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DONANEMAB AND THE FUTURE OF ALZHEIMER'S TREATMENT: BETWEEN INNOVATION AND CAUTION

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ABSTRACT

Introduction and Objective: Alzheimer's disease (AD) is the leading cause of dementia, mainly affecting people over 65. Its growing prevalence, driven by population aging, highlights the need for disease-modifying therapies. This article reviews donanemab - a monoclonal antibody targeting β-amyloid plaques - with emphasis on efficacy, safety, and regulatory landscape.
State of Knowledge: Donanemab may slow cognitive decline in early AD, especially in patients with low-to-intermediate tau levels. Clinical trials showed a 35% reduction in disease progression. However, the treatment is associated with amyloid-related imaging abnormalities (ARIA), particularly in APOE ε4 carriers.
Conclusions: Donanemab offers meaningful clinical benefits in early AD, but safety concerns and inconsistent regulatory decisions complicate its implementation. Though approved in the U.S., Japan, and China, it was denied EMA authorization due to ARIA risks. In Poland, access is limited to individual cases via the Named Patient Import Scheme. Its future use will require precise patient selection and continued safety monitoring. The case of donanemab illustrates the broader tension between therapeutic innovation and patient protection.

KEYWORDS

Alzheimer Disease, Amyloid β-Peptides, Donanemab, Adverse Drug Reactions, Efficacy Studies

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Introduction

Methodology and purpose

The article aims to present the current evidence on the use of donanemab in the treatment of Alzheimer's disease (AD), including a comparison of global recommendations, a review of relevant studies, and a critical analysis of its advantages and limitations. Relevant studies were identified by searching PubMed and Google Scholar with the following terms: "donanemab", "Alzheimer's Disease", "dementia", "TRAILBLAZER-ALZ clinical trial", and "neurodegenerative diseases".

Alzheimer's disease

Alzheimer's disease is the most common neurodegenerative disorder mainly affecting individuals aged over 65 and represents 60% - 80% of dementia cases [1]. AD is characterized by the extracellular accumulation of β-amyloid plaques and the intracellular presence of neurofibrillary tangles, which are primarily composed of hyperphosphorylated tau protein. Additionally, AD is characterized by the loss of synapses and neurons, as well as gliosis [2, 3].

Epidemiology

The most recent data indicate that, by 2050, the prevalence of dementia will double in Europe and triple worldwide, primarily as a consequence of global population aging, which has become a widespread and universal demographic phenomenon [4]. As the proportion of older individuals increases, the incidence of age-related disorders such as dementia - and consequently AD - is expected to rise significantly. Over the coming three decades, the number of older individuals worldwide is expected to rise significantly, exceeding 1.5 billion by 2050. The number of people aged 65 years or older worldwide is projected to more than double, rising from 761 million in 2021 to 1.6 billion in 2050. The number of people aged 80 years or older is growing even faster [5]. Since the risk of AD increases significantly with age, the worldwide growth of the aging population is expected to place a considerable strain on public health systems and long-term care infrastructures across both developed and developing nations; consequently, as AD becomes an increasingly prevalent condition, there is a growing urgency to develop and investigate new therapeutic strategies to address its progression and impact [6].

Treatment strategy

AD is a complex neurodegenerative condition, and the development of effective therapeutic modalities is crucial for managing symptoms, slowing disease progression, and improving overall patient quality of life. Current therapeutic paradigms include both pharmacological and non-pharmacological interventions. Nevertheless, despite the availability of symptomatic treatments, AD continues to progress, with patients experiencing a gradual decline in cognitive function and ability to perform daily activities over time [7, 8].

Given the multifactorial and complex nature of AD, current pharmacological treatment strategies primarily involve selective agents that target specific biological pathways. Among these, three cholinesterase inhibitors - donepezil, rivastigmine, and galantamine - work by enhancing cholinergic neurotransmission through the inhibition of acetylcholinesterase, which increases acetylcholine levels in the brain [9, 10]. In contrast, memantine, an N-methyl-D-aspartate (NMDA) receptor antagonist, exerts its effects by selectively inhibiting the excessive, prolonged activation of NMDA receptors, a process implicated in excitotoxicity and neuronal damage associated with AD pathology [11].

More recently, donanemab has emerged as a promising new therapeutic option. It represents a novel, disease-modifying treatment aimed at slowing disease progression by promoting the clearance of amyloid plaques from the brain [12].

Donanemab

Donanemab is a humanized monoclonal antibody (IgG1) that specifically targets a pyroglutamate-modified, N-terminally truncated form of β-amyloid, known as N3pG-Aβ, which is found exclusively in insoluble amyloid plaques within the brains of individuals with AD [13, 14]. Due to its high specificity, donanemab selectively binds to insoluble, plaque-associated β-amyloid aggregates, reducing interference from soluble β-amyloid species that could otherwise hinder effective targeting of deposited forms [15, 16]. Upon binding to these aggregated forms, donanemab promotes their clearance by activating microglial cells - the resident immune cells of the central nervous system - through Fc receptor-mediated phagocytosis [13, 16]. This immunological mechanism reduces amyloid plaque burden and is believed to slow the clinical progression of Alzheimer's disease. Unlike conventional pharmacotherapies that modulate neurotransmitter systems such as NMDA or cholinergic receptors, donanemab does not act through synaptic signaling pathways. Instead, it adopts a disease-modifying approach by directly targeting a core pathological hallmark of AD: the accumulation of aggregated β-amyloid in the form of insoluble plaques [13, 16, 17].

Treatment with donanemab significantly slowed the decline on the primary outcome measure of integrated Alzheimer's Disease Rating Scale (iADRS) by 40% and demonstrated improvement in all secondary clinical endpoints [18].

Efficacy and safety of donanemab in the TRAILBLAZER-ALZ 2 trial

TRAILBLAZER-ALZ 2 is a Phase 3, placebo-controlled clinical trial conducted by Eli Lilly to evaluate the efficacy and safety of donanemab in patients with early symptomatic AD. The study included 1,736 participants with mild cognitive impairment (MCI) or mild dementia due to AD, confirmed by amyloid and tau PET imaging (Table 1) [13, 17].

Donanemab significantly slowed cognitive and functional decline compared to placebo. Patients with low-to-intermediate tau levels treated with donanemab experienced a 35.1% slower decline on the iADRS over 76 weeks ($p<0.001$) [13]. Functional decline assessed by the Clinical Dementia Rating–Sum of Boxes (CDR-SB) was reduced by 36.0% (mean difference: −0.67; $p<0.001$) [13, 17].

Table 1. Patient Inclusion and Exclusion Criteria for the TRAILBLAZER-ALZ 2 Clinical Trial.

Inclusion Criteria	Exclusion Criteria
• **Gradual and progressive change in memory function** reported by participants or informants for ≥6 months. • **Mini-Mental State Examination (MMSE) score** of 20 to 28 (inclusive) at baseline. • **Positive amyloid PET scan** using 18F-florbetapir or 18F-florbetaben (central read). • **Positive tau PET scan** using 18F-flortaucipir (central read). • **Study partner** who will provide written informed consent to participate.	• **Contraindication to MRI or PET scans**. • **Current treatment with immunoglobulin G (IgG) therapy**. • **Presence of amyloid-related imaging abnormalities (ARIA)**, including: ARIA-E (edema or effusions). ARIA-H (hemorrhages). • **More than 4 cerebral microhemorrhages**. • **More than 1 area of superficial siderosis**. • **Any intracerebral hemorrhage >1 cm**. • **Severe white matter disease** on MRI.

Moreover, the risk of advancing to the next clinical stage of disease was reduced by 38.6% in the donanemab group compared to placebo [13]. Biomarker analysis showed that amyloid plaque clearance occurred in over 80% of treated patients by week 76, and plasma P-tau217 levels decreased by 39.3%, indicating downstream effects on tau pathology [13, 19]. However, safety remains a critical consideration.

Amyloid-related imaging abnormalities (ARIA): Definition and Clinical Revelance

Amyloid-Related Imaging Abnormalities (ARIA) are a spectrum of magnetic resonance imaging (MRI) findings observed in patients undergoing anti-amyloid β immunotherapy for AD [20]. ARIA is categorized into two types: ARIA-E (edema and effusion) and ARIA-H (hemorrhage and hemosiderin deposition) [21]. These abnormalities are primarily associated with monoclonal antibodies targeting β-amyloid, such as aducanumab, lecanemab and donanemab [22].

ARIA-E (Edema and Effusion)

ARIA-E manifests as parenchymal edema or sulcal effusion, detectable on T2-weighted or Fluid-Attenuated Inversion Recovery (FLAIR) MRI sequences. It is often asymptomatic and self-resolving, but in some cases, it may present with symptoms such as headache, confusion, or seizures. The incidence of ARIA-E is dose-dependent and higher in patients carrying the apolipoprotein E4 (APOE ε4) allele [22, 23].

ARIA-H (Hemorrhage and Hemosiderin Deposition)

ARIA-H includes microhemorrhages, superficial cortical siderosis, and macrohemorrhages, identifiable on T2*-weighted gradient recalled echo (GRE) sequences. While most cases are asymptomatic, approximately 5% can lead to severe outcomes, including hospitalization, permanent disability, or death [21, 23].

Clinical Implications

The recognition of ARIA is crucial for patient selection and monitoring during anti-amyloid β immunotherapy. In clinical practice, the development of ARIA may necessitate dose adjustments or discontinuation of therapy. Radiologists play a vital role in identifying ARIA on MRI scans, which informs clinical decisions and ensures patient safety [20].

Amyloid-Related Imaging Abnormalities (ARIA) in the TRAILBLAZER-ALZ 2 Clinical Trial

ARIA were observed in 36.8% of patients, with 24.0% developing ARIA-E and 31.4% ARIA-H; symptomatic ARIA-E occurred in 6.1% of cases. There were three treatment-related deaths in the donanemab group, all linked to severe ARIA events. Infusion-related reactions were also more common with donanemab (8.7%) than with placebo (0.5%) [13, 19]. Importantly, patients who are APOE ε4 homozygotes were found to be at higher risk for ARIA, necessitating careful screening and monitoring [17, 24].

In summary, donanemab shows robust clinical benefit in patients with early symptomatic AD and limited tau pathology. While its efficacy rivals or exceeds that of earlier anti-amyloid agents, the risk of ARIA and need for patient stratification remain key challenges in its broader clinical implementation.

Donanemab: International Regulatory Perspectives

Despite these findings, the European Medicines Agency (EMA) issued a negative opinion on the marketing authorization of donanemab, citing serious and potentially fatal side effects, such as brain swelling and hemorrhages, which occurred in 1.6% of patients, leading to three fatalities [17]. As a result, the EMA determined that the risks outweighed the benefits of the drug [25, 26].

In contrast, Eli Lilly, the manufacturer of donanemab, has obtained regulatory approval for the drug in several countries, including the United States (*U.S. Food and Drug Administration (FDA)* [07.2024]), Japan (*Japan's Ministry of Health, Labour and Welfare* [09.2024]), and China (*China's National Medical Products Administration (NMPA)* [12.2024]). Approval in these regions was based on the same clinical data submitted to the EMA [27]. In the United Kingdom, the Medicines and Healthcare products Regulatory Agency (MHRA) approved donanemab for use in adults with early symptomatic AD who have one or no copies of the APOE ε4 gene. However, the National Institute for Health and Care Excellence (NICE) did not recommend its use in the National Health Service (NHS), primarily due to concerns regarding cost-effectiveness and the drug's high annual treatment cost [28].

Alzheimer Europe - the umbrella organisation of national Alzheimer associations in 36 European countries - expressed regret over the EMA's negative opinion, emphasizing that this decision limits treatment options for Europeans with early-stage AD, especially in comparison to the availability of donanemab in other countries [29]. Donanemab is not currently approved in Poland. Patients interested in accessing the drug may consider the Named Patient Import Scheme, which allows for the importation of unapproved medications for individual use under specific conditions [30].

These approvals reflect the growing recognition of donanemab as a potential treatment option for early symptomatic AD. However, the varying recommendations and restrictions across different countries emphasize the complexities involved in the global adoption of new Alzheimer's therapies.

Conclusions

Donanemab has demonstrated clinically meaningful efficacy in patients with early symptomatic AD characterized by low to intermediate tau burden. Its therapeutic effect on disease progression, reflected in measurable slowing of cognitive and functional decline, positions it as a significant advancement within the anti-amyloid therapeutic class. However, the elevated incidence of ARIA, particularly among APOE ε4 carriers, and the need for precise biomarker-driven patient selection represent critical limitations to its broad clinical applicability [13, 17].

The differing regulatory decisions across countries - ranging from full approval in the United States to rejection in the European Union - highlight the ongoing debate about the risks and benefits of anti-amyloid therapies. These differences reflect not only scientific and clinical uncertainties but also the variable thresholds for evidence across health authorities.

Nonetheless, given the projected increase in AD prevalence due to demographic aging, there remains an urgent and unmet need for innovative and effective therapeutic approaches. Continued translational research, refinement of patient stratification methods, and development of next-generation disease-modifying agents are imperative to address the growing global burden of AD and to improve long-term patient outcomes.

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