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Policy Number: C21304-A

ADUHELM (aducanumab-avwa) MNR

PRODUCTS AFFECTED

ADUHELM (aducanumab-avwa)

COVERAGE POLICY

Coverage for services, procedures, medical devices and drugs are dependent upon benefit eligibility as outlined in the member's specific benefit plan. This Coverage Guideline must be read in its entirety to determine coverage eligibility, if any.

This Coverage Guideline provides information related to coverage determinations only and does not imply that a service or treatment is clinically appropriate or inappropriate. The provider and the member are responsible for all decisions regarding the appropriateness of care. Providers should provide Molina Healthcare complete medical rationale when requesting any exceptions to these guidelines.

Documentation Requirements:

Molina Healthcare reserves the right to require that additional documentation be made available as part of its coverage determination; quality improvement; and fraud; waste and abuse prevention processes. Documentation required may include, but is not limited to, patient records, test results and credentials of the provider ordering or performing a drug or service. Molina Healthcare may deny reimbursement or take additional appropriate action if the documentation provided does not support the initial determination that the drugs or services were medically necessary, not investigational, or experimental, and otherwise within the scope of benefits afforded to the member, and/or the documentation demonstrates a pattern of billing or other practice that is inappropriate or excessive.

DIAGNOSIS:

Alzheimer's disease

REQUIRED MEDICAL INFORMATION:

This clinical policy is consistent with standards of medical practice current at the time that this clinical policy was approved. If a drug within this policy receives an updated FDA label within the last 180 days, medical necessity for the member will be reviewed using the updated FDA label information along with state and federal requirements, benefit being administered and formulary preferencing. Coverage will be determined on a case-by-case basis until the criteria can be updated through Molina Healthcare, Inc. clinical governance. Additional information may be required on a case-by-case basis to allow for adequate review. When the requested drug product for coverage is dosed by weight, body surface area or other member specific measurement, this data element is required as part of the medical necessity review.

A. ALZHEIMER'S DISEASE:

1. Documentation of a diagnosis of mild cognitive impairment (MCI) due to Alzheimer's disease based on NIA-AA diagnostic criteria [A concern regarding cognition reported by the patient or informant or observed by the clinician, Objective evidence of impairment in one or more cognitive domains that is not explained by age or education, Preservation of independence in functional

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- abilities, Not demented] OR diagnosis of Alzheimer disease dementia based on NIA-AA criteria for probable AD dementia [See Appendix]
AND
2. Documentation of Clinical Dementia Rating Scale (CDR) Global Score of 0.5 OR Mini-Mental State Examination (MMSE) score between 24 and 30 OR Montreal Cognitive Assessment (MoCA) score between 24 and 30 [DOCUMENTATION REQUIRED]
AND
 3. Documentation of confirmation of underlying amyloid beta pathology [through PET scan OR lumbar puncture (LP) for CSF (cerebrospinal fluid)]
NOTE: Biogen has a program that will assist with the cost of the CSF testing
<https://www.amyloidbetaconfirmed.com/>
AND
 4. Documentation member is currently in CMS approved randomized controlled trial may be extended to a prospective longitudinal study
AND
 5. Documentation within medical record member does NOT have any medical or neurological condition (other than Alzheimer's Disease) that might be a contributing cause of the member's cognitive impairment

CONTINUATION OF THERAPY:

A. ALZHEIMER'S DISEASE:

1. Adherence to therapy at least 85% of the time as verified by the prescriber or member medication fill history OR adherence less than 85% of the time due to the need for surgery or treatment of an infection, causing temporary discontinuation
AND
2. Prescriber attests to or clinical reviewer has found no evidence of intolerable adverse effects or drug toxicity such as amyloid-related imaging abnormalities (ARIA)
AND
3. Documentation of positive clinical response, as demonstrated by improvement OR stabilization in baseline cognitive scoring using the same testing method as provided during initial authorization request [DOCUMENTATION REQUIRED]
AND
4. Documentation of new MRI prior to the 5th, 7th (first dose of 10mg/kg), 9th and 12th infusions (sixth dose of 10mg/kg), if applicable, showing radiographic stability.
Note: If 10 or more new incident microhemorrhages or > 2 focal areas of superficial siderosis (radiographic severe ARIA-H) is observed, treatment may be continued with caution only after a clinical evaluation and a follow-up MRI demonstrates radiographic stabilization (i.e., no increase in size or number of ARIA-H)
AND
5. Documentation member is able to tolerate the 10mg/kg dosing

DURATION OF APPROVAL:

Initial authorization: 3 months, Continuation of Therapy: 6 months

PRESCRIBER REQUIREMENTS:

Prescribed by or in consultation with a Neurologist, Geriatrician, Geropsychiatric specialist OR Alzheimer's Disease Specialist [If prescribed in consultation, consultation notes must be submitted with initial request and reauthorization requests]

AGE RESTRICTIONS:

50 years of age and older

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QUANTITY:

After an initial titration, the recommended dosage of Aduhelm is 10 mg/kg.

ADUHELM is administered as an intravenous (IV) infusion over approximately one hour every four weeks and at least 21 days apart

Dosing Schedule:

Infusion 1 and 2	1 mg/kg
Infusion 3 and 4	3 mg/kg
Infusion 5 and 6	6 mg/kg
Infusion 7 and beyond	10 mg/kg

Maximum Quantity Limits – 10mg/kg at least 21 days apart

PLACE OF ADMINISTRATION:

The recommendation is that infused medications in this policy will be for pharmacy or medical benefit coverage administered in a place of service that is a non-inpatient hospital facility-based location.

DRUG INFORMATION

ROUTE OF ADMINISTRATION:

Intravenous

DRUG CLASS:

Alzheimer's Treatment - Anti-Amyloid Antibodies

FDA-APPROVED USES:

Indicated for the treatment of Alzheimer's disease.

Treatment with ADUHELM should be initiated in patients with mild cognitive impairment or mild dementia stage of disease, the population in which treatment was initiated in clinical trials. There are no safety or effectiveness data on initiating treatment at earlier or later stages of the disease than were studied. This indication is approved under accelerated approval based on reduction in amyloid beta plaques observed in patients treated with ADUHELM. Continued approval for this indication may be contingent upon verification of clinical benefit in confirmatory trial(s).

COMPENDIAL APPROVED OFF-LABELED USES:

None

APPENDIX

APPENDIX:

Criteria for all-cause dementia: Core clinical criteria

The diagnosis of dementia is intended to encompass the spectrum of severity, ranging from the mildest to the most severe stages of dementia. The methodology for staging of dementia severity was beyond the charge of the workgroup. Dementia is diagnosed when there are cognitive or behavioral (neuropsychiatric) symptoms that:

1. Interfere with the ability to function at work or at usual activities; and
2. Represent a decline from previous levels of functioning and performing; and
3. Are not explained by delirium or major psychiatric disorder.
4. Cognitive impairment is detected and diagnosed through a combination of (1) history-taking from the patient and a knowledgeable informant and (2) an objective cognitive assessment, either a "bedside" mental status examination or neuropsychological testing. Neuropsychological testing should be performed when the routine history and bedside mental status examination cannot

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provide a confident diagnosis.

5. The cognitive or behavioral impairment involves a minimum of two of the following domains:
 - a. Impaired ability to acquire and remember new information—symptoms include repetitive questions or conversations, misplacing personal belongings, forgetting events or appointments, getting lost on a familiar route.
 - b. Impaired reasoning and handling of complex tasks, poor judgment— symptoms include poor understanding of safety risks, inability to manage finances, poor decision-making ability, inability to plan complex or sequential activities.
 - c. Impaired visuospatial abilities—symptoms include inability to recognize faces or common objects or to find objects in direct view despite good acuity, inability to operate simple implements, or orient clothing to the body.
 - d. Impaired language functions (speaking, reading, writing)—symptoms include difficulty thinking of common words while speaking, hesitations; speech, spelling, and writing errors.
 - e. Changes in personality, behavior, or compoirtment—symptoms include uncharacteristic mood fluctuations such as agitation, impaired motivation, initiative, apathy, loss of drive, social withdrawal, decreased interest in previous activities, loss of empathy, compulsive or obsessive behaviors, socially unacceptable behaviors

Probable AD dementia is diagnosed when the patient

1. Meets criteria for dementia described above, and in addition, has the following characteristics:
 - A. Insidious onset. Symptoms have a gradual onset over months to years, not sudden over hours or days.
 - B. Clear-cut history of worsening of cognition by report or observation; and
 - C. The initial and most prominent cognitive deficits are evident on history and examination in one of the following categories.
 - a. Amnesic presentation: It is the most common syndromic presentation of AD dementia. The deficits should include impairment in learning and recall of recently learned information. There should also be evidence of cognitive dysfunction in at least one other cognitive domain, as defined earlier in the text.
 - b. Non-amnesic presentations:
 - Language presentation: The most prominent deficits are in word-finding, but deficits in other cognitive domains should be present.
 - Visuospatial presentation: The most prominent deficits are in spatial cognition, including object agnosia, impaired face recognition, simultanagnosia, and alexia. Deficits in other cognitive domains should be present.
 - Executive dysfunction: The most prominent deficits are impaired reasoning, judgment, and problem solving. Deficits in other cognitive domains should be present.
 - D. The diagnosis of probable AD dementia **should not** be applied when there is evidence of (a) substantial concomitant cerebrovascular disease, defined by a history of a stroke temporally related to the onset or worsening of cognitive impairment; or the presence of multiple or extensive infarcts or severe white matter hyperintensity burden; or (b) core features of Dementia with Lewy bodies other than dementia itself; or (c) prominent features of behavioral variant frontotemporal dementia; or (d) prominent features of semantic variant primary progressive aphasia or nonfluent/agrammatic variant primary progressive aphasia; or (e) evidence for another concurrent, active neurological disease, or a non-neurological medical comorbidity or use of medication that could have a substantial effect on cognition.

Alzheimer's Disease Assessment Scale-Cognitive (ADAS-Cog 13) A 13-item test used to measure the amount or degree of cognitive impairment based on the domains of attention, concentration, memory, language, and praxis. It is currently the preferred test for measuring the effectiveness of anti-dementia treatments, with current studies taking place in the MCI population. Because the ADAS-Cog is given verbally with a heavy emphasis on spoken language, the patient must be fluent in the language in which the test is given. The ADAS-Cog is translated into 81 languages

Clinical Dementia Rating Scale–Sum of Boxes (CDR–SB) Uses a 5-point scale that identifies 6 cognitive and functional performance domains affected by AD and other dementias. Information is acquired through patient interviews and reliable reporting from a caregiver or member of the patient's family. This test has been shown to be effective in correctly staging AD and MCI. This test is available in more than 40 languages

Mini-Cognitive Assessment Instrument (Mini-Cog) A 3-minute assessment used as part of a complete diagnostic workup designed to help detect cognitive impairment in older adults. In the Mini-Cog, the patient is asked to recall 3 items and then draw a clock. The test is accurate and accessible to many populations, including non-English speakers

Mini-Mental State Examination (MMSE) A test that measures cognition and has been used to stage and show changes over time. This assessment is available in more than 70 languages and dialects

Self-Administered Gerocognitive Examination (SAGE) measures cognitive function in the domains of orientation, language, memory, executive function, calculations, abstraction and visuospatial abilities. Takes roughly 10-15 minutes to complete

St Louis University Mental Status (SLUMS) measures cognitive function in the domains of orientation, attention (digit span), numeric calculation, immediate and delayed verbal recall, verbal fluency (animal naming), executive functions, figure recognition/size differentiation and immediate recall of contextual verbal information- takes 7 minutes to complete

Montreal Cognitive Assessment (MoCA) brief screening measure consisting of a 30-point test administered in 10 minutes, focusing on memory, visuospatial, executive and language function and orientation to time and place

BACKGROUND AND OTHER CONSIDERATIONS

BACKGROUND:

An estimated 24 million people worldwide have dementia, the majority of whom are thought to have Alzheimer's disease. Thus, Alzheimer's disease represents a major public health concern and has been identified as a research priority. Although there are licensed treatments that can alleviate symptoms of Alzheimer's disease, there is a pressing need to improve our understanding of pathogenesis to enable development of disease-modifying treatments. Methods for improving diagnosis are also moving forward, but a better consensus is needed for development of a panel of biological and neuroimaging biomarkers that support clinical diagnosis. There is now strong evidence of potential risk and protective factors for Alzheimer's disease, dementia, and cognitive decline, but further work is needed to understand these better and to establish whether interventions can substantially lower these risks. In this Seminar, we provide an overview of recent evidence regarding the epidemiology, pathogenesis, diagnosis, and treatment of Alzheimer's disease, and discuss potential ways to reduce the risk of developing the disease.

On June 7, 2021, the FDA approved Biogen's Aduhelm (aducanumab) for the treatment of Alzheimer's disease (AD). Aduhelm is the first new drug approved for AD in 18 years and is the first drug specifically

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approved to slow the progression of AD. Aduhelm is a monoclonal antibody that targets the buildup of amyloid plaque in the brain and is administered as a once-monthly intravenous infusion.

The approved indication “for the treatment of Alzheimer’s disease” is the broadest label the FDA could have provided. The clinical trials for the drug were conducted on much more specific patient types, those with mild cognitive impairment (MCI) or mild Alzheimer’s dementia. All patients in the trials also received a PET (positron emissions tomography) scan to confirm elevated brain amyloid levels.

The FDA’s approval of Aduhelm was based on results from three Phase 3 trials, EMERGE and ENGAGE, and a Phase 1b trial, PRIME. There has been significant controversy over the trial data, as EMERGE and ENGAGE have conflicting clinical results. In the EMERGE trial, patients treated with high-dose aducanumab showed less cognitive decline than patients receiving placebo.

The efficacy of ADUHELM was evaluated in two double-blind, randomized, placebo-controlled, parallel group studies (Study 1, NCT 02484547 and Study 2, NCT 02477800) in patients with Alzheimer’s disease (patients with confirmed presence of amyloid pathology and mild cognitive impairment or mild dementia stage of disease, consistent with Stage 3 and Stage 4 Alzheimer’s disease, stratified to include 80% Stage 3 patients and 20% Stage 4 patients). The effects of ADUHELM were also supported by a double-blind, randomized, placebo-controlled, dose ranging study (Study 3, NCT 01677572) in patients with Alzheimer’s disease (patients with confirmed presence of amyloid pathology and prodromal or mild dementia stage of disease, consistent with Stage 3 and Stage 4 Alzheimer’s disease, with an enrolled distribution of 43% Stage 3 patients and 57% Stage 4 patients), followed by an optional, dose-blind, long-term extension period.

In Studies 1 and 2, patients were randomized to receive ADUHELM low dose (3 or 6 mg/kg for ApoE ε4 carriers and noncarriers, respectively), ADUHELM high dose (10 mg/kg), or placebo every 4 weeks for 18 months, followed by an optional, dose-blind, long-term extension period. Both studies included an initial titration period of up to 6 months to the maximum target dose. At the beginning of the study, ApoE ε4 carriers were initially titrated up to a maximum of 6 mg/kg in the high dose group, which was later adjusted to 10 mg/kg.

In Studies 1 and 2, patients were enrolled with a Clinical Dementia Rating (CDR) global score of 0.5, a Repeatable Battery for Assessment of Neuropsychological Status (RBANS) delayed memory index score ≤ 85, and a Mini-Mental State Examination (MMSE) score of 24-30. In Study 3, patients were enrolled with a global CDR score of 0.5 or 1.0 and an MMSE score of 20-30. Patients were enrolled with or without concomitant approved therapies (cholinesterase inhibitors and the N-methyl-D-aspartate antagonist memantine) for Alzheimer’s disease. Studies 1 and 2 were terminated prior to their planned completion. Study endpoints were analyzed based on the prespecified statistical analysis plan

Study 1

In Study 1, 1638 patients were randomized 1:1:1 to receive ADUHELM low dose, ADUHELM high dose, or placebo. At baseline, the mean age of patients was 71 years, with a range of 50 to 85 years. A subgroup of 488 patients were enrolled in the amyloid PET sub study; of these, 302 were evaluated at week 78.

The primary efficacy endpoint was the change from baseline on the CDR-Sum of Boxes (CDRSB) at Week 78. In Study 1, treatment with ADUHELM high dose demonstrated reduced clinical decline, as evidenced by a statistically significant treatment effect on change from baseline in CDR-SB compared to placebo (-0.39 [-22%], p = 0.0120)

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Secondary efficacy endpoints included the change from baseline in MMSE score at Week 78, the change from baseline in the Alzheimer's Disease Assessment Scale-Cognitive Subscale (13 items) (ADAS-Cog 13) at Week 78, and the change from baseline in the Alzheimer's Disease Cooperative Study – Activities of Daily Living Inventory (Mild Cognitive Impairment version) (ADCS-ADL-MCI) score at Week 78. In Study 1, statistically significant differences from placebo were observed in the ADUHELM high dose group on all secondary efficacy endpoints evaluated. The estimate of the treatment effect favored ADUHELM across most prespecified subgroups of interest for the secondary efficacy endpoints. The Neuropsychiatric Inventory-10item (NPI-10) was the only tertiary endpoint that assessed efficacy

Table 4: Biomarker Results of ADUHELM in Study 1

Biomarker Endpoint at Week 78¹	ADUHELM High dose	Placebo
Amyloid Beta PET Composite SUVR	N=170	N=159
Mean baseline	1.383	1.375
Change from baseline Difference from placebo	-0.264 -0.278, p<0.0001	0.014
Amyloid Beta PET Centiloid	N=170	N=159
Mean baseline	85.3	83.5
Change from baseline (%) Difference from placebo	-60.8 (-71%) -64.2, p<0.0001	3.4
CSF p-Tau (pg/mL)	N=17	N=28
Mean baseline	100.11	72.55
Change from baseline Difference from placebo	-22.93 -22.44, p=0.0005	-0.49
CSF t-Tau (pg/mL)	N=17	N=28
Mean baseline	686.65	484.00
Change from baseline Difference from placebo	-112.44 -112.05, p=0.0088	-0.39

¹P-values were not statistically controlled for multiple comparisons.

Table 5: Clinical Results of ADUHELM in Study 1

Clinical Endpoint at Week 78	ADUHELM High dose (N=547)	Placebo (N=548)
CDR-SB		
Mean baseline	2.51	2.47
Change from baseline Difference from placebo (%)	1.35 -0.39 (-22%) p=0.0120	1.74
MMSE		
Mean baseline	26.3	26.4
Change from baseline Difference from placebo (%)	-2.7 0.6 (-18%) p=0.0493	-3.3
ADAS-Cog 13		
Mean baseline	22.246	21.867
Change from baseline Difference from placebo (%)	3.763 -1.400 (-27%) p=0.0097	5.162
ADCS-ADL-MCI		
Mean baseline	42.5	42.6
Change from baseline Difference from placebo (%)	-2.5 1.7 (-40%) p=0.0006	-4.3
NPI-10¹		
Mean baseline	4.5	4.3
Change from baseline Difference from placebo (%)	0.2 -1.3 (-87%) p=0.0215	1.5

¹P-value was not statistically controlled for multiple comparisons.

Study 2

In Study 2, 1647 patients were randomized 1:1:1 to receive ADUHELM low dose, ADUHELM high dose, or placebo. At baseline, the mean age of patients was 71 years, with a range of 50 to 85 years. A subgroup of 585 patients were enrolled in the amyloid PET subgroup; of these, 374 were evaluated at week 78.

No statistically significant differences were observed between the ADUHELM-treated and placebo-treated patients on the primary efficacy endpoint, the change from baseline in CDR-SB score at 78 weeks

Table 6: Biomarker Results of ADUHELM in Study 2

Biomarker Endpoint at Week 78¹	ADUHELM High dose	Placebo
Amyloid Beta PET Composite SUVR	N=183	N=204
Mean baseline	1.407	1.376
Change from baseline Difference from placebo	-0.235 -0.232, p<0.0001	-0.003
Amyloid Beta PET Centiloid	N=183	N=204
Mean baseline	90.8	83.8
Change from baseline (%) Difference from placebo	-54.0 (-59%) -53.5, p<0.0001	-0.5
CSF p-Tau (pg/mL)	N=18	N=15
Mean baseline	121.81	94.53
Change from baseline Difference from placebo	-13.19 -10.95, p=0.3019	-2.24
CSF t-Tau (pg/mL)	N=16	N=14
Mean baseline	618.50	592.57
Change from baseline Difference from placebo	-102.51 -69.25, p=0.3098	-33.26

¹P-values were not statistically controlled for multiple comparisons.

The results of the 3 trials were previously debated in a November 2020 meeting of the FDA's Peripheral and Central Nervous System Drugs Advisory Committee, in which, of the 11 members, only 1 member voted for aducanumab (8 members voted no and 2 were uncertain).

In an op-ed published on March 30, 2021, in The Journal of the American Medical Association (JAMA), 3 members of the FDA advisory committee (Ad Comm) who reviewed aducanumab, Biogen's investigational Alzheimer's drug, reasserted their arguments for why the FDA should not approve it. <https://jamanetwork.com/journals/jama/article-abstract/2778191>

CONTRAINDICATIONS/EXCLUSIONS/DISCONTINUATION:

All other uses of ADUHELM (aducanumab-avwa) are considered experimental/investigational and therefore, will follow Molina's Off- Label policy. There are no FDA labeled contraindications to ADUHELM™ (aducanumab-avwa).

OTHER SPECIAL CONSIDERATIONS:

Dilution in 100 mL of 0.9% Sodium Chloride Injection, USP, is required prior to administration. Administer as an intravenous infusion over approximately one hour via a 0.2 or 0.22 micron in-line filter.

CODING/BILLING INFORMATION

Note: 1) This list of codes may not be all-inclusive. 2) Deleted codes and codes which are not effective at the time the service is rendered may not be eligible for reimbursement

HCPCS CODE	DESCRIPTION
J0172	Injection, aducanumab-avwa, 2 mg

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AVAILABLE DOSAGE FORMS:

Aduhelm SOLN 170 mg/1.7 mL (100 mg/mL) single-dose vial
 Aduhelm SOLN 300 mg/3 mL (100 mg/mL) single-dose vial

Diagnosis Codes for ADUHELM™ (aducanumab-avwa):

G30.0: Alzheimer’s disease with early onset
 G30.1: Alzheimer’s disease with late onset
 G30.8: Other Alzheimer’s disease
 G31.84: Mild cognitive impairment, so stated

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5. MMSE Mini-Mental State Examination. PAR website. <https://www.parinc.com/Products/Pkey/237>.
6. 221AD302 Phase 3 Study of Aducanumab (BIIB037) in Early Alzheimer's Disease (EMERGE). Clinical trials.gov
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SUMMARY OF REVIEW/REVISIONS	DATE
REVISION- Notable revisions: Required Medical Information Continuation of Therapy	Q4 2023
REVISION- Notable revisions: Required Medical Information Continuation of Therapy References	Q2 2023
REVISION- Notable revisions: Required Medical Information Coding/Billing Information	Q2 2022
Q2 2022 Established tracking in new format	Historical changes on file