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The Same Angiographic Factors Predict Venous and Arterial Graft Patency

A Retrospective Study

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Abstract: To evaluate the value of angiographic factors in predicting failure of both venous and arterial coronary artery bypass graft.

We retrieved from our angiographic database 148 patients who underwent venous and/or arterial CABG and for whom a control coronary angiography at more than 1 month after surgery was available. Pre-CABG and follow-up angiographies were analyzed in order to evaluate diameter stenosis (DS,%), stenosis length (mm), Bogaty score (extent index), Sullivan score, and Gensini score for the extent of coronary artery disease, and Jeopardy Duke score for the extent of myocardial area supplied by an artery.

Thirty-nine patients (26%) experienced graft failure at follow-up (mean follow-up 11.3 ± 4.6 months). Patients with venous graft failure [26 (20%)] had significantly smaller DS ($P = 0.013$), shorter stenosis length ($P = 0.01$), and lower extent index ($P = 0.015$), Sullivan score ($P = 0.013$), Gensini score ($P = 0.04$) as compared with those without venous graft failure. Patients with arterial graft failure [13 (11%)] had significantly lower DS ($P = 0.008$), shorter stenosis length ($P = 0.001$), and lower extent index ($P = 0.03$) and Sullivan score ($P = 0.023$) as compared with those without arterial graft failure.

Venous and arterial graft failure are associated with less severe stenosis and less extensive atherosclerosis of the grafted vessel.

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Abbreviations: CABG = coronary artery bypass graft, DS = diameter stenosis, FFR = fractional flow reserve, IMA = internal mammary artery, LAD = left anterior descending artery.

INTRODUCTION

Worldwide, more than 800,000 patients undergo coronary artery bypass grafting (CABG) annually. The majority of these patients receive left internal mammary artery (IMA) grafts to the left anterior descending (LAD) coronary artery and saphenous vein grafts or arterial conduits to the remaining vessels. Graft patency, along with completeness of revascularization, are major determinants of long-term outcome following CABG procedures.[1,2]

However, limited information is available on the relation between the preoperative angiographic characteristics of the target vessel and the postoperative graft outcome; moreover, most of the published studies on the argument focused on the patency of arterial but not of venous grafts.[3–5]

In this study, we aimed at evaluating the value of preoperative angiographic features of the target coronary vessels in predicting the failure of both venous and arterial bypass conduits.

METHODS

Patient Population

We retrieved from our electronic angiographic database patients who underwent venous and/or mammary arterial CABG and for whom a control coronary angiography at more than 1 month after surgery was available. From November 2003 to April 2013, we identified 148 patients who underwent a postoperative coronary angiogram beyond the first month after CABG; 51 of them (34.4%) were restudied because involved in study protocols that required control angiography, whereas the remaining underwent repeated coronary angiography for clinical reasons (typical anginal symptoms and/or positive control stress test).

All surgical interventions were performed through a traditional median sternotomy and with central conventional cardiopulmonary bypass. Standard blood cardioplegia (Buckberg cardioplegia) was used for all patients. IMA conduits were harvested in a skeletonized fashion by using the electrocautery. IMAs were always used as "in situ" grafts and prepared using endoluminal papaverine injection. Saphenous vein grafts were harvested in an open standard fashion.

For both venous and arterial grafts, graft failure was defined in the presence of at least 1 critical stenosis (ie, a stenosis with a diameter stenosis [DS] $\geq$70%) in the graft or when graft origin was not visualized either by selective angiography or by aortic root angiography. When analyzing sequential or Y vein grafts, a distal anastomosis (either side to side or end to side) was defined as patent if the contrast medium was seen to flow from the vein graft into the grafted artery. If the graft was occluded at its origin, all associated distal anastomoses were considered occluded. If 1 distal anastomosis of a Y or sequential vein graft was occluded, that site was defined as an occluded graft.

The study was approved by the Ethics Committee of the Catholic University of the Sacred Heart.

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Quantitative Angiographic Measurement

Patients with venous or arterial patent grafts were used respectively as control for venous or arterial graft failure, irrespective of the patency status of a possible coexisting arterial graft for the former and of a possible coexisting venous graft for the latter.

Quantitative coronary angiography was performed with a previously validated automated edge detection method (CMS Medis Imaging System, Wallingford, CT), using a guiding or diagnostic catheter as a reference for calibration. All measurements were performed on images obtained after intracoronary nitrate administration of 200 μg of nitroglycerin. Pre-CABG and follow-up angiographies were analyzed to evaluate DS, stenosis length (mm), Bogaty score (extent index), Sullivan score, Gensini score, and Jeopardy Duke and Bari scores. Two expert angiographers (GN and MR), who were blinded to the clinical and laboratory values, evaluated all angiographic images to assess angiographic scores. Any disagreement between the 2 angiographers was resolved by consensus; when consensus could not be reached, a third experienced angiographer (MG) assessed and classified the parameters under evaluation. All analyses were done after intracoronary nitrates injection.

We chose angiographic scores that accounted both for longitudinal extent of coronary artery disease (Bogaty score,[6] Sullivan score,[7] and Gensini score [8]) and for quantification of the extent of myocardial area affected by stenotic lesions (Jeopardy Duke and Bari scores).[9–11] Bogaty score[6] assesses disease severity (severity index), by investigating the number of diseased vessels, including the number of major epicardial vessels with 70% narrowing of the lumen diameter. The maximum number of vessels diseased was 3 and patients with at least 2 major vessels involved were classified as multivessel disease. Bogaty score determines also extent of disease (extent index), which is usually obtained by dividing the extent score of the entire coronary arterial tree by the number of analyzed segments. In our study, we focused on specific vessels and this score was obtained dividing the extent score of this specific vessel by the total number of analyzed segments. A segment was scored 0 if it appeared angiographically normal, 1 if $<10\%$ of its length appeared abnormal (narrowed and/or irregular), 2 if $>10\%$ up to 50% of its length was abnormal, and 3 if $>50\%$ of its length was abnormal. Moreover, additional quantitative angiographic scores, using the Gensini[8] and Sullivan[7] extent systems, were calculated. The Gensini score[8] quantifies severity of coronary artery disease by a nonlinear points system for the degree of luminal narrowing along with a multiplier for specific coronary tree locations, thereby weighting each lesion score for prognostic significance. The total of the lesion scores is usually summed to give a final Gensini score. Thus, multiple severe proximal lesions gain the highest score. In our study, we focused on specific vessels and this score was thus obtained summing lesion scores referring to these specific vessels. The Sullivan Extent[7] score quantifies the percentage of the coronary intimal surface area affected by atheroma, without specific weighting for the degree of luminal narrowing. The percentage involvement of each vessel is estimated and multiplied by a factor representative of the surface area of that vessel in relation to the entire coronary tree. We used a modified version based on segments of each vessel with reported disease to derive percentage involvement. Four segments of right coronary artery each contributing 25%; 3 segments of left anterior descending artery each contributing 33% with the proximal segment further subdivided into 2; left circumflex artery divided into 3 segments each contributing 33%. Again, in our study, we focused on specific vessels and this score was thus obtained as mean of scores referring to each segment of these specific vessels.

The Jeopardy Duke Score was developed by Dash et al[9] and validated by Califf et al.[10] The coronary tree is divided into 6 segments: the LAD, diagonal branches of the LAD, septal perforating branches, the circumflex coronary artery, obtuse marginal branches, and the posterior descending coronary artery. Each segment distal to $\geq 70\%$ stenosis is assigned 2 points. In our study we focused on specific vessels and this score was thus obtained summing lesion scores referring to these specific vessels. The Duke Coronary Artery Disease Severity Index was developed by Mark et al.[11] The score is discontinuous with higher prognostic weight given to both number of involved vessels and increasing severity of LAD stenosis, with more proximal disease weighted higher. In our study we focused on specific vessels and this score was thus obtained summing lesion scores referring to these specific vessels.

Statistical Analysis

Normal distribution was assessed by the Kolmogorov–Smirnov test. Variables that did not follow a normal distribution were expressed as median and interquartile range, whereas other continuous variables were expressed as means ± standard deviation; categorical variables were expressed as proportions. Continuous variables were compared by Student t test or Mann–Whitney U test as appropriate, whereas categorical variables by χ^2 test or Fisher exact test, as appropriate.

For patients undergoing mammary arterial CABG we performed a 1-to-1 matched analysis without replacement on the basis of the estimated propensity score of each patient. The log odd of arterial graft failure for each patient (the ''logit'') was modeled as a function of the confounders that we identified and included in our dataset. Using the estimated logits, we first randomly selected a patient in the arterial graft failure group and then matched that patient with that patient in the arterial graft patency group with the closest estimated logit value. Patients in the arterial graft patency group who had an estimated logit within 0.25 standard deviations of the selected patients in the arterial graft failure group were eligible for matching. We chose 0.25 standard deviations because this value has been shown to eliminate more than 90% of the bias in observed confounders.[12] If more than 1 patient met this criterion, we randomly selected 1 patient for matching. After all the propensity score matches were performed, we compared the baseline covariates between the 2 groups. Continuous variables were compared using the paired t test or the Wilcoxon signed rank test, as appropriate.

$P < 0.05$ was always required for statistical significance. The software SPSS 20.0 (SPSS Italia, Florence, Italy) was used for statistical analyses.

RESULTS

Overall, 148 patients underwent postoperative coronary angiography beyond 30 days (728 ± 219 days) after surgical revascularization (age 69 ± 9 years, male sex 81%). Within the overall population, 39 patients (26%) experienced graft failure at follow-up (mean follow-up 11.3 ± 4.6 months). Among 129 venous graft patients, 26 were found to have graft failure (20%), while among 116 arterial graft patients, 13 were found to have graft failure (11%).

TABLE 1. Baseline Clinical Characteristics, Therapy on Admission, and Laboratory Data Within Venous Graft Patients

Variables	Patients With Venous Grafts Patency (103)	Patients With Venous Grafts Failure (26)	*P*
Clinical characteristics			
Age (yr ± DS)	68 (± 8)	69 (± 9)	0.50
Male sex [n, (%)]	87 (84.5%)	18 (69.2%)	0.07
Hypertension [n, (%)]	80 (77.7%)	23 (88.5%)	0.28
Current smoker [n, (%)]	62 (60.2%)	17 (65.4%)	0.63
Dyslipidemia [n, (%)]	67 (65%)	21 (80.8%)	0.16
Diabetes mellitus [n, (%)]	44 (42.7%)	12 (46.2%)	0.75
Family history of CAD [n, (%)]	29 (28.2%)	12 (46.2%)	0.08
Acute clinical presentation [n, (%)]	25 (24.3%)	2 (7.7%)	0.10
Pre-ACS [n, (%)]	31 (30.1%)	6 (23.1%)	0.63
Pre-PCI [n, (%)]	20 (19.4%)	4 (15.4%)	0.64
Ejection fraction (%)	47.9 (±7.1)	47.5 (±7.9)	0.82
Therapy on admission			
Beta-blokers [n, (%)]	69 (67%)	21 (80.8%)	0.23
ACE-inhibitors [n, (%)]	44 (42.7%)	15 (57.7%)	0.17
Statins [n, (%)]	52 (50.5%)	12 (46.2%)	0.69
Aspirin [n, (%)]	83 (80.6%)	22 (84.6%)	0.78
Routine laboratory data			
Blood glucose (mg/dL)	97 [87–137]	107 [91–162]	0.26
Total cholesterol (mg/dL)	178 (±54)	161 (±37)	0.14
LDL (mg/dL)	107 (±40)	99 (±31)	0.31
HDL (mg/dL)	43 (±11)	41 (±10)	0.42
Creatinine (mg/dL)	1.1 [0.9–1.2]	1.10 [0.98–1.3]	0.63
ESR (mm/h)	6.0 [4.7–16.0]	8.0 [6.0–15.0]	0.18
Fibrinogen (mg/dL)	328 [275–423]	334 [275–405]	0.93
CK-MB (ng/mL)	101 [44–153]	92 [35–148]	0.56
TnT post (ng/mL)	5.49 [1.93–12.6]	5.98 [1.35–24.4]	0.73

ACE = angiotensin-converting enzyme; ACS = acute coronary syndrome; CAD = coronary artery disease; CK-MB = creatine kinase-MB; ESR = erythrocyte sedimentation rate; HDL = high-density lipoprotein; LDL = low-density lipoprotein; PCI = percutaneous coronary intervention; TnT = Troponin T.

Venous Graft Failure

Patients with patency of venous grafts did not differ with regard to main cardiovascular risk factors, therapy on admission, and baseline laboratory data as compared with those showing venous graft failure (Table 1). Among angiographic data (Table 2), as assessed by coronary angiography performed before coronary bypass, patients showing venous graft failure had significant lower DS ($P = 0.013$) and a shorter stenosis length ($P = 0.001$) (Figure 1A). When assessing angiographic scores toward the target vessel, patients showing venous graft failure had lower extent index, Sullivan score, and Gensini score as compared with those showing graft patency ($P = 0.015$,

TABLE 2. Angiographic Data Within Venous Graft Patients

Variables	Patients Without Venous Grafts Failure (103)	Patients With Venous Grafts Failure (26)	*P*
Angiographic data			
RVDpre (mm)	2.40 (±0.31)	2.31 (±0.33)	0.19
MLDpre (mm)	0.58 (±0.18)	0.65 (±0.16)	0.09
DS (%)	87.6 (±6.5)	84.0 (±6.2)	0.013
Stenosis length (mm)	11.29 [9.27–11.56]	7.70 [4.55–11.44]	0.01
Sullivan target vessel	4.67 [3.33–7.37]	3.66 [1.92–5.42]	0.013
Extent index target vessel	0.33 [0.26–0.42]	0.26 [0.20–0.35]	0.015
Gensini target vessel	8.0 [6.5–8.2]	6.7 [5.6–8.4]	0.04
Jeopardy Score Duke target vessel	27.0 [22.7–29.0]	24.0 [19.0–28.2]	0.13
Jeopardy Score Bari target vessel	3.2 [3.0–3.5]	3.0 [2.3–3.8]	0.09

DS = diameter stenosis; MLD = minimum luminal diameter; RVD = reference vessel diameter.

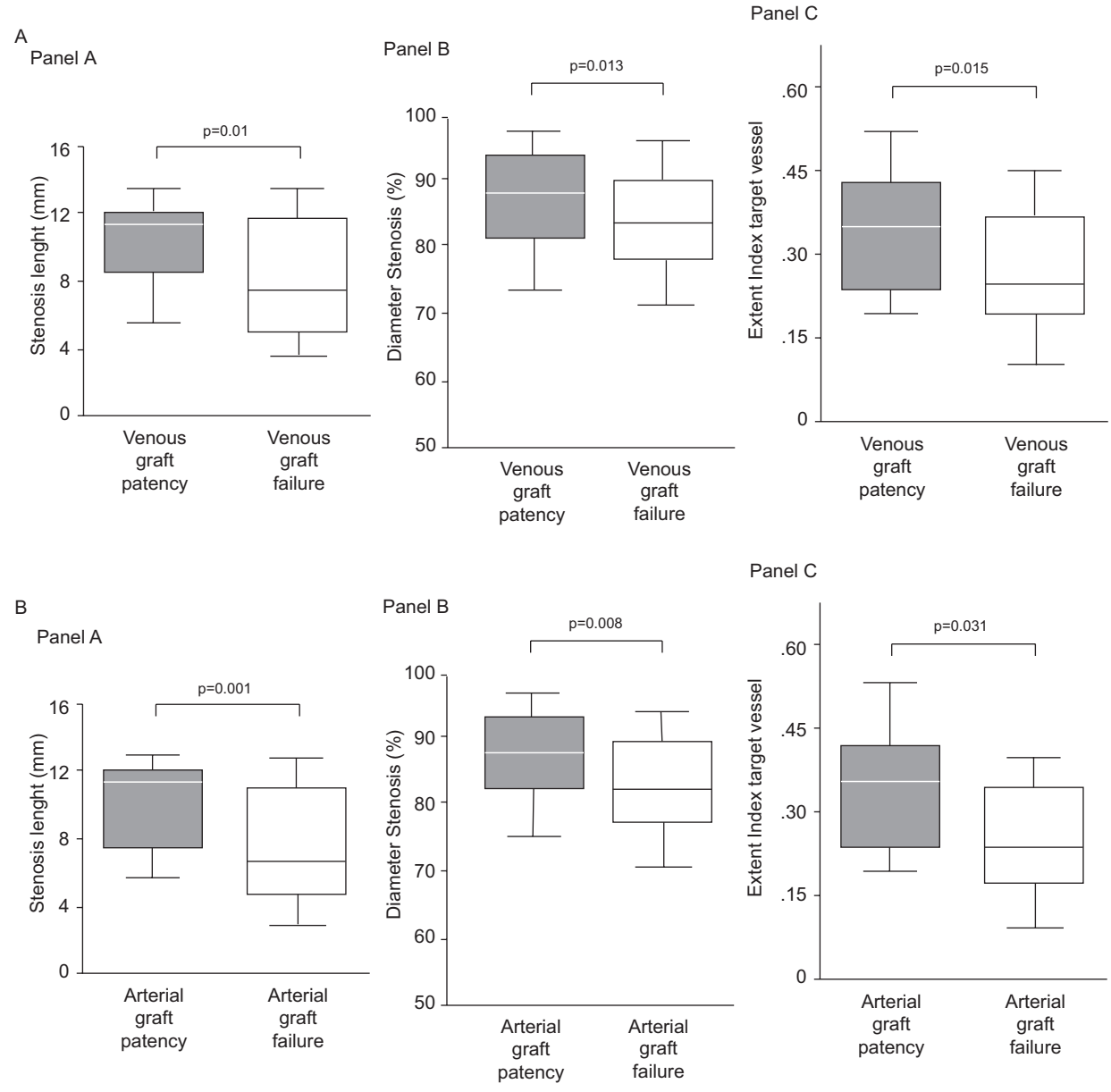

FIGURE 1. A, Stenosis length (mm—Panel A), diameter stenosis (%—Panel B), and extent index (Panel C) in patients with saphenouos venous graft patency and failure. B, Stenosis length (mm—Panel A), diameter stenosis (%—Panel B), and extent index (Panel C) in patients with arterial graft patency and failure.

$P = 0.013$, and $P = 0.04$, respectively), while Jeopardy Duke and Bari scores tended to be lower ($P = 0.13$ and $P = 0.09$, respectively).

Arterial Graft Failure

Patients with arterial graft patency did not differ with regard to main cardiovascular risk factors and therapy on admission as compared with those showing arterial graft failure (Table 3). Among angiographic data (Table 4), as assessed by coronary angiography performed before coronary bypass, patients showing arterial graft failure had significant lower DS ($P = 0.008$) and shorter stenosis length ($P = 0.001$) (Figure 1B). When assessing angiographic scores toward the target vessel, patients showing arterial graft failure had a lower extent index, Sullivan score ($P = 0.03$ and $P = 0.02$, respectively), and tended to have a lower Gensini score ($P = 0.09$) as compared with those showing graft patency. Patients showing arterial graft failure tended to have lower Jeopardy Duke and Bari scores as compared to those showing graft patency ($P = 0.12$ and $P = 0.12$, respectively). Clinical and angiographic features of the propensity score–matched patients are shown in Table 5. Patients with arterial graft patency did not differ with regard to analyzed clinical and angiographic data as compared with those showing arterial graft failure according to propensity score analysis.

TABLE 3. Baseline Clinical Characteristics, Therapy on Admission, and Laboratory Data Within Arterial Graft Patients

Variables	Patients Without Arterial Mammary Grafts Failure (103)	Patients With Arterial Mammary Grafts Failure (13)	*P*
Clinical characteristics			
Age (yr ± DS)	67 (±9)	72 (±6)	0.04
Male sex [n, (%)]	81 (78.6%)	12 (92.3%)	0.46
Hypertension [n, (%)]	83 (80.6%)	12 (92.3%)	0.46
Current smoker [n, (%)]	65 (63.1%)	7 (53.8%)	0.52
Dyslipidemia [n, (%)]	73 (70.9%)	7 (53.8%)	0.21
Diabetes Mellitus [n, (%)]	51 (49.5%)	6 (46.2%)	0.82
Family history of CAD [n, (%)]	34 (33%)	2 (15.4%)	0.40
Acute clinical presentation [n, (%)]	21 (20.1%)	3 (23.7%)	0.73
Pre-ACS [n, (%)]	32 (31.1%)	2 (15.4)	0.34
Pre-PCI [n, (%)]	16 (15.5%)	4 (30.8%)	0.24
Ejection fraction (%)	47 (±7)	46 (±8)	0.54
Therapy on admission			
Beta-blokers [n, (%)]	73 (70.9%)	9 (69.2%)	1.00
ACE-inhibitors [n, (%)]	50 (48.5%)	5 (38.5%)	0.57
Statins [n, (%)]	46 (44.7%)	8 (61.5%)	0.38
Aspirin [n, (%)]	82 (79.6%)	10 (76.9%)	0.73
Routine laboratory data			
Blood glucose (mg/dL)	102 [87–150]	96.0 [88–127]	0.36
Total cholesterol (mg/dL)	182 (±51)	150 (±30)	0.03
LDL (mg/dL)	112 (±41)	82 (±23)	0.001
HDL (mg/dL)	44 (±11)	43 (±12)	0.99
Creatinine (mg/dL)	1.1 [1.0–1.2]	1.1 [.9–1.2]	0.92
ESR (mm/h)	7 [5–16]	7 [4–12]	0.77
Fibrinogen (mg/dL)	324 [275–415]	317 [282–528]	0.54
CK-MB (ng/mL)	87 [38–145]	124 [87–292.9]	0.07
TnT post (ng/mL)	5.49 [1.99–16.10]	5.83 [2.09–17.2]	0.89

ACE = angiotensin converting enzyme; ACS = acute coronary syndrome; CAD = coronary artery disease; CK-MB = creatine kinase-MB; ESR = erythrocyte sedimentation rate; HDL = high-density lipoprotein; LDL = low-density lipoprotein; PCI = percutaneous coronary intervention; TnT = Troponin T.

DISCUSSION

The present study investigates the influence of angiographic features of the target native coronary artery on the long-term patency of venous and arterial bypass grafts. We found that both venous and arterial graft failures are associated with less severe/shorter stenosis and less extensive atherosclerosis of the grafted vessel, thus suggesting that both stenosis features and disease extent influence arterial and venous graft patency.

The correlation between stenosis severity of the target vessel and long-term graft patency is generally accepted for arterial grafts;[13–15] however, very limited information is available on the influence of target vessel stenosis on venous graft patency (although it is generally believed that saphenous

TABLE 4. Angiographic Data Within Arterial Graft Patients

Variables	Patients With Arterial Mammary Grafts Patency (103)	Patients With Arterial Mammary Grafts Failure (13)	*P*
Angiographic data			
RVDpre (mm)	2.35 (±0.29)	2.36 (±0.35)	0.93
MLDpre (mm)	0.57 (±0.17)	0.63 (±0.22)	0.24
DS (%)	87.1 (±6.1)	82.2 (±6.4)	0.008
Stenosis length (mm)	11.30 [8.47–11.57]	6.67 [4.65–9.55]	0.001
Sullivan target vessel	4.67 [3.00–7.00]	2.67 [2.00–4.40]	0.023
Extent index target vessel	0.34 [0.26–0.40]	0.23 [0.17–0.32]	0.031
Gensini target vessel	8.0 [6.4–8.5]	6.5 [5.5–7.83]	0.09
Jeopardy Score Duke target vessel	3.1 [2.5–3.7]	3.0 [2.17–3.4]	0.12
Jeopardy Score Bari target vessel	27.0 [22.7–29.0]	22.0 [18.5–30.2]	0.12

DS = diameter stenosis; MLD = minimum luminal diameter; RVD = reference vessel diameter.

TABLE 5. Baseline and Angiographic Features of the Propensity Score-Matched Patients

Variables	Patients With Arterial Mammary Grafts Patency (13)	Patients With Arterial Mammary Grafts Failure (13)	*P*
Variables			
Age (yr)	70.5 (±.9.5)	73.4 (±9.2)	0.34
Total cholesterol (mg/dL)	163.7 (±38.5)	155 (±34.8)	0.52
LDL (mg/dL)	97.2 (±30.6)	84 (±27)	0.22
DS (%)	81.5 (±6.8)	81.0 (±6.7)	0.82
Stenosis length (mm)	12.3 [7.55–14.50]	7.1 [5.70–9.82]	0.30
Sullivan target vessel	6.73 [3.40–8.54]	3.75 [2.87–5.53]	0.45
Extent index target vessel	0.28 [0.25–0.36]	0.18 [0.15–0.29]	0.54

DS = diameter stenosis; LDL = low-density lipoprotein.

conduits are less sensitive to chronic native competitive flow). The 5-year angiographic follow-up of the RAPCO trial (Radial Artery Patency and Outcome) suggested that the long-term patency of arterial but not of venous conduits was significantly influenced by target vessel run off,[16] but the sample size was too limited to allow definitive conclusions. In recent years, fractional flow reserve (FFR), an index derived from intracoronary pressure measurements and able to assess the functional significance of a coronary stenosis much more accurately than angiography,[17–19] has been demonstrated to predict CABG patency more accurately than angiography.[20,21] Of note this finding applied to both arterial and venous bypass, thus supporting our data of a similar impact of stenosis severity on both graft types.

Another interesting finding of this study is that a higher extent score (expression of more diseased recipient coronary artery) did not significantly affect arterial or venous graft patency, denying the supposed higher occlusion rate of venous grafts when anastomosed to target vessels of poor quality.

We also found that venous and arterial graft patency tended to be associated with a greater Jeopardy score, a well validated method used to calculate jeopardized myocardium based on the size and the distribution of the coronary arteries. Accordingly, in previous studies, both internal thoracic artery and vein graft patency were shown to be higher and more durable in grafts performed to the left anterior descending coronary artery, as compared with left circumflex and right coronary arteries,[22–24] possibly due to the greater amount of myocardium being supplied by the left anterior descending artery, which results in a larger blood flow demand being placed on grafts to this vessel. Thus, grafts with greater blood flow may be less likely to fail. Of note, a hemodynamically significant stenosis is associated not just with the anatomic severity of the lesion but also with the amount of myocardial tissue subtended by the stenosis itself, as demonstrated by the significant inverse correlation observed between the amount of jeopardized myocardium and FFR.[25]

Our study has some inherent limitations. First, study population is small and no sample size calculation was performed. Moreover, as is common in investigation of this type, the majority of enrolled patients underwent a control angiogram for clinical reasons and the study population represents only a small minority of our overall CABG patients. However, our aim was not to evaluate the incidence of graft occlusion but, instead, to identify the angiographic predictors of graft failure. Due to the limited sample size, no target vessel matching was performed between failed and patent grafts; as arterial grafts were usually anastomosed to the LAD and venous conduits to non-LAD target vessels, the confounding effect of the lack of precise matching is likely to be minimal. However, future studies on larger populations allowing a meaningful target vessel matching are warranted to better clarify this issue. Follow-up period of this study is relatively short. Indeed, the present study is a retrospective study and 51 patients (34.4% of study population) underwent repeated coronary angiography because involved in study protocols that required control angiography. This could explain the short mean time to repeated catheterization observed in the present study. However, future investigations with longer follow-up periods are warranted in order to better evaluate the role of complex angiographic parameters in predicting very-late graft failure. The retrospective nature of our study may have some degree of selection bias with respect to the decision-making process of the surgeon in terms of type of graft and target vessel characteristics. Moreover, coronary angiography, albeit quantitative, remains a relatively weak tool to determine the functional repercussion of a stenosis and it is likely that FFR measurements would have provided us with a better predictor of long-term patency. Furthermore, data on baseline flow velocity of implanted graft were not routinely assessed in the present study. Therefore, future studies are needed to investigate this issue. However, these limitations are common to most of the literature regarding graft outcome[26] and our series is to date one of the very few investigations on the angiographic features associated with arterial and venous graft occlusion. Our results should be viewed as hypothesis generating and should elicit further intensive investigation on this argument.

In conclusion, present data indicate that the long-term patency rate of coronary bypass grafts is low when the recipient vessel is only moderately stenosed and this is true for both arterial and venous conduits. Moreover, the extent of disease of the target coronary artery affects the patency rate of arterial and venous graft in a similar way and the amount of jeopardized myocardium is an important predictor of both arterial and venous graft patency.

These findings imply that the decision to use a graft should be carefully considered in light of both anatomic and functional-haemodynamic severity of the stenosis in the recipient vessel.

Our data suggest that the current attitude of preferring venous conduits for less severe target vessel stenosis and arterial conduits in case of more diseased target coronary vessels should be questioned and underscore the need for further investigation on this issue.

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