

RNA-Targeted Therapies (Amvuttra® and Onpattro®) (for Louisiana Only) Retired April 1, 2026

Policy Number: CSLA2025D0072N

Effective Date: October 1, 2025 – March 31, 2026

[➔ Instructions for Use](#)

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Application

This Medical Benefit Drug Policy only applies to the state of Louisiana.

Coverage Rationale

Amvuttra (vutrisiran) is medically necessary for the treatment of cardiomyopathy of wild-type (wtATTR) or hereditary transthyretin-mediated (hATTR) amyloidosis in patients who meet all of the following criteria:

- **Initial Therapy**
 - Diagnosis of wild-type or hereditary ATTR-mediated amyloidosis with cardiomyopathy (ATTR-CM); **and**
 - Documentation of **one** of the following:
 - The patient has a pathogenic TTR mutation (e.g., V30M); **or**
 - Cardiac or noncardiac tissue biopsy demonstrating histologic confirmation of ATTR amyloid deposits; **or**
 - **All** of the following:
 - Echocardiogram or cardiac magnetic resonance imaging suggestive of amyloidosis; **and**
 - Radionuclide imaging (99mTc-DPD, 99mTc-PYP, or 99m Tc-HMDP) showing grade 2 or 3 cardiac uptake*; **and**
 - Absence of light chain amyloidosis
 - and**
 - Patient has New York Heart Association (NYHA) Functional Class I, II, or III heart failure; **and**
 - Physician attests that the patient has an N-terminal pro-B-type natriuretic peptide (NT-proBNP) level that, when combined with signs and symptoms, is considered definitive for a diagnosis of ATTR-CM; **and**
 - Documentation of **one** of the following:
 - History of heart failure, with at least one prior hospitalization for heart failure; **or**
 - Presence of signs and symptoms of heart failure (e.g., dyspnea, edema)
 - and**
 - **One** of the following:
 - Documentation of progressive worsening or an event resulting in hospitalization related to hATTR-mediated amyloidosis with cardiomyopathy (ATTR-CM) while on one of the following:
 - Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis); **or**
 - Attruby (acoramidis)

- Patient continues to have a PND score ≤ IIIb; **or**
- Patient continues to have a FAP stage 1 or 2; **or**
- Patient continues to have a NIS score ≥ 5 and ≤ 130; **or**
- Patient continues to have a KPS score ≥ 60%

and

- Documentation that the patient has experienced a positive clinical response to Amvuttra or Onpattro (e.g., improved neurologic impairment, motor function, quality of life, slowing of disease progression, etc.); **and**
- **One** of the following:
 - For **Amvuttra**, patient is not receiving in combination with **any** of the following:
 - RNA interference agents [e.g., Onpattro (patisiran), Wainua (eplontersen), Tegsedi (inotersen)]; **or**
 - Transthyretin stabilizers [e.g., Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis)]
 - or**
 - For **Onpattro**, patient is not receiving in combination with **any** of the following:
 - RNA interference agents [e.g., Amvuttra (vutrisiran), Wainua (eplontersen), Tegsedi (inotersen)]; **or**
 - Transthyretin stabilizers [e.g., Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis)]

and

- Prescribed by or in consultation with a neurologist; **and**
- Dosing is in accordance with the US Food and Drug Administration prescribing information; **and**
- Authorization is for no more than 12 months

Amvuttra® (vutrisiran) and Onpattro® (patisiran) are unproven and not medically necessary for the treatment of:

- Sensorimotor or autonomic neuropathy not related to hATTR amyloidosis
- Primary or leptomenigeal amyloidosis

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 federal, state, or contractual requirements 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
J0222	Injection, patisiran, 0.1 mg
J0225	Injection, vutrisiran, 1 mg

Diagnosis Code	Description
E85.0	Non-neuropathic hereditary amyloidosis
E85.1	Neuropathic hereditary amyloidosis
E85.4	Organ-limited amyloidosis
E85.82	Wild-type transthyretin-related (ATTR) amyloidosis

Background

Amyloidosis is a broad term that refers to a protein deposition in extracellular tissue, which otherwise typically occur as a component of human plasma. Amyloid deposits manifest in a variety of clinical settings depending on their subtype, location, and prevalence. Immunoglobulin light chain (AL) and transthyretin (ATTR) are the two principal systemic types of amyloidosis. ATTR amyloidosis is a progressive, disabling, and life-threatening condition affecting major organ systems such as the circulatory as well as the central nervous system. This disease is an autosomal transmission disorder which is usually due to a point mutation of the transthyretin (TTR) gene. The disease is caused by misfolded transthyretin (TTR) protein that accumulates as amyloid fibrils in multiple organs, including the nerves, heart, and gastrointestinal tract. ATTR amyloidosis can occur as 'wild-type (wtATTR), often associated with aging, or through mutant proteins (ATTRv indicates a variant subtype while ATTRh indicates a hereditary component). In addition to neuropathic manifestations, hereditary and wild-type ATTR amyloidosis can also cause cardiac disease, typically presenting with signs and symptoms such as dyspnea, lower extremity edema, and/or ascites. This is a result of restrictive cardiomyopathy, often from right ventricular failure. Therapy for cardiac amyloidosis consists of treating the underlying hematologic disorder using traditional agents for heart failure.

Amvuttra (vutrisiran) and Onpattro (patisiran) are double-stranded small interfering RNAs (siRNAs) that target a sequence of mRNA conserved across wild-type and all TTR variants and can thereby degrade and reduce serum levels and protein deposits in tissues of both wild-type and mutated protein. It is formulated as lipid nanoparticles which direct it to the liver, the primary source of circulating TTR. Patisiran therapy is associated with observed lowering of TTR levels in both wild-type and mutant (V30M) forms of TTR.

A genetic testing service is available in the United States and Canada and a genetic counseling service is available in the United States. Medical professionals and patients may access information on the Alnylam Pharmaceuticals website.

Clinical Evidence

Cardiomyopathy of Wild-Type (wtATTR) or Hereditary Transthyretin-Mediated (hATTR) Amyloidosis

The safety and efficacy of vutrisiran in ATTR cardiomyopathy was established in a phase 3 randomized, double-blind study (HELIOS-B) (NCT04153149) in adult patients with ATTR-CM set up in a 1:1 ratio to receive vutrisiran (25 mg) or placebo every 12 weeks for up to 36 months. The primary end point was a composite of death from any cause and recurrent cardiovascular events. A total of 655 patients underwent randomization; 326 were assigned to receive vutrisiran and 329 to receive placebo. The study's primary end point was met, as vutrisiran demonstrated a lower risk of death from any cause and recurrent cardiovascular events compared to placebo (hazard ratio in the overall population, 0.72; 95% confidence interval [CI], 0.56 to 0.93; $p = 0.01$; hazard ratio in the monotherapy population, 0.67; 95% CI, 0.49 to 0.93; $p = 0.02$). A secondary endpoint, lower risk of death from any cause through 42 months, also showed statistical significance (hazard ratio in the overall population, 0.65; 95% CI, 0.46 to 0.90; $p = 0.01$) in favor of vutrisiran. Other secondary endpoints included were the change from baseline at 30 months in functional capacity, as assessed with the 6-minute walk test. In the overall population, the least-squares mean change from baseline in the distance covered on the 6-minute walk test was -45.4 m in the vutrisiran group and -71.9 m in the placebo group (least-squares mean difference, 26.5 m; 95% CI, 13.4 to 39.6; $p < 0.001$). Additionally, patient-reported health status and health-related quality of life, as assessed with the Kansas City Cardiomyopathy Questionnaire–Overall Summary (KCCQ-OS) was also used as a secondary end point. The least-squares mean change from baseline in the KCCQ-OS score was -9.7 points in the vutrisiran group and -15.5 points in the placebo group (least-squares mean difference, 5.8 points; 95% CI, 2.4 to 9.2; $p < 0.001$). At 30 months, in the overall population, 68% of the patients in the vutrisiran group and 61% in the placebo group had improvement or no change in NYHA class (least-squares mean difference, 8.7 percentage points; 95% CI, 1.3 to 16.1; $p = 0.02$), another secondary endpoint used for analysis. Among the study population, patients were permitted to use tafamidis as background therapy in both groups, therefore the trial did not allow for a comparison of vutrisiran alone with tafamidis alone. The trial was not powered to show statistical significance within the subgroup of patients (40% of patients) who were taking tafamidis at baseline. Of the patients who were not already taking tafamidis at the start of the trial, approximately 20% began taking tafamidis at some point during the trial.

Institute for Clinical and Economic Review (ICER)

On October 21st, 2024, ICER released a clinical report entitled, "Disease Modifying Therapies for the Treatment of Transthyretin Amyloid Cardiomyopathy (ATTR-CM)". ICER summary and comments regarding vutrisiran are as follows:

"Results from the HELIOS-B trial show large relative reductions in mortality in all patients and similar (but statistically non-significant) reductions in those receiving or not receiving tafamidis. The population studied was a contemporary population where 40% of patients were receiving tafamidis. Mortality benefit was seen during the open-label extension where both arms may have been receiving vutrisiran, and so those relative effects seen in HELIOS-B may underestimate the actual benefits. The primary composite endpoint of all-cause mortality and recurrent CV events was also reduced by vutrisiran. The absolute reductions in all-cause mortality in HELIOS-B were clinically important. There were no concerns about safety or side effects. As such, we have high certainty that treatment with vutrisiran, compared with no disease specific therapy or when added to tafamidis, provides a substantial net health benefit."

Polyneuropathy of Hereditary Transthyretin-Mediated (hATTR) Amyloidosis

A randomized, double-blind, placebo-controlled, phase III, global study (APOLLO) evaluated the efficacy and safety of patisiran in patients with hATTR amyloidosis with polyneuropathy. Adult patients 18 to 85 years of age were eligible for the study if the investigatory estimated survival to be ≥ 2 years, Neuropathy Impairment Score (NIS) of 5 to 130, and polyneuropathy disability score \leq IIIb. Patients were randomized 2:1 ($n = 148:77$) to receive either intravenous (IV) patisiran 0.3 mg/kg or placebo every 3 weeks. The primary endpoint was to determine the efficacy of patisiran at 18 months based on the difference in the change in modified NIS + 7 (a composite measure of motor strength, sensation, reflexes, nerve conduction, and autonomic function) between the patisiran and placebo groups. Secondary endpoints

evaluated the effect of patisiran on Norfolk-Diabetic Neuropathy quality of life questionnaire score, nutritional status (as evaluated by modified body mass index), motor function (as measured by NIS-weakness and timed 10-m walk test), and autonomic symptoms (as measured by the Composite Autonomic Symptom Score-31 questionnaire). Exploratory objectives include assessment of cardiac function and pathologic evaluation to assess nerve fiber innervation and amyloid burden. Safety of patisiran was also assessed throughout the study. Overall patisiran reduced the mean max serum TTR reduction by 87.8% from baseline in the patisiran treated group over 18 months. The LS mean change in the mNIS + 7 from baseline at 18 months was -33.99 ($p = 9.26 \times 10^{-24}$); (patisiran -6.03; placebo + 27.96). The LS mean change in the Norfolk QoL-DN from baseline at 18 months was -21.1 ($p = 1.10 \times 10^{-10}$); (patisiran -6.7; placebo + 14.4). All secondary endpoints (e.g., NIS-W, R-ODS, COMPASS-31, etc.) also achieved statistical significance at 18 months. The investigators also concluded that patisiran therapy was relatively safe and well tolerated with no increases in the frequency of events for patisiran compared to placebo group by system organ class. Overall, 13 deaths occurred in the APOLLO study, however, none of these were considered related to the study drugs and were consistent with natural history. The majority of infusion-related reactions were mild in severity, with no severe or life-threatening, or serious reactions. These reactions decreased over time and led to treatment discontinuation in only 1 patient. The investigators concluded that patisiran treatment resulted in significant improvement in polyneuropathy relative to placebo while significantly reducing disease symptoms and disability, improvement in quality of life, nutritional status, strength, and ambulation seen with patisiran relative to placebo.^{1,8}

In a subpopulation analysis of the APOLLO trial, investigators evaluated the treatment association of patisiran with regional left ventricular (LV) myocardial strain in cardiac manifestation in hATTR.^{11,12} The prespecified cardiac subpopulation [126 of 225 (56%)] comprised of patients with a baseline LV wall thickness of 13 mm or more and no history of hypertension or aortic valve disease. Of the 126 patients included in the prespecified cardiac subpopulation, 36 patients (28.6%) received placebo and 90 patients (71.4%) received patisiran. At baseline, LV global longitudinal strain (GLS) was impaired and regional longitudinal strains were lowest in the basal segments with apical sparing. There were no differences in regional longitudinal strains between the treatment groups at baseline. Patisiran improved the absolute GLS [least-squares mean (SE) difference, 1.4% (0.6%); 95% CI, 0.3%-2.5%; $p = .02$] compared with placebo at 18 months, with the greatest differential increase observed in the basal region [overall least-squares mean (SE) difference, 2.1% (0.8%); 95% CI, 0.6%-3.6%; $p = .006$] and no significant differences in the mid and apical regions among groups. Patisiran reduced mean left ventricular wall thickness (least-squares mean difference \pm SEM: -0.9 ± 0.4 mm, $p = 0.017$), interventricular septal wall thickness, posterior wall thickness, and relative wall thickness at month 18 compared with placebo. Patisiran also led to increased end-diastolic volume (8.3 ± 3.9 mL, $p = 0.036$), decreased global longitudinal strain ($-1.4 \pm 0.6\%$, $p = 0.015$), and increased cardiac output (0.38 ± 0.19 L/min, $p = 0.044$) compared with placebo at month 18. Patisiran lowered N-terminal prohormone of brain natriuretic peptide at 9 and 18 months (at 18 months, ratio of fold-change patisiran/placebo 0.45, $p < 0.001$). A consistent effect on N-terminal prohormone of brain natriuretic peptide at 18 months was observed in the overall APOLLO patient population ($n = 225$). Median follow-up duration was 18.7 months. The exposure-adjusted rates of cardiac hospitalizations and all-cause death were 18.7 and 10.1 per 100 patient-years in the placebo and patisiran groups, respectively (Andersen-Gill hazard ratio, 0.54; 95% CI, 0.28-1.01). The authors concluded that patisiran prevented the deterioration of LV GLS and decreased mean LV wall thickness over 18 months, suggesting that patisiran may halt or reverse the progression of the cardiac manifestations of hATTR amyloidosis.

The safety and efficacy of vutrisiran was established in a phase 3 randomized, open-label study (NCT03759379) in adult patients with polyneuropathy caused by hATTR amyloidosis. Patients eligible for the study met the following criteria: aged 18-25 years, diagnosis of ATTRv amyloidosis with a documented TTR variant and baseline NIS 5-130, polyneuropathy disability (PND) score \leq IIIb, a KPS \geq 60, and adequate liver and renal function. Patients were randomized 3:1 to receive 25 mg of vutrisiran subcutaneously once every 3 months ($n = 122$), or 0.3mg/kg patisiran intravenously every 3 weeks ($n = 42$) as a reference group. Efficacy assessments were based on a comparison of the vutrisiran with an external placebo group in another study (NCT01960348) composed of a comparable population of adult patients with polyneuropathy caused by hATTR amyloidosis. The primary endpoint was the change from baseline to month 9 in modified Neuropathy Impairment Score + 7 (mNIS + 7). The mNIS + 7 has a total score range from 0 to 304 points, with higher scores representing a greater severity of disease. The least squares mean change from baseline for the mNIS + 7 score was -2.2 for vutrisiran vs. + 14.8 for placebo (difference of -17.0, 95% CI: -21.8, -12.2; $p < 0.001$). The clinical meaningfulness of effects on the mNIS + 7 was assessed by the change from baseline to Month 9 in Norfolk Quality of Life-Diabetic Neuropathy (QoL-DN) total score. The Norfolk QoL-DN scale is a patient-reported assessment that evaluates the subjective experience of neuropathy in the following domains: physical functioning/large fiber neuropathy, activities of daily living, symptoms, small fiber neuropathy, and autonomic neuropathy. The Norfolk QoL-DN has a total score range from -4 to 136, with higher scores representing greater impairment. Additional endpoints were gait speed, as measured by the 10-meter walk test (10MWT), and modified body mass index (mBMI). The mean least squares mean change from baseline for the Norfolk QoL-DN total score was -3.3 for vutrisiran vs. + 12.9 for placebo (difference of -16.2, 95% CI: -21.7, -10.8; $p < 0.001$). The mean least squares mean change from baseline for the 10-meter walk test was 0 for vutrisiran vs. -0.13 for placebo (difference of 0.13, 95% CI; 0.07, 0.19; $p < 0.001$) and 10-meter walk test at Month 9 compared to

placebo in the external study ($p < 0.001$). The mean least squares mean change from baseline for mBMI was 7.6 for vutrisiran vs. -60.2 for placebo (difference of 67.8, 95% CI; 43.0, 92.6; $p < 0.001$).

The most common adverse reactions (at least 5%) were arthralgia (11%), dyspnea (7%), and decreased vitamin A (7%). Patients were instructed to take the recommended daily allowance of vitamin A. Seventy-four percent of patients treated with vutrisiran had normal vitamin A levels at baseline, and 98% of those with a normal baseline developed low vitamin A levels. In some cases, the decreased vitamin A level was reported as an adverse reaction. Two serious adverse reactions of atrioventricular (AV) heart block (1.6%) occurred in patients treated with vutrisiran, including one case of complete AV block. Injection site reactions were reported in 5 (4%) patients treated with vutrisiran. Reported symptoms included bruising, erythema, pain, pruritus, and warmth. Injection site reactions were mild and transient.

Institute for Clinical and Economic Review (ICER)

On October 4, 2018, ICER released a clinical report entitled, "Inotersen and patisiran for Hereditary Transthyretin Amyloidosis: Effectiveness and Value". ICER recommendations are as follows:¹³

- ICER judges the clinical evidence for patisiran to be "incremental" or "better".
- On average, patients on patisiran demonstrated improvement in neuropathy symptoms, as measured by the mNIS + 7. Based on the current body of evidence, there is moderate certainty of a substantial net health benefit with high certainty of at least a small net health benefit compared to best supportive care.

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.

Amvuttra® (vutrisiran) is a transthyretin-directed small interfering RNA indicated for the treatment of the polyneuropathy of hereditary transthyretin-mediated amyloidosis in adults and for cardiomyopathy of wild-type or hereditary transthyretin-mediated amyloidosis in adults to reduce cardiovascular mortality, cardiovascular hospitalizations, and urgent heart failure visits.

Onpattro® (patisiran) contains a transthyretin-directed small interfering RNA and is indicated for the treatment of the polyneuropathy of hereditary transthyretin-mediated amyloidosis in adults.

References

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16. Institute for Clinical and Economic Review: Final Evidence Report - Disease Modifying Therapies for the Treatment of Transthyretin Amyloid Cardiomyopathy (ATTR-CM). October 21, 2024.

Policy History/Revision Information

Date	Summary of Changes
04/01/2026	<ul style="list-style-type: none"> • Retired policy; Louisiana plan membership disenrolled on Apr. 1, 2026
10/01/2025	<p>Coverage Rationale</p> <ul style="list-style-type: none"> • Removed language indicating Amvuttra (vutrisiran) and Onpattro (patisiran) are unproven and not medically necessary for the treatment of transthyretin (ATTR)-mediated amyloidosis with cardiomyopathy (ATTR-CM) • Added language to indicate Amvuttra (vutrisiran) is medically necessary for the treatment of cardiomyopathy of wild-type (wtATTR) or hereditary transthyretin-mediated (hATTR) amyloidosis in patients who meet all of the following criteria: <ul style="list-style-type: none"> Initial Therapy <ul style="list-style-type: none"> ○ Diagnosis of wild-type or hereditary ATTR-mediated amyloidosis with cardiomyopathy (ATTR-CM) ○ Documentation of one of the following: <ul style="list-style-type: none"> ▪ The patient has a pathogenic <i>TTR</i> mutation (e.g., V30M) ▪ Cardiac or noncardiac tissue biopsy demonstrating histologic confirmation of ATTR amyloid deposits ▪ All of the following: <ul style="list-style-type: none"> – Echocardiogram or cardiac magnetic resonance imaging suggestive of amyloidosis – Radionuclide imaging (99mTc-DPD, 99mTc-PYP, or 99m Tc-HMDP) showing grade 2 or 3 cardiac uptake (may require prior authorization and notification) – Absence of light chain amyloidosis ○ Patient has New York Heart Association (NYHA) Functional Class I, II, or III heart failure ○ Physician attests that the patient has an N-terminal pro-B-type natriuretic peptide (NT-proBNP) level that, when combined with signs and symptoms, is considered definitive for a diagnosis of ATTR-CM ○ Documentation of one of the following: <ul style="list-style-type: none"> ▪ History of heart failure, with at least one prior hospitalization for heart failure ▪ Presence of signs and symptoms of heart failure (e.g., dyspnea, edema) ○ One of the following: <ul style="list-style-type: none"> ▪ Documentation of progressive worsening or an event resulting in hospitalization related to hATTR-mediated amyloidosis with cardiomyopathy (ATTR-CM) while on one of the following: <ul style="list-style-type: none"> – Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis) – Attriby (acoramidis) ▪ Documentation of intolerance or contraindication to both of the following: <ul style="list-style-type: none"> – Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis) – Attriby (acoramidis) ○ Patient is not receiving Amvuttra in combination with any of the following: <ul style="list-style-type: none"> ▪ RNA interference agents [e.g., Onpattro (patisiran), Wainua (eplintersen), Tegsedi (inotersen)]

Date	Summary of Changes
	<ul style="list-style-type: none"> ▪ Transthyretin stabilizers [e.g., Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis)] <ul style="list-style-type: none"> ○ Prescribed by or in consultation with a cardiologist ○ Dosing is in accordance with the U.S. Food and Drug Administration (FDA) prescribing information ○ Initial authorization is for no more than 12 months <p>Continuation of Therapy</p> <ul style="list-style-type: none"> ○ Patient has previously received treatment with Amvuttra for the treatment of wtATTR or hATTR amyloidosis ○ Documentation that the patient has experienced a positive clinical response to Amvuttra (e.g., improved symptoms, quality of life, slowing of disease progression, decreased hospitalizations, etc.) ○ Documentation that the patient continues to have New York Heart Association (NYHA) Functional Class I, II, or III heart failure ○ Patient is not receiving Amvuttra in combination with any of the following: <ul style="list-style-type: none"> ▪ RNA interference agents [e.g., Onpattro (patisiran), Wainua (eplontersen), Tegsedi (inotersen)] ▪ Transthyretin stabilizers [e.g., Vyndaqel (tafamidis meglumine) or Vyndamax (tafamidis)] ○ Prescribed by or in consultation with a cardiologist ○ Dosing is in accordance with the U.S. FDA prescribing information ○ Authorization is for no more than 12 months <p>Applicable Codes</p> <ul style="list-style-type: none"> ● Added ICD-10 diagnosis codes E85.0, E85.4, and E85.82 <p>Supporting Information</p> <ul style="list-style-type: none"> ● Updated <i>Background</i>, <i>Clinical Evidence</i>, <i>FDA</i>, and <i>References</i> sections to reflect the most current information ● Archived previous policy version CSLA2024D0072M

Instructions for Use

This Medical Benefit Drug Policy provides assistance in interpreting UnitedHealthcare standard benefit plans. When deciding coverage, the federal, state or contractual requirements for benefit plan coverage must be referenced as the terms of the federal, state or contractual requirements for benefit plan coverage may differ from the standard benefit plan. In the event of a conflict, the federal, state or contractual requirements for benefit plan coverage govern. Before using this policy, please check the federal, state or contractual requirements for benefit plan coverage. 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.

UnitedHealthcare may also use tools developed by third parties, such as the InterQual® criteria, to assist us in administering health benefits. The 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.