Kisunla™ (Donanemab-Azbt)

Policy Number: 2026D0130D
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➲ Instructions for Use

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Related Community Plan Policy

- Monoclonal Antibodies Directed Against Amyloid for the Treatment of Alzheimer's Disease

Coverage Rationale

➲ See Benefit Considerations

Kisunla (donanemab-azbt) is proven for the treatment of Alzheimer's disease (AD) when all of the following criteria are met:

- For **initial therapy**, **all** of the following:
  - Diagnosis of **one** of the following based on National Institute on Aging and the Alzheimer's Association (NIA-AA) criteria:
    - Mild cognitive impairment (MCI) due to Alzheimer's disease; **or**
    - Mild dementia due to Alzheimer's disease

    and
  - Presence of amyloid beta pathology has been confirmed; **and**
  - A baseline brain magnetic resonance imaging (MRI) has been completed within 12 months prior to initiating treatment; **and**
  - Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Leqembi); **and**
  - Kisunla dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
  - Initial authorization will be for no more than 12 months
- For **continuation of therapy**, **all** of the following:
  - Diagnosis of Alzheimer's disease; **and**
  - Follow-up brain MRI has been completed after the initiation of therapy; **and**
  - Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Leqembi); **and**
  - Kisunla dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
  - Reauthorization is for no more than 12 months

Kisunla (donanemab-azbt) is medically necessary for the treatment of Alzheimer's disease (AD) when all of the following criteria are met:

- For **initial therapy**, **all** of the following:
  - Diagnosis of **one** of the following based on National Institute on Aging and the Alzheimer's Association (NIA-AA) criteria:
    - Mild cognitive impairment (MCI) due to Alzheimer's disease; **or**
    - Mild dementia due to Alzheimer's disease

    and

- Submission of medical records (e.g., chart notes, laboratory values) documenting **one** of the following:
  - Mini-Mental State Examination (MMSE) score of 20 to 30
  - Montreal Cognitive Assessment (MoCA) score of 17 to 30
  - Saint Louis University Mental Status (SLUMS) score of 17 to 30

  and
- Submission of medical records (e.g., chart notes, laboratory values) documenting the presence of amyloid beta pathology, as evidenced by positive amyloid positron emission tomography (PET) brain imaging; **and**
- Other differential diagnoses [e.g., dementia with Lewy bodies (DLB), frontotemporal dementia (FTD), vascular dementia, pseudodementia due to mood disorder, vitamin B12 deficiency, encephalopathy, etc.] have been ruled out; **and**
- **One** of the following:
  - Patient is not currently taking an anticoagulant (e.g., warfarin, dabigatran); **or**
  - **Both** of the following:
    - Patient is currently taking an anticoagulant (e.g., warfarin, dabigatran); **and**
    - Counseling has been provided that the combined use of Kisunla with anti-coagulant drugs may increase the risk of cerebral macrohemorrhage and prescriber attests that the patient has shared in decision-making to initiate Kisunla therapy

  and
- Patient has no history of intracerebral hemorrhage within the previous year prior to initiating treatment; **and**
- Counseling has been provided on the risk of amyloid-related imaging abnormalities [ARIA characterized as ARIA with edema (ARIA-E) and ARIA with hemosiderin (ARIA-H)] and patient is aware to monitor for headache, dizziness, visual disturbances, nausea, and vomiting; **and**
- **All** of the following:
  - Counseling has been provided on how testing for apolipoprotein E (ApoE) epsilon 4 (ε4) status informs the risk of developing ARIA when deciding to initiate treatment with Kisunla; **and**
  - Testing for ApoE ε4 status has been offered to the patient and prescriber attests that the patient has shared in decision-making to initiate Kisunla therapy

  and
- A baseline brain magnetic resonance imaging (MRI) has been completed within 12 months prior to initiating treatment; **and**
- Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Leqembi); **and**
- Prescribed by a neurologist, geriatric psychiatrist, or geriatrician who specializes in treating dementia; **and**
- Kisunla dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
- Initial authorization will be for no more than 12 months

- For **continuation of therapy**, **all** of the following:
  - **One** of the following:
    - **Both** of the following:
      - Patient has received Kisunla therapy for less than or equal to 18 months; **and**
      - **One** of the following:
        - Post-treatment amyloid PET brain imaging is positive for amyloid based on visual read; **or**
        - Prescriber attests that amyloid PET imaging will be performed prior to 18 months of total treatment to assess for the effect of Kisunla treatment on amyloid plaque

      or
    - **Both** of the following:
      - Patient has received Kisunla therapy for greater than 18 months; **and**
      - Post-treatment amyloid PET brain imaging is performed at least once per 12 months and is positive for amyloid based on visual read

    and
  - **Both** of the following:
    - Submission of medical records (e.g., chart notes) confirming follow-up brain magnetic resonance imaging (MRI) has been completed after the initiation of therapy; **and**
    - **One** of the following:
      - ARIA has not been observed on MRI; **or**
      - All of the following:
        - ARIA has been observed on MRI; **and**
        - Prescriber attests that continuation of therapy with Kisunla is appropriate based on the severity of the patient's clinical symptoms; **and**
        - **One** of the following:
          - Follow-up MRI demonstrates radiographic resolution and/or stabilization; **or**

- Prescriber attests that continuation of therapy with Kisunla is appropriate based on the radiographic severity of ARIA

and

- Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Leqembi); **and**
- Prescribed by a neurologist, geriatric psychiatrist, or geriatrician who specializes in treating dementia; **and**
- Kisunla dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
- Reauthorization is for no more than 12 months

Kisunla (donanemab-azbt) is unproven and not medically necessary for any indication other than Alzheimer's disease.

Applicable Codes

The following list(s) of procedure and/or diagnosis codes is provided for reference purposes only and may not be all inclusive. Listing of a code in this policy does not imply that the service described by the code is a covered or non-covered health service. Benefit coverage for health services is determined by the member specific benefit plan document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claim payment. Other Policies and Guidelines may apply.

HCPCS Code	Description
J0175	Injection, donanemab-azbt, 2mg

Diagnosis Code	Description
G30.0	Alzheimer's disease with early onset
G30.1	Alzheimer's disease with late onset
G30.8	Other Alzheimer's disease
G30.9	Alzheimer's disease, unspecified

Background

Alzheimer's disease (AD) is the most common cause of dementia and accounts for an estimated 60% to 80% of cases.

AD is characterized by deposition of amyloid-beta Aβ plaques and neurofibrillary tangles (comprised of abnormal tau protein) in the brain, accompanied by synaptic dysfunction and neurodegeneration. The deposition of Aβ (as amyloid plaques) generally begins decades before any symptoms of AD are observed. More specifically, Aβ deposition is followed sequentially by markers of neurodegeneration, accumulation of tau pathology, and brain volume loss. This pre-symptomatic phase of AD will precede the emergence of AD symptoms 10 to 20 years prior.

Tau is the microtubule associated protein (MAP) of a normal mature neuron. Tau is a phosphoprotein that promotes the assembly of tubulin into microtubules and stabilization of their structure. In AD (and certain other related neurodegenerative diseases, called tauopathies), tau protein is abnormally hyperphosphorylated and aggregated into bundles of filaments. In AD, this tau pathology is seen as intraneuronal neurofibrillary tangles of paired helical filaments sometimes admixed with straight filaments. Aggregates of abnormally hyperphosphorylated filaments are also seen in dystrophic neurites surrounding the Aβ plaque core, and in the neuropil as neuropil threads.

Abnormal Aβ may be detected directly via PET imaging using tracers or indirectly by measuring the levels of the long form of Aβ in the CSF. P-tau and t-tau can also be detected using CSF and are used as biomarkers to detect the emergence of AD in patients with MCI.

Age of AD onset:

- Typical AD: AD is characteristically a disease of older age. The incidence and prevalence of AD increase exponentially with age, essentially doubling in prevalence every 5 years after the age of 65 years.
- Early-onset AD: Early-onset AD is less common, and occurs in patients < 65 years of age. These patients often present with symptoms somewhat atypical for this disease, such as language, visual, or mood-behavioral changes rather than predominant memory loss.

- Inherited forms of AD: These forms of AD are rare (< 1% of all AD cases) and routinely present before 65 years of age, frequently in the fifth decade or earlier. Inherited forms of AD typically exhibit an autosomal-dominant inheritance pattern related to mutations in genes that alter Aβ protein production or metabolism, including amyloid precursor protein (APP), presenilin-1 (PSEN1), and presenilin-2 (PSEN2).
- AD associated with Down syndrome: Patients with Down syndrome have an additional gene dose of APP due to trisomy of chromosome 21 and inevitably develop AD pathology. Symptoms tend to emerge at an earlier age, i.e., 10 to 20 years earlier than the general population with AD.

Risk factors for AD:
- Aging is an important risk factor for dementia. AD affects 5% to 10% of people > 65 years of age, and 50% of those ≥ 85 years of age.
- Nonmodifiable risk factors for AD include female gender, Black race, Hispanic ethnicity, and genetic factors such as presence of the APOE gene.
- Modifiable risk factors for all-cause dementia include hypertension, diabetes, diet, and limited cognitive, physical, and social activities.

While the genetic basis for early-onset AD is much better understood, the genetic basis of late-onset AD is considered far more complex, with susceptibility conferred by a variety of more common but less penetrant genetic factors likely interacting with environmental and epigenetic influences. To date, the most firmly established genetic risk factor for late-onset disease is APOE:
- The APOE gene is located on chromosome 19 and exists in 3 alleles: epsilon 2, 3, and 4. The APOE epsilon 4 (ε4) allele has been confirmed to be an important risk factor for AD in many clinical trials.
- Factors that may influence the impact of APOE ε4 on AD risk include female gender, African/African-American race (although there are conflicting data), vascular risk factors (e.g., smoking, diabetes, hypertension, and hypercholesterolemia), and modifier genes/environment.
- Genetic testing is available for the known causative genes in early-onset AD but has not been widely adopted, likely in part because of the current lack of highly effective preventive or therapeutic strategies.

The symptoms at early-stage AD are less pronounced than in later stages of AD, and therefore require measures that are different from those used in later stages.

The Clinical Dementia Rating-Sum of Boxes (CDR-SB) is an integrated scale that assesses both daily function and cognitive effects and was shown to be sufficiently sensitive and specific to detect change over time in early symptomatic AD patients. The scale integrates assessments from 3 domains of cognition (memory, orientation, judgment/problem-solving) and 3 domains of function (community affairs, home/hobbies, personal care). CDR-SB scores range from 0-18, with higher scores indicating greater disease severity. A minimal clinically important difference in CDR-SB has not been clearly defined but has been estimated to be 1-2 points. A CDR-SB score ranging from 0.5 - 4.0 has been reported to correspond to a CDR-G score of 0.5. A CDR-SB score ranging from 4.5-9.0 has been reported to correspond to a CDR-G score of 1.[1]

CDR-SB Score	Disease Severity
0	Normal
0.5 - 4.0	Suggests questionable cognitive impairment to very mild dementia
0.5 - 2.5	Suggests questionable cognitive impairment
3.0 - 4.0	Suggests very mild dementia
4.5 - 9.0	Suggests mild dementia
9.5 - 15.5	Suggests moderate dementia
16.0 - 18.0	Suggests severe dementia

The Mini-Mental State Exam (MMSE) is a widely used performance-based test of global cognitive status. The MMSE is a measure of cognition that includes 11 tasks relating to topics of word recall, attention and calculation, language ability, and visuospatial function. The scale ranges from 0 to 30 with a lower score reflecting greater cognitive impairment. It has several known limitations impacting sensitivity to change, particularly in earlier disease stages: substantial ceiling effects, sensitivity to practice effects, scores are impacted by patients' educational achievement, and learning effects are observed. The minimal clinically important difference of the MMSE in AD is estimated to be 1-3 points, and in early AD to be 1-2 points.

MMSE Score	Disease Severity
25 - 30	Normal to questionable cognitive impairment
19 - 24	Suggests mild dementia
10 - 18	Suggests moderate dementia
0 - 9	Suggests severe dementia

The Alzheimer's Disease Assessment Scale – Cognitive Subscale (13-Item version) (ADAS-Cog13) comprises both cognitive tasks and clinical ratings of cognitive performance. The scale items capture word recall, ability to follow commands, the ability to correctly copy or draw an image, naming, the ability to interact with everyday objects, orientation, word recognition, memory, comprehension of spoken language, word-finding, and language ability, with a measure for delayed word recall and concentration/distractibility. The total score ranges from 0 to 85 with an increase in score over time indicates increasing cognitive impairment. The minimal clinically important difference of the ADAS-COG 13 in early AD is estimated to be 3 points.

The Integrated Alzheimer's Disease Rating Scale (iADRS) is a linear combination of its two components: the ADAS-Cog13 and the Alzheimer's Disease Cooperative Study–Instrumental Activities of Daily Living scale (ADCS-iADL) (range: 0–59; lower scores indicating greater impairment; items: 6a and 7–23). Because worse outcomes are indicated by higher scores on the ADAS-Cog13 and lower scores on the ADCS-iADL, the ADAS-Cog13 score is multiplied by −1 when calculating the iADRS score, such that lower iADRS scores indicate greater impairment. iADRS scores range from 0 to 144. The minimal clinically important difference of the iADRS has been suggested to be 5 points for MCI due to AD and 9 points for AD with mild dementia.

The Montreal Cognitive Assessment (MoCA) is a widely used screening test specifically designed to detect more subtle cognitive deficits that characterize mild cognitive impairment. Like the MMSE, the MoCA is scored on a 30-point scale, with items that assess delayed word recall, visuospatial/executive function, language, attention/concentration, and orientation. Studies examining head-to-head performance of patients on the MMSE and MoCA have shown that the MoCA is more difficult; MoCA scores are consistently lower than those obtained on the MMSE. The MoCA appears to be more sensitive than the MMSE for detecting MCI, though perhaps slightly less specific. A minimum clinically important difference of the MoCA in AD has not been described.

Assessment Scale	Minimal Clinical Important Difference
Clinical Dementia Rating-Sum of Boxes (CDR-SB)	1-2 points
Mini-Mental State Exam (MMSE)	1-3 points
Alzheimer's Disease Assessment Scale – Cognitive Subscale (13-Item version) (ADAS-Cog13)	3 points

The National Institute on Aging and the Alzheimer's Association (NIA-AA) research framework committee created a numeric clinical staging scheme applicable for diagnosing those in the Alzheimer's continuum. The six-stage numeric clinical staging scheme was brought forward largely unchanged (table below) into an Alzheimer's Association 2024 revision of the 2018 framework.

Stage	Numeric Clinical Staging–Applicable Only to Individuals in the Alzheimer's Disease Continuum
Stage 0 Asymptomatic, deterministic gene	• No evidence of clinical change. Biomarkers in normal range.
Stage 1 Asymptomatic, biomarker evidence only	• Performance within expected range on objective cognitive tests. • No evidence of recent cognitive decline or new symptoms.

Stage	Numeric Clinical Staging–Applicable Only to Individuals in the Alzheimer's Disease Continuum
Stage 2 Transitional decline: mild detectable change, but minimal impact on daily function	• Normal performance within expected range on objective cognitive tests. • Decline from previous level of cognitive or neurobehavioral function that represents a change from individual baseline within the past 1 to 3 years, and has been persistent for at least 6 months. • May be documented by evidence of subtle decline on longitudinal cognitive testing, which may involve memory or other cognitive domains but performance still within normal range. • May be documented through subjective report of cognitive decline. • May be documented with recent-onset change in mood, anxiety, motivation not explained by life events. • Remains fully independent with no or minimal functional impact on activities of daily living (ADLs).
Stage 3 Cognitive impairment with early functional impact	• Performance in the impaired/abnormal range on objective cognitive tests. • Evidence of decline from baseline, documented by the individual's report or by observer (e.g., study partner) report or by change on longitudinal cognitive testing or neurobehavioral assessments. • Performs daily life activities independently, but cognitive difficulty may result in detectable functional impact on complex ADLs (i.e., may take more time or be less efficient but still can complete—either self-reported or corroborated by an observer).
Stage 4 Dementia with mild functional impairment	• Progressive cognitive and mild functional impairment on instrumental ADLs, with independence in basic ADLs.
Stage 5 Dementia with moderate functional impairment	• Progressive cognitive and moderate functional impairment on basic ADLs requiring assistance.
Stage 6 Dementia with severe functional impairment	• Progressive cognitive and functional impairment, and complete dependence for basic ADLs.

Kisunla (donanemab-azbt) is a humanized immunoglobulin gamma 1 (IgG1) monoclonal antibody directed against insoluble N-truncated pyroglutamate amyloid beta. The accumulation of amyloid beta plaques in the brain is a defining pathophysiological feature of Alzheimer's disease. Donanemab-azbt reduces amyloid beta plaques.

Benefit Considerations

Some Certificates of Coverage allow for coverage of experimental/investigational/unproven treatments for life-threatening illnesses when certain conditions are met. The member specific benefit plan document must be consulted to make coverage decisions for this service. Some states mandate benefit coverage for off-label use of medications for some diagnoses or under some circumstances when certain conditions are met. Where such mandates apply, they supersede language in the benefit document or in the medical or drug policy.

Clinical Evidence

Multiple investigational anti-Aβ antibodies have been developed with the goal of either reducing production of Aβ or lowering levels of aggregated Aβ present in the brain, the latter of which has been the most pursued approach. Many of these investigational drugs have failed to demonstrate efficacy and/or safety. Some explanations for the failures of previous anti-Aβ antibodies include the following:[5,14]

- Inclusion of patients in clinical trials without evidence of Aβ pathology
- Unknown or no target engagement prior to initiation of Phase 3 study (i.e., poor selectivity of drug for neurotoxic Aβ)

- Lack of robust and sustained inhibition of soluble Aβ oligomers
- Use of subtherapeutic doses (possibly due to decreased brain penetration)
- Inclusion of patients at later stages of AD dementia when significant irreversible neurodegeneration has already occurred

FDA approval for donanemab was based on TRAILBLAZER-ALZ 2, a double-blind, placebo-controlled, parallel-group study (Study 1, NCT04437511) 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). Patients were enrolled with a Mini-Mental State Examination (MMSE) score of ≥ 20 and ≤ 28 and had a progressive change in memory function for at least 6 months. Patients were included in the study based on visual assessment of tau PET imaging with flortaucipir and standardized uptake value ratio (SUVR). Patients were enrolled with or without concomitant approved therapies (cholinesterase inhibitors and the N-methyl-D-aspartate antagonist memantine) for Alzheimer's disease. Patients could enroll in an optional, long-term extension. In TRAILBLAZER-ALZ 2, 1736 patients were randomized 1:1 to receive 700 mg of donanemab every 4 weeks for the first 3 doses, and then 1400 mg every 4 weeks (n = 860) or placebo (n = 876) for a total of up to 72 weeks. The treatment was switched to placebo based on amyloid PET levels measured at Week 24, Week 52, and Week 76. If the amyloid plaque level was < 11 Centiloids on a single PET scan or 11 to < 25 Centiloids on 2 consecutive PET scans, the patient was eligible to be switched to placebo. Additionally, dose adjustments were allowed for treatment-emergent ARIA or symptoms that then showed ARIA-E or ARIA-H on MRI. At baseline, mean age was 73 years, with a range of 59 to 86 years. Of the total number of patients randomized, 68% had low/medium tau level and 32% had high tau level; 71% were ApoE ε4 carriers and 29% were ApoE ε4 noncarriers. Fifty-seven percent of patients were female, 91% were White, 6% were Asian, 4% were Hispanic or Latino, and 2% were Black or African American. The primary efficacy endpoint was change in the integrated Alzheimer's Disease Rating Scale (iADRS) score from baseline to 76 weeks. The iADRS is a combination of two scores: the Alzheimer's Disease Assessment Scale-Cognitive subscale (ADAS-Cog13) and the Alzheimer's Disease Cooperative Study – instrumental Activities of Daily Living (ADCS-iADL) scale. The total score ranges from 0 to 144, with lower scores reflecting worse cognitive and functional performance. Other efficacy endpoints included Clinical Dementia Rating Scale – Sum of Boxes (CDR-SB), ADAS-Cog13, and ADCS-iADL. There were two primary analysis populations based on tau PET imaging with flortaucipir: 1) low/medium tau level population (defined by visual assessment and SUVR of ≥ 1.10 and ≤ 1.46), and 2) combined population of low/medium plus high tau (defined by visual assessment and SUVR > 1.46) population. Patients treated with donanemab demonstrated a statistically significant reduction in clinical decline on iADRS compared to placebo at Week 76 in the combined population [difference, 2.92 (95% CI, 1.51-4.33); p < .001] and the low/medium tau population [difference, 3.25 (95% CI, 1.88-4.62); p < .001]. Patients treated with donanemab demonstrated a statistically significant reduction in clinical decline on CDR-SB compared to placebo at Week 76 in the combined population [difference, -0.7 (95% CI, -0.95 to -0.45); p < .001]. There were also statistically significant differences (p < 0.001) between treatment groups as measured by ADAS-Cog13 and ADCS-iADL at Week 76. Dosing was continued or stopped in response to observed effects on amyloid imaging. The percentages of patients eligible for switch to placebo based on amyloid PET levels at Week 24, Week 52, and Week 76 timepoints were 17%, 47%, and 69%, respectively. Amyloid PET values may increase after treatment with donanemab is stopped. There is no data beyond the 76-week duration of TRAILBLAZER-ALZ 2 to guide whether additional dosing with donanemab may be needed for longer-term clinical benefit.

U.S. Food and Drug Administration (FDA)

This section is to be used for informational purposes only. FDA approval alone is not a basis for coverage.

Kisunla (donanemab-azbt) is indicated for the treatment of Alzheimer’s disease. Treatment with Kisunla should be initiated in patients with mild cognitive impairment or mild dementia stage of disease, the population in which treatment was initiated in the clinical trials.

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Policy History/Revision Information

Date	Summary of Changes

02/01/2026	**Coverage Rationale**
	• Changed initial authorization duration from "no more than ***6*** months" to "no more than ***12*** months"
	• Revised coverage criteria for **continuation of therapy**:
	  ○ Removed criterion requiring one of the following:
	  ▪ The patient has mild cognitive impairment (MCI) due to Alzheimer's disease
	  ▪ The patient has mild dementia due to Alzheimer's disease
	  ▪ The patient has progressed into moderate or severe stages of dementia due to Alzheimer's disease and the prescriber attests that the patient has shared in decision-making to continue Kisunla therapy
	  ○ Replaced criterion requiring:
	  ▪ "The patient has received Kisunla therapy for less than or equal to ***6*** months" with "the patient has received Kisunla therapy for less than or equal to ***18*** months"
	  ▪ "The patient has received Kisunla therapy for greater than ***6*** months, *post-treatment amyloid PET brain imaging obtained between 12 and 18 months of total treatment is positive for amyloid based on visual read,* and *for treatment beyond 18 months of therapy,* post-treatment amyloid PET brain imaging is performed at least once per 12 months and is positive for amyloid based on visual read" with "the patient has received Kisunla therapy for greater than ***18*** months and post-treatment amyloid PET brain imaging is performed at least once per 12 months and is positive for amyloid based on visual read"
	Supporting Information
	• Archived previous policy version 2025D0130C

Instructions for Use

This Medical Benefit Drug Policy provides assistance in interpreting UnitedHealthcare standard benefit plans. When deciding coverage, the member specific benefit plan document must be referenced as the terms of the member specific benefit plan may differ from the standard plan. In the event of a conflict, the member specific benefit plan document governs. Before using this policy, check the member specific benefit plan document and any applicable federal or state mandates. UnitedHealthcare reserves the right to modify its Policies and Guidelines as necessary. This Medical Benefit Drug Policy is provided for informational purposes. It does not constitute medical advice.

This Medical Benefit Drug Policy may also be applied to Medicare Advantage plans in certain instances. In the absence of a Medicare National Coverage Determination (NCD), Local Coverage Determination (LCD), or other Medicare coverage guidance, CMS allows a Medicare Advantage Organization (MAO) to create its own coverage determinations, using objective evidence-based rationale relying on authoritative evidence (Medicare IOM Pub. No. 100-16, Ch. 4, §90.5).

UnitedHealthcare may also use tools developed by third parties, such as the InterQual® criteria, to assist us in administering health benefits. UnitedHealthcare Medical Benefit Drug Policies are intended to be used in connection with the independent professional medical judgment of a qualified health care provider and do not constitute the practice of medicine or medical advice.