OVERVIEW
Lipoprotein-associated phospholipase A2 (Lp-PLA2), is an enzyme that hydrolyzes phospholipids and is primarily associated with low-density lipoproteins (LDLs). Accumulating evidence has suggested that Lp-PLA2 is a biomarker of coronary artery disease (CAD) and may have a proinflammatory role in the progression of atherosclerosis.

MEDICAL CRITERIA
Not applicable

PRIOR AUTHORIZATION
Not applicable

POLICY STATEMENT
BlueCHiP for Medicare and Commercial Products
Measurement of lipoprotein-associated phospholipase A2 (Lp-PLA2) is considered not medically necessary as there is insufficient peer-reviewed scientific literature that demonstrates that the service is effective.

COVERAGE
Benefits may vary between groups/contracts. Please refer to the appropriate Benefit Booklet, Evidence of Coverage, or Subscriber Agreement for limitations of benefits/coverage when services are not medically necessary.

BACKGROUND
Low-density lipoproteins (LDL) have been identified as the major atherogenic lipoproteins and have long been identified by the National Cholesterol Education Project (NCEP) as the primary target of cholesterol-lowering therapy. LDL particles consist of a surface coat composed of phospholipids, free cholesterol, and apolipoproteins, surrounding an inner lipid core composed of cholesterol ester and triglycerides. Traditional lipid risk factors such as low-density lipoprotein-cholesterol (LDL-C), while predictive on a population basis, are weaker markers of risk on an individual basis. Only a minority of subjects with elevated LDL and cholesterol levels will develop clinical disease, and up to 50% of cases of coronary artery disease (CAD) occur in subjects with ‘normal’ levels of total and LDL cholesterol. Thus, there is considerable potential to improve the accuracy of current cardiovascular risk prediction models.

Lipoprotein-associated phospholipase A2 (Lp-PLA2), also known as platelet-activating factor acetylhydrolase, is an enzyme that hydrolyzes phospholipids and is primarily associated with LDLs. Accumulating evidence has suggested that Lp-PLA2 is a biomarker of CAD and may have a proinflammatory role in the progression of atherosclerosis. The recognition that atherosclerosis represents, in part, an inflammatory process has created considerable interest in measurement of proinflammatory factors as part of cardiovascular disease risk assessment.

The evidence for lipoprotein-associated phospholipase A2 (Lp-PLA2) testing in patients who have a risk of cardiovascular disease (CVD) includes studies of analytic validity and studies of the association of Lp-PLA2 and various coronary artery disease outcomes. Outcomes of interest include overall survival, diseases specific...
survival, and test validity. The studies demonstrate that Lp-PLA2 levels are an independent predictor of CVD. To improve outcomes, clinicians must have the tools to incorporate Lp-PLA2 test results into existing risk prediction models, and these models should demonstrate improved classification into risk categories that will improve treatment and health outcomes. Direct evidence for improved health outcomes with the use of Lp-PLA2 in clinical practice is lacking. Although Lp-PLA2 levels are associated with CVD risk, changes in patient management that would occur as a result of obtaining Lp-PLA2 levels in practice are not well-defined. The evidence is insufficient to determine the effects of the technology on health outcomes. Therefore, this service is not medically necessary for BlueCHiP for Medicare and Commercial products as there is insufficient peer-reviewed scientific literature that demonstrates that the service is effective.

CODING
BlueCHiP for Medicare and Commercial Products
The following code is considered not medically necessary:
83698

RELATED POLICIES
Novel Biomarkers in Risk Assessment and Management of Cardiovascular Disease

PUBLISHED
Provider Update, January 2017
Provider Update, October 2015
Provider Update, January 2014
Provider Update, December 2011
Provider Update, January 2011
Provider Update, August 2009
Provider Update, November 2008

REFERENCES


