MASSACHUSETTS

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Medical Policy
Lipid Apheresis

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Policy Number: 465

BCBSA Reference Number: 8.02.04 (For Plan internal use only)
NCD/LCD: NA

Related Policies

Plasma Exchange, #466

Policy

Commercial Members: Managed Care (HMO and POS), PPO, and Indemnity Medicare HMO Blue[SM] and Medicare PPO Blue[SM] Members

Low-density lipoprotein (LDL) apheresis may be considered **MEDICALLY NECESSARY** in individuals with homozygous familial hypercholesterolemia as an alternative to plasmapheresis.

LDL apheresis may be considered **MEDICALLY NECESSARY** in individuals with heterozygous familial hypercholesterolemia who have failed diet therapy and maximum tolerated combination drug therapy* AND who meet the following U.S. Food and Drug Administration approved indications (All LDL levels represent the best achievable LDL level after a program of diet and drug therapy):

1. Functional hypercholesterolemic heterozygotes with LDL $\geq$ 300 mg/dL,
2. Functional hypercholesterolemic heterozygotes with LDL $\geq$ 200 mg/dL* AND documented coronary artery disease.*

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

A scientific statement from American Heart Association (Gidding et al [2015]) 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 >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.

LDL apheresis is considered **INVESTIGATIONAL** for other uses, including nonfamilial hypercholesterolemia, nephrotic syndrome, sudden sensorineural hearing loss, severe diabetic foot ulcerations, peripheral artery disease, preeclampsia, and non–arteritic acute anterior ischemic optic neuropathy.

Therapeutic apheresis with selective high-density lipoprotein delipidation and plasma reinfusion is considered **INVESTIGATIONAL** for all indications, including but not limited to acute coronary syndrome.

Prior Authorization Information

Inpatient

- For services described in this policy, precertification/preauthorization **IS REQUIRED** for all products if the procedure is performed **inpatient**.

Outpatient

- For services described in this policy, see below for products where prior authorization **might be required** if the procedure is performed **outpatient**.

	Outpatient
Commercial Managed Care (HMO and POS)	Prior authorization is **not required**.
Commercial PPO and Indemnity	Prior authorization is **not required**.
Medicare HMO Blue℠	Prior authorization is **not required**
Medicare PPO Blue℠	Prior authorization is **not required**

CPT Codes / HCPCS Codes / ICD Codes

Inclusion or exclusion of a code does not constitute or imply member coverage or provider reimbursement. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage as it applies to an individual member.

Providers should report all services using the most up-to-date industry-standard procedure, revenue, and diagnosis codes, including modifiers where applicable.

The following codes are included below for informational purposes only; this is not an all-inclusive list.

The above medical necessity criteria MUST be met for the following codes to be covered for Commercial Members: Managed Care (HMO and POS), PPO and Indemnity:

CPT Codes

CPT codes:	Code Description
36516	Therapeutic apheresis; with extracorporeal immunoadsorption, selective adsorption or selective filtration and plasma reinfusion

HCPCS Codes

HCPCS codes:	Code Description

S2120	Low density lipoprotein (LDL) apheresis using heparin-induced extracorporeal LDL precipitation

The following ICD Diagnosis Codes are considered medically necessary when submitted with the CPT, ICD Procedure, and HCPCS codes above if <u>medical necessity criteria</u> are met:

ICD-10 Diagnosis Codes

ICD-10-CM Diagnosis codes:	Code Description
E78.01	Familial hypercholesterolemia
I25.10	Atherosclerotic heart disease of native coronary artery without angina pectoris
I25.110	Atherosclerotic heart disease of native coronary artery with unstable angina pectoris
I25.111	Atherosclerotic heart disease of native coronary artery with angina pectoris with documented spasm
I25.112	Atherosclerosic heart disease of native coronary artery with refractory angina pectoris
I25.118	Atherosclerotic heart disease of native coronary artery with other forms of angina pectoris
I25.119	Atherosclerotic heart disease of native coronary artery with unspecified angina pectoris
I25.700	Atherosclerosis of coronary artery bypass graft(s), unspecified, with unstable angina pectoris
I25.701	Atherosclerosis of coronary artery bypass graft(s), unspecified, with angina pectoris with documented spasm
I25.702	Atherosclerosis of coronary artery bypass graft(s), unspecified, with refractory angina pectoris
I25.708	Atherosclerosis of coronary artery bypass graft(s), unspecified, with other forms of angina pectoris
I25.709	Atherosclerosis of coronary artery bypass graft(s), unspecified, with unspecified angina pectoris
I25.710	Atherosclerosis of autologous vein coronary artery bypass graft(s) with unstable angina pectoris
I25.711	Atherosclerosis of autologous vein coronary artery bypass graft(s) with angina pectoris with documented spasm
I25.712	Atherosclerosis of autologous vein coronary artery bypass graft(s) with refractory angina pectoris
I25.718	Atherosclerosis of autologous vein coronary artery bypass graft(s) with other forms of angina pectoris
I25.719	Atherosclerosis of autologous vein coronary artery bypass graft(s) with unspecified angina pectoris
I25.720	Atherosclerosis of autologous artery coronary artery bypass graft(s) with unstable angina pectoris
I25.721	Atherosclerosis of autologous artery coronary artery bypass graft(s) with angina pectoris with documented spasm
I25.722	Atherosclerosis of autologous artery coronary artery bypass graft(s) with refractory angina pectoris
I25.728	Atherosclerosis of autologous artery coronary artery bypass graft(s) with other forms of angina pectoris
I25.729	Atherosclerosis of autologous artery coronary artery bypass graft(s) with unspecified angina pectoris
I25.730	Atherosclerosis of nonautologous biological coronary artery bypass graft(s) with unstable angina pectoris
I25.731	Atherosclerosis of nonautologous biological coronary artery bypass graft(s) with angina pectoris with documented spasm

I25.732	Atherosclerosis of nonautologous biological coronary artery bypass graft(s) with refractory angina pectoris
I25.738	Atherosclerosis of nonautologous biological coronary artery bypass graft(s) with other forms of angina pectoris
I25.739	Atherosclerosis of nonautologous biological coronary artery bypass graft(s) with unspecified angina pectoris
I25.750	Atherosclerosis of native coronary artery of transplanted heart with unstable angina
I25.751	Atherosclerosis of native coronary artery of transplanted heart with angina pectoris with documented spasm
I25.752	Atherosclerosis of native coronary artery of transplanted heart with refractory angina pectoris
I25.758	Atherosclerosis of native coronary artery of transplanted heart with other forms of angina pectoris
I25.759	Atherosclerosis of native coronary artery of transplanted heart with unspecified angina pectoris
I25.760	Atherosclerosis of bypass graft of coronary artery of transplanted heart with unstable angina
I25.761	Atherosclerosis of bypass graft of coronary artery of transplanted heart with angina pectoris with documented spasm
I25.762	Atherosclerosis of bypass graft of coronary artery of transplanted heart with refractory angina pectoris
I25.768	Atherosclerosis of bypass graft of coronary artery of transplanted heart with other forms of angina pectoris
I25.769	Atherosclerosis of bypass graft of coronary artery of transplanted heart with unspecified angina pectoris
I25.790	Atherosclerosis of other coronary artery bypass graft(s) with unstable angina pectoris
I25.791	Atherosclerosis of other coronary artery bypass graft(s) with angina pectoris with documented spasm
I25.792	Atherosclerosis of other coronary artery bypass graft(s) with refractory angina pectoris
I25.798	Atherosclerosis of other coronary artery bypass graft(s) with other forms of angina pectoris
I25.799	Atherosclerosis of other coronary artery bypass graft(s) with unspecified angina pectoris
I25.810	Atherosclerosis of coronary artery bypass graft(s) without angina pectoris
I25.811	Atherosclerosis of native coronary artery of transplanted heart without angina pectoris
I25.812	Atherosclerosis of bypass graft of coronary artery of transplanted heart without angina pectoris

The following CPT code is considered investigational for <u>Commercial Members: Managed Care (HMO and POS), PPO, Indemnity, Medicare HMO Blue and Medicare PPO Blue:</u>

CPT Codes

CPT codes:	Code Description
0342T	Therapeutic apheresis with selective HDL delipidation and plasma reinfusion

Description

Hyperlipidemia

A dominantly inherited disorder, familial hypercholesterolemia (FH) 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 serum LDL cholesterol levels that are approximately 2 to 3 times levels considered acceptable (ie, > 300 mg/dL). Affected male individuals typically develop coronary heart disease (CHD) in their thirties and forties, while women develop the disease in their fifties. Depending on the patient, heterozygous FH may or may not respond adequately to lipid-lowering drugs.

Homozygous hypercholesterolemia is rare, occurring in only 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 CHD by 20 years of age. These individuals typically do not adequately respond to drug or diet modification therapies. In the past, individuals with homozygous FH 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
Low-density lipoprotein 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 plasmapheresis 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 (HDL) delipidation and plasma reinfusion removes plasma from the body, processed through a delipidation device, and then returns it 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.

Summary
This use of low-density lipoprotein (LDL) apheresis has been proposed to treat various types of familial hypercholesterolemia (FH) and other significant hyperlipidemia and to reduce atherosclerosis in cardiovascular disease. Lipid apheresis discriminately removes LDL particles from plasma while leaving other factors intact, allowing the filtrated plasma to be returned to the patient.

Summary of Evidence
Familial Hypercholesterolemia
For individuals with homozygous FH who are unable to achieve target LDL cholesterol (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 (OS), 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. Randomized controlled trials (RCTs) comparing drug therapy alone, apheresis alone, no intervention, usual care, or apheresis plus drug therapy are not feasible and are 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 an improvement in the net health outcome.

For individuals with heterozygous FH who are 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 OS , 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. Randomized controlled trials comparing drug therapy alone, apheresis alone, no intervention, usual care, or apheresis plus drug therapy are not feasible and

are 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 an 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 OS, disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. These studies have reported improvements in lipid levels pretreatment and posttreatment. 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 that the technology results in an improvement in the net health outcome.

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 individuals 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 that the technology results in an improvement in the net health outcome.

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 endpoint, 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 that the technology results in an improvement in the net health outcome.

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, individuals 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 that the technology results in an improvement in the net health outcome.

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 that the technology results in an improvement in the net health outcome.

For individuals with preeclampsia who receive LDL apheresis, the evidence includes a prospective case series. Relevant outcomes are OS, disease-specific survival, 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 that the technology results in an improvement in the net health outcome.

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 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 that the technology results in an improvement in the net health outcome.

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 OS disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. Results have shown improvements in certain biochemical measures (eg, 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 that the technology results in an improvement in the net health outcome.

For individuals with acute coronary syndrome who receive LDL apheresis, the evidence includes RCT. Relevant outcomes are OS, disease-specific survival, change in disease status, morbid events, and treatment-related morbidity. Results revealed a nonsignificant improvement in the mean LDL reduction and percentage change in total plaque volume in the intensive-lipid lowering group (including apheresis) as compared to standard therapy with statins alone. Larger randomized trials, with longer follow-up and clinically relevant outcomes, are needed to determine the impact of LDL apheresis on acute coronary syndrome. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

Policy History

Date	Action
10/2022	Clarified coding information.
4/2022	Clarified coding information and indicated that prior authorization is not required for all products.
1/2022	Clarified prior authorization information
7/2021	Annual policy review. Description, summary, and references updated. Policy statements unchanged.
7/2020	Annual policy review. Description, summary, and references updated. Policy statements unchanged.
6/2019	Annual policy review. Description, summary, and references updated. Policy statements unchanged.
7/2018	Annual policy review. Investigational policy statement on high density lipoprotein apheresis clarified. 7/2018
1/2018	Clarified coding information.
10/2017	Annual policy review. “6-month trial” removed from the second medically necessary policy statement. New investigational indications described. Effective 10/1/2017.
10/2016	Clarified coding information.
11/2015	Annual policy review. New references added.
1/2015	Annual policy review. New investigational indications added. Title changed to Lipid Apheresis. Coding information clarified. Effective 1/1/2015.
6/2014	Updated Coding section with ICD10 procedure and diagnosis codes, effective 10/2015.
4/2014	Coding information clarified.
12/2013	Annual policy review. New references added.
11/2011-4/2012	Medical policy ICD 10 remediation: Formatting, editing and coding updates. No changes to policy statements.
7/2011	Reviewed - Medical Policy Group - Hematology and Oncology. No changes to policy statements.

4/2011	Reviewed - Medical Policy Group - Cardiology and Pulmonology. No changes to policy statements.
9/2010	Reviewed - Medical Policy Group - Hematology and Oncology. No changes to policy statements.
4/2010	Reviewed - Medical Policy Group - Cardiology and Pulmonology. No changes to policy statements.
9/2009	Reviewed - Medical Policy Group - Hematology and Oncology. No changes to policy statements.
5/2009	Annual policy review. New references added.
4/2009	Reviewed - Medical Policy Group - Cardiology and Pulmonology. No changes to policy statements.
11/2008	Annual policy review. New references added. Changes to policy statement.
10/2008	Reviewed - Medical Policy Group - Hematology and Oncology. No changes to policy statements.
4/2008	Reviewed - Medical Policy Group - Cardiology and Pulmonology. No changes to policy statements.
9/2007	Reviewed - Medical Policy Group - Hematology and Oncology. No changes to policy statements.
4/2007	Reviewed - Medical Policy Group - Cardiology and Pulmonology. No changes to policy statements.

Information Pertaining to All Blue Cross Blue Shield Medical Policies

Click on any of the following terms to access the relevant information:
Medical Policy Terms of Use
Managed Care Guidelines
Indemnity/PPO Guidelines
Clinical Exception Process
Medical Technology Assessment Guidelines

References

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