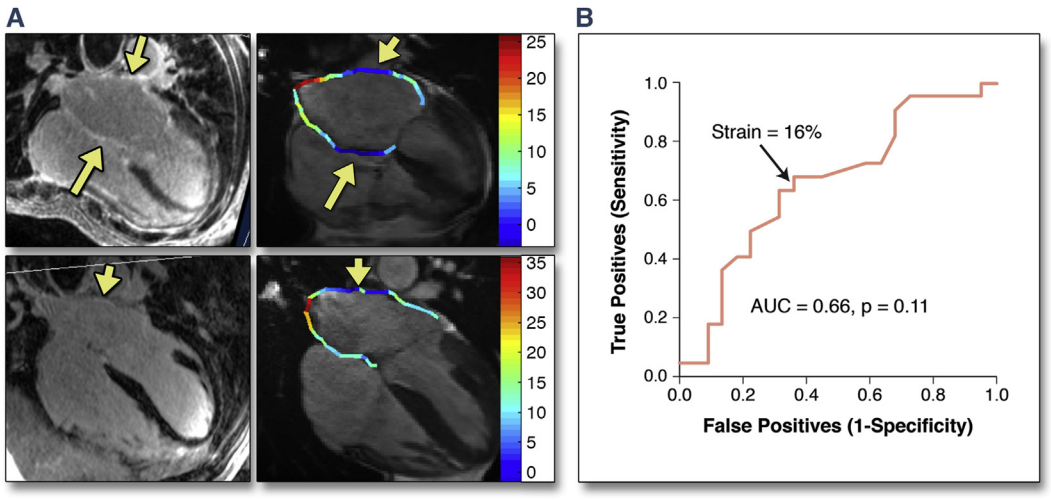
FIGURE 1 LGE Enhancement Is Associated With Low Regional Strain



(A) Reformatted 3-dimensional late gadolinium enhancement (LGE) volumes show LGE enhancement **(left)** located in sites of low strain **(right)** in 2 atrial fibrillation subjects. Strain (in %) is color-coded. **(B)** Using receiver-operating characteristic analysis, strain <16% best predicts LGE enhancement (area under the curve [AUC] = 0.66, p = 0.11, not significant).

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http://dx.doi.org/10.1016/j.jcmg.2016.01.015

Please note: This work was supported by in part by grants from the National Institutes of Health (National Heart, Lung, and Blood Institute R21 HL098573, R21 HL103463, and 1R01HL122560). The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Statin Use Is Associated With Fewer High-Risk Plaques on Coronary CT Angiography

Although there is an ongoing debate whether the detection of individual high-risk plaques (HRP) may help to improve patient outcomes, emerging data from coronary computed tomography angiography (CTA) studies suggest that the presence of HRP features (i.e., positive remodeling and low CT plaque attenuation) is independently associated with a 10- to 20-fold increased risk for future acute coronary syndrome (1). Statin therapy, perhaps via anti-inflammatory properties, may have favorable effects on coronary atherosclerotic plaque composition and lead to plaque stabilization by accelerating plaque calcification and regression of HRP features such as positive remodeling and low CT plaque attenuation (2,3). We performed an observational cross-sectional subanalysis of the ROMICAT (Rule Out Myocardial Ischemia/Infarction Using Computer Assisted Tomography) II trial to determine whether patients on statin therapy had lower prevalence of HRP as compared to those not on statins.

TABLE 1 Unadjusted and Adjusted Associations Between Admission Statin Use and High-Risk Plaque on Coronary CTA for Patients With Any CAD

	High-Risk Plaque			
	Univariable Analyses		Multivariable Analysis	
	OR (95% CI)	p Value	Adjusted OR (95% CI)	p Value
Statin at admission	0.34 (0.16–0.72)	0.005	0.29 (0.12–0.71)	0.007
Age, yrs	0.97 (0.93–1.01)	0.109	0.97 (0.93–1.01)	0.167
Female	0.36 (0.17–0.75)	0.006	0.44 (0.20–0.97)	0.043
Number of risk factors*	0.76 (0.54–1.05)	0.100	0.75 (0.51–1.09)	0.128
Significant CAD, ≥50% stenosis	7.03 (3.26–15.12)	<0.001	6.87 (2.55–18.48)	<0.001
Total plaque volume, per 100 mm^3	1.08 (1.03–1.14)	0.002	1.03 (0.97–1.11)	0.332

N = 222 observations. *Risk factors include arterial hypertension, diabetes mellitus, smoking, and family history of CAD.
CAD = coronary artery disease; CI = confidence interval; CTA = computed tomography angiography; OR = odds ratio.

We included patients with suspected acute coronary syndrome in the emergency department who were enrolled in ROMICAT II, randomized to coronary CTA and who had diagnostic image quality. A detailed description of the patient population and coronary CTA image acquisition of the ROMICAT II trial has been reported previously (4). Data on cardiovascular (CV) risk factors (hypertension, diabetes mellitus, current smoker, family history of coronary artery disease [CAD]) and whether patients were on statin therapy at study entry were collected. The coronary CTA datasets were assessed for the presence of obstructive CAD (>50% stenosis), noncalcified and calcified coronary atherosclerotic plaque, and HRP (defined as the presence of positive remodeling, low CT attenuation plaque, napkin-ring sign, and/or spotty calcium) as described previously (5). Patients with incomplete admission statin data, contraindication to statin, or no CAD on coronary CTA were excluded.

Of the 222 eligible patients (mean age 56.0 ± 7.9 years, 35% female), 34% (n = 75) were on statins on admission, 84% (n = 186) had nonobstructive and 16% (n = 36) had obstructive CAD, and 25% (n = 56) had high-risk plaque. Patients with statins on admission were older (mean age 58.5 ± 7.9 years vs. 54.7 ± 7.7 years; p = 0.001), had a higher mean number of CV risk factors (1.9 ± 1.0 vs. 1.4 ± 0.9; p = 0.001), yet were less likely to have any HRP (13.3% vs. 31.3%; p = 0.003), despite similar proportions of obstructive CAD (16.0% vs. 16.3%; p = 1.000) and nonobstructive CAD (84.0% vs. 83.7%; p = 1.000). After multivariate adjustment for age, sex, number of CV risk factors, total plaque volume, and obstructive CAD, patients on statins were 3× less likely to have HRP than were patients not on statins (odds ratio [OR]: 0.29; 95% confidence interval [CI]: 0.12 to 0.17; p = 0.007) (Table 1). Similar results were seen in patients with nonobstructive CAD (HRP in patients on statins vs. those not on statins: 6.4% vs. 24.4%, respectively; p = 0.002). This association was maintained after multivariate logistic regression analysis (OR: 0.25; 95% CI: 0.08 to 0.80; p = 0.019).

In contrast, patients on statins, as compared to those not on statins, were more likely to have calcified plaque present (93.3% vs. 76.2%; p = 0.001). Similarly, patients with nonobstructive CAD on statins were more likely to have coronary artery calcium than those not on statins (92.1% vs. 72.4%; p = 0.002). After multivariable analysis, the association between coronary artery calcium score and statin was attenuated (OR: 0.92; 95% CI: 0.40 to 2.12; p = 0.860).

Overall, these observational data suggest that patients presenting to the emergency department with suspicion of acute coronary syndrome who are on statin therapy are less likely to have HRP than are those not on statin therapy, independent of the presence of obstructive CAD and CV risk factors. We expand previous observations that statin therapy alters the natural history of composition and morphology of coronary atherosclerosis by CT and intravascular ultrasound, which may be a mechanism by which statins lead to a reduction in CV events, to the setting of coronary CTA in patients with acute chest pain.

ACKNOWLEDGMENTS Travis Hallett, BA, and Jakob Park assisted in the literature review.

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http://dx.doi.org/10.1016/j.jcmg.2016.02.020

Please note: This work was supported by grants from the National Heart, Lung, and Blood Institute (U01HL092040 and U01HL092022). The contents of this letter are solely the responsibility of the authors and do not represent the official views of the National Heart, Lung, and Blood Institute or the U.S. Department of Health and Human Services. Dr. Woodard has received research support from Bayer and Astellas; and funding from the National Institutes of Health. Dr. Truong has received support from the National Institutes of Health/National Heart, Lung, and Blood Institute (K23HL098370 and L30HL093896), St. Jude Medical, American College of Radiology Imaging Network, and Duke Clinical Research Institute. Dr. Ferencik has received grant support from the American Heart Association (13FTF16450001). Dr. Hoffmann has received grant support from National Institutes of Health/National Heart, Lung, and Blood Institute, HeartFlow Inc., Siemens Healthcare, American College of Radiology Imaging Network, and Genentech. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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High Platelet Reactivity and Intrastent Thrombi Assessed by OCT After DES

High platelet reactivity (HPR) on clopidogrel may be related to stent thrombosis after drug-eluting stent (DES) implantation (1). To investigate the relationship between HPR and intrastent thrombi following DES implantation, 202 lesions treated with DES from 109 patients were studied. Coronary angiography and optical coherence tomography (OCT) examination were performed as parts of routine follow-up examination at our institution except for the presence of renal dysfunction or congestive heart failure. OCT imaging and platelet function test were performed at 6 to 9 months (median 202 days). Dual antiplatelet therapy with aspirin and clopidogrel was started before stent implantation and continued for at least 1 year. In patients with acute coronary syndrome who underwent emergent coronary intervention, a loading dose of clopidogrel (300 mg) was started on arrival to the emergency department and a maintenance dose (75 mg) was continued thereafter. In patients with stable angina pectoris who underwent elective coronary intervention, a maintenance dose (75 mg) of clopidogrel was started at least 2 weeks before intervention and continued thereafter. Neither prasugrel nor ticagrelor was used because they were not commercially available at the time of this study.

By OCT, intrastent thrombus was defined as a mass (≥100 μm) with an irregular surface attached to the vessel wall or stent struts that protrude into the lumen (2). Stent and lumen areas were measured, and neointimal area was calculated as stent area minus the lumen area at the minimal stent area site. Blood samples for platelet function tests were obtained during cardiac catheterization and measured within 3 h after OCT imaging using the VerifyNow system (Accumetrics, San Diego, California). The platelet reactivity to adenosine diphosphate was quantified as $P2Y_{12}$ reaction unit (PRU). HPR was defined as PRU ≥230 rather than ≥208, because our study patients were an East-Asian population who have different thrombogenicity from Western populations and thus have different cutoff values (1–3). Data are presented as mean ± SD for continuous variables and as frequency (%) for categorical variables. Student *t* test was used to compare continuous variables and the chi-square test or Fisher exact test was used to compare categorical variables. For lesion-based comparison, no adjustments were made for evaluation of multiple lesions within individuals. Statistical analysis was performed with the SPSS version 22.0 for Windows (SPSS Inc., Chicago, Illinois), and $p < 0.05$ was considered statistically significant.

HPR was documented in 35 patients (32%). Angiography and OCT were performed at 292 days (interquartile range [IQR]: 272 to 545) in patients with HPR and 296 days (IQR: 271 to 595) in patients without HPR ($p = 0.876$). The time from the last dose of clopidogrel to VerifyNow testing was similar between the 2 groups (249 ± 150 min vs. 191 ± 134 min). **Table 1** summarizes patients and lesion characteristics.