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Real-world insights into coronary CTA prognostication: value of semiquantitative scores

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Abstract

Purpose Several semiquantitative coronary computed tomography angiography (CCTA) scores including different parameters describing stenosis degree, plaque burden and plaque features have been developed for diagnostic and prognostic purposes. However, their clinical application is still limited. The aim of the study is to assess the prognostic implication of semiquantitative coronary CCTA scores in clinical practice.

Material and methods In this retrospective, single-center study, 6818 adults who underwent elective CCTA between 2016 and 2020 were screened. A total of 1878 patients were enrolled based on low-to-intermediate pretest risk, good image quality (Likert score ≥ 4), and ≥ 3 years of follow-up. Clinical data were collected, and the following CCTA scores were calculated: CACS, CAD-RADS, Leiden, CT Leaman, SSS and SIS. Prognostic performance for five-point and two-point MACE composite outcomes was evaluated using survival analysis and multivariable models.

Results Over the follow-up period, 10% (187/1878) experienced a five-point MACE and 5.4% (102/1878) a two-point MACE. All CCTA scores stratify the risk of MACE, and all CCTA scores were predictors of outcome with HR increasing as the score increases, with the highest HR in case of severe CAD-RADS (five-point MACE composite outcome: HR 16.35, 95%CI [7.5–35.61]; $p < .001$ and two-point MACE composite outcome: HR 19.49, 95%CI [6.01–63.2]; $p < .001$). CAD-RADS outperformed other scores in multivariable models including age, sex and cardiovascular risk factors with a C-index of 0.75 for five-point MACE and of 0.78 for two-point MACE, always $p < 0.001$. High-risk features were not predictors of outcome.

Conclusions CAD-RADS is the most effective CCTA-derived score for MACE prediction in real-world application.

Keywords Coronary artery disease · Computed tomography · CAD-RADS · Prognosis · Artificial intelligence · Diagnosis

Abbreviations

AHA	American Heart Association
AU	Agatston unit
BMI	Body mass index
CAD	Coronary artery disease
CAD-RADS	Coronary artery disease reporting and data system
CCTA	Coronary computed tomography angiography
CCS	Chronic coronary syndrome
EKG	Electrocardiogram
EMB	Endomyocardial biopsy
HRP	High-risk plaque
MACE	Major adverse cardiac event
MRI	Magnetic resonance imaging

Introduction

Coronary artery disease (CAD) is a major cause of morbidity and mortality worldwide [1]. There were 197.2 million ischemic heart disease cases and 9.1 million CAD-related deaths worldwide in 2019 [2]. Hence, it is essential to have accurate tools to diagnose CAD at different stages and to refine risk stratification to guide personalized strategies for secondary prevention and treatment. Coronary computed tomography angiography (CCTA) can detect coronary atherosclerosis at different stages of disease with a negative predictive value for obstructive CAD close to 100%.

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Therefore, CCTA is frequently used as the initial tool for rule-out CAD [3, 4]. CCTA is also the only imaging modality able to depict coronary wall plaques and their composition in a noninvasive way. Beyond the degree of stenosis, also the coronary vessel, the segment involved [5] and several plaque's features were found associated with the risk of major adverse cardiac events (MACE) [6–9].

Hence, different semiquantitative scores have been developed combining CCTA findings with a range of specific diagnostic, prognostic, standardization or communication purposes, as CAD-RADS, segment involvement score (SIS) [10], segment stenosis score (SSS) [10], Leiden score [11] and CT-adapted Leaman score [12]. However, their value in routine clinical practice is not clear. The aim of this study is to evaluate the prognostic value of different CCTA semiquantitative scores in the setting of CCTA performed in subjects with suspected chronic coronary syndrome (CCS) at a low-to-moderate pretest probability of obstructive CAD.

Methods

Patient population

The study is a single-center retrospective observational study, approved by the Ethics Committee (CET1-124/2023; NCT06029387). Informed consent was obtained from all participants and waiver for died patients.

In this study, 6645 adults (> 18 years old) who underwent elective CCTA between 2016 and 2020 at our hospital were screened. Subjects with suspected CCS at low-to-moderate pretest likelihood of CAD according to ESC 2024 guidelines [13] were considered eligible. Patients with a history of myocardial infarction or previous revascularization were not considered eligible for study inclusion.

A total of 2850 subjects were eligible. CCTA were evaluated by a radiologist (V.M.) with 8 years experience in CCTA, to assess image quality according to five-point Likert scale, based on the presence of motion, artifacts and adequate vessel opacification (5: excellent, no artifacts; 4: good, minor artifacts, good diagnostic quality; 3: adequate, moderate artifacts, acceptable for routine clinical diagnosis; 2: poor, severe artifacts impairing accurate evaluation; 1: non-diagnostic) [14] (Fig. 1).

A Likert scale < 4 was considered an exclusion criteria; hence, 361 subjects were excluded for insufficient image quality. Additionally, subjects were excluded for refusal to consent ($n = 91$), existing cardiomyopathy at the time of CCTA ($n = 267$) or a history of active or past malignant neoplasia ($n = 253$).

Enrollment flowchart is reported in Fig. 2.

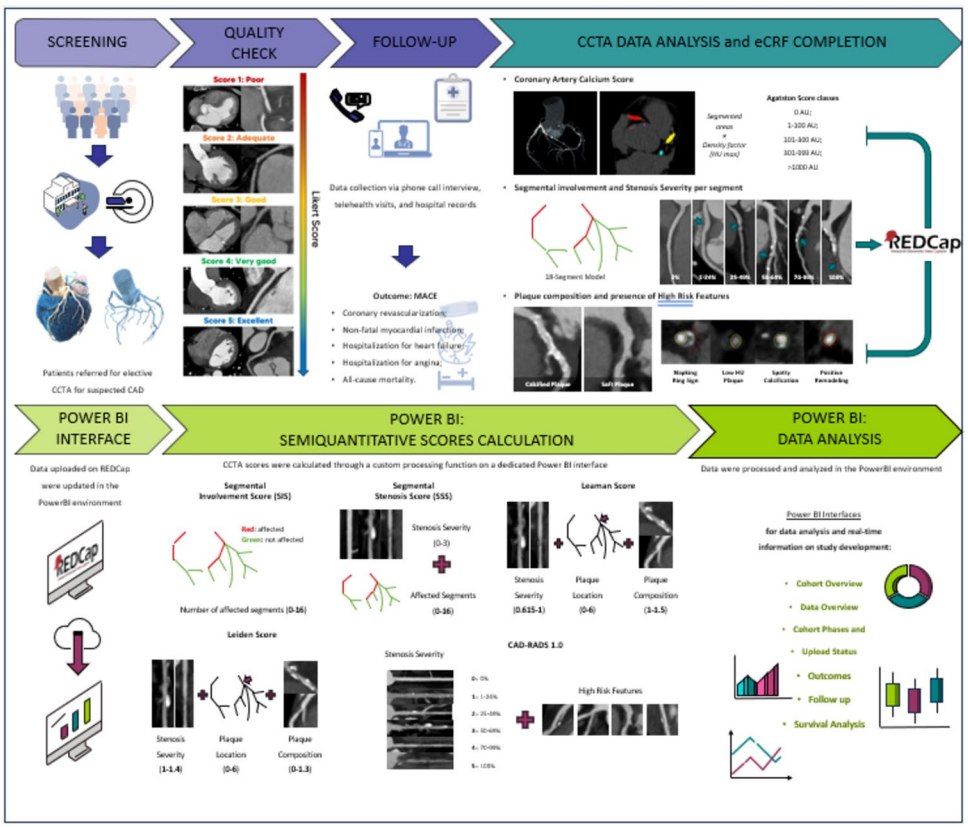

Fig. 1 Study design chart. The study design includes multiple steps: At first, CCTA performed between 2016 and 2020 were screened for eligibility and image quality. Then clinical and outcome data were retrieved. After that, CCTA data from radiological report have been collected in dedicated eCRF that was integrated in a PowerBI-based dashboard for data visualization, real-time calculation of CCTA score and updating of statistical results

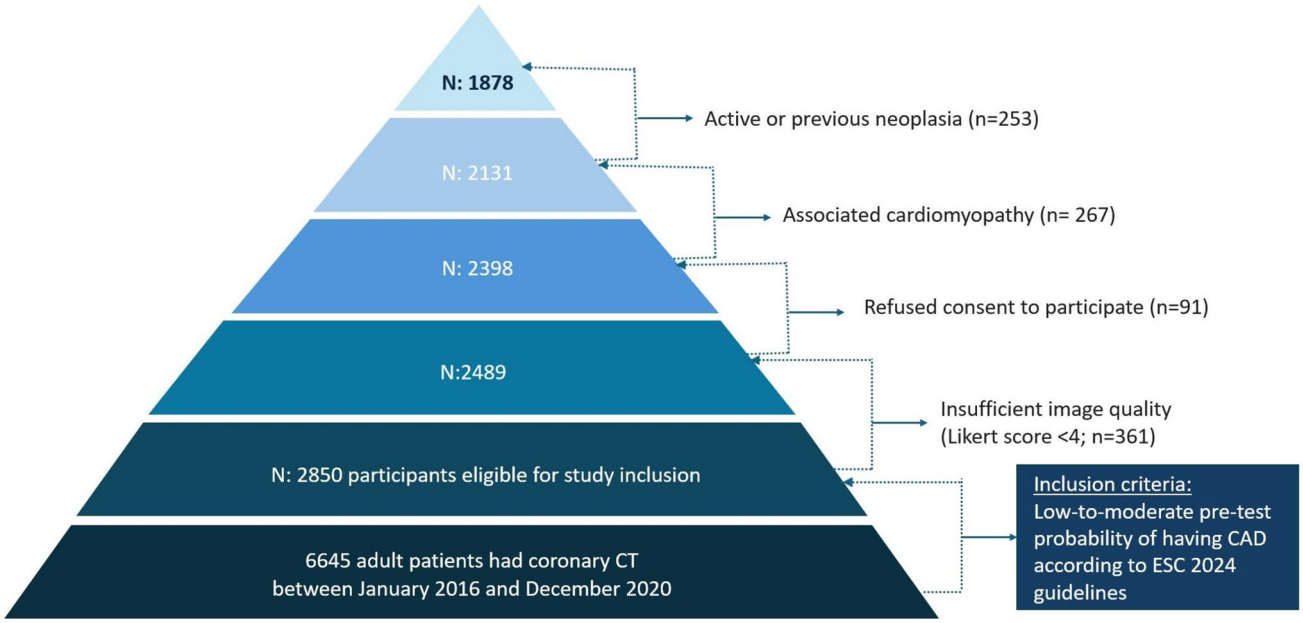

Fig. 2 Enrollment flowchart

Clinical data were obtained from hospital records and medical visits. The following data at the time of CCTA have been collected: (i) demographics (age and sex), (ii) clinical data (symptoms, comorbidities, cardiovascular risk factors, known cardiac disease, EKG, medical therapy) and (iii) other imaging before CCTA (echocardiogram, PET, SPECT, MRI).

Follow-up data were obtained from hospital records, lifelong personal health records from Lombardy region, follow-up medical and/or telehealth visits. Outcome data included the following adapted MACE: late coronary revascularization (after 3 months from CCTA); non-fatal myocardial infarction; hospitalization for heart failure; hospitalization for angina; and all-cause mortality.

The median follow-up time was 62 months (range 50–80).

Clinical, CCTA and outcome data were collected in a dedicated electronic CRF (eCRF) developed in REDCap environment, with variables organized into instruments, applying input validation rules, where relevant, to ensure integrity of data entry. Calculated fields were derived in real time from other variables, improving efficiency and minimizing transcription errors.

CCTA scanning protocol

Patients enrolled were scanned using a 64-slice CT (LightSpeed VCT-XTE scanner, GE-Healthcare) ($n = 479$, 25.5%) or a dual-source CT scanner (SOMATOM Definition Flash; Siemens Healthcare) ($n = 1399$, 74.5%) using a standard protocol including: (i) a prospectively gated precontrast scan for coronary calcium score (CACS); and (ii) a contrast-enhanced CCTA scan acquired with prospective or retrospective gating according to heart rate.

Preparation for CCTA scan included B-blockers to obtain a target heart rate of 65 bpm or less, and sublingual nitroglycerine for coronary vasodilation.

High-iodine concentration contrast agent and saline flush were administered with biphasic or triphasic injection with flow rates of 5–6 mL/s.

CCTA image analysis

CCTA data were retrieved by radiological structured report and collected in the dedicated eCRF, in anonymized fashion, by experienced cardiovascular radiologists (D.V. 10 years and V.M. 8 years of experience), blinded to clinical and outcome data. CCTA preconfigured fields on eCRF were designed to capture detailed segment-level information according to 18 segment model [15], and data regarding stenosis degree, plaque features and plaque composition were collected Supplemental material 1.1. High-risk plaque (HRP) was defined as having at least two high-risk characteristics in a plaque [16], and CACS was quantified at per-patient levels [17]. All the data uploaded and stored on REDCap were parallel updated on a dedicated digital infrastructure (Artificial Intelligence for Preventing Heart Disease, AiPHD) developed in PowerBI (Microsoft) environment. Hence, data were processed

to deliver dynamic and interactive pages with real-time presentation of results about study development and progression, trend of enrollment, list of parameters per patients and cohort, as well as association with risk factors and patient outcome (Fig. 1). The structure of the dedicated PowerBI pages is available in supplemental material 1.2.

CCTA scores (CACS, SIS, SSS, Leaman score, Leiden score, CAD-RADS) have been calculated through custom processing function (supplemental material 1.3) and visualized at patient level, in the overall population and according to the development of MACE.

Statistical analysis

Continuous variables are presented as mean ± standard deviation, and categorical variables are presented as absolute counts and percentages. Normality of continuous data was assessed (Shapiro–Wilk test). When a variable met the assumption of normality, comparisons were made with the Student *t*-test; otherwise, the Mann–Whitney *U* test was applied. Unordered and ordinal categorical variables were compared using the Chi-squared test or Fisher exact test, as appropriate; the Mann–Whitney *U* test was used for ordered, non-normal data.

Follow-up was censored at 36 months. Cumulative event rates were estimated with the Kaplan–Meier method and compared with the log-rank test. Associations between CCTA findings and outcome were quantified with both univariate and multivariable Cox proportional hazards regressions. The analysis was performed for two different composite endpoints: a five-point MACE composite outcome (late coronary revascularization; non-fatal myocardial infarction; hospitalization for heart failure; hospitalization for angina; all-cause mortality) and a two-point MACE composite outcome (non-fatal myocardial infarction; all-cause mortality). Each multivariable model was adjusted for age, sex, hypertension, diabetes mellitus, dyslipidaemia, present and past smoking status and a distinct CCTA score tailored to that model. Results are reported as hazard ratio (HR) with 95% confidence intervals (CIs).

Data imputation was not performed in order to maintain the authenticity of the observed values; therefore, in the prognostic analysis only the variables available for the entire population have been included.

To mitigate over-fitting, we implemented a 10-time repeated tenfold cross-validation scheme and calculated the mean concordance index to summarize prognostic performance. Models were compared with the *Z*-test.

All statistical analyses were conducted in Python (3.9.13).

A two-tailed p-value < 0.05 was considered significant.

Results

A total of 1878 participants were included in the analysis, and the baseline clinical characteristics are reported in Tables 1 and S1.

Participants were mostly men (1222/1878, 65.1%) with a median age of 64 [IQR, 55.0–72.0] years; hypertension was the most frequent cardiovascular risk factor (1237/1878, 66%), followed by smoking (941/1878, 50%) and dyslipidaemia (903/1878, 48%).

At the time of CCTA, 936/1878 (49.8%) participants had symptoms and chest pain was the most frequent (499/936, 53.3%), followed by dyspnea (291/936, 31.1%). EKG data were available in 1338 patients, with evidence of at least one alteration in 607 (45.4%) patients, with repolarization abnormalities resulting the most frequent (198/1338, 14.8%). In total, 717 EKG abnormalities were recorded, as some patients exhibited more than one finding. Echocardiography was available in 970 patients, of whom 204 (21.0%) presented at least one abnormal finding. Overall, 249 echocardiographic abnormalities were documented, since individual patients could exhibit multiple abnormalities.

Most subjects (1170/1878, 62.3%) were receiving pharmacologic therapy before CCTA, especially antihypertensive drugs (990/1878, 52.7%). After CCTA, 41% (777/1878) subjects started at least one new medication, and antiplatelet therapy was the most frequently introduced (434/777, 56%).

CCTA

CCTA findings are reported in Table 2.

Right coronary dominance was most prevalent (84.3%, 1583/1878).

CCTA resulted completely negative, with no evidence of atherosclerosis in 416/1878 participants (22.2%). Most of participants (1305/1878, 69.5%) had non-obstructive CAD (< 50% stenosis) and only 290/1878 (15.4%) had severe CAD (≥ 70% stenosis), of whom 112/290 (38.6%) with multivessel disease.

Median CACS was 57.4 AU [IQR, 0–326.8 AU], and HRP was reported in only 79/1878 subjects (4.2%).

Five-point MACE composite outcome

After 36-month follow-up, 187/1878 (10%) subjects experienced MACE (78 late revascularization, 54 hospitalizations for heart failure or angina, 9 non-fatal myocardial infarction, 93 death).

Subjects experiencing event were similar to those without event in terms of gender (men 129/187, 69% vs 1093/1691, 65%; $p = 0.270$) and BMI (26 [IQR 24–28] vs 26 [IQR

Table 1 Clinical, echocardiography and EKG findings of overall population and according to the occurrence of five-point MACE composite outcome

		Overall ($N=1878$)	Five-point outcome- ($N=1691$)	Five-point outcome + ($N=187$)	*P*-value
Age, median [IQR]		64.0 [55–72]	63.0 [54–71]	70.0 [60.5–78]	< 0.001
Gender, *M* (%)		1222 (65.1)	1093 (64.6)	129 (69.0)	0.270
BMI, median [IQR]	25.7 [23.4–28.5]	25.7 [23.4–28.5]		25.9 [23.6–28.4]	0.701
Cardiovascular risk factors, *n* (%)	Diabetes	265 (14.1)	214 (12.7)	51 (27.3)	< 0.001
	Dyslipidemia	903 (48.1)	803 (47.5)	100 (53.5)	< 0.001
	Active smoker	328 (17.5)	293 (17.3)	35 (18.7)	< 0.001
	Former smoker	613 (32.6)	552 (32.6)	61 (32.6)	< 0.001
	Hypertension	1237 (65.9)	1097 (64.9)	140 (74.9)	< 0.001
Echocardiogram, *n* (%)	Total patients*	970 (51.6)	860 (45.8)	110 (5.8)	
	Reduced ejection fraction	70 (34)	58 (36)	12 (28)	< 0.001
	Dilated left ventricle	38 (19)	30 (19)	8 (18.6)	< 0.001
	Diffuse hypokinesis	39 (19)	34 (21)	5 (11.6)	< 0.001
	Segmental hypokinesis	102 (50)	80 (50)	22 (51)	< 0.001
EKG, *n* (%)	Total patients°	1338 (71)	1194 (71)	144 (77)	
	PVCs-VT	167 (12.5)	158 (13)	9 (6)	< 0.010
	AF	146 (11)	129 (11)	17 (12)	0.173
	Intraventricular conduction delayed	168 (12.6)	148 (12)	20 (14)	0.162
	Q waves	38 (3)	31 (3)	7 (5)	0.052
	repolarization alterations	198 (15)	171 (14)	27 (19)	0.064
Chest pain, *n* (%)		499 (27)	459 (27)	40 (21)	0.010
Type of chest pain, *n* (%)	Atypical	268 (54)	248 (54)	20 (50)	0.366
	Typical	231 (46)	211 (46)	20 (50)	
Dyspnea, *n* (%)		291 (15)	257 (15)	34 (18)	0.149
Type of dyspnea, *n* (%)	At rest	40 (14)	33 (13)	7 (21)	0.373
	Exertional	251 (86)	224 (87)		27 (79)
Palpitations, *n* (%)	218 (11.6)	204 (12.1)	14 (7.5)	0.012	
Syncope, *n* (%)		52 (2.8)	46 (2.7)	6 (3.2)	0.183
Pharmacologic therapy before CCTA, *n* (%)		1170 (62.3%)	1060 (62.7)	110 (58.8)	0.340
Pharmacologic therapy after CCTA, *n* (%)		1450 (77.2%)	1319 (78.0)	131 (70.1)	0.018

*Refers to the total number of patients for whom echocardiographic data were available

°Refers to the total number of patients for whom EKG data were available

23–28] kg/m^2; p = 0.701), but slightly older (70 [IQR 61–78] y.o. vs 63 [IQR 54–71] y.o.; $p < 0.001$) (Table 1).

Symptoms at the time of CCTA were not different between groups, while subjects with MACE had higher prevalence of risk factors, including hypertension (140/187, 74.9% vs 1097/1691, 65%; $p < 0.001$), diabetes (51/187, 27% vs 214/1691, 13%; $p < 0.001$), dyslipidemia (100/187, 53.5% vs 803/1691, 47.5%; $p < 0.001$) and active smoking (35/187, 19% vs 293/1691, 17.3%; $p < 0.001$) (Table 1).

At univariate analysis, diabetes was the main predictor of outcome (HR 2.6, 95%CI [1.86–3.62], $p < 0.001$) among cardiovascular risk factors (Table S2).

The prevalence of pharmacologic therapy before CCTA was not significantly different between the two groups (110/187, 59% vs 1060/1691, 63%; $p = 0.340$), while it was significantly higher after CCTA in subjects without MACE (131/187, 70% vs 1319/1691, 78%; $p = 0.018$).

Subjects experiencing MACE had higher CACS than those without (345 [IQR 62.5–783.0] AU vs 42.7 [IQR 0.0–267] AU; $p < 0.001$) as more prevalent severe classes of all CCTA scores (Table 2).

HRP was not a predictor of outcome (Table 2 and S2).

All CCTA scores resulted able to stratify the risk of MACE (Fig. 3), and all CCTA scores were predictors of outcome with HR increasing as the score increases, with the highest HR in case of severe CAD-RADS (HR 16.35, 95%CI [7.5–35.61]; $p < 0.001$), followed by severe SSS (HR 14.06, 95%CI [6.45–30.66]; $p < 0.001$), severe SIS (HR 11.33, 95%CI [5.25–24.42]; $p < 0.001$), severe Leiden score (HR 9.09, 95%CI [5.17–16.01]; $p < 0.001$), severe CACS (HR

Table 2 CT findings of overall population and according to the occurrence of five-point MACE composite outcome

		Overall ($N=1878$)	Five-point outcome— ($N=1691$)	Five-point outcome + ($N=187$)	*P*-value
Dominance, *n* (%)	Balanced	107 (5.7)	101 (6.0)	6 (3.2)	0.288
	Left dominance	188 (10.0)	170 (10.1)	18 (9.6)	
	Right dominance	1583 (84.3)	1420 (84.0)	163 (87.2)	
CACS, median [IQR], AU		57.4 [0.0–326.8]	42.7 [0.0–267.2]	344.9 [62.5–783.3]	< 0.001
CACS classes, *n* (%)	Absent (0 AU)	698 (37.2)	671 (39.7)	27 (14.4)	< 0.001
	Mild (1–400 AU)	773 (41.2)	697 (41.2)	76 (40.6)	
	Moderate (401–1000 AU)	226 (12.0)	179 (10.6)	47 (25.1)	
	Severe (> 1000 AU)	181 (9.6)	144 (8.5)	37 (19.8)	
CAD-RADS, *n* (%)	Score 0	416 (22.2)	408 (24.1)	8 (4.3)	< 0.001
	Score 1	505 (26.9)	475 (28.1)	30 (16.0)	
	Score 2	384 (20.4)	354 (20.9)	30 (16.0)	
	Score 3	285 (15.2)	237 (14.0)	48 (25.7)	
	Score 4	226 (12.0)	173 (10.2)	53 (28.3)	
	Score 5	62 (3.3)	44 (2.6)	18 (9.6)	
CAD-RADS classes, *n* (%)	Absent (score 0)	416 (22.2)	408 (24.1)	8 (4.3)	< 0.001
	Mild (score 1–2)	889 (47.3)	829 (49.0)	60 (32.1)	
	Moderate (score 3)	285 (15.2)	237 (14.0)	48 (25.7)	
	Severe (score 4–5)	288 (15.3)	217 (12.8)	71 (38.0)	
Leaman score, median [IQR]		3.4 [0.0–8.6]	3.1 [0.0–7.8]	8.6 [3.5–13.4]	< 0.001
Leaman classes, *n* (%)	0	565 (30.1)	546 (32.3)	19 (10.2)	< 0.001
	1–5	533 (28.4)	493 (29.2)	40 (21.4)	
	> 5	780 (41.5)	652 (38.6)	128 (68.4)	
Leiden score, median [IQR]		5.5 [0.0–13.2]	5.0 [0.0–12.2]	12.9 [5.8–19.8]	< 0.001
Leiden classes, *n* (%)	Absent (0)	565 (30.1)	546 (32.3)	19 (10.2)	< 0.001
	Mild (< 5)	340 (18.1)	316 (18.7)	24 (12.8)	
	Moderate (5–20)	794 (42.3)	695 (41.1)	99 (52.9)	
	Severe (> 20)	179 (9.5)	134 (7.9)	45 (24.1)	
SIS, median [IQR]		3.0 [1.0–6.0]	2.0 [1.0–6.0]	6.0 [3.0–9.0]	< 0.001
SIS classes, *n* (%)	Absent (0)	416 (22.2)	408 (24.1)	8 (4.3)	< .001
	Mild (1–2)	511 (27.2)	474 (28.0)	37 (19.8)	
	Moderate (3–4)	282 (15.0)	254 (15.0)	28 (15.0)	
	Severe (≥ 5)	669 (35.6)	555 (32.8)	114 (61.0)	
SSS, median [IQR]		3.0 [1.0–7.0]	2.0 [1.0–6.0]	6.0 [3.0–11.0]	< .001
SSS classes, *n* (%)	Absent (score 0)	419 (22.3)	411 (24.3)	8 (4.3)	< .001
	Mild (score 1–3)	626 (33.3)	579 (34.2)	47 (25.1)	
	Moderate (score 4–8)	516 (27.5)	454 (26.8)	62 (33.2)	
	Severe (score > 9)	317 (16.9)	247 (14.6)	70 (37.4)	
CAD classification ≥ 50%, *n* (%)	Absent	1305 (69.5)	1237 (73.2)	68 (36.4)	< .001
	Single-vessel disease	321 (17.1)	272 (16.1)	49 (26.2)	
	Double-vessel disease	136 (7.2)	101 (6.0)	35 (18.7)	
	Three-vessel disease or LM disease	116 (6.2)	81 (4.8)	35 (18.7)	
CAD classification ≥ 70%, *n* (%)	Absent	1588 (84.6)	1473 (87.1)	115 (61.5)	< .001
	Single-vessel disease	178 (9.5)	139 (8.2)	39 (20.9)	
	Double-vessel disease	70 (3.7)	49 (2.9)	21 (11.2)	
	Three-vessel disease or LM disease	42 (2.2)	30 (1.8)	12 (6.4)	
HRP, *n* (%)		79 (4.2)	71 (4.2)	8 (4.3)	1.000

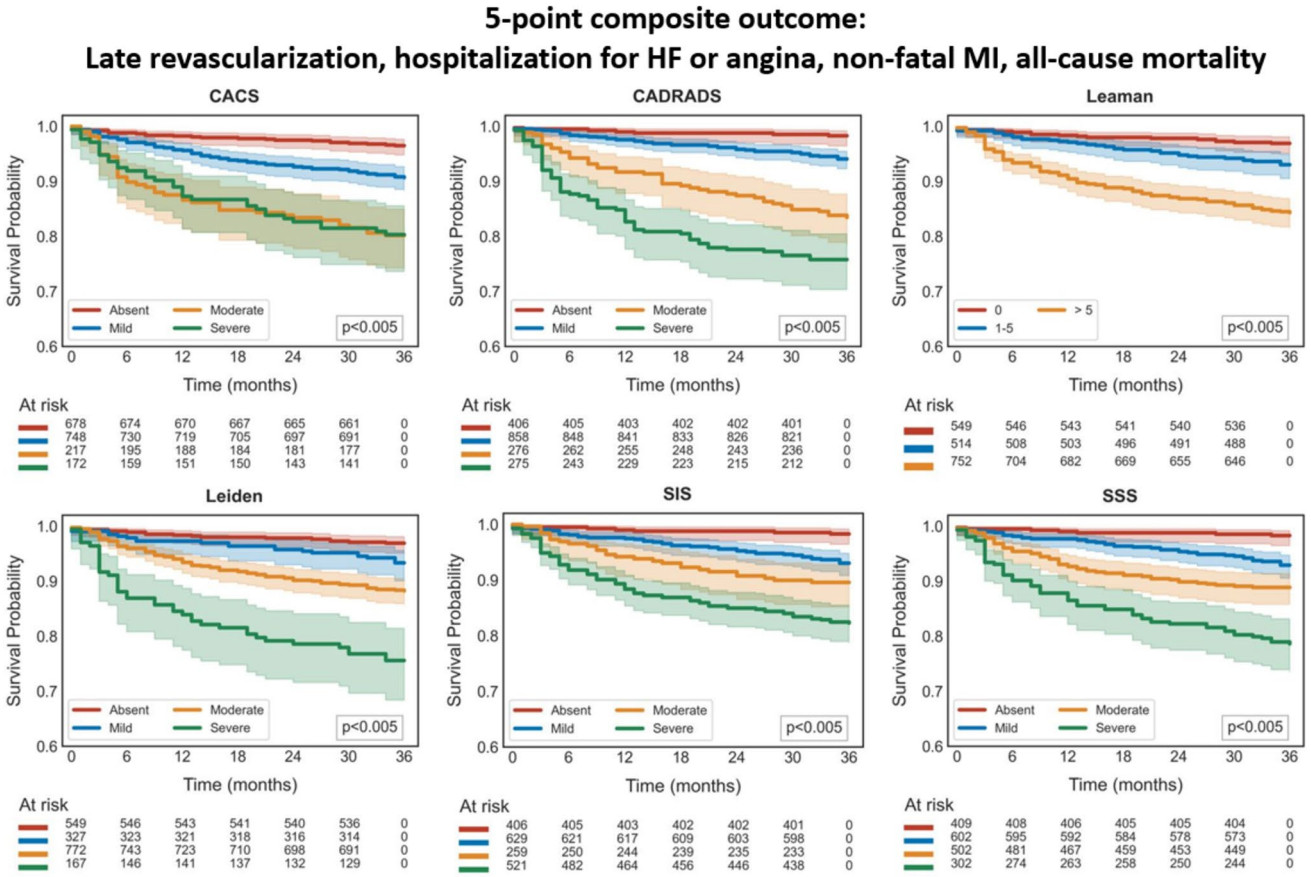

Fig. 3 Kaplan–Meier curves reporting the occurrence of five-point composite outcome (late revascularization, hospitalization for HF or angina, non-fatal MI, all-cause mortality) according to categorized CCTA semiquantitative scores

6.14, 95%CI [3.64–10.36]; $p < 0.001$) and Leaman score > 5 (HR 5.46, 95%CI [3.28–9.07]; $p < 0.001$) (Table S2).

When individually added in a multivariable risk model including age, sex and cardiovascular risk factors, all CCTA scores were significant predictors of outcome and the model including CAD-RADS showed the best performance (C-index 0.75 ± 0.08) significantly higher than the models including other CCTA scores (always $p < 0.01$) (Table 3).

Two-point MACE composite outcome

At 36-month follow-up, 102/1878 (5.4%) subject who experienced non-fatal myocardial infarction or death (Tables S3 and S4) showed no difference in gender (men 70/102, 69% vs 1152/1776, 65%; $p = 0.504$) and BMI (26 [IQR 24–29] vs 26 [IQR 23–28] kg/m^2; $p = 0.736$) compared to subjects without the event, despite they were significantly older (75 [IQR 65–79] vs 63 [IQR 55–71] y.o.; $p < 0.001$).

The prevalence of pharmacologic therapy before CCTA was not significantly different between MACE and no-MACE groups (56/102, 55% vs 1114/1776, 63%; $p = 0.139$), while the rate of pharmacologic therapy after CCTA was significantly higher in patients without the event (53/102, 52% vs 1397/1776, 79%; $p < 0.001$), even considering statins, hypolipidemic drugs, antihypertensive antiarrhythmic and antiheart failure drugs separately (all $p < 0.001$). At univariate analysis among cardiovascular risk factors, diabetes was confirmed the main predictor of outcome (HR 2.85, 95%CI [1.82–4.45], $p < 0.001$) (Table S5).

Subjects who experienced the event had a significantly higher prevalence of CAD ≥ 50% (60/102, 59% vs 513/1776, 29%; $p < 0.001$), higher CACS (325.6 [IQR 80.2–698.8]AU vs 49.4 [0.0–292.5] AU; $p < 0.001$) and higher prevalence of severe CCTA scores (Table S4).

All CCTA categorized scores were able to stratify the risk at Kaplan–Meier survival analysis (Fig. 4), and severe CAD-RADS showed the highest HR (HR 19.49, 95%CI [6.01–63.2]; $p < 0.001$) (Table S5).

At multivariable analysis, all CCTA scores were confirmed predictors of outcome and the one including CAD-RADS showed the highest predictive value (C-index 0.78 ± 0.08) (Table 4).

Table 3 Multivariable models for predicting five-point MACE composite outcome considering each demographic feature, cardiovascular risk factors and one CCTA score for each model

	HR (95% CI)—CAD-RADS range	*p*-value	HR (95% CI)—Leaman range	*p*-value	HR (95% CI)—Leiden range	*p*-value	HR (95% CI)—SSS range	*p*-value	HR (95% CI)—SIS range	*p*-value	HR (95% CI)—CACS range	*p*-value
Age	1.017 (1.001–1.034)	0.035	1.021 (1.004–1.039)	0.015	1.018 (1.001–1.036)	0.037	1.019 (1.002–1.036)	0.03	1.018 (1.001–1.035)	0.049	1.017 (1.0–1.035)	0.051
Sex	1.031 (0.728–1.461)	0.864	0.858 (0.608–1.211)	0.385	0.887 (0.627–1.255)	0.5	0.982 (0.69–1.396)	0.918	0.96 (0.677–1.363)	0.95	0.902 (0.637–1.278)	0.561
Active smoker	1.13 (0.753–1.697)	0.554	1.162 (0.776–1.738)	0.466	1.121 (0.748–1.681)	0.58	1.143 (0.761–1.715)	0.52	1.132 (0.755–1.698)	0.559	1.183 (0.789–1.772)	0.416
Diabetes	1.794 (1.281–2.514)	0.001	1.889 (1.348–2.649)	< 0.001	1.886 (1.345–2.644)	< 0.001	1.788 (1.274–2.51)	0.001	1.827 (1.302–2.564)	< 0.001	1.85 (1.317–2.598)	< 0.001
Dyslipidemia	0.928 (0.679–1.27)	0.642	0.987 (0.722–1.348)	0.933	0.973 (0.712–1.331)	0.865	0.965 (0.706–1.319)	0.823	0.963 (0.704–1.318)	0.767	1.012 (0.741–1.382)	0.94
Former smoker	0.881 (0.624–1.243)	0.471	0.917 (0.651–1.291)	0.619	0.887 (0.628–1.251)	0.494	0.916 (0.65–1.292)	0.618	0.94 (0.668–1.325)	0.72	0.935 (0.663–1.318)	0.699
Hypertension	1.3 (0.889–1.899)	0.176	1.264 (0.864–1.849)	0.228	1.276 (0.872–1.867)	0.21	1.241 (0.848–1.814)	0.266	1.281 (0.876–1.874)	0.234	1.276 (0.87–1.871)	0.212
Mild	2.695 (1.196–6.075)	0.017			1.763 (0.922–3.368)	0.086	3.377 (1.494–7.629)	.003	3.175 (1.386–7.277)	0.004	2.091 (1.277–3.426)	0.003
Moderate	7.198 (3.12–16.603)	< 0.001			2.815 (1.603–4.941)	< 0.001	4.744 (2.069–10.875)	< 0.001	3.914 (1.636–9.362)	0.001	4.135 (2.362–7.237)	< 0.001
Severe	11.587 (5.076–26.446)	< 0.001			5.643 (2.97–10.72)	< 0.001	9.096 (3.942–20.989)	< 0.001	6.805 (3.004–15.415)	< 0.001	3.89 (2.148–7.042)	< 0.001
1–5			1.743 (0.96–3.165)	.068								
> 5			3.513 (2.002–6.164)	< 0.001								
C-index	0.75 ± 0.08		0.69 ± 0.09		0.69 ± 0.09		0.71 ± 0.09		0.69 ± 0.1		0.69 ± 0.09	

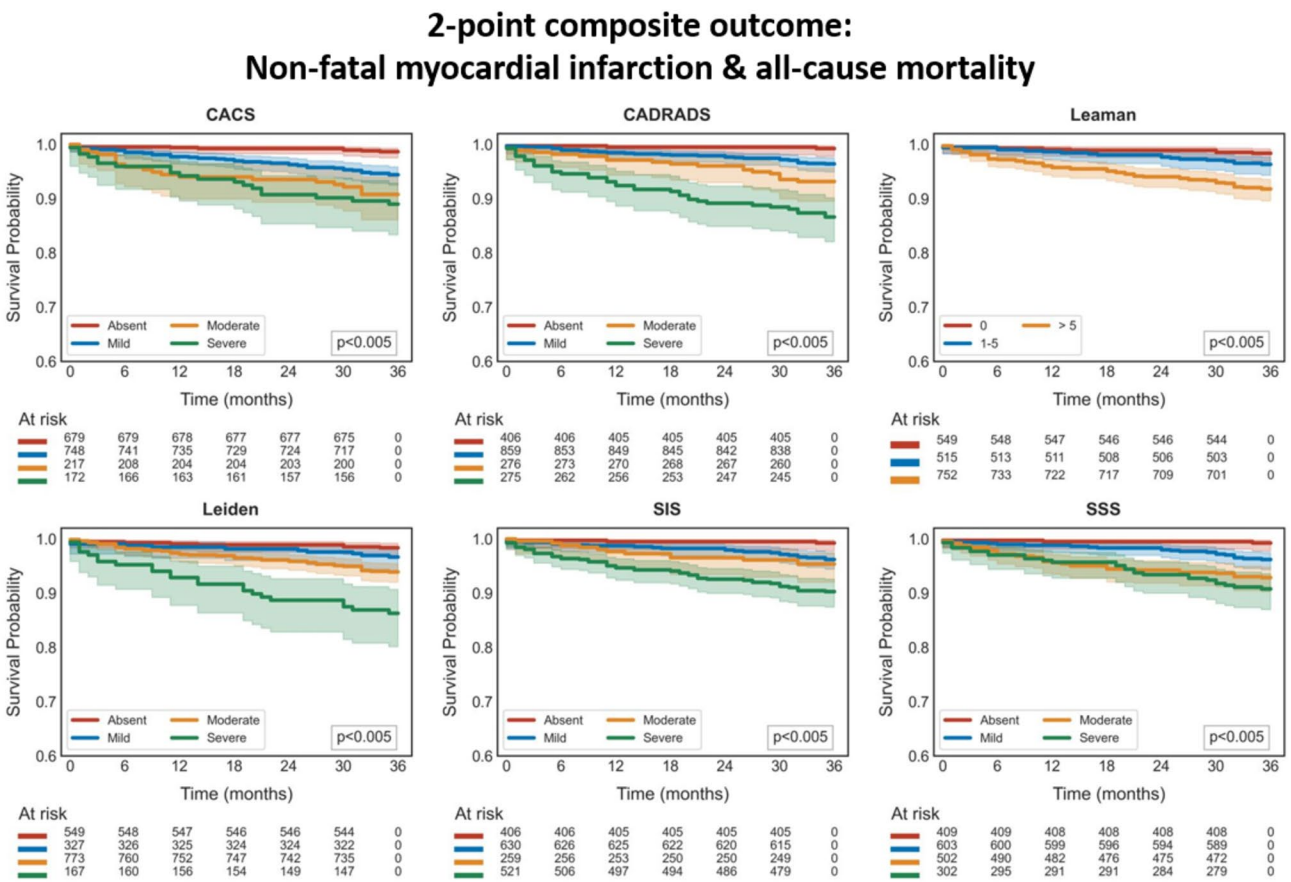

Fig. 4 Kaplan–Meier curves reporting the occurrence of two-point composite outcome (non-fatal myocardial infarction and all-cause mortality) according to categorized CCTA semiquantitative scores

Discussion

In this study, we have investigated the prognostic value of several semiquantitative CCTA scores in a large cohort of 1878 subjects based on data coming from routine clinical practice. All the scores were independent predictors of the outcome over 3 years of follow-up, but CAD-RADS showed the best predictive performance, both for prediction of a five-point MACE composite endpoint, including late revascularization, and for the prediction of a two-point MACE composite endpoint including the non-fatal myocardial infarction and death. The other CCTA scores, namely SIS, SSS, Leaman and Leiden scores, predicted MACE with comparable performance, despite their computation relying on different parameters and criteria for measurement. In fact, SIS includes only data about the presence of atherosclerotic plaque per segment without information about the plaque or the severity of stenosis, SSS contains information about stenosis but lacks of information about plaque features and anatomy, while Leaman and Leiden score provide a more complete description of coronary atherosclerosis evaluating plaque characteristics, location and stenosis degree [11, 18]. Recent studies have demonstrated that assessment of plaque burden and features may enhance risk stratification [19–22]. However, we demonstrated that risk stratification based on subjective estimation of plaque burden and features cannot effectively work in real clinical practice. In fact, CCTA scores including these parameters, such as Leaman and Leiden score, did not resulted to be superior to CAD-RADS, probably being more complex to collect and calculate, and hence potentially affected by limited reliability and applicability in busy daily clinical practice. Similarly, the assessment of HRP was not a predictor of outcome. This result, in contrast to SCOT-HEART study (34), probably reflects the different nature of the data investigated. In our study, we included real-world data retrospectively collected and analyzed, while most of the prospective trials are based on a centralized revision of CCTA and HRP features are collected for study purposes. In fact, as previously demonstrated, HRP features are affected by low reliability with Cohen's *kappa* varying from 0.40 [23] to 0.56 [24] differently from stenosis severity which is more reliable [23], with subsequent variable rate of reporting of HRP in the real clinical practice, and loss of their prognostic implication.

Table 4 Multivariable models for predicting two-point MACE composite outcome (non-fatal myocardial infarction and all-cause of mortality) including demographic data, cardiovascular risk factors and one CCTA score each in categorized fashion

	HR (95% CI)—CAD-RADS	*p*-value	HR (95% CI)—Leaman score	*p*-value	HR (95% CI)—Leiden score	*p*-value	HR (95% CI)—SSS	*p*-value	HR (95% CI)—SIS	*p*-value	HR (95% CI)—CACS	*p*-value
Age	1.066 (1.041–1.092)	< 0.001	1.074 (1.048–1.1)	< 0.001	1.07 (1.044–1.097)	< 0.001	1.07 (1.044–1.097)	< 0.001	1.071 (1.045–1.097)	< 0.001	1.069 (1.042–1.096)	< 0.001
Sex	0.834 (0.516–1.348)	0.459	0.715 (0.445–1.148)	0.165	0.732 (0.455–1.177)	0.198	0.78 (0.481–1.266)	0.315	0.765 (0.473–1.239)	0.277	0.737 (0.457–1.189)	0.211
Active smoker	1.141 (0.64–2.034)	0.654	1.169 (0.657–2.082)	0.595	1.136 (0.637–2.027)	0.666	1.145 (0.642–2.043)	0.646	1.145 (0.643–2.04)	0.646	1.173 (0.659–2.087)	0.588
Diabetes	1.997 (1.269–3.14)	0.003	2.084 (1.325–3.279)	0.001	2.09 (1.328–3.29)	0.001	2.033 (1.291–3.201)	0.002	2.05 (1.302–3.227)	0.002	2.079 (1.318–3.28)	0.002
Dyslipidemia	0.545 (0.352–0.846)	0.007	0.57 (0.368–0.884)	0.012	0.564 (0.364–0.875)	0.011	0.571 (0.368–0.885)	0.012	0.57 (0.367–0.884)	0.012	0.577 (0.373–0.892)	0.013
Former smoker	0.695 (0.429–1.127)	0.14	0.724 (0.448–1.172)	0.189	0.701 (0.432–1.137)	0.149	0.728 (0.45–1.178)	0.197	0.741 (0.458–1.199)	0.222	0.737 (0.455–1.193)	0.215
Hypertension	0.891 (0.552–1.437)	0.636	0.822 (0.509–1.329)	0.424	0.843 (0.522–1.362)	0.485	0.832 (0.515–1.343)	0.451	0.843 (0.522–1.361)	0.484	0.835 (0.516–1.352)	0.463
Mild	2.653 (0.788–8.938)	0.115			1.301 (0.53–3.19)	0.566	3.02 (0.889–10.258)	0.076	3.041 (0.884–10.466)	0.078	2.614 (1.239–5.513)	0.012
Moderate	4.657 (1.313–16.518)	0.017			1.929 (0.898–4.141)	0.092	4.448 (1.302–15.194)	0.017	3.567 (0.99–12.85)	0.052	3.332 (1.436–7.734)	0.005
Severe	8.964 (2.606–30.84)	0.001			3.742 (1.59–8.804)	0.003	5.564 (1.588–19.498)	0.007	4.843 (1.425–16.455)	0.011	3.603 (1.52–8.541)	0.004
1–5			1.298 (0.574–2.935)	0.531								
> 5			2.414 (1.128–5.166)	0.023								
C-index	0.78 ± 0.08		0.74 ± 0.08		0.75 ± 0.08		0.74 ± 0.08		0.75 ± 0.06		0.75 ± 0.08	

CAD-RADS is known to predict significant CAD with excellent sensitivity, specificity and accuracy of 100%, 96.8–98.7% and 98.3–99.3%, depending on the reader [25]. Behind diagnostic values, our study suggested that CAD-RADS can be used not only to standardize reporting, but also for prognostication due to its ability to stratify risk of future cardiovascular events. Some previous studies found similar results to ours, documenting an association between CAD-RADS and MACE [26–29]. In particular, the CONFIRM registry reported an association between CAD-RADS and the risk of non-fatal myocardial infarction and death at 5-year follow-up in a large cohort of 5039 patients [30]. Additionally, our study found incremental prognostic value of CAD-RADS over CACS, with a C-index, respectively, of 0.75 versus 0.69 for five-point MACE and of 0.78 versus 0.75 for two-point MACE (always $p < 0.001$) at 3-year follow-up. A similar finding was found by Bittner [28] in a cohort of 3,840 patients at 2-year follow-up, suggesting the importance of CCTA not only for diagnostic but also for prognostic implication. In fact, interestingly we found that treatment introduction or adjustment after CCTA has a protective role, resulting associated with lower risk of event. In fact, although according 2024 ESC guidelines CACS is suggested as risk modifier in asymptomatic or low-to-moderate likelihood of CAD patients [13], CACS is not able to identify non-calcific atherosclerosis and missing patients that can potentially benefit from optimization of the treatment. On the other hand, CCTA provides a more comprehensive characterization of atherosclerosis. Indeed, in a recent subanalysis of the SCOT-HEART trial, the all-vessels low-attenuation plaque burden was the strongest predictor of outcome (hazard ratio [HR], 1.60 [95%CI, 1.10–2.34] per doubling; $p = 0.014$), suggesting the importance to depict non-calcific plaque.

Therefore, our findings support the importance of CCTA-guided therapy improving patients' risk [31].

Several studies suggested the value in prognostication of plaque burden [32]; however, available software solutions are time-consuming and lack sufficient reproducibility, reducing their applicability in clinical practice and positioning them more as research tools than as viable clinical solutions. For sure, the introduction of photon counting CT and of software based on artificial intelligence will improve automatic plaque burden extraction and characterization [33], with beneficial impact on clinical practice and prognostication.

This study has some limitations: First, this is a retrospective single-center study; hence, data should be confirmed on larger multicenter data. Second, no data about inter-reader agreement were reported. CCTA data analyzed are derived by CCTA radiological reports, reflecting the scope of the study to investigate prognostic role of CCTA quantitative scores in the real clinical practice and not after a centralized revision. Third, risk modifiers such as plaque burden and ischemia were not analyzed.

In conclusion, our study conducted on real-world clinical data, found that CAD-RADS, in its easier form including simply the severity of stenosis, was the semiquantitative score with the best performance in the prediction of 3-year outcome. Our study therefore supports its wider application in clinical reporting for both diagnostic and prognostic purposes. Future studies are needed to investigate the impact of CAD-RADS score modifiers as plaque burden and ischemia over CAD-RADS risk stratification, in order to clarify their value in prognostication.

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Author contributions AP, AC, EB, DV, AnE performed literature review, manuscript drafting and final manuscript editing. AP, DV, EB, VM, AB, FP, ArE, DS, GG, GM, MF, RV and CG performed data collection and image analysis. AC, FB, DS, BMC, CG, CT developed the platform for data collection and automatic analysis. All authors read and approved the final manuscript.

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Data availability Data will be made available on reasonable request.

Declarations

Conflict of interest Beatrice Maria Civelli and Gabriele Chiodini are computer scientist from Porini srl. The remaning authors declare that they have no conflict of interests.

Ethical approval The study has been approved by Ethic Committee (CET1-124/2023).

Consent for publication Informed consent was obtained from all participants.

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