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Native coronary disease progression post coronary artery bypass grafting☆

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ABSTRACT

Background: It remains unclear if graft type impacts native disease progression in the target coronary artery post coronary artery bypass grafting (CABG).
Methods: Patients who underwent repeat angiograms at least 6 months post CABG with ≥1 arterial graft were included. Pre/post CABG angiograms were examined by 2 experienced readers. Progression was defined as new stenosis of ≥50% in a previously normal coronary, an increase in previous stenosis of ≥20%, or a new occlusion. Primary outcome was the occurrence of native disease progression in bypassed vessels. Secondary outcomes included complete occlusion, left main (LM) and distal disease progression. Cox-proportional hazard regression models were used for time-to-event outcomes.
Results: Study population included 98 patients comprising 263 grafts (143 arterial/120 venous grafts). Median time from surgery to catheterization was 559 days (Interquartile Range 374,910).Ninety-one target vessels showed progression (34.6%) with 75 to complete occlusion (28.5%). Progression was not associated with graft choice (HR 0.74(0.49,1.13) $p = 0.163$),but was significantly associated with age($p = 0.034$),previous PCI($p = 0.002$),ACE inhibitor (ACEi) use($p < 0.001$),CAD severity ($p < 0.001$),CCS class III/IV($p = 0.016$) and NYHA class III/IV($p < 0.001$). Progression to occlusion was significantly associated with SVG ($p = 0.019$), as well as previous percutaneous coronary intervention ($p = 0.007$) and ACEi use ($p < 0.001$). LM disease progression was significantly associated with peripheral vascular disease (HR 5.44(1.92, 15.46), $p = 0.001$), and not affected by graft type ($p = 0.754$).
Conclusions: Native CAD progression in non-LM coronaries is multifactorial, while SVG use was only associated with occlusion of non-LM coronaries. The implications of this study warrant consideration for increased arterial grafting in CABG patients, while the negative associations of previous PCI and ACEi use carry important clinical implications, which require further investigation.

1. Introduction

Coronary artery bypass grafting (CABG) remains the gold standard in the treatment of extensive coronary artery disease (CAD), conferring excellent control of symptoms and prolongation of life. [1,2] Although CABG was initially done using reversed saphenous veins (SVGs), surgeons have increasingly used arterial conduits due to their improved long-term patency and resistance to atherosclerosis. [3] The use of the left internal thoracic artery (LITA) to the left anterior descending artery (LAD), has been associated with reduced mortality and improved long-term survival when compared to other conduits. [4] Similar excellent results have been reported with the use of the right internal thoracic artery (RITA) and the radial artery (RA) when compared to SVG. [5–7]

The effectiveness of CABG may also be affected by progression of atherosclerosis in the native coronary arteries. Few studies have examined long-term angiographic disease progression of native coronary arteries following surgical revascularization; nor have they identified the factors which may influence this progression. [8,9] Previous reports have shown proximal native disease progression to be two to six times more frequent than distal disease progression, with higher rates of progression in coronaries bypassed with SVGs when compared to arterial grafts. [8,10,11] However, these studies like others, have been limited by small sample sizes [8,9,12–14], paucity of arterial grafts [10,14], and inconsistency in the grading technique to characterize the native vessel changes. [13–16]

The influence of target-vessel characteristics on native disease progression have also provided mixed results. There is no consensus on

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the risk of native disease progression in the right coronary system (RCS) versus the left coronary system (LCS) post CABG [17,18], and the effects of baseline disease severity on progression are inconsistent. [10,16]

The Ottawa Heart Institute (OHI) is the sole cardiac provider for a large catchment area and it has a database of patients who have undergone CABG surgery, all of whom had ≥one arterial bypass graft done. Thus, the objective of this study was to determine factors associated with the progression of disease in native coronary arteries, including the left main (LM) artery. We hypothesized that arterial grafting would result in less native atherosclerosis progression compared to venous grafts due to the anti-atherogenic properties of arterial endothelium. [19,20]

2. Patients and methods

2.1. Patient population and data

The institutional ethics board provided approval to analyze this de-identified data, which had been prospectively collected before and after cardiac surgery. Individual patient consent was waived. The study population consisted of all patients undergoing CABG between January 2010 and March 2015 (n = 4415). CABG referral at our institution is based on current guidelines for CABG. [21] Patients in this study were mixed, consisting of both acute coronary syndromes and patients with stable angina. Prospective clinical data is routinely collected during surgical admission and forms part of the Cardiac Surgery, Cardiac Anesthesia, and Cardiac Catheterization databases. PCI interventions (Drug Eluting Stents (DES) and Bare Metal Stents (BMS)) prior to and post index CABG were tabulated separately from the PCI registry and merged with the database. All data was reviewed retrospectively. Patients were defined as being on a medication if they were on the drug both at the time of surgery and follow-up angiogram. Patients were included if they underwent both pre- and postoperative catheterizations at this institution, and had ≥1 arterial grafts. Patients were excluded if the post-operative angiogram was <6 months from the date of surgery, to allow time for disease progression and to exclude repeat angiograms that may have been driven by technical failures from the graft anastomoses. Follow-up information including survival and subsequent interventions were collected from the electronic medical record. As the index institution is the sole cardiac surgery and interventional cardiology provider for this region of the province, it is unlikely patients underwent other interventions elsewhere.

2.2. Angiographic analysis

Repeat angiograms post CABG were all driven by patient symptoms or non-invasive testing demonstrating ischemia. Angiograms were examined independently by two experienced readers (cardiac surgeons and/or cardiologists). Interpreters were blinded to patient clinical characteristics and type of conduit (radial, vein, free ITA) with proximal anastomoses to the aorta. To facilitate interpretation, native vessel targets of the bypass grafts were shown to readers on post CABG angiograms. Coronary segments were viewed in at least two projections that best represented the segments and stenosis prior to estimating the degree of stenosis. In the event of interpreter discrepancies, an average of the two scores was taken and classified by consensus among the reviewers. Reviewers attempted to assess for disease progression distal to a target site; however, distal bed comparison between pre and postoperative imaging could only be assessed reliably in 23 target sites. In trials of this nature, it is impossible to accurately determine distal disease progression as these coronary segments are often under filled or not opacified in preoperative angiograms as a consequence of native upstream stenosis. This poses a significant problem when trying to differentiate between under filling and preoperative native disease progression. Thus our focus was on proximal native CAD progression.

2.3. Study definitions and outcomes

Readers evaluated films for left main (LM) disease, graft patency, native vessel disease upstream and downstream from the graft and lesion length. Disease severity was estimated both pre- and post-CABG and coded from 0 to 100% occlusion. Lesion length was categorized as either short (<10 mm), medium (10 to 20 mm), or long (>20 mm).

The primary outcome was the occurrence of disease progression in the most proximal upstream segment from the target graft site in native coronary. Secondary outcomes included left main (LM) progression and progression to complete coronary occlusion. Disease progression was defined as development of a new stenosis of at least 50% in an arterial segment previously considered normal, an increase in stenosis of greater ≥20% in a disease segment, or a new occlusion in a vessel that was previously patent. [10,12] New complete occlusion of a proximal vessel was defined as a new upstream 100% stenosis in a previously patent artery on preoperative angiogram. Progression based on lesion length was defined as an increase large enough to reclassify the lesion into the next categorical length. Non-LM coronaries refer to all coronary arteries other than the left main coronary artery.

3. Statistical analysis

Continuous variables were reported as mean ± SD or median (Interquartile Range [IQR]) for non-normally-distributed variables. Categorical variables were reported as counts and percentages. Student's *t*-tests or Wilcoxon rank-sum tests were used to compare continuous variables between groups. For categorical variables, chi-square tests were used to compare differences. Kaplan-Meier failure curves were generated for atherosclerosis progression data. Cox proportional hazards (PH) regression models were used for time-to-event outcomes. Co-variates in the model included: gender, age, drug use (aspirin, clopidogrel, angiotensin converting enzyme inhibitor, statin), left-sided graft configuration (two arteries versus mixed), vessel system involvement, proximal upstream disease severity, body mass index, previous percutaneous coronary intervention (PCI), carotid disease, peripheral vascular disease (PVD), diabetes, hypertension, creatinine, smoking, class III/IV angina, class III/IV NYHA. Type of graft (arterial versus vein) and the presence of an upstream previous stent was also included in the model of proximal disease progression. Covariates with $p < 0.1$ were tested in the multivariable model with stepwise removal of those with $p > 0.05$. A p value < 0.05 was considered significant. Survival data was analyzed using log-rank testing. Time to

intervention was assessed as a cumulative incidence function using all-cause death as a competing event. All statistical analyses and plots were performed with Stata® version 14.1 (College Station, Texas).

4. Results

4.1. Clinical characteristics

Between January 2010 and March 2015, the total population undergoing CABG was 4415. Cases prior to this time were not considered for evaluation as the angiograms were not available. One hundred and forty-one patients underwent both a pre- and postoperative left heart catheterization with a minimum six-month interval duration. Thirty-three patients were excluded as their angiograms could not be located. A further 9 patients were excluded due to incomplete views of the coronaries (6 patients) and interim

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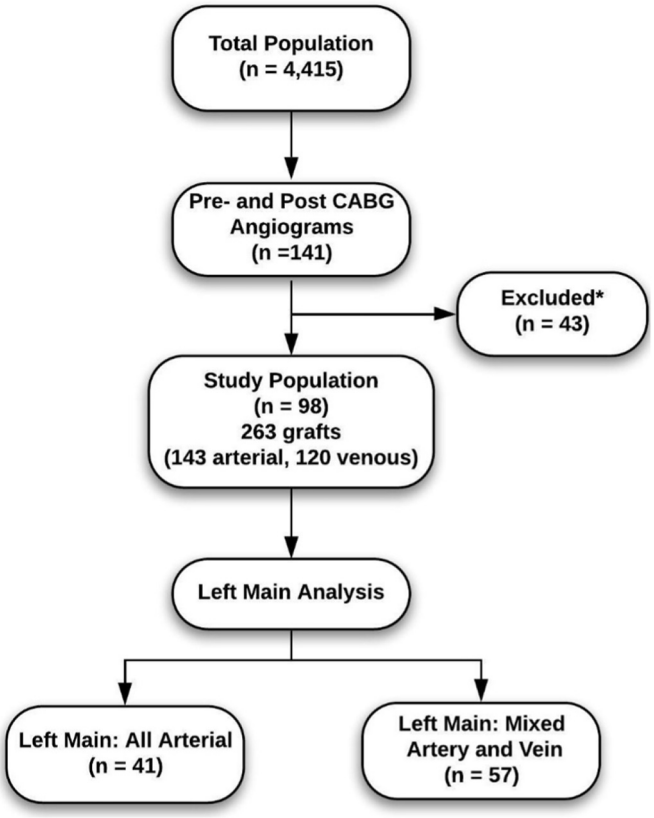

Fig. 1. Flow of patients. Excluded patients: 33 missing angiograms, 6 incomplete coronary views, 3 interim valve replacement surgery, 1 no grafts to left coronary circulation.

Table 1
Patient demographics – all patients and subgroups (arterial only vs mixed arterial/vein configuration).

Characteristic	All patients (n = 98)	All arterial (n = 41)	Mixed (n = 57)	p
Age (years)	66.7 ± 10.1	62.2 ± 11.1	68.8 ± 8.4	0.002
Female gender	21 (21.4)	13 (31.7)	8 (14.0)	0.035
Number of grafts	3 (2,3)	3 (2,3)	3 (2,3)	0.08
Left main stenosis	48 (49%)	18 (43.9)	30 (52.6)	0.39
Days to post-catheterization	559 (374, 910)	613 (404, 1104)	526 (358, 776)	0.36
BMI (kg/m^2)	30.9 ± 5.8	30.5 ± 6.5	31.2 ± 5.3	0.61
Recent MI	36 (63.3)	15 (36.6)	21 (36.8)	0.98
Previous PCI	10 (10.2)	5 (12.2)	5 (8.8)	0.58
Carotid disease	12 (12.2)	4 (9.8)	8 (14.0)	0.52
Peripheral vascular disease	14 (14.3)	4 (9.8)	10 (17.5)	0.28
Diabetes	37 (37.8)	13 (31.7)	24(42.1)	0.30
Hypertension	80 (81.6)	33 (80.5)	47 (82.5)	0.85
Urgent/emergent surgery	50 (51.2)	19 (46.3)	31 (54.4)	0.43
CARE, emergency	2 (2.0)	0 (0.0)	2 (3.5)	0.23
Ventricle class III/IV	9 (9.2)	5 (12.2)	4 (7.0)	0.38
Preoperative Hb (g/L)	133 ± 16	137 ± 15	130 ± 16	0.02
Preoperative Cr (μmol/L)	102 ± 86	98 ± 90	106 ± 83	0.68
COPD	14 (14.3)	6 (14.6)	8 (14.0)	0.93
Smoker	68 (69)	27 (65.6)	41 (71.9)	0.14E
Preoperative ASA use	82 (84)	34 (82.9)	48 (84.2)	0.87
Preoperative clopidogrel use	27 (28)	12 (29.3)	15 (26.3)	0.75
Preoperative ACE inhibitor	35 (36)	15 (36.6)	20 (35.1)	0.88
Preoperative statin use	78 (80)	31 (75.6)	47 (82.5)	0.41
Preoperative CCB	15 (15.3)	3 (7.3)	12 (21.1)	0.06

Data presented as mean ± standard deviation, median (interquartile range [IQR]) or number (%). Abbreviations: BMI – body mass index, MI – myocardial infarction, PCI – percutaneous coronary intervention, CARE – cardiac anesthesia risk evaluation score, Ventricle Class III/IV – ejection fraction < 40%, Hb – hemoglobin, Cr – creatinine, COPD – chronic obstructive pulmonary disease, ASA – aspirin, ACE – angiotensin converting enzyme, CCB – calcium channel blocker.

valve replacement surgery (3 patients). One patient was excluded as there were no grafts to the left circulation at the index surgery. The flow of patients in the study is presented in Fig. 1. The final study population included 98 patients comprising 263 grafts (143 arterial grafts, 120 venous grafts). All conduits had single targets. The median time from date of surgery to date of repeat catheterization was 559 days (IQR 374, 910). The maximum and minimum follow-up times were 191 and 1749 days respectively. Baseline demographics of the entire study population are presented in Table 1. Forty-three patients were on ACEi at the time of discharge post CABG and also at the time of catheterization. An additional 14 patients commenced ACEi in the interim, but the start date was not available.

4.2. Graft type and patency

Four different graft types were used for bypasses including: left internal thoracic artery (LITA), right internal thoracic artery (RITA), radial artery (RA), and saphenous vein grafts (SVG). A total of 96 patients (98%) received a LITA, 34 (35%) a RITA, 12 (12%) a RA, and 78 (80%) a SVG. The LITA had the highest patency rate (96.9%), followed by the RITA (88.2%), SVG (78.3%) and the RA (76.9%). Overall, there was a superior patency rate of ITA versus SVG ($p = 0.010$). Multivariable analysis revealed loss of patency were significantly associated with ACE inhibitor use ($p < 0.001$), smoking ($p = 0.026$), and use of SVG ($p = 0.010$).

4.3. Vessel system involvement – location and baseline disease severity

The risk of proximal disease progression differed significantly depending on the vessel territory involved ($p = 0.029$): progression in the LAD artery was the highest (43.9%), followed by the right coronary (31.1%), and the left circumflex (26.6%). In order to determine the effect of baseline disease severity on upstream native disease progression, all vessels studied were divided into four baseline disease categories: <50%, 49–76%, 75–100%, and > 99%. Native upstream disease progression occurred the most in patients with <50% stenosis at baseline (81.8%), followed by those with baseline disease of 75–100% (39.8%) and 49–76% (29.1%) ($p < 0.001$).

4.4. Change in lesion length

Lesions lengths were analyzed both pre and post CABG. Of the 263 grafts, only 156 target coronaries were able to be analyzed, because of total occlusion of the native artery post-CABG. The majority of lesions did not change in length (n = 132, 84.6%), while 9.6% showed an increase (n = 15) and 5.6% (n = 9) a decrease in length.

4.5. Non-left main progression analysis

Of the 263 vessels studied, 91 showed disease progression (34.6%). Multivariable analysis showed age ($p = 0.034$), previous percutaneous coronary intervention (PCI) ($p = 0.002$), ACE inhibitor use ($p < 0.001$), CAD baseline severity ($p < 0.001$), CCS class III/IV ($p = 0.016$), and NYHA class III/IV ($p < 0.001$), to be significantly associated with non–LM upstream disease progression. The use of SVG was not a factor in native disease progression (HR 0.74 (0.49, 1.13), $p = 0.163$). Upstream native disease progression over time related to downstream graft type is demonstrated in Fig. 2.

There were 75 coronaries in which upstream disease progressed to complete occlusion (28.5%). Factors associated with progression to

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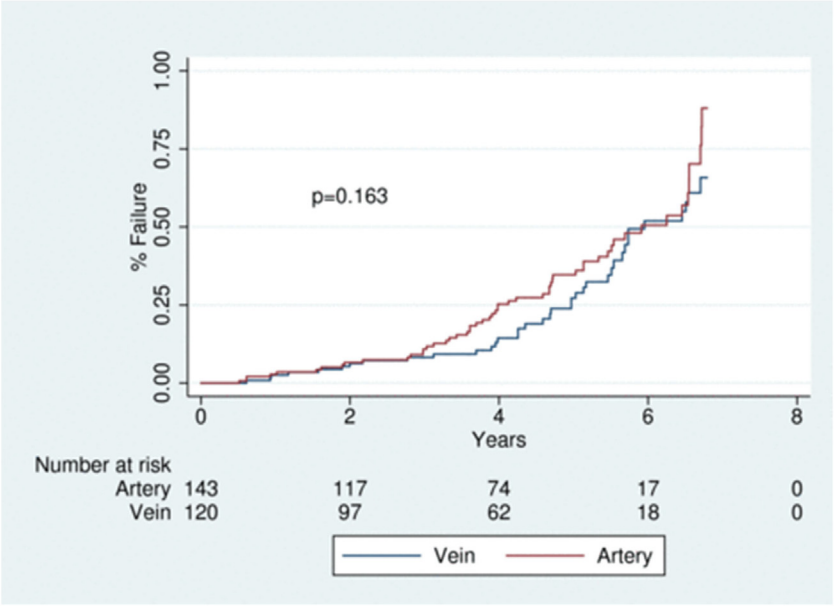

Fig. 2. Non-left main upstream progression related to graft type.

complete upstream occlusion included a previous upstream stent ($p = 0.007$), ACE inhibitor use ($p < 0.001$), and the use of a SVG at the target site ($p = 0.019$). An example of upstream native disease progression to occlusion with SVG use is demonstrated in Fig. 3. Complete occlusion was a subset of upstream native disease progression. Since more vein grafts were associated with upstream occlusion compared to arteries, a

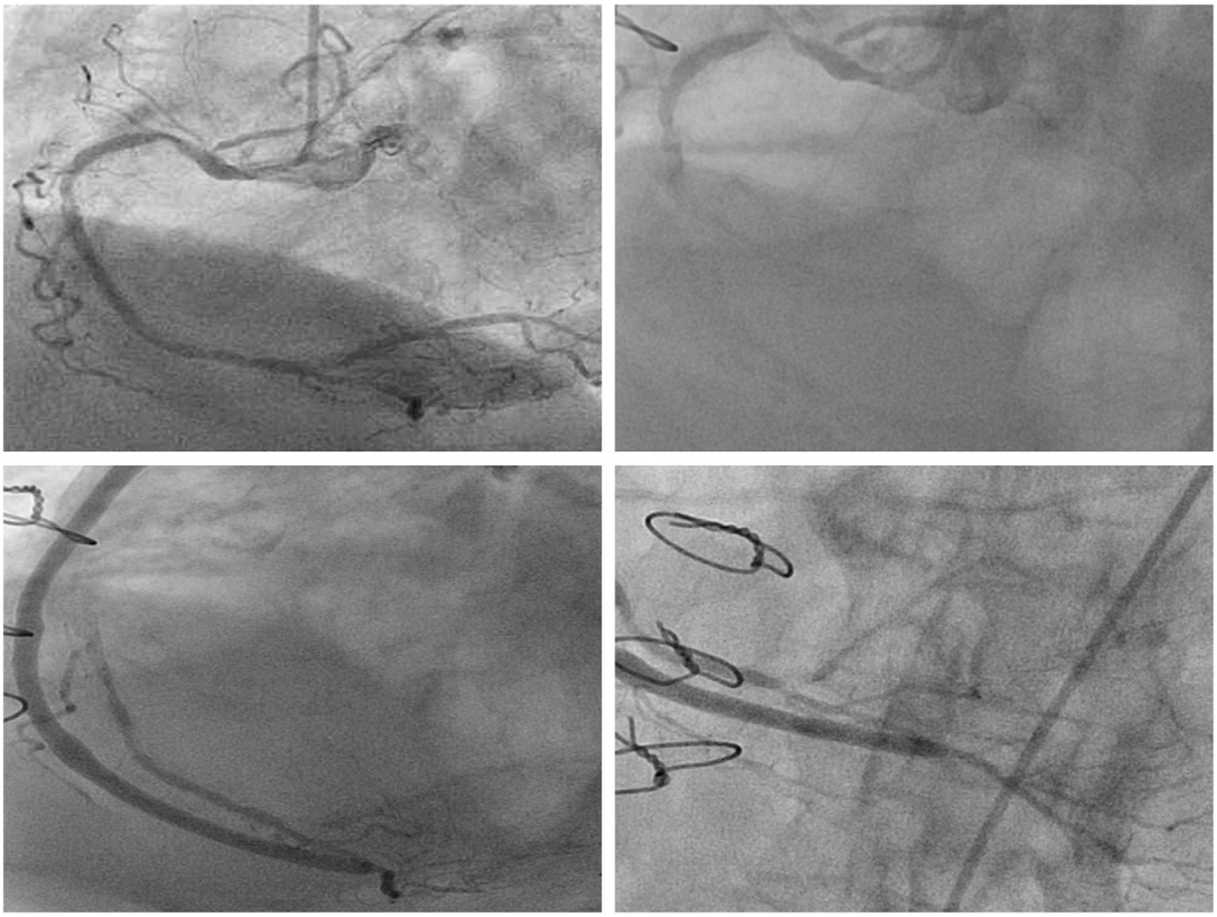

Fig. 3. Native upstream disease progression in the RCA to occlusion post CABG with SVG. Abbreviations: CABG – coronary artery bypass grafting, SVG – saphenous vein graft. Legend: (top left) proximal RCA stenosis of 70% at preoperative angiogram, (top right) Proximal RCA occlusion at follow-up angiogram (542 days post CABG), (bottom left) Patent SVG with retrograde flow to proximal RCA occlusion, (Bottom Right) Patent SVG with distal runoff.

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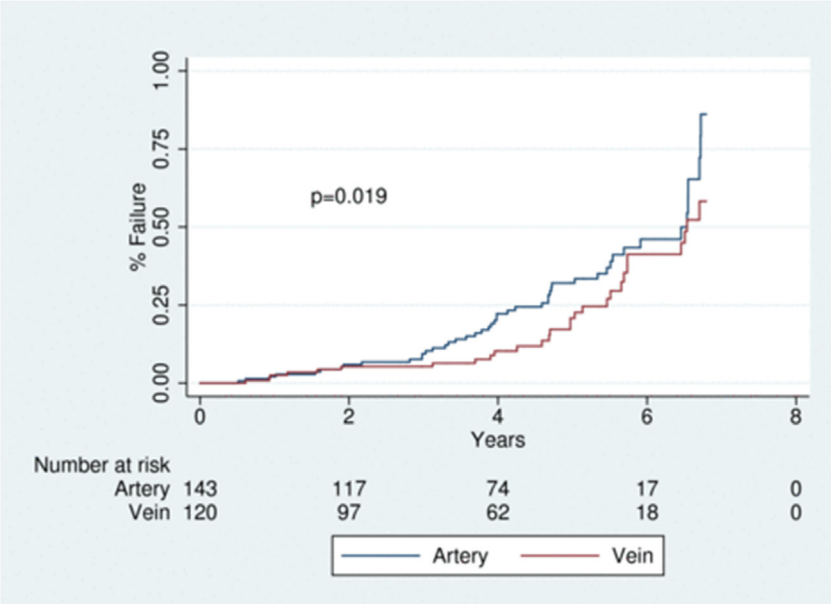

Fig. 4. Non-left main upstream progression to complete occlusion related to target graft type.

significant association was found; whereas in non-occlusive progression there was a greater mix of arteries and veins associated with progression, such that the vein graft effect was no longer seen. Development of upstream complete occlusion over time as related to graft type is demonstrated in Fig. 4 and with concomitant use of ACE in Fig. 5.

4.6. Left main progression analysis

There were two major strategies for grafting of the left coronary system (LCS): 41.8% all arterial grafts versus 58.2% mixed (vein and arterial grafts). Baseline demographics of the two groups are presented in Table 1. Graft configuration to the LM was not significantly related to death (HR 1.79 (0.35, 9.26), $p = 0.485$). There were 16 cases in which LM progression was demonstrated. Multivariable analysis of left main stenosis progression revealed peripheral vascular disease as the only significant covariate (HR 5.44 (1.92, 15.46), p = 0.001). Fig. 6 demonstrates left main progression in patients with and without PVD. Graft configuration to the LM was not significantly related to LM native disease progression (HR 1.18 (0.43, 3.24), p = 0.754).

The median follow up of patients with one artery to the left system was 4.2 years, versus 4.0 years in those with two arteries. There was no difference in survival between groups (p = 0.478). There was no difference in the cumulative incidence of coronary intervention (hazard ratio 1.51 (0.52, 4.38) p = 0.447). Fig. 7 demonstrates the cumulative hazard function of the two groups.

5. Discussion

Native CAD progression post CABG was a common occurrence over a 5-year period, occurring in 34.6% of vessels upstream of a bypass

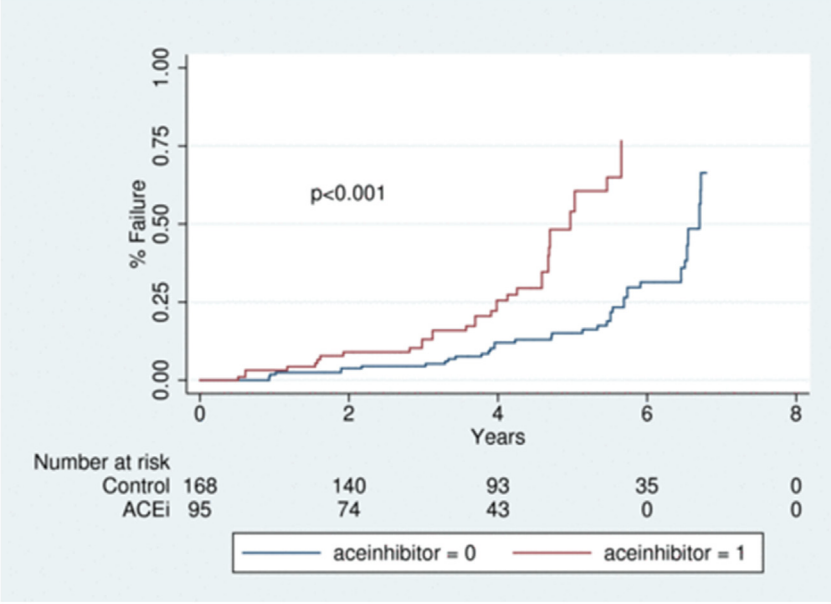

Fig. 5. Non-left main upstream progression to complete occlusion with concomitant use of angiotensin converting enzyme inhibitors.

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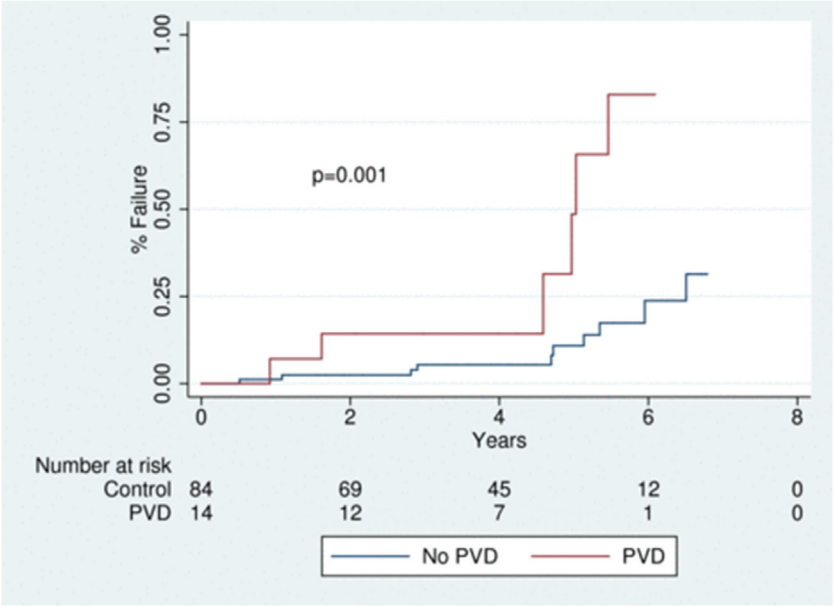

Fig. 6. Left main progression in the presence of peripheral vascular disease.

conduit and 16.3% LM disease progression. Among the predictors of native CAD progression in this study, previous PCI and ACE inhibitor use in non-LM coronaries and the presence of PVD in LM disease progression were key findings. Further to this, we did not find graft type to be associated with either LM or non-LM disease progression. There was also no difference in survival between patients that received two arteries to the left system versus one, with no difference in the cumulative incidence of coronary interventions. Lastly, factors associated with non-LM progression to occlusion included previous PCI, ACEi and SVG use.

CABG results in excellent long-term survival, however its effectiveness can be limited by recurrent symptoms, secondary to graft failure or progression of native atherosclerosis; estimated to affect more than half of patients at 25 years post CABG. [15,22] The mechanisms leading to native CAD progression and occlusion in bypassed coronaries are not fully understood. Studies to date have provided conflicting results [12,15,16], and few have examined the effects of clinical predictors on disease progression proximal to the anastomosis site. The associations we demonstrated of certain patient covariates (age, baseline disease severity, NYHA and CCS III/IV) on non-LM progression were intuitive. Together, these comorbidities reflect higher risk patients with more aggressive disease profiles at baseline.. As such, these patients are prone to further native disease progression despite surgical revascularization, as CABG aims to reconstitute blood flow distal to a stenosis, rather than treating the 'culprit' lesion. Neither hypertension nor diabetes mellitus, well-known atherogenic risk factors of CAD progression [12,15], were related to disease progression in our study. Statin use

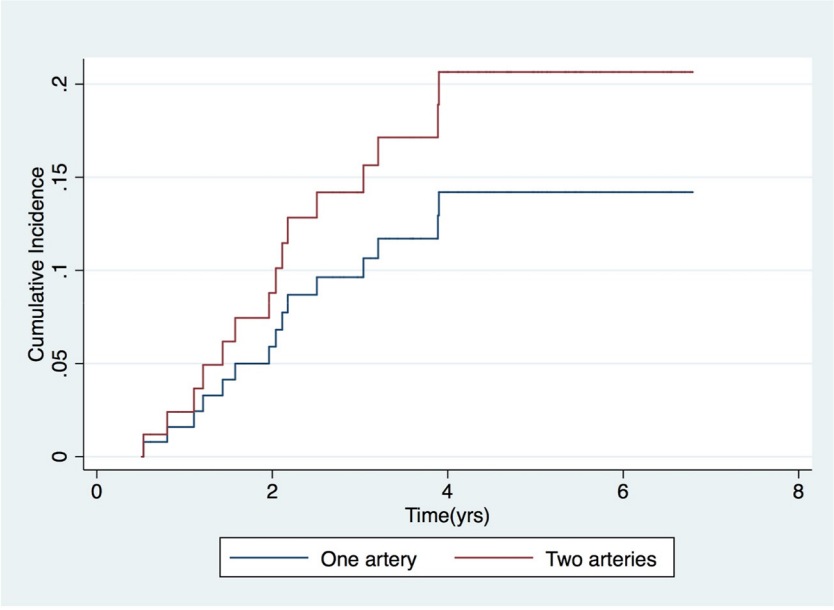

Fig. 7. Cumulative incidence function with all-cause death as a competing event ($p > 0.05$).

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had no effect on disease progression, suggesting the involvement of non-atherosclerotic mechanisms in disease progression, which would be unaffected by these drugs.

Dimitrova et al. previously showed that PCI before CABG was associated with a 20% greater risk of distal disease progression [15], while no studies have examined the relationship of PCI on proximal disease progression. We demonstrated previous PCI in the grafted vessel was also associated with increased risk of native disease progression ($p = 0.002$). This may again reflect patients with more aggressive CAD, as they have now required repeat interventions in a treated coronary. Equally important mechanisms, but beyond the scope of this paper, are the purported proinflammatory states coupled with the presence of denuded coronary endothelium post PCI; which may hasten native CAD progression. [23] Clinical studies examining the impact of PCI prior to CABG are consistent with our findings, showing worse outcomes and increased intervention rates in this group. [23–25]

Unexpectedly, an association of ACEi on disease progression and complete occlusion in native non-LM coronaries ($p < 0.001$ for both) was observed. Data on ACEi use post CABG is sparse and inconsistent. Although several randomized clinical trials have demonstrated mortality and morbidity benefits of ACEi use in patients with cardiovascular disease (CONSENSUS [26] and HOPE [27]), studies to date have failed to answer the effect of these drugs on the reoccurrence of angina, restenosis, or progression of atherosclerosis post CABG. [28,29] Studies by Ribichini et al. and Rouleau et al., have shown ACEi do not reduce risk of graft degeneration or occlusion, and do not appear to improve clinical outcomes post CABG respectively. [28,30] In a study of 3718 patients 65 years or older post CABG, Kalavrouziotis et al. also showed postoperative ACEi use had no independent effect on mortality or recurrent ischemic events after CABG. [31] The double-blind, placebo-controlled IMAGINE study of 2553 patients comparing Quinapril use post CABG also failed to demonstrate a beneficial effect of postoperative ACEi use 3 years post CABG, and was actually associated with an increase in adverse events, particularly recurrent angina. [30] Thus the associations of ACEi use found in our study are not surprising, and suggest ACEi use may be haphazard in patients post CABG; however this warrants confirmation in future studies. An alternative explanation to the association we found with ACEi use, may simply be a reflection of a more unwell cohort, suffering from worse hypertension and LV failure.

In comparison to non-LM progression, the only covariate associated with LM disease progression was peripheral vascular disease ($p = 0.001$). This is consistent with Dimitrova et al., who also found PVD to be an independent predictor of disease progression. [15] PVD causes narrowing of blood vessels outside of the heart and brain, which are larger in caliber than epicardial coronary arteries. PVD may represent a propensity for atherosclerosis progression in larger vessels, including the LM, whereas there may be relative sparing of smaller epicardial vessels. This finding supports patients with PVD to be a higher-risk cohort, raising the possibility of benefits with P2Y12 inhibitor use, with previous studies confirming benefits in this subgroup. [32,33]

Graft configuration to the LM was not significantly related to disease progression ($p = 0.754$) or death ($p = 0.485$), suggesting revascularization of the lateral wall with either artery or SVGs does not affect native LM disease progression or survival. This is in contrast to Hamada et al. and Dimitrova et al., who both showed arterial grafting to have a strong protective effect; however, neither of these studies examined proximal disease progression. [13,15] In a study that did examine proximal disease progression, Manninen et al. found native disease progression was more common in segments bypassed with SVG than with arterial grafts (OR 2.03, $p = 0.001$). [34] It is unclear why a similar result was not found in our study, but it may be related to technical factors, such as the quality of anastomoses performed with SVGs, as well as inherent characteristics of the native vessels bypassed. Overall, there were only 7 deaths in the entire study population during follow up. There was no association between non-LM progression and death ($p = 0.18$), however there was an association between LM progression and death ($p = 0.049$).

Progression to complete occlusion in non-LM coronaries occurred in 28.5% of the patients, and was significantly associated with ACEi use, previous PCI, and SVG (all $p < 0.02$). The latter finding is consistent with Tanake et al., who also showed progression from stenosis to occlusion in the proximal coronary artery post CABG occurred more often in vessels bypassed with SVGs. [35] One plausible explanation for the increased rate of occlusion found in vessels bypassed with SVGs relates to competitive flow between the native vessel and the graft [36]. Unlike SVG, arterial grafts have a greater ability to regulate flow in response to flow competition. [13,37] For example, ITA graft flow decreases much more than SVG flow during competition [37,38], and Griffith et al. suggested that the increased flow in SVGs markedly reduces the flow through the stenotic native coronary, accelerating native disease progression. [39] High competitive flow has also been demonstrated to result in low oscillatory wall shear stress leading to nonlaminar flow [16], endothelial dysfunction [40], and a procoagulant state [41], which have all been linked to enhanced CAD progression and occlusion.

Native disease progression was also dependent on the severity of native disease pre CABG. Previous studies examining the effect of baseline CAD severity on native disease progression have given mixed results. Both Pereg et al. and Pond et al. [9,16], showed a positive correlation with baseline disease stenosis and native disease progression, while Kronke, Cashin, and Cosgrove et al. showed an inverse relationship. [10,14,38] The results of our study are consistent with Cosgrove et al., where bypassed vessels with <50% stenosis showed the greatest degree of progression. This can be attributed to a higher degree of competitive flow that exists when bypassing vessels with <50% stenosis, resulting in the same aforementioned mechanisms that lead to accelerated disease progression. [39–41] Patency rates of ITA grafts were significantly higher than SVG with the LITA having the highest patency. Studies examining patency rates of coronary grafts have consistently shown the superiority of ITA grafts over SVGs [42].

6. Limitations

There are several limitations of this study that must be considered. First, the sample size was small. Secondly, as follow-up angiography was not a routine, it was only based on those that happened to have received them because of symptoms; thus, this may be a bias as only those with suspected events would have had follow-up studies. Accordingly, only short-term results were available based on the available angiograms. As such, the results of this study may not be a true reflection of native CAD progression in entire population. As a consequence of the non-routine nature of the follow-up angiograms, the median follow-up of 1.5 years in the study is likely early to determine the effects of SVG versus arterial grafts on CAD progression, as patency rates of both conduits are still very high. Despite this, SVG was found to be significantly associated with progression to occlusion. Third, coronary angiography is not the best way to assess CAD progression and no quantitative coronary measurements were performed.

7. Conclusion

We found the risk of proximal disease progression post CABG to be multifactorial, and not associated with type of graft used. In contrast, the risk of progression to complete occlusion was more common in segments bypassed with SVG than with arterial grafts.

Declaration of Competing Interest

There are no conflicts of interest to declare.

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