

Does CAC Testing Alter Downstream Treatment Patterns for Cardiovascular Disease?

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An estimated 33% of the adults in the United States are affected by coronary artery disease (CAD),^{1,2} and along with such high prevalence have come substantial and increasing rates of morbidity.¹ While relative mortality rates attributable to cardiovascular disease (CVD) declined 33% in the United States from 1999 to 2009, disease burden remained high. CVD was associated with 1 of every 3 United States deaths in 2009, 32% of the 2.4 million overall. Of 2009 deaths, coronary heart disease alone caused approximately 1 in every 6, and stroke 1 in every 19. The total direct and indirect cost of CVD and stroke was estimated at \$312.6 billion in 2009.³ These sizable and growing burdens have driven efforts to evaluate and better understand cardiac risks in asymptomatic patient populations.^{4,11}

Coronary artery calcium (CAC) scanning, a screening tool that detects subclinical coronary disease in asymptomatic populations, is noninvasive; it can be performed in a few minutes while the patient is fully dressed.^{4,9,12-14} A number of older studies suggest that CAC scores are directly correlated with coronary atherosclerosis, and may represent a marker for plaque burden.¹⁵⁻¹⁹ CAC identifies with calcified plaque,²⁰ and CAC score is considered a predictor of coronary death and nonfatal myocardial infarction (MI).²¹ Furthermore, some studies have reported high sensitivity (true positive for the presence of coronary artery disease), greater accuracy and reproducibility when CAC is measured with coronary computed tomography (CT).^{15,18,19} CAC utilizes radiation, and accumulating evidence about radiation exposure and cancer risk remains a concern, especially in women and middle-aged and younger persons.^{20,22-24} However, results from recent studies suggest that advanced scanning tools may reduce effective radiation doses.^{25,26}

The primary policy concern regarding CAC scanning is whether it provides—on its own, and/or when added to existing tests—a better assessment of future risk of cardiac events than do scoring methods such as the Framingham

ABSTRACT

Objectives

To assess if coronary artery calcium (CAC) scans influence treatment patterns as reflected by subsequent rates of cardiac imaging and therapeutic interventions, and their effect on ischemic events downstream.

Study Design

Longitudinal observational study from January 1, 2005, through August 31, 2011, using a large managed-care medical and pharmacy claims database.

Methods

Two cohorts were evaluated: CAC patients who received CAC testing, and Reference patients, subject to preauthorization, who were denied CAC scans. Patients were adults less than 65 years old. Index date was CAC scan date for CAC and pre-authorization request date for Reference. Patients were stratified into high-risk and non-high-risk categories; outcomes were analyzed only for non-high-risk where CAC scores could potentially modify risk classification. Cardiac imaging, coronary revascularizations, and pharmaceutical interventions were evaluated for 6 months post index and adverse ischemic events were assessed using all available follow-up time.

Results

The study included 2679 CAC and 1135 Reference patients. Among non-high-risk patients, similar proportions of both groups received an imaging test within 6 months (23.2% vs 23.8%, respectively; $P = .5$); revascularization rates and pharmaceutical utilization were similar. Adverse events were rare. Age-sex adjusted incidence rate ratio for adverse events was 1.1 (95% CI, 0.36-3.38) among CAC relative to Reference. High-risk patients, considered inappropriate for CAC testing, represented 20.2% and 23.5% of CAC and Reference, respectively ($P < .05$).

Conclusions

Patients having CAC scans were not associated with fewer downstream ischemic events nor with reduced subsequent imaging and therapeutic interventions among non-high-risk patients. Results also indicated inappropriate testing of high-risk patients.

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Risk Score (FRS)^{14,27} and the National Cholesterol Education Panel (NCEP) Adult Treatment Panel (ATP) III guidelines,²⁸ and whether it leads to improved outcomes for patients.

Anand et al reported, after following 510 asymptomatic patients for an average of slightly more than 2 years, that CAC scores were a better predictor of ischemic events and related short-term cardiovascular outcomes than established measures of cardiovascular risk factors such as the FRS.²⁹

Similarly, based on data from the multi-center prospective longitudinal trial, Multi-Ethnic Study of Atherosclerosis (MESA), Detrano et al concluded that CAC scores yielded better predictive information relative to the FRS.¹² Using data from the MESA trial, Polonsky et al added CAC scores to FRS risk factors to examine the prediction of incident CHD (including soft events such as revascularizations). The authors concluded that the addition of CAC scores produced significant net reclassification improvement, an indicator of the amount of adjustments between risk categories.³⁰ Similar reclassification rates were observed in a study by Erbel et al that added CAC scores to both the FRS and the NCEP ATP III scores when predicting hard events (ie, nonfatal myocardial infarction and coronary death).³¹

CAC testing, however, may not be useful for everyone. For high-risk patients, CAC scoring does not produce any improvement in event prediction, management, or outcomes.⁵ The 2010 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) Task Force on Practice Guidelines indicated that CAC measurements were reasonable for the assessment of cardiovascular risk among asymptomatic adults at intermediate risk, defined as 10% to 20% risk of a cardiac event within 10 years.^{6,12,14} The guidelines also indicated that the measurement of CAC may be appropriate for patients at low to intermediate risk, defined at 6% to 10% risk of a cardiac event within 10 years.^{6,14,32} The guidelines indicated, however, that CAC was not appropriate for individuals at low (6% or lower) risk of experiencing a cardiac event within 10 years.^{4,6,12,14}

While CAC scans may potentially influence the reclassification of risk for certain subgroups of asymptomatic patients, the impact of CAC testing on treatment as evidenced by subsequent imaging, therapeutic interventions, and ischemic events in the real-world setting is still largely unknown.³³ There is some evidence that CAC scanning

Take-Away Points

- This is the first study to compare downstream differences post scan in the rates of additional diagnostic testing, therapeutic interventions, and ischemic events between patients who received coronary artery calcium (CAC) scans, and controls, who were denied CAC scans because their health plans did not cover the procedure, within a large, real-world, managed-care population.
- The findings in this study, consistent with those of prior, largely clinical studies, indicated that there were no significant differences in treatment patterns or ischemic events during the post scan, follow-up period.
- While questions about the value of CAC scans persist, they are still being ordered for asymptomatic patients, raising policy questions that may be resolved by additional studies in larger patient populations and for longer durations.

may improve cardiac risk management,³⁴ but in a statement to AHA health professionals, Wexler et al pointed out that the number of people with coronary calcium could be 10 to 100 times greater than those who will ever get heart disease, and that CAC may not always be actionable, especially in those with no known risk factors.³⁵ Furthermore, evidence is lacking as to whether screening asymptomatic adults impacts morbidity or mortality from CAD.

The objective of this study was to explore if CAC testing resulted in any downstream modifications in treatment patterns and cardiac outcomes of non-high-risk patients whose physicians ordered CAC scans and either 1) received them (the CAC group), or 2) were denied them for insurance reasons, citing noncovered services (the Reference group, or References).

METHODS

Data Source and Study Design

This longitudinal observational study utilized medical and pharmacy claims within the HealthCore Integrated Research Database (HIRD) to identify patients who received a CAC procedure between January 1, 2005, and August 31, 2011. Patient records maintained by AIM Specialty Health, a specialty services company that manages radiology benefits, were used to identify patients who were denied the procedure during that same time frame. The HIRD represents a clinically rich and geographically diverse repository of longitudinal claims data for 45 million lives covered by about 14 health insurance plans in the Northeast, Midwest, South, and West regions of the United States. Researchers had access only to a limited data set, and the acquisition and handling of patient data complied with all applicable state and federal privacy regulations including the Health Insurance Portability and Accountability Act. No IRB approval was required for

this nonexperimental study, which utilized data containing no personal patient identifiers.

Study Population

Two population groupings were included in the study. The CAC group consisted of patients not requiring pre-authorization for a CAC scan (Current Procedural Terminology [CPT] codes 0144T or 75571) and receiving the procedure in an outpatient setting between January 1, 2006, and April 30, 2011. The index date for CAC patients was defined as the date of the CAC scan. The Reference group comprised patients whose physicians requested a CAC scan but were denied in the pre-authorization process, administered by AIM Specialty Health, because the procedure was not covered in the patients' health plan benefits. Reference patients did not have a CPT code for a CAC scan during the study period, and were identified via pre-authorization records maintained by AIM Specialty Health. Given the limited clinical information on cardiovascular risks in claims data, the selected reference population was the best comparator group to the CAC group. Physicians recommended CAC testing for patients in both groups. The patients in 1 group were denied the procedure because of restrictions in their health plan coverage, not because of their clinical characteristics. The index date for Reference patients was defined as the prior approval request date for the CAC procedure.

Both CAC and Reference patients were further subcategorized into high-risk and non-high-risk groups on the basis of pre existing comorbidities identified within their medical claims. Patients were classified as high risk if they had at least 1 diagnosis of diabetes, myocardial infarction (MI), angina pectoris, ischemic heart disease other than MI and angina, peripheral artery disease, thrombotic stroke/transient ischemic attack, congestive heart failure, or cerebrovascular disease during the 12 months prior to the index date. This risk classification methodology was based on conditions included in the NCEP ATP III guidelines,²⁸ plus heart failure, an approach that was described in detail in prior studies.^{36,37}

Inclusion/Exclusion Criteria

For inclusion in the analysis, both CAC and Reference patients were required to be between the ages of 18 and 64 years at the index date. To evaluate pre existing cardiac risk, all patients were required to have at least 12 months of continuous health plan eligibility prior to the index date. In addition, at least 6 months of continuous health plan eligibility post index was required to perform the analysis of downstream utilization. No minimum post index continuous eligibility was required for the assess-

ment of adverse ischemic events. All patients identified as high risk were excluded from downstream utilization and outcome measures, as CAC testing would not be considered "screening" in these patients.

Downstream Utilization and Outcome Measures

Downstream utilization and outcomes were analyzed only for patients in the non-high-risk group because prior evidence suggested that the results of CAC tests could potentially influence a reclassification of the risk and, therefore, treatment pathway of patients may be altered only in the non-high-risk group.^{5,6} Among the measures of interest for the non-high-risk patients were downstream cardiac imaging tests, coronary revascularizations, and pharmaceutical interventions in the 6 months following the index date. Cardiac imaging tests of interest included stress echocardiography, myocardial nuclear imaging, cardiac magnetic resonance imaging, diagnostic cardiac catheterization, cardiac positron emission tomography, and coronary CT angiography. The revascularizations evaluated included coronary artery bypass surgery (CABG) and percutaneous coronary intervention (PCI). All tests and interventions were identified via CPT and *International Classification of Disease, Ninth Revision, Clinical Modification (ICD-9-CM)* procedure codes in patients' medical claims. Cardiac pharmaceutical interventions included angiotensin-converting-enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), beta-blockers, calcium channel blockers, diuretics, nitrates, and statins identified from National Drug Code/General Product Identifier (NDC/GPI) coding from pharmacy claims.

Also of interest was the average total cost per month for cardiac pharmaceutical interventions. This was calculated as the sum of the total costs (plan-paid plus patient out-of-pocket costs) for cardiac pharmaceutical interventions divided by the months of prescription eligibility. Monthly costs were analyzed as a proxy for medication utilization rate.

Adverse ischemic events of interest, which were identified from *ICD-9-CM* diagnostic codes in medical claims for hospital inpatient stays lasting at least 3 days but no longer than 180 days, included acute myocardial infarction (410.x0 and 410.x1), ischemic stroke (433.x1 and 434.x1), and unstable angina pectoris (411.1x) after the index date.

Statistical Analysis

All comparisons between patients in the CAC and Reference groups were conducted with χ^2 or Fisher exact tests for categorical variables and with 2-sample *t* tests for continuous demographic variables. Wilcoxon ranked-sum test

■ **Table 1.** Demographic Characteristics of Study Population

	All			CAC			Reference			P ^a
	N/Mean	%/SD	Median	N/Mean	%/SD	Median	N/Mean	%/SD	Median	
Patients (N)	3814	100.00		2679	100.00		1135	100.00		
Age (y)	52.76	7.72	54.00	52.79	7.61	54.00	52.70	7.97	54.00	.75
18-34	70	1.84		47	1.75		23	2.03		.40
35-44	531	13.92		358	13.36		173	15.24		
45-54	1410	36.97		993	37.07		417	36.74		
55-64	1803	47.27		1281	47.82		522	45.99		
Female	1543	40.46		1092	40.76		451	39.74		.56
Region										
Northeast: CT, ME, NH, NY	555	14.55		500	18.66		55	4.85		<.0001
Midwest: OH, IN, WI, MO	1125	29.50		706	26.35		419	36.92		
South: VA, KY, GA	680	17.83		500	18.66		180	15.86		
West: CA, NV	1454	38.12		973	36.32		481	42.38		
High risk for CHD	808	21.19		540	20.16		268	23.61		.02
CCI score	0.42	0.97	0.00	0.40	0.94	0.00	0.47	1.04	0.00	.03
0 (no comorbidity)	2837	74.38		2031	75.81		806	71.01		.01
1-2	855	22.42		567	21.16		288	25.37		
3-4	87	2.28		60	2.24		27	2.38		
5 or higher	35	0.92		21	0.78		14	1.23		

CCI indicates Charlson Comorbidity Index; CHD, coronary heart disease.

^aCategorical variables: χ^2 square or Fisher tests / continuous variables: 2-sample *t* tests

was applied for cost variables. Comparisons of ischemic adverse events during follow-up between the CAC and Reference groups were conducted with Cox proportional-hazards models. All statistical analyses were performed with SAS version 9.2 (SAS Institute Inc, Cary, North Carolina). A statistical significance level of .05 was utilized.

RESULTS

Demographic Characteristics of Study Population

The total study population (N = 3814) included 2679 patients in the CAC cohort and 1135 in the control (Reference) group. Patients in the CAC and control groups were comparable in age (mean 52.7 years) and gender (40% female) distribution (Table 1). Patients in the high-risk category accounted for 20.2% and 23.5% of the CAC and Reference groups, respectively (*P* < .05). The Reference group had a greater pre existing comorbidity burden (mean Charlson Comorbidity Index [CCI] score 0.47) vs the CAC group (mean CCI score 0.40), *P* < .05), although the difference between the groups may not be clinically significant. Approximately three-fourths of the patients in each cohort had CCI scores of zero, indicating that no relevant comorbidities were present.

Post Index Interventions

Among the patients categorized as non-high-risk, similar proportions in both the CAC and Reference groups received at least 1 subsequent (not CAC) imaging test within 6 months post index (23.2% vs 23.8%, *P* = .5). Overall, the proportions were comparable between the 2 groups, regardless of the type of imaging test (Table 2). The rates of therapeutic interventions (revascularizations) were similar between the groups: CAC (0.3%) and Reference (0.65%) for CABG (*P* = .20), and 3.29% and 4.43% for PCI (*P* = .15), respectively. Medication utilization was comparable for the 2 groups, with slightly more than one-third of the patients on statins (*P* = .86), and approximately one-fifth receiving ACE inhibitors or ARBs in each group (*P* = .61). Similar monthly mean (\pm SD) total costs were associated with the pharmaceutical therapeutic interventions between 2 groups (\$31.50 [\pm \$52.79]) for CAC versus \$29.95 [\pm \$49.43] for Reference, *P* = .65).

Adverse Cardiac Events

The median follow-up periods were 689 days for CAC and 501 days for Reference. Adverse events were rare in both groups during these relatively short follow-up periods (0.85% in CAC vs 0.79% in Reference). The propor-

Table 2. Therapeutic Interventions in the 6-month Post Index Period

	CAC		Reference		P ^b
	N / Mean	% / SD	N / Mean	% / SD	
Non-high-risk patients who had 6-month medical benefit^a					
Non-high-risk patients who had 6-month medical and Rx benefit^a	1066	54.72	663	86.33	
No. of different cardiac imaging tests performed within 183 days following index date					
Did not have any testing	1496	76.80	585	76.17	.52
Had 1 type of test	364	18.69	152	19.79	
Had 2 types of test	81	4.16	26	3.39	
Had 3 or more types of test	7	0.36	5	0.65	
Stress echocardiography	257	13.19	112	14.58	.34
Myocardial nuclear imaging	187	9.60	64	8.33	.30
Cardiac magnetic resonance imaging	3	0.15	3	0.39	.36
Diagnostic cardiac catheterization	40	2.05	23	2.99	.14
Cardiac positron emission tomography	7	0.36	1	0.13	.45
Coronary CT angiography	43	2.21	13	1.69	.40
Therapeutic intervention performed within 183 days following index date					
Coronary artery bypass surgery	6	0.31	5	0.65	.20
Percutaneous coronary intervention	64	3.29	34	4.43	.15
CHD prevention medication utilization within 183 days following index date (among people with both medical and Rx benefit)					
Inhibitors or ARBs	186	17.45	122	18.40	.61
Beta-blockers	4	0.38	4	0.60	.50
Calcium channel blockers	54	5.07	38	5.73	.55
Diuretics	9	0.84	9	1.36	.31
Nitrates	16	1.50	4	0.60	.11
Statins	379	35.55	233	35.14	.86
Cost per month associated with CHD prevention medication utilization within 183 days following index date (among people with both medical and Rx benefit)					
Total	\$31.50	\$52.79	\$29.25	\$49.43	.65
Plan-paid	\$21.10	\$39.48	\$18.73	\$35.95	.75
Out-of-pocket	\$10.40	\$19.97	\$10.52	\$21.26	.30

ACE indicates angiotensin-converting-enzyme; ARB, angiotensin receptor blocker; CHD, coronary heart disease; CT, computed tomography; Rx, prescription.

^aOnly patients who had 6-month continuous medical eligibility from index date and were classified as non-high-risk for CHD were included in Table 2, regardless of whether there was an occurrence of cardiovascular event after index date.

^bP value for utilization was based on χ^2 / Fisher exact test; P value for cost was based on Wilcoxon rank-sum test.

tions of adverse cardiac events were comparable for CAC and Reference patients regardless of event type (Table 3). The age-sex adjusted incidence rate ratio for adverse events was 1.1 (95% CI, 0.36-3.38) among non-high-risk patients in the CAC versus Reference cohorts (Figure).

DISCUSSION

This study demonstrated no significant difference in the rates of subsequent cardiac imaging utilization and

therapeutic interventions, nor in the incidence of ischemic events, between the CAC and Reference cohorts. Slightly less than one-fourth of the patients in the CAC and Reference groups received a cardiac imaging test during the 6-month follow-up period, and the proportions remained comparable regardless of test type. The 2 groups had similar rates of revascularizations (PCI and CABG), as well as similar utilization patterns and costs for drug interventions, indicating no notable change in treatment patterns post CAC testing. During the follow-up period,

■ **Table 3.** Adverse Cardiac Events During Post Index Period

	CAC		Reference		P ^a
	N	%	N	%	
Patients [N]^b	2139	100.00	867	100.00	
1 to 90 days of follow-up	88	4.11	40	4.61	<.01
91 to 183 days of follow-up	109	5.10	62	7.15	
184 to 365 days of follow-up	298	13.93	227	26.18	
More than 365 days of follow-up	1644	76.86	538	62.05	
Mean follow-up time	810.9	27.03	618.6	20.62	<.01
Median follow-up time	689.0	22.97	501.0	16.70	<.01
Adverse cardiac events during the entire follow-up period	14	0.85	4	0.74	.79
Acute myocardial infarction ^c	8	0.49	2	0.37	.73
Ischemic stroke ^d	4	0.24	0	0.00	.58
Hospital admission for unstable angina pectoris ^e	4	0.24	3	0.56	.42

^aCategorical variables: χ^2 or Fisher tests; mean follow-up time: 2-sample *t* tests; median follow-up time: Wilcoxon rank-sum tests.

^bPatients were followed from the index date to the end of study period, end of plan enrollment, or first occurrence of any of adverse cardiac event, whichever occurred first. Continuous eligibility following index date was not required for this analysis. Patients classified as high risk were excluded.

^cAcute myocardial infarction is defined as hospitalization with *ICD-9-CM* diagnosis codes 410.x0 or 410.x1 and a length of stay between 3 and 183 days.

^dIschemic stroke is defined as hospitalization with *ICD-9-CM* diagnosis code 433.x1 or 434.x1, and a length of stay between 3 and 183 days.

^eUnstable angina pectoris was identified by *ICD-9-CM* diagnosis code 411.1x.

ischemic adverse events were rare in both the CAC and Reference groups.

The FRS—a multivariate statistical model incorporating age, gender, smoking status, blood pressure, cholesterol, and diabetes, among other risk factors—has been used successfully to evaluate the risk of coronary events among people without a diagnosis of heart disease.³⁸ Relative to the FRS, CAC score has been suggested as an approach that could enhance the prediction of risk in this population.^{5,21,38-40} In this study, the association between CAC scanning and subsequent cardiac imaging, revascularization, pharmaceutical-based cardiac interventions, and adverse ischemic events was assessed in a real-world setting among managed care patients. To facilitate comparison, the study included a substantive control cohort and confined the analysis to non-high-risk patients, among whom the impact of CAC measurements had the potential to be most evident.^{5,6}

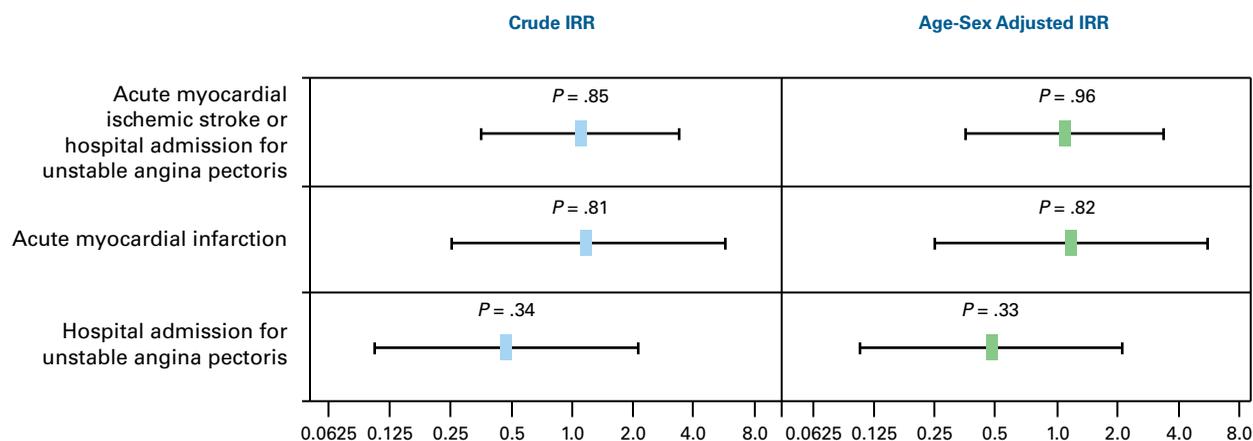
The results suggest that CAC testing did not substantially impact referrals for additional screening, therapeutic interventions, drug utilization, and costs during 6 months of follow-up. Similarly, CAC testing did not change the rate of downstream adverse events during a median follow-up of 689 and 501 days for CAC and Reference patients, respectively. Another important finding was that 1 in 5 CAC tests in the CAC group was performed on high-risk patients, and would be deemed inappropriate per ACCF/AHA guidelines.⁵ This is

probably a conservative estimate because not all factors that could result in a high-risk classification were necessarily captured with the claims-based identification criteria used in this study. This could have resulted in an understatement of the proportion of CAC scans that were inappropriate.

Evidence from prior studies has demonstrated that CAC testing alone or in combination may predict cardiac events better than available scoring systems such as the FRS and the NCEP ATP III guidelines.^{12,29-31} Yet it does not appear that improved predictive ability influences clinical decision making. One possible explanation might be that the marginal improvements associated with CAC over existing algorithms were not sufficient to persuade providers to modify treatment patterns for asymptomatic patients.

The study by Polonsky et al showed only a modest improvement in the predictive accuracy (as measured by area under the receiver operating characteristic [ROC] curve) when CAC scores were added to FRS scores.³⁰ The result for FRS score alone was 0.76 (95% CI, 0.72-0.79), which increased to 0.81 (95% CI, 0.78-0.84) ($P < .001$) following the addition of CAC to scores.³⁰ An improvement of such modest size might not have clinical consequences. In addition, most of the reclassifications were from moderate to low risk, which might also not carry clinical importance.³⁰ In another study, after adding CAC scores to FRS and NCEP ATP III scores,

■ **Figure.** Incidence Rate Ratios (IRRs) for Adverse Cardiac Events During Follow-up Period



Erbel et al showed that the areas under the ROC curve improved from 0.681 to 0.749 ($P < .003$) and from 0.653 to 0.755 ($P = .0001$), respectively.³¹ While this represents better improvement following the addition of CAC scores relative to results from the Polonsky et al study, it was still in the modest range.^{30,31}

The current study relied on secondary (administrative claims) data, from which it was not possible to access patients' actual CAC scores. Based on prior studies^{12,29-31} with CAC scans, however, it would seem reasonable to expect the risk status of some of the patients in the CAC cohort to change based on their CAC scores. The evidence in this study, however, did not indicate notable modifications in how patients utilized healthcare resources nor in how providers managed their patients' pharmaceutical interventions and cardiac procedures post scan. This finding is consistent with the prevailing view that no beneficial evidence is available on how CAC testing influences treatment.⁴¹

Nonetheless, earlier studies and AHA scientific statements⁴² have suggested a role for CAC as an independent predictor of cardiovascular events. The findings of our study have implications for the management of treatment in light of concerted efforts by payers to comprehend how procedures such as CAC testing may be integrated into their offerings in an effort to improve health outcomes.⁴³ The results of diagnostic procedures may influence changes or improvements in treatment patterns. When treatment patterns remain largely unchanged regardless of test results, as was observed with CAC scans in this study, however, questions about the value of the test may have merit.

Limitations

As is typical of claims-based data analyses, which have inherent limitations including the lack of clinical indicators on disease severity, the results of this study should be interpreted with caution. This consideration could have directly affected the identification of high-risk patients (and consequently, non-high-risk patients), which was accomplished exclusively through the presence of specific pre existing diagnoses within medical claims. It is not inconceivable that that some patients may have been misclassified despite this solid methodological approach—although we believe that the portion would likely be miniscule. Furthermore, any misclassification was more likely in the low-risk population versus the high-risk. Part of the reason for this is that if low- and intermediate-risk patients had cardiac events prior to the initiation of the health plan coverage captured in our database, such patients would truly belong in the high-risk category. Nonetheless, the study findings were unlikely to be affected because they focused on treatment pattern differences between these 2 populations.

A broader limitation was the inability to identify and account for any patients in the control group who were denied the CAC scan as a noncovered benefit but still received the test via self-pay or other financial arrangements. Finally, although the median follow-up durations—689 days for CAC and 501 days for Reference—were substantially longer than the follow-up periods for the therapeutic interventions, they may still not represent sufficient time in which to observe a comprehensive range of adverse events following the establishment of patients' risk status with CAC procedures, as was observed in earlier

studies and suggested by the FRS 10-year mortality risk for cardiovascular diseases.^{12,34,44}

We recognized that short follow-up times would present a challenge when this study was initiated with administrative claims as the primary data source. Enrollees often change health plans as they move from one job to another, so it was anticipated that within claims, the researchable follow-up periods could well be of shorter duration. Still, events and risks within a shorter time frame for a general managed care population would be an important addition to the field, and could fill an important gap because incidences over a shorter follow-up duration have only been reported for specific populations, such as patients with diabetes, or breast cancer. As a result, the findings in this study, while limited to the impact of CAC scans on treatment patterns in the short term rather than long term, may address an important gap. We believe that future studies using longer follow-up periods would contribute important information on the long-term impact of CAC scans on subsequent treatment patterns.

Finally, although this study only included working, commercially insured subjects, differences in socioeconomic circumstances could have influenced the type and quality of healthcare insurance they accessed. Such differences were likely minimized, however, because all coverage was employer-determined. In this study, the procedure was not covered by employers who concurred with the health plan's position that CAC did not satisfy its medical necessity criteria. Also included, however, were patients from self-insured employer groups with independent medical policies that allowed CAC scans as a covered benefit. Such extended coverage could sometimes be reflected by the business and financial circumstances of employers; neither that nor the socioeconomic status of individual patients was determinable from our study data. This could have introduced a bias, although one of miniscule significance given that all patients were working and were receiving employer-provided health insurance.

CONCLUSIONS

In this study, non-high-risk patients having CAC scans were not associated with fewer ischemic events nor reduced use of additional imaging tests. While this study was designed to evaluate the non-high-risk category in which the predictive value of CAC was expected to be greatest, almost no difference was seen in additional testing, cardiac interventions, drug utilization, and adverse event rates between the CAC and Reference groups. In addition, a substantial number of high-risk patients inap-

ropriately received CAC scans, which provided no additional predictive value, exposed them unnecessarily to radiation harm, and increased their healthcare costs. This will undoubtedly have policy implications for payers and providers. Additional studies in real-world settings over longer durations could help to further elucidate how or if CAC testing modifies treatment patterns post scan.

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