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ORIGINAL ARTICLE

# Coronary Artery Calcium Identified on Non-Gated Chest CT Scans: A Wasted Opportunity for Preventive Cardiological Care

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**Background** Coronary artery calcium (CAC) evaluated on dedicated cardiac computed tomography (CT) is an independent predictor of cardiovascular events. This study aimed to evaluate the correlation between CAC detected on non-gated standard chest CT and coronary lesions on coronary angiography (CAG) and determine its impact on prognosis.

**Methods** Consecutive patients who underwent CAG due to acute coronary syndrome and had prior non-contrasted non-gated chest CT were included and retrospectively evaluated. Coronary artery calcium was evaluated by quantitative (Agatston score) and qualitative (visual assessment) assessment.

**Results** A total of 114 patients were included in this study. The mean time difference between chest CT and CAG was 23 months. Coronary artery calcium was visually classified as mild, moderate, and severe in 31%, 33%, and 16% of patients, respectively. Moderate or severe CAC was an independent predictor of significant lesions on CAG (OR 22; 95% CI 8–61; p<0.001) and all-cause mortality (OR 4; 95% CI 2–9; p=0.001). Quantitative CAC evaluation accurately predicted significant lesions on CAG (AUC 0.81; p<0.001). While significant CAC was identified in 80% of chest CTs, formal reporting was 25%.

**Conclusion** Coronary artery calcium evaluation with chest CT was feasible and strongly associated with severity of coronary disease on CAG and mortality. Although the identification of CAC on chest CT represents a unique opportunity for cardiovascular risk stratification for preventive care, CAC underreporting is frequent.

**Keywords** Coronary artery calcium • Coronary disease • Computed tomography • Coronary angiography • Risk stratification

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# Introduction

Coronary artery disease (CAD) is the leading cause of mortality in developed countries [1]. Detection of coronary artery calcification (CAC) is a strong predictor of CAD, adverse cardiovascular events, and all-cause mortality [2,3]. Although CAC scoring is traditionally performed with electrocardiography (ECG)-gated computed tomography (CT) scan with standard reconstruction and acquisition parameters, CAC can also be detected on non-gated chest CT examination [4]. Recent publications suggest a good correlation between CAC scores from non-gated chest CT scans and formal CAC testing [5–8].

Standard non-gated chest CT scans are used for numerous clinical indications, and incidental coronary calcification is a frequent finding [9,10]. In 2007, 13.6 million non-gated chest CT examinations were performed in the United States, in contrast to 0.7 million ECG-gated CT examinations for calcium scoring [11]. Thus, although the primary indication for performing a chest CT is not to evaluate CAC, the importance of assessing CAC on non-gated chest CT has been recognised, and recent guidelines recommended that CAC should be evaluated and reported on all non-gated chest CT scans, regardless of the indication [12,13]. The routine reporting of CAC on non-gated chest CT scans may flag, to a referring provider, a patient at high risk of cardiovascular events, for which preventative therapy or complementary investigations may be pursued. Jacobs et al. demonstrated that simple visual grading of CAC on non-gated chest CT strongly correlates with future cardiovascular events [14]. Although there are current evidence and recommendations, a recent survey found that 17% of non-cardiac and 32% of cardiac imagers were aware of the existing data correlating calcium scores on non-gated chest CTs [15]; this may justify the underreporting of CAC, representing a wasted opportunity for preventive cardiology care [10,13,14].

Although previous studies have demonstrated the correlation between CAC on non-gated chest CT and major adverse cardiac events [16,17], none have studied its correlation with coronary angiography (CAG). This study aimed to evaluate the correlation between CAC documented on standard non-gated chest CT and coronary lesions on coronary angiography and determine its correlation with prognosis. The primary outcome was to evaluate the relationship between CAC and significant lesions on CAG. The second outcome was to assess the prognostic value of CAC and document the rate of CAC reporting on chest CT scans.

# Methods

This study was a single-centre, retrospective, observational cohort study performed at a tertiary university hospital between 2017–2021. It selected adult outpatients who underwent CAG due to acute coronary syndrome and had previous non-contrasted non-gated chest CT. Patients who underwent chest CT <3 months before CAG or with a previous history of percutaneous coronary intervention (PCI) or coronary artery bypass graft were excluded from the analysis. In cases of multiple CTs available for analysis, the most recent chest CT was chosen. Electronic medical records were abstracted for baseline demographics and cardiovascular risk factors. Due to the retrospective analysis of data, the need for informed consent was waived by the institution.

## Chest computed tomography protocol and coronary artery calcification evaluation

The CT scans were obtained from a 64-slide or 16-slice multidetector CT (Siemens Medical Solutions USA Inc, Malvern, PA, USA). Slice thickness varied according to the CT indication and corresponding protocol. All CT studies were reviewed for study purposes without additional processing using Patient Archiving and Communication System (PACS) software. All studies were analysed in the axial plane. No other plane reconstructions were included for analysis.

Coronary artery calcium was evaluated by an investigator blinded to clinical information, including the CAG report. Coronary artery calcium was identified and quantified using two methods:

a) Qualitative assessment, in which CAC was visually quantified according to the extent of global coronary artery tree calcification as absent (0 points), mild (1 point), moderate (2 points), or severe (3 points).
b) Quantitative assessment, in which CAC was obtained by using the Agatston method with the traditional 130-HU threshold. The score was calculated separately for each of the main coronary artery branches: left main trunk, left anterior descending (LAD), circumflex (CX), and right coronary (RCA), and then summed into a single score for the entire coronary artery tree. For risk-stratification, the Agatston score was further stratified into three risk categories, which have been used in previous studies [4,7,8]: no CAC (0), mild CAC ($\leq$100), moderate CAC (100–400), and severe CAC ($\geq$400).

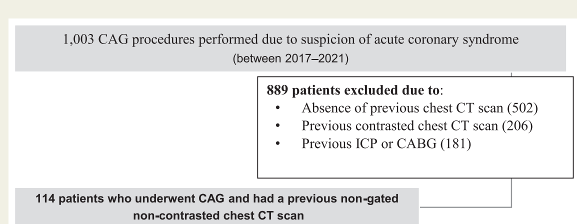


**Figure 1** Study flowchart.
Abbreviations: CAG, coronary angiography; CT, computed tomography; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft.

**Table 1** Comparison of demographics for patients with CAC and without CAC at the time of non-gated chest computed tomography.

| Variable | No CAC (n=23) | CAC (n=91) | p-value |
|---|---|---|---|
| Age, median (Q1-Q3) (yr) | 53 (47–66) | 72 (63–80) | p<0.001 |
| Gender - Male, n (%) | 11 (48) | 27 (30) | p=0.099 |
| Comorbidities | | | |
| Arterial hypertension, n (%) | 15 (65) | 74 (81) | p=0.095 |
| Dyslipidemia, n (%) | 11 (48) | 60 (66) | p=0.109 |
| Diabetes mellitus, n (%) | 3 (13) | 40 (44) | p=0.006 |
| Chronic kidney disease, n (%) | 4 (17) | 40 (44) | p=0.019 |
| Chronic obstructive pulmonary disease, n (%) | 2 (9) | 23 (25) | p=0.086 |
| Smoking, n (%) | 5 (22) | 40 (44) | p=0.049 |
| Cerebrovascular disease, n (%) | 1 (4) | 8 (9) | p=0.480 |
| Peripheral arterial disease, n (%) | 2 (9) | 7 (8) | p=0.873 |
| Coronary angiography | | | |
| Significant lesions, n (%) | 4 (17) | 61 (67) | p<0.001 |

Abbreviation: CAC, coronary artery calcification.

Parameters of CT scan protocols were extracted (slice thickness, tube potential [kVp], and tube current-time product [mAs]). To determine sensitivity and specificity and predict significant lesions on CAG, subgroups of patients were formed according to the slice thickness (2 mm, 3 mm, or 5 mm), and no reconstructions of slice thickness were made. No control of heart rate or rhythm was performed; thus, these data were not collected.

The CT report was reviewed to determine whether CAC was mentioned in either the text body or in the conclusion of the CT report. Report of CAC was considered if any mention of coronary calcification or atherosclerosis was made. Reporting of aortic calcification, valvular calcification, unspecified vascular calcifications, or non-coronary atherosclerosis was not considered indicative of reporting CAC.

## Statistical analysis

The statistical software used to analyse the data was SPSS Version 26 (IBM Corp, Armonk, NY, USA). Categorical variables were presented as frequency rates/percentages and continuous variables as median with interquartile range. Categorial and continuous variables were compared using Pearson Chi-square and Mann-Whitney tests, respectively. The comparison of means was performed using analysis of variance (ANOVA). The comparison of non-normally distributed continuous variables was reported as median with interquartile range and analysed using the Mann-Whitney test. Statistical significance was defined as p<0.05. Receiver operating characteristic (ROC) curve analysis was performed. Cox regression was used for the multivariate-adjusted factor analysis to study the impact of CAC severity on survival. Kaplan-Meier analysis was performed for event-free survival.

# Results

A total of 114 patients were included in the study. The study flowchart is shown in Figure 1. The most frequent reasons for performing chest CT were lung cancer screening (26%) and aetiological investigation of dyspnoea (24% of cases, mostly in the Emergency Department, due to suspected respiratory

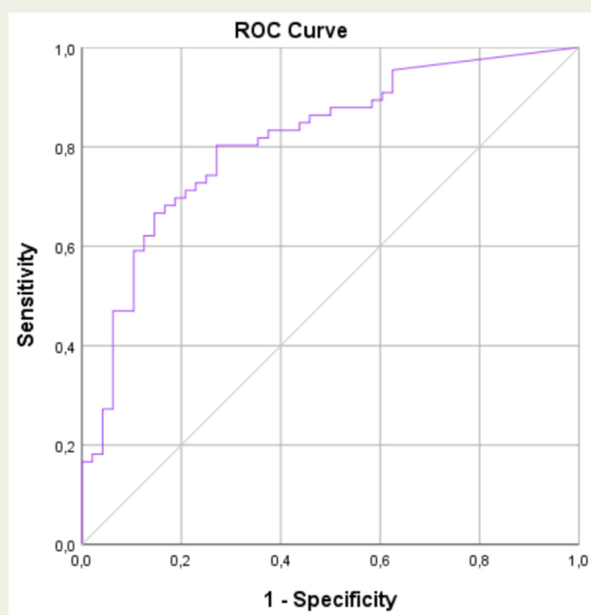


**Figure 2** Receiver operating characteristics (ROC) curve demonstrating the accuracy of quantitative coronary artery calcium score (Agatston score) to predict the risk of significant lesions on coronary angioplasty.

**Table 2** **Coronary artery calcium score for each of the main vessels (left main trunk, left anterior descending artery, circumflex artery and right coronary artery) according to the presence of significant lesions on CAG.**

| | CAC score (median, IQR) | | |
|---|---|---|---|
| | Significant lesions | No lesions or non-significant lesions | p-value |
| Left main trunk | 76 (0–217) | 0 (0–0) | p=0.029 |
| Left anterior descending artery | 813 (300–1429) | 59 (0–352) | p<0.001 |
| Circumflex artery | 314 (51–1064) | 6 (0–155) | p<0.001 |
| Right coronary artery | 500 (207–1245) | 43 (0–206) | p<0.001 |

Abbreviation: CAC, coronary artery calcium.

infection or pulmonary embolism), followed by investigation of obstructive and interstitial pulmonary disease (12%) and complications due to SARS-COV2 infection (7% of cases).

Coronary artery calcium was identified in 91 patients (80%). The baseline characteristics of patients with and without CAC are shown in Table 1. Patients with CAC were older, had a higher prevalence of diabetes and chronic kidney disease, and were over twice as likely to smoke. Significant coronary artery lesions on CAG were substantially higher in patients with CAC on previous chest CT than those without CAC.

All patients underwent CAG due to acute coronary syndrome (30% unstable angina, 52% non-ST-elevation myocardial infarction, and 18% ST-elevation myocardial infarction). The CAG revealed significant coronary lesions in 65 patients (57%); of those, 56 patients (86%) were revascularised (80% PCI and 20% coronary artery bypass graft). Regarding patients who underwent PCI (n=45), 31 underwent PCI due to single-vessel disease (LAD n=19, CX n=5, and RCA n=7) and 14 patients underwent multivessel PCI (LAD+CX n=4, LAD+RCA n=4, CXR+CA n=2, and three-vessel disease n=2).

The mean time interval between chest CT and CAG was 23±19 months. Patients with no lesions on CAG had significantly lower CAC quantitative scores compared with patients with non-significant lesions (15 [IQR 0–0] vs 536 [IQR 86–675]; p<0.001). Additionally, patients with significant lesions had significantly higher CAC compared with those with non-significant lesions (1,823 [IQR 354–2,255) vs 536 [IQR 86–675]; p<0.001). For CAC visual assessment, patients with no lesions on CAG had a median CAC visual assessment score of 0.07 [IQR 0–0], which is significantly lower than those who had non-significant lesions (1.0 [IQR 1–1]; p<0.001). Patients with significant lesions on CAG had a higher CAC visual assessment score (1.9 [IQR 1.8–3.0]; p<0.001].

The presence of moderate or severe CAC on visual assessment was an independent predictor of significant lesions on CAG (OR 22; 95% CI 8–61; p<0.001). Using ROC analysis, quantitative CAC assessment accurately predicted the presence of significant lesions on CAG (AUC 0.81; 95% CI 0.73–0.89; p<0.001), as shown in Figure 2.

The absence of any CAC was associated with a low risk of significant coronary lesions on CAG. Considering the patients without CAC on chest CT by visual assessment (n=23), 83% had no or non-significant lesions on CAG, and 13% underwent PCI. On the other hand, 94% of patients with severe CAC by visual assessment (n=18) had significant lesions on CAG.

Patients with significant lesions on CAG had significantly higher CAC for the corresponding vessels, as demonstrated in Table 2. In addition, the most severely calcified artery on CT scan often matched the culprit vessel of future acute coronary syndrome, with 79% concordance for LAD (p=0.027) and 67% concordance for both CX and RCA (p<0.001 and p=0.001, respectively).

This study included a heterogeneous CT scan protocol, reflecting the different indications for chest CT. As shown in Table 3, the sensitivity and specificity for significant lesions on CAG were >90% for slice thickness of <2 mm. In comparison, the sensitivity reduced to 71% for both 3-mm and 5-mm slice thicknesses.

Although patients with CAC were older and had a higher prevalence of diabetes, chronic kidney disease, and smoking, no difference was found in age or prevalence of cardiovascular risk factors according to the severity of CAC. However, patients with severe CAC by visual assessment had significantly higher peak troponin at the time of acute coronary syndrome compared with those with mild or

**Table 3** **Median CAC score calculated in different slice thickness CT scans (≤2 mm, 3 mm, and 5 mm).**

| Slice thickness | Sensitivity (%) | Specificity (%) |
|---|---|---|
| ≤2 mm (n=26) | 94% | 90% |
| 3 mm (n=76) | 71% | 85% |
| 5 mm (n=12) | 71% | 100% |

Sensitivity and specificity to detect significant lesions on CAG according to different slice thicknesses.
Abbreviation: CAC, coronary artery calcium.

**Table 4** Comparison of laboratory parameters, findings on coronary angiography and 10-year mortality between patients with different CAC severity levels according to visual assessment (none, mild, moderate and severe).

| Variable | No CAC (n=23) | Mild CAC (n=35) | Moderate CAC (n=38) | Severe CAC (n=18) | p-value |
|---|---|---|---|---|---|
| CTnT-hs (admission), median (Q1–Q3) (ng/L) | 21 (8–244) | 60 (27–902) | 83 (47–467) | 633 (26–2052) | p=0.054 |
| CTnT-hs (peak), median (Q1–Q3) (ng/L) | 23 (8–358) | 430 (34–1425) | 242 (92–242) | 1780 (87–1780) | p=0.008 |
| NTproBNP (admission), median (Q1–Q3) (ng/L) | 392 (109–1387) | 777 (612–6715) | 7141 (1153–33177) | 6637 (2965–19273) | p=0.001 |
| LDL (admission), median (Q1–Q3) (mg/dL) | 102 (67–127) | 81 (62–106) | 79 (58–112) | 80 (66–86) | p=0.310 |
| Coronary angiography | | | | | |
| No lesions, n (%) | 13 (57) | 1 (3) | 0 (0) | 0 (0) | p<0.001 |
| Non-significant lesions, n (%) | 6 (26) | 23 (66) | 5 (13) | 1 (6) | p<0.001 |
| Significant lesions, n (%) | 4 (17) | 11 (31) | 33 (87) | 17 (94) | p<0.001 |
| Outcomes | | | | | |
| 10-year mortality, n (%) | 0 (0) | 12 (34) | 18 (47) | 12 (67) | p<0.001 |

Abbreviations: CAC, coronary artery calcium; CTnT-hs, high sensitivity troponin T; NTproBNP, N-terminal pro-B-type natriuretic peptide; LDL, low-density lipoprotein.

moderate CAC (1,780 vs 315 ng/L; p=0.024), as shown in Table 4.

Regarding the prognostic prediction, moderate or severe CAC was an independent predictor of all-cause mortality (OR 4; 95% CI 2–9; p=0.001]. The overall 10-year mortality of the cohort was 37% (n=42). The CAC qualitative score was shown to be directly and incrementally associated with reduced survival (log-rank test p<0.001), according to survival proportion by Cox regression analysis adjusted for age, diabetes, chronic kidney disease, and smoking history (Figure 3). For those with severe CAC, a significative reduction in the overall estimated survival at 10 years was expected (33%), mainly from those without CAC, mild CAC, or moderate CAC (100%, 66%, and 53%, respectively). Regarding the quantitative assessment of CAC, 10-year mortality was 7% for patients with CAC ≤100, 27% for patients with CAC 100–400, and 56% for patients with CAC ≥400.

Considering only patients with CAC (n=91), multivessel coronary disease was associated with a higher risk of mortality compared with single-vessel coronary disease (OR 2.76; 95% CI 1.51–5.07; p=0.001], independent of the CAC score absolute value. The cumulative 10-year mortality was 37% for patients with single-vessel disease and 65% for multivessel disease (p=0.012), as shown in Figure 4 (log-rank test p=0.022). Coronary artery calcium strongly correlated with the SYNTAX score (p<0.001).

From a clinical perspective, patients with CAC were at higher risk of recurrent acute coronary syndrome during the follow-up than those without CAC (13% vs 30%; p=0.038). While CAC was identified in 80% of the reviewed CTs, it was reported in 25% of the cases. Even in patients with severe CAC, two of 18 reports mentioned it. Examples of unreported severe CAC are shown in Figures 5A and B. There was no difference in reporting based on age, gender, or CAC extent (p=0.257). Two reports specified the location of CAC, and none mentioned the absence of CAC.

Approximately 40% of patients with CAC documented on chest CT (n=91) were not on statin therapy at the time of CAG. Considering patients with CAC documented on CAG, 27 (29%) were under aspirin treatment prior to the current episode of acute coronary syndrome, mainly for secondary prevention (nine patients due to previous cerebrovascular disease; eight patients due to peripheral disease; and five patients due to chronic coronary syndrome).

# Discussion

This study highlights the strong correlation and potential utility of standard non-gated chest CT to identify patients at higher risk of significant CAD and mortality. It is believed that this study is the first to correlate CAC documented on non-gated chest CT with CAG findings.

Coronary artery disease is one of the leading causes of morbidity and mortality worldwide [1]. Approximately 50% of all cardiovascular disease-related deaths occur in asymptomatic patients, making risk stratification of the utmost importance to reduce the burden of associated cardiovascular morbidity and mortality. More advanced risk stratification tools, including the quantification of CAC, have emerged as strong predictors of adverse cardiovascular events, including myocardial infarction and death [18,19]. Although CAC scoring is traditionally performed with electrocardiogram-gated CT with standard reconstruction and acquisition parameters, as described by Agatston et al., calcium within the coronary arteries can be recognised on non-gated chest CT [7,8,20]. Despite the possibility of motion affecting the assessment of coronary calcium on non-gated CT, published data suggest a good

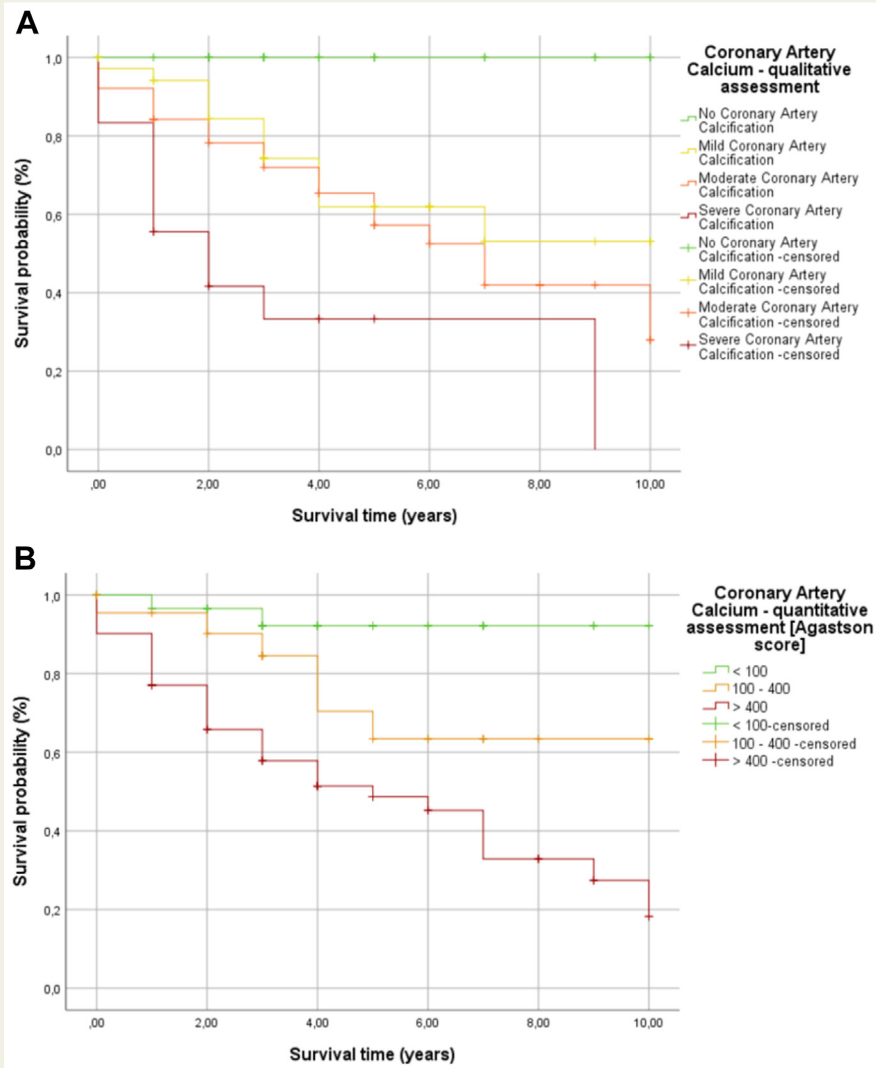


**Figure 3** Cox regression survival analysis. (A) Stratified by severity of coronary artery calcium (CAC) evaluated by visual/qualitative assessment (non-CAC, mild CAC, moderate CAC, and severe CAC); log-rank $p<0.001$; (B) Stratified by quantitative assessment: mild ($\leq$100), moderate (100-400), and severe (>400); log-rank $p<0.001$.

correlation among CAC scores between non-gated chest CT scans and formal CAC [6,21,22]. In a meta-analysis of 661 patients undergoing both gated and non-gated CT scans, the correlation coefficient for agreement of CAC scores was 0.94 (95% CI 0.89–0.97) [4].

The current study evaluated the correlation between CAG performed due to acute coronary syndrome suspicion and CAC documented in a previous non-gated chest CT. It documented a high prevalence of CAC in this cohort (80%) with an expected association with age and comorbidities such as diabetes, chronic kidney disease, and smoking.

Although the mean time difference between CT scan and CAG was around 2 years, moderate or severe CAC on CT scan was an independent predictor of significant lesions on CAG. In line with those documented in previous studies, Agatston scores obtained with non-gated chest CT scans were higher than expected in those with dedicated gated CT scans. However, as demonstrated in the current study, it maintains its value for cardiovascular risk stratification. Independent of quantitative or qualitative assessment, patients with significant lesions on CAG had significantly higher CAC scores than those with no lesions or non-significant lesions. In addition, compared with patients with mild or moderate CAC, severe CAC was associated with higher peak high-sensitivity troponin T at the time of acute coronary syndrome, suggesting more extensive myocardial infarction.

Although multivessel CAC in gated CTs is associated with a worse prognosis, the same has not been clearly demonstrated in non-gated CTs [23,24]. The current findings

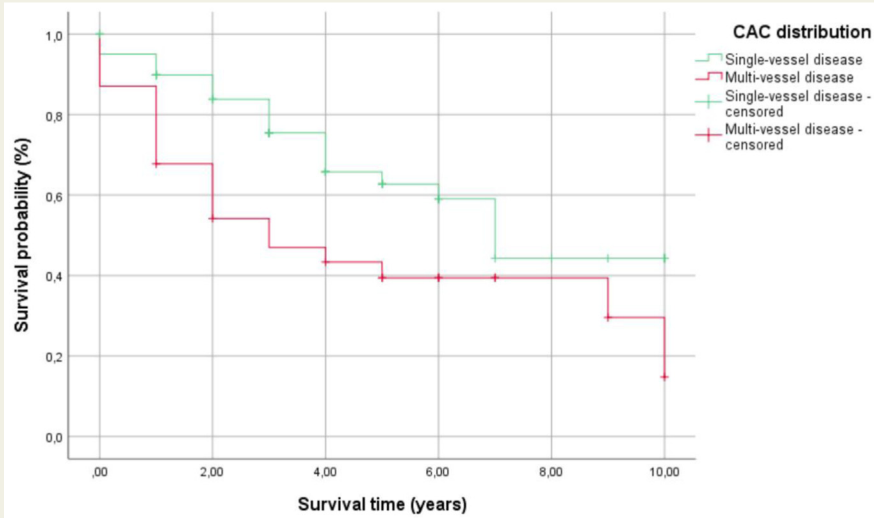


**Figure 4** Kaplan-Meier survival analysis stratified by distribution of coronary artery calcium (single-vessel vs multi-vessel coronary disease); log-rank p=0.022.
Abbreviation: CAC, coronary artery calcium.

support this hypothesis, adding further value to the prognostic information that can be derived from non-gated CTs.

In the current analysis, CAC was shown to be directly and incrementally associated with reduced survival, whether it was evaluated by qualitative (visual) or quantitative assessment (Agatston score). The 10-year mortality rate was as high as 67% for patients with severe CAC by visual assessment. On the other hand, the absence of CAC was a strong protector for cardiovascular events, as 83% had no lesions or non-significant lesions documented on CAG, and with an excellent outcome. Patients with CAC were at higher risk of recurrent coronary syndrome during follow-up, demonstrating that the higher risk is maintained at long-term follow-up.

This study included patients with chest CT studies that employed a heterogeneous set of CT protocols with a range of section widths. In line with previous studies [25], the current study also demonstrated lower sensitivity for CAC detection with wider sections compared with thinner sections (≤2 mm), although the global sensitivity remained high. Therefore, it is possible that CAC determined from 3-mm and 5-mm sections may be underestimated, and a

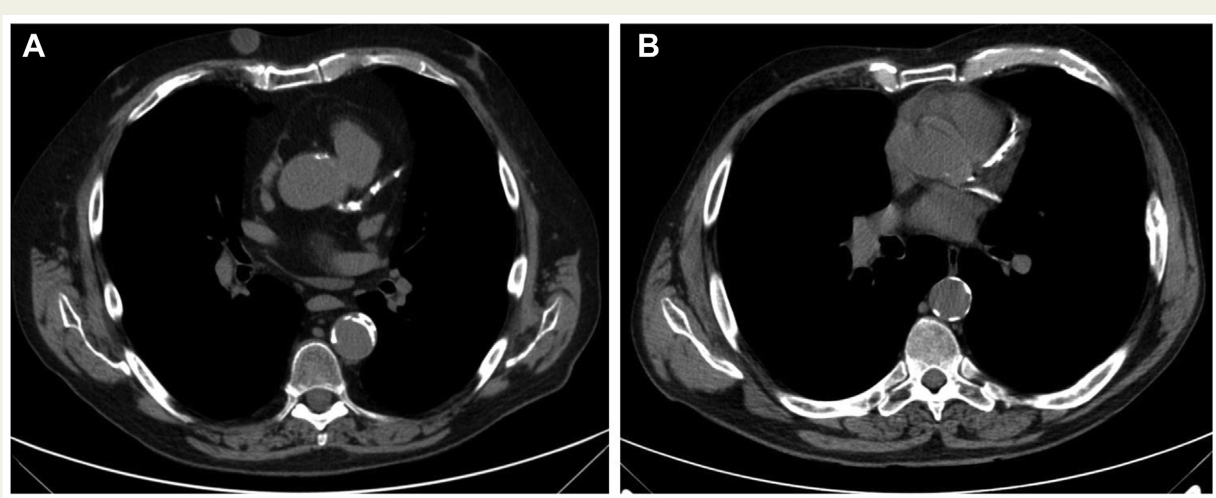


**Figure 5** Case examples. A. Axial CT image demonstrating non-reported severe calcification in the left anterior descending artery. B. Axial CT image showing non-reported extensive calcification involving the left anterior descending and circumflex arteries.

more accurate estimation of CAC might be achieved by using ≤2 mm sections.

Given the prognostic value of CAC, the simple presence or absence of CAC provides significant value in cardiovascular risk stratification. It represents a simple and reproducible scoring method with a minimum of additional reviewing time that can be applied to a range of CT protocols in daily clinical practice without dedicated software. Specifically, the absence of CAC identifies patients with a low risk for subsequent cardiovascular events.

Most final chest CT reports in the current study did not comment on the presence of CAC. This finding aligns with those previously described by Williams et al., which reported that CAC was recorded in 44% of final CT reports of patients with known CAC [13]. The routine reporting of CAC on non-gated chest CT scans may flag patients at higher risk of cardiovascular events for which preventive therapy and lifestyle modification can be aggressively pursued. Current guidelines recommended that CAC noted on non-gated chest CT should be reported, as even simple qualitative visual estimation of CAC severity has demonstrated a correlation with subsequent adverse cardiac events [9,12]. This is particularly interesting considering the expected increase in chest CT utilisation due to recent recommendations for lung cancer screening in smokers, which target a particular high cardiovascular-risk population [26,27]. Previous studies have demonstrated that >70% of patients who underwent lung cancer screening had coronary calcification, despite more than one-third not being under statin therapy [28]. Thus, identifying CAC in these patients may offer a unique opportunity for primary prevention. In addition to increasing awareness of radiologists and cardiac imaging specialists to the importance of reporting these findings, it is also essential to alert clinicians to its prognostic significance, as previous data report that 23% of clinicians were aware that it was even reported, and 4% said that they would make clinical decisions based on this finding [29].

Although coronary CT remains the gold standard for non-invasive assessment of coronary anatomy, routine reporting of CAC on non-gated chest CT could affect millions of people and may be enough when the only purpose is cardiovascular risk stratification. Thus, these results encourage including CAC as a clinically relevant finding on chest CT reports. Coronary artery calcification can be reported as an Agatston score, with the caveat that the number is likely higher than a dedicated calcium-scoring CT, or by simple visual assessment, which could be easily incorporated into structured reporting systems. However, despite its prognostic value, there are no clear guidelines or recommendations for managing patients with CAC detected on non-gated CTs concerning the decision to proceed to a dedicated coronary CT scan or CAG, particularly if they are asymptomatic. To promote the routine implementation of CAC reports on chest CTs in daily practice, more studies are necessary to guide clinicians about the appropriate preventive care strategies.

## Limitations

This study had several limitations, including only using data from a single institution and a relatively small sample size. It aimed to establish the correlation between CAC documented in a previous non-gated chest CT and CAG. Thus, patients were selected based on CAG referral and represented a high-risk cohort with an expected higher prevalence of coronary disease, which may have overestimated the prevalence and severity of CAC and may not be generalisable to other practice settings. In addition, it used a heterogenous CT protocol, particularly with a wide range of slice thickness and no control of heart rate or rhythm, which may have influenced the visual assessment of CAC. However, it intended to demonstrate the feasibility of assessing CAC in everyday practice and its prognostic value irrespective of the CT protocol. Although it found good correlation between non-gated CT and CAG for all main coronary arteries, difficulty of CAC delimitation in the left main trunk is recognised, due to the imprecise identification of calcification in its bifurcation from those of the LAD or CX arteries.

# Conclusion

Coronary artery calcium evaluation during standard chest CT was feasible and strongly associated with the extent and severity of CAD on CAG and mortality. However, CAC underreporting was frequent. Incorporating CAC into standard chest CT report represents a potentially major advance in the early detection of CAD. As more thoracic CT scans are routinely performed, identification and awareness of the presence of CAC on routine chest CT could provide a unique opportunity for cardiovascular risk stratification. It is very important that radiologists and clinicians become comfortable with the interpretation and application of CAC as a prognostic marker.

# Authors' Contribution

BVS conceived the idea, analysed the data, and took the lead in writing the manuscript. AGA discussed the concepts and the methodology. BG, AMM, and CO collected data. MNM, RP, CJ, JR, AGA, and FJP supported and reviewed the manuscript.

# Conflict of Interest

The authors report no relationships that could be construed as a conflict of interest.

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