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Use of Coronary Computed Tomographic Angiography Findings to Modify Statin and Aspirin Prescription in Patients with Acute Chest Pain

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Abstract

Coronary CT angiography (CCTA) is used in low-intermediate chest pain patients presenting to the Emergency Department (ED) for its reliability in excluding acute coronary syndrome (ACS). However, its influence on medication modification in this setting is unclear. We sought to determine whether knowledge of CCTA-based CAD was associated with change in statin and aspirin prescription. We utilized the CCTA arm of the ROMICAT II multicenter, randomized control trial (R-II) and comparison cohort from the observational ROMICAT I cohort (R-I). In R-II, subjects were randomly assigned to CCTA to guide decision-making, whereas in R-I patients underwent CCTA with results *blinded* to caregivers and managed according to standard-care. Our

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final cohort consisted of 277 subjects from R-I and 370 from R-II. ACS rate was similar (6.9% vs 6.2% respectively, $p=.75$). For subjects with CCTA- detected obstructive CAD without ACS, initiation of statin was significantly higher after disclosure of CCTA results (0% in R-I vs 20% in R-II, $p=.009$). Conversely, for subjects without CCTA-detected CAD, aspirin prescription was lower with disclosure of CCTA results (16% in R-I vs 4.8% in R-II, $p=.001$). However, only 68% of subjects in R-II with obstructive CAD were discharged on statin and 65% on aspirin. *In conclusion*, physician knowledge of CCTA results leads to improved alignment of aspirin and statin with the presence and severity of CAD, though still many patients with CCTA-detected CAD are not discharged on aspirin or statin. Our findings suggest opportunity for practice improvement when CCTA is performed in the ED.

Keywords

computed tomography; medication; prevention; statin; coronary artery disease; emergency department

INTRODUCTION

The presence and severity of CCTA-detected coronary artery disease (CAD) provides prognostic value for cardiovascular events and mortality.¹⁻⁹ Despite this strong association between CAD detected by CCTA and outcomes, there is limited evidence on whether CCTA findings can influence the prescription of cardiovascular preventive medical therapies such as aspirin and statin. By using both the observational Rule Out Myocardial Infarction using Computed Angiographic Tomography (ROMICAT) I cohort in which low-intermediate risk acute chest pain subjects underwent CCTA in the ED with *results blinded to caretakers*, and the multi-center ROMICAT II cohort with similar inclusion criteria but with CCTA *results disclosed to providers*, we sought to determine whether physician knowledge of CCTA results would result in better alignment of medical therapy prescription with the presence of CAD on CCTA.^{10,11}

METHODS

We utilized data from two trials that included low-intermediate risk subjects presenting with acute chest pain to the ED and suspicion for ACS^{10,11}: (1) the observational ROMICAT I (R-I) study in which 368 subjects were managed according to standard care, but underwent CCTA with results blinded to healthcare providers, and (2) the multicenter, randomized controlled ROMICAT II (R-II) trial in which 501 of the enrolled 1000 subjects were randomized to undergo CCTA, the results of which were disclosed to care providers. Thus, the two studies differed as to whether CCTA results were disclosed to care providers. Our institutional review board approved these study protocols and all patients provided informed consent.

Although the trials' inclusion criteria were very similar, there were some differences in exclusion criteria. Therefore, in order to create comparable cohorts, we excluded subjects from the R-I trial that would have met exclusion criteria for the R-II trial. More precisely, we excluded patients who were <40 years or >74 years of age ($n=65$), and those who had a self-

reported history of CAD (n=17). See flow diagram (Figure 1). In the R-II cohort we excluded 28 subjects that were randomized to the CCTA arm but did not undergo CCTA. Finally, we excluded subjects with incomplete medication data (9 in R-I and 103 in R-II). Thus, the final study population consisted of 277 subjects from R-I, and 370 subjects from R-II.

Two physicians blinded to individual patient CCTA results performed medication data collection. Admission and discharge medication prescription data was collected for each patient by systematically reviewing medical records during the index ED visit and hospitalization, including ED admission and discharge notes, as well as inpatient admission and discharge summary if the patient was admitted to the hospital (143 subjects in R-I and 82 subjects in R-II). Contraindication to aspirin (aspirin) therapy was defined as a severe aspirin allergy (anaphylaxis, angioedema, etc), or documented intolerance due to a history of major gastrointestinal bleed (requiring transfusion) or intracranial bleed. Contraindication to statin therapy was defined as a history of statin-induced myopathy, rhabdomyolysis, or other documented statin intolerance.

Risk factors were assessed at the time of subject enrollment, based on self-report as well as review of medical records for the index hospitalization. Hypertension was defined as a systolic blood pressure of at least 140 mm Hg or diastolic blood pressure of at least 90 mm Hg or current antihypertensive treatment. Diabetes mellitus was defined as a fasting plasma glucose ≥ 126 or treatment with a hypoglycemic agent. Peripheral vascular disease was defined as having a history transient ischemic attack, cerebrovascular event, or peripheral arterial disease. Family history of CAD was defined as having a first-degree female (<65 years of age) or male (<55 years of age) relative with a documented history of myocardial infarction or sudden cardiac death. Subjects were classified as smokers if they had smoked at least 1 cigarette per day in the year before the study. Lipid profiles were collected as part of the study protocol in R- I subjects at the time of admission, and collected retrospectively in R-II subjects (admission laboratory data or most recent lipid panel within 1 year prior to admission). The 10-year Framingham Risk Score was calculated for each patient, and patients were stratified into the following three groups: $<10\%$, $10\text{--}20\%$, and $>20\%$ for 10-year risk of a cardiovascular event. The definition of ACS during the index hospitalization in both the R-I and R-II cohorts have been published previously.^{10–12} Briefly, in both trials, ACS was defined as either an acute myocardial infarction or unstable angina pectoris. Establishment of this diagnosis was based on an outcome panel of experienced physicians who reviewed prospectively collected patient clinical information as well as medical records during the index hospitalization. The outcome panel was blinded to the findings of CCTA, and disagreement was resolved by the adjudication of an additional cardiologist.

Based upon CCTA results, patients were categorized into having no CAD, non-obstructive CAD (most severe stenosis $<50\%$), or obstructive CAD (most severe stenosis $\geq 50\%$ or inconclusive results). In the R-I cohort, this analysis was performed in a core laboratory, while in the multi-center R-II trial, we relied on individual site interpretation data, to evaluate the influence of CCTA results on medication prescription.

Baseline demographics of the two patient cohorts were compared with use of independent sample t-test for continuous variables, Fisher's exact test for categorical variables, and the Wilcoxon rank-sum test for ordinal variables. Continuous variables were expressed as mean $\pm$ SD; categorical variables were described by frequency. Step-wise logistic regression analysis was employed to assess the association between disclosure of CCTA results (R-II cohort) and changes in medical therapy, controlling for confounding baseline variables. All analyses were performed using Stata (Version 13.1, StatCorp, College Station, Texas). For all analyses, a two-tailed p value < 0.05 was required to reject the null hypothesis.

RESULTS

Baseline characteristics of our R-I (52.3 ± 7.9 years, 58% male) and R-II (53.8 ± 8.0 years, 53% male) comparable cohorts are shown in Table 1. Overall clinical characteristics were similar between the cohorts. Furthermore, there was a higher prevalence of diabetes and hypertension in the R-II cohort, while mean total cholesterol and LDL-cholesterol were higher in R-I. There were no significant differences in Framingham Risk category and ACS rate between R-I and RII. Complete admission and discharge prescription data for aspirin and statin were obtainable in 277 subjects in the R-I cohort and 370 subjects in the R-II cohort. (Figure 1) Of these, a total of 14 subjects were identified as having a contraindication to aspirin, and 14 subjects were identified as having a contraindication to statin. (See Table 2) Overall, there was no significant difference in the overall change in aspirin or statin therapy from admission to discharge between the R-I and R-II cohorts. Most subjects who had a change in medical therapy from admission to discharge were "upgraded" as opposed to "downgraded"; in other words, they were not on aspirin or statin at admission, but were prescribed the medication at discharge.

Overall, upgrade of statin therapy occurred in 9.0% (33/365) of subjects in R-II and 6.7% (18/268) of subjects in R-I. Although the change in statin therapy from admission to discharge was not significantly different between the two cohorts, there was a higher rate of statin therapy at both admission and discharge in the R-II cohort compared to the R-I cohort [admission: 31% vs 23%, $p=.038$; discharge: 40% vs 28%, $p=.003$]. The distribution of CCTA-detected CAD findings is shown in Table 3. Roughly half of subjects in both cohorts had CCTA-detected CAD. Obstructive CAD was found in 16% of R-I and 25% of R-II individuals.

Statin Therapy

In subjects with *CCTA-detected obstructive CAD*, 30% of subjects in the R-II cohort were started on statin therapy, while 23% of subjects in the R-I cohort were started on statin therapy. (Table 4) Stratifying the subjects with obstructive CAD by the presence or absence of clinical ACS unveiled a marked difference in statin prescription between the R-I and R-II cohorts. (Table 5) Specifically, subjects with obstructive CAD without ACS were significantly more likely to be prescribed statin therapy in R-II compared to R-I [upgrade to statin: 20% (14/69) versus 0% (0/27), $p=.009$]. Similar findings were noted when only including subjects who were admitted to the hospital. There was no difference in rates of statin prescription in the subjects with both obstructive CAD and ACS. Among patients with

no CAD and non-obstructive CAD, there were no significant differences between the R-I and R-II cohorts with respect to change in statin therapy.

Aspirin Therapy

In subjects with *no CAD or non-obstructive CAD*, compared to the R-I cohort there was a significant reduction in prescription of aspirin therapy in the R-II cohort (no CAD: 16% versus 5% upgrade, $p=.001$; non-obstructive CAD: 22% versus 11% upgrade, $p=.046$). For obstructive CAD, there was no significant difference in aspirin initiation between the R-I and R-II cohorts.

We performed multiple univariable regression analyses with upgrade in statin therapy as the dependent variable and baseline characteristic confounding factors and an interaction term of having obstructive CAD without ACS in the ROMICAT II trial as independent variables. Parameters significantly associated with an upgrade in statin therapy were: having obstructive CAD but not ACS in the ROMICAT II trial (the interaction term), having ACS, an increase in FRS category, having obstructive CAD, and increased LDL ($p<.001$).

To control for demographic and clinical differences between patients in the two trials, we built two multivariable logistic regression models that included all parameters of the univariable analyses with a p -value <0.2 . Then, in a stepwise approach, we excluded variables with a p -values >0.1 . In both models we included a binary variable for being a patient in the ROMICAT II trial while in the second model we included also the interaction term. In both models having ACS and increasing LDL cholesterol were associated with a higher chance of receiving an upgrade in statin therapy. In the first model having nonobstructive or obstructive CAD or being in R- II positively correlated with an upgrade in statin therapy. However, these relationships disappeared when including the interaction term in model 2.

DISCUSSION

The main finding of this study is that disclosure of CCTA findings in patients presenting to the ED with acute chest pain is associated with differential changes in the prescription of statin and aspirin therapy between presentation and discharge. Thus, a 20% increase in statin prescription occurred in patients with obstructive CAD detected by CCTA without ACS but no significant change in those without obstructive CAD. A decrease in aspirin prescription was seen in patients with either no CAD or nonobstructive CAD, whereas no change was observed in those with obstructive CAD. Furthermore, the discharge prescription rates of 68% for statins and 65% for aspirin in obstructive CAD without ACS suggest that many acute chest pain patients presenting to the ED who are noted to have CAD on CCTA are not initiated on aspirin or statin therapy.

This is the first study to show the influence of CCTA findings on medical therapy prescription in the management of low-intermediate risk acute chest pain patients presenting to the emergency department. In an era in which multiple diagnostic testing algorithms could potentially be used for triaging such patients, the stronger association of CCTA results to

influence downstream preventive medical therapy compared to standard care approaches is particularly appealing.

Similar to prior studies we have shown a graded change in medical therapy that corresponds with the degree of CAD severity on CCTA.^{13–15} However, the major strength of our analysis is the comparison with a control group that underwent standard of care management, but also had undergone CCTA with the results blinded to providers (R-I). This allowed us to truly evaluate the impact of CCTA results beyond current standard of care in specific subgroups of CAD severity.

Prior studies that have shown an association between the severity of CAD detected by CCTA and prescription of medical therapy in stable patients were limited by this lack of control group that underwent standard care testing and management.^{13,14} In the SPARC trial, although CCTA was compared to other imaging modalities with respect to downstream medication impact, the anatomic CAD status of subjects undergoing SPECT or PET was not definitively known unless patients underwent subsequent CCTA or cardiac catheterization. Furthermore, this registry-based study evaluated patients with intermediate to high likelihood of coronary artery disease, including nearly a third of subjects with diabetes mellitus.¹⁵

Despite provider knowledge of CCTA-detected CAD in our R-II cohort, only 65% of subjects with obstructive CAD were discharged on aspirin, and only 68% were discharged on statin therapy. However, rates of discharge medical therapy were significantly higher in those with obstructive CAD *and* ACS (96% for statin and 100% for aspirin) than those without ACS (59% for statin and 54% for aspirin), suggesting ready acceptance of appropriate secondary prevention therapies for patients who have had an ACS event according to current guidelines.^{16,17} Adoption of secondary prevention aspirin and statin therapy for those with obstructive CAD but not ACS appears to less well accepted despite numerous studies showing an adverse prognosis in patients with CCTA-detected obstructive CAD.^{1–9}

When CCTA results were disclosed to providers, those patients with either no CAD or nonobstructive CAD were initiated on aspirin significantly less often. This suggests that care providers are less likely to initiate aspirin if they are aware that a patient has either no CAD or non-obstructive CAD. This cautious decision-making appears to be in line with contemporaneous concerns regarding whether potential long-term bleeding risks outweigh the benefits of institution aspirin in the primary prevention setting. In addition, subjects without any significant CAD were less likely to be evaluated by an internist or cardiologist; therefore it is conceivable that decisions regarding such as instituting preventive guideline based therapy were deferred to the outpatient setting.

We evaluated medical therapy at the time of admission and discharge only, and did not have any systematic follow-up data regarding medication prescription in the outpatient setting. Temporal trends in treatment patterns may have contributed to differences in medical therapy prescription between the R-I and R-II cohorts, which were recruited approximately four years apart. Further, although we describe specific associations between medical

therapy modification and CCTA-based CAD findings, it is still possible that other unmeasured factors may have influenced a decision to initiate aspirin or statin therapy.

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Figure 1.

Flow chart summarizing selection of patient population utilized in the analysis. CCTA = coronary computed tomography angiography; MI = Myocardial Infarction; R-I = ROMICAT I; R-II = ROMICAT II

Table 1

Baseline Patient Characteristics Stratified by Trial

Variable	R-I (N = 277)	R-II (N = 370)	P value
Age (years), mean $\pm$ SD	52.3 $\pm$ 7.9	53.8 $\pm$ 8.0	0.026
Men	161 (58%)	196 (53%)	0.20
Hypertension	105 (38%)	194 (52%)	<0.001
Diabetes mellitus	29 (11%)	64 (17%)	0.017
Dyslipidemia	105 (38%)	163 (44%)	0.13
Former or current smoker	141 (51%)	183 (50%)	0.75
Family history of premature CAD	74 (27%)	98 (27%)	1.00
Cholesterol (mg/dl), mean $\pm$ SD			
Total	200 $\pm$ 40	188 $\pm$ 39	<0.001
High-Density Lipoprotein	52 $\pm$ 15	50 $\pm$ 18	0.41
Low-Density Lipoprotein	118 $\pm$ 36	108 $\pm$ 37	0.003
Framingham Risk	(N = 252)	(N = 243)	0.91
<10%	191 (76%)	182 (75%)	
10%-20%	48 (19%)	46 (19%)	
>20%	13 (5.2%)	15 (6.2%)	
Acute Coronary Syndrome	19 (6.9%)	23 (6.2%)	0.75

CAD = Coronary Artery Disease; N = Number; SD = Standard Deviation; R-I = ROMICAT I; R-II = ROMICAT I

Table 2

Admission and Discharge Medical Therapy Stratified by Trial

	R-I (N = 277)	R-II (N = 370)	P value
Contraindication to Aspirin	6 (2.2%)	8 (2.2%)	1.00
Aspirin Therapy*	N = 271	N = 362	
Admission Medication	64 (24%)	88 (24%)	0.85
Discharge Medication	120 (44%)	143 (40%)	0.25
Change in Medication	58 (21%)	59 (16%)	0.10
Upgrade in Therapy	57 (21%)	57 (16%)	0.095
Downgrade in Therapy	1 (0.4%)	2 (0.6%)	1.00
Contraindication to Statin	9 (3.3%)	5 (1.4%)	0.11
Statin Therapy*	N = 268	N = 365	
Admission Medication	62 (23%)	112 (30.7%)	0.038
Discharge Medication	76 (28%)	145 (39.7%)	0.003
Change in Medication	18 (6.7%)	33 (9.0%)	0.31
Upgrade in Therapy	16 (6.0%)	33 (9.0%)	0.18
Downgrade in Therapy	2 (0.8%)	0 (0.0%)	0.18

* Excluding patients with contraindication. R-I = ROMICAT I; R-II = ROMICAT II

Table 3

Coronary CT Angiography Findings Stratified by Trial

	R-I (N = 277)	R-II (N = 370)	P value
Presence and Severity of CAD			0.02
No CAD	144 (52%)	172 (47%)	
Non-Obstructive CAD (<50%)	89 (32%)	106 (29%)	
Obstructive CAD (>50% or indeterminate)	44 (16%)	92 (25%)	
CCTA Results Disclosed	No	Yes	

CAD = Coronary Artery Disease; N = Number; R-I = ROMICAT I; R-II = ROMICAT II

Admission and Discharge Medical Therapy Stratified by Trial and Coronary CT Angiography Findings

Table 4

	No CAD			Non-obstructive CAD			Obstructive CAD		
	R-I (N = 144)	R-II (N = 172)	P	R-I (N = 89)	R-II (N = 106)	P	R-I (N = 44)	R-II (N = 92)	P
Aspirin *	(N = 140)	(N = 168)		(N = 87)	(N = 102)		(N = 44)	(N = 92)	
Admission Medication	30 (21%)	40 (24%)	0.68	21 (24%)	25 (25%)	1.00	13 (30%)	23 (25%)	0.68
Discharge Medication	52 (37%)	47 (28%)	0.09	40 (46%)	36 (35%)	0.14	28 (64%)	60 (65%)	0.85
Change in Medication	24 (17%)	9 (5.4%)	0.001	19 (22%)	11 (11%)	0.046	15 (34%)	39 (4%)	0.45
Upgrade in Therapy	23 (16%)	8 (4.8%)	0.001	19 (21.8%)	11 (11%)	0.046	15 (34%)	38 (41%)	0.46
Downgrade in Therapy	1 (0.7%)	1 (0.6%)	1.00	0 (0.0%)	0 (0.0%)	—	0 (0.0%)	1 (1.1%)	1.00
Statin *	(N = 139)	(N = 172)		(N = 86)	(N = 102)		(N = 43)	(N = 91)	
Admission Medication	22 (16%)	49 (29%)	0.01	24 (28%)	28 (28%)	1.00	16 (37%)	35 (39%)	1.00
Discharge Medication	21 (15%)	51 (30%)	0.003	29 (33%)	32 (31%)	0.76	26 (61%)	62 (68%)	0.44
Change in Medication	3 (2.2%)	2 (1.2%)	0.66	5 (5.8%)	4 (3.9%)	0.73	10 (23%)	27 (30%)	0.54
Upgrade in Therapy	1 (0.7%)	2 (1.2%)	1.00	5 (5.8%)	4 (3.9%)	0.73	10 (23%)	27 (30%)	0.54
Downgrade in Therapy	2 (1.4%)	0 (0.0%)	0.20	0 (0.0%)	0 (0.0%)	—	0 (0.0%)	0 (0.0%)	—

* Excluding patients with contraindication

Admission and Discharge Medical Therapy Stratified by Trial and Acute Coronary Syndrome for Obstructive Coronary Artery Disease Patients

Table 5

	No ACS		ACS		P value
	R-I (N = 28)	R-II (N = 70)	R-I (N = 16)	R-II (N = 22)	P value
Aspirin*					
Admission Medication	8 (29%)	17 (24%)	5 (31%)	6 (27%)	1.00
Discharge Medication	13 (46%)	38 (54%)	15 (94%)	22 (100%)	0.42
Change in Medication	5 (18%)	23 (33%)	10 (63%)	16 (73%)	0.73
Upgrade in Therapy	5 (18%)	22 (31%)	10 (63%)	16 (73%)	0.73
Downgrade in Therapy	0 (0.0%)	1 (1.4%)	0 (0.0%)	0 (0.0%)	—
Statin*					
Admission Medication	11 (41%)	27 (39%)	5 (31%)	8 (36%)	1.00
Discharge Medication	11 (41%)	41 (59%)	15 (94%)	21 (96%)	1.00
Change in Medication	0 (0.0%)	14 (20%)	10 (63%)	13 (59%)	1.00
Upgrade in Therapy	0 (0.0%)	14 (20%)	10 (63%)	13 (59%)	1.00
Downgrade in Therapy	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	—

* Excluding patients with contraindication