Coronary Artery Disease, Coronary Revascularization, and Outcomes in Chronic Advanced Systolic Heart Failure

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Coronary artery disease, coronary revascularization, and outcomes in chronic advanced systolic heart failure

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Abstract

Background—Associations between coronary artery disease (CAD) and outcomes in systolic heart failure (HF) and that between coronary artery bypass graft (CABG) and outcomes in patients with HF and CAD have not been examined using propensity-matched designs.

Methods—Of the 2707 patients with advanced chronic systolic HF in the Beta-Blocker Evaluation of Survival Trial (BEST), 1593 had a history of CAD, of whom 782 had prior CABG. Using propensity scores for CAD we assembled a cohort of 458 pairs of CAD and no-CAD patients. Propensity scores for prior CABG in those with CAD were used to assemble 500 pairs of patients with and without CABG. Matched patients were balanced on 68 baseline characteristics.

Results—All-cause mortality occurred in 33% and 24% of matched patients with and without CAD respectively, during 26 months of median follow-up (hazard ratio {HR} when CAD was compared with no-CAD, 1.41; 95% confidence interval {CI}, 1.11–1.81; P=0.006). HR's (95% CIs) for CAD-associated cardiovascular mortality, HF mortality, and sudden cardiac death (SCD) were 1.53 (1.17–2.00; P=0.002), 1.44 (0.92–2.25; P=0.114) and 1.76 (1.21–2.57; P=0.003) respectively. CAD had no association with hospitalization. Among matched patients with HF and CAD, all-cause mortality occurred in 32% and 39% of those with and without prior CABG respectively (HR for CABG, 0.77; 95% CI, 0.62–0.95; P=0.015).

Conclusions—In patients with advanced chronic systolic HF, CAD is associated with increased mortality, and in those with CAD, prior CABG seems to be associated with reduced all-cause mortality but not SCD.

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Keywords

chronic heart failure; coronary artery disease; coronary artery bypass graft; revascularization; mortality; hospitalization

1. Introduction

Coronary artery disease (CAD) is a major risk factor for heart failure (HF) [1,2]. Damage from ongoing myocardial ischemia may adversely affect disease progression and prognosis in chronic HF [2–6]. However, if the history of CAD has an independent association with outcomes in patients with advanced systolic HF has not been previously examined in propensity-matched studies. Furthermore, whether a history of prior coronary artery bypass graft (CABG) surgery is associated with improved outcomes in advanced systolic HF patients with CAD has also not been examined in propensity-matched studies. Therefore, the objectives of this study were to determine (1) association between CAD and outcomes in a propensity-matched population of advanced chronic systolic HF patients, and (2) association between prior CABG and outcomes in a propensity-matched population of advanced chronic systolic HF patients with CAD.

2. Materials and Methods

2.1. Source of data

Details of the design and results of the Beta-Blocker Evaluation of Survival Trial (BEST) trial have been published previously [7]. Briefly, 2708 patients, ≥18 years old with advanced chronic systolic HF, were enrolled from 90 different sites across the United States and Canada between May 1995 and December 1998, and were then randomized to receive either bucindolol or placebo [7]. Only patients with left ventricular ejection fraction ≤35% were included and those with acute decompensated HF were excluded. Patients had a mean of 49 months of HF duration and all patients had New York Heart Association (NYHA) class III–IV symptoms. Over 90% of patients were receiving angiotensin-converting enzyme (ACE) inhibitors, diuretics, and digitalis.

2.2. History of CAD and prior CABG

Of the 2707 patients in the public-use copy of the BEST dataset, obtained from the NHLBI, 1593 had a history of CAD. Of these, 56% patients had a history of an ST-elevation acute myocardial infarction (AMI) that occurred ≥6 months before randomization, 79% had at least one >70% coronary artery stenosis by angiography, 49% had a prior CABG, and 26% had a prior a percutaneous coronary intervention (PCI). Patients were excluded if they had a history of AMI within the 6 months before randomization, if they had angina pectoris requiring treatment with >6 nitroglycerin tablets per week, or if they had undergone PCI or a CABG surgery within the 2 months before randomization. However, data on the number of patients excluded for these reasons were not available. Overall, 3% patients had implantable cardioverter defibrillators (ICD), of whom 77% had CAD.

2.3. Study outcomes

Primary outcomes for the current analysis were all-cause mortality and HF hospitalization. Secondary outcomes were cause-specific mortalities, including sudden cardiac death (SCD), and all-cause hospitalization. Study outcomes were ascertained by blinded investigators. Patients were followed for a median, 26 months (range, 0.07 to 50 months).

2.4. Assembly of a balanced cohort

Because of significant imbalances in baseline characteristics between patients with and without a CAD (Table 1), we used propensity score matching to assemble a cohort in which patients with and without CAD would be well-balanced in all measured baseline covariates[8,9]. We estimated propensity scores for CAD for each of the 2707 participants using a non-parsimonious multivariable logistic regression model, checking for plausible interactions, in which CAD was used as the dependent variable, and 68 measured baseline characteristics (Figure 1) were included as covariates.

We used a greedy matching protocol, described elsewhere in detail, to match 487 pairs of patients with and without CAD who had similar propensity scores [10–17]. To determine the efficacy of the propensity score model to assemble a balanced cohort, we estimated absolute standardized differences for each measured baseline covariate between patients with and without CAD and data were presented as Love plots (Figure 1) [10,12,14,18–20]. Values of absolute standardized difference are not affected by sample size and values <10% are considered of inconsequential bias [10,12,14,21].

We then repeated the above processes for all 1593 patients with HF and CAD of whom 782 (49%) had prior CABG. Propensity scores for CABG were estimated and used to assemble a balanced cohort of 500 pairs of CAD patients with and without prior CABG.

2.5. Statistical analysis

For descriptive analyses, we used Pearson chi-square and Wilcoxon rank-sum tests for the pre-match, and McNemar's test and paired sample t-test for the post-match comparisons of baseline covariates between HF patients with and without CAD, and CAD patients with and without prior CABG, as appropriate. Kaplan-Meier and Cox regression analyses were used to determine the associations of CAD and CABG with various outcomes. Formal sensitivity analyses were conducted to quantify the degree of hidden bias that would need to be present to invalidate our main conclusions [22]. Select subgroup analyses of matched patients were conducted to determine heterogeneity of the associations of CAD with all-cause mortality. All statistical tests were two-sided, and tests with p-values <0.05 were considered significant. All statistical analyses were done using SPSS for windows version 15 [23].

3. Results

3.1. Baseline characteristics

Matched patients had a mean age of 60 (±11) years, 22% were women and 26% were African American. Pre-match imbalances and post-match balances in baseline characteristics are displayed in Table 1 and Figure 1. Post-match absolute standardized differences for all measured covariates were <10% (most were <5%), suggesting substantial covariate balance across the groups (Figure 1).

3.2. A history of CAD and outcomes

All-cause mortality occurred in 33% and 24% of matched patients with and without CAD (hazard ratio {HR} when CAD was compared with no-CAD, 1.41; 95% confidence interval {CI}, 1.11–1.81; P=0.006; Figure 2a and Table 2). In the absence of a hidden bias, a sign-score test for matched data with censoring provided strong evidence (P=0.035) that patients without CAD clearly outlived those with CAD. An unmeasured covariate that is a near-perfect predictor of mortality could potentially explain away this association if it would increase the odds of CAD by only 2.19%. The association of CAD with all-cause mortality was homogenous across various patient subgroups, except that the CAD-associated all-cause mortality increase was significantly higher in women (Figure 3). Before matching, all-cause

mortality occurred in 37% and 24% of patients with and without CAD (HR when CAD was compared with no-CAD, 1.69; 95% CI, 1.47–1.95; P<0.001; Table 2).

SCD occurred in 16% and 9% of matched patients with and without CAD, respectively (HR when CAD was compared with no-CAD, 1.76; 95% CI, 1.21–2.57; P=0.003; Figure 2b and Table 2). Before matching, SCD occurred in 17% and 10% of patients with and without CAD, respectively (HR when CAD was compared with no-CAD, 1.77; 95% CI, 1.43–2.21; P<0.001; Table 2). In the absence of a hidden bias, a sign-score test for matched data with censoring provides strong evidence (P=0.010) that patients without CAD clearly had fewer SCD than those with CAD. An unmeasured covariate would need to increase the odds of CAD by 14.63% before it could potentially explain away this association. Associations of CAD with other outcomes among matched patients are displayed in Table 2.

3.3. Prior CABG and outcomes in patients with HF and CAD

Pre-match imbalances and post-match balances in baseline characteristics by prior CABG surgery are displayed in Table 3. All-cause mortality occurred in 32% and 39% matched patients with and without a history of prior CABG, respectively (HR when CABG was compared with no-CABG 0.77; 95% CI 0.62–0.95; P=0.015; Figure 4 and Table 4). A sign-score test for matched data with censoring did not provide clear evidence (P=0.117) that those with prior CABG surgery outlived those without. Associations of prior CABG with other outcomes are displayed in Table 4.

4. Discussion

The findings of the current study demonstrate that in patients with advanced chronic systolic HF, a history of CAD was a strong and independent predictor of all-cause and cardiovascular mortality, which was primarily driven by an increase in SCD. Consistent with these findings, we also observed that CAD had no association with hospitalization. We also observed that in patients with HF and CAD, a prior CABG surgery was associated with a significant reduction in all-cause mortality but not in SCD. Despite a beneficial role of revascularization in the treatment of CAD in general, its role in HF patients with CAD is less clear [24]. However, findings from the current study suggest that judicious use of coronary revascularization may be beneficial in these patients.

SCD is common in advanced systolic HF and is often associated with myocardial fibrosis, increased left ventricular stress, and repolarization abnormalities, all of which may be more common in those with CAD [25–30]. CAD-associated mortality in HF may be mediated through a variety of mechanisms including SCD, pump failure, AMI, renal failure and stroke. However, findings from our study suggest that the CAD-associated increased mortality was primarily cardiovascular, driven by an increase in SCD. This is consistent with our observation that CAD had no association with hospitalization as SCD is likely to preclude hospitalization. The predominance of SCD in BEST participants with advanced systolic HF is intriguing as SCD has been shown to be less common in patients with more advanced HF [31].

We observed that mortality due to AMI and HF was low and not significantly associated with a history of CAD suggesting that recurrent AMI may play a lesser role in disease progression to advanced systolic HF. Although we had no data on incident AMI, in patients with advanced systolic HF and CAD, AMI may be under diagnosed. Autopsy studies from these patients suggest that AMI in these patients may be small and/or subclinical and often associated with SCD [32,33]. Therefore, it is possible that some deaths classified as SCD in our study may have actually been caused by AMI. A high rate of plaque rupture or coronary thrombosis has been documented in patients with CAD who suffered SCD [34,35]. Taken

together, these findings suggest that CAD may increase arhythmogenicity of the myocardial substrate, thus increasing the risk for SCD.

Despite lack of a significant association between CABG and reduction in SCD, likely due to small numbers of events, findings from our study suggest that a prior CABG may improve long-term survival in HF patients with CAD. These findings are consistent with previous reports of an association between history of revascularization and improved survival in patients with HF [36–39]. However, our study is distinguished by the use of propensity score matching to assemble a balanced study cohort while remaining blinded to study outcomes [9]. Randomized controlled trials for coronary revascularization are difficult to perform as it may be nearly impossible to randomize patients before coronary angiography is performed, and yet results of angiography can strongly influence study participant selection. The randomized Surgical Treatment for Ischemic Heart Failure (STICH) trial, due to be completed by December 2012, will likely provide more definitive evidence in this regard [24].

The long-term prognosis of patients with HF and CAD is directly related to the angiographic extent and severity of CAD [5,40]. Revascularization may reduce mortality by reducing the amount of jeopardized myocardium, improving left ventricular ejection fraction, stimulating ventricular reverse remodeling, and possibly by reducing SCD [41]. The role of revascularization in HF patients with CAD may be important as recent reports suggest that the optimal use of recommended evidence-based pharmacotherapy with statins may not improve prognosis in these patients [42,43].

There are several limitations in the current study. We had no data regarding the severity of CAD, completeness of revascularization, and surgical mortality among CABG patients. It is possible that more patients without prior CABG had nonviable myocardium [44,45]. However, we had no data on myocardial viability. Although half of the patients were receiving beta-blockers, bucindolol is not currently approved for use in HF. Finally, it is possible that some patients with asymptomatic CAD may have been misclassified as having no CAD, which may have underestimated the association between CAD and outcomes in our study.

In conclusion, findings from the current study suggest that in patients with advanced chronic systolic HF, a history of CAD is associated with increased mortality, which is primarily driven by an increase in SCD, and in those with CAD, a prior CABG seems to be associated with reduced mortality. A history of CAD may be used to identify advanced systolic HF patients at increased risk of SCD who might benefit from targeted therapies, including judicious use of coronary revascularization.

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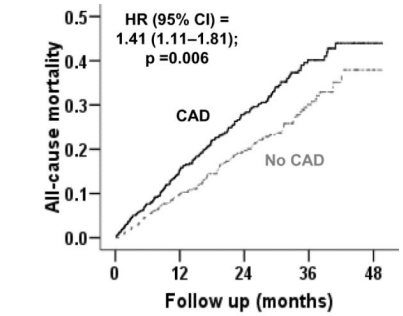
Fig. 1.

Love plots displaying pre- and post-match absolute standardized differences for covariates between patients with and without history of coronary artery disease (ACE=angiotensin-converting enzyme; ARB=angiotensin receptor blocker; LV=left ventricular; NYHA=New York Heart Association; RV=right ventricular)

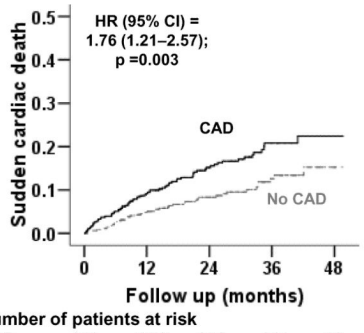
Figure 2a



Number of patients at risk

CAD	458	379	256	111	10
No CAD	458	350	241	96	10

Figure 2b



Number of patients at risk

CAD	458	379	256	111	10
No CAD	458	350	241	96	10

Fig. 2.

Kaplan-Meier plots for all-cause mortality (**2a**) and sudden cardiac death (**2b**) by history of coronary artery disease (CAD) (CI= confidence interval; HR=hazard ratio)

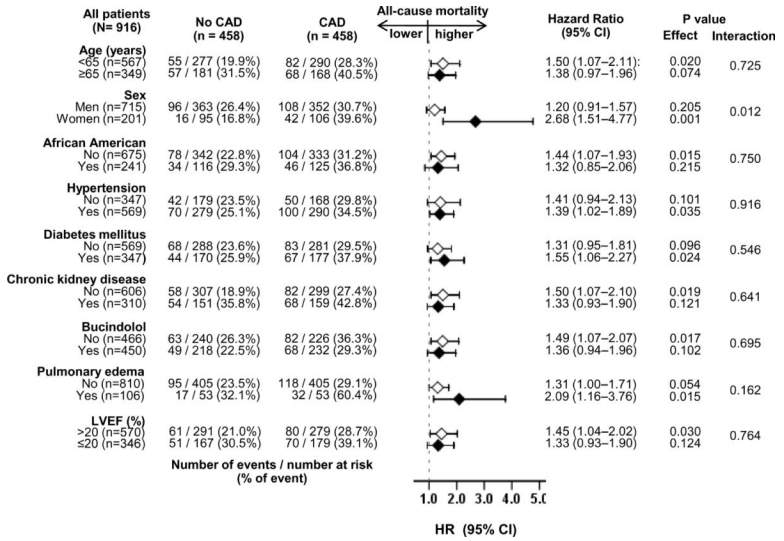
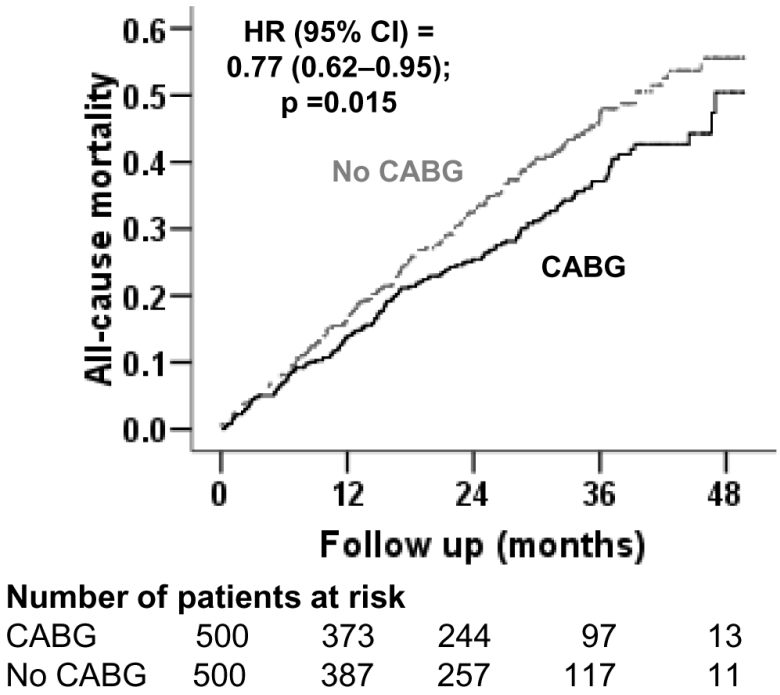


Fig. 3.

Association of a history of coronary artery disease (CAD) with all cause mortality in subgroups of propensity score-matched patients in the BEST trial (CI = confidence interval; HR = hazard ratio; LV = left ventricular)



Number of patients at risk

CABG	500	373	244	97	13
No CABG	500	387	257	117	11

Fig. 4.

Kaplan-Meier plots for all-cause mortality by coronary artery bypass graft (CABG) surgery among advanced systolic heart failure patients (CI= confidence interval; HR=hazard ratio)

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Table 1

Baseline characteristics, by a history of coronary artery disease (CAD), before and after propensity score matching

	Pre-match			Post-match		
n (%) or mean (±SD)	**No-CAD (n=1114)**	**CAD (n=1593)**	**P value**	**No-CAD (n=458)**	**CAD (n=458)**	**P value**
Age, years	55 (±13)	64 (±10)	<0.0001	60 (±12)	60 (±11)	0.564
Female	363 (33)	230 (14)	<0.0001	95 (21)	106 (23)	0.425
African American	357 (32)	270 (17)	<0.0001	116 (25)	125 (27)	0.548
Body mass index, kg/m^2	38 (±9)	36 (±7)	<0.0001	37 (±9)	37 (±8)	0.448
Current smoker	182 (16)	292 (18)	0.179	78 (17)	79 (17)	1.000
Smoking duration, years	16 (±16)	25 (±17)	<0.0001	20 (±18)	21 (±16)	0.723
Past medical history						
Duration of HF, months	47 (±47)	51 (±49)	0.013	50 (±48)	49 (±47)	0.744
Angina pectoris	242 (22)	1158 (73)	<0.0001	198 (43)	188 (41)	0.474
Diabetes mellitus	316 (28)	648 (41)	<0.0001	170 (37)	177 (39)	0.682
Hypertension	586 (53)	1009 (63)	<0.0001	279 (61)	290 (63)	0.505
Hyperlipidemia	278 (25)	892 (56)	<0.0001	163 (36)	172 (38)	0.676
Atrial fibrillation	236 (21)	417 (26)	0.003	111 (24)	102 (22)	0.531
Ventricular fibrillation	49 (4)	217 (14)	<0.0001	39 (9)	32 (7)	0.470
Peripheral vascular disease	88 (8)	353 (22)	<0.0001	57 (12)	57 (12)	1.000
Chronic kidney disease	303 (27)	703 (44)	<0.0001	151 (33)	159 (35)	0.631
Pacemaker	70 (6)	161 (10)	<0.0001	35 (8)	42 (9)	0.494
Implanted cardiac defibrillator	21 (2)	69 (4)	<0.0001	14 (3)	14 (3)	1.000
Medications						
Bucindolol	555 (50)	799 (50)	0.863	218 (48)	232 (51)	0.387
ACE inhibitors or angiotensin receptor blocker	1088 (98)	1520 (95)	0.002	445 (97)	443 (97)	0.851
Digitalis	1054 (95)	1440 (90)	<0.0001	428 (93)	426 (93)	0.896
Diuretics	1048 (94)	1476 (93)	0.148	431 (94)	430 (94)	1.000
Vasodilators	333 (30)	851 (53)	<0.0001	173 (38)	187 (41)	0.351
Anti-coagulants	617 (55)	953 (60)	0.021	260 (57)	267 (58)	0.688
Clinical features						

	Pre-match			Post-match		
n (%) or mean (±SD)	**No-CAD (n=1114)**	**CAD (n=1593)**	**P value**	**No-CAD (n=458)**	**CAD (n=458)**	**P value**
S3 gallop	528 (47)	650 (41)	0.001	199 (43)	215 (47)	0.335
Jugular venous distension	497 (45)	739 (46)	0.361	205 (45)	205 (45)	1.000
Leg edema	275 (25)	455 (29)	0.025	124 (27)	126 (28)	0.942
Pulmonary rales	104 (9)	255 (16)	<0.0001	51 (11)	63 (14)	0.271
NYHA class III	1041 (93)	1440 (90)	0.005	424 (93)	422 (92)	0.804
Heart rate, beats per minute	84 (±14)	80 (±12)	<0.0001	82 (±14)	83 (±13)	0.562
Blood pressure, mm Hg						
Systolic	117 (±19)	117 (±18)	0.975	119 (±19)	119 (±18)	0.939
Diastolic	72 (±11)	70 (±11)	<0.0001	72 (±11)	72 (±11)	0.674
Chest X-ray						
Cardiothoracic ratio >0.5	871 (78)	1176 (74)	0.009	339 (74)	344 (75)	0.761
Pulmonary edema	105 (9)	203 (13)	0.007	53 (12)	53 (12)	1.000
Electrocardiography						
Left bundle branch block	340 (31)	339 (21)	<0.0001	123 (27)	114 (25)	0.550
Right bundle branch block	33 (3)	145 (9)	<0.0001	22 (5)	25 (6)	0.766
Multi Gated Acquisition Scan						
Left ventricular ejection fraction, %	23 (±7)	23 (±7)	0.333	23 (±8)	23 (±7)	0.317
Right ventricular ejection fraction, %	35 (±12)	35 (±12)	0.373	35 (±12)	34 (±12)	0.989
Laboratory values						
Hemoglobin, g/dL	14 (±2)	14 (±2)	0.867	14 (±2)	14 (±2)	0.468
White blood cell, 10^3 /μL	7.4 (±2.1)	7.6 (±2.2)	0.081	7.5 (±2.2)	7.3 (±1.9)	0.318
Platelet, 10^3 /μL	233 (79)	214 (63)	<0.0001	223 (66)	222 (68)	0.895
Potassium, mEq/L	4.26 (±0.47)	4.35 (±0.48)	<0.0001	4.31 (±0.46)	4.32 (±0.48)	0.679
Creatinine, mg/dL	1.15 (±0.37)	1.31 (±0.42)	<0.0001	1.21 (±0.38)	1.23 (±0.38)	0.449
Total cholesterol, mg/dl	197 (±49)	193 (±49)	0.030	195 (±46)	197 (±50)	0.456
Norepinephrine, pg/ml	497 (±299)	527 (±311)	0.013	501 (±314)	515 (±316)	0.529

ACE = angiotensin converting enzyme; NYHA = New York Heart Association; PCI = Percutaneous Coronary Intervention; STEMI = ST-elevation during index acute myocardial infarction

Table 2

Association between coronary artery disease (CAD) and outcomes in patients with advanced systolic heart failure (HF)

	Events (%)		HR (95% CI)	P value
Pre-match	**No-CAD (n=1114)**	**CAD (n= 1593)**		
All-cause mortality	270 (24%)	589 (37%)	1.69 (1.47–1.95)	<0.001
Cardiovascular mortality	222 (20%)	508 (32%)	1.77 (1.51–2.07)	<0.001
HF mortality	75 (7%)	187 (12%)	1.95 (1.49–2.55)	<0.001
Sudden cardiac death	116 (10%)	268 (17%)	1.77 (1.43–2.21)	<0.001
All-cause hospitalization	659 (59%)	1044 (66%)	1.28 (1.16–1.41)	<0.001
HF hospitalization	393 (35%)	651 (41%)	1.30 (1.14–1.47)	<0.001
Post-match	**No-CAD (n=458)**	**CAD (n = 458)**		
All-cause mortality	112 (24%)	150 (33%)	1.41 (1.11–1.81)	0.006
Cardiovascular mortality	89 (19%)	129 (28%)	1.53 (1.17–2.00)	0.002
HF mortality	33 (7%)	45 (10%)	1.44 (0.92–2.25)	0.114
Sudden cardiac death	43 (9%)	72 (16%)	1.76 (1.21–2.57)	0.003
All-cause hospitalization	294 (64%)	284 (62%)	1.01 (0.85–1.18)	0.947
HF hospitalization	161 (35%)	178 (39%)	1.22 (0.98–1.50)	0.073

CI = confidence internal; HR = hazard ratio

Table 3

Baseline characteristics of heart failure (HF) patient with coronary artery disease (CAD), by coronary artery bypass graft surgery (CABG), before and after propensity matching

n (%) or mean (±SD)	Before matching			After matching		
	CABG		P value	CABG		P value
	No (n=811)	Yes (n=782)		No (n=500)	Yes (n=500)	
Age, years	63 (±11)	65 (±9)	0.001	63 (±11)	63 (±9)	0.751
Female	146 (18)	84 (11)	<0.0001	71 (14)	73 (15)	0.927
African American	199 (25)	71 (9)	<0.0001	68 (14)	65 (13)	0.838
Body mass index, kg/m^2	36 (±8)	36 (±7)	0.992	36 (±8)	36 (±7)	0.683
Current smoker	168 (21)	124 (16)	0.012	85 (17)	94 (19)	0.512
Smoking duration, years	25 (±18)	25 (±17)	0.269	25 (±18)	26 (±17)	0.318
Past medical history						
STEM	467 (58)	427 (55)	0.231	285 (57)	278 (56)	0.705
Prior PCI	200 (25)	221 (28)	0.103	140 (28)	149 (30)	0.530
Duration of HF, months	51 (±49)	52 (±49)	0.562	52 (±51)	53 (±50)	0.891
Angina pectoris	525 (65)	633 (81)	<0.0001	373 (75)	376 (75)	0.878
Diabetes mellitus	339 (42)	309 (41)	0.353	207 (41)	204 (41)	0.903
Hypertension	527 (65)	482 (62)	0.166	314 (63)	311 (62)	0.897
Hyperlipidemia	416 (51)	476 (61)	<0.0001	285 (57)	299 (60)	0.405
Atrial fibrillation	187 (23)	230 (29)	0.004	120 (24)	126 (25)	0.721
Ventricular fibrillation	97 (12)	120 (15)	0.049	67 (13)	67 (13)	1.000
Peripheral arterial disease	161 (20)	192 (25)	0.024	100 (20)	111 (22)	0.419

n (%) or mean (±SD)	Before matching			After matching		
	CABG		P value	CABG		P value
	No (n=811)	Yes (n=782)		No (n=500)	Yes (n=500)	
Chronic kidney disease	328 (40)	375 (48)	0.003	210 (42)	225 (45)	0.371
Pacemaker	58 (7)	103 (13)	<0.0001	44 (9)	41 (8)	0.824
Implanted cardiac defibrillator	28 (4)	41 (5)	0.079	22 (4)	21 (4)	1.000
Medications						
Bucindolol	396 (49)	403 (52)	0.280	256 (51)	240 (48)	0.356
ACE inhibitors or angiotensin receptor blocker	776 (96)	744 (95)	0.604	475 (95)	479 (96)	0.652
Digitalis	732 (90)	708 (91)	0.851	451 (90)	454 (91)	0.830
Diuretics	749 (92)	727 (93)	0.640	460 (92)	462 (92)	0.905
Vasodilators	432 (53)	419 (54)	0.900	258 (52)	266 (53)	0.664
Anti-coagulants	488 (60)	465 (60)	0.773	299 (60)	300 (60)	1.000
Clinical features						
S3 gallop	340 (42)	310 (40)	0.354	203 (41)	194 (39)	0.616
Jugular venous distension	356 (44)	383 (49)	0.042	226 (45)	244 (49)	0.294
Leg edema	204 (25)	251 (32)	0.002	135 (27)	136 (27)	1.000
Pulmonary râles	121 (15)	134 (17)	0.228	81 (16)	83 (17)	0.933
NYHA class III	741 (91)	699 (89)	0.179	452 (90)	448 (90)	0.760
Heart rate, beats per minute	82 (±13)	78 (±12)	<0.0001	80 (±12)	80 (±11)	0.809
Blood pressure, mm Hg						
Systolic	117 (±18)	117 (±17)	0.632	117 (±18)	117 (±17)	0.708

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n (%) or mean (±SD)	Before matching			After matching		
	CABG		P value	CABG		P value
	No (n=811)	Yes (n=782)		No (n=500)	Yes (n=500)	
Diastolic	71 (±11)	69 (±10)	<0.0001	70 (±11)	70 (±10)	0.892
Chest X-ray						
Cardiothoracic ratio >0.5	585 (72)	591 (76)	0.118	356 (71)	364 (73)	0.628
Pulmonary edema	108 (13)	95 (12)	0.484	58 (12)	61 (12)	0.848
Electrocardiography						
Left bundle branch block	161 (20)	178 (23)	0.156	107 (21)	111 (22)	0.815
Right bundle branch block	69 (9)	76 (10)	0.401	38 (8)	40 (8)	0.904
Multi Gated Acquisition Scan						
Left ventricular ejection fraction, %	22 (±7)	23 (±7)	0.003	23 (±7)	23 (±7)	0.615
Right ventricular ejection fraction, %	35 (±12)	34 (±11)	0.458	35 (±12)	35 (±12)	0.996
Laboratory values						
Hemoglobin, g/dL	14 (±2)	14 (±2)	0.074	14 (±2)	14 (±2)	0.997
White blood cell, $10^3/\mu L$	7.5 (±2.4)	7.6 (±2.0)	0.680	7.6 (±2.5)	7.6 (±2.0)	0.761
Platelet, $10^3/\mu L$	221 (±65)	207 (±60)	<0.0001	214 (±59)	215 (±63)	0.733
Potassium, mEq/L	4.3 (±0.5)	4.4 (±0.5)	0.551	4.4 (±0.5)	4.4 (±0.5)	0.431
Creatinine, mg/dL	1.3 (±0.4)	1.3 (±0.4)	0.247	1.3 (±0.4)	1.3 (±0.4)	0.401
Total cholesterol, mg/dl	195 (±53)	191 (±44)	0.065	192 (±48)	195 (±46)	0.366
Norepinephrine, pg/ml	512 (±307)	543 (±314)	0.048	520 (±346)	531 (±305)	0.606

ACE = angiotensin converting enzyme; NYHA = New York Heart Association; PCI = Percutaneous Coronary Intervention; STEMI = ST-elevation during index acute myocardial infarction

Table 4

Association of a history of coronary artery bypass graft (CABG) and outcomes in a propensity-matched cohort of heart failure (HF) patients with coronary artery disease

Outcomes	Events (%)		HR (95% CI)	P value
	No-CABG (n=500)	CABG (n=500)		
All-cause mortality	194 (39%)	158 (32%)	0.77 (0.62–0.95)	0.015
Cardiovascular mortality	164 (33%)	138 (28%)	0.80 (0.64–1.00)	0.050
HF mortality	53 (11%)	47 (9%)	0.83 (0.56–1.24)	0.365
AMI mortality	1 (0.2%)	6 (1%)	6.28 (0.76–52.19)	0.089
Sudden cardiac death	92 (18%)	77 (15%)	0.80 (0.59–1.08)	0.147
All-cause hospitalization	315 (63%)	338 (68%)	1.13 (0.97–1.32)	0.120
HF hospitalization	207 (41%)	199 (40%)	0.93 (0.56–1.12)	0.434

AMI = acute myocardial infarction; CI = confidence internal; HF = heart failure; HR = hazard ratio