision algorithm based on clinical trial data and regulatory guidance.

standardized monitoring protocols to ensure safe and effective implementation.

While these advances represent important progress, their modest clinical benefits and significant implementation challenges underscore the continued need for more effective and accessible therapies. Future developments in blood-based biomarkers, combination therapies, and precision medicine approaches may further improve outcomes for patients with Alzheimer's disease.

The successful implementation of these therapies will require coordinated efforts across healthcare systems, regulatory agencies, and payers to ensure equitable access while maintaining appropriate safety standards. Clinicians must remain vigilant in patient selection, monitoring, and communication to optimize the risk-benefit ratio for each individual patient.

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None.

Conflict of Interest

None to declare.

Funding Sources

None.

References

1. Alzheimer's disease facts and figures. Alzheimers Dement. 2023;19:1598-695.
2. Jack Jr CR, Bennett DA, Blennow K, Carrillo MC, Dunn B, Haeberlein SB, et al. NIA-AA research framework: toward a biological definition of Alzheimer's disease. Alzheimers Dement. 2018;14:535-62.
3. Cummings J, Zhou Y, Lee G, Zhong K, Fonseca J, Cheng F. Alzheimer's disease drug development pipeline: 2023. Alzheimers Dement (N Y). 2023;9:e12385.
4. van Dyck CH, Swanson CJ, Aisen P, Bateman RJ, Chen C, Gee M, et al. Lecanemab in early Alzheimer's disease. N Engl J Med. 2023;388:9-21.
5. Sims JR, Zimmer JA, Evans CD, Lu M, Ardayfio P, Sparks J, et al. Donanemab in early symptomatic Alzheimer disease: The TRAILBLAZER-ALZ 2 randomized clinical trial. JAMA. 2023;330:512-27.
6. Espay AJ, Kepp KP, Herrup K. Lecanemab and donanemab as therapies for Alzheimer's disease: an illustrated perspective on the data. eNeuro. 2024;11:ENEURO.0319-23.
7. Satlin A, Wang J, Logovinsky V, Berry S, Swanson C, Dhadda S, et al. Design of a Bayesian adaptive phase 2 proof-of-concept trial for BAN2401, a putative disease-modifying monoclonal antibody for the treatment of Alzheimer's disease. Alzheimers Dement (N Y). 2016 4;2:1-12.
8. Mintun MA, Lo AC, Duggan Evans C, Wessels AM, Ardayfio PA, Andersen SW, et al. Donanemab in early Alzheimer's disease. N Engl J Med. 2021;384:1691-704.
9. Jin M, Noble JM. What's in It for Me? Contextualizing the potential clinical impacts of lecanemab, donanemab, and other anti-β-amyloid monoclonal antibodies in early Alzheimer's disease. eNeuro. 2024;11:ENEURO.0088-24.2024.
10. Huang LK, Kuan YC, Lin HW, Hu CJ. Clinical trials of new drugs for Alzheimer disease: a 2020–2023 update. J Biomed Sci. 2023;30:83.
11. Chertkow H, Phillips N, Rockwood K, Anderson N, Andrew MK, Bartha R, et al. Impact of a national dementia research consortium: the Canadian Consortium on Neurodegeneration in Aging (CCNA). J Alzheimers Dis. 2024;102:535-61.
12. Tarawneh R, Pankratz VS. The search for clarity regarding "clinically meaningful outcomes" in Alzheimer disease clinical trials: CLARITY-AD and Beyond. Alzheimers Res Ther. 2024;16:37.
13. Honig LS, Barakos J, Dhadda S, Kanekiyo M, Reyderman L, Irizarry M, et al. ARIA in patients treated with lecanemab (BAN2401) in a phase 2 study in early Alzheimer's disease. Alzheimers Dement (N Y) 2023;9:e12377.
14. Zeng B, Tang C, Wang J, Yang Q, Ren Q, Liu X. Pharmacologic and nutritional interventions for early Alzheimer's disease: a systematic review and network meta-analysis of randomized controlled trials. J Alzheimers Dis. 2024;99:1173-86.
15. Barakos J, Sperling R, Salloway S, Jack C, Gass A, Fiebach JB, et al. MR Imaging features of amyloid-related imaging abnormalities. AJNR Am J Neuroradiol. 2013;34:1958-65.
16. Salloway S, Chalkias S, Barkhof F, Burkett P, Barakos J, Purcell D, et al. Amyloid-Related imaging abnormalities in 2 phase 3 studies evaluating aducanumab in patients with early Alzheimer disease. JAMA Neurol. 2022;79:13-21.
17. Zimmer JA, Ardayfio P, Wang H, Khanna R, Evans CD, Lu M, et al. Amyloid-related imaging abnormalities with donanemab in early symptomatic Alzheimer disease: secondary analysis of the TRAILBLAZER-ALZ and ALZ 2 randomized clinical trials. JAMA Neurol. 2025;82:461-9.
18. Reiman EM, Arboleda-Velasquez JF, Quiroz YT, Huentelman MJ, Beach TG, Caselli RJ, et al. Exceptionally low likelihood of Alzheimer's dementia in APOE2 homozygotes from a 5,000-person neuropathological study. Nat Commun. 2020;11:667.
19. O'Brien RJ, Resnick SM, Zonderman AB, Ferrucci L, Crain BJ, Pletnikova O, et al. Neuropathologic studies of the Baltimore Longitudinal Study of Aging (BLSA). J Alzheimers Dis. 2009;18:665-75.
20. Wise J. Lecanemab: European drug agency upholds decision to recommend Alzheimer's drug. BMJ. 2025;388:r439.
21. Rabinovici GD, Karlawish J, Knopman D, Snyder HM, Sperling R, Carrillo MC. Testing and disclosures related to amyloid imaging and Alzheimer's disease: common questions and fact sheet summary. Alzheimers Dement. 2016;12:510-5.
22. Rabinovici GD, Carrillo MC, Forman M, DeSanti S, Miller DS, Kozauer N, et al. Multiple comorbid neuropathologies in the setting of Alzheimer's disease neuropathology and implications for drug development. Alzheimers Dement. 2017;3:83-91.
23. Ostrowitzki S, Lasser RA, Dorflinger E, Scheltens P, Barkhof F, Nikolcheva T, et al. A phase III randomized trial of gantenerumab in prodromal Alzheimer's disease. Alzheimers Res Ther. 2017;9:95.
24. Logovinsky V, Satlin A, Lai R, Swanson C, Kaplow J, Osswald G, et al. Safety and tolerability of BAN2401 - a clinical study in Alzheimer's disease with a protofibril selective Aβ antibody. Alzheimers Res Ther. 2016;8:14.