



Universiteit
Leiden
The Netherlands

Prognostic value of coronary anatomy and myocardial innervation imaging in cardiac disease

Veltman, Caroline Emma

Citation

Veltman, C. E. (2016, March 10). *Prognostic value of coronary anatomy and myocardial innervation imaging in cardiac disease*. Retrieved from <https://hdl.handle.net/1887/38453>

Version: Corrected Publisher's Version

License: [Licence agreement concerning inclusion of doctoral thesis in the Institutional Repository of the University of Leiden](#)

Downloaded from: <https://hdl.handle.net/1887/38453>

Note: To cite this publication please use the final published version (if applicable).

Cover Page



Universiteit Leiden



The handle <http://hdl.handle.net/1887/38453> holds various files of this Leiden University dissertation

Author: Veltman, Caroline Emma

Title: Prognostic value of coronary anatomy and myocardial innervation imaging in cardiac disease

Issue Date: 2016-03-10

Chapter 3

Influence of coronary vessel dominance on short-and long-term outcome in patients after ST-segment elevation myocardial infarction.

Veltman CE, van der Hoeven BL, Hoogslag GE, Boden H, Kharbanda RK, de Graaf MA, Delgado V, van Zwet EW, Schalij MJ, Bax JJ, Scholte AJ.

European Heart Journal 2015; 36(17): 1023-30.

ABSTRACT

Aims:

Prognostic importance of coronary vessel dominance in patients with ST-elevation myocardial infarction (STEMI) remains uncertain. The aim of this study was to assess influence of coronary vessel dominance on the short- and long-term outcome after STEMI.

Methods and Results:

Coronary angiographic images of consecutive patients presenting with first STEMI were retrospectively reviewed to assess coronary vessel dominance. Patients were followed after STEMI during a median period of 48 (IQR 38-61) months for the occurrence of all-cause mortality and the composite of reinfarction and cardiac death. The population comprised 1131 patients of which 971 (86%) patients had a right dominant system, 102 (9%) a left dominant system and 58 (5%) a balanced system. After 5 years of follow-up the cumulative incidence of all-cause mortality was significantly higher in patients with a left dominant system, compared to a right dominant and balanced system (log-rank $P=0.013$). Moreover, a left dominant system was an independent predictor for 30-day mortality (OR 2.51, 95% CI 1.11-5.67, $P=0.027$) and the composite of reinfarction and cardiac death within 30-days after STEMI (OR 2.25, 95%CI 1.09-4.61, $P=0.028$). In patients surviving first 30-days post-STEMI, coronary vessel dominance had no influence on long-term outcome.

Conclusions:

A left dominant coronary artery system is associated with a significantly increased risk of 30-day mortality and early reinfarction after STEMI. After surviving the first 30-days post-STEMI, coronary vessel dominance had no influence on long-term outcome.

INTRODUCTION

Coronary vessel dominance, defined by the coronary artery that supplies the posterior descending artery (PDA) and posterolateral branches, influences the relative contribution of the different coronary arteries to the total left ventricular blood flow.¹ A right dominant system has a reported prevalence of 87-89%.²⁻⁴ In patients with a left dominant system approximately 60% of the left ventricular myocardium is supplied by the PDA and posterolateral branches originating from the LCx.⁵ This less well-balanced coronary circulation might have a negative influence on prognosis of patients with coronary artery disease (CAD). Currently, the prognostic importance of coronary vessel dominance in patients presenting with first ST-segment elevation myocardial infarction (STEMI) remains uncertain. The aim of this study was to assess the influence of coronary vessel dominance on the 30-day mortality and long-term outcome after STEMI.

METHODS

Patients

The population consisted of consecutive patients presenting with first STEMI at the Leiden University Medical Center between 2004 and 2008. Patients with previous myocardial infarction, previous percutaneous coronary intervention (PCI) and/or previous coronary artery bypass grafting were excluded. The diagnosis STEMI was defined based on criteria of typical chest pain, elevated cardiac enzyme levels, and typical changes on the electrocardiogram.⁶ All patients were treated according to the institutional MISSION! Protocol,⁷ based upon the European Society of Cardiology and American College of Cardiology/American Heart Association guidelines.^{8,9} The protocol includes primary PCI, optimal medical therapy initiated early during hospitalization and two-dimensional echocardiography performed within 48 hours of admission to assess residual left ventricular function.¹⁰

Demographic, clinical, angiographic and echocardiographic data were prospectively collected in the departmental Cardiology Information System (EPD-Vision®, Leiden University Medical Center, Leiden, The Netherlands) and retrospectively analyzed. Patients with uninterpretable coronary angiographic images for coronary vessel dominance were excluded from analysis.

Primary percutaneous coronary intervention and angiographic data analysis

Images of the coronary angiography and PCI were obtained using standardized angiographic projections according to the guidelines of the American College of Cardiology/American Heart Association,¹¹ and stored digitally. All images were retrospectively reviewed by two

experienced observers. During the analysis coronary vessel dominance, the culprit vessel and culprit lesion and severity of coronary artery disease (CAD) were recorded. A coronary artery system was classified as right dominant if the PDA and posterolateral branch originated from the RCA, left dominant if the PDA and the posterolateral branch originated from the LCx artery, and balanced if the PDA originated from the RCA in combination with posterolateral branches originating from the LCx artery.^{4,12} The culprit vessel was determined on the coronary artery territory subtended by the regions of acute electrocardiographic changes. If the culprit vessel had more than two lesions, the most severe proximal stenosis or a stenosis identified with thrombus was considered the culprit lesion.¹³ The extent of CAD was expressed as the presence of one-, two- or three-vessel disease (stenosis causing $\geq 50\%$ luminal narrowing). Complete revascularization was defined as treating all present significant coronary artery stenosis ($\geq 70\%$ luminal narrowing) during primary PCI or during secondary revascularization before discharge.

Follow-up and endpoint definitions

After discharge, patients were followed according to the institutional STEMI protocol.^{7,14} By reviewing medical records, retrieval of survival status through municipal civil registries and telephone interviews, data on the occurrence of adverse events after discharge were collected. The adverse events included nonfatal reinfarction and all-cause mortality during follow-up. Nonfatal reinfarction was defined based on criteria of typical chest pain, elevated cardiac enzyme levels and typical changes on the electrocardiogram.⁶ The primary cause of death was recorded and all deaths were classified as cardiac unless unequivocally proven non-cardiac.

The primary endpoint was all-cause mortality. The secondary endpoint was the composite of reinfarction and cardiac death. Short- and long-term outcome after STEMI were investigated using these endpoints. Short-term outcome was defined as within the first 30 days post-STEMI.

Follow-up was updated regularly until 2012. Patients with < 2 years of follow-up after STEMI, but who were alive according to the municipal civil registries, were considered lost to follow-up. Data of these patients were included up to the last date of follow-up.

Statistical analysis

Continuous variables are presented as mean \pm standard deviation or as median and interquartile range. Categorical variables are presented as number and percentages. Differences in baseline characteristics between the three coronary vessel dominance groups were evaluated with one-way analysis of variance (ANOVA) and chi-squared tests, where appropriate. To estimate the cumulative incidences of the primary and secondary endpoint during long-term follow-up Kaplan-Meier analyses stratified for coronary vessel dominance was performed. In addition, to investigate the difference in short-term outcome between

the different coronary vessel dominance groups multivariate binary logistic regression analysis was performed with the endpoints 30-day mortality and the occurrence of reinfarction within 30-days post-STEMI. The number of covariables included in the analysis was adjusted to the number of events and complete availability, resulting in inclusion of the clinical risk factors age, gender, diabetes, hypertension and smoking. The results of the binary logistic regression analysis were reported as adjusted odds ratios (OR's) with 95% confidence intervals (CIs). Subsequently, the influence of coronary vessel dominance on long-term outcome was evaluated using Cox regression analysis in a subgroup of patients surviving the first 30 days after STEMI. In the multivariate Cox regression model the covariables were selected based upon univariate significance of P-value ≤ 0.20 and/or significant difference in baseline distribution among the vessel dominance groups, resulting in inclusion age, gender, diabetes, hypertension, smoking, Killip class during STEMI, three-vessel disease, peak cardiac troponin T, glomerular filtration rate (eGFR), left-ventricular ejection fraction and finally reinfarction within the first 30 days post-STEMI was corrected for to adjust for its effect on long-term outcome in the survivors of the 30 days post-STEMI. The results of the Cox regression analysis were reported as adjusted hazard ratios (HRs) with 95% CIs. Statistical analysis was performed using SPSS software (version 20.0, SPSS, Inc., Chicago, IL, USA). A P-value of <0.05 , by a two-sided test, was considered statistically significant.

RESULTS

Patient and angiographic characteristics

A total of 1156 consecutive patients were included. Twentyfive patients (2%) were excluded from the analysis due to uninterpretable coronary vessel dominance. The final population comprised 1131 patients of which 971 (86%) patients had a right dominant system, 102 (9%) patients had a left dominant system and 58 (5%) patients had a balanced system. The patients characteristics and angiographic data are presented in Table 1. Overall baseline characteristics were similar between coronary vessel dominance groups. However, patients with a balanced system tended to be younger ($p=0.026$) and patients with a left dominant system more frequently had hypertension ($p=0.036$). The RCA was most often the culprit vessel in patients with a right dominant system, whereas in patients with a left dominant or balanced system the LAD artery was most often the culprit vessel. Moreover, a relatively high incidence of the LCx as culprit vessel was observed in patients with a left dominant system ($P<0.001$). Importantly, the majority of patients presented with single vessel disease. Complete revascularization was achieved in 789 patients (70%). A trend towards a slightly lower left ventricular ejection fraction at discharge was observed in patients with a left dominant system.

Table 1. Patient characteristics and angiographic data

| | Total n=1131 | Right Dominant n=971 | Left Dominant n=102 | Balanced n=58 | p-value |
|--|-----------------|----------------------------|---------------------------|------------------|---------|
| Gender (male) | 850 (75%) | 731 (75%) | 73 (72%) | 46 (79%) | 0.536 |
| Age (years) | 61±12 | 61±12 | 63±13 | 58±11 | 0.026* |
| Diabetes | 117 (10%) | 96 (10%) | 12 (12%) | 9 (16%) | 0.351 |
| Hypercholesterolemia | 191 (17%) | 160 (17%) | 22 (22%) | 9 (16%) | 0.414 |
| Hypertension | 361 (32%) | 304 (31%) | 43 (42%) | 14 (24%) | 0.036 |
| Current smoking | 549 (49%) | 468 (48%) | 48 (47%) | 33 (57%) | 0.426 |
| Presenting in Killip class ≥2 during STEMI | 66 (6%) | 54 (6%) | 7 (7%) | 5 (9%) | 0.574 |
| Culprit vessel | | | | | |
| LM | 7 (0.6%) | 6 (0.6%) | 1 (1%) | 0 (0%) | 0.749 |
| RCA | 438 (39%) | 421 (43%) | 3 (3%) | 14 (24%) | <0.001 |
| LAD | 512 (45%) | 418 (43%) | 62 (61%) | 32 (55%) | 0.001 |
| LCx | 174 (15%) | 126 (13%) | 36 (35%) | 12 (21%) | <0.001 |
| Extent of CAD | | | | | |
| One-vessel disease | 606 (54%) | 515 (53%) | 59 (58%) | 32 (55%) | 0.632 |
| Two-vessel disease | 350 (31%) | 305 (31%) | 31 (30%) | 14 (24%) | 0.504 |
| Three-vessel disease | 175 (16%) | 151 (16%) | 12 (12%) | 12 (21%) | 0.319 |
| Complete revascularization | 789 (70%) | 684 (70%) | 69 (68%) | 36 (62%) | 0.358 |
| eGFR(mL/min/1.73m ²) | 100±34 | 100±35 | 97±34 | 106±28 | 0.220 |
| eGFR ≤60 mL/min/1.73m ² | 131 (12%) | 115 (12%) | 13 (13%) | 3 (5%) | 0.276 |
| Peak cardiac troponin T level(µg/L) | 6.1±6.5 | 6.1±6.4 | 6.4±6.8 | 6.5±6.7 | 0.543 |
| Peak cardiac troponin T level ≥3.5 µg/L | 627 (56%) | 539 (56%) | 55 (54%) | 33 (57%) | 0.967 |
| LV ejection fraction at discharge(%) | 47±9 | 47±9 | 45±9 | 49±10 | 0.053 |
| LV ejection fraction≤40% | 235 (22%) | 200 (22%) | 22 (24%) | 13 (24%) | 0.853 |

* left dominant versus balanced coronary artery systems.

Follow-up

Follow-up was completed in 1076 patients (95%), with 55 patients (5%) lost to follow-up. The median follow-up was 48 months (IQR 38-61). The primary endpoint occurred in 119 patients (11%): 77 patients (7%) died from cardiac death and 42 patients (4%) died from non-cardiac causes. Furthermore, 92 patients (8%) had reinfarction, of which 9 were fatal. The secondary endpoint occurred in 154 patients (14%).

The cumulative incidence of both the primary and secondary endpoint after 5 years post-STEMI was higher in patients with a left dominant system (Figure 1).

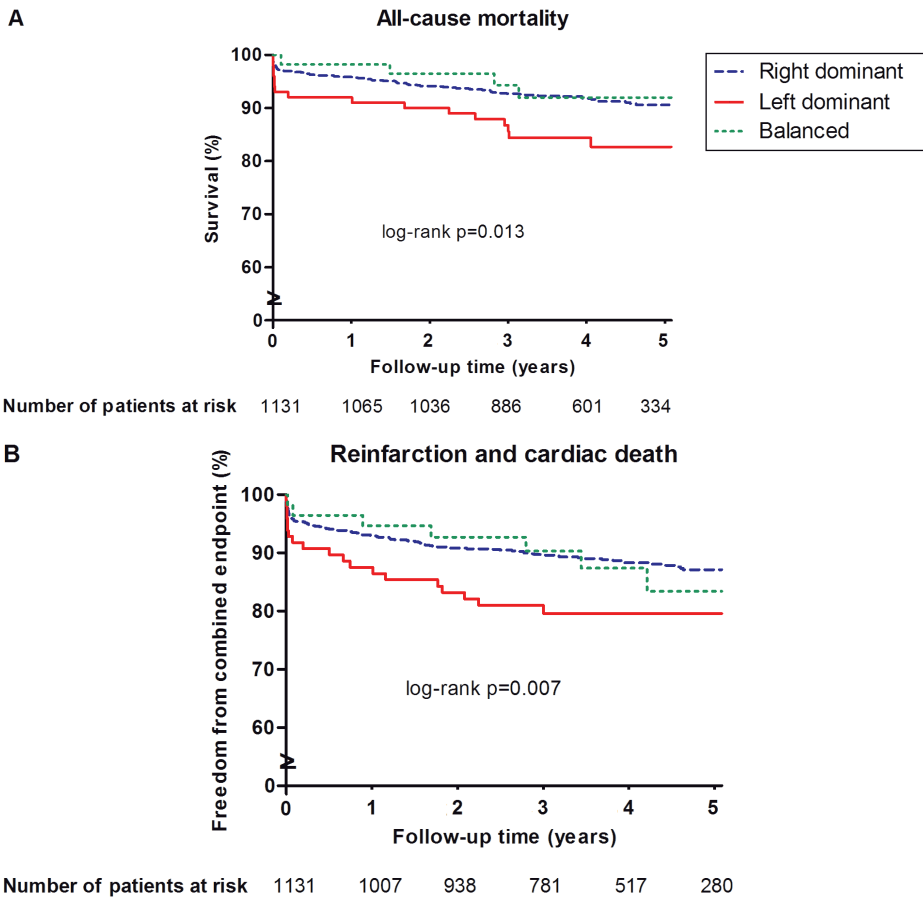


Figure 1. Kaplan-Meier curves for the primary and secondary endpoint in the total patient population stratified according to coronary vessel dominance.

Patients with left dominant coronary artery system had statistically significant worse outcome with higher cumulative incidence of all-cause mortality (A, log-rank $P=0.013$) and higher cumulative incidence of the composite of reinfarction and cardiac death (B, log-rank $P=0.007$) during long-term follow-up after STEMI.

Coronary vessel dominance and 30-day adverse outcome

Among 119 patients who died, 45 patients (4%) died within the first 30 days after STEMI of which 25 patients (56%) died within 24 hours after STEMI. The majority was classified as cardiac death (43 of the 45 patients: cardiogenic shock (20 patients), ventricular fibrillation (3 patients), left ventricular free wall rupture (4 patients), heart failure (9 patients), reinfarction due to in-stent restenosis (4 patients) or undetermined (3 patients). Two patients were classified as non-cardiac death as they died due to an infection after coronary bypass surgery and a gastro-intestinal bleeding after PCI. Furthermore, 24 patients (2%) had reinfarction within the first 30 days after STEMI. The secondary endpoint was reached in 63 patients (5.5%) at 30-days post-STEMI.

Patients who died within 30 day post-STEMI were more likely to be female, of advanced age and less likely to smoke (Table 2). Furthermore, these patients presented with a higher Killip class during STEMI and were more likely to have three-vessel disease. Importantly, the culprit lesion was more frequently located in the left main (LM) artery or the dominant LCx artery compared to patients who survived the first 30 days post-STEMI. Finally, a higher prevalence of a left dominant system was observed in patients who died within the first 30 days post-STEMI (Table 2).

Multivariate logistic regression analyses demonstrated that coronary vessel dominance and age were independent predictors of both the primary and secondary endpoint within 30 days post-STEMI (Table 3). A left dominant system was associated with an OR of 2.51 (95%CI 1.11-5.67) for 30-day mortality and with an OR of 2.25 (95%CI 1.09-4.61) for reinfarction or cardiac death within 30 days post-STEMI.

Table 2. Differences in baseline characteristics between patients who died within the first 30 days post-STEMI and survivors of the first 30 days post-STEMI

| | Deceased within the first 30 days post-STEMI n=45 | Survivors of the first 30 days post-STEMI n=1086 | p-value |
|----------------------------------|--|---|---------|
| Gender(female) | 18 (40%) | 263 (24%) | 0.016 |
| Age(years) | 73±12 | 60±12 | <0.001 |
| Diabetes | 7 (16%) | 110 (10%) | 0.194 |
| Hypercholesterolemia | 8 (19%) | 183 (17%) | 0.764 |
| Hypertension | 15 (35%) | 346 (32%) | 0.677 |
| Current smoking | 12 (29%) | 537 (49%) | 0.011 |
| Killip class ≥2 during STEMI | 27 (61%) | 39 (4%) | <0.001 |
| Extent of CAD | | | |
| One-vessel disease | 12 (27%) | 594 (55%) | <0.001 |
| Two-vessel disease | 15 (33%) | 335 (31%) | 0.724 |
| Three-vessel disease | 18 (40%) | 157 (15%) | <0.001 |
| Culprit vessel | | | |
| LM | 5 (11%) | 2 (0.2%) | <0.001 |
| RCA | 14 (31%) | 424 (39%) | 0.285 |
| LAD | 19 (42%) | 493 (45%) | 0.675 |
| LCx | 7 (16%) | 167 (15%) | 0.974 |
| Dominant LCx | 5 (11%) | 31 (3%) | 0.002 |
| Coronary vessel dominance | | | |
| Right dominant | 35 (78%) | 936 (86%) | 0.113 |
| Left dominant | 9 (20%) | 93 (9%) | 0.009 |
| Balanced | 1 (2%) | 57 (5%) | 0.367 |

CAD=coronary artery disease; LAD=left anterior descending; LCx=left circumflex; LM=left main ; RCA=right coronary; STEMI=ST-segment elevation myocardial infarction.

Table 3. Multivariate logistic regression analysis to assess independent correlates of 30-day adverse events post-STEMI

| | Primary endpoint All-cause Mortality | | | Secondary endpoint Reinfarction or cardiac death | | |
|----------------------------------|---|-------------|---------|---|-------------|---------|
| | OR | 95%CI | p-value | OR | 95%CI | p-value |
| Age | 1.10 | (1.06-1.14) | <0.001 | 1.06 | (1.03-1.09) | 0.001 |
| Gender(male) | 1.06 | (0.52-2.17) | 0.871 | 1.30 | (0.69-2.44) | 0.419 |
| Diabetes | 1.72 | (0.63-4.70) | 0.520 | 1.93 | (0.96-3.88) | 0.067 |
| Hypertension | 0.59 | (0.26-1.32) | 0.453 | 0.78 | (0.44-1.39) | 0.395 |
| Current smoking | 0.89 | (0.37-2.12) | 0.799 | 0.72 | (0.39-1.33) | 0.288 |
| Coronary Vessel Dominance | | | | | | |
| Right | Reference(1.0) | | | Reference(1.0) | | |
| Left | 2.51 | (1.11-5.67) | 0.027 | 2.25 | (1.09-4.61) | 0.028 |
| Balanced | 0.73 | (0.10-5.64) | 0.761 | 1.33 | (0.39-4.53) | 0.650 |

CI=confidence interval; OR=odds ratio; STEMI=ST-segment elevation myocardial infarction.

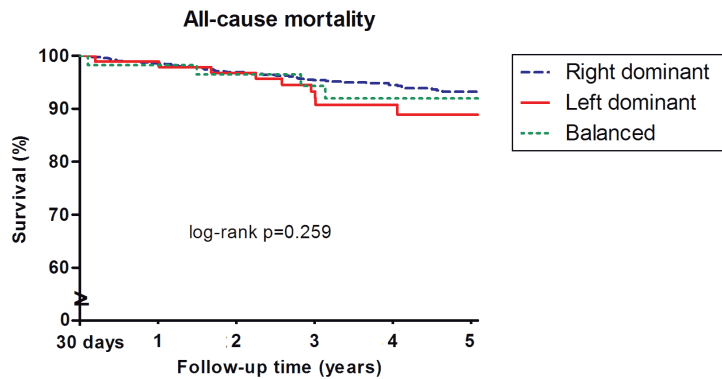
Coronary vessel dominance and long-term outcome in survivors of the first 30 days post-STEMI

A total of 1086 patients survived the first 30 days post-STEMI. The primary endpoint was reached in 74 patients (7%) and the secondary endpoint was reached in 98 patients (9%) during long-term follow-up. No differences in the cumulative incidence of the primary endpoint were observed between patients (log-rank $P=0.259$; Figure 2A). Moreover, no differences in cumulative incidences for the secondary endpoint were observed (log-rank $p=0.117$; Figure 2B). Multivariate Cox regression analysis showed that coronary vessel dominance was not associated with the occurrence of either the primary or secondary endpoint during long-term follow-up in patients who survived the first 30 days post-STEMI (Table 4). Age, gender, three-vessel disease and peak cardiac troponin T levels were independently associated with the primary endpoint of all-cause mortality in this subgroup of patients. Independent predictors of the secondary endpoint were gender, Killip class during STEMI, three-vessel disease, left ventricular ejection fraction at discharge and a reinfarction within the first 30 days post-STEMI (Table 4).

DISCUSSION

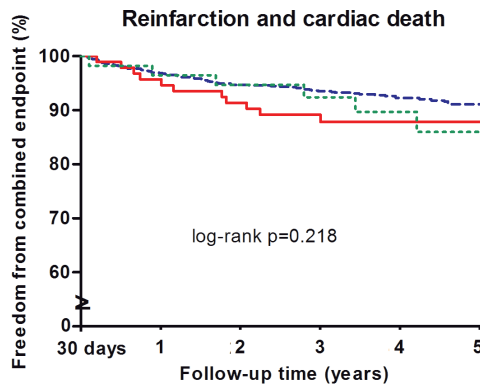
The main finding of this study is that a left dominant system is associated with an increased risk of all-cause mortality and reinfarction or cardiac death post-STEMI. Importantly, a left dominant system had a more than 2-fold increased risk of mortality within the first 30 days post-STEMI, whereas in patients surviving the first 30 days post-STEMI cumulative

A



Number of patients at risk 1086 1065 1036 886 601 334

B



Number of patients at risk 1086 1041 995 839 565 313

Figure 2. Kaplan-Meier curves for the primary and secondary endpoint in patients who survived the first 30 days after STEMI stratified according to coronary vessel dominance.

The survivors of the first 30 days post-STEMI had similar long-term outcome showing no statistically significant difference in cumulative incidences of all-cause mortality(A, log-rank $P=0.259$) or the composite of reinfarction and cardiac death(B, log-rank $P=0.218$) between patients with a right dominant, left dominant and balanced coronary artery system.

incidences of the primary and secondary endpoint were observed comparable to a right dominant or balanced system at 5 years of follow-up after STEMI.

Prognostic value of coronary vessel dominance

Currently, a left dominant system is considered to be a normal variant of the coronary anatomy without particular prognostic value. However, a left dominant system is less common than a right dominant system which may reflect a biological disadvantage. A study screening 1620 postmortem angiograms, showed that the prevalence of a left dominant system decreased with age¹⁵, suggesting a higher death rate among patients with a left

Table 4. Multivariate Cox regression analysis in survivors of the first 30 days post-STEMI

| | All-cause Mortality | | | Reinfarction or cardiac death | | |
|---|---------------------|-------------|---------|-------------------------------|-------------|---------|
| | HR | 95%CI | p-value | HR | 95%CI | p-value |
| Age | 1.05 | (1.02-1.09) | 0.001 | 0.99 | (0.97-1.02) | 0.727 |
| Gender(male) | 1.95 | (1.02-3.70) | 0.042 | 2.46 | (1.29-4.67) | 0.006 |
| Diabetes | 1.70 | (0.84-3.45) | 0.138 | 1.32 | (0.70-2.51) | 0.389 |
| Hypertension | 0.96 | (0.56-1.63) | 0.874 | 1.03 | (0.65-1.63) | 0.910 |
| Current smoking | 1.68 | (0.99-2.87) | 0.056 | 1.29 | (0.82-2.01) | 0.267 |
| Killip class during STEMI | 1.37 | (0.96-1.95) | 0.083 | 1.38 | (0.98-1.93) | 0.064 |
| Three-vessel disease | 1.93 | (1.12-3.33) | 0.018 | 2.12 | (1.31-3.41) | 0.002 |
| eGFR(mL/min/1.73m ²) | 0.99 | (0.98-1.00) | 0.067 | 0.99 | (0.99-1.00) | 0.198 |
| Peak cardiac troponin T level (µg/L) | 1.06 | (1.03-1.10) | <0.001 | 1.01 | (0.97-1.04) | 0.637 |
| LV ejection fraction at discharge (%) | 0.97 | (0.95-1.00) | 0.064 | 0.96 | (0.94-0.99) | 0.002 |
| Reinfarction within 30 days post-STEMI [†] | 1.99 | (0.58-6.82) | 0.276 | 3.44 | (1.44-8.19) | 0.005 |
| Coronary Vessel Dominance | | | | | | |
| Right dominant | Reference (1.00) | | | Reference (1.00) | | |
| Left dominant | 1.52 | (0.73-3.19) | 0.264 | 1.61 | (0.87-2.99) | 0.131 |
| Balanced | 0.93 | (0.28-3.03) | 0.900 | 1.03 | (0.41-2.59) | 0.944 |

eGFR=estimated glomerular filtration rate; LV=left ventricle; STEMI=ST-segment elevation myocardial infarction.

dominant coronary artery system. An explanation could be that a larger amount of myocardium is at risk in these patients, resulting in more extensive myocardial infarction in case of a left coronary artery occlusion.

Limited information is available about the prognostic value of coronary vessel dominance in patients with STEMI. In 27,289 patients presenting with acute coronary syndrome(ACS), Goldberg et al. showed that the presence of a left dominant system was associated with an increased mortality (HR: 1.13, 95% CI1.00-1.28).¹⁶ Accordingly, a more recent registry (the CathPCI registry), observed a higher in-hospital mortality after PCI in patients with a left dominant system (OR 1.29, 95%CI 1.17-1.42).¹⁷ However, both registries represent a heterogeneous patient populations, including patients with STEMI as well as non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina, having varying risks.^{18,19} In the study by Goldberg, the increased risk of mortality in the presence of a left dominant system was more pronounced in patients presenting with STEMI.¹⁶ In contrast, in the subanalysis of the CathPCI registry the increased risk in patients with a left dominant system only remained significant in patients presenting with NSTEMI.¹⁷

Our study is the first to include only patients with STEMI, demonstrating significantly worse outcome with a left dominant system compared to a right dominant or balanced system. A primary endpoint of all-cause mortality and secondary endpoint of reinfarction or cardiac death were studied at both short- and long-term follow-up, extending the earlier

findings.^{16,17} Differences in outcome compared to the CathPCI registry can be explained by differences in patient population and type of PCI used.

Coronary vessel dominance and 30-day outcome

In this study a more than 2-fold increased risk for 30-day mortality was found in patients with a left dominant system compared to a right dominant system. This can be explained by the impact coronary anatomy has on the extent of the infarcted area and recovery of left ventricular function post-STEMI. Ilija et al. showed that acute occlusion of a proximal dominant LCx artery resulted in a higher proportion of patients presenting with cardiogenic shock and higher in-hospital mortality rate as compared to patients with a proximal LAD artery occlusion, underlining the importance of a dominant LCx artery which supplies approximately 60% of the left ventricular myocardium.⁵ Another factor contributing to the poor outcome of patients with a left dominant system could be lack of collateral circulation in patients with a left dominant system.^{20,21}

Additionally, this study showed a higher risk of early reinfarction in patients with a left dominant system. Yip et al. showed that a left dominant system was independently predictive of failed reperfusion in patients with LCx artery infarction.²²

Coronary vessel dominance and long-term outcome after surviving the first month after STEMI

Coronary vessel dominance had impact on outcome during the first 30 days post-STEMI, but had no prognostic significance during long-term follow-up. This could be explained by the fact that all patients received secondary prevention post-STEMI.¹⁴ Therefore, the influence of coronary vessel dominance on long-term outcome may be less prominent once surviving the first 30 days post-STEMI.

Limitations

This is a retrospective analysis with all inherent limitations. Second, although we adjusted for a wide range of potential confounders in the multivariate analysis, the possibility of unmeasured confounding remains. In patients who died within the first 30 days post-STEMI, covariates such as peak cardiac troponin T level and left ventricular ejection fraction at discharge were incomplete or unavailable. Hence, these factors could not be corrected for in the multivariate analysis on 30-day outcome. Finally, hospitalization for heart failure has been used as clinical endpoint in patients after STEMI, but was not assessed in the current study.

CONCLUSIONS

This study demonstrated that patients with a left dominant system have worse outcome after STEMI, with a 2-fold increased risk of 30-day mortality and an increased risk of reinfarction during the first month post-STEMI. In patients surviving the first 30 days post-STEMI, coronary vessel dominance had no influence on long-term outcome.

REFERENCES

1. Leaman DM, Brower RW, Meester GT, Serruys P, van den Brand M. Coronary artery atherosclerosis: severity of the disease, severity of angina pectoris and compromised left ventricular function. *Circulation* 1981;63:285-299.
2. Angelini P, Velasco JA, Flamm S. Coronary anomalies: incidence, pathophysiology, and clinical relevance. *Circulation* 2002;105:2449-2454.
3. Cademartiri F, La GL, Malago R, Alberghina F, Meijboom WB, Pugliese F, Maffei E, Palumbo AA, Aldrovandi A, Fusaro M, Brambilla V, Coruzzi P, Midiri M, Mollet NR, Krestin GP. Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. *Eur Radiol* 2008;18:781-791.
4. Gorlin R Coronary anatomy. *Major Probl Intern Med* 1976;11:40-58.:40-58.
5. Ilija R, Cafri C, Weinstein JM, Gueron M. Acute myocardial infarction due to occlusion of the dominant left circumflex artery proximally. *Am J Cardiol* 2003;92:54-55.
6. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, Thygesen K, Alpert JS, White HD, Jaffe AS, Katus HA, Apple FS, Lindahl B, Morrow DA, Chaitman BR, Clemmensen PM, Johanson P, Hod H, Underwood R, Bax JJ, Bonow JJ, Pinto F, Gibbons RJ, Fox KA, Atar D, Newby LK, Galvani M, Hamm CW, Uretsky BF, Steg PG, Wijns W, Bassand JP, Menasche P, Ravkilde J, Ohman EM, Antman EM, Wallentin LC, Armstrong PW, Simoons ML, Januzzi JL, Nieminen MS, Gheorghide M, Filippatos G, Luepker RV, Fortmann SP, Rosamond WD, Levy D, Wood D, Smith SC, Hu D, Lopez-Sendon JL, Robertson RM, Weaver D, Tendera M, Bove AA, Parkhomenko AN, Vasilieva EJ, Mendis S, Bax JJ, Baumgartner H, Ceconi C, Dean V, Deaton C, Fagard R, Funck-Brentano C, Hasdai D, Hoes A, Kirchhof P, Knutti J, Kolh P, McDonagh T, Moulin C, Popescu BA, Reiner Z, Sechtem U, Sirnes PA, Tendera M, Torbicki A, Vahanian A, Windecker S, Morais J, Aguiar C, Almahmeed W, Arnar DO, Barili F, Bloch KD, Bolger AF, Botker HE, Bozkurt B, Bugiardini R, Cannon C, de LJ, Eberli FR, Escobar E, Hlatky M, James S, Kern KB, Moliterno DJ, Mueller C, Neskovic AN, Pieske BM, Schulman SP, Storey RF, Taubert KA, Vranckx P, Wagner DR. Third universal definition of myocardial infarction. *J Am Coll Cardiol* 2012;60:1581-1598.
7. Antoni ML, Boden H, Hoogslag GE, Ewe SH, Auger D, Holman ER, van der Wall EE, Schalij MJ, Bax JJ, Delgado V. Prevalence of dyssynchrony and relation with long-term outcome in patients after acute myocardial infarction. *Am J Cardiol* 2011;108:1689-1696.
8. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC, Jr., Alpert JS, Anderson JL, Faxon DP, Fuster V, Gibbons RJ, Gregoratos G, Halperin JL, Hiratzka LF, Hunt SA, Jacobs AK. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction). *Circulation* 2004;110:e82-292.
9. Van de Werf F, Ardissino D, Betriu A, Cokkinos DV, Falk E, Fox KA, Julian D, Lengyel M, Neumann FJ, Ruzyllo W, Thygesen C, Underwood SR, Vahanian A, Verheugt FW, Wijns W. Management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J* 2003;24:28-66.

10. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr* 2005;18:1440-1463.
11. Scanlon PJ, Faxon DP, Audet AM, Carabello B, Dehmer GJ, Eagle KA, Legako RD, Leon DF, Murray JA, Nissen SE, Pepine CJ, Watson RM, Ritchie JL, Gibbons RJ, Chaitlin MD, Gardner TJ, Garson A, Jr., Russell RO, Jr., Ryan TJ, Smith SC, Jr. ACC/AHA guidelines for coronary angiography. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (Committee on Coronary Angiography). Developed in collaboration with the Society for Cardiac Angiography and Interventions. *J Am Coll Cardiol* 1999;33:1756-1824.
12. Veltman CE, de Graaf FR, Schuijff JD, van Werkhoven JM, Jukema JW, Kaufmann PA, Pazhenkottil AP, Kroft LJ, Boersma E, Bax JJ, Schalij MJ, van der Wall EE. Prognostic value of coronary vessel dominance in relation to significant coronary artery disease determined with non-invasive computed tomography coronary angiography. *Eur Heart J* 2012;33:1367-1377.
13. Antoni ML, Yiu KH, Atary JZ, Delgado V, Holman ER, van der Wall EE, Schuijff JD, Bax JJ, Schalij MJ. Distribution of culprit lesions in patients with ST-segment elevation acute myocardial infarction treated with primary percutaneous coronary intervention. *Coron Artery Dis* 2011;22:533-536.
14. Liem SS, van der Hoeven BL, Oemrawsingh PV, Bax JJ, van der Bom JG, Bosch J, Viergever EP, van RC, Padmos I, Sedney MI, van Exel HJ, Verwey HF, Atsma DE, van der Velde ET, Jukema JW, van der Wall EE, Schalij MJ. MISSION!: optimization of acute and chronic care for patients with acute myocardial infarction. *Am Heart J* 2007;153:14-11.
15. Knaapen M, Koch AH, Koch C, Koch KT, Li X, van Rooij PC, Tijssen JG, Peters RJ, van der Wal AC, Damman P, de Winter RJ. Prevalence of left and balanced coronary arterial dominance decreases with increasing age of patients at autopsy. A postmortem coronary angiograms study. *Cardiovasc Pathol* 2013;22:49-53.
16. Goldberg A, Southern DA, Galbraith PD, Traboulsi M, Knudtson ML, Ghali WA. Coronary dominance and prognosis of patients with acute coronary syndrome. *Am Heart J* 2007;154:1116-1122.
17. Parikh NI, Honeycutt EF, Roe MT, Neely M, Rosenthal EJ, Mittleman MA, Carrozza JP, Jr., Ho KK. 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. *Circ Cardiovasc Qual Outcomes* 2012;5:775-782.
18. Braunwald E, Antman EM, Beasley JW, Califf RM, Chaitlin MD, Hochman JS, Jones RH, Kereiakes D, Kupersmith J, Levin TN, Pepine CJ, Schaeffer JW, Smith EE, III, Stewart DE, Theroux P, Gibbons RJ, Alpert JS, Faxon DP, Fuster V, Gregoratos G, Hiratzka LF, Jacobs AK, Smith SC, Jr. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction--summary article: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol* 2002;40:1366-1374.

19. O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Jr., Chung MK, de Lemos JA, Ettinger SM, Fang JC, Fesmire FM, Franklin BA, Granger CB, Krumholz HM, Linderbaum JA, Morrow DA, Newby LK, Ornato JP, Ou N, Radford MJ, Tamis-Holland JE, Tommaso CL, Tracy CM, Woo YJ, Zhao DX. 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2013;61:e78-e140.
20. Ilia R, Carmel S, Cafri C, Gueron M. Coronary collaterals in patients with normal and impaired left ventricular systolic function. *Int J Cardiol* 1998;63:151-153.
21. Chen YL, Hang CL, Fang HY, Tsai TH, Sun CK, Chen CJ, Chen SM, Yang CH, Hsieh YK, Wu CJ, Fu M, Yip HK. Comparison of prognostic outcome between left circumflex artery-related and right coronary artery-related acute inferior wall myocardial infarction undergoing primary percutaneous coronary intervention. *Clin Cardiol* 2011;34:249-253.
22. Yip HK, Wu CJ, Fu M, Yeh KH, Yu TH, Hung WC, Chen MC. Clinical features and outcome of patients with direct percutaneous coronary intervention for acute myocardial infarction resulting from left circumflex artery occlusion. *Chest* 2002;122:2068-2074.

