Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Age changes what a risk-factor count tells the referring physician: recorded cardiometabolic burden and obstructive coronary artery disease on CT angiography in a single-centre cross-sectional study

Xiaohua Sun
Taizhou People's Hospital
Huimin Gu
Taizhou People's Hospital
Dongrui Liu
wbaoc1177@hhu.edu

Taizhou People's Hospital

Research Article

Keywords: Coronary computed tomography angiography, cardiometabolic risk factors, multimorbidity, effect modification, glycated haemoglobin, general practice

Posted Date: November 4th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11131474/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

General practitioners considering coronary computed tomography angiography (CCTA) usually have only admission diagnoses and recent laboratory results. Whether the number of cardiometabolic risk factors in those records means the same at every age, and whether HbA1c and eGFR add discrimination, is untested.

Methods

Single-centre cross-sectional study of adult CCTA examinations from the general practice and cardiology pathways of a tertiary hospital, 2021 to 2025. The exposure was the number of hypertension, diabetes, dyslipidaemia and renal impairment recorded from diagnoses and laboratory results within 30 days before CCTA. Obstructive coronary artery disease (CAD) was read from reports by a validated text rule. After excluding prior revascularisation and incomplete laboratory data, 5,225 patients were analysed with patient-clustered logistic regression.

Results

Obstructive CAD was reported in 1,018 patients (19.5%), similarly in both pathways. Each additional risk factor carried an adjusted odds ratio of 1.70 (95% CI 1.57 to 1.84); adjusted prevalence rose from 9.2% with none to 43.9% with four. Age modified the association (interaction $P < 0.001$): the per-factor odds ratio was 2.20 under 55 years and 1.46 at 75 or older; obstructive CAD without recorded risk factors was 0.4% and 21.2%. Adjusted odds ratios were 4.41 for HbA1c of 8.0% or higher and 1.64 for eGFR below 60 mL/min/1.73 m².

Conclusions

The recorded risk-factor count shows a dose-response relationship with obstructive CAD that weakens with age. A clean record reassures in younger patients but much less in older ones. HbA1c and eGFR refine risk; risk-factor review before CCTA should be read by age.

Background

Ischaemic heart disease remains the leading contributor to disease burden worldwide and in China, where the number of people living with cardiovascular disease continues to rise (1, 2). Coronary computed tomography angiography (CCTA) is now a first-line test for stable chest pain, and its reporting system, CAD-RADS, flags possible obstructive disease at grade 3 or higher (3–5). Coronary stenosis rarely reflects a single disease, because hypertension, diabetes, dyslipidaemia and renal impairment

tend to coexist and are now grouped by the American Heart Association under the cardiovascular-kidney-metabolic (CKM) syndrome (6). This study asks whether the number of such risk factors recorded in the chart before CCTA carries the same meaning at every age, and whether glycaemic control and eGFR separate risk further.

That more risk factors mean more coronary disease is clinical common sense. CKM syndrome is common. Most adults in the United States are at stage 1 or above, roughly one in four Chinese adults is at stage 3 or 4, and mortality rises with each stage (7, 8). Mortality also multiplies with the number of coexisting cardiometabolic diseases, which is the classical basis for grading by count (9). Individual risk factors have well-described relationships with CCTA findings. Hypertension is associated with more extensive and more severe coronary atherosclerosis (10), and obstructive CAD is more often detected in patients with diabetes than without (11). Reduced kidney function independently predicts adverse events in CCTA populations (12), and metabolic phenotypes relate to CAD and events in stable chest pain (13). Recent coronary imaging studies framed around CKM have mostly relied on formal staging or on more complex models (14, 15). Chinese routine data have not yet been used to relate a risk-factor count taken directly from admission records and laboratory results to the CCTA result of the same episode of care. Nor has that relationship been examined across age groups and laboratory bands.

The interaction between age and risk factors matters for pre-test assessment. The CONFIRM registry showed a dose-response relationship between CCTA findings and mortality that differed by age and sex (16, 17). Among symptomatic patients aged 18 to 45 years, stenosis of 50% or more was found in only 1.8% (18). Young patients with no risk factors and a zero calcium score have very low event rates, whereas those with several risk factors have markedly higher rates (19). In a large registry spanning younger and older patients, obstructive disease predicted adverse events in both groups (20). A multicentre Chinese cohort of 16,300 CCTA examinations found that plaque burden increases nonlinearly with age and that atherosclerosis begins about 20 years later in women than in men (21). Among 11,234 Chinese CCTA patients, current pre-test probability models underestimated obstructive CAD in the young (22). Newer clinical likelihood models have been calibrated against CCTA findings in several cohorts (23), and machine-learning work on CAD-RADS and CCTA yield in older adults offers further reference points (24, 25). CAD-RADS grades carry prognostic value (26), but CCTA reports have their own limits: they may underestimate plaque progression, agree imperfectly with invasive angiography, and lose specificity in advanced chronic kidney disease (27–29). From a primary care standpoint, a Swedish cross-sectional study of 483 patients referred directly from primary health care centres found suspected significant stenosis in 18% (30). That yield matched the referring physicians' pre-test probability estimates. These studies point to an age-dependent meaning of a risk factor, but they used events as the endpoint or pre-test information as model input. The relationship between chart-recorded risk factors and the CCTA result of the same episode, across age groups, still needs direct testing.

Research on routine data has to state where the data came from, how they were linked, how variables were defined and whether the data are fit for the question (31, 32). Diagnostic codes record disease with

limited sensitivity, and the measured level of multimorbidity depends heavily on the data source (33, 34). The process by which data come to be recorded can itself introduce selection bias (35). An odds ratio from logistic regression and an absolute prevalence in a defined population are different quantities, and the latter needs an explicit standard population (36). With these points in mind, we studied five years of adult CCTA examinations ordered through the general practice and cardiology pathways of one hospital and asked three questions. First, do the two pathways differ in case mix and prevalence, and can they be pooled? Second, in the first-evaluation population, does the number of risk factors recorded before CCTA show a dose-response relationship with obstructive CAD, and does age modify it? Third, do HbA1c and eGFR levels separate risk beyond the presence or absence of diabetes or renal impairment?

Methods

Study Design and Setting

This was a single-centre retrospective cross-sectional study of adult patients who underwent CCTA between January 2021 and December 2025 after attending the general practice or cardiology service of a tertiary hospital in Jiangsu, China. Data sources were de-identified radiology reports, hospital information system records, electronic medical records and laboratory system extracts. Most CCTA at this hospital is ordered by cardiology and general practice. We chose these two pathways for two reasons. The study was designed from a general practice perspective and focused on risk-factor information that a general practitioner can read directly from the chart. Cardiology orders the most CCTA at this hospital, and its patients are mostly referred for cardiovascular symptoms. It therefore served as the reference pathway for testing whether the relationship between risk factors and CCTA findings changes with the referral population. The stratified comparison of the two pathways was pre-specified in the protocol. The small number of CCTA examinations ordered by other departments was not extracted. The unit of analysis was the examination, and a de-identified patient identifier linked repeat examinations of the same patient. There was no follow-up. Reporting follows the relevant items of STROBE and CODE-EHR (31, 32); the STROBE checklist is provided as Additional file 2.

Study Population and Data Sources

During the study period 8,118 CCTA examinations were performed in adults. Six were invalid because the examination was refunded, repeated or did not assess the coronary arteries, leaving 8,112 valid examinations. Following the definition of a population without known CAD used in the CONFIRM registry (16), we excluded 758 examinations in patients with prior stenting, percutaneous coronary intervention or bypass grafting, leaving 7,354. We then excluded 2,129 examinations in which at least one laboratory result needed for risk-factor classification was missing within 30 days before CCTA, giving a main analysis population of 5,225 examinations (n = 5,225; Fig. 1). All eligible examinations in the period were included; no sample size was calculated.

Outcome: Obstructive CAD on CCTA

The outcome was obstructive CAD on the formal CCTA report, defined as maximal coronary stenosis of 50% or more, or a described severity equivalent to CAD-RADS grade 3 or higher (5, 37). A fixed text rule identified coronary descriptions only, handled percentages, occlusion and mild, moderate and severe wording, and excluded carotid, aortic and other non-coronary stenoses. “Equivalent to CAD-RADS” means a grade derived from the report text; not every report contained a structured CAD-RADS field.

The rule was developed on a separate sample. Two hundred positive and borderline reports, stratified by department and wording, were reviewed by two attending physicians against the same report text. Discordant or uncertain records were used to revise the rule. This sample contributed to rule development and was not used for validation. After the rule was frozen, 400 of the remaining reports were drawn at random, stratified by rule result (200 positive, 200 negative), and 150 of these (75 and 75) were randomly selected for human review. Two general practitioners (XS and HG), blinded to the rule result, independently read each report and judged whether maximal stenosis reached 50%. Inter-reader agreement was summarised with Cohen’s kappa. Reports with discordant readings were read jointly until consensus was reached, and the consensus served as the reference standard for agreement, kappa, sensitivity and specificity of the rule. Thirty-eight reports whose stenosis could not be classified by the text rule were adjudicated one by one by a single investigator. Six were invalid examinations, and five described only grafts or stents after prior revascularisation without describing the native vessels. The remaining 27 were judged as stenosis below 50% and included as such. The rule was not changed after validation.

Exposure: Cardiometabolic Risk Factors Recorded Before CCTA

Risk factors were classified from admission diagnoses and from the valid laboratory result closest to the examination date within 30 days before CCTA, including the day of the examination. Information generated after the examination was not used. Hypertension was defined by diagnosis. Diabetes was defined by diagnosis or HbA1c of 6.5% or higher. Dyslipidaemia was defined by diagnosis or by any one of four laboratory criteria: low-density lipoprotein cholesterol of 3.4 mmol/L or higher, total cholesterol of 5.2 mmol/L or higher, triglycerides of 1.7 mmol/L or higher, or high-density lipoprotein cholesterol below 1.0 mmol/L. Renal impairment was defined by a diagnosis of chronic kidney disease or by eGFR below 60 mL/min/1.73 m². The reported eGFR was used when available; otherwise it was calculated from creatinine, age and sex with the 2021 CKD-EPI Eq. (38). Because records did not cover three months or more, a single reduced eGFR was treated as a marker of renal impairment and not as a diagnosis of chronic kidney disease (39). Laboratory values entered threshold decisions only when their units matched the threshold.

Each positive risk factor scored one point, giving a count from 0 to 4; two or more defined cardiometabolic multimorbidity. A patient without the relevant diagnosis and without the relevant laboratory result before CCTA could not be classified for that factor and was not included in the main analysis. Smoking, body mass index and complete medication records were incompletely documented

and were not analysed. A recorded diagnosis of chest pain, chest tightness or angina served as a proxy for a symptomatic indication.

Laboratory Grading

HbA1c was graded as below 5.7%, 5.7 to 6.4%, 6.5 to 7.9% and 8.0% or higher. eGFR was graded as 90 or higher, 60 to 89 and below 60 mL/min/1.73 m², with the lowest band split into 45 to 59 and below 45. Graded analyses were restricted to patients with the relevant result. Adjusted odds ratios were estimated first against the reference band and then re-estimated after adding the risk-factor count. The second estimate tests whether the laboratory level separates risk once the corresponding risk factor has already been counted.

Statistical Analysis

Continuous variables are presented as mean and standard deviation and compared with Welch's t test; categorical variables are presented as counts and percentages and compared with the chi-squared test. The analysis proceeded in three steps.

The first step compared case mix and prevalence between the cardiology and general practice pathways. The difference was expressed as the odds ratio for cardiology versus general practice, and age, sex, recording period (2021 to 2023 or 2024 to 2025) and the risk-factor count were added in sequence to trace its source. To characterise the two referral populations we also compared three proportions: a recorded symptom of chest pain, chest tightness or angina; a non-CAD cardiac diagnosis (atrial fibrillation, valvular disease, heart failure, cardiomyopathy or arrhythmia); and a metabolic diagnosis (diabetes, hyperuricaemia, fatty liver, obesity or metabolic syndrome). Prevalence was reported for three referral profiles: symptomatic record; no symptomatic record with multimorbidity; and no symptomatic record with fewer than two risk factors. The effect of the count was estimated within each pathway and among patients without a symptomatic record, with interaction tests. If the difference between pathways could be explained by case mix and the effect of the count was consistent, subsequent analyses were pooled with pathway as a covariate.

The second step was the primary model: logistic regression with the risk-factor count as a continuous variable, adjusted for age, sex, pathway and recording period, with standard errors clustered by patient. Age was modelled with a restricted cubic spline with knots at 40, 59, 70 and 83 years. Departure from linearity in the count and nonlinearity in age were tested with robust Wald tests. To report absolute prevalence, predicted probabilities from a model with the count as a categorical variable were marginally standardised to the covariate distribution of the main analysis population. This gave the adjusted prevalence at each count and the prevalence difference and ratio relative to zero. Confidence intervals used the patient-clustered covariance and the delta method (36). Effect modification by age was tested in two ways. An interaction between continuous age and the count was added to the primary model. The

same model was then fitted within age groups (below 55, 55 to 64, 65 to 74 and 75 years or older), and crude prevalence with Wilson 95% confidence intervals was tabulated for each age group by count cell.

The third step examined the individual risk factors and the laboratory bands. The four factors were estimated in one mutually adjusted model. HbA1c and eGFR bands were analysed as described above. The symptom record was used as a covariate and as a stratification variable. The age-modification, laboratory-band and symptom analyses were carried out after the primary result was fixed. They were specified after the primary result and are reported as exploratory analyses. Sensitivity analyses included the 2,129 examinations with incomplete pre-test laboratory data (unclassifiable risk factors treated as not recorded), included patients with prior revascularisation, and restricted the data to 2024 to 2025 with adjustment for year. Two-sided P below 0.05 was taken as statistically significant; interpretation rests mainly on effect estimates and their confidence intervals. Analyses used Python with statsmodels; scripts, variable definitions and aggregate outputs are archived locally.

Use of Large Language Models

Large language models (Anthropic Claude and OpenAI Codex) were used, under the authors' direction, for code drafting, checking of statistical implementation, verification of reference metadata against PubMed and Crossref, and manuscript drafting and translation. No patient-level data were entered into any such tool. All 38 rule-unclassifiable reports were adjudicated by an investigator, and all 150 validation reports were read by the two general practitioners. As an internal quality check only, a large language model had earlier re-read the 400 sampled reports and agreed with the rule in 397; this result was not used as validation evidence. The authors reviewed and take responsibility for all text, citations and analyses.

Ethics Approval

The study used de-identified hospital data, and no patient-level data left the hospital-authorised local environment. Ethics approval and the consent waiver are stated in the Declarations.

Results

Participants

After the exclusions described above, the main analysis population comprised 5,225 examinations in 5,225 different patients (n = 5,225), of which 1,018 (19.5%) showed obstructive CAD on CCTA. Mean age was 62.8 years (SD 12.9); 25.2%, 27.4%, 27.0% and 20.4% of patients were aged below 55, 55 to 64, 65 to 74 and 75 years or older, and 2,850 (54.5%) were men. The risk-factor count was 0, 1, 2, 3 and 4 in 14.6%, 34.5%, 34.6%, 15.0% and 1.2% of examinations. Cardiometabolic multimorbidity was present in 2,655 patients (50.8%), and 1,243 (23.8%) had a recorded symptom of chest pain or angina. Patients with obstructive CAD were older, more often men, and more often positive for each of the four risk factors

(Table 1). The 2,129 examinations excluded for incomplete pre-test laboratory data had an obstructive CAD prevalence of 20.0%, similar to the main analysis population.

Table 1
Characteristics and recorded risk factors by CCTA result

Characteristic	Total (n = 5,225)	Non-obstructive (n = 4,207)	Obstructive (n = 1,018)
Age, years	62.8 ± 12.9	61.3 ± 13.0	69.0 ± 10.7
Men	2,850 (54.5)	2,189 (52.0)	661 (64.9)
Hypertension	3,153 (60.3)	2,403 (57.1)	750 (73.7)
Diabetes	1,544 (29.5)	1,037 (24.6)	507 (49.8)
Dyslipidaemia	3,080 (58.9)	2,437 (57.9)	643 (63.2)
Renal impairment	246 (4.7)	157 (3.7)	89 (8.7)
Risk-factor count	1.54 ± 0.96	1.43 ± 0.93	1.95 ± 0.95
Recorded chest pain or angina	1,243 (23.8)	925 (22.0)	318 (31.2)

Values are mean ± SD or n (%). Risk factors were classified from admission diagnoses and laboratory results within 30 days before CCTA.

Validation of outcome ascertainment

Of the 150 validation reports, the two readers agreed independently on 139 of 149 (93.3%; one report was left unrated by one reader), with a kappa of 0.87. The 11 discordant or unrated reports all reached consensus on joint reading. Against the consensus, the text rule agreed on 149 of 150 (99.3%), kappa 0.99; sensitivity was 100% (74/74; 95% CI 95.1 to 100%) and specificity 98.7% (75/76; 95% CI 92.9 to 100%). The single disagreement was a report describing a left anterior descending stenosis of “about 50%”, which the rule classified as positive and the readers, by consensus, as negative.

Cardiology and general practice pathways

Of the 7,354 first-evaluation examinations, 6,044 came through cardiology and 1,310 through general practice, with crude prevalence of 19.8% and 18.6%. In the main analysis population the figures were 19.8% and 18.1%, a difference that was not statistically significant (odds ratio for cardiology versus general practice 1.12, 95% CI 0.93 to 1.34, P = 0.24; 1.05, 0.87 to 1.27, after adjustment for age and sex). The referral populations differed (Table 2). General practice patients were slightly younger: 27.4% were under 55 (cardiology 23.8%) and 15.7% were 75 or older (cardiology 22.2%). Cardiology patients more often had recorded hypertension (61.7% vs 53.0%) and renal impairment (4.5% vs 3.2%); general practice patients more often had diabetes (29.7% vs 25.3%) and dyslipidaemia (50.6% vs 46.7%); multimorbidity was similar (43.6% vs 43.0%). A symptom of chest pain, chest tightness or angina was recorded in 24.0%

of cardiology and 17.9% of general practice patients. A non-CAD cardiac diagnosis such as atrial fibrillation, valvular disease or heart failure was recorded in 38.6% of cardiology patients but only 13.7% of general practice patients. Metabolic diagnoses were similar (35.3% vs 37.0%). By referral profile, patients with no symptomatic record and multimorbidity made up 43.1% of the general practice pathway (cardiology 39.1%), and patients with a symptomatic record 17.2% (cardiology 25.2%). Among patients with a symptomatic record, prevalence was 27.0% in cardiology and 16.2% in general practice. Among those without, it was similar in the two pathways: 22.7% vs 24.2% with multimorbidity, and 11.5% vs 12.3% with fewer than two risk factors. The per-factor odds ratio was 1.71 (95% CI 1.56 to 1.86) in cardiology and 1.72 (1.41 to 2.10) in general practice, interaction P = 0.92; among patients without a symptomatic record it was 1.65 (1.49 to 1.84) and 1.75 (1.41 to 2.18). Cardiology therefore mainly saw symptomatic referrals and work-up of other cardiac disease, and general practice mainly saw asymptomatic multimorbid patients. Because prevalence was similar and the effect of the count was the same, the subsequent analyses were pooled across the 5,225 examinations with pathway as a covariate.

Table 2
Case mix of the cardiology and general practice pathways (7,354 first-evaluation examinations)

Characteristic	Cardiology (n = 6,044)	General practice (n = 1,310)
Age, years	63.4 ± 13.1	62.1 ± 11.9
Age below 55 years	1,436 (23.8)	359 (27.4)
Age 75 years or older	1,343 (22.2)	206 (15.7)
Men	3,278 (54.2)	724 (55.3)
Hypertension	3,730 (61.7)	694 (53.0)
Diabetes	1,529 (25.3)	389 (29.7)
Dyslipidaemia	2,822 (46.7)	663 (50.6)
Renal impairment	269 (4.5)	42 (3.2)
Multimorbidity (2 or more factors)	2,636 (43.6)	563 (43.0)
Recorded chest pain, chest tightness or angina	1,449 (24.0)	235 (17.9)
Non-CAD cardiac diagnosis	2,335 (38.6)	180 (13.7)
Metabolic diagnosis	2,133 (35.3)	485 (37.0)
Incomplete pre-test laboratory data	1,758 (29.1)	371 (28.3)
Obstructive CAD on CCTA	1,199 (19.8)	244 (18.6)

Values are mean ± SD or n (%). Counts were derived from proportions and totals; the last digit follows the source tables. Risk factors were classified from admission diagnoses and laboratory results within 30

days before CCTA; for patients with incomplete laboratory data, an unclassifiable factor was treated as not recorded. Non-CAD cardiac diagnosis: atrial fibrillation, valvular disease, heart failure, cardiomyopathy or arrhythmia. Metabolic diagnosis: diabetes, hyperuricaemia, fatty liver, obesity or metabolic syndrome.

Dose-response relationship between the risk-factor count and obstructive CAD

There were 765, 1,805, 1,810, 782 and 63 examinations with 0, 1, 2, 3 and 4 risk factors, with 63, 262, 385, 275 and 33 cases of obstructive CAD; crude prevalence rose from 8.2% to 52.4%. The adjusted odds ratio per additional risk factor was 1.70 (95% CI 1.57 to 1.84). The test for departure from linearity in the count gave P = 0.614; nonlinearity in age gave P = 0.029, and the age spline was retained in all subsequent models. Adjusted prevalence at 0 to 4 risk factors was 9.2%, 15.1%, 20.9%, 31.9% and 43.9% (Table 3, Fig. 2). Relative to no risk factor, four risk factors carried a prevalence difference of 34.7 percentage points (95% CI 22.4 to 47.0) and a prevalence ratio of 4.78 (3.34 to 6.84).

Table 3
Crude and adjusted prevalence of obstructive CAD by risk-factor count

Risk-factor count	Examinations	Obstructive CAD, n (crude %)	Adjusted prevalence, % (95% CI)
0	765	63 (8.2)	9.19 (7.30 to 11.50)
1	1,805	262 (14.5)	15.06 (13.50 to 16.77)
2	1,810	385 (21.3)	20.87 (19.14 to 22.71)
3	782	275 (35.2)	31.93 (28.94 to 35.08)
4	63	33 (52.4)	43.90 (32.39 to 56.11)

Adjusted prevalence was obtained by marginal standardisation of a model with the count as a categorical variable, an age spline, sex, pathway and recording period to the main analysis population; confidence intervals used the patient-clustered robust delta method.

Age modification of the dose-response relationship

The interaction between age and the risk-factor count was statistically significant (robust Wald P < 0.001) with a negative coefficient. By age group, the adjusted odds ratio per additional risk factor fell from 2.20 (95% CI 1.73 to 2.80) below 55 years to 2.02 (1.70 to 2.41) at 55 to 64 and 1.57 (1.37 to 1.80) at 65 to 74. At 75 years or older it was 1.46 (1.27 to 1.68) (Table 4).

The age difference is more striking in absolute terms (Table 4, Fig. 3). Among 243 patients under 55 with no recorded risk factor, one (0.4%) had obstructive CAD; with one, two and three risk factors the prevalence was 5.5%, 10.2% and 19.3%. Among 118 patients aged 75 or older with no recorded risk factor, 25 (21.2%) had obstructive CAD, and with three risk factors the prevalence was 45.9%. Cells with four risk factors held 4 to 27 examinations per age group and are not interpreted.

Table 4
Risk-factor count and obstructive CAD within age groups

Age group	Examinations	Obstructive CAD, n (%)	0 factors, %	1 factor, %	2 factors, %	3 factors, %	OR per factor (95% CI)
Below 55 years	1,317	104 (7.9)	0.4 (1/243)	5.5 (26/477)	10.2 (45/443)	19.3 (29/150)	2.20 (1.73 to 2.80)
55 to 64 years	1,431	219 (15.3)	3.8 (8/213)	10.1 (50/496)	17.8 (90/506)	32.0 (65/203)	2.02 (1.70 to 2.41)
65 to 74 years	1,409	348 (24.7)	15.2 (29/191)	18.8 (89/474)	26.1 (128/490)	39.1 (92/235)	1.57 (1.37 to 1.80)
75 years or older	1,068	347 (32.5)	21.2 (25/118)	27.1 (97/358)	32.9 (122/371)	45.9 (89/194)	1.46 (1.27 to 1.68)

Models within each age group were adjusted for age (continuous within the group), sex, pathway and recording period. Cells with four risk factors (4, 13, 19 and 27 examinations) are not shown. Interaction $P < 0.001$.

Individual risk factors and laboratory grading

In the mutually adjusted model, diabetes had the strongest association with obstructive CAD (odds ratio 2.41, 95% CI 2.07 to 2.80), followed by hypertension (1.50, 1.28 to 1.76), renal impairment (1.39, 1.03 to 1.88) and dyslipidaemia (1.37, 1.18 to 1.60) (Table 5).

By HbA1c band, prevalence was 10.9%, 16.3%, 30.4% and 39.1% for below 5.7%, 5.7 to 6.4%, 6.5 to 7.9% and 8.0% or higher. After adjustment for age, sex, pathway and recording period, the odds ratios relative to the lowest band were 1.32 (1.07 to 1.64), 2.71 (2.14 to 3.44) and 4.41 (3.28 to 5.92). After further adjustment for the risk-factor count, the odds ratio for HbA1c of 8.0% or higher remained 2.65, indicating that among patients with diabetes, worse glycaemic control went with higher prevalence. By eGFR band, prevalence was 15.6%, 27.5% and 35.0% for 90 or higher, 60 to 89 and below 60 mL/min/1.73 m². After adjustment, 60 to 89 did not differ from 90 or higher (odds ratio 1.16, 0.96 to 1.39), whereas below 60 carried an odds ratio of 1.64 (1.18 to 2.27): 1.62 (1.13 to 2.31) for 45 to 59 and 1.72 (0.91 to 3.23, 58 examinations only) for below 45. HbA1c and eGFR bands were associated with prevalence within every age group, and the relative gradient was steeper in younger patients (Fig. 4).

Table 5
Individual risk factors and laboratory bands in relation to obstructive CAD

Variable	Examinations, n (%)	Prevalence, %	Adjusted OR (95% CI)	OR after adding the count
Hypertension (mutually adjusted)	3,153 (60.3)	23.8	1.50 (1.28 to 1.76)	
Diabetes (mutually adjusted)	1,544 (29.5)	32.8	2.41 (2.07 to 2.80)	
Dyslipidaemia (mutually adjusted)	3,080 (58.9)	20.9	1.37 (1.18 to 1.60)	
Renal impairment (mutually adjusted)	246 (4.7)	36.2	1.39 (1.03 to 1.88)	
HbA1c below 5.7%	1,200 (23.2)	10.9	1 (reference)	
HbA1c 5.7 to 6.4%	2,643 (51.1)	16.3	1.32 (1.07 to 1.64)	1.27
HbA1c 6.5 to 7.9%	967 (18.7)	30.4	2.71 (2.14 to 3.44)	1.68
HbA1c 8.0% or higher	363 (7.0)	39.1	4.41 (3.28 to 5.92)	2.65
eGFR 90 or higher	3,662 (70.1)	15.6	1 (reference)	
eGFR 60 to 89	1,326 (25.4)	27.5	1.16 (0.96 to 1.39)	1.13
eGFR below 60	237 (4.5)	35.0	1.64 (1.18 to 2.27)	
of which 45 to 59	179 (3.4)	35.2	1.62 (1.13 to 2.31)	0.82
of which below 45	58 (1.1)	34.5	1.72 (0.91 to 3.23)	0.83

The four risk factors were entered in one model adjusted for the age spline, sex, pathway and recording period. Laboratory-band models used the same covariates; percentages use patients with the relevant result as denominator (HbA1c 5,173; eGFR 5,225). eGFR below 60 is itself a criterion for renal impairment, so its odds ratio after adding the count has no independent meaning. eGFR in mL/min/1.73 m².

Symptom records

Prevalence was 25.6% in the 1,243 examinations with a recorded symptom of chest pain, chest tightness or angina and 17.6% in those without. Adjusted for age, sex, pathway, recording period and the risk-factor count, the odds ratio for a symptom record was 2.06 (95% CI 1.74 to 2.43). The per-factor odds ratio

pointed the same way with (1.90, 1.63 to 2.21) and without (1.66, 1.51 to 1.83) a symptom record, interaction P = 0.125.

Sensitivity analyses

Including the 2,129 examinations with incomplete pre-test laboratory data (7,354 examinations, unclassifiable factors treated as not recorded), the per-factor odds ratio was 1.66 (95% CI 1.56 to 1.78). In the 5,764 examinations with complete laboratory data including prior revascularisation, it was 1.72 (1.60 to 1.85) with an age by count interaction P < 0.001. Restricted to 2024 to 2025 with adjustment for year (4,741 examinations), it was 1.69 (1.56 to 1.83). All analyses pointed in the same direction.

Discussion

Main findings

In 5,225 first-evaluation CCTA examinations, the number of cardiometabolic risk factors recorded before the scan showed a clear dose-response relationship with obstructive CAD, and that relationship weakened with age. Each additional factor roughly doubled the odds in patients under 55 and raised them by about half at 75 or older. Patients under 55 with no recorded risk factor almost never had obstructive disease (0.4%), whereas one in five of those aged 75 or older with an equally clean record did. HbA1c and eGFR refined the picture. Prevalence was 3.6 times higher with HbA1c of 8.0% or above than below 5.7%, and 2.2 times higher with eGFR below 60 than at 90 or above. The cardiology and general practice pathways served different populations, yet prevalence and the effect of the count were the same.

Comparison with previous studies

The age dependence of the discriminating value of risk factors agrees with evidence from elsewhere. Only 1.8% of symptomatic patients aged 18 to 45 had stenosis of 50% or more (18). Young patients with no risk factors have very low coronary event rates, whereas those with three or more have rates many times higher (19). Our 0.4% prevalence in patients under 55 with no recorded risk factor sits in the same range. The 19.3% seen with three risk factors shows how wide the discriminating span is in younger patients. CONFIRM had already reported that the prognostic meaning of CCTA findings differs by age and sex (16). A recent large registry found obstructive disease to predict events in both younger and older patients (20). Plaque burden in Chinese CCTA patients increases nonlinearly with age (21), which is consistent with the steadily rising prevalence we saw in older patients even at zero recorded risk factors. In 11,234 Chinese CCTA patients the prevalence of obstructive CAD was 16.9%, and current pre-test probability models underestimated it in the young (22); our 19.5% in the first-evaluation population is close.

The prevalence we observed in the general practice pathway is also close to that reported from primary care in Sweden. Among 483 patients referred directly from primary health care centres for chest pain or

dyspnoea, suspected significant stenosis was found in 18% (30). In our general practice pathway it was 18.6% overall and 18.1% in the main analysis population. The Swedish study asked whether general practitioners' pre-test probability estimates matched the CCTA yield. We asked a complementary question: how much the risk factors already in the chart tell the referring physician, and whether that information means the same thing at 45 as at 80.

The size of each factor's association matches earlier reports. In CONFIRM, obstructive CAD was found in 37% of patients with diabetes and 27% without (11); we found 32.8% and 13.9%. Hypertension is associated with the extent and severity of coronary atherosclerosis (10), reduced kidney function independently predicts adverse events in CCTA populations (12), and metabolic phenotypes relate to CAD and events (13). The HbA1c gradient extends these results: prediabetes was already associated with higher prevalence, and poorly controlled diabetes carried far higher risk than well-controlled diabetes. Our position differs from that of pre-test probability models. CONFIRM and the recent clinical likelihood models use symptoms, calcium scores and other inputs and have been externally validated (17, 23). Our question is upstream of those models: how much discrimination the chart alone provides, by age and by laboratory band, before calcium scoring or structured symptom assessment is added. A triage threshold would build on that information rather than replace it. Imaging studies built on CKM staging generally need fuller information (14, 15); a four-item count and two laboratory bands are simpler, but closer to what most hospitals can take from the chart at once.

Why two pathways, and what differed between them

We started from a general practice perspective and used cardiology as the reference pathway for two reasons. General practitioners mostly see multimorbid patients without typical cardiovascular symptoms, and what they can act on is the risk-factor record in the chart. Our question was how much that record tells them about the CCTA result. Cardiology orders the most CCTA at this hospital, its patients are mostly referred for symptoms, and their pre-test probability is higher. If the relationship between risk factors and CCTA findings is the same on a high-probability pathway and a low-probability one, the relationship does not depend on the referral population and pooling is justified.

The referral populations did differ. Cardiology patients more often had a symptom record, were more often scanned to work up atrial fibrillation, valvular disease or heart failure, were older and more often had recorded hypertension. General practice patients were younger, more often asymptomatic and multimorbid, and more often had diabetes and dyslipidaemia. Among patients with a symptom record, prevalence was higher in cardiology than in general practice, consistent with cardiology "chest pain" being more often typical angina. Among patients without a symptom record, prevalence was similar in the two pathways, overall prevalence was similar, and the effect of the count was the same in both pathways and among asymptomatic patients. In other words, the higher pre-test probability of cardiology raised prevalence among its symptomatic referrals but did not change the relationship between risk factors and CCTA findings. This supports pooling, and it also means that general practitioners can read the risk-factor record by the same standard as cardiologists.

Implications for general practice

Four points follow. First, risk-factor review should be read by age. In younger patients, the presence and number of recorded risk factors discriminates strongly: obstructive disease is rare with none and approaches one in five with three. In older patients, one in five has obstructive disease even with no recorded risk factor, and a clean chart should not lower vigilance. Second, laboratory levels separate risk more finely than the presence or absence of a diagnosis. Diabetes was the strongest single factor, and HbA1c of 8.0% or higher carried far higher risk than well-controlled disease. eGFR below 60 was already associated with higher prevalence, whereas 60 to 89 was not. Third, about three in ten patients lacked the laboratory results needed to classify their risk factors within 30 days before CCTA. Their prevalence was similar to that of patients with complete results, so pre-test metabolic and renal assessment has room to improve. Fourth, a recorded symptom of chest pain or angina went with higher prevalence, and symptoms and risk factors should be weighed together. These four points describe what the chart already tells the referring physician. Whether acting on them changes referral decisions or outcomes is the question for a prospective comparison with current practice.

Methodological considerations

Classifying risk factors only from pre-test diagnoses and laboratory results avoided work-up bias, in which the CCTA result feeds back into what gets recorded (40). The price was the exclusion of about three in ten patients for missing pre-test laboratory data. Their prevalence was similar to that of the main analysis population, and including them moved the per-factor odds ratio from 1.70 to 1.66, so this exclusion did not shape the result. The age-modification, laboratory-band and symptom analyses were specified after the primary result and are reported as exploratory. Three features argue that the age interaction is real rather than a product of multiplicity: it was significant both in the main analysis population and in the population including prior revascularisation, the age-group odds ratios declined monotonically, and every age group held more than 1,000 examinations. Excluding prior revascularisation brought the main analysis population closer to the setting in which patients without known CAD are assessed. Because diagnostic codes have limited sensitivity and measured multimorbidity depends on the data source (33, 34), we counted patients who met laboratory thresholds as positive alongside those with a diagnosis. Adjusted prevalence conveys absolute magnitude to clinical readers more readily than odds ratios, with confidence intervals conditional on the covariate distribution of the main analysis population (36).

Limitations

First, the outcome was taken from the formal CCTA report. The classification rule was validated on 150 reports by two physicians reading blind and reaching consensus, with 99% agreement, but the validation used report text alone and did not revisit the images. The stenosis grade on CCTA does not fully agree with invasive angiography and loses specificity in advanced chronic kidney disease, which is inherent to the method (28, 29). Our outcome is the stenosis described on CCTA and was not confirmed by angiography. Second, the low prevalence in young patients without risk factors may partly reflect referral

selection: most of these patients were scanned for symptoms, and the finding cannot be extrapolated to asymptomatic populations. Third, smoking, body mass index, complete medication data and coronary calcium scores were incompletely recorded, and symptoms were captured only as diagnostic records. The four-item count is deliberately restricted to what the chart records before the scan; it is not a full risk score, and confounding by unmeasured factors remains. Fourth, the data come from one hospital and only from the general practice and cardiology pathways, which account for most but not all CCTA there. More than 80% of examinations came through cardiology, all examinations from 2021 to 2023 came through general practice, and recording period therefore partly overlaps with pathway. The process by which data come to be recorded may itself introduce selection bias (35). Fifth, a single eGFR below 60 counted as renal impairment, but without records spanning three months it cannot be equated with chronic kidney disease (39). A single HbA1c of 6.5% or higher counted as diabetes but likewise does not replace a clinical diagnosis. Sixth, the design was cross-sectional without follow-up, and no inference about later events, causation or clinical benefit is possible.

Future research

Radiologists could revisit the original images for a subset of validation reports to assess agreement between report text and images. The age modification and the added value of laboratory grading need testing in an independent population under a pre-specified hypothesis. Their incremental value for pre-test decisions should be assessed alongside symptoms and calcium scores. A prospective study could then test whether presenting the risk-factor record, HbA1c and eGFR by age improves the completeness of risk-factor assessment and the rate of attendance for follow-up.

Conclusions

This study asked whether, in first-evaluation patients undergoing CCTA, the number of cardiometabolic risk factors recorded before the scan shows a dose-response relationship with obstructive CAD, whether age modifies that relationship, whether HbA1c and eGFR levels separate risk further, and whether the cardiology and general practice pathways can be analysed together.

Across 5,225 CCTA examinations we first compared the two pathways using their case mix, prevalence and per-pathway odds ratios, and found different referral populations but similar prevalence and the same effect of the count. In the pooled population we used patient-clustered logistic regression, interaction testing, age stratification, laboratory grading and marginal standardisation, checked against sensitivity analyses that included incomplete laboratory data, prior revascularisation and a restricted recording period. These analyses showed a per-factor odds ratio of about 1.70, with adjusted prevalence rising from about 9% with no risk factor to about 44% with four. The relationship weakened with age: the per-factor odds ratio was 2.20 below 55 years and 1.46 at 75 or older. Prevalence with no recorded risk factor was 0.4% below 55 and 21.2% at 75 or older. HbA1c of 8.0% or higher and eGFR below 60 mL/min/1.73 m² carried adjusted odds ratios of 4.41 and 1.64. Risk-factor review therefore

discriminates most in younger patients, should be weighed against age as the baseline risk in older patients, and should use glycaemic control and eGFR to identify those at highest risk.

Abbreviations

CAD
coronary artery disease
CAD-RADS
Coronary Artery Disease Reporting and Data System
CCTA
coronary computed tomography angiography
CI
confidence interval
CKD-EPI
Chronic Kidney Disease Epidemiology Collaboration
CKM
cardiovascular-kidney-metabolic
CODE-EHR
Core Minimum Standards for Reporting Studies Using Electronic Health Records
eGFR
estimated glomerular filtration rate
HbA1c
glycated haemoglobin
OR
odds ratio
SD
standard deviation
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate

The study was approved by the Clinical Research Ethics Committee of Taizhou People's Hospital (approval number LSKY 2026-146-01, expedited review, 8 September 2026; protocol version 1.0 dated 31 August 2026). The committee waived the requirement for informed consent because the study analysed de-identified retrospective data. The study was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Funding

This study received no specific funding.

Author Contribution

XS conceived the study, served as principal investigator for the ethics application, independently read the validation reports and contributed to interpretation. HG independently read the validation reports, took part in the consensus reading and contributed to interpretation. DL designed the analysis, curated the data, performed the statistical analysis and drafted the manuscript. All authors revised the manuscript and read and approved the final version.

Acknowledgements

Not applicable.

Data Availability

The datasets analysed during the current study are not publicly available because they contain patient-level clinical information held under hospital data governance, but aggregate results, variable definitions and analysis scripts are available from the corresponding author on reasonable request and with the permission of Taizhou People's Hospital.

References

1. GBD 2021 Diseases and Injuries Collaborators. Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: A systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2024;403(10440):2133–61. 10.1016/S0140-6736(. 24)00757-8 PubMed PMID: 38642570.
2. The Writing Committee of the Report on Cardiovascular Health and Diseases in China. Report on cardiovascular health and diseases in China 2023: An updated summary. Biomed Environ Sci. 2024;37(9):949–92. 10.3967/bes2024.162 . PubMed PMID: 39401992.
3. Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain: A report of the American College of Cardiology/American Heart Association Joint Committee on

Clinical Practice Guidelines. Circulation. 2021;144(22):e368–454. 10. 1161/CIR.0000000000001029 PubMed PMID: 34709879.

4. Vrints C, Andreotti F, Koskinas KC, Rossello X, Adamo M, Ainslie J, et al. 2024 ESC guidelines for the management of chronic coronary syndromes. Eur Heart J. 2024;45(36):3415–537. 10.1093/eurheartj/ehae177 . PubMed PMID: 39210710.
5. Cury RC, Leipsic J, Abbara S, Achenbach S, Berman D, Bittencourt M, et al. the American College of Cardiology (ACC), the American College of Radiology (ACR), and the North America Society of Cardiovascular Imaging (NASCI). J Cardiovasc Comput Tomogr. 2022;16(6):536–57. 10.1016/j.jcct.2022.07.002 . PubMed PMID: 35864070. CAD-RADS™ 2.0–2022 Coronary Artery Disease-Reporting and Data System: An Expert Consensus Document of the Society of Cardiovascular Computed Tomography (SCCT).
6. Ndumele CE, Rangaswami J, Chow SL, Neeland IJ, Tuttle KR, Khan SS, et al. Cardiovascular-Kidney-Metabolic Health: A Presidential Advisory From the American Heart Association. Circulation. 2023;148(20):1606–35. 10.1161/cir.0000000000001184 . PubMed PMID: 37807924.
7. Aggarwal R, Ostrominski JW, Vaduganathan M. Prevalence of cardiovascular-kidney-metabolic syndrome stages in US adults, 2011–2020. JAMA. 2024;331(21):1858–60. 10.1001/jama.2024.6892 . PubMed PMID: 38717747.
8. Zheng C, Tian Y, Chen Z, Wang X, Zhang L, Cao X, et al. Prevalence and mortality of cardiovascular-kidney-metabolic syndrome in China: A nationwide population-based study. JACC Asia. 2025;5(7):898–910. 10.1016/j.jacasi.2025.04.007 . PubMed PMID: 40498431.
9. Emerging Risk Factors Collaboration, Di Angelantonio E, Kaptoge S, Wormser D, Willeit P, Butterworth AS, et al. Association of cardiometabolic multimorbidity with mortality. JAMA. 2015;314(1):52–60. 10.1001/jama.2015.7008 . PubMed PMID: 26151266.
10. Nakanishi R, Baskaran L, Gransar H, Budoff MJ, Achenbach S, Al-Mallah M, et al. Relationship of hypertension to coronary atherosclerosis and cardiac events in patients with coronary computed tomographic angiography. Hypertension. 2017;70(2):293–9. 10.1161/HYPERTENSIONAHA.117.09402 . PubMed PMID: 28607128.
11. Rana JS, Dunning A, Achenbach S, Al-Mallah M, Budoff MJ, Cademartiri F, et al. Differences in prevalence, extent, severity, and prognosis of coronary artery disease among patients with and without diabetes undergoing coronary computed tomography angiography: Results from 10,110 individuals from the CONFIRM registry. Diabetes Care. 2012;35(8):1787–94. 10.2337/dc11-. 2403 PubMed PMID: 22699296.
12. Dahl JN, et al. Prognostic impact of reduced kidney function in patients with suspected coronary artery disease undergoing CCTA. Eur J Prev Cardiol. 2025. 10.1093/eurjpc/zwaf. 218 PubMed PMID: 40249751.
13. Kammerlander AA, Mayrhofer T, Ferencik M, Pagidipati NJ, Karady J, Ginsburg GS, et al. Association of metabolic phenotypes with coronary artery disease and cardiovascular events in patients with

stable chest pain. Diabetes Care. 2021;44(4):1038–45. 10.2337/dc20-. 1760 PubMed PMID: 33558267.

14. Xi Y, Xu Y, Shu Z. Impact of hypertension on coronary artery plaques and FFR-CT in type 2 diabetes mellitus patients: Evaluation utilizing artificial intelligence processed coronary computed tomography angiography. Front Artif Intell. 2024;7:1446640. 10.3389/frai.2024.1446640 . PubMed PMID: 39507325.
15. Zhu S, Zhang H, Liu Y, Bu W, Wu Q, Wang J, et al. Development of an optimized risk evaluation system for cardiovascular-kidney-metabolic syndrome-associated coronary heart disease based on tabular prior-data fitted network. Digit Health. 2025;11:20552076251379379. doi:10.1177/20552076251379379 PubMed PMID: 40964606.
16. Min JK, Dunning A, Lin FY, Achenbach S, Al-Mallah M, Budoff MJ, et al. Age- and sex-related differences in all-cause mortality risk based on coronary computed tomography angiography findings: Results from the International Multicenter CONFIRM registry of 23,854 patients without known coronary artery disease. J Am Coll Cardiol. 2011;58(8):849–60. 10.1016/j.jacc.2011.02.074 . PubMed PMID: 21835321.
17. Al'Aref SJ, Maliakal G, Singh G, van Rosendael AR, Ma X, Xu Z, et al. Machine learning of clinical variables and coronary artery calcium scoring for the prediction of obstructive coronary artery disease on coronary computed tomography angiography: Analysis from the CONFIRM registry. Eur Heart J. 2020;41(3):359–67. 10.1093/eurheartj/ehz565 . PubMed PMID: 31513271.
18. Lorenzatti D, et al. Interaction between risk factors, coronary calcium, and CCTA plaque characteristics in patients aged 18–45 years. Eur Heart J Cardiovasc Imaging. 2024;25(8):1071–82. 10.1093/ehjci/jeae094 . PubMed PMID: 38578944.
19. Mortensen MB, et al. Interplay of risk factors and coronary artery calcium for CHD risk in young patients. JACC Cardiovasc Imaging. 2021;14(12):2387–96. 10.1016/j.jcmg.2021.05.003 . PubMed PMID: 34147446.
20. Huck DM, et al. Adverse cardiovascular events among younger and older patients referred for coronary CTA: The Mass General Brigham CCTA Registry. JACC Cardiovasc Imaging. 2026;19(5):604–17. 10.1016/j.jcmg.2025. 12.012 PubMed PMID: 41711627.
21. Yang X, Zhang J, Song Y, Jia D, Hong Q, Zhan Y, et al. Deciphering age- and sex-specific patterns of coronary artery atherosclerosis from a large Chinese cohort. Nat Commun. 2025;16(1):10616. 10.1038/s41467-025-64940-8 . PubMed PMID: 41274881.
22. Zheng Y, et al. Performance of the 2019 ESC pre-test probability model in predicting obstructive coronary artery disease in a Chinese population using coronary computed tomography angiography outcomes. J Cardiovasc Comput Tomogr. 2024;18(4):408–15. 10.1016/j.jcct.2024.04.011 . PubMed PMID: 38702270.
23. Rasmussen LD, Schmidt SE, Knuuti J, Spiro J, Rajwani A, Lopes PM, et al. Clinical likelihood models calibrated against observed obstructive coronary artery disease on computed tomography

angiography. Eur Heart J Cardiovasc Imaging. 2025;26(5):802–13. 10.1093/ehjci/jeaf049 . PubMed PMID: 39918232.

24. Dai Y, Ouyang C, Luo G, Cao Y, Peng J, Gao A, et al. Risk factors for high CAD-RADS scoring in CAD patients revealed by machine learning methods: a retrospective study. PeerJ. 2023;11:e15797. 10.7717/peerj.15797 . PubMed PMID: 37551346.
25. Laggoune J, Nerlekar N, Munnur RK, Ko BS, Cameron JD, Seneviratne S, et al. The utility of coronary computed tomography angiography in elderly patients. J Geriatr Cardiol. 2019;16(7):507–13. 10.11909/j.issn.1671-5411. 2019.07.006 PubMed PMID: 31447889.
26. Bittner DO, Mayrhofer T, Budoff M, Szilveszter B, Foldyna B, Hallett TR, et al. Prognostic value of coronary CTA in stable chest pain: CAD-RADS, CAC, and cardiovascular events in PROMISE. JACC Cardiovasc Imaging. 2020;13(7):1534–45. 10.1016/j.jcmg.2019.09.012 . PubMed PMID: 31734213.
27. Szilveszter B, Vattay B, Bossoussou M, Vecsey-Nagy M, Simon J, Merkely B, et al. CAD-RADS may underestimate coronary plaque progression as detected by serial CT angiography. Eur Heart J Cardiovasc Imaging. 2022;23(11):1530–9. 10.1093/ehjci/jeab215 . PubMed PMID: 34687544.
28. Tekinhatun M, Akbudak I, Özbek M, Turmak M. Comparison of coronary CT angiography and invasive coronary angiography results. Ir J Med Sci. 2024;193(5):2239–48. 10.1007/s11845-024-. 03745-y PubMed PMID: 38965116.
29. Cheng XS, et al. Coronary computed tomography angiography in diagnosing obstructive coronary artery disease in patients with advanced chronic kidney disease: A systematic review and meta-analysis. Cardiorenal Med. 2021;11(1):44–51. doi:10.1159/000510402 PubMed PMID: 33321489.
30. Stertman E, Gabro F, Sandstedt M, Sysoev O, Lauermann J, Östgren CJ, et al. Coronary computed tomography angiography in primary care patients with chest pain or dyspnea: A cross-sectional study. BMC Prim Care. 2025;26(1):178. 10.1186/s12875-025-02877-z . PubMed PMID: 40394490.
31. Kotecha D, Asselbergs FW, Achenbach S, Anker SD, Atar D, Baigent C, et al. CODE-EHR best practice framework for the use of structured electronic healthcare records in clinical research. BMJ. 2022;378:e069048. 10.1136/bmj-2021-069048 . PubMed PMID: 36562446.
32. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Ann Intern Med. 2007;147(8):573–7. 10.7326/0003-4819-147-8-200710160-00010 . PubMed PMID: 17938396.
33. Quan H, Li B, Saunders LD, Parsons GA, Nilsson CI, Alibhai A, et al. Assessing validity of ICD-9-CM and ICD-10 administrative data in recording clinical conditions in a unique dually coded database. Health Serv Res. 2008;43(4):1424–41. 10.1111/j.1475-6773.2007.00822. .x PubMed PMID: 18756617.
34. MacRae C, et al. Impact of data source choice on multimorbidity measurement: A comparison study of 2.3 million individuals in the Welsh National Health Service. BMC Med. 2023;21(1):309. 10.1186/s12916-023-02970-z . PubMed PMID: 37582755.

35. Haneuse S, Daniels M. A general framework for considering selection bias in EHR-based studies: What data are observed and why? EGEMS (Wash DC). 2016;4(1):1203. 10.13063/2327-9214.1203 PubMed PMID: 27668265.
36. Muller CJ, MacLehose RF. Estimating predicted probabilities from logistic regression: different methods correspond to different target populations. Int J Epidemiol. 2014;43(3):962–70. 10.1093/ije/dyu. 029 PubMed PMID: 24603316.
37. Narula J, Chandrashekhar Y, Ahmadi A, Abbara S, Berman DS, Blankstein R, et al. SCCT 2021 expert consensus document on coronary computed tomographic angiography: A report of the Society of Cardiovascular Computed Tomography. J Cardiovasc Comput Tomogr. 2021;15(3):192–217. .001 PubMed PMID: 33303384.
38. Inker LA, Eneanya ND, Coresh J, Tighiouart H, Wang D, Sang Y, et al. New Creatinine- and Cystatin C-Based Equations to Estimate GFR without Race. N Engl J Med. 2021;385(19):1737–49. 10.1056/nejmoa2102953 . PubMed PMID: 34554658.
39. Kidney Disease: Improving Global Outcomes CKD Work Group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease [Internet]. 2024. Available from: https://kdigo.org/wp-content/uploads/2024/03/KDIGO-2024-CKD-Guideline.pdf
40. Ransohoff DF, Feinstein AR. Problems of spectrum and bias in evaluating the efficacy of diagnostic tests. N Engl J Med. 1978;299(17):926–30. 10. 1056/NEJM197810262991705 PubMed PMID: 692598.

Figures

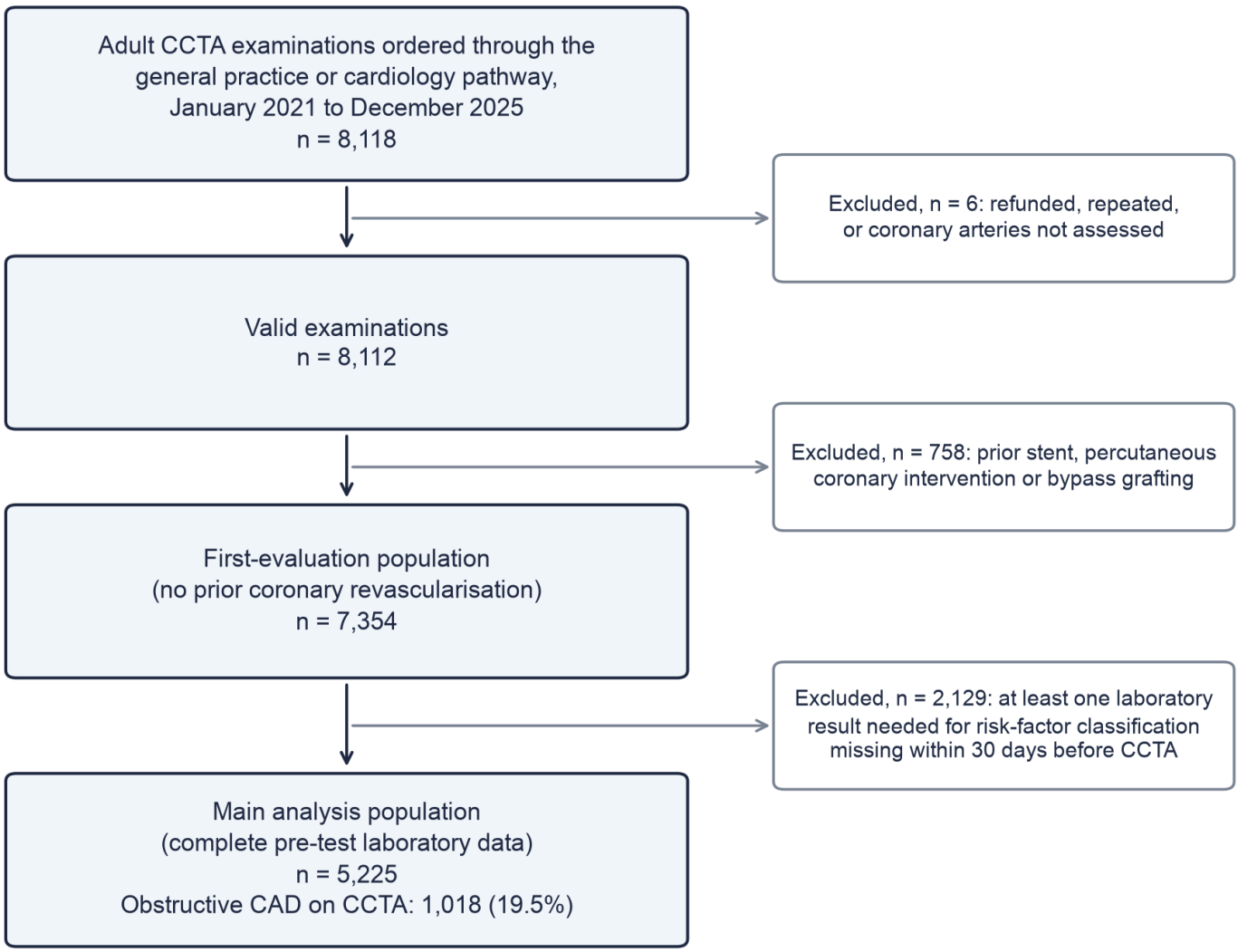


Figure 1

Flow of examinations into the main analysis population. CAD, coronary artery disease; CCTA, coronary computed tomography angiography.

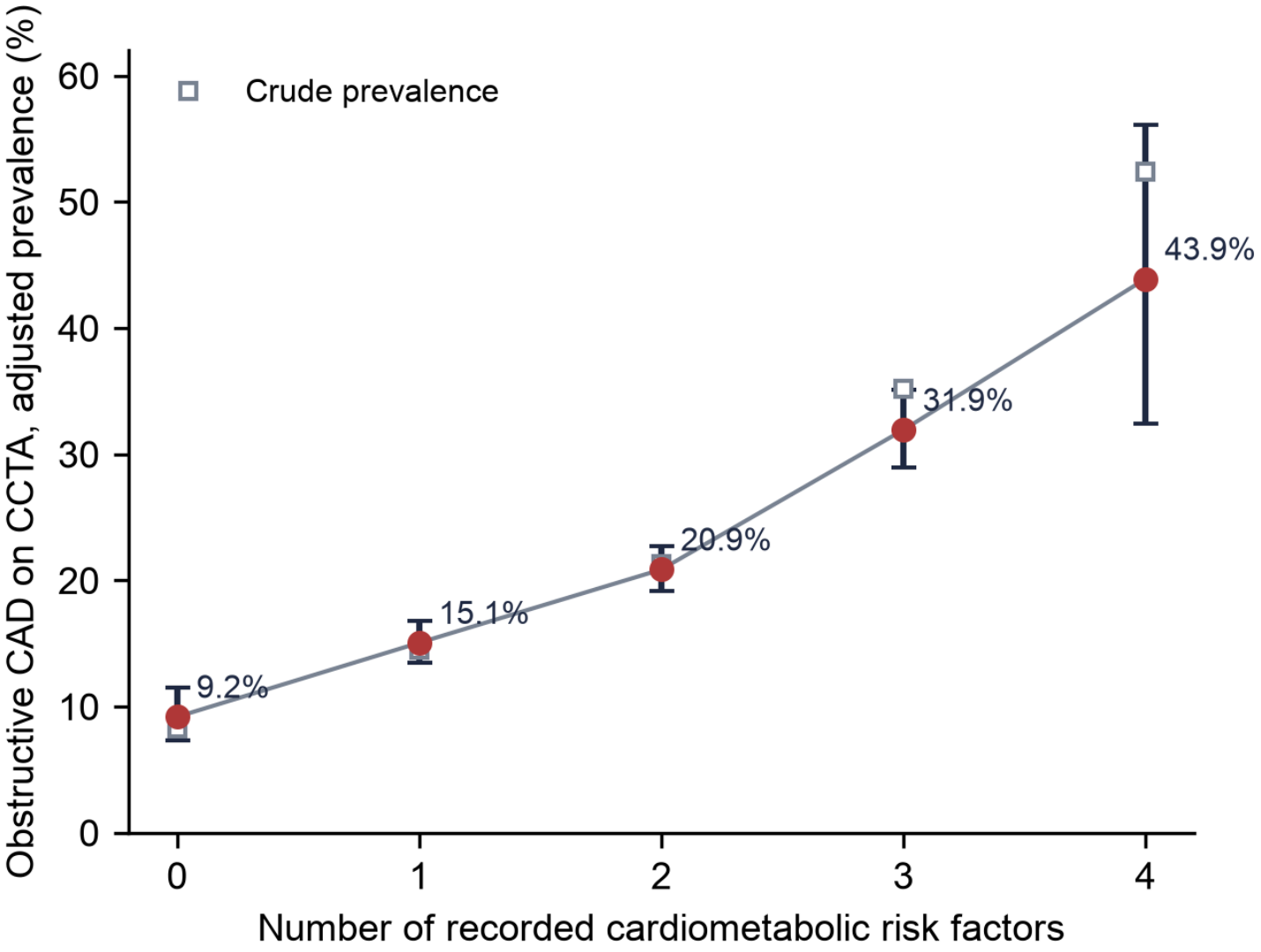


Figure 2

Adjusted prevalence of obstructive CAD on CCTA by number of recorded cardiometabolic risk factors. Circles show adjusted prevalence with 95% confidence intervals from marginal standardisation to the main analysis population; open squares show crude prevalence; the line is a visual guide only.

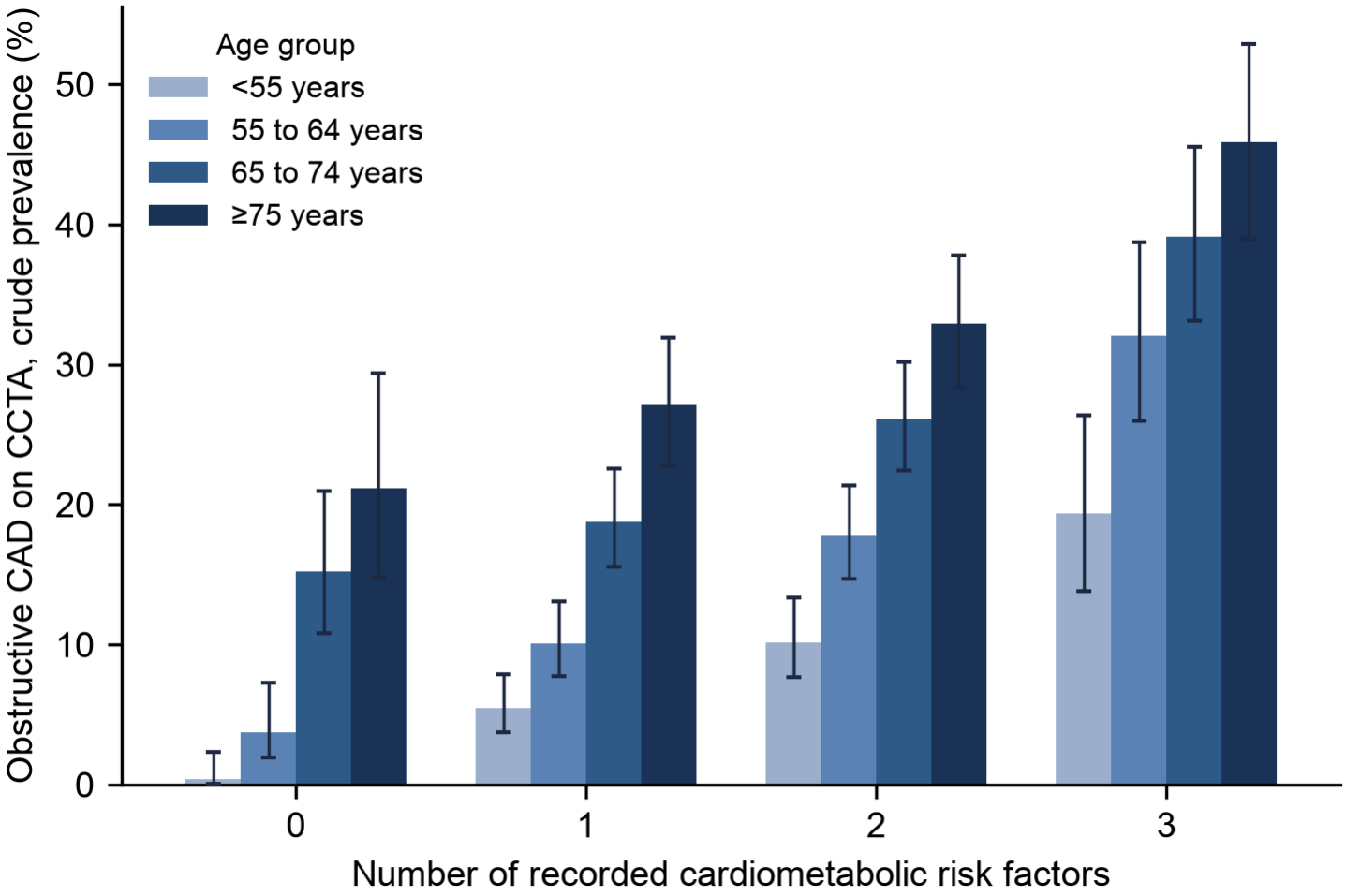


Figure 3

Crude prevalence of obstructive CAD on CCTA by number of recorded cardiometabolic risk factors within age groups. Error bars are Wilson 95% confidence intervals; cells with fewer than 30 examinations are not shown.

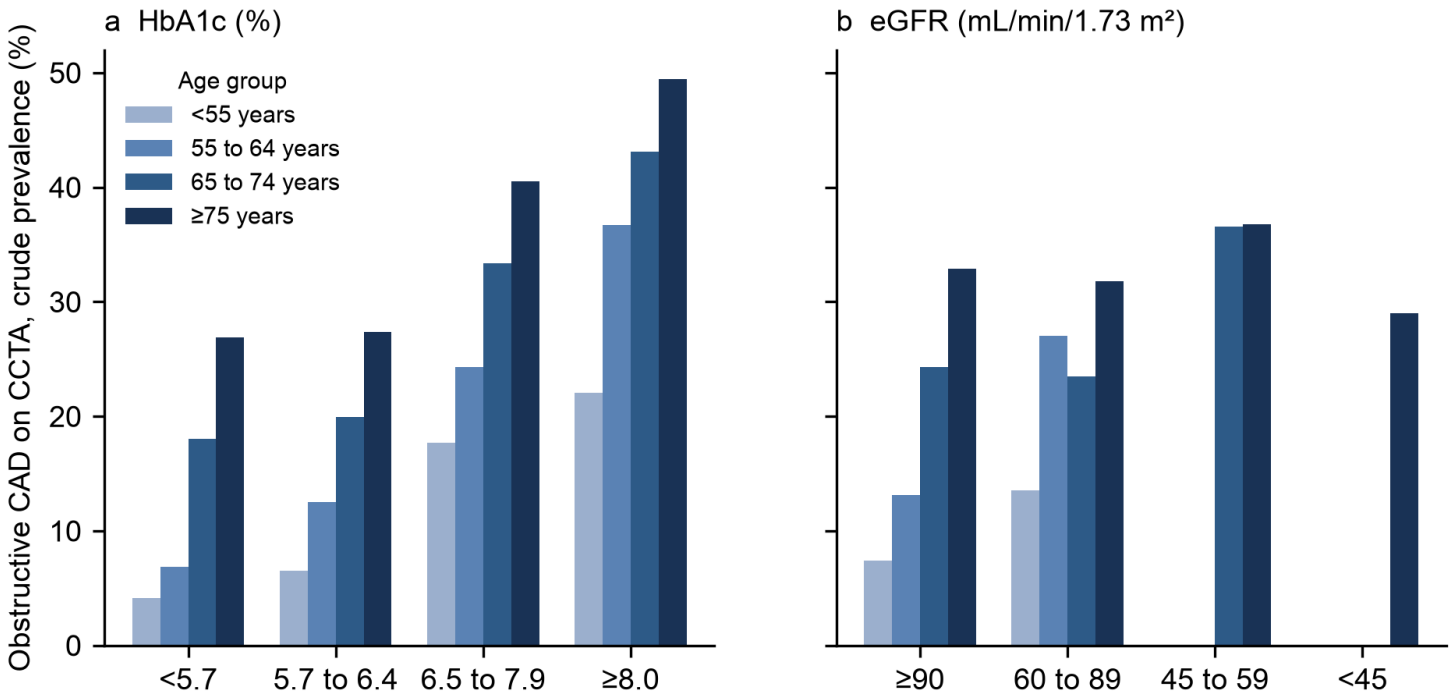


Figure 4

Crude prevalence of obstructive CAD on CCTA by HbA1c band (a) and eGFR band (b) within age groups. Cells with fewer than 30 examinations are not shown. eGFR in mL/min/1.73 m².

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile1Supplementarytables.docx
- Additionalfile2STROBEchecklist.docx