

PHARMACY / MEDICAL POLICY – 5.01.626

Amyloid Antibodies for the Treatment of Alzheimer’s Disease

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Replaces: N/A

RELATED MEDICAL POLICIES: N/A

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Introduction

Alzheimer’s disease (AD) is a progressive disease that leads to loss in memory, language, and thinking and mostly affects adults over 65 years of age. The loss of memory is the most common initial symptom but other symptoms in people with mild AD can also include changes in behavior or mood. One common finding in people with AD is the development of plaques (amyloid beta plaques) between brain cells and tangles of twisted fiber (tau protein) within the brain cells. These plaques and tangles in the brain of people with AD is often more extensive than people without AD and research regarding the role of these plaques and tangles is ongoing.

Note: The Introduction section is for your general knowledge and is not to be taken as policy coverage criteria. The rest of the policy uses specific words and concepts familiar to medical professionals. It is intended for providers. A provider can be a person, such as a doctor, nurse, psychologist, or dentist. A provider also can be a place where medical care is given, like a hospital, clinic, or lab. This policy informs them about when a service may be covered.

Policy Coverage Criteria

Drug	Medical Necessity
<p>Leqembi (lecanemab-irmb)</p>	<p>Leqembi (lecanemab-irmb) may be considered medically necessary for the treatment of adults with Alzheimer’s disease when ALL the following are met:</p> <ul style="list-style-type: none"> • The individual has been diagnosed with Alzheimer’s disease <p>AND</p> <ul style="list-style-type: none"> • The individual has documented cognitive test results that indicate mild cognitive impairment OR mild Alzheimer’s disease dementia <p>AND</p> <ul style="list-style-type: none"> • The individual has documented presence of beta-amyloid protein deposition as evidenced by ONE of the following: <ul style="list-style-type: none"> ○ Positive amyloid positron emission tomography (PET) scan ○ Cerebrospinal fluid (CSF) biomarker testing documents abnormalities suggestive of beta-amyloid accumulation in the brain <p>AND</p> <ul style="list-style-type: none"> • A baseline brain magnetic resonance imaging (MRI) has been completed within 12 months prior to initiating treatment <p>AND</p> <ul style="list-style-type: none"> • The individual does not have cognitive impairment due to other medical conditions (e.g., dementia with Lewy bodies, frontotemporal dementia, vascular dementia, vitamin B12 deficiency, encephalopathy, pseudodementia due to mood disorder, or untreated thyroid disease) <p>AND</p> <ul style="list-style-type: none"> • Leqembi (lecanemab-irmb) will not be used in combination with other monoclonal antibodies for the treatment of Alzheimer’s disease <p>AND</p> <ul style="list-style-type: none"> • Leqembi (lecanemab-irmb) is prescribed by or in consultation with a specialist in dementia such as a neurologist, geriatric psychiatrist, neuropsychiatrist, or geriatrician <p>AND</p> <ul style="list-style-type: none"> • The dose is limited to 10 mg/kg every 2 weeks



Drug	Investigational
Aduhelm (aducanumab-avwa)	The use of Aduhelm (aducanumab-avwa) is considered investigational for all indications including treatment of Alzheimer’s disease.
Leqembi (lecanemab-irmb)	All other uses of Leqembi (lecanemab-irmb) for conditions not outlined in this policy are considered investigational.

Length of Approval	
Approval	Criteria
Initial authorization	Leqembi (lecanemab-irmb) may be approved for up to 12 months
Re-authorization criteria	<p>Future re-authorization of Leqembi (lecanemab-irmb) may be approved up to 12 months in duration when ALL the following are met:</p> <ul style="list-style-type: none"> • The drug-specific policy coverage criteria are met • Medical records demonstrate that the individual continues to show a positive clinical response to therapy • A brain magnetic resonance imaging (MRI) is completed to check for radiographically observed amyloid related imaging abnormalities (ARIA) when warranted

Documentation Requirements
<p>The individual’s medical records submitted for review for all conditions should document that medical necessity criteria are met. The record should include the following:</p> <ul style="list-style-type: none"> • Office visit notes that contain the diagnosis, relevant history, physical evaluation, and medication history

Coding

Code	Description
HCPCS	
J0172	Injection, aducanumab-avwa (Aduhelm), 2 mg
J0174	Injection, lecanemab-irmb (Leqembi), 1 mg



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Evidence Review

Description

Alzheimer's disease is a neurodegenerative disorder leading to progressive, irreversible destruction of neurons and loss of cognitive function and memory. Over time, individuals progress to severe dementia, loss of independence, and death. Extracellular deposits of amyloid beta (A- β), referred to as amyloid plaques are considered a hallmark of the disease. Beta-amyloid monomers lead to formation of beta oligomers and fibrils and are deposited as plaques and then interact with tau fibrils, leading to formation of neuro-fibrillary tangles. These pathophysiological changes and clinical manifestations of Alzheimer's disease are progressive and occur along a continuum, and accumulation of A- β may begin 20 years or more before symptoms arise. Aducanumab is a human IgG1 anti-A- β antibody targeting amyloid aggregates. The drug is administered by intravenous infusion every 4 weeks. Binding of antibody is intended to lead to clearance of amyloid from the brain. On June 7, 2021, the US Food and Drug Administration (FDA) approved Aduhelm (aducanumab) for the treatment of Alzheimer's disease. It was approved under accelerated approval based on reduction in A- β plaques observed in individuals treated with aducanumab. Leqembi (lecanemab-irmb) is an anti-amyloid beta protofibril antibody that is administered once every 2 weeks. On January 6, 2023, the FDA approved Leqembi for the treatment of Alzheimer's disease. Continued approval of Aduhelm and Leqembi may be contingent upon verification of clinical benefit in confirmatory trial.

Background

Alzheimer's Disease

Alzheimer's disease is a fatal neurodegenerative disease that causes progressive loss in memory, language, and thinking, with the eventual loss of ability to perform social and functional activities in daily life. Survival after a diagnosis of dementia due to Alzheimer's disease generally ranges between 4 and 8 years; however, life expectancy can be influenced by other factors, such as comorbid medical conditions. It is estimated that 6.2 million Americans aged 65 and older are



currently living with Alzheimer's disease dementia, and the number is projected to reach over 12 million by 2050.

Pathophysiology

The pathologic hallmarks of Alzheimer's disease are extracellular deposits of beta-amyloid (A- β), referred to as amyloid plaques, and intracellular aggregates of hyperphosphorylated tau in the form of neurofibrillary tangles. There are different forms of amyloid such as plaques, oligomers, and monomers, and the roles of these different forms and how specifically they are pathophysiologically associated with Alzheimer's disease is not well understood. Generally referred to as "amyloid hypothesis", it is believed that aggregation of A- β oligomers in the brain leads to amyloid plaques and is thought to be the primary driver of the disease process. Amyloid aggregation is thought to precede accumulation of tau pathology and neurodegeneration. These changes in the brain result in widespread neurodegeneration and cell death, and ultimately cause the clinical signs and symptoms of dementia.

Salient known risk factors for Alzheimer's disease are older age, genetics, and family history. Of these, increasing age has the largest known impact on the risk of developing Alzheimer's disease. While several genes have been found to increase the risk of Alzheimer's disease, the $\epsilon 4$ allele of the apolipoprotein E (ApoE) gene is the strongest known genetic risk factor. Having 1 copy of the gene is associated with a 2- to 3-fold increase in developing Alzheimer's disease while 2 copies of the gene may increase risk of Alzheimer's disease by as much as 15 times. Approximately two-thirds of pathology-confirmed Alzheimer's disease cases are $\epsilon 4$ positive (homozygous or heterozygous), compared with about 15% to 20% of the general population. Autosomal dominant genetic mutations are estimated to account for less than 1% of Alzheimer's disease cases.

The pathophysiological changes and clinical manifestations of Alzheimer's disease are progressive and occur along a continuum, and accumulation of A- β may begin 20 years or more before symptoms arise. National Institute on Aging-Alzheimer's Association (NIA-AA) have created a "numeric clinical staging scheme" (Table 1) that avoids traditional syndromal labels and is applicable for only those in the Alzheimer's continuum. This staging scheme reflects the sequential evolution of Alzheimer's disease from an initial stage characterized by the appearance of abnormal Alzheimer's disease biomarkers in asymptomatic individuals. As biomarker abnormalities progress, the earliest subtle symptoms become detectable. Further progression of biomarker abnormalities is accompanied by progressive worsening of cognitive symptoms, culminating in dementia. This numeric cognitive staging scheme is not designed to be used in a clinical setting but to be used for interventional trials such as those of aducanumab.



The phase 3 randomized controlled trials for aducanumab were stratified to include 80% of stage 3 individuals and 20% of stage 4 individuals. This numeric staging scheme is very similar to the categorical system for staging Alzheimer's disease outlined in the Food and Drug Administration (FDA) guidance for industry pertaining to developing drugs for treatment of early Alzheimer's disease.

Many tests are available in the market to detect the underlying core pathology such use of certain biomarkers in the cerebrospinal fluid (CSF) (e.g., decreased A- β and increased CSF tau protein levels) and on imaging (e.g., amyloid on positron emission tomography [PET] scans). Approved amyloid PET tracers in the US include [18F]-florbetapir, [18F]-flutemetamol and [18F]-florbetaben. In addition, there are several CSF tests for A- β confirmation that are currently in development in the US. CSF tests and amyloid PET tracers are routinely used in the enrollment of participants in contemporary Alzheimer's disease studies.

Current Treatment

Current treatment goals for individuals with Alzheimer's disease are often directed to maintain quality of life, treat cognitive symptoms, and manage behavioral and psychological symptoms of dementia. Treatment remains largely supportive, including creation and implementation of individualized dementia care plans, caregiver education and support, care navigation, care coordination, and referral to community-based organizations for services (e.g., adult day care, caregiver training, etc.).¹¹ Non-pharmacologic treatments include physical activity as well as behavioral strategies to ameliorate neuropsychiatric symptoms (e.g., agitation, delusions, disinhibition), and problem behaviors (e.g., resistance to care, hoarding, obsessive-compulsive behaviors). Currently FDA-approved drugs for Alzheimer's include cholinesterase inhibitors donepezil, rivastigmine, and galantamine, and the N-methyl-D-aspartate antagonist memantine. Cholinesterase inhibitors are indicated in mild, moderate, and severe AD, while memantine is approved for moderate-to-severe AD. These drugs, either alone or in combination, focus on managing cognitive and functional symptoms of the disease and have not been shown to alter disease trajectory. The evidence for efficacy is limited and associated with significant side effects.



Table 1. National Institute on Aging-Alzheimer’s Association Numerical Clinical Staging for Individuals in the Alzheimer’s Continuum^a

Stage	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6
Severity	Pre-clinical	Pre-clinical	MCI due to Alzheimer’s disease	Mild Dementia	Moderate Dementia	Severe Dementia
Clinical Features	<ul style="list-style-type: none"> • Performance within expected range on objective cognitive tests. • No evidence of recent cognitive decline or new neurobehavioral symptoms. 	<ul style="list-style-type: none"> • Normal performance within expected range on objective cognitive tests. • Transitional cognitive decline (change from individual baseline within past 1 to 3 years, and persistent for at least 6 months). • Mild neurobehavioral changes may coexist or may be the primary complaint rather than cognitive. • No functional impact on daily life activities 	<ul style="list-style-type: none"> • Performance in the impaired/ abnormal range on objective cognitive tests. • Evidence of decline from baseline. • Performs daily life activities independently, but cognitive difficulty may result in detectable but mild functional impact on the more complex activities of daily life. 	<ul style="list-style-type: none"> • Substantial progressive cognitive impairment affecting several domains, and/or neurobehavioral disturbance. • Clearly evident functional impact on daily life, affecting mainly instrumental activities. • No longer fully independent /requires occasional assistance with daily life activities. 	<ul style="list-style-type: none"> • Progressive cognitive impairment or neurobehavioral changes. • Extensive functional impact on daily life with impairment in basic activities. • No longer independent and requires frequent assistance with daily life activities 	<ul style="list-style-type: none"> • Progressive cognitive impairment or neurobehavioral changes. • Clinical interview may not be possible. • Complete dependency due to severe functional impact on daily life with impairment in basic activities, including basic self-care.

Adapted from Table 6, Jack et al (2018)

^aApplicable only to individuals in the Alzheimer’s continuum that fall into 1 of the 4 biomarker groups: 1) A+T+N+ 2) A+T-N- 3) A+T+N- 4) A+T-N+ where A: Aggregated A β or associated pathologic state (CSF A β 42, or A β 42/A β 40 ratio or Amyloid PET), T: Aggregated tau (neurofibrillary tangles) or associated pathologic state (CSF phosphorylated tau or Tau PET) and N: Neurodegeneration or neuronal injury (anatomic MRI, FDG PET or CSF total tau)

For stages 1 to 6: Cognitive test performance may be compared to normative data of the investigators choice, with or without adjustment (choice of the investigators) for age, sex, education, etc.

For stages 2 to 6: Although cognition is the core feature, neurobehavioral changes—for example, changes in mood, anxiety, or motivation—may coexist.



For stages 3 to 6: Cognitive impairment may be characterized by presentations that are not primarily amnesic.

CSF: cerebrospinal fluid; FDG: fluorodeoxyglucose; MCI: mild cognitive impairment; MRI: magnetic resonance imaging; PET: positron emission tomography.

Summary of Evidence

For individuals with early Alzheimer's disease (MCI or mild dementia due to Alzheimer's disease) who receive aducanumab, the evidence includes 2 RCTs and 1 dose-finding and proof of concept phase I trial. Relevant outcomes are disease-specific survival, change in disease status, functional outcomes, health status measures, quality of life, and treatment-related mortality and morbidity. ENGAGE (study 301) and EMERGE (study 302) were identical randomized, double-blind, placebo-controlled studies that enrolled individuals with early Alzheimer's disease. The majority of individuals had a diagnosis of MCI due to Alzheimer's disease (81.6%) and approximately two-thirds were apolipoprotein E ϵ 4 carriers. The primary clinical outcome was a change in mean score on the Clinical Dementia Rating Sum of Boxes (CDR-SB). Both trials were terminated early following a prespecified interim analysis for futility. In study 301, there was no treatment benefit observed in either the high- or low-dose arms at week 78. In study 302, a statistically significant difference in change from baseline in CDR-SB was observed in the high-dose arm (difference vs. placebo -0.39 [95% CI, -0.69 to -0.09]) but not the low-dose arm at week 78. The observed change of 0.39 was well below the range of 1 to 2 points reported as the minimal clinically important difference (MCID) in published literature. Approval by the FDA was based on the reduction in A- β plaques, which was observed in both trials and at all doses. However, there are no satisfactory data clearly establishing that individual changes in amyloid correlate with or predict long term cognitive and functional changes. In the absence of clinical data convincingly demonstrating a clinical effect, it cannot be concluded that the observed reduction in amyloid will translate into a clinical benefit to individuals. Cognitive decline in early Alzheimer's disease generally occurs over years, and thus the follow-up duration may not be sufficient to conclude whether a drug is effective for this disease or whether the safety profile might change with longer follow-up. A confirmatory, prospective and adequately powered trial is necessary to assess the net health benefit of aducanumab in individuals with early Alzheimer's disease. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

The efficacy of Leqembi was evaluated in a double-blind, placebo-controlled, parallel-group dose-finding trial, Study 201 (NCT01767311) in adult individuals with AD (patients with confirmed presence of amyloid pathology and MCI or mild dementia consistent with Stage 3 and Stage 4 AD). The study assessed three doses across two regimens of Leqembi. Study 201 had a 79-week double-blind, placebo-controlled period, followed by an open-label extension (OLE) period for up to 260 weeks, which was initiated after a gap period (range, 9 to 59 months);



mean, 24 months) off treatment. Following the precedent set by the accelerated approval of Aduhelm based on the surrogate endpoint of A β plaque reduction combined with generally consistent and favorable results on clinical endpoints, Study 201 met the criteria for accelerated approval of Leqembi according to the FDA. Change from baseline in brain amyloid plaque, as measured by 18F-florbetapir PET and quantified by a composite standard uptake value ratio (SUVR), was assessed in a subset of individuals at Weeks 53 and 79. This data served as the endpoint supporting accelerated approval. Compared with placebo, the Leqembi 10 mg/kg biweekly arm demonstrated a statistically significant reduction in brain amyloid plaque at Week 79 (mean difference of -0.31 SUVR or -73.5 Centiloids; $P < 0.001$).

Safety

For Aduhelm, data with limited follow-up are available to analyze safety because the phase 3 trials were stopped prematurely due to futility. Pooled safety data from the 2 phase 3 clinical trials showed that about 35% (compared to 3% in the placebo arm) of individuals on aducanumab experienced amyloid-related imaging abnormalities (ARIA), whose clinical effects can range from asymptomatic to severe. Although the majority of individuals were asymptomatic or had symptoms such as headache, confusion, or dizziness that resolved with temporary stoppage of the drug, 6.2% of participants receiving the high dose of aducanumab discontinued the drug due to ARIA. The incidence of ARIA-edema was higher in ApoE $\epsilon 4$ carriers than non-carriers (42% and 20%, respectively). The majority of ARIA-edema radiographic events occurred early in treatment (within the first 8 doses), although ARIA can occur at any time. Among individuals treated with a planned dose of aducanumab 10 mg/kg who had ARIA-edema, the maximum radiographic severity was mild in 30%, moderate in 58%, and severe in 13% of individuals (refer to prescribing label for classification of severity of ARIA). Resolution occurred in 68% of ARIA-edema individuals by 12 weeks, 91% by 20 weeks, and 98% overall after detection. Ten percent of all individuals who received aducanumab 10 mg/kg had more than 1 episode of ARIA-edema.

An increase in falling adverse events was observed in the high-dose as compared to placebo across the 2 phase 3 studies (15% vs. 12%, respectively). FDA statistical review, reported a hazard ratio of 1.33 ($p = .016$) suggesting a 33% relative increase in hazard of falling for 10 mg/kg compared to placebo. A quantitative integration of benefit and risk was not done, but if the high dose increases falls it could be a significant risk for the Alzheimer's disease population.

For Leqembi, in Study 201 the most common adverse reactions reported in at least 5% of patients treated with Leqembi 10 mg/kg biweekly ($n = 161$) and having at least 2% higher incidence than in individuals on placebo ($n = 245$) were infusion-related reactions (Leqembi



20%; placebo 3%), headache (Leqembi 14%; placebo 10%), ARIA-E (Leqembi 10%; placebo 1%), cough (Leqembi, 9%; placebo, 5%) and diarrhea (Leqembi, 8%; placebo, 5%). The most common adverse reactions leading to discontinuation of Leqembi were infusion-related reactions (e.g. flu-like symptoms, nausea, vomiting and changes in blood pressure), which led to discontinuation in 2% (4/161) of patients treated with Leqembi compared to 1% (2/245) of individuals on placebo. Individuals were excluded from enrollment in Study 201 for baseline use of anticoagulant medications. Antiplatelet medications such as aspirin and clopidogrel were allowed. Individuals who received Leqembi and an antithrombotic medication (i.e. aspirin, other antiplatelets, or anticoagulants) did not have an increased risk of amyloid-related imaging abnormalities (ARIA) with hemosiderin deposition (ARIA-H; includes microhemorrhage and superficial siderosis) compared to individuals who received placebo and an antithrombotic medication. The majority of exposures to antithrombotic medications were to aspirin; few individuals were exposed to other antiplatelet drugs or anticoagulants, limiting any meaningful conclusions about the risk of ARIA or intracerebral hemorrhage in individuals taking other antiplatelet drugs or anticoagulants. Because intracerebral hemorrhages greater than 1 cm in diameter have been observed in individuals taking Leqembi, additional caution should be exercised when considering the administration of antithrombotics or a thrombolytic agent (e.g. tissue plasminogen activator [tPA]) to an individual already being treated with Leqembi. Additionally, individuals were excluded from enrollment in Study 201 for the following risk factors for intracerebral hemorrhage: prior cerebral hemorrhage greater than 1 cm in greatest diameter, more than 4 microhemorrhages, superficial siderosis, evidence of vasogenic edema, evidence of cerebral contusion, aneurysm, vascular malformation, infective lesions, multiple lacunar infarcts or stroke involving a major vascular territory, and severe small vessel or white matter disease. Caution should be exercised when considering the use of Leqembi in individuals with these risk factors. The approved label does not contain a Boxed Warning or require a REMS program. However, it does include a warning about ARIA with a recommendation for MRI monitoring for signs and symptoms and discussion of concomitant antithrombotic medication use. Of concern, three deaths have been reported in the OLE trial of Leqembi (ALZFORUM, 2022); two individuals had received anticoagulants, and another had a history of treatment with tPA.

Practice Guidelines and Position Statements

Guidelines or position statements will be considered for inclusion if they were issued by, or jointly by, a US professional society, an international society with US representation, or National Institute for Health and Care Excellence (NICE). Priority will be given to guidelines that are



informed by a systematic review, include strength of evidence ratings, and include a description of management of conflict of interest.

Institute for Clinical and Economic Review

The Institute for Clinical and Economic Review (ICER) evaluated the effectiveness and value of aducanumab for Alzheimer's disease and released their final evidence report on August 5, 2021. The report concludes, "given the certainty that harms can occur in individuals treated with aducanumab and uncertainty about benefits, we rate the evidence to be insufficient to determine the net health benefit of aducanumab ("I") in individuals with MCI and mild AD." The conclusion about uncertainty of benefits stems from a number of methodologic issues raised in the report that includes use of phase Ib trial to provide a "second" positive trial as supportive evidence, post-hoc analyses to explain failure of study 301, and role of functional blinding due to amyloid-related imaging abnormalities. ICER evaluated the effectiveness and value of lecanemab for Alzheimer's disease and released their final evidence report on April 17, 2023. ICER rated the current evidence to be promising but inconclusive (P/I) to determine whether lecanemab provides a net health benefit over supportive care alone in individuals with mild cognitive impairment due to Alzheimer's disease or mild Alzheimer's disease.

Regulatory Status

In June 2021, aducanumab (Aduhelm; Biogen) was approved by the US FDA for treatment of Alzheimer's disease. This indication was approved under accelerated approval based on reduction in A- β plaques observed in individuals treated with aducanumab. Continued approval for this indication may be contingent upon verification of clinical benefit in confirmatory trial(s).

In July 2021, FDA amended the approved label to emphasize the disease stages studied in the clinical trials. The amended label states, "Treatment with aducanumab should be initiated in individuals 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."

The FDA, under the accelerated approval regulations (21 CFR 601.41), requires that Biogen conduct a randomized, controlled trial to evaluate the efficacy of aducanumab-awwa compared to an appropriate control for the treatment of Alzheimer's disease. The trial should be of sufficient duration to observe changes on an acceptable endpoint in the individual population



enrolled in the trial. The expected date of trial completion is August 2029 and final report submission to the FDA by February 2030.

In January 2023, lecanemab (Leqembi; Eisai) was approved by the US FDA for the treatment of Alzheimer's disease. Continued approval for this indication may be contingent upon verification of clinical benefit in confirmatory trial(s).

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History

Date	Comments
10/01/21	New policy, approved September 14, 2021. The use of Aduhelm (aducanumab) is considered investigational for all indications including treatment of Alzheimer's disease. Added HCPC code J3590 to report Aduhelm.
1/1/22	Coding update, Added HCPCS code J0172 and removed HCPCS code J3590.
12/01/22	Annual Review, approved November 21, 2022. No changes to policy statement. Changed the wording from "patient" to "individual" throughout the policy for standardization.
05/01/23	Annual Review, approved April 19, 2023. No changes to policy statement. The use of Leqembi (lecanemab-irmb) is considered investigational for all indications including treatment of Alzheimer's disease. Added HCPC code to report Leqembi.
08/01/23	Coding update. Added HCPCS code J0174 for Leqembi. Removed UNL code J3590 (previously used to report Leqembi)
05/01/24	Annual Review, approved April 9, 2024. Added coverage criteria for Leqembi (lecanemab-irmb) for the treatment of Alzheimer's disease.

Disclaimer: This medical policy is a guide in evaluating the medical necessity of a particular service or treatment. The Company adopts policies after careful review of published peer-reviewed scientific literature, national guidelines and local standards of practice. Since medical technology is constantly changing, the Company reserves the right to review and update policies as appropriate. Member contracts differ in their benefits. Always consult the member benefit booklet or contact a member service representative to determine coverage for a specific medical service or supply. CPT codes, descriptions and materials are copyrighted by the American Medical Association (AMA). ©2024 Premera All Rights Reserved.



Scope: Medical policies are systematically developed guidelines that serve as a resource for Company staff when determining coverage for specific medical procedures, drugs or devices. Coverage for medical services is subject to the limits and conditions of the member benefit plan. Members and their providers should consult the member benefit booklet or contact a customer service representative to determine whether there are any benefit limitations applicable to this service or supply. This medical policy does not apply to Medicare Advantage.



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Language Assistance

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