MEDICAL POLICY

POLICY TITLE	LIPID APHERESIS
POLICY NUMBER	MP-4.024

Effective Date:	11/1/2022

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I. POLICY

Low-density lipoprotein (LDL) apheresis may be considered **medically necessary** in patients with homozygous familial hypercholesterolemia as an alternative to plasmapheresis.

LDL apheresis may be considered **medically necessary** in patients with heterozygous familial hypercholesterolemia (FH) who have failed diet therapy and maximum tolerated combination drug***** therapy **AND** who meet the following FDA-approved indications (all LDL levels represent the best achievable LDL level after a program of diet and drug therapy):

- Functional hypercholesterolemic heterozygotes with LDL greater than or equal to 300 mg/dl;
- Functional hypercholesterolemic heterozygotes with LDL greater than or equal to 200 mg/dl AND documented coronary artery disease. *****

LDL apheresis is considered **investigational** for other uses, including nonfamilial hypercholesterolemia, sudden sensorineural hearing loss, severe diabetic foot ulcerations, peripheral artery disease, preeclampsia, and non–arteritic acute anterior ischemic optic neuropathy, as there is insufficient evidence to support a conclusion concerning the health outcomes or benefits associated with this procedure.

Therapeutic apheresis with selective high-density lipoprotein (HDL) delipidation and plasma reinfusion is considered **investigational,** for all indications, including but not limited to acute coronary syndrome.as there is insufficient evidence to support a conclusion concerning the health outcomes or benefits associated with this procedure.

** For definitions of maximum tolerated drug therapy and documented coronary artery disease, please see Policy Guidelines section.*

POLICY GUIDELINES

A scientific statement from American Heart Association for the treatment of heterozygous familial hypercholesterolemia (FH) has indicated that adults should be treated with available pharmacotherapy with an initial goal of reducing low-density lipoprotein cholesterol (LDL-C) by at least 50%, usually with a statin. This treatment can be followed by achieving an LDL-C of less than 100 mg/dL (absent coronary artery disease [CAD] or other major risk factors]) or 70 mg/dL

(presence of CAD or other major risk factors). The following approach for pharmacotherapy is suggested:

- High-intensity statin therapy to target greater than 50% LDL-C reduction, such as rosuvastatin or atorvastatin.
- If the patient is adherent and LDL-C is above the target goal after 3 months, consider adding ezetimibe.
- If the patient is adherent and LDL-C is above the target goal after 3 months, consider adding a PCSK9 inhibitor or colesevelam (or other bile acid sequestrant or niacin).
- If the patient is adherent and LDL-C is above the target goal after 3 months, proceed to complex therapy combination such as a 4-drug combination plus LDL apheresis.

Documented CAD includes a history of myocardial infarction, coronary artery bypass surgery, percutaneous transluminal coronary angioplasty or alternative revascularization procedure, or progressive angina documented by exercise or nonexercise stress test.

The frequency of LDL apheresis varies, but typically averages once every 2 weeks to obtain an interapheresis level of LDL-C at less than 120 mg/dL. Patients with homozygous FH may be treated more frequently. Patients are simultaneously treated with diet and drug therapy.

Cross-reference:
MP 4.031 Plasma Exchange

II. PRODUCT VARIATIONS TOP

This policy is only applicable to certain programs and products administered by Capital Blue Cross and subject to benefit variations as discussed in Section VI. Please see additional information below.

FEP PPO - Refer to FEP Medical Policy Manual. The FEP Medical Policy manual can be found at: https://www.fepblue.org/benefit-plans/medical-policies-and-utilization-management-guidelines/medical-policies

III. DESCRIPTION/BACKGROUND TOP

Hyperlipidemia

A dominantly inherited disorder, familial hypercholesterolemia results from a variant in the gene that encodes for the specific cell surface receptor responsible for low-density lipoprotein (LDL) uptake by the cells. The heterozygous form affects about 1 in 500 people. The number of LDL receptors is halved in this condition, resulting in serumLDL cholesterol levels that are approximately 2 to 3 times levels considered acceptable (ie, greater than 300 mg/dL). Affected male patients typically develop coronary heart disease in their thirties and forties, while women develop the disease in their fifties. Depending on the patient, heterozygous familial hypercholesterolemia may or may not respond adequately to lipid-lowering drugs.

Homozygous hypercholesterolemia is rare, occurring in only in 1 in 1 million subjects. Due to the total lack of functioning LDL receptors, serum levels of LDL cholesterol may be elevated 6-fold (>500 mg/dL). Homozygotes may develop severe aortic stenosis and coronary heart disease by 20 years of age. These patients typically do not adequately respond to drug or diet modification therapies. In the past, patients with homozygous familial hypercholesterolemia may have been treated with plasma exchange, but the advent of LDL apheresis provides a more targeted approach by permitting selective removal of LDL from plasma.

Treatment

Low-Density Lipoprotein

LDL apheresis (also referred to as lipid apheresis) involves the extracorporeal removal of apolipoprotein B (apo B)-containing lipoproteins, including LDL, lipoprotein(a), and very low-density lipoprotein.

The apheresis procedure is designed to isolate plasma. The LDLs are then selectively removed from the plasma by immunoadsorption, heparin-induced extracorporeal LDL precipitation, dextran sulfate adsorption, or double-filtration plasma pheresis of lipoprotein. In immunoadsorption, polyclonal antihuman apo B antibodies from sheep selectively bind and remove LDL, because apo B is the protein moiety of LDL. In heparin-induced extracorporeal LDL precipitation, LDL and other particles containing apo B are precipitated by heparin at an acidic pH. Dextran sulfate adsorption removes LDL by binding the positively charged apo B to dextran sulfate particles bound to cellulose.

High-Density Lipoprotein

Therapeutic apheresis with selective HDL delipidation and plasma reinfusion removes plasma from the body, processed through a delipidation device, and then returned to the patient. The delipidation procedure selectively removes cholesterol from HDL, converting the major α-HDL to pre-β-like HDL, a form of HDL that enhances cholesterol transport to the liver and is thought to reduce atherosclerosis development and burden. The plasma with pre-β-like HDL is then reinfused into the patient.

Regulatory Status

Two LDL apheresis systems have been approved by the U.S. Food and Drug Administration (FDA) for marketing. In 1996, the Liposorber LA-15® System (Kaneka Pharma), dextran sulfate device, was approved by the FDA through the premarket approval process for use to "acutely remove LDL-C from the plasma of high-risk patient populations for whom diet has been ineffective or not tolerated."

In 1997, the HELP® System (B. Braun), a heparin-induced extracorporeal LDL precipitation, was approved by the FDA through the premarket approval process for the same indication. FDA product code: MMY.

In 2013, the Liposorber LA-15® System was approved for additional indications through the humanitarian device exemption process for the treatment of pediatric patients with primary focal segmental glomerulosclerosis when the following conditions apply:

- "Standard treatment options, including corticosteroid and/or calcineurin inhibitor treatments, are unsuccessful or not well-tolerated, and the patient has a GFR [glomerular filtration rate] greater than or equal to 60 mL/min/1.73 m^2 or
- The patient is post renal transplantation."

No devices have been approved by the FDA specifically for HDL delipidation. The Lipid Sciences Plasma Delipidation System-2 (Lipid Sciences) was tested in clinical studies, but the company ceased business operations in 2012.

IV. RATIONALE

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SUMMARY OF EVIDENCE

Familial Hypercholesterolemia

For individuals with homozygous FH and unable to achieve target LDL-C with maximally tolerated pharmacotherapy who receive LDL apheresis, the evidence includes multiple nonrandomized prospective and retrospective small cohort studies and a systematic review. Relevant outcomes are overall survival, disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. Studies have reported reductions in LDL-C levels after apheresis, with means ranging from 57% to 75%. Currently, the direct evidence does not demonstrate that reductions in LDL-C levels seen with LDL apheresis will reduce adverse cardiovascular events. RCTs comparing drug therapy alone, apheresis alone, no intervention, usual care, or apheresis plus drug therapy are not feasible, and unlikely to resolve any clinical uncertainty because lipid apheresis is generally used as a treatment of last resort when maximally tolerated pharmacotherapy has failed to achieve target LDL-C levels. The evidence is sufficient to determine that the technology results in a meaningful improvement in the net health outcome.

For individuals with heterozygous FH and unable to achieve target LDL-C with maximally tolerated pharmacotherapy who receive LDL apheresis, the evidence includes multiple nonrandomized prospective and retrospective small cohort studies as well as a systematic review.

Relevant outcomes are overall survival, disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. Studies have reported reductions in LDL-C levels after apheresis with means ranging from 58% to 63%. Currently, there is no direct evidence that reductions in LDL-C levels seen with LDL apheresis will reduce adverse cardiovascular events. RCTs comparing drug therapy alone, apheresis alone, no intervention, usual care, or apheresis plus drug therapy are not feasible, and unlikely to resolve any clinical uncertainty because lipid apheresis is generally used as a treatment of last resort when maximally tolerated pharmacotherapy has failed to achieve target LDL-C levels. The evidence is sufficient to determine that the technology results in a meaningful improvement in the net health outcome.

Nonfamilial Hypercholesterolemia

For individuals with non-FH who receive LDL apheresis, the evidence includes multiple retrospective and prospective nonrandomized cohort studies. Relevant outcomes are overall survival, disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. These studies have reported improvements in lipid levels pre- and post treatment. Randomized trials in patient populations that are well-characterized regarding previous treatments, lipid levels, and comorbidities are necessary to demonstrate improvements in health outcomes. The evidence is insufficient to determine the effects of the technology on health outcomes.

Nephrotic Syndrome

For individuals with treatment-resistant nephrotic syndrome who receive LDL apheresis, the evidence includes multiple nonrandomized prospective and retrospective cohort studies. Relevant outcomes are symptoms, change in disease status, and treatment-related morbidity. Using variable schedules of LDL apheresis with short-term follow-up, these studies have reported that LDL apheresis may improve proteinuria and lipid abnormalities in patients with steroid-resistant nephrotic syndrome. Additional studies with concurrent controls and longer term follow-up are necessary to determine whether outcomes are improved with the use of LDL apheresis in nephrotic syndrome. The evidence is insufficient to determine the effects of the technology on health outcomes.

Other Indications

For individuals with sudden sensorineural hearing loss who receive LDL and fibrinogen apheresis, the evidence includes 2 RCTs. Relevant outcomes are symptoms, change in disease status, and treatment related morbidity. One RCT compared LDL apheresis with the standard treatment of prednisolone, hydroxyethyl starch, and pentoxifylline; it reported no statistically significant differences in hearing recovery between groups. The second RCT compared the combination of a single lipid apheresis procedure plus standard treatment with standard treatment alone; it reported statistically significant differences in hearing recovery with the addition of apheresis to standard treatment. An a priori primary end point, power calculations, and the statistical plan to control for type I error for multiple comparisons were not reported in the second trial. Further evaluation and replication of these findings are required given the inconsistent reporting. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals with severe diabetic foot ulcerations who receive LDL apheresis, the evidence includes a single prospective case series. Relevant outcomes are symptoms, change in disease status, morbid events, and treatment-related morbidity. In the case series, patients underwent from 1 to 7 treatment procedures and were followed for 2 to 73 months. Authors reported improved wound healing and reductions in the risk of lower leg amputations but results were insufficient to ascertain the effects on outcomes. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals with peripheral artery disease who receive LDL apheresis, the evidence includes a single prospective case series. Relevant outcomes are change in disease status and treatment-related morbidity. Improvements in symptomatic parameters such as coldness,

numbness, and resting pain were reported, but insufficient to ascertain the effects on outcomes. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals with preeclampsia who receive LDL apheresis, the evidence includes a prospective case series. Relevant outcomes are overall survival, disease-specific survival, change in change in disease status, morbid events, and treatment-related morbidity. Improvements in gestation were reported, but insufficient to ascertain the effects on outcomes. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals with non–arteritic acute anterior ischemic optic neuropathy who receive LDL apheresis, the evidence includes a prospective case series. Relevant outcomes are symptoms, change in change in disease status, and treatment-related morbidity. Improvement in visual outcomes was reported, but insufficient to ascertain the effects on outcomes. The evidence is insufficient to determine the effects of the technology on health outcomes.

Acute Coronary Syndrome

For individuals with acute coronary syndrome who receive selective high-density lipoprotein (HDL) delipidation and plasma reinfusion, the evidence includes an RCT. Relevant outcomes are overall mortality, disease-specific survival, change in change in disease status, morbid events, and treatment related morbidity. Results have shown improvements in certain biochemical measures (e.g., pre-β-like HDL and α-HDL levels). There were no significant changes in atheroma volume. Larger randomized trials, with longer follow-up and clinically relevant outcomes, are needed to determine the impact of delipidated HDL plasma on acute coronary syndrome. The evidence is insufficient to determine the effects of the technology on health outcomes.

V. DEFINITIONS

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ADSORPTION refers to a natural process whereby molecules of a gas or liquid adhere to the surface of a solid.

HEMOLYTIC UREMIC SYNDROME is a rare kidney disorder marked by renal failure, microangiopathic hemolytic anemia, and platelet deficiency.

IDIOPATHIC THROMBOCYTOPENIA PURPURA is a deficiency of platelets that results in bleeding into the skin and other organs.

PATHOGENIC MEANS capable of causing or producing a disease.

PHERESIS refers to the removal of blood or other body fluids from a patient, separating certain elements (e.g., immunoglobulins, platelets, or red blood cells) and reinfusing the remaining elements into the patient.

PLASMA refers to the watery straw-colored fluid part of the lymph and the blood in which the leukocytes, erythrocytes, and platelets are suspended. Plasma is made up of water, electrolytes, proteins, glucose, fats, bilirubin, and gases and is essential for carrying the cellular elements of the blood through circulation, transporting nutrients, maintaining the acid-base balance of the body, and transporting wastes from the tissue.

PLATELET refers to the smallest cells in the blood, essential for coagulation and for hemostasis.

VI. BENEFIT VARIATIONS

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The existence of this medical policy does not mean that this service is a covered benefit under the member's health benefit plan. Benefit determinations should be based in all cases on the applicable health benefit plan language. Medical policies do not constitute a description of benefits. A member's health benefit plan governs which services are covered, which are excluded, which are subject to benefit limits, and which require preauthorization. There are different benefit plan designs in each product administered by Capital Blue Cross. Members and providers should consult the member's health benefit plan for information or contact Capital Blue Cross for benefit information.

VII. DISCLAIMER

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Capital Blue Cross's medical policies are developed to assist in administering a member's benefits, do not constitute medical advice and are subject to change. Treating providers are solely responsible for medical advice and treatment of members. Members should discuss any medical policy related to their coverage or condition with their provider and consult their benefit information to determine if the service is covered. If there is a discrepancy between this medical policy and a member's benefit information, the benefit information will govern. If a provider or a member has a question concerning the application of this medical policy to a specific member's plan of benefits, please contact Capital Blue Cross' Provider Services or Member Services. Capital Blue Cross considers the information contained in this medical policy to be proprietary and it may only be disseminated as permitted by law.

VIII. CODING INFORMATION

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Note: This list of codes may not be all-inclusive, and codes are subject to change at any time. The identification of a code in this section does not denote coverage as coverage is determined by the terms of member benefit information. In addition, not all covered services are eligible for separate reimbursement.

Investigational; therefore, not covered:

Procedure Codes								
0342T								

Covered when medically necessary:

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Procedure Codes								
36516	S2120							

ICD-10-CM Diagnosis Codes	Description
E78.01	Familial hypercholesterolemia

IX. REFERENCES

TOP

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21. *Blue Cross Blue Shield Association Medical Policy Reference Manual. 8.02.04 Lipid Apheresis, September 17,2021.(Archived)*

Other:

1. *Mosby's Medical, Nursing, & Allied Health Dictionary, 6th edition.*
2. *Taber's Cyclopedic Medical Dictionary, 20th edition.*

X. POLICY HISTORY

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MP 4.024	**CAC 02/25/2003**
	CAC 06/29/2004
	CAC 01/25/2005
	CAC 07/26/2005
	CAC 02/28/2006 Consensus
	CAC 06/27/2006
	CAC 07/25/2006
	CAC 07/31/2007
	CAC 05/27/2008
	CAC 05/26/2009 Consensus
	CAC 07/27/2010 Plasma exchange procedure removed and cross-reference to separate plasma exchange policy added.
	CAC 02/28/2012 Adopted BCBSA. Title changed to match BCBSA. Deleted information related to pheresis procedure, extracorporeal immunoadsorption using protein A Columns, since there is no longer U.S. sales activity. Policy statement unchanged related to low-density lipid apheresis. Remains medically necessary.
	CAC 06/04/2013 Consensus review. No changes to the policy statements. References updated. FEP variation added to refer to the FEP manual. No coding changes.
	CAC 03/25/2014 Consensus review. No changes to the policy statements. References updated. Rationale and additional guideline added.
	CAC 01/27/2015 Minor revision. Added policy statement indicating therapeutic apheresis with selective high-density lipoprotein (HDL) delipidaton and plasma reinfusion and plasma is considered investigational. Title changed to “Lipid Apheresis”. Background, rationale, and references updated. Medicare variation added for this review. Policy coded.
	CAC 01/26/2016 Consensus review. No changes to the policy statements. References and rationale updated. Coding reviewed.
	01/01/2017 Administrative update. Product variation section reformatted. New diagnosis code E78.01 added effective 10/01/2016
	CAC 05/23/2017 Consensus. No change to policy statements. References and rationale reviewed. Coding Reviewed.
	09/26/2017 Minor review. “6-month trial” removed from the second medically necessary policy statement. The investigational statement on LDL apheresis for other uses revised to include non-FH, sudden sensorineural hearing loss, severe diabetic foot ulcerations, peripheral artery disease, and non–arteritic acute anterior ischemic optic neuropathy. The Policy Guidelines section was revised by deleting the statement “Maximum tolerated drug therapy is defined as a trial of drugs from at least 2 separate classes of hypolipidemic agents such as bile acid sequestrants, HMG-CoA reductase inhibitors, fibric acid derivatives, or niacin/nicotinic acids.” Background, rationale and references updated. Coding reviewed

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	06/06/2018 Consensus review. The policy statement on high density lipoprotein apheresis considered investigational was clarified**.** Background and references updated. Rationale revised.
	06/11/2019 Consensus review. No change to policy statements. Background and references updated.
	06/25/2020 Consensus review. No changes to policy statements.
	10/22/2021 Consensus review. No change to policy statement. References reviewed and updated.
	07/21/2022 Consensus review. No change to policy statement. References reviewed and updated. Coding table format updated.

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