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Cardiac Catheterizations in Patients With Acute Coronary Syndrome and Prior Coronary Bypass Surgery: Impact of Native *vs* Graft *vs* Absent Culprit Lesions on Clinical Outcomes and Treatment Strategy

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ABSTRACT

Background: In patients with prior coronary artery bypass graft surgery (CABG), acute coronary syndrome (ACS) is not uncommon. This study investigated treatment strategy and compared clinical outcomes for native, graft and absent culprit lesions.

Methods: Single-center retrospective cohort study. From July 2010 to July 2019, 642 consecutive ACS patients with prior CABG were screened for eligibility. The primary endpoint was major adverse cardiovascular events (MACE) at 1 year, a composite of all-cause mortality, myocardial infarction, stroke and ischemia-driven revascularization.

Results: A total of 549 patients were included, with 215 (39.2 %) having native culprits, 256 (46.6 %) graft culprits and 78 (14.2 %) no clear culprits. Patients with native culprits were treated with native PCI in 94.0 %, re-CABG in 0.9 % and optimal medical therapy (OMT) in 5.1 %. Patients with graft culprits were treated with native PCI in 14.1 %, graft PCI in 81.2 %, re-CABG in 0.8 % and OMT in 3.9 %. All patients without a clear culprit received OMT. The cumulative incidence of 1-year MACE was 24.7 % for native *vs* 26.2 % for graft *vs* 21.8 % for absent culprits. Kaplan-Meier curves did not differ significantly. In patients with graft culprit, no significant difference in 1-year MACE was observed between native PCI and graft PCI (30.6 % *vs* 25.5 %, $p = 0.36$).

Conclusions: This retrospective study shows that in ACS patients with prior CABG, MACE occurred frequently and was comparable for native, graft and absent culprits. Native PCI as treatment strategy for patients with a graft culprit was relatively common, with no significant difference in MACE as compared to graft PCI.

1. Introduction

In patients with an acute coronary syndrome (ACS), prior coronary artery bypass graft surgery (CABG) is not uncommon and associated with worse clinical outcomes [1–6]. As current guidelines recommend an invasive treatment strategy in these patients, coronary angiography and percutaneous coronary intervention (PCI) are often performed to detect and treat culprit lesions [7,8]. These culprits can be located in native coronary arteries, bypass grafts, or can be absent.

Studies investigating the clinical impact of a culprit's location or its absence are limited. A few small studies have compared clinical outcomes for native *vs* graft culprit lesions, showing conflicting results [4,9,10]. In addition, a large registry comparing native *vs* graft PCI, found higher unadjusted mortality and event rates for native PCI [11]. However, this study focused on the treated vessel type, ignoring the fact that patients with a graft culprit can also be treated with PCI of a native coronary artery to achieve reperfusion. Of note, studies investigating the clinical impact of native PCI as treatment strategy for graft culprits in ACS patients are currently lacking.

To obtain more knowledge with respect to invasively managed ACS patients with prior CABG, the main goal of this study was to compare patients with a native *vs* graft *vs* absent culprit lesion, investigating subsequent treatment strategy and potential differences in major adverse

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cardiovascular events (MACE). The secondary objective was to compare clinical outcomes for native PCI *vs* graft PCI in patients presenting with a graft culprit.

2. Material and methods

2.1. Study design and patient population

We performed a single-center retrospective cohort study in a tertiary care facility with on-site cardiothoracic surgery. All consecutive ACS patients with prior CABG undergoing coronary angiography or PCI between July 1st 2010 and June 30th 2019 at the Erasmus University Medical Center in Rotterdam, the Netherlands, were screened for eligibility.

Acute coronary syndrome included ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation ACS (NSTE-ACS) and was diagnosed by the treating physician according to the guidelines at the time of admission. Non-ST-segment myocardial infarction and unstable angina were both considered NSTE-ACS. Patients undergoing coronary angiography or PCI within 48 h post-CABG were excluded, as well as patients with ACS-related subclavian stenosis or prior heart transplantation.

After assessment of procedural records and angiographic findings, included patients were divided into 3 groups: 1) native culprit; 2) graft culprit; or 3) absent culprit. A native culprit was defined as a significant lesion (at least 50 % angiographic stenosis) in an ungrafted native coronary artery or distal to the anastomosis of a grafted native coronary artery. A graft culprit was defined as a significant lesion in a coronary arterial bypass graft or saphenous venous graft (SVG). Acute angiographic findings (*e.g.* thrombus) and/or dynamic electrocardiogram changes were used to help identify the culprit lesion. In case of any uncertainty regarding the presence or location of a culprit, patients were discussed in a consensus meeting (FG, KM, WD). If patients underwent multiple procedures during the study period, the first cardiac catheterization was listed as the index procedure.

The Ethical Committee of the Erasmus University Medical Center waived the need for informed consent, since the study was not subject to the Dutch Research on Humans Subjects Act.

2.2. Procedure description and treatment strategy

All procedures were performed in line with treatment protocols at that time. Patients received heparin and in case of transradial access a spasmolytic cocktail consisting of verapamil and nitroglycerin.

After coronary angiography, the following treatment strategies were distinguished for native, graft and absent culprits: 1) PCI of native coronary artery, 2) PCI of graft, 3) *Re*-CABG, or 4) optimal medical therapy (OMT). In case angiographic findings were first discussed with a cardiac surgeon, the recommended treatment strategy of the (*ad-hoc*) Heart Team was selected as the treatment strategy for this study.

If PCI was performed, procedural success was defined as thrombolysis in myocardial infarction (TIMI) 3 flow with <30 % angiographic stenosis. Hemostasis was achieved with a transradial compression device in case of transradial access, and an arterial closure device or manual compression in case of transfemoral access. Medication use was prescribed according to the guidelines at the time of the cardiac catheterization, including aspirin, a P2Y12 inhibitor (clopidogrel, prasugrel or ticagrelor) for 1 year, and a statin. In case of atrial fibrillation, a vitamin K antagonist or a direct oral anticoagulant was added to the prescription.

2.3. Study endpoints

The primary study endpoint was MACE at 1 year, a composite of all-cause mortality, myocardial infarction (MI), stroke and ischemia-driven revascularization. Stroke and MI were diagnosed by the treating physician based on guidelines at the time of event and ischemia-driven revascularization was defined as revascularization driven by symptoms and/or objective evidence of ischemia. The other endpoint of interest was MACE at 30 days.

2.4. Data collection

All patient and procedural data was obtained from the hospital's electronic medical record system and entered in an electronic database. To determine if events had occurred, all medical records were screened and data on all-cause mortality was extracted from the municipal civil registry. A questionnaire was sent to every patient alive to complete follow-up and a telephone survey was conducted if patients did not respond. Only after explicit patient's permission, the treating physician was asked for additional information to complete follow-up data. Procedural records and angiographic findings were assessed to obtain data on the selected treatment strategy.

2.5. Statistical analysis

Continuous variables were presented as medians (interquartile ranges (IQR)) and were compared with Mann-Whitney *U* test and Kruskal-Wallis one-way ANOVA test. Categorical variables were reported as counts with percentages and compared with the Pearson's $\chi 2$ test or Fisher's exact test. The Kaplan-Meier method was used to visualize cumulative incidence at 1 year. Log-rank test was performed to assess whether curves differed significantly. Furthermore, unadjusted and adjusted hazard ratios (HRs) with 95 % confidence intervals (CIs) were calculated. For the adjusted HRs a propensity score, including all presented baseline variables, was obtained. To identify if baseline characteristics were potential predictors for native or graft culprits, multivariable logistic regression was performed. SPSS version 28.0 was used for statistical analysis and p values below 0.05 were considered significant.

3. Results

3.1. Study population and baseline characteristics

After screening 642 consecutive patients, a total of 549 patients were included in the study. Eighty-nine patients were excluded due to cardiac catheterization within 48 h post-CABG, 3 had ACS-related subclavian stenosis and 1 had undergone prior heart transplantation. In total, 215 patients (39.2 %) had a native culprit lesion and 256 patients (46.6 %) had a graft culprit lesion. In 78 patients (14.2 %) no clear culprit was present.

Baseline characteristics are shown in Table 1. Median age was 72 to 74 years and did not differ significantly between groups. Patients with a graft or absent culprit were more often male. Cardiovascular risk factors were common and did not differ significantly between groups. Patients presented with STEMI in 18.1 %, 21.5 % and 7.7 % for native, graft and absent culprit lesions, respectively ($p = 0.02$). In most patients, CABG was performed with use of the left internal mammary artery (LIMA) and a SVG. The median time between (first) CABG and ACS presentation was 11 years (IQR 5–18) for patients with a native culprit, 15 years (IQR 8–20) for patients with a graft culprit, and 13 years (IQR 8–18) for patients without a clear culprit.

3.2. Procedural characteristics and treatment strategy

Procedural characteristics are shown in Table 2. A transfemoral access strategy was used in 49.3 to 52.7 %. In the native culprit group, the left circumflex artery was the most common infarct-related artery (36.7 %), followed by the right coronary artery (31.6 %) and the left anterior descending (22.8 %). In patients with a graft culprit, SVG was the infarct-related artery in 92.2 %, followed by the LIMA in 6.3 %.

Table 1
Baseline characteristics.

Variable	Native Culprit ($n = 215$)	Graft Culprit ($n = 256$)	p	Absent Culprit ($n = 78$)	p
Age (years)	72 (66–79)	74 (66–79)	0.11	73 (66–78)	0.23
Male	152 (70.7)	207 (80.9)	0.01	64 (82.1)	0.02
Hypertension	170 (79.1)	207 (80.9)	0.64	65 (83.3)	0.74
Hypercholesterolemia	166 (77.2)	202 (78.9)	0.67	63 (80.8)	0.82
Diabetes	83 (38.6)	97 (37.9)	0.87	30 (38.5)	0.99
Family history	65 (30.2)	76 (29.7)	0.89	32 (41.0)	0.16
Current smoker	23 (10.7)	30 (11.7)	0.73	7 (9.0)	0.78
Prior stroke	34 (15.8)	36 (14.1)	0.59	11 (14.1)	0.85
Prior MI	99 (46.0)	124 (48.4)	0.61	37 (47.4)	0.88
Prior PCI	84 (39.0)	104 (40.6)	0.74	34 (43.6)	0.80
Atrial fibrillation	43 (20.0)	42 (16.4)	0.31	21 (26.9)	0.11
Peripheral artery disease	46 (21.4)	60 (23.4)	0.60	16 (20.5)	0.80
Chronic kidney disease	49 (22.8)	67 (26.2)	0.41	13 (16.7)	0.21
Clinical presentation					
NSTE-ACS	176 (81.9)	201 (78.5)	0.37	72 (92.3)	0.02
STEMI	39 (18.1)	55 (21.5)	0.37	6 (7.7)	0.02
CABG					
Time since (years)[a]	11 (5–18)	15 (8–20)	<0.01	13 (8–18)	0.02
Graft anatomy known	202 (94.0)	247 (96.5)	0.20	75 (96.2)	0.40
LIMA used	171 (79.5)	209 (81.6)	0.63	69 (88.5)	0.24
RIMA used	21 (9.8)	11 (4.3)	0.02	7 (9.0)	0.05
SVG used	194 (90.2)	241 (94.1)	0.11	69 (88.5)	0.16

Values are median (interquartile range) or n (%).
ACS = acute coronary syndrome, CABG = coronary artery bypass graft, LIMA = left internal mammary artery, MI = myocardial infarction, NSTE-ACS = non-ST-segment elevation acute coronary syndrome, PCI = percutaneous coronary intervention, RIMA = right internal mammary artery, STEMI = ST-segment elevation myocardial infarction, SVG = saphenous vein graft.
[a] Time since first CABG, in case of multiple surgeries.

Of patients with native culprit, 202 (94.0 %) were treated with native PCI, 0 (0.0 %) with graft PCI, 2 (0.9 %) with re-CABG and 11 (5.1 %) with OMT (Fig. 1). Of patients with graft culprit, 36 (14.1 %) were treated with native PCI, 208 (81.2 %) with graft PCI, 2 (0.8 %) with re-CABG and 10 (3.9 %) with OMT. All 78 patients with no clear culprit were treated with OMT (100.0 %). If PCI was performed, drug-eluting stents (DES) were used in 90.1 % to 91.4 % of patients and procedural success did not differ significantly for native *vs* graft culprits (93.6 % *vs* 93.0 %, $p = 0.82$). Embolic protection was used in 17.2 % of patients with a graft culprit.

Table 2
Procedural characteristics.

Variable	Native Culprit ($n = 215$)	Graft Culprit ($n = 256$)	p	Absent Culprit ($n = 78$)	p
Transradial access	109 (50.7)	121 (47.3)	0.28	38 (48.7)	0.42
Culprit vessel					
RCA	68 (31.6)	–	[a]	–	[a]
LM	19 (8.8)	–	[a]	–	[a]
LAD	49 (22.8)	–	[a]	–	[a]
LCx	79 (36.7)	–	[a]	–	[a]
LIMA	–	16 (6.3)	[a]	–	[a]
RIMA	–	4 (1.6)	[a]	–	[a]
SVG	–	236 (92.2)	[a]	–	[a]
PCI performed	202 (94.0)	244 (95.3)	0.51	0 (0.0)	<0.01
Lesion class 2B or C	164 (79.7)	213 (87.3)	0.03	–	[a]
Implanted stents	1 (1–2)	1 (1–2)	0.60	–	[a]
Total stent length (mm)	26 (16–44)	24 (16–43)	0.87	–	[a]
Drug eluting stent	182 (90.1)	223 (91.4)	0.37	–	[a]
Procedural success	189 (93.6)	227 (93.0)	0.82	–	[a]
Embolic protection device	0 (0.0)	42 (17.2)	< 0.01	–	[a]
Procedure time (min)	64 (49–83)	63 (49–84)	0.90	42 (35–59)	<0.01
Fluoroscopy time (min)	17 (11–26)	16 (11–24)	0.49	10 (7–16)	<0.01
Radiation exposure ($cGycm^2$)	5479 (3247–9870)	5302 (3318–8400)	0.36	3874 (2274–5034)	<0.01
Contrast (ml)	150 (100–218)	150 (100−200)	0.42	100 (70–125)	<0.01

Values are median (interquartile range) or n (%).
LAD = left anterior descending, LCx = left circumflex, LM = left main, RCA = right coronary artery. Other abbreviations as in Table 1.
[a] Not applicable.

Procedure time, fluoroscopy time, radiation dose, and contrast use were significantly lower in patients without a clear culprit ($p < 0.01$ for all), but comparable for patients with native and graft culprits.

3.3. Clinical outcomes

Cumulative event rates for MACE were 24.7 % for native, 26.2 % for graft and 21.8 % for absent culprit lesions (Fig. 2), with 1-year follow-up available in 97.1 % of patients. Kaplan-Meier event curves did not differ significantly for patients with a native *vs* graft culprits ($p = 0.73$), a native *vs* absent culprit ($p = 0.62$) and a graft *vs* absent culprit ($p = 0.44$). Also after multivariable propensity score adjustment, HRs were not significantly different for patients with a native *vs* graft culprit (0.97; 95 % CI 0.67–1.41, $p = 0.88$), a native *vs* absent culprit (0.90; 95 % CI 0.50–1.62, $p = 0.72$) and a graft *vs* absent culprit (1.03; 95 % CI 0.58–1.84, $p = 0.91$) (Fig. 2). Individual components of MACE did not differ significantly at 30 days and 1 year, except for ischemia-driven revascularization (lower for absent culprit lesions at 1 year, $p = 0.03$) (Table 3). In addition, a subgroup analysis comparing native, graft and absent culprit lesions in STEMI patients, showed no significant differences in 1-year MACE nor in its individual components (supplemental table 1).

In patients with a graft culprit lesion undergoing PCI, 1-year cumulative MACE rates were 30.6 % for native PCI and 25.5 % for graft PCI ($p = 0.36$) (Fig. 3). The propensity score adjusted HR was 1.76 (95 % CI, 0.77–4.01, $p = 0.18$).

3.4. Predictors for graft culprit lesions

After multivariable analysis, prolonged time since (first) CABG was the only independent predictor for graft culprits as compared to native culprits (OR 1.03; 95 % CI, 1.00–1.06, p = 0.03) (supplemental table 2).

4. Discussion

This retrospective study compared native *vs* graft *vs* absent culprit lesions in ACS patients with prior CABG undergoing cardiac catheterization. The main findings are: 1) Clinical outcomes did not differ significantly between groups, 2) PCI of a native coronary artery was a relatively common treatment strategy for patients with a graft culprit (14.1 %) and not significantly associated with impaired clinical outcomes as compared to graft PCI.

This study illustrates that in current clinical practice almost half of ACS patients with prior CABG present with a graft culprit (46.6 %). With the long-term patency of SVG being inferior as compared to arterial coronary bypass grafts, graft culprits were expectedly located in a SVG in 92.2 % [12]. Interestingly, the median time since (first) CABG for patients with a graft culprit was 15 years, while previous data from our institution showed the highest re-intervention incidence between 8 and 13 years [13]. A possible explanation for this discrepancy is the treatment of graft failure (re-CABG or PCI) prior to the index procedure and the exclusion of patients with type 5 myocardial infarction (within 48 h post-CABG). In the present study, ACS was frequently caused by a native culprit as well (39.2 %), demonstrating that interventional cardiologists should pay specific attention to ungrafted perfusion areas and native coronary lesions located distal to the anastomosis. Moreover, 14.2 % of patients had not a clear culprit based on coronary angiography and were treated with OMT. Identifying these patients on forehand

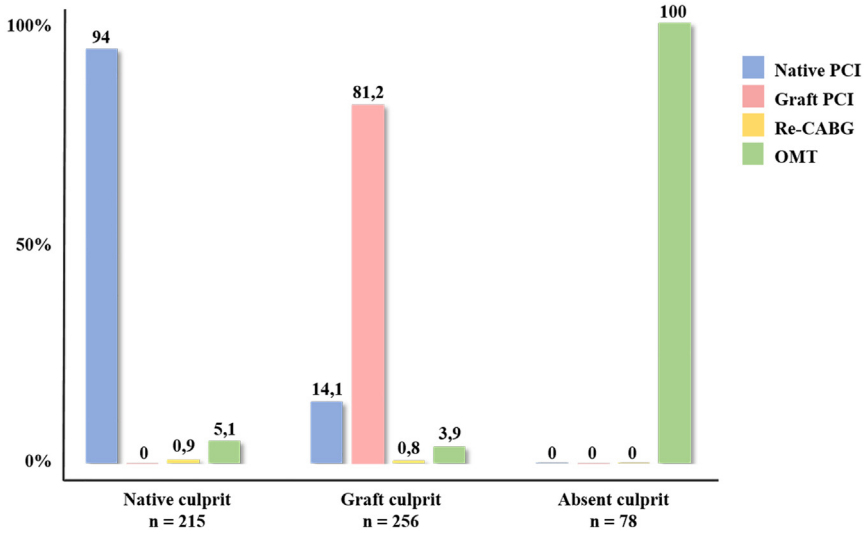

Fig. 1. Treatment strategies for native, graft and absent culprit lesions
CABG = coronary artery bypass graft surgery, OMT = optimal medical therapy, PCI = percutaneous coronary intervention.

would potentially reduce the need for an invasive approach. However, since baseline characteristics as compared to patients with a native and/or graft culprit were largely comparable in this study, this seems rather unfeasible. To the best of our knowledge, this is the largest study providing insight in the clinical impact of native, graft and absent culprit lesions in ACS patients with prior CABG. Major adverse cardiovascular events did not differ significantly between patients with a native, graft or absent culprit. Likewise, single components of MACE were comparable, except for lower rates of ischemia-driven revascularization in patients without a clear culprit. In patients with a native culprit, MACE was mainly driven by all-cause mortality, while in patients with a graft culprit, MI and ischemia-driven revascularization contributed relatively more to the composite endpoint.

Similar to our findings, a prospective registry by Kohl et al., including 249 STEMI patients with prior CABG, compared native *vs* graft *vs* absent culprits and observed no significant differences in MACE (a composite of stroke, recurrent infarction, recurrent ischemia, or death) at 30 days and 1 year [9]. Other studies only compared native *vs* graft culprits, not investigating patients without a clear culprit. Conversely to our findings, a post-hoc analysis of the randomized APEX-AMI study, including 128 STEMI patients with prior CABG, reported an increased 90-day mortality for graft culprits [4]. This survival difference could be explained by the fact that our study covered a later time period, in which the ability and experience to successfully treat graft culprits has substantially increased [14]. In a more recent prospective registry by Gharacholou et al., including 296 STEMI patients with prior CABG, long-term mortality and MACE (a composite of cardiac death, myocardial infarction, and target vessel revascularization) did not differ significantly between patients with a native or graft culprit [10]. Of note, our study also included NSTE-ACS patients, but a subgroup analysis for patients with only ST-segment elevation showed similar results as compared to the main analysis.

A large registry by Shoaib et al. investigated the impact of CABG in NSTE-ACS patients and compared outcomes based on the treated vessel type [11]. Of 18,369 patients with prior CABG, 48.0 % underwent native PCI and 52.0 % graft PCI, with higher unadjusted mortality rates in the native PCI group. However, this study ignored the fact that patients with a graft culprit can also be treated with PCI of a native coronary artery to achieve reperfusion [11]. This was illustrated in the study by Gharacholou et al., in which 2.1 % of STEMI patients with a graft culprit were treated with native PCI [10]. However, 7.1 % of patients with a native culprit were treated with primary PCI of the graft, a finding that seems reasonless from a clinical perspective.

In our study, 14.1 % of patients with a graft culprit were treated with native PCI, demonstrating that this was a relatively common reperfusion strategy. In this subset of patients, the event rates were numerically higher for native PCI as compared to graft PCI (30.6 *vs* 25.5 %), but Kaplan-Meier curves did not differ significantly. Although this retrospective study did not include assessment of the pathoanatomy of individual cases and specific culprit lesion characteristics (*i.e.* thrombus burden, total occlusion, graft size and number of prior interventions), the main reason to perform native PCI in these patients is primarily the complexity of the graft culprit (including in-stent restenosis) and/or a relatively easy accessible native coronary lesion. Avoiding high-risk PCI of these complex graft lesions and performing native PCI instead, could be the crux to better outcomes in this specific subset of patients. Nevertheless, patients with complex failing grafts for which PCI of a diseased native coronary artery is the final resort, will remain at the highest risk of future events. Larger dedicated studies are needed to further investigate the efficacy of native and graft PCI in this subset of patients. Given the improbability of performing a large randomized controlled trial, a collaborative multicenter prospective registry which includes detailed information on lesion and interventional techniques and equipment use, would be of important additional value.

Overall, 81.2 % of patients were treated with PCI, 0.7 % underwent re-CABG, and 18.0 % was treated medically, illustrating that 4 out of every 5 patients was invasively treated in the present study. Conversely, in a large registry with 42,147 CABG patients presenting with myocardial infarction, PCI was only performed in 30.0 %, while 69.2 % was treated with medication only [15]. This large numerical difference might be explained by the fact that patients in our study were 9 years younger and treated in a tertiary care high-volume PCI center with a broad experience in treating grafts percutaneously [16]. In addition, a small trial by Lee et al., including 60 NSTE-ACS patients with prior CABG, randomized to invasive management ($n = 31$) or medication only ($n = 29$), found no statistical differences in safety and efficacy outcomes between both groups [17]. These findings suggest that in patients without ST-segment elevation an invasive approach might be less beneficial. However, sample size was small and only one third of the invasively managed patients was actually treated with PCI. In the absence of larger trials, there seems no reason to deny coronary angiography in ACS patients with prior CABG.

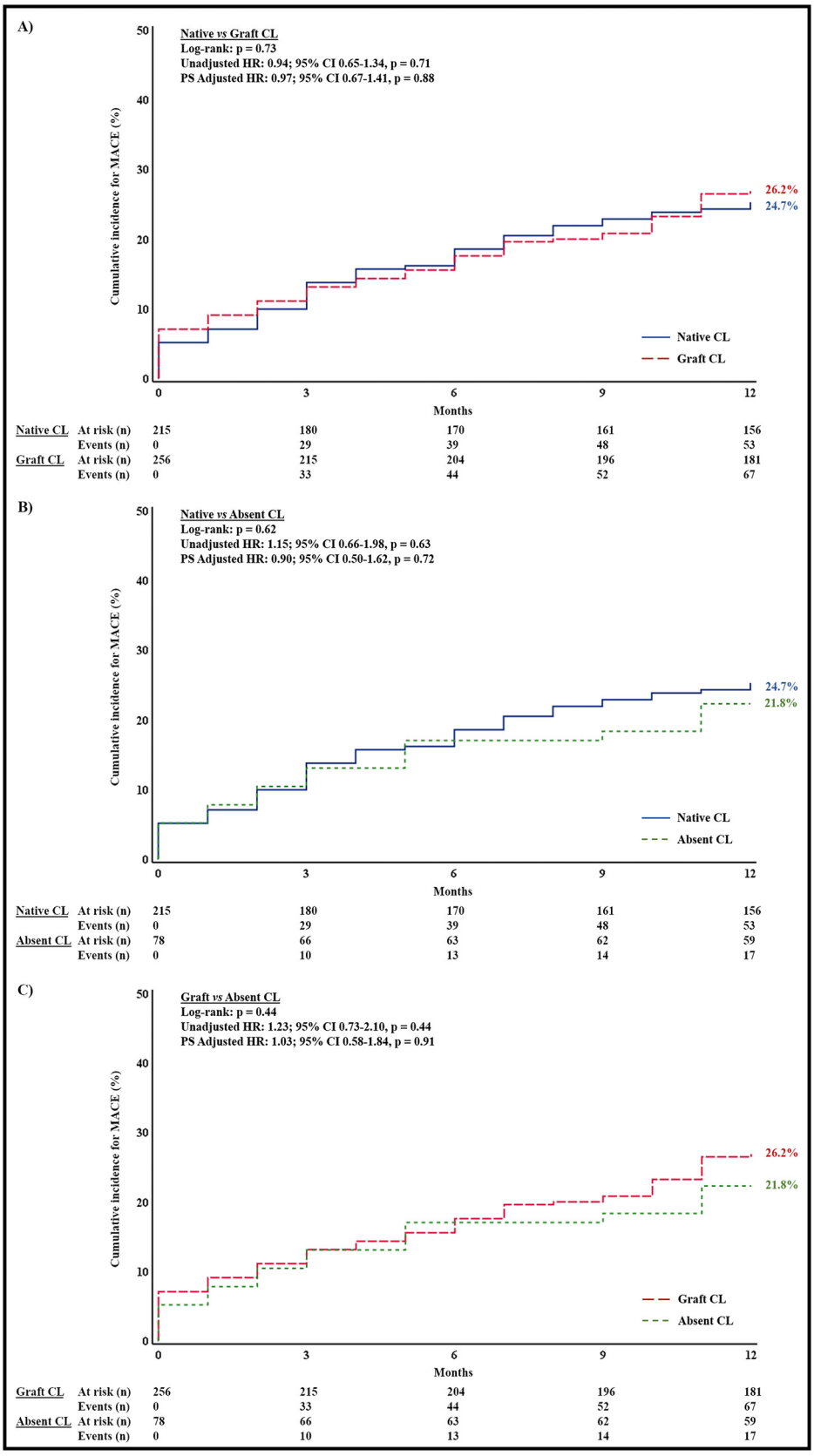

Fig. 2. Native *vs* graft *vs* absent culprit lesions; Kaplan Meier event curves with unadjusted and adjusted hazard ratios for major adverse cardiovascular events
CI = confidence interval, CL = culprit lesion, HR = hazard ratio, MACE = major adverse cardiovascular event, PS = propensity score.

Table 3
Clinical outcomes for native *vs* graft *vs* absent culprit lesions.

Outcome	Native Culprit (n = 215)	Graft Culprit (n = 256)	p	Absent Culprit (n = 78)	p
30 days					
MACE	14 (6.5)	19 (7.4)	0.70	6 (7.7)	0.91
All-cause mortality	11 (5.1)	9 (3.5)	0.39	4 (5.1)	0.66
Myocardial infarction	1 (0.5)	2 (0.8)	1.00	0 (0.0)	0.70
Stroke	0 (0.0)	5 (2.0)	0.07	2 (2.6)	0.09
Ischemia-driven revascularization	2 (0.9)	6 (2.3)	0.30	1 (1.3)	0.47
	1 year				
MACE	53 (24.7)	67 (26.2)	0.71	17 (21.8)	0.73
All-cause mortality	28 (13.0)	25 (9.8)	0.27	10 (12.8)	0.50
Myocardial infarction	9 (4.2)	19 (7.4)	0.14	3 (3.8)	0.24
Stroke	1 (0.5)	5 (2.0)	0.23	3 (3.8)	0.11
Ischemia-driven revascularization	23 (10.7)	34 (13.3)	0.39	2 (2.6)	0.03

Values are n (%).
MACE = major adverse cardiovascular even.

4.1. Limitations

First, all forms of bias related to the retrospective nature of this study should be considered, since they might have impacted our findings. To correct for potential differences in baseline characteristics between study groups, propensity score multivariable logistic regression was performed and adjusted hazard ratios were obtained. Second, specific procedural data regarding lesion characteristics (*e.g.* thrombus burden, total occlusion) and treatment techniques (*e.g.* use of intracoronary imaging, direct stenting, intracoronary medication for no-reflow) was not included in the present study. Availability of this data would have provided more insight in potential differences between native and graft culprit lesions and their treatment strategy. Finally, follow-up of clinical outcomes was mainly based on written patient questionnaires. Underreporting of adverse events due to suboptimal documentation by patients could not be completely ruled out.

5. Conclusions

This retrospective study showed that in ACS patients with prior CABG, MACE rates were comparable for graft, native and absent culprits. In patients with a graft culprit, PCI of a native coronary artery was a relatively common treatment strategy and was not associated with a significant difference in MACE as compared to graft PCI.

CRediT authorship contribution statement

Frederik T.W. Groenland: Conceptualization, Methodology, Investigation, Writing – original draft, Visualization, Project administration. **Jay Yee:** Methodology, Investigation, Writing – original draft. **Karim D. Mahmoud:** Conceptualization, Methodology, Writing – review & editing. **Rutger-Jan Nuis:** Methodology, Formal analysis, Visualization, Writing – review & editing. **Jeroen M. Wilschut:** Investigation, Formal analysis, Writing – review & editing. **Roberto Diletti:** Conceptualization, Methodology, Writing – review & editing. **Joost Daemen:** Conceptualization, Methodology, Writing – review & editing. **Nicolas M. Van Mieghem:** Conceptualization, Methodology, Writing – review & editing. **Wijnand K. den Dekker:** Conceptualization, Methodology, Investigation, Writing – review & editing, Supervision.

Declaration of competing interest

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Submission declaration

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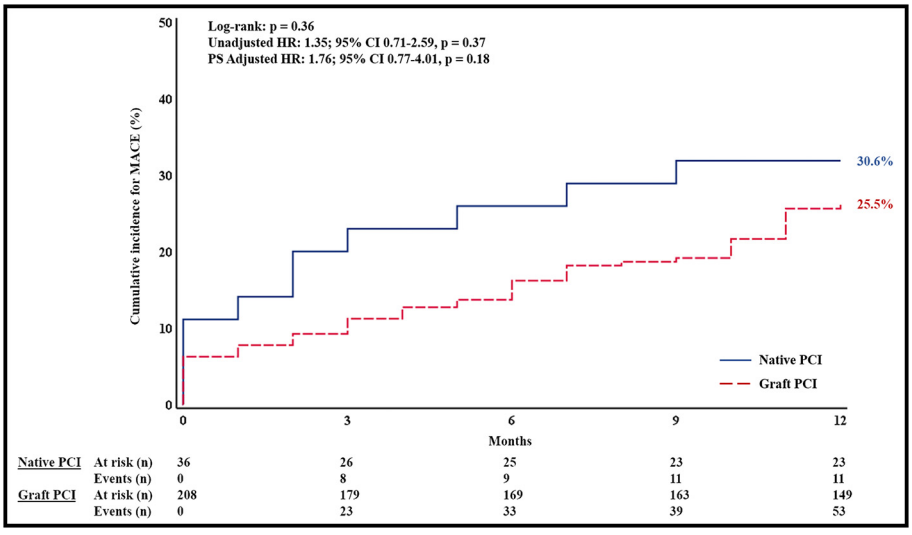

Fig. 3. Native *vs* graft PCI for graft culprit lesions; Kaplan Meier event curves with unadjusted and adjusted hazard ratios for major adverse cardiovascular events
CI = confidence interval, HR = hazard ratio, MACE = major adverse cardiovascular event, PS = propensity score.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.carrev.2022.06.257.

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