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MEDICAL POLICY – 5.01.530

Egrifta WR (tesamorelin)

Effective Date:	Jun. 1, 2025	RELATED MEDICAL POLICIES:
Last Revised:	May 12, 2025	None
Replaces:	N/A	

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Introduction

Lipodystrophy is a condition that affects how the body processes and stores fat. It can cause either a buildup or a loss of fat. Up to one half of people with HIV experience lipodystrophy. It's not known whether it's the virus or the drugs used to treat the virus that causes lipodystrophy. Egrifta WR is a drug that can be used to reduce the excess fat in the belly area in people with HIV. There have been no studies looking at whether Egrifta WR improves long-term heart health. Because of this and the lack of other scientific evidence about how it affects overall health, the use of Egrifta WR in those with HIV is considered cosmetic. All other uses are investigational (unproven).

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	Cosmetic
Egrifta WR (tesamorelin)	**Use of Egrifta WR (tesamorelin) for the reduction of excess abdominal fat in HIV-infected individuals with lipodystrophy is considered cosmetic.**

Drug	Investigational
Egrifta WR (tesamorelin)	**All other uses of Egrifta WR (tesamorelin) not outlined above are considered investigational at this time.** **Egrifta WR (tesamorelin) is subject to the product's US Food and Drug Administration (FDA) dosage and administration prescribing information.**

Coding

Code	Description
HCPCS	
J3490	Unclassified drugs (use to report: Egrifta WR)

Note: CPT codes, descriptions and materials are copyrighted by the American Medical Association (AMA). HCPCS codes, descriptions and materials are copyrighted by Centers for Medicare Services (CMS).

Related Information

N/A

Evidence Review

Description

Egrifta (tesamorelin) is a growth hormone releasing factor (GRF) analog FDA-approved on November 10, 2010, for the reduction of excess abdominal fat in human immunodeficiency (HIV)–infected individuals with lipodystrophy. Growth hormone-releasing factor (GRF), also known as growth hormone-releasing hormone (GHRH), is a 44-amino acid peptide that is released from the hypothalamus and binds to receptors on pituitary cells (somatotropes) that release growth hormone (GH). The interaction of GRF with its somatotrope receptors stimulates GH release. Tesamorelin is a synthetic growth GRF analogue consisting of the 44 amino acid sequence of human GRF with a hexenoyl moiety (a 6-carbon chain with a double bond at position 3) attached at the N-terminal. The hexenoyl moiety slows degradation of Egrifta (tesamorelin), increasing its half-life. Tesamorelin is self-administered as a daily subcutaneous injection.

Disease Characteristics

The human immunodeficiency virus (HIV) lipodystrophy syndrome is characterized by fat loss (lipoatrophy) in the limbs, buttocks and face; localized fat accumulation (lipohypertrophy) at the back of the neck ("buffalo hump"), the upper trunk and breasts, around the abdominal viscera, and in lipomata; hyperlipidemia; insulin resistance; and hyperglycemia. Before the availability of antiretroviral therapy, severe wasting and decreased cholesterol levels were common metabolic abnormalities in advanced acquired immunodeficiency syndrome (AIDS). With the introduction of effective antiretroviral therapy, central lipohypertrophy with peripheral lipoatrophy was seen more commonly. Individuals can have pure lipoatrophy, pure lipohypertrophy, or a mixture of both morphologic features.

Epidemiology and Risk Factors

A 2005 cohort study in the United States followed 452 HIV-infected individuals for 1 year to assess risk factors for progression of morphologic abnormalities. Baseline prevalence rates were 35% for lipoatrophy, 44% for lipohypertrophy, and 14% for mixed morphology. Risk factors for lipohypertrophy included female sex and higher levels of body fat and serum triglyceride at baseline. Additional risk factors include use of protease inhibitors (e.g., ritonavir and indinavir), increasing age, longer duration of antiretroviral therapy (ART), which may be a surrogate for longer duration of HIV infection, and a low-fiber diet. In contrast, risk factors for lipoatrophy include low baseline triceps skin-fold values, smaller hips, higher HIV ribonucleic acid (RNA)

levels, and use of the nucleoside reverse transcriptase inhibitors (NRTIs), abacavir or stavudine. The findings suggest that lipoatrophy and lipohypertrophy are different syndromes.

Pathophysiology

Lipodystrophy is thought to result from mitochondrial toxicity and derangements in adipocytokines (adipose derived proteins) that regulate energy homeostasis, including those that regulate growth hormone (GH). In HIV seropositive or seronegative obese individuals, the amount of GH secreted per burst is decreased, and the response to growth hormone-releasing factor (GF) and arginine stimulation testing is blunted. Additionally, deficiency of adiponectin and elevation of leptin are seen in HIV lipohypertrophy. Adiponectin is an adipocyte-derived hormone that functions as an insulin sensitizer by reducing triglyceride levels and inhibiting gluconeogenesis in the liver. Leptin is a hormone involved in the central regulation of energy homeostasis and insulin resistance. In HIV-infected individuals, leptin levels seem to correlate with body fat phenotype, with the lowest levels seen in individuals with lipoatrophy, and the highest levels, consistent with a state of leptin resistance, in individuals with lipohypertrophy.

Clinical Presentation

Abdominal lipohypertrophy reflects an excess of visceral adipose tissue (VAT) that results in an increased abdominal girth. "Buffalo hump" results from an accumulation of fat in the dorsocervical area. Both men and women may develop accumulation of extra breast fat or symmetrical deposits of subcutaneous fat nodules on their trunk and extremities (lipomata). In many cases, it may be difficult to distinguish the syndrome from simple obesity, in which visceral fat is also accompanied by increased amounts of subcutaneous fat in the abdominal wall. The distinguishing feature of HIV-associated lipodystrophy is that visceral fat deposition is accompanied by normal or decreased (but not increased) amounts of subcutaneous fat. These body changes are distressing and can be stigmatizing for sufferers.

To assess 10-year coronary heart disease (CHD) risk estimates in individuals with HIV lipodystrophy, 91 HIV-infected individuals were compared with 273 HIV-seronegative controls from the Framingham Offspring Study who were matched for age, gender and body mass index.

Ten-year risk estimates were significantly higher in HIV-infected individuals with evidence of fat redistribution compared to matched controls (7.4% ± 0.6% vs. 5.3% ± 0.3%, p=0.002). When these individuals were matched to controls for waist-to-hip ratio measurements, the CHD risk was similar (7.6% ± 0.6% vs. 7.6% ± 0.4%; p=0.9). The CHD risk estimate was greatest in HIV-

infected individuals who had primary lipoatrophy (9.2% ± 1.8%) compared with those who had either abdominal lipohypertrophy (4.3% ± 0.7%, $p<0.05$) or mixed fat redistribution (7.6% ± 0.8%, p-value not reported). HIV-infected individuals without fat redistribution did not have a greater CHD risk estimate than did controls (4.1% ± 0.7% vs. 3.3% ± 0.3%, $p=0.27$). The authors conclude that "although CHD risk is increased in HIV-infected individuals with fat redistribution, the pattern of fat distribution is a potential important component in determining the risk in this population."

Diagnosis

There are a wide range of screening techniques for abdominal obesity. A simple measure is the waist circumference, which is considered more accurate than the waist circumference: hip circumference ratio. An abdominal circumference >102 cm for men and >88 cm for women is considered abnormal. Quantitative measurements of visceral adipose tissue by computed tomography (CT) or magnetic resonance imaging (MRI) are usually done in research trials only. Dual-energy x-ray absorptiometry (DEXA) cannot distinguish between visceral and subcutaneous adipose tissue and is not recommended.

Principal Therapeutic Options

Potential interventions for lipohypertrophy include exercise, surgical interventions, and medical therapy. Both aerobic and resistance exercise should be encouraged for HIV-infected individuals with central or generalized fat accumulation. Moderate exercise is well tolerated by HIV-infected individuals and does not increase viral load. In a small study of resistance exercise training in HIV-infected individuals who complained of increased abdominal girth, there was a significant decline in total fat, measured by dual-energy x-ray absorptiometry (DEXA), and the majority of fat loss occurred in the truncal region.

Medical therapies for lipohypertrophy, predominantly abdominal, include metformin, somatropin and pioglitazone. Each of these alternatives is associated with significant and potentially serious side effects.

Rationale

Efficacy

The clinical development program of Egrifta (tesamorelin) included 2 randomized, placebo-controlled, double-blind, Phase III pivotal trials conducted in the US, Canada, and Europe. The design of the trials was similar. Each consisted of a 26-week Main Phase followed by a 26-week Extension Phase. A total of 816 HIV-infected adults with clinically-defined lipodystrophy receiving antiretroviral therapy for at least 8 weeks were enrolled. Individuals with diabetes mellitus requiring medication were excluded. Individuals self-administered tesamorelin (Egrifta) 2 mg or placebo subcutaneously daily. The primary efficacy outcome was percent reduction in visceral adipose tissue (VAT) as measured by single-slice CT scan at the L4-L5 level. The minimum clinically meaningful reduction was defined a priori as 8%. Secondary endpoints included effects on lipid profile and individual-reported outcomes related to belly image. The FDA questioned the content validity of the scale used to assess the individual-reported outcomes. Individuals who completed the Main Phase trials were invited to enroll in the Extension Phases (total n=578). Individuals in the tesamorelin group were randomized to continue tesamorelin (Egrifta) or switch to placebo. Individuals in the placebo group were switched to tesamorelin (Egrifta).

In one main phase trial, Trial 10, VAT in the Egrifta (tesamorelin) group decreased by 18% compared to an increase of 2% in the placebo group (mean treatment difference [95% confidence interval (CI)] -20% [-24% to -15%]). In the other Main Phase trial, Trial 11, VAT in the Egrifta (tesamorelin) group decreased by 14% compared to a decrease of 2% in the placebo group (mean treatment difference [95% CI] -12% [-16% to -7%]). In the Extension Phase trials, reductions in VAT were maintained in individuals that continued Egrifta (tesamorelin) for an additional 26 weeks and reverted back toward baseline in individuals that switched to placebo.

In Trial 10, mean triglyceride level decreased by 48 mg/dL in the Egrifta (tesamorelin) group and increased by 5 mg/dL in the placebo group, a statistically significant difference ($p<0.001$). In Trial 11, mean triglyceride level decreased by 19 mg/dL in the Egrifta (tesamorelin) group and increased by 1 mg/dL in the placebo group, a difference that was not statistically significant ($p=0.10$). Similarly, reductions in total cholesterol: high-density lipoprotein (HDL) cholesterol ratio and non-HDL cholesterol were statistically significant in Trial 10 but not in Trial 11. In the Extension Phase trials, Egrifta (tesamorelin) effects on triglyceride levels were variable and of similar magnitude to the Main Phase trial effects.

Individual-reported outcomes related to belly image were inconsistent across trials. The mean between-group difference in improvement from baseline belly size evaluation to week 26 was

1.5 points in Trial 10 (p=0.75) and 2.9 points in Trial 11 (p=0.21). Responder criteria required an improvement of 50 points. For belly appearance distress, the mean between-group difference in improvement was 5.4 points in Trial 10 (p=0.08) and 3.1 points in Trial 11 (p=0.02). Responder criteria required an improvement of 25 points.

The effect of VAT reduction on cardiovascular (CV) risk reduction was not a requirement for FDA-approval of Egrifta (tesamorelin). Instead, clinical correlations with secondary supportive efficacy measures of improved individual-reported body image and lipid abnormalities were expected. FDA did not consider the observed changes in these measures to be robust and concludes that the clinical benefit of the observed reductions in VAT is uncertain.

Safety

The most common adverse events seen with Egrifta (tesamorelin) treatment in clinical trials were:

- Hypersensitivity reactions (e.g., rash, urticaria);
- Adverse events associated with the use of growth hormone (e.g., arthralgia, peripheral edema, hyperglycemia, carpal tunnel syndrome) which occurred in 25.6% of Egrifta (tesamorelin) individuals and 13.7% of placebo individuals during the Main Phase trials; and
- Injection site reactions (erythema, pruritis, pain, urticaria, irritation, swelling, hemorrhage) which occurred in 24.5% of Egrifta (tesamorelin) individuals and 14.4% of placebo individuals overall.

Hypersensitivity

Hypersensitivity reactions occurred in 27 of 740 Egrifta (tesamorelin)-treated individuals (3.6%) compared with 1 of 263 placebo-treated individuals (0.4%). These reactions included pruritus, erythema, flushing, urticaria, and other rash. Anaphylaxis was not observed. Of these 27 individuals, 23 (85%) tested positive for anti- Egrifta (tesamorelin) antibodies, compared to a seropositive rate of approximately 50% for the pivotal trial population as a whole. The presence or titer of anti- Egrifta (tesamorelin) antibodies did not appear to correlate with the clinical severity of the hypersensitivity reaction.

Glucose Intolerance

During the Main Phase trials and their extensions, there were no clinically significant changes in mean values for fasting blood glucose, fasting serum insulin, homeostasis model assessment of insulin resistance (HOMA-IR), or glycosylated hemoglobin (HbA1c). However, during the Main Phase trials, the proportion of individuals with HbA1c in the diabetic range (≥6.5%) increased from 2.1% at baseline to 6.6% at week 26 in the Egrifta (tesamorelin) group and from 1.2% to 2.5% in the placebo group, a statistically significant difference (odds ratio [95% confidence interval] 3.6 [1.5, 12.0]).[1, 2] In the Extension Phase trials, the proportion of individuals with HbA1c in the diabetic range decreased from 4.9% at week 26 to 1.5% at week 52 in the T-T group, and from 5.2% to 4.3% in the T-P group.

Increased IGF-1 and Cancer Risk

In Phase III trials, 15 individuals developed cancer, including 8 individuals during the Main Phase trials and 7 individuals during the Extension Phase trials. No specific pattern of cancers differentiated Egrifta (tesamorelin) from placebo. Because of the suspected link between high insulin-like growth factor (IGF)-1 levels and the risk of tumorigenesis, IGF-1 values for all individuals who developed cancer during clinical trials were reviewed. Twelve of 15 IGF-1 values (80%) fell within the normal range (not more than 2 standard deviations [SDs] from mean normal). Three individuals had IGF-1 levels above the upper limit of normal. Basal cell carcinoma developed in 2 of these individuals and lung cancer in the third.

Conclusion

The primary efficacy outcome was percent reduction in VAT as determined by abdominal computed tomography (CT) scan. Significant reductions in VAT that exceeded the agreed-upon minimum clinically meaningful threshold (8% reduction) were demonstrated with Egrifta (tesamorelin) in both phase 3 trials. The effect of VAT reduction on cardiovascular (CV) risk reduction was not a requirement for FDA-approval of Egrifta (tesamorelin). Instead, clinical correlations with secondary supportive efficacy measures of improved individual-reported body image and lipid abnormalities were expected. The changes in these measures were not robust or consistent. Therefore, the clinical benefit of the observed reductions in VAT is uncertain.

The assumed efficacy of tesamorelin in reducing risk of CV events is therefore based on population-based risk analyses that assume that reducing VAT in HIV lipodystrophy individuals

will produce risk profiles comparable to those of non-HIV infected individuals with similar VAT. The available evidence does not adequately demonstrate that this assumption is warranted.

Although Egrifta (tesamorelin) is FDA-approved for the reduction of excess abdominal fat in HIV-infected individuals with lipodystrophy, an improvement in net health outcome has not been demonstrated. Specifically:

- Long-term cardiovascular benefit has not been studied.
- Long-term risks of elevated IGF-1 levels are unknown.
- There are no data to support improved compliance with antiretroviral therapies.
- Effects on quality of life measures were not assessed.
- Individual-reported outcomes related to belly image were inconsistent across trials.

Given the relatively low prevalence of HIV-lipodystrophy and of CV event rates, FDA considers a randomized trial to assess CV outcomes infeasible. Instead, post-marketing requirements include a long-term observational study to assess long-term risks associated with Egrifta (tesamorelin) including major adverse cardiac events (MACE), cancer risk, and hypersensitivity reactions and a double-blind, randomized, placebo-controlled clinical trial of HIV-infected individuals with lipodystrophy and type 2 diabetes mellitus to assess the risk of retinopathy.

2012 Update

A literature search of the MEDLINE database conducted from January 2011 through February 2012 did not identify any additional published studies that would prompt reconsideration of the policy statements.

2013 Update

A literature search of the MEDLINE database conducted from January through December 2012 did not identify any additional published studies that would prompt reconsideration of the policy statements.

2014 Update

A literature search conducted from January 2013 through February 2014 found no new evidence that would change this policy.

2015 Update

A literature search conducted from January 2014 through February 2015 found no new evidence that would change this policy.

2016 Update

A literature search conducted from June 1, 2015, through December 6, 2016, found no new evidence that would change this policy.

2017 Update

A literature search conducted from June 1, 2016, through August 11, 2017, found no new evidence that would change this policy.

2018 Update

A literature search conducted from July 1, 2017, through June 12, 2018, found no new evidence that would change this policy.

2019 Update

Reviewed Egrifta (tesamorelin) prescribing information and conducted a literature search from June 1, 2018, through July 20, 2019. No new evidence found that would change this policy.

2020 Update

Reviewed Egrifta SV (tesamorelin) prescribing information. No new evidence found that would change this policy. Updated policy and drug name from Egrifta (tesamorelin) to Egrifta SV (tesamorelin). Egrifta SV has different dosage, administration, and storage requirements than Egrifta. Egrifta is no longer available.

2021 Update

Reviewed Egrifta SV (tesamorelin) prescribing information. No new evidence found that would change this policy.

2022 Update

Reviewed Egrifta SV (tesamorelin) prescribing information and the management of lipodystrophy in HIV-infected individuals. No new evidence found that would change this policy.

2023 Update

Reviewed Egrifta SV (tesamorelin) prescribing information. No new evidence found that would change this policy.

2024 Update

Reviewed Egrifta SV (tesamorelin) prescribing information and the management of lipodystrophy in HIV-infected individuals. No new evidence found that would change this policy.

2025 Update

Reviewed Egrifta SV (tesamorelin) prescribing information and the management of lipodystrophy in HIV-infected individuals. Clarified that the medications listed in this policy are subject to the product's FDA dosage and administration prescribing information.

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History

Date	Comments
03/08/11	Add to Prescription Drug Section - New Policy. Reviewed and recommended by P&T January 2011.
04/25/12	Replace policy. Policy updated with literature review; no changes to policy statements.
04/16/13	Replace policy. Policy updated with literature review; no changes to policy statements. Reference 31 added.
04/14/14	Annual Review. Policy updated with literature review; no changes to policy statements. Reference 32 added.

Date	Comments
04/24/15	Annual review. Policy updated with literature review; no changes to policy statements.
01/01/17	Annual review, approved December 13, 2016. Policy updated with literature review; no changes to policy statements.
09/01/17	Annual Review, approved August 22, 2017. Title changed to Egrifta (tesamorelin). Policy updated with literature review; no changes to policy statements.
07/01/18	Annual Review, approved June 22, 2018. Policy updated with literature review; no changes to policy statements.
09/01/19	Annual Review, approved August 6, 2019. Policy updated with literature review; no changes to policy statements.
10/01/20	Annual Review, approved September 1, 2020. Updated policy and drug name listed in policy from Egrifta (tesamorelin) to Egrifta SV (tesamorelin). Egrifta is no longer available. No changes to policy statements.
09/01/21	Annual Review, approved August 3, 2021. No changes to policy statement.
11/01/22	Annual Review, approved October 10, 2022. No changes to policy statement. Changed the wording from "patient" to "individual" throughout the policy for standardization.
06/01/23	Annual Review, approved May 22, 2023. Reviewed Egrifta SV (tesamorelin) prescribing. No new evidence found that would change this policy.
07/01/24	Annual Review, approved June 24, 2024. No changes to policy statement.
04/01/25	Annual Review, approved March 24, 2025. Clarified that the medications listed in this policy are subject to the product's FDA dosage and administration prescribing information.
06/01/25	Interim Review, approved May 12, 2025. Updated policy title from Egrifta SV (tesamorelin) to Egrifta WR (tesamorelin). Changed drug name listed in policy from Egrifta SV to Egrifta WR.

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.