

Policy Number	RAD604.009
Policy Effective Date	11/15/2025

Computed Tomography to Detect Coronary Artery Calcification

Table of Contents
Coverage
Policy Guidelines
Description
Rationale
Coding
References
Policy History

Related Policies (if applicable)
None

Disclaimer

Carefully check state regulations and/or the member contract.

Each benefit plan, summary plan description or contract defines which services are covered, which services are excluded, and which services are subject to dollar caps or other limitations, conditions or exclusions. Members and their providers have the responsibility for consulting the member's benefit plan, summary plan description or contract to determine if there are any exclusions or other benefit limitations applicable to this service or supply. **If there is a discrepancy between a Medical Policy and a member's benefit plan, summary plan description or contract, the benefit plan, summary plan description or contract will govern.**

Legislative Mandates

EXCEPTION: TEXAS contracts only: Texas House Bill 1290, effective September 1, 2009, bars excluding coverage for cardiac computed tomography scanning measuring coronary artery calcification (including screening for atherosclerosis and abnormal artery structure and/or function) performed once every five years. Patients must be:

- Male older than 45 years of age and younger than 76 years of age, or female older than 55 years of age and younger than 76 years of age, **AND**
 - Diabetic, **or**
 - At risk of developing coronary heart disease, based on a score derived from the Framingham Heart Study coronary prediction algorithm that is intermediate or higher.

EXCEPTION: NEW MEXICO contracts only: Effective January 1, 2021, New Mexico House Bill 126 provides coverage for a heart artery calcium scan for eligible insureds between 45 and 65 years of age who have an intermediate risk of developing coronary heart disease as determined by a health care provider based upon a score calculated from an evidence-based algorithm widely used in the medical community to assess a person's ten-year cardiovascular disease risk, including a score calculated using a pooled cohort equation. Coverage is required for the scan to be provided every five years if an eligible insured has previously received a heart artery calcium score of zero. Coverage is not required for further heart artery calcium scans if an eligible insured receives a heart artery calcium score greater than zero.

Coverage

The use of computed tomography to detect coronary artery calcification **is considered not medically necessary**.

Policy Guidelines

When quantitative assessment is performed as part of the same encounter as contrast-enhanced cardiac computed tomography (codes 75572-75573) or coronary computed tomography angiography (code 75574), it is included in the service.

The primary fast computed tomography methods for this determination are electron beam computed tomography and multidetector computed tomography.

Description

Several types of fast computed tomography (CT) imaging, including electron-beam CT and spiral CT, allow the quantification of calcium in coronary arteries. Coronary artery calcium (CAC) is associated with coronary artery disease (CAD). The use of CAC scores has been studied in the prediction of future risk of CAD and in the diagnosis of CAD in symptomatic individuals.

Coronary Artery Calcium

Coronary artery calcium is associated with CAD. The development of fast CT scanners has allowed the measurement of CAC in clinical practice. Coronary artery calcium has been evaluated in several clinical settings. The most widely studied indication is for the use of CAC in the prediction of future risk of CAD in patients with subclinical disease, with the goal of instituting appropriate risk-reducing therapy (e.g., statin treatment, lifestyle modifications) to improve outcomes. Also, CAC has been evaluated in patients with symptoms potentially consistent with CAD, but in whom a diagnosis is unclear.

Detection

Electron-beam computed tomography (EBCT; also known as ultrafast CT) and spiral CT (or helical CT) may be used as an alternative to conventional CT scanning due to faster throughput. In both methods, the speed of image acquisition gives them unique value for imaging a moving heart. The rapid image acquisition time virtually eliminates motion artifact related to cardiac contraction, permitting visualization of the calcium in the epicardial coronary arteries. Electron-beam computed tomography software permits quantification of calcium area and density, which are translated into calcium scores. Calcium scores have been investigated as a technique for detecting CAC, both as a diagnostic technique in symptomatic patients to rule out an atherosclerotic etiology of symptoms or, in asymptomatic patients, as an adjunctive method for risk stratification for CAD.

Electron-beam computed tomography and multidetector CT were initially the primary fast CT methods for measurement of CAC. A fast CT study for CAC measurement takes 10 to 15 minutes and requires only a few seconds of scanning time. More recently, computed tomography angiography has been used to assess coronary calcium. Because of the basic similarity between EBCT and computed tomography angiography in measuring coronary calcium, it is expected that computed tomography angiography provides information on coronary calcium that is similar to EBCT.

Computed tomography scan-derived coronary calcium measures have been used to evaluate coronary atherosclerosis. Coronary calcium is present in coronary atherosclerosis, but atherosclerosis detected may or may not be causing ischemia or symptoms. Coronary calcium measures may be correlated with the presence of critical coronary stenoses or serve as a measure of the patient's proclivity toward atherosclerosis and future coronary disease. Thus, coronary calcium could serve as a variable to be used in a risk assessment calculation to determine appropriate preventive treatment in asymptomatic patients. Alternatively, in other clinical scenarios, coronary calcium scores might help determine whether there is an atherosclerotic etiology or component to the presenting clinical problem in symptomatic patients, thus helping to direct further workup for the clinical problem. In this second scenario, a calcium score of 0 usually indicates that the patient's clinical problem is unlikely to be due to atherosclerosis and that other etiologies should be more strongly considered. In neither case does the test determine a specific diagnosis. Most clinical studies have examined coronary calcium for its potential use in estimating the risk of future coronary heart disease events.

Nomenclature

Coronary calcium levels can be expressed in many ways. The most common method is the Agatston score, which is a weighted summed total of calcified coronary artery area observed on CT. This value can be expressed as an absolute number, commonly ranging from 0 (low-risk) to 400 (high-risk). These values can be translated into age- and sex-specific percentile values. Different imaging methods and protocols will produce different values based on the specific algorithm used to create the score, but the correlation between any 2 methods appears to be high, and scores from 1 method can be translated into scores from a different method.

Regulatory Status

Many models of CT devices, including EBCT and other ultrafast CT devices, have been cleared for marketing by the U.S. Food and Drug Administration (FDA) through the 510(k) process. U.S. FDA product code: JAK.

Rationale

Medical policies assess whether a medical test is clinically useful. A useful test provides information to make a clinical management decision that improves the net health outcome. That is, the balance of benefits and harms is better when the test is used to manage the condition than when another test or no test is used to manage the condition.

The first step in assessing a medical test is to formulate the clinical context and purpose of the test. The test must be technically reliable, clinically valid, and clinically useful for that purpose. Medical policies assess the evidence on whether a test is clinically valid and clinically useful. Technical reliability is outside the scope of these reviews, and credible information on technical reliability is available from other sources.

Coronary Artery Calcium Scoring in Asymptomatic Individuals

Clinical Context and Test Purpose

The purpose of coronary artery calcium (CAC) scoring using computed tomography (CT) in asymptomatic individuals is to assess who may benefit from preventive interventions targeted to minimize the risk of atherosclerotic cardiovascular disease (ASCVD).

The following PICO was used to select literature to inform this policy.

Populations

The population of interest is individuals who are asymptomatic with risk of coronary artery disease (CAD).

Interventions

The intervention of interest is CAC scoring using fast CT imaging, including electron-beam computed tomography (EBCT) and spiral CT, in combination with standard risk stratification.

Coronary artery calcium scoring is usually initiated or used to modify cardiac risk-reduction interventions in individuals asymptomatic for CAD.

Comparators

The following tool is currently being used to make decisions about managing cardiovascular disease (CVD) in asymptomatic patients: CAD risk factor stratification based on standard risks, such as the Framingham Risk Score (FRS).

Outcomes

The outcomes of interest include overall survival (OS), test accuracy, test validity, morbid events (e.g., major adverse cardiac events [MACEs]), as well as the need for invasive coronary angiography (ICA) and revascularization.

Intermediate or surrogate outcomes of interest are changes in cardiac risk profile indicators such as smoking, hyperlipidemia, or hypertension.

Study Selection Criteria

For the evaluation of clinical validity of CAC scoring using CT, studies that meet the following eligibility criteria were considered:

- Reported on the accuracy of the marketed version of the technology;
- Included a suitable reference standard;

- Patient/sample clinical characteristics were described;
- Patient/sample selection criteria were described;
- The study reported on a minimum of 1000 patients.

Clinically Valid

A test must detect the presence or absence of a condition, the risk of developing a condition in the future, or treatment response (beneficial or adverse).

Systematic Reviews

Haq et al. (2024) conducted a systematic review and meta-analysis to evaluate whether CAC could predict the risk of cardiovascular events and mortality in adults aged less than 50 years. (1) Six observational studies (N=45,919) included patients with a mean age of 43.1 years. A CAC of 1 to 100 was associated with a higher risk of cardiovascular events compared to a zero CAC (hazard ratio [HR], 1.85; 95% confidence interval [CI], 1.08 to 3.16; $p=.012$; $I^2=65.5\%$) but there was no difference in mortality ($p=.2917$). Compared to a zero CAC, CAC greater than 100 was associated with a higher risk of cardiovascular events (HR, 6.57; 95% CI, 3.23 to 13.36; $p<.0001$; $I^2=72.6\%$) and a higher mortality risk (HR, 2.91; 95% CI, 2.23 to 3.80; $p<.0001$).

Bell et al. (2022) evaluated the incremental gain of CAC scoring in addition to traditional cardiovascular risk assessments for primary prevention in a systematic review and meta-analysis of cohort studies. (2) Six studies (N=17,961) were included. Mean patient age ranged from 50 to 75.1 years; 38.4% to 59.4% of patients in each study were women and 38% to 100% were White. The C statistic for the traditional CVD risk assessments ranged from 0.693 (95% CI, 0.661 to 0.726) to 0.80. The addition of CAC scoring resulted in a gain of 0.036 (95% CI, 0.020 to 0.052). When CAC score reclassified low risk patients to intermediate or high risk, 85.5% to 96.4% of patients did not have a CVD event during follow-up (range, 5.1 to 10 years). Of those originally classified as high risk and reclassified as low risk after CAC scoring, 91.4% to 99.2% did not have a CVD event during follow-up. Although the CAC score did add some additional discrimination to traditional CVD risk assessment, the authors cautioned that costs, rates of incidental findings, and radiation risks may offset the benefit.

Sarwar et al. (2009) conducted a systematic review and meta-analysis to examine the prognostic utility of CAC scoring in categorizing asymptomatic patients according to their risk for adverse events. (3) Thirteen studies assessing the relation between CAC and adverse cardiovascular outcomes (N=71,595 asymptomatic patients; 65% men) were included in the analysis. Among the participants, 29,312 (41%) did not have any evidence of CAC (range, 22% to 80% of patients per study). During a mean follow-up of 50 months (range, 32 to 102 months), 154 (0.47%) of 29,312 patients without CAC and 1749 (4.14%) of 42,283 patients with CAC had cardiovascular events. The pooled relative risk was 0.15 (95% CI, 0.11 to 0.21; $p<.001$).

Observational Studies

From a pool of 27,125 patients who had had coronary computed tomography angiography (CCTA) for CAD, Han et al. (2018) evaluated 3145 asymptomatic elderly patients between 52 and 62 years of age to compare the prognostic value of CCTA and CAC score. (4) In this

multicenter, prospective, observational study, the authors found that adding CCTA improved the level of discrimination of a model that only included FRS and CAC score (C statistic: 0.75 vs. 0.70; $p=.015$). The authors did not correlate the potential impact of CCTA results with treatment choices and downstream events. The study had a relatively short follow-up, and substantial disparity in the duration of risk prediction, FRS in particular.

Numerous observational studies have used data available from the Multi-Ethnic Study of Atherosclerosis (MESA) cohort to evaluate CAC in patients asymptomatic for CVD. (5) The MESA cohort of 6814 asymptomatic men and women 45 to 84 years of age is designed to study the characteristics of subclinical CVD and the risk factors that predict progression to symptomatic CVD. Approximately 38% of the patients in MESA were White, 28% African American, 22% Hispanic, and 12% Asian. Cainzos-Achirica et al. (2020) assessed whether use of CAC improved appropriate aspirin use for primary prevention compared with other risk calculators. (6) In multivariable regression analysis, a CAC score ≥ 100 was independently associated with an increased risk of CVD events compared with those with a CAC score of 0 (HR, 3.9; 95% CI, 2.5 to 6.1). The pooled cohort equations and an estimated cardiovascular risk threshold of $>20\%$ failed to identify optimal candidates for aspirin; however, a CAC score of at least 100 was able to identify subgroups of patients where aspirin would yield benefit. Gepner et al. (2017) prospectively compared the use of CAC with carotid plaque scores in order to predict CVD, coronary heart disease (CHD), and stroke/transient ischemic attack (TIA) events. (7) After 11.3 years of follow-up among 4955 participants (mean age, 61.6 years), 709 CVD, 498 CHD, and 262 stroke/TIA events had occurred. Coronary artery calcium score significantly reclassified non-CVD events (3%; 95% CI, 2% to 5%) and CHD events (13%; 95% CI, 5% to 18%). Carotid plaque score did not consistently reclassify CVD or CHD events or nonevents. Budoff et al. (2018) evaluated the relationship between CAC and incident ASCVD (stroke, cardiovascular death, or nonfatal myocardial infarction [MI]). (8) After a median follow-up of 11.1 years, there were 498 total CHD events in the cohort (7.3%). Results were stratified by categories of race/ethnicity, age, sex, and education. Event rates increased with increasing CAC levels across all demographic subgroups and tests for interaction with age, sex, or race/ethnicity were all non-significant, demonstrating that CAC was independently associated with events. Event rates in the CAC=0 group ranged from 1.3% to 5.6%, and in the CAC >300 group ranged from 13.1% to 25.6%. Blaha et al. (2016) evaluated the accuracy of change in risk classification by calculating the net reclassification improvement (NRI) for each of the 13 negative risk markers. (9) During a median of 10.3 years of follow-up among a cohort of 6814, 710 CVD events occurred. Among all the negative risk markers, a CAC score of 0 was the strongest, with an adjusted mean diagnostic likelihood ratio of 0.41 for all CHD. Net reclassification improvement for downward reclassification (10-year CVD risk, $<7.5\%$) of CVD events with CAC scores of 0 in participants with a pretest 10-year CVD risk of 7.5% or higher ($n=3833$ [3227 participants without events and 606 with events]) was 0.14, higher than other negative risk markers included in the study. Polonsky et al. (2010) also used data from MESA to determine whether incorporation of calcium score into a risk model based on traditional risk factors would improve the classification of risk. (10) During a median of 5.8 years of follow-up among a final cohort of 5878, 209 CHD events occurred, of which 122 were MI, death from CHD, or resuscitated cardiac arrest. Addition of CAC score in the model resulted in significant improvements in risk prediction

compared with the model without CAC score (NRI, 0.25; 95% CI, 0.16 to 0.34; $p<.001$). Subjects reclassified to high-risk had a similar risk of CHD events as those originally classified as high-risk.

In 2017, Ferencik et al. evaluated whether the distribution of CAC in individual coronary arteries and segments, as well as CAC in the proximal dominant coronary artery, as detected by cardiac CT predicts incident major CHD events independent of traditional CAC score in 1268 asymptomatic subjects without prevalent major CHD from the offspring and third generation cohorts of the Framingham Heart Study. (11) Results revealed a total of 42 major CHD events occurring during a median follow-up period of 7.4 years. Both the number of coronary arteries with CAC (HR, 1.68 per artery, 95% CI, 1.10 to 2.57; $p=.02$) and the presence of CAC in the proximal dominant coronary artery (HR, 2.59; 95% CI, 1.15 to 5.83; $p=.02$) were associated with major CHD events after multivariable adjustment.

Nakanishi et al. (2016) conducted a study among 13,092 consecutive asymptomatic individuals without known CAD (mean age, 58 years) clinically referred for a CAC scan between 1997 and 2011 at a university medical center; the study examined the predictive value of CAC for 5- and 15-year mortality rates among men and women. (12) Coronary artery calcium showed an incremental prognostic value over traditional risk factors among men at 5 years (area under curve [AUC], 0.702 vs. 0.655; $p=.002$) as well as at 15 years (AUC, 0.723 vs. 0.656; $p<.001$). In women, the incremental prognostic value of CAC was not statistically significant at 5 years (AUC, 0.650 vs. 0.612; $p=.065$), but was statistically significant at 15 years (AUC, 0.690 vs. 0.624; $p<.001$).

Elias-Smale et al. (2011) conducted a study among 2153 asymptomatic participants (69.6 years) who underwent a multidetector CT scan in the Rotterdam Study. (13) During a median follow-up of 3.5 years, 58 CHD events (MI or death) occurred. Participants were classified into low (<5%), intermediate (5% to 10%), and high (>10%) 5-year risk categories based on a refitted Framingham risk model. For the outcome of CHD, the C statistic improved from 0.693 for the refitted Framingham model to 0.743 by addition of coronary calcium. Reclassification of subjects occurred most substantially in the intermediate-risk group (5-year risk, 5% to 10%) where 56% of persons were reclassified. Addition of CAC scoring reclassified 56% of persons: 36% moved to low-risk while 20% moved to high-risk, leading to a net gain in reclassification of 18% in persons with an event and a net decline in reclassification of 3% in persons without an event, resulting in an NRI of 15% ($p<.01$).

Erbel et al. (2010) assessed NRI and risk prediction based on CAC scoring in comparison with traditional risk factors in 4129 subjects without overt CAD at baseline in the Heinz Nixdorf Recall study. (14) Results revealed that 93 coronary deaths and nonfatal MIs occurred after 5 years of follow-up (cumulative risk, 2.3%; 95% CI, 1.8% to 2.8%). Reclassifying intermediate risk subjects with CAC <100 to the low-risk category and with CAC $\geq$ 400 to the high risk category yielded an NRI of 21.7% ($p=.0002$) and 30.6% ($p<.0001$) for the FRS, respectively. Adding CAC scores to the FRS and National Cholesterol Education Panel ATP III categories improved the AUC from 0.681 to 0.749 ($p<.003$) and from 0.653 to 0.755 ($p=.001$), respectively. The authors concluded that limiting CAC scoring to intermediate risk subjects assists in correctly identifying

a high proportion of individuals at highest risk and may contribute to reducing the number of coronary events in the general population; however, clinicians need to be aware that this may not be applicable across the board, particularly for patients in a low-risk category. In 2018, Lehmann et al. published additional 10-year follow-up data from Heinz Nixdorf and concluded that CAC progression is associated with coronary and CV event rates, but only weakly adds to risk prediction. (15) The authors stated that what counts is the most recent CAC value and risk factor assessment.

A number of additional studies have reported that CAC scoring adds predictive information. (16-24)

Clinically Useful

A test is clinically useful if the use of the results informs management decisions that improve the net health outcome of care. The net health outcome can be improved if patients receive correct therapy, more effective therapy, or avoid unnecessary therapy or testing.

Direct evidence of clinical utility is provided by studies that have compared health outcomes for patients managed with and without the test. Because these are intervention studies, the preferred evidence would be from randomized controlled trials (RCTs).

Direct Evidence - Systematic Reviews

Tables 1 and 2 summarize, respectively, the characteristics and results of systematic reviews relevant to the assessment of the clinical utility of CAC scoring.

Scheu et al. (2025) performed a systematic review of RCTs and prospective cohort studies of CAC screening in asymptomatic adults. (25) Changes in cardiovascular therapy, risk factors, and health behavior were compared between patients who underwent screening and control patients (i.e., did not undergo screening, or were blinded to screening results). Of the 8 included studies (N=51,554), 7 were RCTs and one was an observational cohort. Results were summarized descriptively; meta-analysis was not performed due to heterogeneity. Changes reported with CAC screening were improved blood pressure (1 study), improved lipids (5 studies), increased adherence to statins (1 study), increased motivation to change lifestyle (1 study), and more self-reported physical activity (1 study). No studies reported benefit in cardiovascular events or all-cause mortality.

Gupta et al. (2017) performed a systematic review and meta-analysis evaluating the odds of initiating or continuing pharmacological (i.e., aspirin, lipid-lowering, and blood pressure-lowering medications) and lifestyle preventive therapies in asymptomatic CAD patients with nonzero versus 0 CAC scores as detected on cardiac CT. (26) Results revealed that the odds of aspirin, lipid-lowering, and blood pressure-lowering medication initiation, lipid-lowering medication continuation, an increase in exercise, and dietary changes were significantly higher in patients with nonzero CAC versus 0 CAC scores. However, the odds of aspirin or blood pressure-lowering medication continuation were not significantly increased in the nonzero CAC group. Statistical heterogeneity was present across studies for many of the outcomes; potential

sources of heterogeneity included variations in sample size and the proportion of patients with 0 versus nonzero CAC, whether patients were shown their CAC scan, and differences in clinical characteristics of study populations.

Mamudu et al. (2014) conducted a systematic review of studies evaluating the effects of CAC screening on behavioral modification, risk perception, and medication adherence in asymptomatic adults. (27) Fifteen studies were selected (3 RCTs, 12 observational studies). The size of the study populations ranged from 56 to 6814 individuals. Reviewers primarily provided descriptive results of the studies given the lack of standardization across studies regarding CAC measures and outcome variables. Coronary artery calcium screening improved medication adherence. However, the impact of CAC screening on behavioral and lifestyle factors (body mass index, diet, exercise, smoking), the perception of CAD risk, and psychosocial effects were not statistically significant compared with baseline.

Whelton et al. (2012) published a meta-analysis of RCTs that evaluated the impact of CAC scores on cardiac risk profiles and cardiac procedures. (28) Four trials were identified (N=2490); the individual trials ranged in size from 50 to 1934 patients. Reviewers pooled data from 4 trials on the impact of calcium scores on blood pressure, from 3 to evaluate the impact on low-density lipoprotein, and from 2 to determine the impact on high-density lipoprotein. Pooled analysis did not show a significant change in any of these parameters when incorporating calcium scores. Similarly, in 4 studies that looked at the rates of smoking cessation following calcium scores, no significant change was found. Two studies included rates of coronary angiography and 2 included rates of revascularization. Pooled analysis of these studies did not show a significant change after the measurement of coronary calcium.

Table 1. Characteristics of Systematic Reviews Assessing the Clinical Utility of CAC Score for Asymptomatic Patients

Study	Dates	Trials	Participants	N (Range)	Design	Duration (Range)	Outcomes
Scheu et al. (2025) (25)	2003-2024	8	Asymptomatic for CAD	51,554 (56 to 43,447)	SR of RCTs and prospective cohorts	6 to 60 months	Change in cardiovascular risk factors, use of pharmacological prevention, health-related behaviors, cardiovascular events, all-cause mortality
Gupta et al. (2017) (26)	2006-2011	6	Asymptomatic for CAD	11,256 (505 to 6814)	SR and MA of RCTs and observational cohorts	1.6 to 6 y (mean follow-up)	Initiation or continuation of pharmacological and lifestyle

							preventive therapies
Mamudu et al. (2014) (27)	1996-2014	15	Asymptomatic for CAD	16,983 (56 to 6814)	SR of RCTs and Prospective Cohorts	3 mo to > 8 y	Positive behavioral change, risk perception, medication adherence
Whelton et al. (2012) (28)	2003-2011	4	Asymptomatic for CAD	2490 (50 to 1934)	MA of RCTs	1 to 4 y	CVD and CAD risk factors, 10-y FRS event rate, incident clinical disease

CAC: coronary artery calcium; CAD: coronary artery disease; CVD: cardiovascular disease; FRS: Framingham risk score; MA: meta-analysis; mo: month; N: Number; RCT: randomized controlled trial; SR: systematic review; y: year.

Table 2. Results of Systematic Reviews Assessing the Impact of CAC Score on Clinical Risk Profile, Cardiac Procedures, and Pharmacological and Lifestyle Preventive Therapies Among Asymptomatic Patients

Study	Treatment	Comparator	Trials	Measure	Association	95% CI
Gupta et al. (2017) (26)	CAC score of 0	Nonzero CAC score	4	Aspirin initiation	2.61	1.81 to 3.78
	CAC score of 0	Nonzero CAC score	3	Lipid lowering medication initiation	2.86	1.85 to 4.41
	CAC score of 0	Nonzero CAC score	2	Blood pressure lowering medication initiation	1.94	1.61 to 2.33
	CAC score of 0	Nonzero CAC score	3	Aspirin continuation	1.28	0.75 to 2.18
	CAC score of 0	Nonzero CAC score	4	Lipid lowering medication continuation	2.26	1.56 to 3.28
	CAC score of 0	Nonzero CAC score	2	Blood pressure lowering medication continuation	1.38	0.86 to 2.23
	CAC score of 0	Nonzero CAC score	3	Increased exercise	1.84	1.41 to 2.41
	CAC score of 0	Nonzero CAC score	2	Dietary change	1.94	1.52 to 2.49

Whelton et al. (2012) (28)	CAC screen	No CAC screen	4	Mean change in systolic BP	0.23	-2.25 to 2.71
	CAC screen	No CAC screen	3	Mean change in diastolic BP	-0.42	-1.18 to 0.35
	CAC screen	No CAC screen	3	Mean change in LDL	0.23	-5.96 to 6.42
	CAC screen	No CAC screen	2	Mean change in HDL	-1.18	-5.50 to 3.14
	CAC screen	No CAC screen		RR of smoking cessation	1.15	0.77 to 1.71
	CAC screen	No CAC screen		RR of angiography	1.17	0.68 to 1.99
	CAC screen	No CAC screen		RR of revascularization	1.35	0.69 to 2.63

BP: blood pressure; CAC: coronary artery calcium; CI: confidence interval; HDL: high-density lipoprotein; LDL: low-density lipoprotein; RR: relative risk

Direct Evidence - Randomized Controlled Trials

Randomized controlled trials by Rozanski et al. (2011) (29) and O'Malley et al. (2003) (30), both included in the Whelton et al. (2012) (28) systematic review, and Whitmore et al. (2025) (31) which was included in the Scheu et al. (2025) (25) systematic review, captured the effect of incorporating CAC scoring in clinical practice on CAD risk factors and overall CAD risk.

Whitmore et al. (2025) conducted an RCT to assess the effect of repeated CAC testing over 3 years on cardiovascular risk reduction in asymptomatic patients. (31) All patients had a family history of premature CAD and a CAC score of 1 to 400. Patients were randomized to a cardiovascular risk reduction program (including visualizing CAC images and statin therapy) or standard care. At 3 years, FRS scores had decreased by 3.4% (95% CI, 2.4% to 4.4%; $p \leq .001$) in the program group, which was a greater reduction than in the control group. Reductions in low-density lipoprotein were also larger in program group than the control group (-1.2 mmol/L; 95% CI, -1.4 to -1.0; $p < .001$). There were no differences between groups in blood pressure or body mass index at 36 months, but there was a significant difference in adherence to daily exercise and lifestyle behaviors ($p \leq .001$).

Rozanski et al. (2011) conducted an RCT to evaluate the impact of CT scanning for CAC on cardiac risk factors. (29) A total of 2137 healthy volunteers were randomized in a 2:1 ratio to CT scanning ($n=1424$) or no CT scanning ($n=713$) and followed for 4 years. At baseline, both groups received one session of risk factor counseling by a nurse practitioner. The primary endpoint was a 4-year change in CAD risk factors and FRS. At the 4-year follow-up, there was a differential dropout among the groups, with 88.2% (1256/1424) of follow-up in the scan group and 81.9% (584/713) in the no-scan group. Compared with the no-scan group, the scan group showed a net favorable change in systolic blood pressure ($p=.02$), low-density lipoprotein cholesterol

($p=.04$), and waist circumference for those with increased abdominal girth ($p=.01$), and a tendency to weight loss among overweight subjects ($p=.07$). While there was a mean rise in FRS in the no-scan group (0.7), FRS remained static in the scan group (0.002; $p=.003$). Downstream medical testing in the scan group was comparable with those of the no-scan group, balanced by lower and higher resource utilization for subjects with normal CAC scans and CAC scores of 400 or higher, respectively.

This trial highlights the potential benefit of CAC screening in modifying the cardiac risk profile but is not definitive in demonstrating improved outcomes. Trial limitations included differing intensities of interventions between groups and differential dropout. It is possible that the small differences reported in the trial resulted from bias related to these methodologic limitations. Also, this trial did not compare the impact of other types of risk factor intervention, most notably more intensive risk factor counseling.

O'Malley et al. (2003) conducted an RCT among a consecutive sample of 450 asymptomatic active-duty U.S. Army personnel aged 39 to 45 years to assess the effects of incorporating EBCT as a motivational factor into a cardiovascular screening program. (30) The program offered intensive case management or usual care and assessed treatment impact on 10-year FRS over 1 year. The authors used a 2 x 2 factorial design and patients were randomized to 1 of the 4 intervention arms: EBCT results provided in the setting of intensive case management ($n=111$) or usual care ($n=119$) or EBCT results withheld in the setting of intensive case management ($n=124$) or usual care ($n=96$). Mean absolute risk change in 10-year FRS between groups receiving and not receiving results was +0.30 and +0.36 ($p=.81$), respectively. The trial was not powered for clinical endpoints. EBCT did not produce any benefits regarding a difference in FRS at 1 year.

Nerlecker et al. (2025) conducted an open-label RCT (CAUGHT-CAD) in 365 asymptomatic patients with intermediate risk for CAD based on family history that compared CAC score-informed prevention and usual care. (32) This study was not included in any of the aforementioned systematic reviews. Prevention strategies included lifestyle education, patient review of CCTA images, blood pressure control, and lipid-lowering therapy. After 3 years, the change in the primary outcome (total plaque volume) was smaller in the CAC score-informed group than the usual care group (between group difference, 9.5 mm³; 95% CI, 2.4 to 23.8; $p=.009$). Total cholesterol, low-density lipoprotein, and triglycerides were all significantly better with CAC score-informed therapy (all $p<.001$). Waist circumference and body mass index were similar between groups.

Chain of Evidence

Indirect evidence on clinical utility rests on clinical validity. If the evidence is insufficient to demonstrate test performance, no inferences can be made about clinical utility.

Section Summary: Coronary Artery Calcium Scoring in Asymptomatic Individuals

Multiple observational cohort studies and systematic reviews of these studies have consistently demonstrated the incremental prognostic value of CAC scoring in predicting CVD events

compared to standard risk stratification alone among asymptomatic populations over the intermediate and long-term; however, studies have reported mixed findings on whether the use of the score is key to improved cardiovascular outcomes or improvements in other clinical outcomes that lead to cardiovascular risk reduction.

Coronary Artery Calcium Scoring in Symptomatic Patients

In certain clinical situations, such as individuals presenting with chest pain, it is uncertain whether the symptoms are due to CAD. Coronary calcium measurement has been proposed as a method to rule out CAD in certain individuals if their CAC score is 0. The presence of any coronary calcium can be a sensitive, but not specific, test for coronary disease because CAD rarely occurs in the absence of coronary calcium. False-positives occur because the calcium may not be associated with an ischemic lesion. The absence of any coronary calcium can be a specific test for the absence of coronary disease and direct the diagnostic workup toward other causes of the patient's symptoms. In this context, coronary calcium measurement is not used to make a positive diagnosis, but as a diagnostic "filter" to rule out an atherosclerotic cause for the patient's symptoms.

Clinical Context and Test Purpose

The use of CAC scoring with CT in symptomatic individuals can rule out the atherosclerotic etiology of CAD.

The following PICO was used to select literature to inform this policy.

Populations

The population of interest is individuals who have signs and/or symptoms suggestive of CAD.

Interventions

The intervention of interest is CAC scoring using fast CT imaging, including EBCT and spiral CT. Computed tomography CAC scoring is utilized when individuals require evaluation for persistent stable angina or experience onset of acute chest pain.

Comparators

The following test is currently being used to make decisions about managing CAD: standard diagnostic testing, which includes functional testing and exercise electrocardiography.

Outcomes

The outcomes of interest include OS, test accuracy, test validity, and morbid events (e.g., MACEs, need for ICA and revascularization).

Study Selection Criteria

For the evaluation of clinical validity of CAC scoring using CT, studies that meet the following eligibility criteria were considered:

- Reported on the accuracy of the marketed version of the technology;
- Included a suitable reference standard;

- Patient/sample clinical characteristics were described;
- Patient/sample selection criteria were described;
- The study reported on a minimum of 1000 patients.

Clinically Valid

A test must detect the presence or absence of a condition, the risk of developing a condition in the future, or treatment response (beneficial or adverse).

Systematic Reviews

Chaikriangkrai et al. (2016) conducted a systematic review and meta-analysis to examine the prognostic value and accuracy of a CAC score of 0 for identifying patients presenting with acute chest pain at acceptable low-risk for future cardiovascular events. (33) The systematic review included only prospective cohort studies that used multidetector CT or EBCT to calculate CAC scores using the Agatston method and reported MACEs at 1 month and beyond the index emergency department visit. Eight studies evaluating 3556 patients with a median follow-up of 10.5 months were selected. Reviewers conducted a subgroup analysis of 6 studies in predominantly White patients (N=2432) to estimate the prognostic accuracy indices of CAC scores (0, >0) for cardiovascular events (MACEs, all-cause deaths, nonfatal MI). Pooled sensitivity, specificity, as well as positive and negative likelihood ratios were 96% ($I^2=0\%$), 60% ($I^2=15.1\%$), 2.36 ($I^2=0\%$), and 0.07 ($I^2=0\%$), respectively (Table 3).

The systematic review by Sarwar et al. (2009), mentioned prior in this policy examined the clinical, diagnostic, and prognostic significance of a CAC score of 0. (3) Eighteen studies from 1992 to 2007, in which 10,355 symptomatic patients with suspected CAD underwent CAC testing as well as ICA, were selected in the analysis to examine the diagnostic accuracy of CAC scoring for stenosis on ICA. A total of 5805 (56%) patients had significant coronary stenosis (defined as >50%) on ICA. Pooled data revealed that the presence of calcium had a sensitivity, a specificity, as well as a positive and a negative likelihood ratio of 98%, 40%, 1.63, and 0.06, respectively, for predicting coronary artery stenosis. The summary negative predictive value was 92% (95% CI, 88% to 95%; $p<.001$). The summary positive predictive value was 68% (95% CI, 64% to 72%; $p<.001$) (Table 3).

Lo-Kioeng-Shioe et al. (2019) conducted a systematic review and meta-analysis of 18 observational studies (N=34,041) to assess the ability of CAC to predict risk of MACE, defined as the composite of late cardiac revascularization, hospitalization for unstable angina pectoris or heart failure, nonfatal MI, and cardiac death or all-cause mortality, in stable patients with suspected CAD. (34) Of 1601 cardiovascular events, 158 occurred in patients with a CAC score of 0. The pooled risk ratio for MACE in patients with CAC >0 was 5.71 (95% CI, 3.98 to 8.19), and risk increased with increasing levels of CAC. The pooled relative risk for incidence of all-cause mortality or nonfatal MI was 3.64 (95% CI, 2.68 to 4.96).

Table 3. Pooled Diagnostic Performance of CAC Score for CAD Among Symptomatic Individuals

Test	Studies	N	Sensitivity (95% CI), %	Specificity (95% CI), %	LR+ (95% CI)	LR- (95% CI)
Chaikriangkrai et al. (2016) (33)						
CAC score (0, >0)	6	2432	96 (93 to 98)	60 (58 to 62)	2.36 (2.22 to 2.51)	0.07 (0.04 to 0.14)
Sarwar et al. (2009) (3)						
CAC score (0, >0)	18	10,355	98 (97 to 98)	40 (38 to 41)	1.63 (1.59 to 1.67)	0.06 (0.05 to 0.07)

CAC: coronary artery calcium; CAD: coronary artery disease; CI: confidence interval; LR: likelihood ratio.

Randomized Controlled Trials

Lubbers et al. (2016) conducted a multicenter RCT to compare the effectiveness and safety of a cardiac CT algorithm with functional testing in patients with symptoms (stable chest pain or angina equivalent symptoms) suggestive of CAD. (35) A total of 350 patients with stable angina were prospectively randomized 2:1 to cardiac CT or functional testing, such as exercise electrocardiography, myocardial perfusion imaging, or stress echocardiography. Patients in the cardiac CT arm (n=242) initially underwent calcium scanning followed by CCTA if the Agatston score was between 1 and 400. Coronary artery disease was ruled out if the patients had a CAC score of 0. The original primary endpoint of the trial was the proportion of patients undergoing catheter angiography followed by revascularization, but because of insufficient funding, the authors could not assess that endpoint and chose clinical effectiveness as the alternative primary outcome, defined as the absence of chest pain complaints after 1 year. After 1-year, fewer patients randomized to CT reported angina symptoms than those in the functional testing group (39% vs. 25%; p=.012), although the proportion of patients with similar or worsened symptoms was comparable (26% vs. 29%; p=.595). The tiered protocol study design is a strength of this trial, but the unplanned change in endpoints limits analysis and conclusions.

Observational Studies

Pursnani et al. (2015) published results from a subgroup analysis of the Rule Out Myocardial Infarction using Computed Assisted Tomography II trial. (36) This analysis evaluated the incremental diagnostic value of CAC scoring plus CCTA in low- to intermediate-risk patients presenting to the emergency department with symptoms (chest pain or angina equivalent of ≥ 5 minutes duration within 24 hours) suggesting acute coronary syndrome (ACS). The Rule Out Myocardial Infarction using Computed Assisted Tomography II trial randomized patients with possible ACS to CCTA as part of an initial evaluation or to the standard emergency department evaluation strategy, as directed by local caregivers. As part of the trial protocol, all patients undergoing CCTA had a CAC scan; the present analysis included 473 patients who underwent both CCTA and CAC scanning. Among these patients, the ACS rate (defined as unstable angina and MI during the index hospitalization) was 8% (n=38). Patients with lower CAC scores were less likely to have a discharge diagnosis of ACS. Among 253 patients with a CAC score of 0, 2 (0.8%) patients were diagnosed with ACS (95% CI, 0.1% to 2.8%). Receiver operating characteristic curve analysis was used to predict the risk of ACS by CAC score greater than 0, continuous CAC score, CCTA results, and combined CAC and CCTA score. The optimal cut-point of CAC for ACS detection was 22 (C statistic, 0.81), with 318 (67%) patients having a

CAC score of less than 22. All CCTA strategies had high sensitivity for ACS detection, without significant differences in stenosis thresholds. Coronary artery calcium was inferior to CCTA for predicting ACS (C range, 0.86 vs. 0.92; $p=.03$). The addition of CAC score to CCTA (i.e., using selective CCTA only for patients with CAC score >22 or >0) did not significantly improve the detection of ACS (CAC plus CCTA $C=0.93$ vs. CCTA $C=0.92$; $p=.88$). Overall, this trial suggested that CAC scoring did not provide incremental value beyond CCTA in predicting the likelihood of ACS in a low- to intermediate-risk population presenting to the emergency department.

Chaikriangkrai et al. (2015) retrospectively evaluated whether CAC added incremental value to CCTA for predicting coronary artery stenosis in 805 symptomatic patients without known CHD. (37) Coronary artery calcium score was significantly associated with the presence of coronary artery stenosis on CCTA. Both CAC score and the presence of CCTA stenosis were significantly associated with MACE rates, including cardiac death, nonfatal MI, and late coronary revascularization. Patients with more than 50% stenosis on CCTA had higher MACE rates, compared with those who had a normal CCTA (4.5% vs. 0.1%; $p<.001$) and with those who had less than 50% stenosis (4.5% vs. 1.4%; $p=.002$). Those with a CAC score of more than 400 had higher MACE rates than those with scores between 1 and 100 (4.2% vs. 1.4%; $p=.014$) and those with scores of 0 (4.2% vs. 0%; $p<.001$). The addition of CAC score to a risk prediction model for MACE, which included clinical risk factors and CCTA stenosis, significantly improved the model's predictive performance (global c^2 score, 108 vs. 70; $p=.019$).

Hulten et al. (2014) published results from a retrospective cohort study among symptomatic patients without a history of CAD to evaluate the accuracy of CAC scoring for excluding coronary stenosis, using CCTA as the criterion standard. (38) The study included 1145 patients who had symptoms possibly consistent with CAD who underwent noncontrast CAC scoring and contrast-enhanced CCTA from 2004 to 2011. For detection of greater than 50% stenosis, CAC had a sensitivity, specificity, and negative predictive value of 98%, 55%, and 99%, respectively. For the prediction of cardiovascular death or MI, the addition of either or both CAC and CCTA to a clinical prediction score did not significantly improve prognostic value.

Dharampal et al. (2013) retrospectively evaluated a cohort of 1975 symptomatic patients (those with chest pain referred by their cardiologist for CCTA) who underwent a clinical evaluation and CAC scoring and CCTA or ICA. (39) The primary outcome was obstructive CAD ($\geq 50\%$ stenosis) on ICA or CCTA (if ICA was not done). The authors evaluated the NRI with the addition of CAC score to a clinical prediction model for patients who had an intermediate probability of CHD (10% to 90%) after clinical evaluation based on chest pain characteristic, age, sex, risk factors, and electrocardiogram. Discrimination of CAD was significantly improved by incorporating the CAC score into the clinical evaluation (AUC, 0.80 vs. 0.89; $p<.001$).

Yoon et al. (2012) conducted a prospective study among 136 Korean patients (58% men; mean age, 56 years) who presented to the emergency department with acute chest pain and nondiagnostic electrocardiograph to examine the diagnostic usefulness of the "zero calcium score criteria" as a decision-making strategy to rule out significant CAD as the etiology of acute chest pain. (40) All patients underwent 64-slice CT for calcium scoring and CCTA. Ninety-two

(68%) of 136 patients did not show detectable CAC, and 14 (15%) of these 92 without CAC had 50% or more stenosis on CCTA. Sensitivity, specificity, positive predictive value, and negative predictive value of a CAC score of 0 for the detection of 50% or more stenosis were 66% (95% CI, 50% to 80%), 83% (95% CI, 74% to 90%), 64% (95% CI, 48% to 77%), and 85% (95% CI, 75% to 91%), respectively. A calcium score of 0 did not necessarily guarantee the absence of significant CAD in an Asian population presenting to the emergency department with chest pain.

Gottlieb et al. (2010) conducted a prospective multicenter study to evaluate whether the absence of coronary calcium could be used to rule out 50% or more coronary stenosis or the need for revascularization. (41) The authors compared the diagnostic performance of 64-detector CT with that of ICA. Among 291 patients with suspected CAD included in the study, 214 (73%) were male, and the mean age was 59.3 years. Fifty-six percent of the patients had 50% or more stenosis. Among 72 patients with a CAC score of 0, 14 (19%) had at least 1 coronary artery with 50% or more stenosis. The overall sensitivity for a CAC score of 0 to predict the absence of 50% or more stenosis was 45%, specificity was 91%, the negative predictive value was 68%, and the positive predictive value was 81%. Additionally, 9 (12.5%) patients with a CAC score of 0 underwent revascularization within 30 days of calcium scoring.

Clinically Useful

A test is clinically useful if the use of the results informs management decisions that improve the net health outcome of care. The net health outcome can be improved if patients receive correct therapy, more effective therapy, or avoid unnecessary therapy or testing.

Direct evidence of clinical utility is provided by studies that have compared health outcomes for patients managed with and without the test. Because these are intervention studies, the preferred evidence would be from RCTs.

Direct Evidence - Observational Studies

Yerramasu et al. (2014) prospectively assessed an evaluation algorithm including CAC scoring for patients presenting to a rapid access chest pain clinic with stable chest pain possibly consistent with CHD. (42) Three hundred patients presenting with acute chest pain to 1 of 3 chest pain clinics underwent CAC scoring. If the CAC score was 1000 or more Agatston units, ICA was performed; if the CAC score was less than 1000, CCTA was performed. All patients with a CAC score of 0 and low pretest likelihood of CHD had no obstructive CHD on CCTA and were event-free during follow-up. Of the 18 patients with CAC scores from 400 to 1000, 17 (94%) had greater than 50% obstruction on subsequent CCTA and were referred for further evaluation, 14 (78%) of whom had obstructive CHD. Of 15 patients with CAC scores 1000 or more and who were referred for coronary angiography, obstructive CHD was present in 13 (87%). This study suggested that CAC scoring can be used in the acute chest pain setting to stratify decision-making for further testing.

ten Kate et al. (2013) prospectively evaluated the accuracy of cardiac CT, including CAC scoring with or without CCTA, in distinguishing heart failure due to CAD from heart failure due to non-CAD causes. (43) Data on the predictive ability of a negative CAC score in ruling out CAD was

also included. The study included 93 symptomatic patients with newly diagnosed heart failure of unknown etiology, all of whom underwent CAC scoring. Those with a CAC score greater than 0 underwent CCTA and, if the CCTA was positive for CAD (>20% luminal diameter narrowing), ICA was recommended. Forty-six percent of patients had a CAC score of 0. At a mean follow-up of 20 months, no patient with a CAC score of 0 had a MI, underwent percutaneous coronary intervention, had a coronary artery bypass graft, or had signs of CAD.

Chain of Evidence

Indirect evidence on clinical utility rests on clinical validity. If the evidence is insufficient to demonstrate test performance, no inferences can be made about clinical utility.

Because the clinical validity of CAC scoring for symptomatic patients has not been established, a chain of evidence supporting the clinical utility of CAC scoring in this population cannot be constructed.

Section Summary: Coronary Artery Calcium Scoring in Symptomatic Patients

Systematic reviews and meta-analyses have reported a very low negative likelihood ratio for CAC score in predicting MACEs and significant coronary stenosis, suggesting the potential value of a calcium score of 0 in ruling out an atherosclerotic etiology of the disease. However, multiple observational studies with angiographic (CCTA or ICA) interventions have suggested that a CAC score of 0 may not rule out the presence of significant atherosclerotic CAD among symptomatic patients. Currently, evidence from nonrandomized, observational studies has suggested a very low short- or long-term risk of cardiovascular events or death in patients having calcium scores of 0 compared with those having positive (>0) calcium scores. However, considering the inconsistency in evidence regarding the diagnostic accuracy of calcium scoring and lack of evidence from RCTs, further research is needed to examine the clinical utility of ruling out atherosclerotic CAD based on a CAC score of 0.

Summary of Evidence

For individuals who are asymptomatic with the risk of coronary artery disease (CAD) who receive coronary artery calcium (CAC) scoring, the evidence includes multiple systematic reviews, randomized controlled trials (RCTs), and nonrandomized observational studies. Relevant outcomes are overall survival (OS), test accuracy and validity, morbid events, and resource utilization. There is extensive evidence on the predictive value of CAC score screening for cardiovascular disease (CVD) among asymptomatic patients, and this evidence has demonstrated that scanning has incremental predictive accuracy above traditional risk factor measurement. However, high-quality evidence demonstrating that the use of CAC scores in clinical practice leads to changes in patient management or in individual risk behaviors that improve cardiac outcomes is limited. One meta-analysis of RCTs reported no significant change in coronary risk profile, downstream testing, or revascularization following screening using CAC scoring compared with no CAC scoring. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

For individuals with signs and/or symptoms suggestive of CAD who receive CAC scoring before other diagnostic testing, the evidence includes systematic reviews, RCTs, and nonrandomized observational studies. Relevant outcomes are OS, test accuracy and validity, morbid events, and resource utilization. Coronary artery calcium scoring has potential as a diagnostic test to rule out CAD in patients presenting with symptoms or as a "gatekeeper" test before invasive imaging is performed. Evidence from observational studies has suggested that negative results on CAC scoring rule out CAD with good reliability. However, the evidence has been inconsistent, with some studies reporting a lack of value when using a 0 calcium score to rule out CAD. Further prospective trials would be needed to demonstrate that such a strategy is effective in practice and is at least as effective as alternative strategies for ruling out CAD. To demonstrate that use of calcium scores improves the efficiency or accuracy of the diagnostic workup of symptomatic patients, rigorous studies defining exactly how CAC scores would be used in combination with other tests to triage patients would be necessary. The evidence is insufficient to determine that the technology results in an improvement in the net health outcome.

Practice Guidelines and Position Statements

American Heart Association/American College of Cardiology

The American College of Cardiology and American Heart Association (2018) Clinical Practice Guidelines on the Management of Blood Cholesterol state, "When risk status is uncertain, a coronary artery calcium (CAC) score is an option to facilitate decision making in adults 40 to 75 years of age." (44) The guidelines further note, "One purpose of CAC scoring is to reclassify risk identification of patients who will potentially benefit from statin therapy. This is especially useful when the clinician and patient are uncertain whether to start a statin. Indeed, the most important recent observation has been the finding that a CAC score of 0 indicates a low ASCVD risk for the subsequent 10 years. Thus, measurement of CAC potentially allows a clinician to withhold statin therapy in patients showing 0 CAC."

With regard to the prognostic significance of CAC, the guideline "makes use of the available data to predict the risk associated with CAC." (44) The guideline notes that "these data need to be amplified by new and ongoing studies to guide treatment decisions" and that "particular uncertainty exists about the predictive value of intermediate CAC scores." Additionally, there are concerns regarding the predictive significance of a CAC score of 0, which must be further verified in follow-up studies. For patients with a 0 score, "it is currently uncertain when and if follow-up CAC measurements should be done to reassess risk status."

The American College of Cardiology and American Heart Association (2019) Guideline on the Primary Prevention of Cardiovascular Disease is in line with the blood cholesterol guideline stating that adults (40 to 75 years of age) who are being evaluated for cardiovascular disease prevention should initially undergo 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimation with a clinician-patient risk discussion before starting pharmacological therapy. (45) The guideline also notes that assessing for other risk-enhancing factors can help guide decision making "about preventive interventions in select individuals, as can CAC scanning." The guideline specifically states the following recommendation regarding assessment of cardiovascular risk and CAC:

- In adults at intermediate risk ($\geq 7.5\%$ to $< 20\%$ 10-year ASCVD risk) or selected adults at borderline risk (5% to $< 7.5\%$ 10-year ASCVD risk), if risk-based decisions for preventive interventions remain uncertain, it is reasonable to measure a CAC score to guide clinician-patient risk discussion [Class (Strength) of Recommendation: IIa; Level (Quality) of Evidence: B-NR]. A IIa class of recommendation is of moderate strength based on moderate quality nonrandomized studies.

The American Heart Association, American College of Cardiology (2021) Guideline on Evaluation and Diagnosis of Chest Pain includes a recommendation for CAC as first-line testing in patients with stable chest pain with no known coronary artery disease and low likelihood of obstruction. (46) The guidelines recommend the addition of CAC may also be useful for intermediate-high risk patients with stable chest pain and no known coronary artery disease undergoing stress testing.

Special Report - American Heart Association/American College of Cardiology

The American Heart Association and the American College of Cardiology (2019) issued a special report on the use of risk assessment tools to guide decision-making in the primary prevention of ASCVD. (47) This report includes an algorithm of clinical approaches to incorporate CAC measurement in risk assessment for borderline- and intermediate-risk patients:

"For borderline-risk (10-year risk 5% to $< 7.5\%$) and intermediate-risk (7.5% to $< 20\%$) patients who are undecided regarding statin therapy, or when there is clinical uncertainty regarding the net benefit, consider the value of additional testing with measurement of CAC. If CAC is measured, interpret results as follows:

- a. CAC score of 0 indicates that a borderline- or intermediate-risk individual is reclassified to a 10-y event rate lower than predicted, and below the threshold for benefit from a statin. Consider avoiding or postponing statin therapy unless there is a strong family history of premature ASCVD, history of diabetes mellitus, or heavy cigarette smoking. Consider repeat CAC measurement in 5 years if patient remains at borderline or intermediate risk.
- b. CAC score 1 to 99 and < 75 th percentile for age/sex/race/ethnicity indicates that there is subclinical atherosclerosis present. This may be sufficient information to consider initiating statin therapy, especially in younger individuals, but does not indicate substantial reclassification of the 10-y risk estimate. Consider patient preferences and, if statin decision is postponed, consider repeat CAC scoring in 5 years.
- c. CAC score 100 or ≥ 75 th percentile for age/sex/race/ethnicity indicates that the individual is reclassified to a higher event rate than predicted, that is above the threshold for statin benefit. Statin therapy is more likely to provide benefit for such patients."

National Institute for Health and Care Excellence

For patients with "stable chest pain who cannot be excluded by clinical assessment alone," the National Institute for Health and Care Excellence recommended CT using 64-slice imaging. (48)

U.S. Preventive Services Task Force Recommendations

The U.S. Preventive Services Task Force (2018, update in progress) updated its recommendations on the use of nontraditional or novel risk factors in assessing coronary heart disease risk in asymptomatic adults with no known cardiovascular disease. (49, 50) Calcium score was 1 of 3 nontraditional risk factors considered. Reviewers concluded the current evidence was insufficient to assess the balance of benefits and harms of adding any of the nontraditional risk factors studied to traditional risk assessment in asymptomatic adults with no known cardiovascular disease.

Ongoing and Unpublished Clinical Trials

Some ongoing trials that might influence this policy are listed in Table 4.

Table 4. Summary of Key Trials

NCT Number	Trial Name	Planned Enrollment	Completion Date
NCT05700877	Screening and Intervention for Subclinical Coronary Artery Disease in Patients with Type 2 Diabetes: THE STENO INTEN-CT STUDY	7300	Mar 2029
NCT03972774	Assessment of Patients With Suspected Coronary Artery Disease by Coronary Calcium First Strategy Versus Usual Care Approach	2500	Nov 2029
NCT04075162	Community Benefit of No-charge Calcium Score Screening Program	77,000	Dec 2032
NCT03439267	Effectiveness of a Proactive Cardiovascular Primary Prevention Strategy, With or Without the Use of Coronary Calcium Screening, in Preventing Future Major Adverse Cardiac Events	5,765	May 2027
NCT05314140	Towards Optimal Screening and Management of Coronary Artery Disease in Diabetes: TOSCANA Study	2000	Jun 2026
NCT05267990	Impact of a Coronary Artery Calcium-guided Primary Prevention of Major Coronary Heart Disease for Asymptomatic Coronary Artery Disease in Diabetes: a Prospective Cohort Study	2000	Dec 2028

NCT: national clinical trial.

Coding

Procedure codes on Medical Policy documents are included **only** as a general reference tool for each policy. **They may not be all-inclusive.**

The presence or absence of procedure, service, supply, or device codes in a Medical Policy document has no relevance for determination of benefit coverage for members or reimbursement for providers. **Only the written coverage position in a Medical Policy should be used for such determinations.**

Benefit coverage determinations based on written Medical Policy coverage positions must include review of the member's benefit contract or Summary Plan Description (SPD) for defined coverage vs. non-coverage, benefit exclusions, and benefit limitations such as dollar or duration caps.

CPT Codes	75571, 75572, 75573, 75574, 0710T, 0711T, 0712T, 0713T
HCPCS Codes	G0680, S8092

*Current Procedural Terminology (CPT®) ©2024 American Medical Association: Chicago, IL.

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Centers for Medicare and Medicaid Services (CMS)

The information contained in this section is for informational purposes only. HCSC makes no representation as to the accuracy of this information. It is not to be used for claims adjudication for HCSC Plans.

The Centers for Medicare and Medicaid Services (CMS) does not have a national Medicare coverage position. Coverage may be subject to local carrier discretion.

A national coverage position for Medicare may have been developed since this medical policy document was written. See Medicare's National Coverage at <<https://www.cms.hhs.gov>>.

Policy History/Revision	
Date	Description of Change
11/15/2025	Document updated. Coverage unchanged. Added references 1, 25, 31, and 32.
01/01/2025	Reviewed. No changes.
06/01/2024	Document updated with literature review. Coverage for the use of computed tomography (CT) to detect coronary artery calcification changed from experimental, investigational and/or unproven to not medically necessary. References revised; no new references added.
01/15/2023	Document updated with literature. Coverage unchanged. References 1, 4, 5 and 42 added.
01/01/2022	Reviewed. No changes.
01/01/2021	Document updated with literature review. Coverage unchanged. References 5, 8, 11, 12, 22, 28, 38, 39 and 40 added, other references updated or removed.
01/15/2020	Reviewed. No changes.
12/15/2018	Document updated with literature review. Coverage unchanged. References 2-9, 12, 18-19, 22, 25-26, 31-32, and 40-42 added and some references removed.
03/01/2017	Reviewed. No changes.
03/01/2016	Document updated with literature review. Coverage unchanged.

10/01/2015	Reviewed. No changes.
02/01/2014	Document updated with literature review. Coverage unchanged. Title changed from Cardiac Computed Tomography (CCT) for Calcium Scoring. CPT/HCPCS codes updated.
09/01/2009	Coverage revised to include Texas Contracts ONLY Legislative Mandate for allowance of CCT screening effective on 9/1/2009.
02/01/2009	CPT/HCPCS code(s) updated
02/15/2007	New medical document originating from RAD604.006