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MALAYSIAN HEALTH TECHNOLOGY ASSESSMENT SECTION

LECANEMAB (BAN2401) FOR ALZHEIMER'S DISEASE

EXECUTIVE SUMMARY

Alzheimer's disease (AD) is a progressive, neurodegenerative disease that severely impairs a person's memory, thinking, learning, and organisational abilities, which eventually inhibits their capacity to perform basic everyday tasks. Since there were no available disease-modifying therapies at the time, medications to manage symptoms (cognitive and behavioural) were commonly utilised to increase independence and preserve function. Lecanemab also called BAN2401 is a humanised IgG1 monoclonal antibody that reduce the brain amyloid plaques and slowed clinical decline in early Alzheimer's disease. It has been demonstrated to be safe and well-tolerated at various doses in the completed phase 2b trial.

Keywords: lecanemab, BAN2401, Alzheimer's disease, Amyloid plaques

INTRODUCTION

Worldwide, around 55 million people suffer from dementia, with over 60% living in low- and middle-income countries.[1] This figure is predicted to rise to 78 million in 2030 and 139 million in 2050, as the proportion of older people in the population increases in almost every country.[1]

According to the Alzheimer's disease Foundation Malaysia 2016, it is predicted that currently there are about 50,000 people in Malaysia with this disease.[2] The estimated number of older people with this disease worldwide is expected to double for every 20 years.[2] As derived from the Malaysian National Health and Morbidity Survey 2018: Elderly Health, the overall prevalence of probable dementia was 8.5% (95% CI 6.97 to 10.22).[3] The prevalence was

found to be higher among females, those with no formal education and those in rural areas in Malaysia.[4]

In the Alzheimer's brain, abnormal levels of amyloid beta (Aβ) together will form plaques that collect between neurons and disrupt cell function. This toxic amyloid-beta (Aβ) aggregates (protofibrils) exists in various conformational states, including soluble monomers, soluble aggregates of increasing size (e.g., oligomers, protofibrils), and insoluble fibrils and plaque.[5] Soluble Aβ aggregates have been shown to be more toxic than monomers or insoluble fibrils, and reducing these soluble Aβ aggregates could represent an effective treatment approach in early stages of AD.[6] The clinical trials of lecanemab completed to date have yielded promising results in patients with early AD including reduced brain amyloid, slowed clinical decline and reduce the life-time probability for institutional care.[7, 8]

THE TECHNOLOGY

Lecanemab also known as BAN2401 is a humanised IgG1 monoclonal antibody that selectively binds to neutralise and eliminate soluble, toxic amyloid-beta (Aβ) aggregates (protofibrils) that contribute to the neurodegenerative process in Alzheimer's disease (AD).[9] It has at least a 1000-fold higher selectivity for protofibrils compared to monomers and 10-15 times better binding to protofibrils than to fibrils.[10] This potentially allows the immune system to clear the protein before it forms the toxic clumps that drive the death of nerve cells (neurons) in the brains of those with Alzheimer's.[11] Lecanemab is administered to the patients by intravenous infusion biweekly or monthly. It was licensed to Eisai, which in March 2014 signed a collaboration agreement with Biogen for joint development of this therapeutic antibody.[12] There are currently two published lecanemab trials on its safety, tolerability, and pharmacokinetics.

In March 2019, Eisai began a Phase 3 trial called Clarity AD, in 250 sites across the world. It is ongoing and completed enrollment in March 2021 with 1,795 patients with early symptomatic AD, who receive 10 mg/kg drug or placebo every two weeks for 18 months, followed by a two-year open-label extension. The primary outcome in the core study is change in Clinical Dementia Rating (CDR) at 18 months, with secondary outcomes of brain amyloid, AD Composite Score (ADCOMS), and Alzheimer's Disease Assessment Scale-cognitive subscale (ADAS-Cog14).[13] In the extension phase, primary outcomes will be change in Clinical Dementia Rating-Sum-of-Boxes (CDR-SB) as well as safety. The trial is set to run until 2024.

In February 2020, the Alzheimer's Therapeutic Research Institute announced that the Alzheimer's Clinical Trial Consortium (ACTC) would conduct a large Phase 3 study of

BAN2401, called AHEAD 3-45 and co-funded by the NIH and Eisai. AHEAD 3-45 started in July 2020 as a four-year trial that comprises two sub-studies in a combined 1,400 people who are cognitively normal but have elevated brain amyloid.

The Food and Drug Administration (FDA) had granted its breakthrough therapy designation to lecanemab in mid-2021. In December 2021, fast track designation was granted after the biologics license application for accelerated FDA approval for lecanemab to treat patients with early AD was filed on September 21, 2021, with a decision expected in the first half of 2022.[13]

PATIENT GROUP AND INDICATION

Lecanemab is indicated for the individuals with Alzheimer's disease in the early stage. It may have an effect on disease pathology by reducing the amyloid-beta (Aβ) and to slow down the progression of the disease.

Alzheimer's disease is the commonest type of dementia (60 - 70%), and patients often present initially with gradual episodic memory impairment.[14] Alzheimer disease occurs on a continuum, progressing from asymptomatic preclinical AD, to mild cognitive impairment due to AD, and to mild, moderate, and severe AD.[14] In early-onset of AD (less than 65 years old), patients may present with behavioral (frontal), visual (posterior cortical atrophy) or language (logopenic) variants with relatively well-preserved memory until the later stages of the disease.[15]

The pathogenesis of Alzheimer's disease is closely related to the processing of the amyloid precursor protein (APP), which results in the synthesis of various amyloid-beta (A) peptides.[16] They are found as insoluble aggregates in senile plaques, the histopathological hallmark of the disease.[16] In particular, the concentration of Aβ1–42 in the CSF undergoes a characteristic drop during disease progression, which is interpreted as the consequence of the ongoing parenchymal Aβ deposition in senile plaques.[16]

CURRENT PRACTICE

Management of AD patients is complex as the development of psychiatric and behavioural disturbances upon pharmacological treatment might overlap with the symptoms of cognitive decline.[17] The diagnosis of dementia should be based on detailed history and physical examination, and supported by cognitive, functional and behavioral evaluation.[17] There are no disease-modifying therapies accessible at this time but the medications aimed at

managing symptoms (cognitive and behavioral), improve independence and preserve function.[18]

According to the Malaysian Clinical Practice Guidelines on the Management of Dementia, to improve cognitive function in mild to moderate dementia, cognitive stimulation therapy and physical activity should be offered as a non-pharmacological intervention.[17]

Meanwhile, for the pharmacological treatment, the guidelines recommended several drugs that can help to manage the symptoms.[17] Donepezil should be offered in AD of all severity and rivastigmine and galantamine is an option in mild to moderate AD.[17] Memantine may be considered in moderate to severe AD as monotherapy or in combination with acetylcholinesterase inhibitors (AChEI).[17]

Besides that, an amyloid beta-directed monoclonal antibody called Aducanumab (brand name: Aduhelm) have been approved by FDA for the treatment of AD with mild cognitive impairment or mild dementia stage of disease.[16]

In the management of dementia, caregivers should be actively involved and supported. Advance care planning should be considered in the management of dementia once the diagnosis is established.[17]

EFFICACY AND SAFETY

Based on available evidence up to 30 June 2022, there are one phase 1 study done to test the safety, tolerability, and pharmacokinetics of lecanemab and one phase 2b study conducted in patients with early Alzheimer's disease.

The first randomised, multicenter, double-blind, phase 2b (CORE) clinical trial on lecanemab was conducted in 854 randomised subjects with early Alzheimer's disease.[19] This placebo-controlled Bayesian design clinical trial employing response adaptive randomisation across placebo and five lecanemab arms (2.5 mg/kg biweekly, 5 mg/kg monthly, 5 mg/kg biweekly, 10 mg/kg monthly, 10 mg/kg biweekly).[19] The primary endpoint was Bayesian analysis of 12-month clinical change on the Alzheimer's Disease Composite Score (ADCOMS) for the ED90 dose, and the secondary endpoints included 18-month Bayesian and frequentist analyses of brain amyloid reduction using positron emission tomography; clinical decline on ADCOMS, Clinical Dementia Rating-Sum-of-Boxes (CDR-SB), and Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog14); changes in CSF core biomarkers; and total hippocampal volume (HV) using volumetric magnetic resonance imaging.[19]

At 12 months, the 10-mg/kg biweekly ED90 dose showed a 64% probability to be better than placebo by 25% on ADCOMS, which missed the 80% probability threshold for the primary outcome.[19] At 18 months, 10-mg/kg biweekly lecanemab reduced brain amyloid (−0.306 SUVr units) while showing a drug-placebo difference in favor of active treatment by 27% and 30% on ADCOMS, 56% and 47% on ADAS-Cog14, and 33% and 26% on CDR-SB versus placebo according to Bayesian and frequentist analyses, respectively. CSF biomarkers were supportive of a treatment effect.[19] Marginally greater total hippocampal volume reduction (7.56% increased volume decline) was observed at 10mg/kg biweekly compared to placebo without nominal significance.[19] Whole brain volume and ventricular volume results reflect increased volume decline in the treatment groups compared to the placebo group. According to volumetric MRI results, lecanemab therapy at 10 mg/kg biweekly increased hippocampus volume loss by a little amount; however, the effects on whole brain volume loss and total ventricular volume increase were more significant.[19] Lecanemab was well-tolerated with 9.9% incidence of amyloid-related imaging abnormalities-edema/effusion at 10 mg/kg biweekly.[19]

An open label extension (OLE) study with 10 mg/kg IV biweekly lecanemab dosing was implemented after analysis of the phase 2b study, with an intervening gap period off-treatment ranging from 9-54 months (mean 24 months) in 121 subjects plasma samples.[20] This study aimed to evaluate longitudinal plasma Aβ 42:40 ratio that determine the presence or absence of amyloid plaques in the brain (C2N PrecivityAD assay) and the relationship to longitudinal amyloid PET in the phase 2b CORE study, gap period, and open label extension.[20]

Lecanemab produced dose dependent reductions in PET SUVr, with corresponding increases in plasma Aβ 42:40 ratios in Core and OLE. In the OLE, reductions in PET SUVr were dependent on OLE baseline SUVr; PET reductions in the OLE were inversely correlated with increases in plasma Aβ 42:40 ratios.[20] Changes in plasma Aβ 42:40 ratio were inversely correlated with changes in PET SUVr at the group and individual levels in the Core (group: r=-0.939, p=0.0056; individual: r=-0.49; p<0.0001) and OLE (group: r=- 0.907, p=0.0007; individual: r=-0.31 p=0.0012) studies.[20] Thus, Lecanemab dose-dependent reduction in brain amyloid PET SUVr correlate with increases in plasma Aβ 42:40 ratio.[20]

On the basis of lecanemab trial data and available literature, a disease simulation model was used to assess the long-term clinical outcomes of lecanemab for patients with early AD. Data from the phase 2b study and other published literature was used in the evidence-based

stimulation model to compare lecanemab plus Standard of Care (SoC) versus SoC alone in a cohorts of patients with mild cognitive impairment due to AD and mild AD dementia.

Lecanemab treatment was estimated to slow the rate of disease progression, resulting in an extended duration of mild cognitive impairment due to AD and mild AD dementia and shortened duration in moderate and severe AD dementia.[21] Compared with SoC alone, the proportions of patients on lecanemab plus SoC who progressed to mild AD, moderate AD, and severe AD were reduced by 7%, 13%, and 10%, respectively.[21] The mean time to mild, moderate, and severe AD dementia was longer for patients in the lecanemab plus SoC group than for patients in the SoC group by 2.51, 3.13, and 2.34 years, respectively.[21] On base-case analysis, lecanemab was associated with 0.73 incremental life years (LY) and 0.75 incremental quality-adjusted LYs (QALY), and the caregiver QALYs lost was reduced by 0.03 years.[21] Patients treated with lecanemab plus SoC were estimated to spend 11.6 more years in community care and 0.13 fewer years in institutional care compared with the SoC group, which translated into overall incremental survival of 1.03 years (8.40 years for lecanemab plus SoC versus 7.37 years for SoC).[21] The model also predicted a lower lifetime probability of admission to institutional care in lecanemab plus SoC versus SoC group (25% versus 31%).[21] A subgroup analysis by age and disease severity at baseline also revealed a potentially greater impact on disease progression with earlier initiation of treatment with lecanemab. The incremental mean times for transition to mild and moderate AD dementia were 2.53 and 3.34 years, respectively, when treating MCI due to AD in a subgroup analysis compared to SoC.[21]

In terms of safety, a multicenter Phase 1 trial had been conducted to test the safety, tolerability, and pharmacokinetics of single (SAD) and multiple ascending intravenous doses (MAD) of lecanemab in 80 people with mild to moderate AD.[22] The pharmacokinetics in serum, cerebrospinal fluid (CSF) and changes in Aβ levels were also assessed. In the SAD cohorts, serum concentrations of BAN2401 were measured pre dose, at 0, 0.5, 1, 2, 4, 8, and 24 h post dose, and at 10, 21-, 28-, 90- and 180-days post dose.[22] A multiple ascending dose part investigated doses of 0.3, 1, and 3 mg/kg administered every four weeks with a total of four doses over four months and a dose of 10 mg/kg biweekly, with a total of seven doses over four months.[22]

Lecanemab was well-tolerated at all doses tested, up to 10mg/kg every two weeks for four months, with amyloid-related imaging abnormalities (ARIA, E for edema, H for hemorrhage) occurring at the same rate in both placebo and BAN2401. Treatment-related adverse events (TEAEs) were all categorised as mild or severe.[22] The most frequently observed TEAEs with a single dose of lecanemab were dizziness (8.3 vs. 8.3 % on placebo), fatigue (5.6 vs 8.3 %

on placebo), and sinusitis (5.6 vs 0 % on placebo).[22] Two subjects developed new asymptomatic ARIA-H in the 0.3mg/kg and 1mg/kg SAD cohort but remained asymptomatic, clinically stable throughout the study and the ARIA-H had completely resolved by day 180.[22]

The most frequently observed TEAEs in subjects treated with multiple doses of lecanemab were upper respiratory tract infection (16.7 vs. 12.5 % on placebo), headache (12.5 vs. 25 % on placebo), and orthostatic hypotension (12.5 vs. 0 % on placebo).[22] In the 1 mg/kg MAD cohort, new asymptomatic ARIA-Hs emerged during the study in the two subjects on placebo and in one subject on active treatment.[22] The subject on lecanemab (1 mg/kg) had two new ARIA-Hs identified on MRI after three doses. All three of these subjects remained clinically stable throughout the study.[22] In overall, asymptomatic ARIA-H occurred in 3/60 (5 %) subjects treated with BAN2401, which is a lower incidence than that observed in subjects treated with placebo (2/20, 10 %). No symptomatic ARIA-E or ARIA-H seen with either SAD or MAD cohorts.[22]

Only a slight increase of plasma Aβ was observed and there were no measurable effects of BAN2401 on CSF biomarkers.[22] Results of the non-compartmentalized PK analyses indicate that BAN2401 is characterized by linear PK, with dose-proportional exposure and first order elimination.[22] The mean serum half-life of BAN2401 was approximately seven days, which was reliably determined when given at doses of 10 mg/kg or higher.[22] The 10 mg/kg biweekly dose achieved minimum steady state levels of approximately 40 µg/mL after three doses, with an accumulation factor of approximately 1.4.[22] Over a 14-day interval at steady state with multiple dosing at 10 mg/kg biweekly, the CSF; serum ratio increased to 0.29 %, which may suggest a longer half-life in CSF vs. plasma.[22]

In the phase 2b clinical trials, lecanemab was generally well-tolerated, with the key adverse event being ARIA-E with an incidence rate of less than 10% at the two highest doses for the overall population and 14.3% for ApoE4-positive subjects.[19] Apart from ARIA-E and infusion reactions, the incidence rates of adverse events, serious adverse events, and TEAEs were consistent with those expected for this population and similar across placebo and lecanemab treatment groups.[19] ARIA-H (new cerebral microhaemorrhages, cerebral macrohaemorrhages, and superficial siderosis) regardless of the presence of ARIA-E were observed in 13 (5.3%) subjects in the placebo group (N = 245) compared to 65 (10.7%) subjects in the lecanemab groups (N = 609).[19] Although there was a higher incidence of ARIA-H on lecanemab than placebo, there were no dose-related trends in the incidence of ARIA-H on lecanemab.[19]

ESTIMATED COST

According to the early economic assessment by Institute for Clinical and Economic Review (ICER), it was estimated that the potential economic value of lecanemab with standard of care (SoC) over a broad range of willingness-to-pay thresholds from $50,000 (220,275 MYR; 1 USD= 4.41 MYR) to $200,000 (881,100 MYR; 1 USD= 4.41 MYR) per QALY gained as recommended by the ICER.[8] Lecanemab plus SoC was predicted to result in a gain of 0.61 QALYs and a decrease in total non-treatment costs of $8,707 (38,358.69 MYR; 1 USD= 4.41 MYR) per person from the healthcare payer perspective (Societal perspective: 0.64 QALYs gain and $11,214 (49,403.28 MYR; 1 USD= 4.41 MYR) decrease) compared to the SoC for patients with early AD who have confirmed presence of amyloid pathology.[8]

In the scenario analyses, lecanemab had a greater impact on total non-treatment costs and QALYs when initiated at earlier ages in patients with mild cognitive impairment due to AD, and in patients earlier in their tau pathology (lower quintiles).[8] The estimated gain in QALYs was 0.67–0.88 in the mild cognitive impairment due to AD subset vs. 0.32 in the mild AD dementia subset.[8]

The potential annual value-based price (VBP) of lecanemab was estimated at $9,249 (40,746.47 MYR; 1 USD= 4.41 MYR) to $35,605 (156,857.83 MYR; 1 USD= 4.41 MYR) (Societal perspective: $10,400 (45,817.20 MYR; 1 USD= 4.41 MYR) to $38,053 (167,642.49 MYR; 1 USD= 4.41 MYR)) based on this early economic assessment.[8]

POTENTIAL IMPACT

Alzheimer's disease is a progressive and neurodegenerative disease that affects elderly and cause memory, thinking and behavior problem. It may also cause a substantial burden to the caregivers and to the healthcare systems. Currently, no disease modifying therapy available yet except for aducanumab, a monoclonal antibody directed at beta-amyloid plaques that have been approved since 2021 with several drawback issues.[23] Most patients with AD and related dementia receive informal care from family and friends. Moreover, the pharmacological therapy offered to AD patients is merely advised to help with symptom management.[17]

Lecanemab offers alternative to the patients with early AD and mild cognitive impairment due to AD. It may have a potential to reduce beta-amyloid plaques and improve cognitive behaviour.[19] The estimated lifetime risk of disease progression to mild, moderate and severe AD dementia from baseline could potentially be reduced in lecanemab plus SoC,

respectively, compared to SoC alone.[21] Besides of estimated to potentially slow the rate of disease progression, it could potentially maintain treated patients for a longer duration in earlier stages of AD as early AD which can lead to an increase in disease-free years and sizable cost-savings for care, providing significant benefits to patients, their caregivers and society.[21]

However, the ongoing phase 3 clinical trials (Clarity AD and AHEAD 3-45) data and longer-term clinical trial follow-up is needed as further evidence to ascertain the clinical efficacy, safety and cost effectiveness of lecanemab.

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