

Left and Codominant Coronary Artery Circulations Are Associated With Higher In-Hospital Mortality Among Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndromes

Report From the National Cardiovascular Database Cath Percutaneous Coronary Intervention (CathPCI) Registry

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Background—Left or codominant coronary arterial circulation may represent less well-balanced myocardial perfusion and thus confer worse prognosis in acute coronary syndrome, especially for culprit lesions arising from the left coronary artery.

Methods and Results—We related left and codominance, relative to right dominance, with in-hospital mortality in 207926 percutaneous coronary interventions (PCI) for acute coronary syndromes from July 1, 2009 through June 30, 2010 in the National Cardiovascular Data Registry Cath Percutaneous Coronary Intervention (CathPCI) Registry database version 4. Generalized estimating equations and logistic regression analyses were used in unadjusted and multivariable adjusted models. Models were adjusted using the validated National Cardiovascular Data Registry mortality risk model. We performed subgroup analyses and formally tested for effect modification by the epicardial coronary artery containing the culprit lesion. Left coronary dominance was associated with higher in-hospital mortality in unadjusted (odds ratio=1.29, 95% confidence interval [CI], 1.17–1.42) and adjusted models (1.19, 95% CI, 1.06–1.34). Codominance was associated with worsened mortality only in adjusted models (odds ratio=1.16, 95% CI, 1.01–1.34). Addition of coronary dominance to the National Cardiovascular Data Registry risk model did not materially change model discrimination or calibration. The odds of death for left versus right dominance among those with left circumflex or left main culprit lesions was 1.25 (95% CI, 1.02–1.53), for right coronary artery lesions was 1.19 (95% CI, 0.83–1.71), and for left anterior descending artery lesions was 1.09 (95% CI, 0.93–1.28). There was no statistical evidence for effect modification by culprit lesion vessel ($P=0.8$).

Conclusions—Left and codominance are associated with modestly increased post-percutaneous coronary intervention in-hospital mortality in patients with acute coronary syndrome. Confirmation of these findings with angiographic core laboratory verification of coronary dominance and longer term follow-up will be desirable. (*Circ Cardiovasc Qual Outcomes*. 2012;5:775-782.)

Key Words: coronary dominance ■ left dominance ■ acute coronary syndrome ■ risk stratification ■ mortality

In a right-dominant circulation, the right coronary artery (RCA) supplies the posterior portion of the interventricular septum and gives off the posterior descending artery. This contrasts with a left-dominant circulation, in which the left circumflex (LCX) artery supplies this territory. In a codominant circulation, supply of the posterior interventricular septum is

shared by the RCA and LCX. The prevalence of left dominance is ≈8%, whereas codominance has ≈7% population prevalence.¹

Left and codominance are generally considered to be normal variants with no particular prognostic significance. However, the relatively low prevalence of left and codominance may reflect a small biologic disadvantage relative to right dominance.

Received December 8, 2011; accepted September 22, 2012.

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This article was handled independently by Thomas M. Maddox, MD, MSc as a Guest Editor. The Editors had no role in the evaluation of the article or the decision about its acceptance.

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Circ Cardiovasc Qual Outcomes is available at <http://circoutcomes.ahajournals.org>

DOI: 10.1161/CIRCOUTCOMES.111.964593

WHAT IS KNOWN

- Two prior studies have suggested that left-dominant circulation may confer excess short- and long-term mortality after acute myocardial infarction.

WHAT THE STUDY ADDS

- Both left and right coronary artery circulation are associated with excess in-hospital death after percutaneous coronary intervention for acute coronary syndrome in the United States in American College of Cardiology's National Cardiovascular Data Registry Cath Percutaneous Coronary Intervention Registry.
- This association between left dominance and codominance and in-hospital death was independent of 23 demographic, clinical, and angiographic characteristics known to be associated with in-hospital death during percutaneous coronary intervention for acute coronary syndrome.
- The specific location of culprit vessel lesion did not modify the association between left and right dominance and in-hospital death in percutaneous coronary intervention for acute coronary syndrome.

Furthermore, it is possible that left and codominance may represent less well-balanced circulation with more myocardium at risk. This may be particularly true in patients with acute coronary syndrome (ACS) because of culprit lesions in the LCX and left main (LM) territories with either left-dominant or codominant systems (territories that supply the posterior descending artery).

A prior Canadian study of 27 289 patients, whose primary indication for cardiac catheterization was ACS, demonstrated that left dominance was associated with an increased hazard of death during a 3.5-year follow-up with a hazards ratio of 1.13 (95% confidence interval [CI], 1.00–1.28). In that study, codominance had a similar mortality as right dominance. However, there may have been insufficient statistical power to detect an association and to study associations within culprit lesion vessel anatomic subgroups. In a more recent Dutch study of 1425 men and women referred for coronary computed tomography angiography, during a 2-year follow-up period, nonfatal myocardial infarction and all-cause mortality were increased with left dominance relative to right (hazard ratio, 3.20; 95% CI, 1.67–6.13; $P < 0.001$).²

Thus, the aim of our study was to determine whether left and codominant coronary circulation, relative to right dominance, are associated with an increased risk of in-hospital mortality in ACS patients undergoing percutaneous coronary intervention (PCI). A secondary aim of our study was to assess whether a potentially increased risk of death was more pronounced in those with LCX or LM culprit lesions compared with RCA or left anterior descending artery (LAD) culprit lesions. We used a large and detailed US observational registry, the National Cardiovascular Data Registry (NCDR) and its Cath Percutaneous Coronary Intervention (CathPCI) Registry, to address these questions.

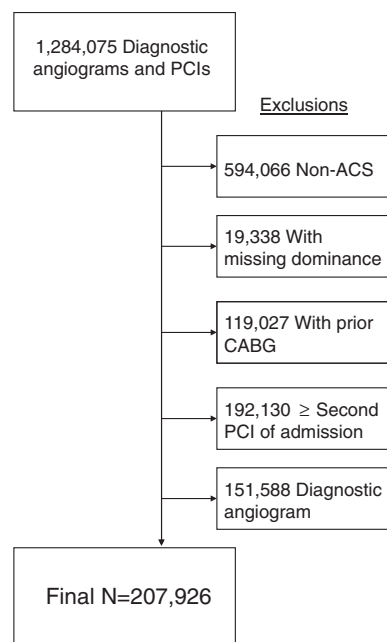


Figure 1. Creation of study sample. PCI indicates percutaneous coronary intervention; ACS, acute coronary syndrome; and CABG, coronary artery bypass graft.

Materials and Methods

NCDR Database

The NCDR CathPCI Registry is a predominantly US-based voluntary reporting system for diagnostic cardiac catheterization and PCI procedures. The registry is jointly sponsored by the American College of Cardiology and the Society for Cardiovascular Angiography and Interventions. Details of the NCDR have been previously described.^{3,4} Demographic, clinical, procedural, and institutional data elements from diagnostic catheterization and PCI procedures were collected at >1100 participating centers. Data collected using CathPCI Registry version 4 for procedures performed from July 1, 2009 through June 30, 2010 were used in this study; this version of the data set was selected because it was the most recent version and contained information on coronary artery dominance. ACS was defined as patients presenting with unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), or ST-segment elevation myocardial infarction (STEMI).

A comprehensive description of CathPCI Registry version 4 data elements and definitions is available at: http://www.ncdr.com/WebNCDR/NCDRDocuments/CathPCI_v4_CodersDictionary_4.4.pdf.

Exclusions

The creation of our study sample is shown in Figure 1. We chose to exclude those with prior coronary artery bypass graft to assess fully the effect of specific culprit vessel site without confounding by bypass grafts. We chose to use only the first PCI of each hospital admission as we defined culprit vessel lesion on the basis of the vessel that was addressed with the first PCI.

Exposure

Dominance was determined independently at each participating site. The NCDR definition of dominance was provided as follows: right dominance was if the posterior descending artery and posterolateral branch arises from the RCA, left dominance if the posterior descending artery and posterolateral branch arises from the LCX, and codominance if the RCA supplies the posterior descending artery and LCX supplies the posterolateral branch. The CathPCI Registry Clinical Support Team (comprising interventional cardiologists and clinically trained NCDR personnel) was available to answer general questions

Table 1. Demographic, Clinical History, Clinical Presentation, and Angiographic Characteristics of the Study Population

	Total (n=207926)	Right Dominance (n=169809)	Codominance (n=17143)	Left Dominance (n=20974)
Demographics				
Age, median (IQR)	62 (54–72)	62 (54–72)	62 (53–71)	62 (54–72)
Women	34.0	34.5	32.3	31.3
Race				
White	88.1	88.4	85.7	87.7
Black	8.3	8.1	10.5	8.8
Asian	2.2	2.2	2.3	2.1
American–Indian/Alaskan native	0.5	0.5	0.5	0.5
Native Hawaiian/Pacific islander	0.2	0.2	0.2	0.2
Hispanic	4.7	4.6	5.5	5.2
Post procedure LOS	2.0 (2.0–4.0)	2.0 (2.0–4.0)	3.0 (2.0–4.0)	3.0 (2.0–4.0)
Clinical history				
Body mass index, kg/m ² , median (IQR)	28.9 (25.6–33.2)	28.9 (25.6–33.1)	29.2 (25.8–33.6)	28.9 (25.5–33.1)
Hypertension	76.0	75.8	77	76.9
Diabetes mellitus	30.6	30.2	32.2	31.8
Peripheral vascular disease	8.6	8.6	8.5	9.3
Congestive heart failure	7.6	7.5	8.4	8.2
Cerebrovascular disease	9.3	9.3	9.8	9.5
Chronic lung disease	13.5	13.4	14.0	13.5
Hemodialysis	1.9	1.8	2.3	2.16
Glomerulofiltration rate, median (IQR)	75.9 (60.3–91.8)	75.9 (60.5–91.8)	76.3 (60.3–92.4)	75.2 (58.9–91.5)
Prior percutaneous intervention	28.9	29.2	27.4	27.8
Prior valve surgery	0.6	0.6	0.5	0.7
Clinical presentation factors				
Cardiogenic shock	3.2	3.1	3.4	3.6
ACS type				
Unstable angina	46.9	46.7	47.0	48.1
NSTEMI	26.0	26.0	25.5	26.3
STEMI	27.1	27.3	27.5	25.7
Preprocedure IABP placement	0.5	0.5	0.6	0.8
PCI status				
Elective	25.7	25.5	27.3	26.2
Urgent	44.9	45.1	42.7	45.4
Emergency	28.8	28.9	29.3	27.7
Salvage	0.5	0.5	0.6	0.7
NYHA functional class				
Class 1	10.9	10.9	10.7	10.8
Class 2	25.2	25.5	24.2	23.9
Class 3	33.4	33.1	35.2	34.1
Class 4	30.1	30.1	29.4	30.7
Angiographic characteristics				
Coronary lesion >50% with subacute thrombosis	0.2	0.2	0.2	0.2
Preprocedure TIMI flow				
0	24.1	24.3	24.4	22.6
1	9.8	9.4	11.2	11.8
2	18.7	18.3	20.4	20.2
3	47.2	47.8	43.7	45.3

(continued)

Table 1. (Continued)

	Total (n=207926)	Right Dominance (n=169809)	Codominance (n=17143)	Left Dominance (n=20974)
SCAI lesion class				
I	41.1	40.8	41.8	42.2
II	34.0	34.1	32.8	33.8
III	6.9	6.8	7.6	6.9
IV	17.9	18.1	17.6	16.9
Culprit vessel (PCI lesion location)				
LAD	41.4	34	42.7	51.8
LCX	21.3	18.9	27.8	35.4
RCA	36.7	40.5	29	12
LMCA	0.6	0.6	0.5	0.9

LOS indicates length of stay; IQR, interquartile range; ACS, acute coronary syndrome; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; IABP, intra-aortic balloon pump; PCI, percutaneous coronary intervention; NYHA, New York Heart Association; TIMI, Thrombolysis In Myocardial Infarction; SCAI, Society for Cardiovascular Angiography and Interventions; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; and LMCA, left main coronary artery.

Values are percentages or median with IQR as specified.

and provide guidance about dominance definitions to participating CathPCI Registry sites.

Covariates

We adjusted for 2 nested models. Model 2 included demographic, clinical history, clinical presentation, and angiographic characteristics and was a previously published risk model for mortality after PCI derived from 181 775 procedures in the CathPCI Registry (NCDR mortality risk model).⁵ Model 1 was a subset of Model 2, the NCDR mortality risk model and included all variables aside from those hypothesized to be in the causal pathway between coronary dominance and death in ACS. Specifically, Model 1 did not include the following clinical presentation/acuity variables: cardiogenic shock, STEMI, New York Heart Association Functional Class, preprocedure intra-aortic balloon pump, left ventricular ejection fraction, and PCI status (elective, urgent, emergent, salvage).

Statistical Analysis

We performed descriptive statistics on our study population stratified by coronary artery dominance. We used generalized estimating equations to account for clustering by hospital/site, along with logistic regression in unadjusted and multivariable adjusted models. Generalized estimating equations models used an exchangeable working correlation matrix. We considered 2 exposures separately: left- versus right-dominant (referent) and co- versus right-dominant (referent). The main outcome was in-hospital all-cause mortality. To assess the potential implications of coronary dominance on estimation of ACS mortality risk, we assessed the discrimination or C-statistic of the NCDR risk model with and without coronary dominance included, as well as assessment of model discrimination using a visual test of observed versus expected mortality along with the Hosmer-Lemeshow (HL) test.⁶ We performed multivariable adjusted models in subgroups according to culprit vessel site—the RCA, LAD, or LCX/LM. We did not assess LM independently because of small sample size and chose to combine it with LCX because we were interested in knowing whether or not culprit vessels that specifically supplied the PDA in a left dominant system accounted for potentially worse outcomes among persons with left dominance. We formally tested for modification of the effect of coronary artery dominance on mortality by the culprit vessel undergoing PCI for ACS. We repeated our primary analysis among subgroups of patients presenting with (1) STEMI and (2) NSTEMI. Given our finding that the

association between codominance and mortality became stronger and more statistically significant upon multivariable adjustment, we performed secondary analysis to further explore these findings. Specifically, we created univariate models of codominance and mortality adding 1 covariate from the NCDR model at a time to determine which particular variable most elevated the odds ratio (OR) upon adjustment. We then explored the distribution of dominance patterns with this covariate.

All analyses were performed using SAS version 9.2. Two-tailed 95% CIs and *P* values are given, with 2-sided *P* values <0.05 regarded as statistically significant.

The study was reviewed by the Committee on Clinical Investigations (the Institutional Review Board) of Beth Israel Deaconess Medical Center and deemed exempt from review (given previously abstracted data with no patient or personal health identifiers). The study was also approved by the Research and Publications Committee of the CathPCI Registry.

Results

Among 207 926 admissions for ACS, 34% of the patients were women, and the median age was 62 years (interquartile range 54–72). There were 82% right, 8% codominant, and 10% left dominant patients in the study sample. Descriptive demographic, clinical, and angiographic factors for the study population are shown in Table 1. There were small but statistically significant differences in several of these participants among the 3 coronary dominance groups. In particular, there was a higher prevalence of women and white patients in those with right as compared with co- or left dominance. There was a higher prevalence of black and Hispanic/Latino patients with codominance. The median post-PCI length of stay was higher among those with left dominance (3 days, interquartile range 2–4) and codominance (3 days, interquartile range 2–4) versus right dominance (2 days, interquartile range 2–4). There was a higher prevalence of hypertension, diabetes mellitus, peripheral vascular disease, and end-stage renal disease requiring hemodialysis among those with codominance and those with left dominance. The prevalence of shock and preprocedure intra-aortic balloon pump was higher in those with left dominance. The prevalence of slow or TIMI flow <

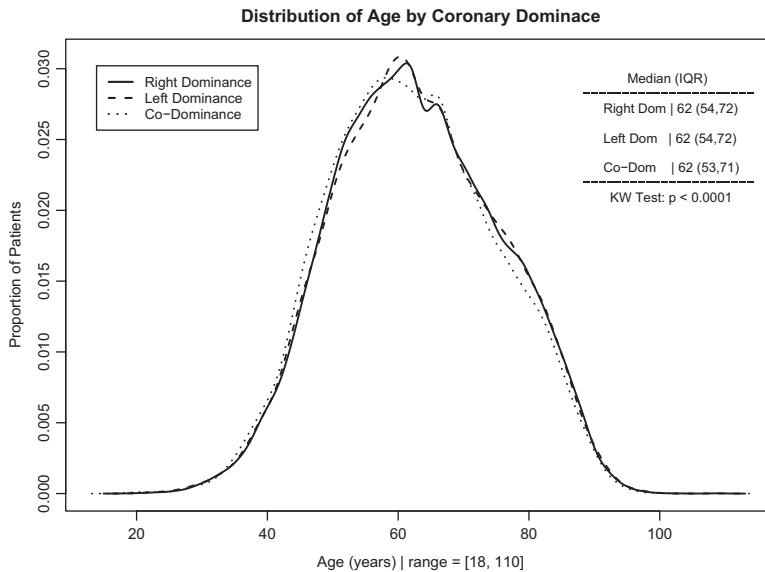


Figure 2. Age distribution by coronary dominance.

grade 3 was higher among those with left dominance and with codominance. All above comparisons had $P < 0.01$. Persons with codominant circulation tended to be younger than those with right or left dominance (Figure 2).

Primary Analysis

Left coronary dominance was associated with higher in-hospital mortality in unadjusted (OR=1.29, 95% CI, 1.17–1.42) and in both Model 1 and Model 2 (Table 2). Codominance was not significantly associated with mortality in unadjusted models (OR=1.11, 95% CI, 0.99–1.24) but was significantly related to mortality in adjusted models (Model 2 OR=1.16, 95% CI, 1.01–1.34). The C-statistic of the risk model for mortality after PCI without coronary dominance was identical to the C-statistic for the risk model which included coronary dominance (0.921). The inclusion of coronary dominance did not materially improve calibration of the risk model for in-hospital mortality in ACS (HL statistic < 0.0001 for models with and without coronary dominance, observed rates of in-hospital mortality closely mirrored expected rates in models with and without coronary dominance Figure 3A and 3B).

Secondary Analysis

The adjusted OR for post-PCI in-hospital mortality was highest for left dominance versus right among those with LCX or LM culprit lesions (OR=1.25, 95% CI, 1.02–1.53) compared with RCA (OR=1.19, 95% CI, 0.83–1.71) or LAD lesions (1.09, 95% CI, 0.93–1.28) (Table 3). However, there was no statistical evidence for effect modification by culprit vessel among those with left dominant circulations as compared with right ($P=0.8$). The adjusted ORs for post-PCI in-hospital mortality were similar for codominance versus right dominance among all culprit vessel lesion subgroups: RCA, LAD and LCX, or LM, and none were statistically significant (Table 3). The adjusted odds death for left dominance or codominance among those with STEMI ($n=56410$) were not significantly different (left versus right dominance: OR=1.12 [0.96–1.30]; co- versus right dominance: OR= 1.10 [0.91–1.32]). The adjusted odds of post-PCI mortality for left versus right dominance among those with NSTEMI ($n=54073$) was significantly different (left versus right dominance: OR= 1.29 [1.05–1.60]), whereas co- versus right dominance was not (OR= 1.23 [0.93–1.62]).

Table 2. Main Results: Dominance and Post-PCI in-Hospital Mortality in Acute Coronary Syndrome

	Unadjusted GEE OR		Model 1 GEE OR: NCDR Mortality Risk Model Without Clinical Presentation Variables*		Model 2 GEE OR: NCDR Mortality Risk Model†	
	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value
Right dominant (reference group)	1.0	...	1.0	...	1.0	...
Codominant	1.11 (0.99–1.24)	0.05	1.15 (1.02–1.31)	0.03	1.16 (1.01–1.34)	0.04
Left Dominant	1.29 (1.17–1.42)	<0.001	1.21 (1.09–1.35)	<0.001	1.19 (1.06–1.34)	0.003

PCI indicates percutaneous coronary intervention; GEE, generalized estimating equations; OR, odds ratio; CI, confidence interval; and NCDR, National Cardiovascular Data Registry.

*Model 1: Covariates include age, male, white race, glomerular filtration rate, body mass index, diabetes mellitus, peripheral vascular disease, congestive heart failure, prior valvular surgery, cerebrovascular disease, lung disease, prior PCI, hemodialysis, cardiogenic shock, coronary lesion $>50\%$ with subacute thrombosis, preprocedural Thrombolysis In Myocardial Infarction (TIMI) flow, highest lesion severity using the Society for Cardiovascular Angiography and Interventions (SCAI) lesion class, and lesion location (eg, proximal LAD).

†Model 2: Fully adjusted NCDR Mortality Risk Model. [Model 1 covariates+clinical presentation covariates (cardiogenic shock, ST-segment elevation myocardial infarction, New York Heart Association Functional Class, preprocedural intra-aortic balloon pump, left ventricular ejection fraction, PCI status [elective, urgent, emergent, and salvage]).

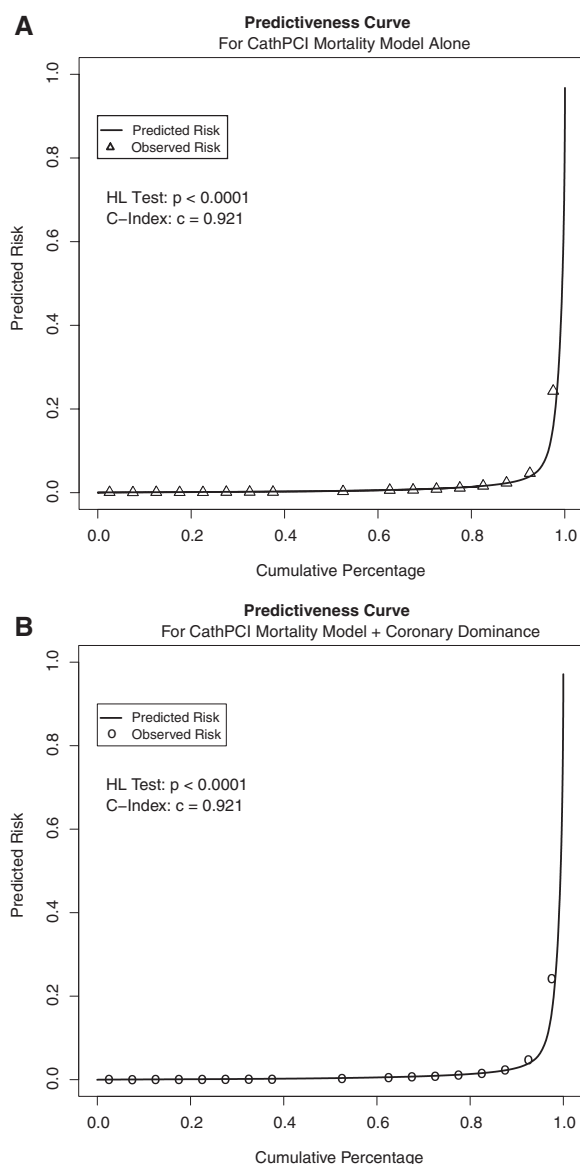


Figure 3. **A**, Predictiveness curve for National Cardiovascular Data Registry (NCDR) model alone. **B**, Predictiveness curve for NCDR model + coronary dominance. HL test indicates Hosmer-Lemeshow test.

The covariate that most greatly increased the OR for codominance relative to right dominance in multivariable models compared with unadjusted models was age. The age distribution of persons with codominance was younger than those with right dominance (Figure 2) and age is known to be positively associated with mortality in the NCDR mortality risk model, thus resulting in an increased multivariable adjusted OR for codominance and mortality as compared with the unadjusted model.

Discussion

Summary of Findings

Left coronary dominance was associated with a 1.19-fold increased odds of in-hospital mortality after PCI for ACS, and

codominance was associated with a 1.16-fold increased odds of death after PCI for ACS after accounting for 23 demographic, clinical, and angiographic characteristics. Inclusion of coronary dominance into the risk model for mortality after PCI for ACS did not materially change model discrimination or calibration.

The adjusted OR for post-PCI in-hospital mortality was highest for left dominance versus right among those with LCX or LM culprit lesions as compared with RCA or LAD lesions. There was no evidence for statistically significant effect modification by culprit lesion site ($P=0.8$). However, our study was likely statistically underpowered to detect effect modification.

Prior Studies of Coronary Dominance and Mortality in ACS

In the prior Canadian Assert Study of 27289 persons undergoing PCI for ACS, left dominance as compared with right was associated with increased risk of death during a mean 3.5 years of follow-up multivariable adjusted hazard ratio=1.13 (1.00–1.28).⁷ This hazard of death was similar in magnitude to our increased multivariable increased odds of in-hospital death OR=1.19 (1.06–1.34). Unlike the earlier study, we excluded those with prior coronary artery bypass graft and limited our study to only those who underwent PCI. In the earlier study, codominance was not associated with an increased risk of death: hazard ratio=1.03 (0.90–1.18), however, this study may have been underpowered to detect an association given the relatively low prevalence of codominance.

Among the subset of participants in the Assert study who received intervention to coronary artery branches that supplied the posterior descending artery, there was an elevated hazard of death that was statistically significant, hazard ratio=1.60 (1.16–2.20), compared with those who had lesions outside the posterior descending artery dilated or were medically treated. Our finding that the subgroup of persons with left dominance and LM/LCX PCI had the highest odds of death is consistent with this finding. However, we extended the earlier literature by demonstrating that the odds of death given left dominance was highest in those with LM or LCX culprit lesions as compared with RCA or LAD lesions. We may have been underpowered to detect effect modification by anatomic lesion.

Our analysis among subgroups of STEMI and NSTEMI showed fairly similar odds of death in ACS for left and codominant patients versus right-dominant patients. Lack of significant differences in the post-PCI mortality of left and codominance among STEMI and codominance in NSTEMI was likely because of reduction of statistical power in these smaller subgroup analyses.

Inclusion of dominance into the existing risk model for mortality after PCI for ACS did not materially change model discrimination or calibration. The finding that inclusion of dominance did not affect discrimination was not surprising given that (1) the existing model that did not include coronary dominance already contained >20 covariates, 2) the existing model had a relatively high C-statistic of 0.92, and 3) given that coronary dominance had a relatively modest effect size in relation to mortality.^{8–10} The HL test being significant even when plotting of calibration curves indicate that there is

Table 3. Coronary Dominance and In-Hospital Mortality by Site of Culprit Lesion During Percutaneous Coronary Intervention for Acute Coronary Syndrome

	% With RCA Culprit Lesion (Within Dominance Category)	Right Coronary Artery	% With LAD Culprit Lesion (Within Dominance Category)	Left Anterior Descending Artery	% With LM or LCX Culprit Lesion (Within Dominance Category)	LM/LCX Artery
Right dominant (reference group)	35.8	1.0	16.8	1.0	LM=0.6, LCX=46.8	1.0
Codominant	35.7	1.17 (0.90–1.52)	18.6	1.13 (0.93–1.36)	LM=0.5, LCX=45.2	1.09 (0.83–1.43)
Left dominant	34.8	1.19 (0.83–1.71)	23.4	1.09 (0.93–1.28)	LM=0.9, LCX=40.9	1.25 (1.02–1.53)**

PCI indicates percutaneous coronary intervention; RCA, right coronary artery; LAD, left anterior descending artery; LM, left main; and LCX, left circumflex.

Odds ratios are for mortality relative to right dominance within each culprit vessel subgroup.

Adjusted odds ratios and 95% confidence intervals are presented.

Covariates include age, male, white race, glomerular filtration rate, body mass index, diabetes mellitus, peripheral vascular disease, congestive heart failure, prior valvular surgery, cerebrovascular disease, lung disease, prior PCI, hemodialysis, cardiogenic shock, ST-segment elevation myocardial infarction, New York Heart Association Functional Class, preprocedure intra-aortic balloon pump, left ventricular ejection fraction, PCI status (elective, urgent, emergent, and salvage), coronary lesion >50% with subacute thrombosis, preprocedure Thrombolysis In Myocardial Infarction (TIMI) flow, highest lesion severity using the Society for Cardiovascular Angiography and Interventions (SCAI) lesion class, and lesion location (eg, proximal LAD).

** $P=0.03$, remainder of comparisons within culprit vessel subgroups were not statistically significant.

P value for interaction by culprit vessel lesion site among left dominant versus right= 0.8.

good calibration is also not surprising in this setting—it has been shown that the HL test is very sensitive to sample size. Specifically, when the sample size is large as in our study, the HL will almost always lead to a significant test regardless of good model fit.¹¹

Strengths and Limitations

The strengths of our study were the use of a large, nationwide sample not specifically selected for either our exposure (coronary dominance) or dependent variable (in-hospital mortality). We were able to control for a multitude of potential confounding factors given the richness of the NCDR database. Several limitations should also be acknowledged. Coronary dominance was site-reported at each participating hospital and was not validated prospectively by an angiographic core laboratory. This could have led to some degree of exposure misclassification. Because of the fact that left dominant and codominant patients had slightly higher rates of cardiac comorbidities, it is possible that residual confounding was present and explained the association between dominance and in-hospital mortality. Our definition of culprit vessel was based on the first lesion treated by PCI rather than that identified specifically by the interventional cardiologist. For this study, we were only able to study in-hospital mortality and not long-term mortality, which also may be related to coronary artery dominance. Our study was likely underpowered to detect statistical effect modification of culprit lesion site given 3 dominance categories and 3 potential coronary artery territories for which we considered potential interaction. Finally, we did not adjust for testing multiple comparisons.

Future Directions

Analyses of dominance and death after PCI for ACS using angiographic core laboratory verification of dominance are important future opportunities for investigation. Well-powered investigations regarding long-term mortality given codominance and left dominance would be interesting areas for further study.

Conclusions

Left and codominant coronary artery circulation confer modestly increased risk of in-hospital mortality after PCI for ACS, particularly in lesions in the LM/LCX territory.

Acknowledgment

We thank Dr Todd Seto for his thoughtful review of the article.

Disclosures

None.

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