

ORIGINAL ARTICLE

Association between coronary artery calcifications and pre-bronchodilator airflow limitation among individuals with LDCT-detected emphysema: findings from a lung cancer screening program

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Summary

Introduction: Coronary artery calcifications (CAC) and emphysema are common findings detected during low-dose computed tomography (LDCT) lung cancer screening. This study investigated the association between CAC and pulmonary function parameters among individuals with CT-detected emphysema.

Materials and Methods: This cross-sectional study included 323 participants with radiographically detected emphysema identified through a lung cancer screening program. All participants underwent pre-bronchodilator spirometry. We evaluated associations among CAC, pre-bronchodilator airflow limitation, and pulmonary function parameters. We used multivariable logistic regression to identify factors independently associated with CAC.

Results: Coronary artery calcifications were present in 216 participants (66.9%). Participants with CAC were significantly older than those without CAC (68.2 ± 6.7 vs. 61.1 ± 7.1 years, $p < 0.001$) and were more frequently male (59.3% vs. 32.7%, $p < 0.001$). Pre-bronchodilator airflow limitation was significantly more common among participants with CAC (50.0% vs. 35.5%; $p = 0.014$). No significant differences were observed in FEV₁ or FVC values. In multivariable logistic regression analysis, age and male sex were independently associated with CAC, whereas pre-bronchodilator airflow limitation was not independently associated with CAC after adjustment.

Conclusion: Among individuals with CT-detected emphysema, coronary artery calcifications were associated with a higher prevalence of pre-bronchodilator airflow limitation. However, age and male sex were the strongest independent predictors of CAC, suggesting that shared demographic and cardiovascular risk factors may account for much of the observed overlap between pulmonary and cardiovascular abnormalities. These findings support the value of integrated cardiopulmonary assessment within lung cancer screening programs.

Keywords: emphysema, coronary artery calcification, lung cancer screening, spirometry, airflow limitation

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) and cardiovascular disease (CVD) are major contributors to global morbidity and mortality, accounting for a substantial proportion of disability-adjusted life years (DALYs) worldwide (1,2). COPD is currently the third leading cause of death worldwide, whereas CVD remains the leading cause of global mortality. Together, these conditions impose a substantial burden on healthcare systems and public health worldwide.

The coexistence of COPD and cardiovascular disease has been consistently observed across multiple populations. In a systematic review and meta-analysis of 29 datasets, Chen et al. reported that patients with COPD were more than twice as likely to have CVD as matched controls (OR 2.46, 95% CI 2.02–3.00) (3). Moreover, COPD was associated with a two- to fivefold increased risk of ischemic heart disease, cardiac arrhythmias, heart failure, pulmonary circulatory disorders, and peripheral arterial disease (4). This close relationship is largely explained by shared risk factors, particularly cigarette smoking, and by common pathophysiological mechanisms, including systemic inflammation, oxidative stress, endothelial dysfunction, and accelerated vascular aging (5).

The introduction of low-dose computed tomography (LDCT) lung cancer screening (LCS) programs has created a unique opportunity to identify smoking-related abnormalities beyond lung cancer. In addition to pulmonary nodules, LDCT frequently detects emphysema and coronary artery calcifications (CAC), two of the most common non-malignant findings in screening populations. The SUMMIT study, which included more than 11,000 participants undergoing LDCT as part of a Lung Health Check program, reported CAC in 64.2% and emphysema in 33.4% of screened individuals (6). Similarly, a recent scoping review of 32 lung cancer screening studies found CAC prevalence ranging from 14.8% to 98%, highlighting the high burden of subclinical atherosclerotic disease among individuals eligible for screening (7). These observations suggest that lung cancer screening populations are an ideal setting for investigating the interplay between pulmonary and cardiovascular abnormalities.

Coronary artery calcifications are widely recognized as a marker of subclinical atherosclerosis and an established predictor of future cardiovascular events and mortality. In a case-cohort study nested within a randomized lung cancer screening trial, Jacobs et al. demonstrated a progressive increase in all-cause mortality with higher CAC burden, with multivariable-adjusted hazard ratios of 3, 6, and 11 for CAC scores of 1–100, 101–1000, and >1000, respectively (8). Furthermore, a meta-analysis of more than 20,000 participants from lung cancer screening programs found that the presence of CAC was associated with a twofold increase in both cardiovascular and

all-cause mortality (9). These findings underscore the clinical importance of identifying and reporting CAC during LDCT screening.

Although emphysema, airflow limitation, and cardiovascular disease frequently coexist, the relationship between coronary artery calcifications and pulmonary functional impairment remains incompletely understood. It remains unclear whether coronary artery calcifications primarily reflect the severity of pulmonary impairment or represent an independent manifestation of smoking-related systemic disease.

Alongside radiological assessment, spirometry remains the cornerstone for detecting and characterizing airflow limitations in individuals at risk for COPD. Previous studies have shown that a substantial proportion of participants undergoing lung cancer screening have previously unrecognized airflow limitations, often in the setting of CT-detected emphysema. The simultaneous availability of imaging and functional respiratory data offers a unique opportunity to investigate the relationship between coronary artery calcifications and pulmonary functional impairment. Determining whether CAC is associated with airflow limitation or reduced lung function may improve risk stratification in smokers and individuals with smoking-related lung damage.

Therefore, the present study aimed to investigate the association between coronary artery calcifications and pre-bronchodilator spirometric airflow limitation among individuals with CT-detected emphysema who participated in a lung cancer screening program.

MATERIALS AND METHODS

Study Design and Population

The Lung Cancer Screening Program was launched in November 2024 at the Clinic for Pulmonary Diseases of the University Clinical Center of Serbia, with the Ministry of Health involved.

This cross-sectional study was conducted within an LCS program for individuals aged 50 years or older with a smoking history of more than 30 years who underwent LDCT screening. Between November 2024 and May 2025, we screened 3,076 individuals. Of these, 323 participants with radiologically detected emphysema were contacted by phone to undergo spirometry and were subsequently classified based on the presence or absence of coronary artery calcifications on LDCT. All invited participants completed a pre-bronchodilator spirometric assessment to evaluate pre-bronchodilator airflow limitation. Spirometry exclusion criteria included a prior diagnosis of chronic respiratory disease.

Assessment of Emphysema and Coronary Artery Calcifications

Experienced thoracic radiologists independently interpreted LDCT examinations on a Siemens Definition Edge 128-slice CT scanner using a standardized low-dose acquisition protocol (20 mA, 100 kV). Scans were obtained during a single full-inspiration breath-hold and reconstructed with a 1-mm slice thickness using the BF37 lung kernel. All image acquisition and reconstruction parameters followed the standardized protocol used in the national lung cancer screening program. Radiological emphysema was identified using the conventional densitometric threshold of -950 Hounsfield units on inspiratory CT images, a threshold widely validated for assessing emphysematous lung destruction (10,11). For the present analysis, emphysema was categorized as present or absent. Coronary artery calcifications were evaluated visually on LDCT images and likewise categorized as present or absent.

Pulmonary Function Testing

Pre-bronchodilator spirometry was performed to characterize respiratory impairment in the lung cancer screening cohort rather than to establish a definitive diagnosis of COPD. It was conducted using a Vyntus SPIRO system (Vyaire Medical, Germany) with SentrySuite software, following the recommendations of the European Respiratory Society and the American Thoracic Society (ERS/ATS) (12,13). Measured spirometric variables included: Forced Expiratory Volume in One Second (FEV_1), Forced Vital Capacity (FVC), and FEV_1/FVC ratio. Predicted FEV_1 values were used to describe the severity of airflow limitation, and pre-bronchodilator airflow limitation was defined as an FEV_1/FVC ratio below 0.70 (14).

Statistical methods, presentation, and interpretation of results

Statistical analyses were conducted using IBM SPSS Statistics version 29. Continuous variables are presented as mean $\pm$ standard deviation (SD), and categorical variables are presented as absolute numbers and percentages.

Comparisons between participants with and without CAC were performed using the independent-samples t-test for continuous variables and the chi-square test for categorical variables. Associations between CAC and pre-bronchodilator airflow limitation were evaluated using Pearson's chi-square test.

To identify factors independently associated with CAC, a multivariable binary logistic regression was conducted. CAC presence was used as the dependent variable, and age, sex, and pre-bronchodilator airflow limitation were included as independent variables. Results are presented as odds ratios (ORs) with corresponding 95%

confidence intervals (CIs). Statistical significance was set at $p < 0.05$. All analyses were performed using IBM SPSS Statistics version 29.

RESULTS

A total of 323 individuals with LDCT-detected emphysema were included in the analysis. Coronary artery calcifications were present in 216 participants (66.9%), while 107 participants (33.1%) had no evidence of CAC on low-dose computed tomography, as shown in **Figure 1**.

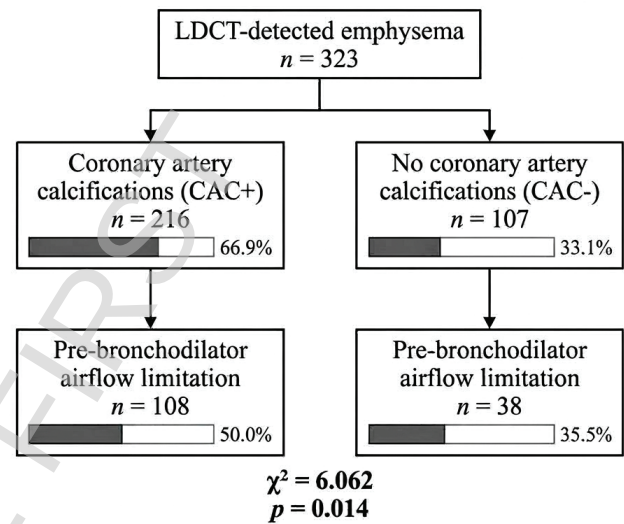


Figure 1. Association between coronary artery calcifications and pre-bronchodilator airflow limitation in individuals with LDCT-detected emphysema.

Among 323 participants with LDCT-detected emphysema, coronary artery calcifications (CAC) were identified in 216 (66.9%) individuals. Pre-bronchodilator airflow limitation was present in 108 of 216 participants (50.0%) with CAC compared with 38 of 107 participants (35.5%) without CAC. The prevalence of airflow limitation was significantly higher among participants with CAC (Pearson $\chi^2 = 6.062$, $p = 0.014$). Abbreviations: CAC, coronary artery calcifications; LDCT, low-dose computed tomography.

Baseline characteristics according to CAC status are presented in **Table 1**.

Table 1. Baseline Characteristics According to Coronary Artery Calcification Status

	CAC present (n=216)	CAC absent (n=107)	p value
Age, years	68.2 $\pm$ 6.7	61.1 $\pm$ 7.1	<0.001
Male sex, n (%)	128 (59.3)	35 (32.7)	<0.001
BMI, kg/m ²	26.3 $\pm$ 4.5	25.8 $\pm$ 4.3	0.309

Data are presented as mean $\pm$ standard deviation or n (%). Abbreviations: CAC, coronary artery calcifications; BMI, body mass index.

Table 2. Pulmonary Function According to Coronary Artery Calcification Status

	CAC present (n=216)	CAC absent (n=107)	p value
Pre-bronchodilator airflow limitation, n (%)	108 (50.0)	38 (35.5)	0.014
No airflow limitation, n (%)	108 (50.0)	69 (64.5)	
FEV ₁ (% predicted)	98.5 ± 19.9	99.7 ± 16.2	0.580
FVC (% predicted)	114.2 ± 18.1	115.0 ± 15.3	0.704

Data are presented as mean ± standard deviation or n (%). Abbreviations: CAC, coronary artery calcifications; FEV₁, forced expiratory volume in one second; FVC, forced vital capacity.

Participants with CAC were significantly older than those without CAC (68.2 ± 6.7 vs. 61.1 ± 7.1 years, $p < 0.001$) and more frequently male (59.3% vs. 32.7%, $p < 0.001$). BMI did not differ significantly between groups (26.3 ± 4.5 vs. 25.8 ± 4.3 kg/m², $p = 0.309$).

Pulmonary function characteristics according to CAC status are presented in **Table 2**.

Pre-bronchodilator airflow limitation was more common among participants with CAC than among those without CAC (50.0% vs. 35.5%, $p = 0.014$). No significant differences were observed in FEV₁ (98.5 ± 19.9% vs. 99.7 ± 16.2%, $p = 0.580$) or FVC (114.2 ± 18.1% vs. 115.0 ± 15.3%, $p = 0.704$).

Table 3 presents the multivariable logistic regression analysis of factors associated with CAC.

Table 3. Factors Independently Associated with Coronary Artery Calcification

	OR	95% CI	p value
Male sex	2.503	1.452–4.314	0.001
Age	1.152	1.106–1.199	<0.001
Pre-bronchodilator airflow limitation	1.517	0.881–2.613	0.133

Model performance: Omnibus $\chi^2 = 82.94$, $p < 0.001$, Nagelkerke $R^2 = 0.315$, Classification accuracy = 75.2%. Abbreviations: OR, odds ratio; CI, confidence interval;

Multivariable logistic regression analysis showed that age and male sex were independently associated with CAC. Each additional year of age increased the odds of CAC by approximately 15% (OR 1.152, 95% CI 1.106–1.199; $p < 0.001$). Male participants had 2.5-fold higher odds of CAC than female participants (OR 2.503, 95% CI 1.452–4.314; $p = 0.001$). Although pre-bronchodilator airflow limitation was associated with higher odds of CAC (OR 1.517, 95% CI 0.881–2.613), the association did not reach statistical significance after adjustment ($p = 0.133$).

DISCUSSION

The present study investigated the association between coronary artery calcifications and spirometric pulmonary function parameters among individuals with LDCT-detected emphysema enrolled in a lung cancer screening program. The principal finding was that participants with CAC had a significantly higher prevalence of pre-bronchodilator airflow limitation than those

without CAC. These findings support a close relationship between smoking-related pulmonary abnormalities and subclinical coronary atherosclerosis in individuals undergoing lung cancer screening.

Our findings are broadly consistent with previous studies showing an association between airflow obstruction and coronary atherosclerosis. Chae et al. reported an independent association between coronary calcium burden and lower FEV₁/FVC ratios after adjusting for age, body mass index, and smoking exposure (15). Similarly, Kim et al., using coronary CT angiography in 1,888 asymptomatic individuals, demonstrated that airflow limitation was associated with higher coronary calcium scores, greater atherosclerotic burden, and a higher prevalence of obstructive coronary artery disease, even after adjusting for traditional cardiovascular risk factors (16). Furthermore, Rasmussen et al. showed that COPD was independently associated with higher coronary artery calcification risk categories among long-term smokers in the Danish Lung Cancer Screening Trial (17). Together, these findings support the concept that pulmonary and cardiovascular abnormalities frequently coexist in smoking-exposed populations.

Although coronary artery calcifications were associated with pre-bronchodilator airflow limitation in unadjusted analyses, this association was attenuated after adjustment for age and sex. These findings suggest that the coexistence of coronary artery calcifications and airflow limitation may be largely explained by shared demographic and cardiovascular risk factors. Cigarette smoking, aging, systemic inflammation, oxidative stress, endothelial dysfunction, and accelerated vascular aging are recognized contributors to both atherosclerosis and chronic airway disease. The high cumulative tobacco exposure characteristic of the study population may have further contributed to the coexistence of pulmonary and cardiovascular abnormalities. Supporting this interpretation, Gudmundsson et al. demonstrated that airflow obstruction remained associated with atherosclerosis burden independently of traditional cardiovascular risk factors and inflammatory markers (18). In contrast, Sheen et al. reported an association between reduced FEV₁/FVC ratios and increased vascular stiffness in both smokers and non-smokers (19). Interestingly, we observed no significant differences in FEV₁ between participants with and without CAC. This finding suggests that coronary artery calcifications may be more closely related to

airflow obstruction than to the degree of pulmonary impairment measured by FEV₁. Similar observations have been reported previously, as increased cardiovascular risk has been described across COPD severity categories without a clear dose–response relationship between spirometric impairment and coronary calcification burden (16,17). These findings support the concept that cardiovascular risk may already be elevated in individuals with relatively preserved lung function. Together, these observations suggest that pulmonary and cardiovascular manifestations may arise from a shared biological substrate rather than representing completely independent disease processes. In our cohort, age and male sex emerged as the strongest independent predictors of CAC, consistent with established epidemiological patterns of coronary atherosclerosis.

The clinical implications of these findings may extend beyond the respiratory system. Williams et al. reported significantly higher coronary artery calcium (CAC) scores in COPD patients than in smokers with normal spirometry in the ECLIPSE cohort, and CAC also predicted subsequent mortality (20). Similarly, Mota et al. reported that COPD was associated with more severe and more extensively calcified coronary lesions (21). At the same time, Zagaceta et al. identified CAC as one of the strongest non-invasive predictors of future cardiovascular events in COPD (22). Taken together, these observations support routine evaluation and reporting of CAC during lung cancer screening as part of an integrated cardiopulmonary risk assessment strategy.

Several limitations should be acknowledged. First, the cross-sectional design precludes causal conclusions. Second, CAC was assessed visually on LDCT scans rather than by quantitative Agatston scoring, preventing evaluation of dose–response relationships. Third, spirometry was based on pre-bronchodilator measurements, reflecting the study's screening-oriented design rather than a formal COPD diagnosis. Information on current smoking status and cumulative smoking exposure was not available for analysis. Although all participants met the screening eligibility criterion of a smoking history exceeding 30 years, the potential contribution of smoking burden to the observed associations could not be directly assessed. Finally, the study was conducted within a single lung cancer screening cohort, which may limit generalizability.

CONCLUSIONS

Among individuals with CT-detected emphysema participating in a lung cancer screening program, coronary artery calcifications were associated with a higher prevalence of pre-bronchodilator airflow limitation. However, this association was attenuated after adjustment for age and sex, suggesting that shared risk factors may account for much of the observed overlap between pulmonary and cardiovascular abnormalities. These findings further support the value of integrated cardiopulmonary assessment within lung cancer screening programs.

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Conflicts of Interest: S. Dimic-Janjic reports receiving honoraria for lectures from AstraZeneca, Berlin-Chemie Menarini, Boehringer Ingelheim, Takeda, and Providens (Chiesi), and serves as President of the Respiratory Society of Serbia. The remaining authors declare no conflicts of interest.

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Ethical Approval: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the University Clinical Center of Serbia (approval number: 1048/1, dated 13/5/2025).

Informed Consent: Informed consent was obtained from all study participants.

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POVEZANOST KALCIFIKACIJA KORONARNIH ARTERIJA I EMFIZEMA OTKRIVENIH NISKODOZNIM CT PREGLEDOM SA PRE-BRONHODILACIONOM OPSTRUKCIJOM DISAJNIH PUTEVA KOD ISPITANIKA IZ PROGRAMA SKRININGA NA KARCINOM PLUĆA

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Sažetak

Uvod: Prisustvo kalcifikacija koronarnih arterija (KKA) i emfizema predstavlja čest nalaz tokom skrininga karcinoma pluća niskodoznom kompjuterizovanom tomografijom (NDCT). Cilj ovog istraživanja bio je da ispita povezanost između KKA i pokazatelja plućne funkcije kod osoba sa CT-detekovanim emfizemom.

Metode: U ovu studiju preseka uključena su 323 ispitanika sa radiološki potvrđenim emfizemom identifikovana u programu skrininga karcinoma pluća. Svim ispitanicima učinjena je pre-bronhodilataciona spirometrija. Analizirana je povezanost između prisustva KKA i pre-bronhodilatacionih spirometrijskih pokazatelja plućne funkcije. Za procenu faktora nezavisno povezanih sa KKA korišćena je multivarijantna logistička regresija.

Rezultati: KKA su bile prisutne kod 216 (66,9%) ispitanika. Ispitanici sa KKA bili su stariji (68.2 ± 6.7 vs. 61.1 ± 7.1 godina, $p < 0.001$) i češće muškog pola (59.3% vs. 32.7%, $p < 0.001$). Pre-bronhodilataciona opstrukcija bila je zna-

čajno češća kod ispitanika sa KKA nego kod ispitanika bez KKA (50,0% prema 35,5%; $p = 0,014$). Nisu uočene značajne razlike u vrednostima forsiranog ekspiratornog volumena u prvoj sekundi (FEV₁) i forsiranog vitalnog kapaciteta (FVC). U multivarijantnoj analizi starost i muški pol bili su nezavisno povezani sa prisustvom KKA, dok pre-bronhodilataciona opstrukcija nije ostala nezavisno povezana sa KKA nakon korekcije.

Zaključak: Kod osoba sa NDCT-detekovanim emfizemom kalcifikacije koronarnih arterija povezane su sa većom učestalošću pre-bronhodilatacione opstrukcije. Međutim, starost i muški pol predstavljali su najznačajnije nezavisne prediktore prisustva KKA, što ukazuje da zajednički faktori rizika mogu objasniti značajan deo preklapanja između plućnih i kardiovaskularnih abnormalnosti. Ovi nalazi potvrđuju značaj integrisane kardiopulmonalne procene u okviru programa skrininga karcinoma pluća.

Gljučne reči: emfizem, koronarne arterijske kalcifikacije, skrining karcinoma pluća, spirometrija, bronho-opstrukcija

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