Ultrasonographic Measurement of Carotid Intimal-Medial Thickness as an Assessment of Subclinical Atherosclerosis

Policy # 00251
Original Effective Date: 02/17/2010
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Applies to all products administered or underwritten by Blue Cross and Blue Shield of Louisiana and its subsidiary, HMO Louisiana, Inc.(collectively referred to as the "Company"), unless otherwise provided in the applicable contract. Medical technology is constantly evolving, and we reserve the right to review and update Medical Policy periodically.

Note: Computed Tomography to Detect Coronary Calcification is addressed in medical policy 00031.

Services Are Considered Investigational
Coverage is not available for investigational medical treatments or procedures, drugs, devices or biological products.

Based on review of available data, the Company considers ultrasonographic measurement of carotid intimal-medial thickness (CIMT) as a technique of identifying subclinical atherosclerosis for use in the screening, diagnosis or management of atherosclerotic disease to be **investigational.***

Background/Overview

Coronary heart disease (CHD) accounts for 30.8% of all deaths in the United States. Established major risk factors for CHD have been identified by the National Cholesterol Education Program (NCEP) Expert Panel. These risk factors include elevated serum levels of low-density lipoprotein cholesterol (LDL-C), and total cholesterol, and reduced levels of high-density lipoprotein cholesterol (HDL-C). Other risk factors include a history of cigarette smoking, hypertension, family history of premature CHD, and age.

The third report of the NCEP Adult Treatment Panel (ATP III) established various treatment strategies to modify the risk of CHD, with emphasis on target goals of LDL-C. Pathology studies have demonstrated that levels of traditional risk factors are associated with the extent and severity of atherosclerosis. ATP III recommended use of the Framingham criteria to further stratify those patients with 2 or more risk factors for more intensive lipid management. However, at every level of risk factor exposure, there is substantial variation in the amount of atherosclerosis, presumably related to genetic susceptibility and the influence of other risk factors. Thus, there has been interest in identifying a technique that can improve the ability to diagnose those at risk of developing CHD, as well as to measure disease progression, particularly for those at intermediate risk.

The carotid arteries can be well-visualized by ultrasonography, and ultrasonographic measurement of the CIMT has been investigated as a technique to identify and monitor subclinical atherosclerosis. B-mode ultrasound is most commonly used to measure CIMT. The intima-media thickness (IMT) is measured and averaged over several sites in each carotid artery. Imaging of the far wall of each common carotid artery yields more accurate and reproducible IMT measurements than imaging of the near wall. Two echogenic lines are produced, representing the lumen-intima interface and the media-adventitia interface. The distance between these 2 lines constitutes the IMT.

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FDA or Other Governmental Regulatory Approval

U.S. Food and Drug Administration (FDA)
In February 2003, SonoCalc®‡ (SonoSite) was cleared for marketing by the U.S. FDA through the 510(k) process. FDA determined that this software was substantially equivalent to existing image display products for use in the automatic measurement of the IMT of the carotid artery from images obtained from ultrasound systems. Subsequently, other devices have been cleared for marketing by FDA through the 510(k) process. Product code: LLZ.

Centers for Medicare and Medicaid Services (CMS)
There is no national coverage determination (NCD). In the absence of an NCD, coverage decisions are left to the discretion of local Medicare carriers.

Rationale/Source

Measurement of CIMT is primarily meant to assess risk for future disease, and therefore can be evaluated as a prognostic measure. Assessment of a prognostic measure typically focuses on 3 categories of evidence: (1) technical performance; (2) prognostic value (i.e., significant association between the test result and health outcomes); and (3) effect on health outcomes (i.e., demonstration that use of the prognostic information clinically can alter clinical management and/or improve health outcomes compared with patient management without use of the prognostic tool). In some cases, it is important to evaluate whether the test provides incremental information above the standard workup to determine whether the test has utility in clinical practice.

The literature on use of CIMT for cardiac risk stratification consists of numerous cohort studies and systematic reviews of these cohort studies. The following review includes the largest prospective cohort studies and the most important systematic reviews of these studies. A summary of the key literature follows.

PROGNOSTIC VALUE

Systematic Reviews
In 2010, Mookadam et al conducted a systematic review of the role of CIMT in predicting individual cardiovascular event risk and as a tool for assessing therapeutic interventions. Reviewers concluded that CIMT is an independent risk factor for cardiovascular events and may be useful in determining treatment when there is uncertainty regarding the approach or patient reluctance. However, they recommended further study to identify the best approaches to screening and interventions to prevent progression of atherosclerosis.

In a 2012 meta-analysis, the USE Intima-Media Thickness (USE-IMT) collaboration investigators sought to determine whether common CIMT measurements can assist in estimating the 10-year risk of first-time myocardial infarction (MI) or first-time stroke when added to the Framingham Risk Score. Using individual data for 45,828 patients from 14 population-based cohort studies, Den Ruijter et al found risk of first-time MI

or stroke was related positively to both the Framingham Risk Score and the adjusted common CIMT. The mean common CIMT was 0.73 mm, and it increased in every cohort with patient age during a median follow-up of 11 years. For every 0.1-mm difference in common CIMT, the hazard ratio (HR) for risk of MI or stroke, which occurred in 4007 patients, was 1.12 (95% confidence interval [CI], 1.09 to 1.14) for women and 1.08 (95% CI, 1.05 to 1.11) for men. However, adding common CIMT measurements to the Framingham Risk Score did not improve risk prediction and resulted in reclassification of risk in only 6.6% of patients. The added value of mean common CIMT in reclassifying risk was only 0.8% (95% CI, 0.1% to 1.6%) and did not differ between men and women. The C statistic of the Framingham Risk Score model with and without CIMT was similar between men (0.759; 95% CI, 0.752 to 0.766) and women (0.757; 95% CI, 0.749 to 0.764), suggesting the addition of CIMT in risk assessment offered limited benefit.

In another 2012 meta-analysis of individual participant data pooled from 16 studies (total N=36,984 patients), Lorenz et al examined CIMT progression from 2 ultrasound screenings taken 2 to 7 years apart (median, 4 years). Patients were followed for a mean of 7 years, during which time 1339 strokes, 1519 MI, and 2028 combined end points (MI, stroke, vascular death) occurred. Mean CIMT of the 2 ultrasound results was predictive of cardiovascular risk using the combined end point (adjusted HR=1.16; 95% CI 1.10 to 1.22). In sensitivity analyses, no associations were found between cardiovascular risk and individual CIMT progression regardless of CIMT definition, end point, and adjustments. As an example, for the combined end points, an increase of 1 SD in mean common CIMT progression resulted in an overall estimated HR of 0.97 (95% CI, 0.94 to 1.00) when adjusted for age, sex, and mean common CIMT; the HR was 0.98 (95% CI, 0.95 to 1.01) when adjusted for vascular risk factors. These data confirmed that CIMT is a predictor of cardiovascular risk, but did not demonstrate that changes in CIMT over time are predictive of future events.

A 2013 meta-analysis of 15 articles by van den Oord et al found similar results on the added value of CIMT. Six cohort studies (total N=32,299 patients) were evaluated to examine the predictive value of CIMT when added to traditional cardiovascular risk factors. While a CIMT increase of 0.1 mm was predictive for MI (HR=1.15; 95% CI, 1.12 to 1.18) and for stroke (HR=1.17; 95% CI, 1.15 to 1.21), the addition of CIMT did not statistically increase risk prediction over traditional cardiovascular risk factors (p=0.8).

Studies have found that including carotid plaques in CIMT measurements increases the predictive value of cardiovascular risk over CIMT assessed only in plaque-free sites. However, the meta-analysis by Lorenz found no difference in the main results between studies that included CIMT with carotid plaque and plaque-free CIMT. The 2012 systematic review by Peters et al found adding carotid plaque to the traditional CIMT model increased the C statistic from 0.01 to 0.06.

Prospective Cohort Studies
Numerous prospective cohort studies have evaluated the association between CIMT and future cardiovascular events. Some of the larger trials are discussed below.

In the Atherosclerosis Risk in Communities (ARIC) study, trialists evaluated risk factors associated with increased CIMT in 15,800 subjects. CIMT had a graded relation with increasing quartiles of plasma total cholesterol, LDL-C, and triglycerides. CIMT also correlated with the incidence of CHD in a subgroup of patients enrolled in the trial after 4 to 7 years of follow-up. Among the 12,841 subjects studied, there were 290 incident events. The HR rates for women and men, adjusted for age and sex, comparing extreme CIMT (i.e., ≥1 mm) with nonextreme CIMT (i.e., <1 mm), were 5.07 for women and 1.85 for men. The strength of the relation was reduced by including major CHD risk factors but remained elevated for higher measurements of CIMT. Authors concluded that mean CIMT was a noninvasive predictor of future CHD incidence.

The Rotterdam cohort study started in 1989 and recruited 7983 men and women ages 55 years and older. Its main objective was to investigate the prevalence and incidence of risk factors for chronic diseases, including cardiovascular disease (CAD), in elderly people. One aspect of the study sought to determine whether progression of atherosclerosis in asymptomatic elderly subjects is a prelude to cardiovascular events. Measurements of CIMT were used to assess the progression of atherosclerosis. Increasing CIMT was associated with increasing risks of stroke and MI.

O'Leary et al (1999) performed CIMT measurement on 4476 asymptomatic subjects ages 65 years or older without clinical CAD in the Cardiovascular Health Study. The incidence of cardiovascular events correlated with measurements of CIMT; this association remained significant after adjusting for traditional risk factors. Authors concluded that increases in CIMT were directly associated with an increased risk of MI and stroke in older adults without a history of CAD.

The longitudinal Carotid Atherosclerosis Progression Study (CAPS) included 4904 subjects. All subjects received a baseline CIMT measurement as well as traditional risk factor analysis, and were followed for 10 years (mean follow-up, 8.5 years; range, 7.1-10.0 years). Adverse events were MI in 73 (1.5%) patients, angina or MI in 271 (5.5%) patients, and death in 72 (1.5%) subjects. Lorenz et al (2010) retrospectively reviewed the CAPS data. They modeled the predictive value of CIMT on the cardiovascular adverse events within that decade. Because the thresholds of CIMT measurements that would lead to reclassification of risk are unknown, the authors used 24 models of reclassification and 5 statistical tests. Each model compared the predictive value of traditional risk factors alone with those risk factors with the addition of CIMT. None the reclassification models improved with the addition of CIMT measurements. They concluded that their retrospective analysis did not support use of CIMT as a clinically useful risk classification tool when used with traditional risk factor analysis.

In the Multi-Ethnic Study of Atherosclerosis (MESA) trial, an ongoing cohort study of atherosclerosis, CIMT was found to be a modestly better predictor of stroke but a worse predictor of CHD than coronary artery calcium (CAC) score at a median follow-up of 3.9 years among 6698 adults asymptomatic at baseline. In a 2010 article from the MESA trial, CIMT results in 4792 healthy, nondiabetic adults who were not on lipid-lowering medications were compared across 6 different lipid groups, including normolipemia and several types of common dyslipidemias. Mean CIMT values were increased only for the combined hyperlipidemia

(defined as any HDL-C level, LDL-C ≥160, and triglyceride ≥150) and simple hypercholesterolemia (defined as any HDL-C level, LDL-C ≥160, and triglyceride <150) groups. In another MESA report (2011) on 6760 patients with elevated high-sensitivity C-reactive protein (hsCRP) as defined by the JUPITER study, CIMT increases correlated with obesity but only mildly with hsCRP. A 2015 report from MESA trial of 6125 individuals with a family history of premature CHD identified 382 atherosclerotic CAD events at a mean follow-up of 10.2 years. The study found that CAC improved the risk estimation atherosclerotic CAD events but CIMT did not.

In the Bogalusa Heart Study (N=991 subjects), obesity along with overweight and elevated metabolic risk were associated with increased CIMT. In this study population, 41% of patients were found to have increased CHD risk. In the CARDIA study, clotting factor VII was associated with increases in CIMT in 1254 subjects. CIMT has also been used as a surrogate outcome measure in atherosclerosis treatment research studies.

In 2010, Raiko et al compared CAD risk scoring tools for identification of CHD risk to CIMT results in 2204 healthy adults, ages 24 to 39 years, from the Cardiovascular Risk in Young Finns study. The CAD risk scoring tools evaluated included the Framingham, Reynolds Risk Score, Systematic Coronary Risk Evaluation (SCORE), PROCAM, and FINRISK. In this population-based follow-up study, the authors found all CAD risk scores performed equally well in predicting subclinical atherosclerosis, as measured by high CIMT 6 years later.

The BioImage study enrolled 5808 asymptomatic individuals from the United States. All patients were evaluated by 3-dimensional carotid ultrasound and by CAC score, and followed for a mean of 2.7 years. The primary end point was major cardiovascular events, defined as cardiovascular death, MI, and ischemic stroke. Carotid plaque burden was an independent predictor of outcomes, with a HR of 2.36 (95% CI, 1.13 to 4.92) for individuals in the highest tertile. The coronary calcium score was also an independent predictor of outcomes, with HRs similar to carotid plaque. Both carotid plaque and CAC score led to significant net reclassification, with a net reclassification index of 0.23.

Section Summary: Prognostic Value
Evidence from large, prospective cohort studies has established that CIMT is an independent risk factor for CAD. However, systematic reviews have concluded that the ability of CIMT to reclassify patients into clinically relevant categories is modest and may not be clinically important. The uncertainty concerning the ability to reclassify patients into clinically relevant categories limits the potential for CIMT to improve health outcomes.

EFFECT ON HEALTH OUTCOMES
In a 2011 study by Johnson et al, 355 patients, ages 40 years with 1 or more CAD risk factors, received carotid ultrasound screenings to prospectively determine whether abnormal results would change physician and patient behaviors. Results were considered abnormal (when CIMT was >75th percentile or with the presence of carotid plaque) in 266 patients. Self-reported questionnaires were completed before the carotid

ultrasound, immediately after the ultrasound, and 30 days later to determine behavioral changes. Physician behavior in prescribing aspirin and cholesterol medication changed significantly ($p<0.001$ and $p<0.001$, respectively) after identification of abnormal carotid ultrasound results. Abnormal ultrasound results predicted reduced dietary sodium (odds ratio [OR], 1.45; $p=0.002$) and increased fiber intake (OR=1.55, $p=0.022$) in patients, but no other significant changes. Health outcomes were not evaluated in this study, and the short-term follow-up limits interpretation of results.

The evidence on reclassification of cardiovascular risk offers a potential chain of evidence to improve outcomes. If a measure is able to reclassify patients into risk categories that have different treatment approaches, then clinical management changes may occur that lead to improved outcomes. Because the ability to reclassify patients into clinically relevant categories with CIMT is modest at best, the clinical utility of this measure for reclassification is uncertain.

Section Summary: Effect on Health Outcomes
There is no direct evidence on the clinical utility of measuring CIMT for cardiac risk stratification. The available evidence on reclassification into clinically relevant categories does not indicate that use of CIMT will improve health outcomes.

SUMMARY OF EVIDENCE
For individuals who are undergoing cardiac risk assessment who receive ultrasonic measurement of CIMT, the evidence includes large cohort studies and systematic reviews. Relevant outcomes are test accuracy and morbid events. Some studies correlate increased CIMT with many other commonly used markers for risk of CHD and with risk for future cardiovascular events. A 2012 meta-analysis of individual participant data by Lorenz et al found that CIMT was associated with increased cardiovascular events although CIMT progression over time was not associated with increased cardiovascular event risk. In a 2012 systematic review by Peters et al, the added predictive value of CIMT was modest, and the ability to reclassify patients into clinically relevant categories was not demonstrated. The results from these reviews and other studies have demonstrated the predictive value of CIMT is uncertain, and that the predictive ability for any level of population risk cannot be determined with precision. In addition, available studies do not define how use of CIMT in clinical practice improves outcomes. There is no scientific literature that directly tests the hypothesis that measurement of CIMT results in improved patient outcomes and no specific guidance on how measurements of CIMT should be incorporated into risk assessment and risk management. The evidence is insufficient to determine the effects of the technology on health outcomes.

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02/04/2010	Medical Policy Committee approval
02/17/2010	Medical Policy Implementation Committee approval. New policy.
02/03/2011	Medical Policy Committee review
02/16/2011	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
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02/15/2012	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
02/07/2013	Medical Policy Committee review
02/20/2013	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
02/06/2014	Medical Policy Committee review
02/19/2014	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
01/01/2015	Coding Update
02/05/2015	Medical Policy Committee approval
02/18/2015	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
08/03/2015	Coding update: ICD10 Diagnosis code section added; ICD9 Procedure code section removed.
02/04/2016	Medical Policy Committee approval
02/17/2016	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
01/01/2017	Coding update: Removing ICD-9 diagnosis codes
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03/15/2017	Medical Policy Implementation Committee approval. Coverage eligibility unchanged.
03/01/2018	Medical Policy Committee review
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05/16/2018	Coding update

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Coding

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Code Type	Code
CPT	0126T, 93895
HCPCS	No codes
ICD-10 Diagnosis	Z13.6

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B. Whether the medical treatment, procedure, drug, device, or biological product requires further studies or clinical trials to determine its maximum tolerated dose, toxicity, safety, effectiveness, or effectiveness as compared with the standard means of treatment or diagnosis, must improve health outcomes, according to the consensus of opinion among experts as shown by reliable evidence, including:
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