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Differences in the association of total versus local coronary artery calcium with acute coronary syndrome and culprit lesions in patients with acute chest pain: The coronary calcium paradox

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Abstract

Background and aims—Total coronary artery calcium (CAC) burden is associated with an increased cardiovascular risk, while local CAC may represent stable plaques. We determined differences in relationship of total CAC with acute coronary syndrome (ACS) and local CAC with culprit lesions in patients with suspected ACS.

Methods—We performed computed tomography (CT) for CAC and CT angiography to assess the presence of significant stenosis and high-risk plaque (positive remodeling, low CT attenuation, napkin-ring sign, spotty calcium) in 37 patients with ACS and 223 controls. Total and segmental Agatston score were measured. Culprit lesions were assessed in subjects with ACS.

Results—Patients (n=260) with vs. without ACS had higher total CAC score (median 229, 25th–75th percentile 75–517 vs. 27, 25th–75th percentile 0–99, $p<0.001$), higher prevalence of significant stenosis (78% vs. 7%, $p<0.001$) and high-risk plaque (95% vs. 59%, $p<0.001$). In those with ACS, culprit (n=41) vs. non-culprit (n=200) lesions, had similar segmental CAC score (median 22, 25th–75th percentile 4–71 vs. 14, 25th–75th percentile 0–51; $p=0.37$), but higher prevalence of significant stenosis (81% vs. 11%, $p<0.001$) and high-risk plaque (76% vs. 51%, $p=0.005$). Significant stenosis (odds ratio 40.2, 95%CI 15.6–103.9, $p<0.001$) and high-risk plaque (odds ratio 3.4, 95%CI 1.3–9.1, $p=0.02$), but not segmental CAC score (odds ratio 1.0, 95%CI 1.0–1.0, $p=0.47$) were associated with culprit lesions of ACS.

Conclusions—Total CAC burden was associated with ACS but segmental CAC was not associated with culprit lesions. Our findings suggest that total but not local CAC is a marker of ACS risk and support the hypothesis that extensive local CAC is a marker of plaque stability.

Graphical abstract

Keywords

coronary artery calcium; acute coronary syndrome; coronary artery calcium score; coronary artery calcium density; cardiac computed tomography; culprit lesions; high-risk coronary plaque

Introduction

Total burden of coronary artery calcium (CAC) as detected by cardiac computed tomography (CT) is an established marker of increased cardiovascular risk [1–4]. However, extensive CAC is not typically found in culprit coronary plaques of acute coronary syndrome (ACS), which are characterized by thin-cap fibroatheroma, large necrotic core, positive remodeling and microcalcifications [5–12]. Recently, Mauriello et al. showed that extent of CAC was higher in subjects with myocardial infarction as compared to controls, but extensive CAC was not associated with high-risk plaque features in post-mortem histology analysis [13]. Thus, there is dissociation between the prognostic value of total CAC burden and the relationship of local CAC to culprit lesions responsible for acute events.

Extensive local CAC with high density may be a marker of plaque stability despite the fact that higher total CAC score is a marker of increased cardiovascular risk. The diffuse distribution of CAC with smaller and less dense individual calcified plaques as well as overall lower CAC plaque density have been described as factors increasing cardiovascular risk independently and incrementally to total CAC score [14–17]. This coronary calcium paradox is further emphasized by the failure of therapeutic interventions to slow CAC progression. The faster progression of CAC scores was associated with increased risk of cardiovascular events [1,18], but statin therapy, which significantly decreased the risk of cardiovascular events, did not slow CAC progression [19–21]. Studies using serial intravascular ultrasound and coronary CT angiography (CTA) for plaque assessment provided some explanation for coronary calcium paradox. While statins slowed or even halted coronary plaque volume progression in a dose-dependent manner, they accelerated the progression of densely calcified plaque volume [22–24].

To better understand the complex relationship between CAC and risk of ACS, we examined the relationship of total CAC burden with ACS and between local CAC and culprit lesions in patients with suspected ACS.

Patients and methods

Patient population

This is a secondary analysis of the Rule Out Myocardial Infarction/Ischemia Using Computer Assisted Tomography II (ROMICAT II) trial [25]. ROMICAT II enrolled patients who presented to the emergency departments of nine hospitals in the United States with chest pain and clinical suspicion of ACS between April 2010 and January 2012. We studied 260 patients who were randomized to the coronary CTA arm of the ROMICAT II trial, underwent coronary CTA and had coronary plaque present (Fig. 1). Baseline clinical characteristics, medical history and cardiovascular risk factors (arterial hypertension, hyperlipidemia, diabetes mellitus, and former or current smoking) were collected. All study participants provided written consent, and local institutional review boards approved the study.

Coronary CTA

CT images were acquired using a 64 or greater multi-detector row scanner using either retrospectively ECG-gated or prospectively ECG-triggered protocols. The investigators in the study used the scanners from three vendors (Siemens, General Electric, Toshiba). The protocol consisted of a non-contrast scan for the detection and quantification of CAC followed by a contrast-enhanced coronary CTA scan. The images were transferred to a core laboratory for further analysis.

Coronary artery calcium analysis

An independent reader blinded to clinical outcomes performed the analysis of CAC on a dedicated cardiac workstation (TeraRecon, San Mateo, CA) using non-contrast CT images. The total CAC amount was quantified using the Agatston method [26]. The amount of CAC in each coronary segment was also measured using the Agatston method. We also measured calcium density score at the patient and segmental level using the method described by Criqui et al [16]. The coronary segments as defined in contrast-enhanced coronary CTA were used to facilitate segmental assignment of CAC.

Qualitative coronary CTA analysis

Three experienced cardiac CT core laboratory readers (at least 5 years of experience in cardiac CT and level III training) performed the image analysis on a dedicated cardiac workstation (TeraRecon, San Mateo, CA). Coronary CTA analysis was performed in each coronary segment as defined by the model of the Society of Cardiovascular Computed Tomography [27]. For each coronary segment, the reader determined whether the image quality was sufficient to evaluate for the presence of stenosis and coronary plaque with confidence. Coronary segments with non-diagnostic image quality (202 out of 6855 segments, 2.9%) were treated as non-informative for the purpose of the analysis.

Each evaluable coronary segment was assessed for the presence of 50% stenosis, 70% stenosis and coronary atherosclerotic plaque [27,28]. The segments with coronary plaque were qualitatively evaluated for the presence of high-risk plaque features. We determined the presence of following high-risk plaque features: positive remodeling (remodeling index >1.1), low CT attenuation plaque (<30 HU), napkin-ring sign (ring-like peripheral higher attenuation surrounding a central core of low attenuation of a non-calcified coronary plaque) and spotty calcium (calcified plaque with a maximum diameter <3 mm in any direction, length of the calcification <1.5 times vessel diameter and width of the calcification $<2/3$ of vessel diameter) as reported previously [12,29].

Definition and adjudication of acute coronary syndrome

ACS was defined as acute myocardial infarction or unstable angina pectoris according to the American College of Cardiology/American Heart Association Guidelines [30]. ACS during index hospitalization was a predefined end-point in the ROMICAT II trial and was adjudicated by an independent clinical events committee [25].

Definition of culprit lesions of ACS

In order to determine the location of culprit lesions of ACS, an investigator, who was blinded to the coronary CTA readings, reviewed all available clinical records in patients diagnosed with ACS during the index hospitalization. The records included discharge summaries, invasive coronary angiography reports and additional cardiac testing (e.g. stress echocardiograms, nuclear myocardial perfusion stress tests). The primary decision on the location of culprit lesions was made by reviewing the invasive coronary angiography reports. Invasive coronary angiography was performed in 32 out of 37 patients with ACS. We assigned the culprit lesion to coronary segments in which percutaneous coronary intervention was performed. In patients who underwent coronary artery bypass grafting (n=4), we assigned culprit lesions to all segments with $\geq 70\%$ stenosis or $\geq 50\%$ stenosis for the left main coronary artery. There were 6 patients who underwent invasive coronary angiography, but did not undergo revascularization. The culprit lesion in those patients was assigned to the coronary segment with the highest degree of diameter stenosis on invasive coronary angiography. In patients with no invasive coronary angiography (n=5), we determined the culprit vessel of ACS based on the artery subtending the territory with ischemia on stress testing. The culprit segment of ACS was then assigned to the coronary segment with the highest degree of diameter stenosis in the culprit vessel as measured on coronary CTA.

Statistical analysis

All statistical analyses were performed using Stata 13.1 (StataCorp LP, College Station, Texas). Continuous data are presented as mean $\pm$ standard deviation or median and 25th–95th percentile. Comparisons between groups were performed with an independent student t-test or the Wilcoxon rank-sum test for continuous variables and Fisher's exact test for categorical variables. We performed a multivariable multilevel mixed-effects logistic regression analysis that accounted for the group structure (i.e. multiple segments per patient) and thus possible autocorrelation effects in the data. The dependent variable was culprit coronary segment of ACS. The independent variables were segmental CAC score, the presence of high-risk plaque and $\geq 70\%$ stenosis in any coronary artery or $\geq 50\%$ stenosis in the left main coronary artery. For all analyses, a 2-tailed *p* value of <0.05 was required to reject the null hypothesis.

Results

Study population

We studied 501 patients randomized to the coronary CTA arm of the ROMICAT II trial (Fig. 1). Of those, 28 patients did not undergo coronary CTA and 1 had non-evaluable coronary CTA scan. We also excluded patients who did not have any evidence of coronary atherosclerotic plaque (n= 210) and patients with insufficient image quality for the evaluation of high-risk plaque features and quantification of CAC (n=2). There were 20 patients with CAC score >500 and coronary CTA was diagnostic in all of them. No patients were excluded from the analysis due to high CAC score. The final population included 260 patients, whose baseline characteristics are summarized in Table 1. ACS during the index hospitalization was diagnosed in 37 patients (myocardial infarction n=5, unstable angina

pectoris n=32). Although there were fewer women among patients with than without ACS, there were no significant differences in the prevalence of cardiovascular risk factors.

Total CAC measures and ACS

Patients diagnosed with ACS during the index hospitalization had higher total CAC scores as compared to patients without ACS (Table 2). The rate of ACS increased across the categories of CAC score of 0 (ACS rate 1.6%, n=1/64), 1–100 (ACS rate 8.3%, n=9/109), 101–300 (ACS rate 23.9%, n=11/46), and >300 (ACS rate 39.0%, n=16/41). Similarly, patients diagnosed with ACS had higher total CAC volume. We did not find difference in calcium density score in patients with and without ACS.

We also observed association of total CAC with obstructive coronary artery disease (CAD). The prevalence of significant CAD (< 50% stenosis) was 3.1% (n=2/64), 12.8% (n=14/109), 19.6% (n=9/46), and 48.8% (n=20/41) for patients with Agatston score of 0, 1–100, 101–300, and >300, respectively ($p<0.001$). The results were similar when significant CAD was defined as ≥ 70% stenosis: 0% (n=0/64), 5.5% (n=6/109), 15.2% (n=7/46), and 26.8% (n=11/41) for patients with Agatston score of 0, 1–100, 101–300, and >300, respectively ($p<0.001$). The prevalence of high-risk plaque (defined as the presence of any high-risk plaque feature) increased with increasing CAC score: 34.4% (n=22/64), 70.6% (n=77/109), 73.9% (n=34/46), and 80.5% (n=33/41) for patients with Agatston score of 0, 1–100, 101–300, and >300, respectively ($p<0.001$).

Patients diagnosed with ACS during the index hospitalization had a higher prevalence of significant CAD (< 50% and ≥ 70% stenosis) and high-risk plaque features (Table 2).

None of traditional cardiovascular factors individually or the numbers of risk factors were associated with ACS during the index hospitalization in the logistic regression analysis. CAC score was a significant predictor of ACS (OR: 1.001, CI95%: 1.001 – 1.002, $p=0.001$). In multivariable analysis, CAC score was associated with ACS (OR: 1.001, CI95%: 1.001 – 1.002, $p=0.002$) after adjustment for number of cardiovascular risk factors. The model including CAC score and risk factors had a better diagnostic performance for ACS as compared to model with risk factors only (AUC 0.76, 95%CI 0.66 – 0.85 vs. 0.58, 95%CI 0.49 – 0.68; $p<0.001$).

Local CAC and culprit lesions

We compared culprit (n= 41) and (n= 200) non-culprit coronary segments in patients with ACS (Table 3). There was no significant difference in segmental CAC score, CAC volume and calcium density score between culprit and non-culprit segments. In contrast, we observed a higher prevalence of high-risk plaque features in culprit as compared to non-culprit coronary segments of the same patients. Individual high-risk features (positive remodeling, low <30 HU plaque, napkin-ring sign) except for spotty calcium were more often present in culprit segments of ACS.

In multivariable multilevel mixed-effects logistic regression analysis, the presence of high-risk plaque (OR 4.2, 95%CI 1.6–11.2, $p<0.01$) was associated with culprit lesions of ACS after adjusting for ≥ 70% stenosis in any artery or ≥ 50% stenosis in the left main coronary

artery (OR 28.9, 95%CI 10.7–77.7, $p<0.001$) and segmental CAC score (OR 1.0, 95%CI 1.0–1.0, $p=0.63$). Similar results were observed when the analysis was performed without patients who underwent coronary artery by-pass surgery ($n=4$), in whom the determination of culprit lesions is more problematic (high-risk plaque: OR 3.3, 95%CI 1.1–9.6, $p=0.03$; 70% stenosis in any artery or 50% stenosis in the left main coronary artery: OR 32.0, 95%CI 9.6–106.1, $p<0.001$; segmental CAC score: OR 1.0, 95%CI 1.0–1.0, $p=0.78$).

Segmental analysis of CAC and high-risk plaque features

In the analysis of individual coronary segments with CAC present, we found an inverse relationship between segmental CAC score and the presence of high-risk plaque (Fig. 2A). The prevalence of high-risk plaque in coronary segments decreased with increasing segmental CAC ($p<0.001$). The prevalence of any high-risk plaque feature was 68% ($n=141/207$), 45% ($n=94/207$) and 38% ($n=78/206$) in the lower, mid and higher tertiles of segmental CAC scores, respectively. Segmental CAC score was lower in coronary segments with any high-risk plaque ($n=313$ segments) as compared to segments without high-risk plaque ($n=307$ segments) (CAC score: 9.4, 25th–75th percentile 0.9 – 36.0 vs. 11.3, 25th–75th percentile 0 – 51.1, $p=0.011$).

Similar observation was made for segmental calcium density score (Figure 2B). The prevalence of high-risk plaque in coronary segments decreased with increasing segmental calcium density score ($p<0.001$). Segmental calcium density score was lower in coronary segments with any high-risk plaque as compared to segments without high-risk plaque (calcium density score: 2.5, 25th–75th percentile 1.7 – 3.0 vs. 3.0, 25th–75th percentile 2.5 – 3.3, $p<0.001$).

We also found an inverse relationship between segmental CAC score and the presence of high-risk plaque in non-culprit coronary segments (Supplementary Figure 1A). The prevalence of high-risk plaque in coronary segments decreased with increasing segmental CAC ($p<0.001$). The prevalence of any high-risk plaque feature was 67% ($n=128/192$), 44% ($n=85/192$) and 36% ($n=69/192$) in the lower, mid and higher tertiles of segmental CAC scores, respectively. Similar observation was made for segmental calcium density score. The prevalence of high-risk plaque in coronary segments decreased with increasing segmental calcium density score (lower tertile 67%, $n=128/192$, mid tertile 49%, $n=95/192$, higher tertile 31%, $n=59/192$, $p<0.001$). In contrast, we did not find significant difference in the prevalence of high-risk plaque in tertiles of segmental CAC score in the analysis of culprit segments (Supplementary Fig. 1B, $p=0.24$). The prevalence of any high-risk plaque feature was 87% ($n=13/15$), 60% ($n=9/15$) and 64% ($n=9/14$) in the lower, mid and higher tertiles of segmental CAC scores, respectively. There was no significant difference in the prevalence of high-risk plaque in tertiles of calcium density (lower tertile 80%, $n=12/15$, mid tertile 67%, $n=10/15$, higher tertile 64%, $n=9/14$, $p=0.71$).

Discussion

Our study suggests that there are differences in relationship of total CAC with ACS and local CAC with culprit lesions of ACS in patients with acute chest pain. We demonstrated that a higher total CAC score was strongly associated with the diagnosis of ACS in patients

presenting to the emergency department with suspected ACS. In contrast, local more extensive CAC was not associated with the presence of culprit lesions and high-risk plaque.

Histopathology of coronary calcium and high-risk plaque

The studies of coronary plaques in subjects who suffered sudden cardiac death revealed histopathologic features of culprit coronary plaques, which included the presence of thick-cap fibroatheroma, large necrotic core, positive remodeling and microcalcifications.[5,6] Extensive calcification was associated with stable forms of coronary plaque such as fibroatheroma and healed plaque ruptures.[6] The progression of calcification is part of the natural history of plaque. Micro-calcifications within plaque lipid pools coalesce into speckles and fragments during atheroma progression and the fragmented calcified areas eventually form calcified sheets.[31] The histopathologic findings provide insight into the interplay between CAC and cardiovascular events. While the overall plaque burden is associated with higher total amounts of CAC [32–34] and is a marker of increased risk of events [1–4], the presence and severity of local CAC is not helpful in detecting high-risk lesions causing acute events.[13]

Coronary artery calcium and risk of cardiovascular events

There is an extensive body of literature that supports the role of total CAC burden on non-contrast cardiac CT for the prediction of future cardiovascular events.[1–4] While the progression of total CAC scores is also associated with adverse cardiovascular outcomes [1,18], the attempts to slow the progression of CAC with statins have been disappointing. Despite the well-established role of statins in decreasing the risk of cardiovascular events[35], statins failed to slow down CAC progression in large prospective trials.[19–21] These results were surprising to many, given the close correlation between CAC as determined by CT and overall coronary plaque burden, including non-calcified plaque [32–34] and the fact that statins have been shown to slow progression of coronary atherosclerosis or even lead to plaque regression.[36,37]

Recently published studies using serial evaluation of coronary atherosclerotic plaques by intravascular ultrasound provide insight into the seemingly paradoxical effects of statins on CAC.[22,23] Both studies showed that statins slowed the progression of coronary atherosclerosis, but led to the increase of dense, more advanced, calcium. Similar observations were made in a study of patient who underwent serial coronary CTA after ACS. [24] Patients on high-dose statin as compared to those on moderate-dose statin showed similar overall progression of plaque volume, but significantly greater progression of densely calcified plaque. Thus, an increase in lesion calcification correlates with conditions that improve clinical outcomes and may represent a plaque-stabilizing effect of statins beyond their effect on atherosclerotic plaque progression.[38] It appears therefore that while having a large total CAC burden is a strong prognosticator for cardiovascular events, an increase of CAC over time observed with statin treatment may be associated with more stable coronary plaque characteristics and possibly improved outcomes.

Recent observations from the Multi-Ethnic Study of Atherosclerosis further extend our understanding of the relationship between CAC and cardiovascular events.[15,16] The study

showed that more diffuse distribution of CAC and lower density of individual CAC plaques were associated with increased cardiovascular risk independently and incrementally to total CAC score. Thus, when comparing two subjects with the same total CAC, subject with lower CAC density and more diffuse distribution of CAC over the entire coronary tree had higher risk of subsequent events. This result suggests that subjects with more diffuse coronary atherosclerosis, but low density of CAC are in an active stage in the natural history of coronary atherosclerosis with large amounts of non-calcified plaque and possibly other high-risk plaque features that are not visualized by a non-contrast coronary CT scan. The studies using coronary CTA, which permits also visualization of non-calcified plaque components, demonstrated that the presence of high-risk plaque features was associated with an increased risk of future cardiovascular events.[11,39,40]

Imaging characteristics of culprit lesions of ACS

Intravascular imaging methods have demonstrated plaque characteristics that correlate with histopathologic findings in culprit lesions of ACS. Large plaque burden, positive remodeling, large necrotic core, thin-cap fibroatheroma, and spotty calcium were found more often in culprit lesions of ACS as compared to lesions of patients with stable angina pectoris.[7–9,41] Similar results were observed in studies using coronary CTA for plaque characterization. Culprit plaques of patients with ACS are characterized by large plaque burden, positive remodeling, spotty calcium, low HU plaque and napkin-ring sign.[10,12,42] Furthermore, high-risk plaque features were independent and incremental to stenosis in diagnosing ACS and detecting lesion specific ischemia.[12,43,44] Morphologic assessment of high-risk plaque can be combined with measurements of serum biomarker such as high-sensitivity troponin for more accurate prediction of cardiovascular events.[45] Combination of high-sensitivity troponin and advanced analysis of coronary CTA for high-risk plaque can also improve diagnostic accuracy for ACS in patients with acute chest pain.[46]

Relationship between coronary calcium and culprit lesions in patients with suspected ACS

The results of our study provide another piece to the puzzle of the complex relationship between CAC and the risk of cardiovascular events. In line with epidemiological studies, we observed that total amount of CAC was associated with ACS. In the population of patients presenting to the emergency department with symptoms suggestive of ACS, the presence of significant coronary stenosis, high-risk plaque features and higher total CAC score were all associated with the diagnosis of ACS during the index hospitalization. This result confirmed that coronary plaque burden as measured by CAC score was a strong predictor of increased risk of an acute event. However, the segmental analysis of coronary plaque revealed a paradoxical dissociation between CAC and high-risk plaque features. The coronary segments with high CAC scores were less likely to harbor high-risk plaque features such as positive remodeling, low HU plaque, napkin-ring sign and spotty calcium. Similarly, high-risk plaque features were less often seen in coronary segments with higher CAC density. Our observations were further strengthened by the observation that presence of significant coronary stenosis and high-risk plaque features, but not higher local CAC score, was found in the culprit lesions of ACS. These results confirmed that local extensive CAC was less likely to be present at the site of the culprit lesion and represented a more stable form of coronary atherosclerosis.

Limitations

Some limitations of the current analysis should be recognized. The accurate detection of coronary segment boundaries on a non-contrast cardiac CT scan can be difficult and may complicate the assignment of CAC to the correct segment. We permitted the use of contrast-enhanced coronary CTA to help determine the coronary segment boundaries in non-contrast scans. However, the reader of non-contrast CT scans was blinded to the CTA results. The assignment of culprit lesion was performed retrospectively from available clinical data, and invasive coronary angiography was not performed in 5 of 37 patients with ACS. In these latter patients we used results of non-invasive testing to determine the culprit coronary artery and then assigned the lesion with the highest degree of stenosis on coronary CTA as the culprit lesion.

Conclusions

We found differences in the relationship of total CAC burden with ACS and that between local CAC and culprit lesions of ACS in patients presenting to the emergency department with acute chest pain. While total CAC burden was a strong predictor of ACS, culprit lesions of ACS were characterized by significant stenosis and high-risk plaque, but not by higher segmental CAC. The prevalence of high-risk plaque was inversely related to segmental amount of CAC. These findings suggest that local extensive CAC may represent more stable stage of atherosclerosis, while the culprit lesions are characterized by smaller amounts of CAC.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Total coronary artery calcium burden was a good predictor of acute coronary syndrome (ACS) in acute chest pain patients
- Culprit lesions of ACS were characterized by significant stenosis and high-risk plaque
- Culprit lesions of ACS had smaller amounts of coronary calcium
- The prevalence of high-risk plaque was inversely related to segmental coronary calcium score
- Local extensive coronary calcium may represent more stable stage of atherosclerosis

Figure 1.

Study population enrollment, inclusion and exclusion.

(A)

(B)

Figure 2.

The relationship of high-risk plaque features with segmental coronary calcium scores and coronary calcium density score.

(A) The prevalence of any high-risk plaque features stratified by tertiles of segmental coronary calcium scores. The prevalence of high-risk plaque in coronary segments decreased with increasing segmental coronary calcium score ($p < 0.001$).

(B) The prevalence of any high-risk plaque features stratified by tertiles of segmental calcium density score. The prevalence of high-risk plaque in coronary segments decreased with increasing segmental calcium density score ($p < 0.001$).

Table 1

Baseline characteristics of study patients stratified according to diagnosis of acute coronary syndrome

	Total (n=260)	No ACS (n=223)	ACS (n=37)	<i>p</i> value
Age (years)	56.1 ± 7.8	55.9 ± 7.8	57.2 ± 8.3	0.383
Women, n (%)	97 (37.3)	91 (40.8)	6 (16.2)	0.005
Cardiovascular risk factors, n (%)				
Hypertension	157 (60.4)	135 (60.5)	22 (59.5)	1.000
Diabetes mellitus	53 (20.4)	46 (20.6)	7 (18.9)	1.000
Dyslipidemia	142 (54.6)	117 (52.5)	25 (67.6)	0.109
Former or current smoker	145 (55.8)	120 (53.8)	25 (67.6)	0.153
Family history of premature CAD	65 (25.0)	54 (24.2)	11 (29.7)	0.539

ACS = Acute coronary syndrome; CAD = coronary artery disease.

Table 2

Coronary artery disease characteristics stratified according to diagnosis of acute coronary syndrome

	Total (n=260)	No ACS (n=223)	ACS (n=37)	p value
Agatston score, median (25 th –75 th percentile)	36.6 (0.2–137.4)	26.6 (0.0–98.7)	229.3 (75.4–517.3)	<0.001
Coronary artery calcium volume in mm ³ , median (25 th –75 th percentile)	38.1 (0.7–119.8)	28.8 (0.0–86.2)	186.8 (62.4–438.4)	<0.001
Calcium density score, median (25 th –75 th percentile)	2.9 (2.3–3.2)	2.9 (2.3–3.3)	3.0 (2.6–3.2)	0.316
Significant CAD, n (%)				
50% stenosis	45 (17.3)	16 (7.2)	29 (78.4)	<0.001
70% stenosis	24 (9.2)	4 (1.8)	20 (54.1)	<0.001
Any high-risk plaque, n (%)	166 (63.9)	131 (58.7)	35 (94.6)	<0.001
Positive remodeling	55 (21.2)	33 (14.8)	22 (59.5)	<0.001
Low CT attenuation plaque	39 (15.0)	23 (10.3)	16 (43.2)	<0.001
Napkin-ring sign	26 (10.0)	14 (6.3)	12 (32.4)	<0.001
Spotty calcium	151 (58.1)	116 (52.0)	35 (94.6)	<0.001

ACS = acute coronary syndrome; CAD = coronary artery disease, HU = Hounsfield units.

Table 3

Coronary artery disease characteristics in coronary segments with and without culprit lesions in 37 patients with acute coronary syndrome during the index hospitalization

	Total (n=241)	Non-culprit lesions (n=200)	Culprit lesions (n=41)	p value
Agatston score, median (25 th –75 th percentile)	14.3 (0.5–52.8)	14.1 (0.3–51.1)	22.3 (3.6–70.8)	0.372
Coronary artery calcium volume in mm ³ , median (25 th –95 th percentile)	17.3 (1.5–50.9)	16.7 (0.8–48.7)	23.2 (4.2–77.9)	0.340
Calcium density score, median (25 th –95 th percentile)	2.7 (2.0–3.3)	2.7 (1.9–3.3)	2.6 (2.0–3.3)	0.616
Significant CAD, n (%)				
50% stenosis	54 (22.4)	21 (10.5)	33 (80.5)	<0.001
70% stenosis	31 (12.9)	9 (4.5)	22 (53.7)	<0.001
Any high-risk plaque, n (%)	133 (55.2)	102 (51.0)	31 (75.6)	0.005
Positive remodeling	38 (15.8)	18 (9.0)	20 (48.8)	<0.001
Low CT attenuation plaque	24 (10.0)	9 (4.5)	15 (36.6)	<0.001
Napkin-ring sign	18 (7.5)	6 (3.0)	12 (29.3)	<0.001
Spotty calcium	121 (50.2)	98 (49.0)	23 (56.1)	0.493

ACS = Acute coronary syndrome; CAD = coronary artery disease.