

Clinical, SMuRF-Related, and Angiographic Predictors of Long-Term All- Cause Mortality in Patients Aged ≤ 40 Years with Acute Coronary Syndrome: A Retrospective Cohort Study

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Abstract

Background Acute coronary syndrome (ACS) in adults aged ≤ 40 years is uncommon, and evidence on long-term mortality predictors in this age group remains limited. We evaluated clinical, angiographic, and standard modifiable cardiovascular risk factor (SMuRF)-related predictors of all-cause mortality in a large cohort of very young patients with ACS.

Methods This retrospective cohort study included 903 consecutive patients aged ≤ 40 years who were treated for ACS at a tertiary center between January 2011 and June 2024. ACS presentation, cardiovascular risk factors, SMuRF status, coronary angiographic findings, and mortality were assessed. SMuRFs were defined as hypertension, diabetes mellitus, hyperlipidemia, and current smoking, while obesity was analyzed separately. Survival was evaluated using Kaplan–Meier analysis and Cox proportional hazards regression. Sensitivity analyses included administrative censoring at 10 years and a parsimonious Cox model.

Results The cohort included 528 patients with ST-segment elevation myocardial infarction, 293 with non-ST-segment elevation myocardial infarction, and 82 with unstable angina. During a median follow-up of 4.93 years [95% confidence interval (CI) 4.69–5.18], 51 deaths occurred. Obesity was present in 180 patients (19.9%), left anterior descending artery (LAD) involvement in 436 (48.3%), and multivessel disease in 190 (21.0%). In the multivariable Cox model, obesity [adjusted hazard ratio (HR) 2.677, 95% CI 1.430–5.010; $p = 0.002$] and LAD involvement [adjusted HR 3.586, 95% CI 1.849–6.956; $p < 0.001$] were independently associated with mortality. SMuRF-less patients comprised 10.6% of the cohort and had higher proportions of female sex, obesity, LAD involvement, and multivessel disease than SMuRF-positive patients, although mortality did not differ significantly between the groups. Kaplan–Meier analysis showed lower survival among patients with obesity and among those with LAD involvement. Mortality occurred in 6.3% of SMuRF-less and 5.6% of SMuRF-positive patients. The 10-year sensitivity analysis supported the main findings.

Conclusions In patients aged ≤ 40 years with ACS, obesity and LAD involvement were independently associated with long-term all-cause mortality, whereas SMuRF status was not. These findings support risk assessment that considers cardiometabolic burden and coronary anatomy alongside conventional risk-factor status in very young patients with ACS.

Introduction

Cardiovascular disease remains the leading cause of mortality worldwide, and acute coronary syndrome (ACS) continues to account for a substantial proportion of premature morbidity, mortality, and health care use [1, 2]. Although ACS is traditionally considered a disease of middle-aged and older adults, events occurring in early adulthood have become an increasingly important clinical and public health concern. Kraler et al. emphasized that contemporary ACS should not be interpreted solely through electrocardiographic and biomarker categories because its mechanisms vary according to age, sex, coronary anatomy, and individual risk-factor profiles [2]. This perspective is particularly relevant in young

adults, for whom an ACS event may lead to decades of secondary prevention, recurrent risk, psychosocial burden, and loss of productive life years [3, 4].

The definition of “young” ACS varies across studies, with upper age thresholds commonly ranging from 40 to 55 years [3, 5, 6]. However, patients aged ≤ 40 years represent a particularly informative subgroup because they may have had shorter cumulative exposure to conventional risk factors and may show different clinical, angiographic, and prognostic patterns from older patients. In young ACS cohorts, ST-segment elevation myocardial infarction (STEMI) is frequently the predominant presentation, single-vessel disease is common, and the left anterior descending artery (LAD) is often involved [5–7]. Freedman et al. showed in the CARDIA cohort that sex differences in cardiovascular risk emerge by early midlife, with coronary heart disease occurring earlier in men than in women [4]. These observations suggest that ACS in very young adults should be evaluated as a distinct clinical entity rather than simply as an earlier presentation of disease typically seen later in life.

Traditional risk factors remain central to premature ACS, but their prognostic significance in very young patients is still incompletely defined. Smoking, hypertension, diabetes mellitus, hyperlipidemia, obesity, and family history have all been associated with early coronary disease, although their distribution varies across populations and between sexes [3, 6]. Simonsson et al. recently found in a nationwide Swedish cohort that three in four young patients with acute myocardial infarction had at least one recognized risk factor, with obesity becoming the most common and increasingly prevalent risk factor over time [8]. Obesity may be particularly relevant in young adults because it can reflect prolonged cardiometabolic exposure before hypertension, diabetes, or dyslipidemia becomes clinically apparent [8, 9].

Another unresolved issue is ACS among patients without standard modifiable cardiovascular risk factors (SMuRFs), commonly defined by the absence of hypertension, diabetes mellitus, hyperlipidemia, and current smoking [10, 11]. These patients may not be identified as high risk by conventional prevention strategies because they do not fit traditional risk profiles before the index event. Figtree et al. argued that SMuRF-less coronary disease challenges existing prevention models and may require greater attention to unmeasured or underrecognized risk pathways [10]. However, studies of SMuRF-less ACS have reported inconsistent outcomes, suggesting that this group may represent a heterogeneous phenotype rather than a uniformly high- or low-risk population [11, 12].

ACS in young adults is also mechanistically heterogeneous. Nonatherothrombotic mechanisms, including spontaneous coronary artery dissection (SCAD), coronary embolism, vasospasm, and myocardial infarction with nonobstructive coronary arteries, are increasingly recognized in younger patients, particularly women. Al-Gully et al. demonstrated that SCAD is frequently underdiagnosed in young women presenting with ACS, with 41% of cases not correctly identified at the initial presentation, often leading to inappropriate treatment [13]. In addition, angina with nonobstructive coronary arteries, ischemia with nonobstructive coronary arteries, and myocardial infarction with nonobstructive coronary arteries require diagnostic approaches that extend beyond the identification of obstructive epicardial stenosis alone [14]. These considerations support a broader assessment of young ACS that integrates

conventional risk factors, coronary anatomy, sex-related patterns, nonatherothrombotic mechanisms, and long-term outcomes.

Despite the expanding literature, important gaps remain. Many studies have focused on in-hospital or short-term outcomes, whereas long-term mortality data in patients aged ≤ 40 years remain limited. Angiographic predictors are often reported descriptively but are less frequently incorporated into multivariable survival models. Moreover, the relationships among SMuRF status, coronary involvement, ACS presentation type, and long-term mortality remain insufficiently characterized in very young cohorts. Therefore, this study aimed to evaluate clinical, angiographic, and SMuRF-related predictors of long-term all-cause mortality in patients aged ≤ 40 years with ACS. We also examined factors associated with ACS presentation and performed sensitivity analyses to assess the robustness of the mortality findings.

Methods

Study Design and Population

This retrospective cohort study was conducted at Süleyman Demirel University Research and Practice Hospital, a tertiary center in Türkiye. Between January 2011 and June 2024, 27,647 patients aged 12–91 years were screened. Among them, 928 patients aged ≤ 40 years with acute coronary syndrome (ACS) were identified. ACS comprised ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA), diagnosed from clinical presentation, electrocardiographic findings, cardiac biomarkers, and coronary angiography.

Patients were excluded when the final diagnosis was inconsistent with ACS, myocardial injury was due to nonischemic or secondary causes, or baseline, angiographic, follow-up, or outcome data were unavailable. Twenty-five patients were excluded, leaving 903 consecutive patients in the analytical cohort. Vital status and follow-up information were obtained from electronic records and follow-up files. During follow-up, 51 patients died, and 852 patients without documented death were censored at last follow-up. Consecutive screening, predefined eligibility criteria, standardized definitions, and data extraction were used to reduce selection and information bias.

Data Collection and Definitions

Demographic, clinical, laboratory, electrocardiographic, echocardiographic, angiographic, and follow-up data were extracted from electronic medical records, discharge summaries, laboratory databases, echocardiography reports, catheterization reports, and follow-up files. Risk factors were hypertension, diabetes mellitus, hyperlipidemia, current smoking, obesity, alcohol use, substance use, and family history of premature cardiovascular disease. Obesity was defined as body mass index ≥ 30 kg/m². Hypertension, diabetes mellitus, and hyperlipidemia were recorded from prior diagnosis, treatment, or hospital confirmation. Smoking was defined as current smoking at presentation, and family history as premature cardiovascular disease in a first-degree relative.

Standard modifiable cardiovascular risk factors (SMuRFs) were hypertension, diabetes mellitus, hyperlipidemia, and current smoking. Patients with none were classified as SMuRF-less, and those with at least one as SMuRF-positive. Obesity, family history, alcohol use, and substance use were analyzed separately.

ACS presentation was classified as STEMI, NSTEMI, or UA. Involvement of the left anterior descending artery (LAD), circumflex artery (CX), and right coronary artery (RCA) was recorded. Coronary involvement was categorized as no vessel, single-vessel, double-vessel, or triple-vessel disease. Multivessel disease was defined as involvement of at least two major epicardial coronary arteries.

Exploratory nonobstructive and mechanistic classifications were assigned descriptively from clinical records, angiographic reports, electrocardiographic findings, and biomarker data. Angina with nonobstructive coronary arteries (ANOCA) indicated angina or equivalent symptoms without obstructive epicardial disease or biomarker evidence of infarction. Ischemia with nonobstructive coronary arteries (INOCA) required objective ischemia without obstructive epicardial disease or infarction. Myocardial infarction with nonobstructive coronary arteries (MINOCA) indicated infarction without obstructive epicardial disease. Nonobstructive arteries were defined as <50% stenosis in major epicardial vessels. Isolated slow coronary flow was not classified as INOCA without documented ischemia. Spontaneous coronary artery dissection (SCAD) was identified from angiographic reports and, when MINOCA criteria were met, reported as a MINOCA subgroup. Because these conditions were uncommon, they were described only and were not entered into multivariable survival models.

The primary outcome was all-cause mortality. Follow-up extended from the index ACS date to death or the last available follow-up. Patients alive at the last assessment were censored, and median follow-up was estimated by the reverse Kaplan–Meier method.

Statistical Analysis

The minimum required sample size was estimated using G*Power. The calculation used a linear multiple regression fixed model with an R^2 increase, nine total predictors, and one tested predictor. Assuming a medium effect size ($f^2 = 0.15$), $\alpha = 0.05$, and 80% power, the required total sample size was 55. The final cohort of 903 patients exceeded this requirement. Continuous variables were summarized as mean $\pm$ standard deviation or median with interquartile range, according to distribution. Categorical variables were reported as frequencies and percentages. Age was compared using the Mann–Whitney U test because it was non-normally distributed. Categorical variables were compared using Pearson's chi-square or Fisher's exact test, as appropriate, and omnibus p values were reported for variables with more than two categories. Missing data were not imputed; each analysis used available complete data.

Survival was evaluated using Kaplan–Meier curves and log-rank tests. Cox proportional hazards regression was used to assess mortality predictors, with hazard ratios, 95% confidence intervals, and p values reported. The proportional hazards assumption was assessed using Schoenfeld residuals. Sensitivity analyses applied administrative censoring at 10 years and a parsimonious Cox model

containing age, sex, obesity, and LAD involvement. Multinomial logistic regression evaluated factors associated with ACS presentation, with UA as the reference category. Tests were two-sided, and $p < 0.05$ indicated statistical significance. Analyses used IBM SPSS Statistics version 30.0.0, R version 4.4.2, and G*Power version 3.1.9.7.

Ethical Approval

The study was approved by the Süleyman Demirel University School of Medicine Ethics Committee (approval date: November 5, 2024; decision no. 70). The requirement for informed consent was waived because anonymized retrospective data were used. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Results

Study Population and Baseline Characteristics

The final analytical cohort included 903 patients aged ≤ 40 years with ACS. During follow-up, 51 deaths occurred, and 852 patients were censored. The median follow-up duration, estimated using the reverse Kaplan-Meier method, was 4.93 years [95% CI: 4.69–5.18]. Baseline demographic, clinical, and risk-factor characteristics according to mortality status are presented in Table 1.

STEMI was the most frequent presentation, occurring in 528 patients (58.5%), followed by NSTEMI in 293 patients (32.4%) and UA in 82 patients (9.1%). Obesity was present in 180 patients (19.9%). Overall all-cause mortality during follow-up was 5.6% (51/903). Among deceased patients, STEMI accounted for 36 deaths (70.6%). LAD involvement was more frequent among deceased patients than among censored patients (76.5% vs 46.6%; $p < 0.001$), as was obesity (35.3% vs 19.0%; $p = 0.005$). Smoking was less frequent among deceased patients than among censored patients (58.8% vs 69.4%; $p = 0.115$).

Table 1 Baseline Characteristics According to Mortality Status

Variable	Overall (n = 903)	Censored (n = 852)	Event/death (n = 51)	p
Age, years	36.0 [32.0–39.0]	36.0 [32.0–39.0]	36.0 [31.0–38.0]	0.313*
Male sex	750 (83.1%)	707 (83.0%)	43 (84.3%)	0.805
Alcohol use	6 (0.7%)	4 (0.5%)	2 (3.9%)	0.041†
Hypertension	180 (19.9%)	165 (19.4%)	15 (29.4%)	0.081
Hyperlipidemia	208 (23.0%)	193 (22.7%)	15 (29.4%)	0.265
Diabetes mellitus	105 (11.6%)	98 (11.5%)	7 (13.7%)	0.630
Family history	333 (36.9%)	310 (36.4%)	23 (45.1%)	0.210
Smoking	621 (68.8%)	591 (69.4%)	30 (58.8%)	0.115
Obesity	180 (19.9%)	162 (19.0%)	18 (35.3%)	0.005
Substance use	5 (0.6%)	3 (0.4%)	2 (3.9%)	0.028†
SMuRF status				0.787
SMuRF-less	96 (10.6%)	90 (10.6%)	6 (11.8%)	
SMuRF-positive	807 (89.4%)	762 (89.4%)	45 (88.2%)	
ACS type				0.193
STEMI	528 (58.5%)	492 (57.7%)	36 (70.6%)	
NSTEMI	293 (32.4%)	281 (33.0%)	12 (23.5%)	
UA	82 (9.1%)	79 (9.3%)	3 (5.9%)	
LAD involvement	436 (48.3%)	397 (46.6%)	39 (76.5%)	<0.001
CX involvement	146 (16.2%)	137 (16.1%)	9 (17.6%)	0.768
RCA involvement	231 (25.6%)	216 (25.4%)	15 (29.4%)	0.519

Values are n (%) unless otherwise stated. Age is presented as the median [IQR] and was compared using the Mann-Whitney U test. Categorical variables were compared using chi-square or Fisher's exact test, as appropriate. For SMuRF status and ACS type, p values represent overall omnibus tests. Missing data were not imputed; denominators are shown where they differ from the full cohort. ANOCA, INOCA, and MINOCA are reported descriptively in Supplementary Table S1. Statistical significance was defined as $p < 0.05$. *Mann-Whitney U test; †Fisher's exact test.

Angiographic Findings

Detailed angiographic and exploratory non-obstructive/mechanistic findings are provided in Supplementary Table S1. LAD involvement was the most frequent coronary finding and was identified in

436 patients (48.3%). CX involvement was present in 146 patients (16.2%), and RCA involvement was present in 231 patients (25.6%). Regarding the extent of coronary involvement, 297 patients (32.9%) had no major vessel involvement, 416 (46.1%) had single-vessel disease, 173 (19.2%) had double-vessel disease, and 17 (1.9%) had triple-vessel disease. Overall, multivessel disease was present in 190 patients (21.0%), whereas 713 patients (79.0%) did not have it. SCAD was recorded in four female patients aged 18–40 years. Among these patients, one death occurred during follow-up, whereas the remaining three patients were censored. Because of the small number of SCAD cases, this finding was reported descriptively and was not included in multivariable survival modeling.

Kaplan-Meier Survival Analyses

Kaplan-Meier analysis demonstrated significantly lower survival among patients with obesity compared with those without obesity (log-rank $p = 0.0019$). Survival was also significantly lower among patients with LAD involvement compared with those without LAD involvement (log-rank $p < 0.001$). These curves are presented in Fig. 1A and 1B.

Additional Kaplan-Meier analyses are summarized in Supplementary Table S2 and displayed in Supplementary Figs. S1–S3. Smoking and hypertension did not show statistically significant survival differences in the log-rank analyses, with $p = 0.17$ for both variables.

Cox Regression Analyses for Mortality

During a median follow-up of 4.93 years [95% CI: 4.69–5.18], 51 deaths occurred among 903 patients. Univariable Cox regression results are shown in Table 2. Candidate clinical and angiographic variables were evaluated for their association with mortality. Obesity was associated with mortality in univariable analysis [HR: 2.501; 95% CI: 1.377–4.542; $p = 0.003$], as was LAD involvement [HR: 3.666; 95% CI: 1.914–7.022; $p < 0.001$]. Alcohol use and substance use also showed statistically significant associations in univariable analysis; however, these estimates were based on sparse exposed cases and are reported descriptively in Table 2.

Table 2 Univariable Cox Proportional Hazards Regression Analysis for Mortality

Predictor	B	SE	Wald	HR	95% CI for HR	p
Age, per 1-year increase	-0.035	0.025	1.923	0.966	0.920–1.014	0.165
Female sex	-0.147	0.385	0.146	0.863	0.405–1.837	0.702
Alcohol use	3.318	0.759	19.131	27.611	6.242–122.133	<0.001
Hypertension	0.417	0.308	1.842	1.518	0.831–2.773	0.175
Hyperlipidemia	0.112	0.309	0.132	1.119	0.611–2.050	0.716
Diabetes mellitus	-0.028	0.408	0.005	0.973	0.437–2.166	0.946
Family history	0.422	0.284	2.207	1.525	0.874–2.661	0.137
Smoking	-0.386	0.285	1.826	0.680	0.389–1.190	0.177
Obesity	0.917	0.305	9.057	2.501	1.377–4.542	0.003
Substance use	3.420	0.756	20.457	30.563	6.944–134.521	<0.001
SMuRF-positive	-0.122	0.436	0.079	0.885	0.377–2.079	0.779
LAD involvement	1.299	0.332	15.346	3.666	1.914–7.022	<0.001
CX involvement	0.071	0.184	0.150	1.074	0.749–1.541	0.698
RCA involvement	0.047	0.103	0.212	1.048	0.857–1.282	0.646
ACS type overall	NA	NA	3.227	NA	NA	0.199
STEMI vs UA	0.667	0.601	1.232	1.949	0.600–6.332	0.267
NSTEMI vs UA	0.148	0.646	0.053	1.160	0.327–4.112	0.818

HR, hazard ratio; CI, confidence interval; UA, unstable angina; LAD, left anterior descending artery; CX, circumflex artery; RCA, right coronary artery; SMuRF, standard modifiable cardiovascular risk factor. UA was the reference category for ACS type. Male sex, absence of each binary risk factor, SMuRF-less status, and absence of vessel involvement were used as reference categories. Alcohol and substance-use estimates should be interpreted cautiously because of sparse exposed cases. Statistical significance was defined as $p < 0.05$.

In the multivariable Cox proportional hazards model including age, sex, hypertension, hyperlipidemia, diabetes mellitus, family history, smoking, obesity, and LAD involvement; obesity and LAD involvement remained independently associated with all-cause mortality. The results are presented in Table 3. Obesity was associated with a 2.68-fold higher hazard of mortality [adjusted HR: 2.677; 95% CI: 1.430–5.010; $p = 0.002$], and LAD involvement was associated with a 3.59-fold higher hazard [adjusted HR: 3.586; 95% CI: 1.849–6.956; $p < 0.001$]. Smoking was not independently associated with mortality in this model [adjusted HR: 0.829; 95% CI: 0.439–1.564; $p = 0.562$]. The multivariable Cox model is also presented graphically in Supplementary Fig. S4.

Table 3 Multivariable Cox Proportional Hazards Regression Analysis for Mortality

Predictor	B	SE	Wald	Adjusted HR	95% CI for adjusted HR	p
Female sex	-0.640	0.421	2.317	0.527	0.231–1.202	0.128
Age, per 1-year increase	-0.037	0.026	2.040	0.964	0.916–1.014	0.153
Hypertension	0.423	0.318	1.767	1.526	0.818–2.846	0.184
Hyperlipidemia	0.032	0.311	0.010	1.032	0.561–1.900	0.919
Diabetes mellitus	-0.113	0.426	0.071	0.893	0.388–2.058	0.791
Family history	0.352	0.292	1.453	1.422	0.802–2.519	0.228
Smoking	-0.188	0.324	0.336	0.829	0.439–1.564	0.562
Obesity	0.985	0.320	9.481	2.677	1.430–5.010	0.002
LAD involvement	1.277	0.338	14.277	3.586	1.849–6.956	<0.001

HR, hazard ratio; CI, confidence interval; LAD, left anterior descending artery. The multivariable model included age, sex, hypertension, hyperlipidemia, diabetes mellitus, family history, smoking, obesity, and LAD involvement. Alcohol and substance use were excluded because of sparsely exposed cases. Statistical significance was defined as $p < 0.05$.

Proportional Hazards Assessment and Sensitivity Analyses

The proportional hazards assumption was assessed using Schoenfeld residuals. In the full follow-up Cox model, evidence of non-proportionality was detected for selected variables, including age and obesity. Therefore, sensitivity analyses were performed using administrative censoring at 10 years.

After administrative censoring at 10 years, 46 deaths were included in the sensitivity analysis. The global proportional hazards test was no longer significant. In the parsimonious 10-year Cox model including age, female sex, obesity, and LAD involvement, obesity and LAD involvement remained independently associated with mortality, whereas age and female sex were not statistically significant predictors (Supplementary Table S3). Obesity was associated with a 2.49-fold higher mortality hazard [95% CI: 1.34–4.64; $p = 0.004$], and LAD involvement was associated with a 3.83-fold higher mortality hazard [95% CI: 1.89–7.74; $p < 0.001$]. The global proportional hazards test was not significant in this model ($p = 0.061$). Statistical significance was defined as $p < 0.05$.

Multinomial Logistic Regression for ACS Presentation Type

Multinomial logistic regression was performed to evaluate factors associated with ACS presentation type, with UA used as the reference category. The model evaluated adjusted associations for STEMI

versus UA and NSTEMI versus UA. Detailed adjusted ORs, 95% CIs, and p values are provided in Table 4.

Table 4 Adjusted Multinomial Logistic Regression Analysis for ACS Type

Predictor	STEMI vs UA Adjusted OR	95% CI	<i>p</i>	NSTEMI vs UA Adjusted OR	95% CI	<i>p</i>	Overall <i>p</i>
Age, per 1-year increase	0.847	0.793–0.905	<0.001	1.387	1.267–1.518	<0.001	<0.001
Female sex	19.160	2.540–144.506	0.004	29.057	3.809–221.678	0.001	<0.001
Hypertension	0.843	0.448–1.587	0.597	0.555	0.280–1.099	0.091	0.025
Hyperlipidemia	0.743	0.423–1.303	0.300	1.030	0.572–1.855	0.922	0.255
Diabetes mellitus	0.902	0.419–1.943	0.793	0.818	0.359–1.861	0.631	0.885
Family history	0.830	0.501–1.377	0.471	1.082	0.634–1.847	0.772	0.352
Smoking	1.313	0.725–2.376	0.369	1.118	0.596–2.096	0.729	0.609
Obesity	1.374	0.694–2.722	0.362	1.345	0.656–2.757	0.418	0.639
LAD involvement	1.193	0.734–1.939	0.477	0.740	0.440–1.245	0.257	0.034
CX involvement	1.173	0.591–2.327	0.649	1.112	0.545–2.266	0.771	0.895
RCA involvement	0.761	0.443–1.304	0.320	0.638	0.358–1.138	0.128	0.316

OR, odds ratio; CI, confidence interval; ACS, acute coronary syndrome; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; UA, unstable angina. UA was used as the reference category. Age was entered as a continuous variable. Statistical significance was defined as $p < 0.05$.

SMuRF-Stratified Analysis

SMuRF-stratified findings are presented in Supplementary Table S4. Overall, 96 patients (10.6%) were classified as SMuRF-less, and 807 patients (89.4%) were classified as SMuRF-positive. Age did not differ significantly between groups, with a median age of 36.0 years [IQR: 31.5–38.0] among SMuRF-less patients and 36.0 years [IQR: 32.0–39.0] among SMuRF-positive patients ($p = 0.419$).

SMuRF-less patients had a higher proportion of female sex than SMuRF-positive patients (39.6% vs 14.3%, $p < 0.001$). The ACS presentation type did not differ significantly by SMuRF status (overall $p = 0.672$). STEMI occurred in 53 SMuRF-less patients (55.2%) and 475 SMuRF-positive patients (58.9%), NSTEMI in 35 (36.5%) and 258 (32.0%), and UA in 8 (8.3%) and 74 (9.2%), respectively.

Obesity was more frequent among SMuRF-less patients than among SMuRF-positive patients (37.5% vs 17.8%, $p < 0.001$). LAD involvement was also more common in the SMuRF-less group (68.8% vs 45.8%, $p < 0.001$). CX involvement did not differ significantly between groups (21.9% vs 15.5%, $p = 0.108$), and RCA involvement was also similar (28.1% vs 25.3%, $p = 0.546$). The extent of vessel involvement differed significantly by SMuRF status (overall $p < 0.001$). Compared with SMuRF-positive patients, SMuRF-less patients had a lower frequency of no vessel involvement (14.6% vs 35.1%), a higher frequency of single-vessel disease (56.3% vs 44.9%), and a higher frequency of multivessel disease (29.2% vs 20.1%).

Despite these clinical and angiographic differences, mortality did not differ significantly between SMuRF-less and SMuRF-positive patients. Death occurred in 6 SMuRF-less patients (6.3%) and 45 SMuRF-positive patients (5.6%) ($p = 0.787$).

Discussion

Principal Findings and Prognostic Factors

In this cohort of 903 patients aged ≤ 40 years with ACS, long-term mortality was most consistently associated with obesity and LAD involvement, whereas SMuRF status itself was not associated with a significant mortality difference. These findings suggest that prognosis in very young ACS patients may be more strongly shaped by the combination of cardiometabolic burden and coronary anatomical involvement than by the conventional SMuRF construct alone. Nevertheless, the SMuRF-less subgroup revealed a different phenotype. The finding that SCAD was observed only in female patients is consistent with possible sex-specific non-atherothrombotic mechanisms in young ACS, although the small number of cases requires cautious interpretation.

The present findings add to the growing evidence that ACS in very young adults is not simply an age-shifted form of late-life coronary disease. Contemporary mechanistic frameworks describe ACS as a heterogeneous syndrome involving plaque rupture, plaque erosion, coronary spasm, SCAD, embolic events, and microvascular dysfunction, with variation according to age, sex, coronary anatomy, and risk-factor profile [2]. Rallidis et al. [3] identified premature myocardial infarction as a separate clinical entity characterized by particular angiographic features, risk-factor patterns, and therapeutic considerations. Large cohort studies from the United States and India also reveal that very young individuals with myocardial infarction or coronary artery disease carry high risk-factor burden and clinically significant disease patterns across different populations [15, 16]. In the present study, the main mortality-related findings involved both cardiometabolic and angiographic factors, namely obesity and LAD involvement, rather than SMuRF status alone. This supports an integrated risk-stratification approach in young ACS that includes both clinical and coronary anatomical information.

Obesity was one of the two principal mortality-related predictors in this cohort. This finding is clinically important because obesity in young adults may capture cumulative cardiometabolic exposure before overt hypertension, diabetes mellitus, or hyperlipidemia is diagnosed. Obesity is linked to cardiovascular disease through insulin resistance, systemic inflammation, dyslipidemia, endothelial dysfunction, thrombogenicity, and adverse cardiac remodeling [8, 9]. Mahendiran et al. reported increasing obesity among young ACS patients hospitalized in Switzerland over two decades [17]. Our findings extend this literature by showing that obesity was not only frequent but also independently associated with long-term mortality in patients aged ≤ 40 years. This is particularly relevant because age-weighted risk models may underestimate lifetime cardiovascular risk in young adults with substantial metabolic burden.

The prognostic importance of LAD involvement is another major finding. LAD disease was the most frequent angiographic finding and remained independently associated with mortality. This association is clinically plausible because LAD involvement often corresponds to a larger myocardial territory at risk and may be linked to greater left ventricular injury, arrhythmogenic substrate, and adverse remodeling. Yagel et al. reported that LAD involvement was associated with worse long-term prognosis in young ACS patients treated with percutaneous coronary intervention [5]. In the present cohort, LAD involvement remained independently associated with mortality after adjustment for clinical risk factors. These findings support the integration of coronary anatomical information, especially LAD involvement, into long-term prognostic assessment in young ACS.

Smoking was not identified as one of the main independent mortality-related predictors in this cohort. This finding should not be interpreted as evidence that smoking is clinically unimportant in young ACS. Biery et al. showed in the Partners YOUNG-MI registry that smoking cessation within 1 year after myocardial infarction was associated with substantially lower all-cause and cardiovascular mortality among young adults [18]. Therefore, smoking cessation remains a central objective of primary and secondary prevention, although obesity and LAD involvement showed the most consistent mortality-related associations in the present study.

SMuRF Status, Mechanistic Considerations, and Clinical Implications

The SMuRF-stratified analysis further supports the need to interpret conventional risk-factor status cautiously. SMuRF-less patients represented 10.6% of the cohort and had higher proportions of female sex, obesity, LAD involvement, and multivessel disease. However, mortality did not differ significantly between SMuRF-less and SMuRF-positive patients. Ball et al. reported that SMuRF-less ACS patients undergoing percutaneous coronary intervention more often presented with STEMI, cardiac arrest, or both, although short-term outcomes were similar and adjusted long-term mortality was lower [11]. In contrast, SMuRF-less ACS has been associated with higher mortality in some pooled and registry-based analyses [19–21]. Kong et al. reported increased mortality among ACS patients without conventional modifiable risk factors in a large meta-analysis [19]. These apparently divergent findings suggest that SMuRF-less status is not a uniform prognostic category. Instead, it may represent a heterogeneous

phenotype influenced by age, sex, treatment patterns, unmeasured risk factors, obesity, and coronary anatomy.

The higher prevalence of obesity among SMuRF-less patients is especially relevant because obesity is not included in the conventional SMuRF definition. A patient without hypertension, diabetes mellitus, hyperlipidemia, or current smoking may still carry clinically meaningful risk when obesity, adverse coronary anatomy, or sex-specific mechanisms are present [10]. Therefore, SMuRF-less status should not be interpreted as the absence of cardiovascular risk. Rather, it should prompt clinicians to consider non-standard or under-recognized risk pathways, including adiposity-related cardiometabolic stress, inherited susceptibility, inflammatory pathways, psychosocial factors, and sex-specific mechanisms.

The SCAD findings, although descriptive, are clinically relevant. SCAD was recorded in four female patients aged from 18 to 40 years, and one death occurred during follow-up. Non-atherothrombotic mechanisms, including SCAD, coronary embolism, vasospasm, and MINOCA, are increasingly recognized in younger adults, particularly women [13]. Hayes et al. described SCAD as an important cause of ACS, myocardial infarction, and sudden death, especially among young women and individuals with few conventional atherosclerotic risk factors [22, 23]. Due to the identification of only four SCAD cases, formal modeling was not suitable. Nevertheless, this observation supports careful diagnostic assessment in young women with ACS, particularly when traditional risk factors are absent or angiographic patterns are atypical.

The diagnostic implications extend beyond SCAD alone. Young patients with ACS may have a broader range of etiologies and mimics than older patients, including coronary anomalies, vasospasm, thrombophilia, substance-related ischemia, myocarditis, SCAD, and non-obstructive coronary syndromes [24]. Reynolds et al. showed that multimodality imaging using optical coherence tomography and cardiac magnetic resonance imaging can help identify underlying mechanisms in women with MINOCA [25]. These observations are relevant to young ACS because angiography alone may not fully explain the clinical presentation in selected patients. Diagnostic interpretation should therefore integrate symptoms, electrocardiography, biomarkers, coronary anatomy, imaging findings, and clinical context [14, 26].

The multinomial logistic regression analysis further illustrates the variability of ACS presentation in this age range. No single traditional risk factor fully explained ACS presentation type. Younger age was associated with STEMI presentation, whereas older age was associated with NSTEMI. This suggests that age-related variation in plaque morphology, thrombogenicity, or coronary vasomotion may influence ACS type, even within the ≤ 40 -year age range. STEMI, NSTEMI, and UA may reflect overlapping but distinct mechanisms, including plaque disruption, thrombosis, vasospasm, microvascular dysfunction, non-obstructive coronary disease, and non-atherothrombotic causes. Thus, a multifaceted approach that integrates coronary anatomy, sex-specific mechanisms, risk-factor profile, presentation type, and follow-up risk may help interpret young ACS.

The proportional hazards sensitivity analyses strengthen the survival findings. In the model using the complete follow-up period, Schoenfeld residual testing suggested non-proportionality for selected

variables. After administrative censoring at 10 years, however, the global proportional hazards test was no longer significant, and obesity and LAD involvement remained associated with mortality. The persistence of obesity and LAD involvement after 10-year administrative censoring, including in the parsimonious Cox model, supports the robustness of these variables as the main mortality-related predictors in this cohort.

These findings have practical implications. Young adults with ACS require long-term follow-up even when the early prognosis appears favorable. Obesity should be considered a major prognostic marker rather than only a background risk factor. LAD involvement should prompt careful risk stratification because it identifies a higher-risk angiographic subgroup. SMuRF-less status should not by itself reassure clinicians, particularly when obesity, LAD involvement, multivessel disease, or possible sex-specific or non-atherothrombotic mechanisms are present. Guideline-directed secondary prevention, including lipid management where indicated, remains essential [27]. Byrne et al. provided contemporary ESC guidance for ACS management across the full ACS spectrum [28]. In addition, structured evaluation of chest pain is essential to avoid missed ACS diagnoses, especially in younger patients and women [29]. Existing ACS risk prediction models face important limitations in simultaneously integrating clinical, angiographic, and cardiometabolic variables for individual prognostication [30]. Consistent with our findings, young STEMI cohorts have shown a higher prevalence of LAD as the culprit vessel compared with older patients, with smoking frequently reported as a prominent risk exposure [31]. More recently, machine-learning-based risk scores incorporating LAD involvement and other angiographic variables have been developed to refine risk stratification in STEMI and may offer improved individualized prognostication [32]. Together, these data support a more structured approach to young ACS that combines guideline-directed secondary prevention with individualized assessment of cardiometabolic risk, coronary anatomy, and sex-specific mechanisms.

Limitations

Several limitations warrant consideration. First, the single-center retrospective design may introduce selection, information, and documentation bias and may limit generalizability to other settings. Second, the primary endpoint was all-cause mortality; cause-specific cardiovascular mortality, recurrent myocardial infarction, repeat revascularization, heart failure hospitalization, medication adherence, and post-discharge lifestyle modification were not systematically available. Third, residual confounding remains possible because emerging or underrecognized risk factors, including inflammatory biomarkers, lipoprotein(a), genetic predisposition, socioeconomic status, psychosocial stressors, pregnancy-related vascular history, and detailed medication exposure, were not fully captured. Finally, SCAD was reported descriptively because only four cases were identified, and some multivariable and multinomial regression estimates require cautious interpretation because of the modest number of deaths, sparse subgroup counts, and wide confidence intervals.

Conclusion

In this cohort of patients aged ≤ 40 years with ACS, obesity and LAD involvement were independently associated with long-term all-cause mortality, whereas SMuRF status itself was not associated with a significant mortality difference. SMuRF-less patients showed a distinct clinical and angiographic phenotype, including higher proportions of female sex, obesity, LAD involvement, and multivessel disease. These findings indicate that risk assessment in very young ACS should extend beyond the conventional SMuRF construct and include obesity, coronary anatomical involvement, and possible sex-specific mechanisms. Early recognition of both standard and under-recognized risk factors may improve risk stratification and secondary prevention in this population.

Abbreviations

ACS: Acute coronary syndrome

ANOCA: Angina with nonobstructive coronary arteries

BMI: Body mass index

CAD: Coronary artery disease

CI: Confidence interval

CMR: Cardiac magnetic resonance

CX: Circumflex artery

ECG: Electrocardiogram

HR: Hazard ratio

INOCA: Ischemia with nonobstructive coronary arteries

IQR: Interquartile range

LAD: Left anterior descending artery

MINOCA: Myocardial infarction with nonobstructive coronary arteries

NSTEMI: Non-ST-segment elevation myocardial infarction

OCT: Optical coherence tomography

OR: Odds ratio

PCI: Percutaneous coronary intervention

RCA: Right coronary artery

SCAD: Spontaneous coronary artery dissection

SD: Standard deviation

SMuRF: Standard modifiable cardiovascular risk factor

STEMI: ST-segment elevation myocardial infarction

UA: Unstable angina

Declarations

Ethics approval and consent to participate

This study was approved by the Süleyman Demirel University School of Medicine Ethics Committee, Isparta, Türkiye (decision no. 70; November 5, 2024). The requirement for informed consent was waived because anonymized retrospective data were used. The study was conducted in accordance with the principles of the Declaration of Helsinki. Clinical trial number: not applicable.

Consent for publication

Not applicable.

Availability of data and materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

OG: Conceptualization; methodology; data curation; formal analysis; investigation; project administration; supervision; validation; visualization; writing - original draft; writing - review and editing; resources.

BU: Data curation; investigation; resources.

BAU: Clinical supervision; investigation; validation; writing - review and editing; resources.

YT: Clinical review; investigation; validation; writing - review and editing.

All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

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References

1. Palaniappan LP, Allen NB, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, et al. 2026 heart disease and stroke statistics: a report of US and global data from the American Heart Association. *Circulation*. 2026;153:e275-e906.
<https://doi.org/10.1161/cir.0000000000001412>
2. Kraler S, Mueller C, Libby P, Bhatt DL. Acute coronary syndromes: mechanisms, challenges, and new opportunities. *Eur Heart J*. 2025;46:2866-2889.
<https://doi.org/10.1093/eurheartj/ehaf289>
3. Rallidis LS, Xenogiannis I, Brilakis ES, Bhatt DL. Causes, angiographic characteristics, and management of premature myocardial infarction: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2022;79:2431-2449.
<https://doi.org/10.1016/j.jacc.2022.04.015>
4. Freedman AA, Colangelo LA, Ning H, Borrowman JD, Lewis CE, Schreiner PJ, et al. Sex differences in age of onset of premature cardiovascular disease and subtypes: The Coronary Artery Risk Development in Young Adults Study. *J Am Heart Assoc*. 2026;15:e044922.
<https://doi.org/10.1161/JAHA.125.044922>
5. Yagel O, Shadafny N, Eliaz R, Dagan G, Leibowitz D, Tahiroglu I, et al. Long-term prognosis in young patients with acute coronary syndrome treated with percutaneous coronary intervention. *Vasc Health Risk Manag*. 2021;17:153-159. <https://doi.org/10.2147/vhrm.s298436>
6. Kassam N, Ngunga M, Varwani M, Msunza M, Jeilan M. Acute coronary syndrome patterns in the young: risk factor profile and in-hospital outcomes in a tertiary referral hospital in Kenya. *BMC Cardiovasc Disord*. 2024;24:192.
<https://doi.org/10.1186/s12872-024-03832-z>
7. Shrateh ON, Al-Tawil M, Awad A, Zeer ZMM, Owais TA, Sinokrot A, et al. Acute coronary syndrome in young (≤ 45 years) patients: a multi-centre observational study. *Ann Med Surg (Lond)*. 2024;86:3303-

<https://doi.org/10.1097/ms9.00000000000002125>

8. Simonsson M, Mohammad MA, Sederholm Lawesson S, Hofmann R, Faxén J, Erlinge D, et al. Trends in risk factors among young patients with acute myocardial infarction: a nationwide cohort study. *Eur Heart J Qual Care Clin Outcomes*. 2025;11:835-846. <https://doi.org/10.1093/ehjqcco/qcaf034>
9. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. 2021;143:e984-e1010.

<https://doi.org/10.1161/CIR.0000000000000973>

10. Figtree GA, Vernon ST, Harmer JA, Gray MP, Arnott C, Bachour E, et al. Clinical pathway for coronary atherosclerosis in patients without conventional modifiable risk factors: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2023;82:1343-1359.

<https://doi.org/10.1016/j.jacc.2023.06.045>

11. Ball J, Dinh DT, Brennan A, Ajani A, Clark DJ, Freeman M, et al. Prevalence and outcomes of patients with SMuRF-less acute coronary syndrome undergoing percutaneous coronary intervention. *Open Heart*. 2024;11:e002733.

<https://doi.org/10.1136/openhrt-2024-002733>

12. Justin Paul G, Sankaran S, Saminathan K, Iliyas M, Sethupathy S, Saravanan S, et al. Outcomes of ST segment elevation myocardial infarction without standard modifiable cardiovascular risk factors: newer insights from a prospective registry in India. *Glob Heart*. 2023;18:13.

<https://doi.org/10.5334/gh.1189>

13. Al-Gully J, Montero-Cabezas JM, Eikenboom J, den Exter PL, van der Kley F, Jukema W, et al. Diagnosis and treatment of unrecognized spontaneous coronary artery dissection in young women presenting with an acute coronary syndrome. *Am J Cardiol*. 2025;255:116-125.

<https://doi.org/10.1016/j.amjcard.2025.08.017>

14. Parlati ALM, Nardi E, Sucato V, Madaudo C, Leo G, Rajah T, et al. ANOCA, INOCA, MINOCA: The New Frontier of Coronary Syndromes. *J Cardiovasc Dev Dis*. 2025;12:64.

<https://doi.org/10.3390/jcdd12020064>

15. Yang J, Biery DW, Singh A, Divakaran S, DeFilippis EM, Wu WY, et al. Risk factors and outcomes of very young adults who experience myocardial infarction: The Partners YOUNG-MI Registry. *Am J*

Med. 2020;133:605-612.e1.

<https://doi.org/10.1016/j.amjmed.2019.10.020>

16. Singh M, Kumar R, Kansal V, Singhal A, Kapoor A, Singh G. Young onset coronary artery disease in India: A descriptive observational study. *Clin Epidemiol Glob Health*. 2024;30:101851.

<https://doi.org/10.1016/j.cegh.2024.101851>

17. Mahendiran T, Hoepli A, Foster-Witassek F, Rickli H, Roffi M, Eberli F, et al. Twenty-year trends in the prevalence of modifiable cardiovascular risk factors in young acute coronary syndrome patients hospitalized in Switzerland. *Eur J Prev Cardiol*. 2023;30:1504-1512.

<https://doi.org/10.1093/eurjpc/zwad077>

18. Biery DW, Berman AN, Singh A, Divakaran S, DeFilippis EM, Collins BL, et al. Association of smoking cessation and survival among young adults with myocardial infarction in the Partners YOUNG-MI Registry. *JAMA Netw Open*. 2020;3:e209649. <https://doi.org/10.1001/jamanetworkopen.2020.9649>

19. Kong G, Chin YH, Chong B, Goh RSJ, Lim OZH, Ng CH, et al. Higher mortality in acute coronary syndrome patients without standard modifiable risk factors: Results from a global meta-analysis of 1,285,722 patients. *Int J Cardiol*. 2023;371:432-440.

<https://doi.org/10.1016/j.ijcard.2022.09.062>

20. Figtree GA, Vernon ST, Hadziosmanovic N, Sundström J, Alfredsson J, Arnott C, et al. Mortality in STEMI patients without standard modifiable risk factors: a sex-disaggregated analysis of SWEDEHEART registry data. *Lancet*. 2021;397:1085-1094.

[https://doi.org/10.1016/S0140-6736\(21\)00272-5](https://doi.org/10.1016/S0140-6736(21)00272-5)

21. Gong W, Yan Y, Liu J, Wang X, Zheng W, Que B, et al. In-hospital mortality and treatment in patients with acute coronary syndrome with or without standard modifiable cardiovascular risk factors: A scientific report from the CCC-ACS Project. *J Am Heart Assoc*. 2024;13:e029252.

<https://doi.org/10.1161/jaha.122.029252>

22. Hayes SN, Kim ESH, Saw J, Adlam D, Arslanian-Engoren C, Economy KE, et al. Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. *Circulation*. 2018;137:e523-e557.

<https://doi.org/10.1161/cir.0000000000000564>

23. Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, et al. Spontaneous coronary artery dissection: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2020;76:961-984.

<https://doi.org/10.1016/j.jacc.2020.05.084>

24. Krittanawong C, Kumar A, Virani SS, Bangalore S, Wang Z, Johnson KW, et al. Acute myocardial infarction: Etiologies and mimickers in young patients. *J Am Heart Assoc.* 2023;12:e029971.

<https://doi.org/10.1161/JAHA.123.029971>

25. Reynolds HR, Maehara A, Kwong RY, Sedlak T, Saw J, Smilowitz NR, et al. Coronary optical coherence tomography and cardiac magnetic resonance imaging to determine underlying causes of myocardial infarction with nonobstructive coronary arteries in women. *Circulation.* 2021;143:624-640.

<https://doi.org/10.1161/CIRCULATIONAHA.120.052008>

26. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). *Circulation.* 2018;138:e618-e651.

<https://doi.org/10.1161/cir.0000000000000617>

27. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol. *J Am Coll Cardiol.* 2019;73:e285-e350.

<https://doi.org/10.1016/j.jacc.2018.11.003>

28. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J.* 2023;44:3720-3826.

<https://doi.org/10.1093/eurheartj/ehad191>

29. 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. *Circulation.* 2021;144:e368-e454.

<https://doi.org/10.1161/CIR.0000000000001029>

30. Haghbayan H, Gale CP, Chew DP, Brieger D, Fox KA, Goodman SG, Yan AT. Clinical risk prediction models for the prognosis and management of acute coronary syndromes. *Eur Heart J Qual Care Clin Outcomes.* 2021;7:222-228.

<https://doi.org/10.1093/ehjqcco/qcab018>

31. Liang MT, Pang Y, Gao LL, Han LJ, Yao HC. Clinical risk factors and outcomes of young patients with acute ST segment elevation myocardial infarction: a retrospective study. *BMC Cardiovasc Disord.* 2023;23:353.

<https://doi.org/10.1186/s12872-023-03392-8>

32. Sritharan HP, Nguyen H, Ciofani J, Bhindi R, Allahwala UK. Machine-learning based risk prediction of in-hospital outcomes following STEMI: the STEMI-ML score. *Front Cardiovasc Med*. 2024;11:1454321.

<https://doi.org/10.3389/fcvm.2024.1454321>

Figures

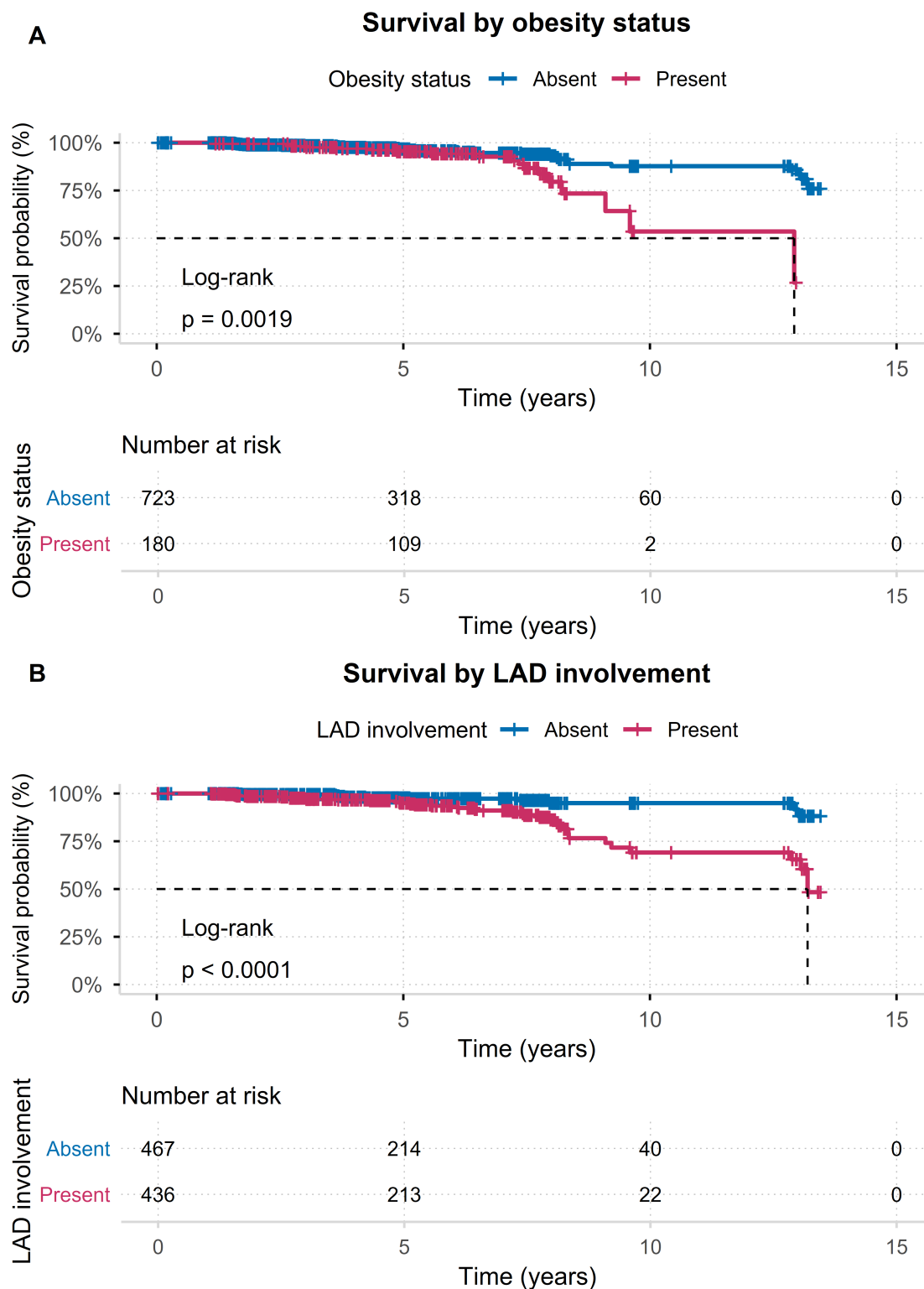


Figure 1

Kaplan-Meier survival curves for key mortality-related variables. (A) Survival probability was significantly lower among patients with obesity than among those without obesity (log-rank $p = 0.0019$). (B) Survival probability was significantly lower among patients with left anterior descending artery (LAD) involvement than among those without LAD involvement (log-rank $p < 0.001$). Number-at-risk tables are shown below the survival curves. Statistical significance was defined as $p < 0.05$.

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