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ORIGINAL ARTICLE

The risk factors for radial artery and saphenous vein graft occlusion are different

Ramez Bahar[a], Stig E. Hermansen[a], Øystein Dahl-Eriksen[b], Rolf Busund[a,c], Per E. Dahl[a], Amjid Iqbal[b], Jan T. Mannsverk[b], Truls Myrmel[a,c], Terje K. Steigen[b,c], Thor S. Trovik[b], Dag G. Sørlie[a,c] and Kristian Bartnes[a,c]

[a]Department of Cardiothoracic and Vascular Surgery and Department of Cardiology, University Hospital North Norway, Tromsø, Norway; [b]Department of Cardiology, University Hospital North Norway, Tromsø, Norway; [c]Institute of Clinical Medicine, University of Tromsø The Arctic University of Norway, Tromsø, Norway

ABSTRACT

Objectives. To determine risk factors for radial artery and saphenous vein graft occlusion during long-term follow-up after coronary artery bypass grafting (CABG). *Methods:* From a cohort of 119 patients who had received a radial artery graft, 76 – of whom 55 also had at least one saphenous vein graft – underwent a preplanned direct angiography and anthropometric, biochemical, and endothelial function assessment 7.6–12.1 (mean 8.9) years after CABG. Comorbidity, medication, and smoking habits were also recorded. The association between these parameters and conduit longevity was analyzed in univariable and multivariable logistic regression models. *Results:* Radial artery graft occlusions were associated with higher plasma levels of high-sensitive C-reactive protein and patency was best among patients with pharmacologically treated hypertension. The sole independent risk factor identified for saphenous vein graft occlusion was tobacco smoking 8–12 years postoperatively. *Conclusion:* Our data support the contention that the pathogenesis of radial artery graft failure is distinct from vein graft disease and is related to hypertension status and systemic inflammation. These risk factors are potential targets for preventive measures. Accordingly, the study supports the eventual design of personalized secondary prevention regimens.

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Coronary artery bypass grafting (CABG); graft disease; radial artery; saphenous vein; ischemic heart disease

Introduction

To secure an optimal outcome after coronary artery bypass surgery, conduit selection and design of the secondary preventive regimen should be customized according to each patient's susceptibility to graft failure. Therefore, we need precise knowledge about modifiable risk factors. These would best be identified and quantified by prospective monitoring of broadly characterized patient cohorts with high follow-up rates and no clinically driven selection bias. Long-term graft patency should be evaluated by the gold-standard method; direct angiography. The Radial Artery BypAss GrAft Study of Tromsø (RABAGAST) was designed accordingly to compare the radial artery and saphenous vein as alternatives to complement internal mammary artery grafting [1,2]. Our studies show that the patency of radial artery and saphenous vein bypasses were similar per protocol angiographies after 2–3 as well as after 8–12 years. We here report independent risk factors for occlusion for the two graft types.

Materials and methods

The study protocol encompasses all 119 patients revascularized with radial artery grafts, mostly in combination with internal mammary (93%) and saphenous vein (71%) grafts, at The University Hospital North Norway from April 4th, 2001 to October 7th, 2003. The patient characteristics, treating institution, surgical approach, ethics approval, computerized data analysis, and per protocol assessments of graft patency have been described [1,2]. Skeletonized saphenous vein and pedicled radial artery grafts were harvested using an open technique and soaked in 0.9% NaCl with 30% autologous blood, papaverine 0.6 mg/ml and heparin 0.5 U/ml.

7.6–12.1 years postoperatively, 76 patients were subjected to direct angiography solely for the study purpose [2]. These represented 64% of the original cohort and 74% of those alive, as 17 were deceased, 6 had contraindications to the procedure (impaired renal function, 4; previous stroke, 1; contrast agent hypersensitivity, 1), and 20 declined. At the follow-up, fasting blood samples and urine were collected and analyzed by the standard methods of our hospital's laboratory except for plasma adiponectin which was quantified with an enzyme-linked immunosorbent assay at the Hormone laboratory of Oslo University Hospital. The diameters of the radial artery not harvested for grafting and the ipsilateral brachial artery were measured with an Acuson

CONTACT Kristian Bartnes kristian.bartnes@unn.no Department of Cardiothoracic and Vascular Surgery, University Hospital North Norway, Tromsø, N-9038, Norway

Sequoia ultrasonographic apparatus (Siemens, Malvern, PA) before and after near-maximal exercise with hand grippers. The endothelial function represented by the digital pulse amplitude was assessed with an Endo-PAT2000 device (Itamar-Medical, Caesarea, Israel) as described [3]. Information about co-morbidity, medication, and smoking habits was obtained from the hospital records, patient interviews, and questionnaires by patients and their primary care physicians.

Parameters linked to atherosclerosis or graft disease were tested for a potential association with long-term conduit patency. The reason for also including endothelial function assessment was our experience that 2–3 years postoperatively, radial artery graft patency was negatively associated with diabetes mellitus whereas angiotensin converting enzyme (ACE)-inhibitors appeared protective (1). We included anthropometric data (radial and brachial artery size and body mass index) since the female gender was associated with early radial artery graft failure, suggesting that vessel dimensions might influence the outcome.

Statistical computation was performed with SAS software (Cary, NC). Logistic regression models were used to estimate unadjusted odds ratios (ORs) for each independent variable and to estimate ORs in a multivariable model. Due to the low number of events, we restricted the multivariable models to only include two independent variables. The two strongest independent variables were retained in each model, using a forward selection procedure.

Results

The occlusion rates of the radial artery and saphenous vein grafts have previously been shown to be similar (0.18 and 0.16, respectively) after a mean of 8.9 years [2]. Failure of one type of graft was not linked to the failure of the other, as the pattern of graft occlusions was very close to that of a random distribution (Figure 1). Radial artery bypass occlusion was associated with elevated levels of plasma high sensitivity C-reactive protein (hsCRP), diastolic blood pressure, and absence of treatment for hypertension, whereas saphenous vein graft occlusion was linked to smoking (Table 1). No relation to graft longevity was found regarding endothelial function, vascular dimensions or body size. By multivariable logistic regression analysis, hsCRP independently predicted radial artery graft occlusion, as did smoking for saphenous vein grafts (Table 2).

According to guidelines, measures to maintain vein graft patency include platelet inhibition, lipid-lowering and smoking cessation [4]. At the 8–12 years follow-up, the vast majority of patients were on a statin and a platelet inhibitor (Table 1). Only two had a plasma low density lipoprotein (LDL) level below the recommended limit of 1.4 mmol/L [5] (data not shown). The proportion of smokers was moderately reduced from 0.36 to 0.29 over the 8–12 postoperative years. The proportion using calcium channel blockers, which may sustain radial artery graft patency [6], was 0.96 at discharge and 0.38 and 0.30 at 2–3 and 8–12 years, respectively ([1], Table 1).

Discussion

Elevated levels of hsCRP, a marker of systemic inflammation, predict cardiovascular events [7–9] and have been linked to saphenous vein graft failure [10–12]. Our finding of elevated hsCRP as an independent risk factor for radial artery graft occlusion is novel and suggests that this conduit is vulnerable to inflammatory mediators, possibly CRP itself which is known to impact endothelial function, vascular smooth muscle cells and monocytes [11]. Inflammation is a modifiable risk factor in patients with coronary artery disease as treatment with a monoclonal antibody targeting interleukin-1β improved the outcome after myocardial infarction [13]. Therefore, if reproduced in other studies, the implication of the RABAGAST trial that inflammatory mediators harm radial artery grafts identifies chronic inflammation as a potential new target for preventive measures after surgery. Larger cohorts with a substantial number of rheumatic patients appear suitable to further explore the

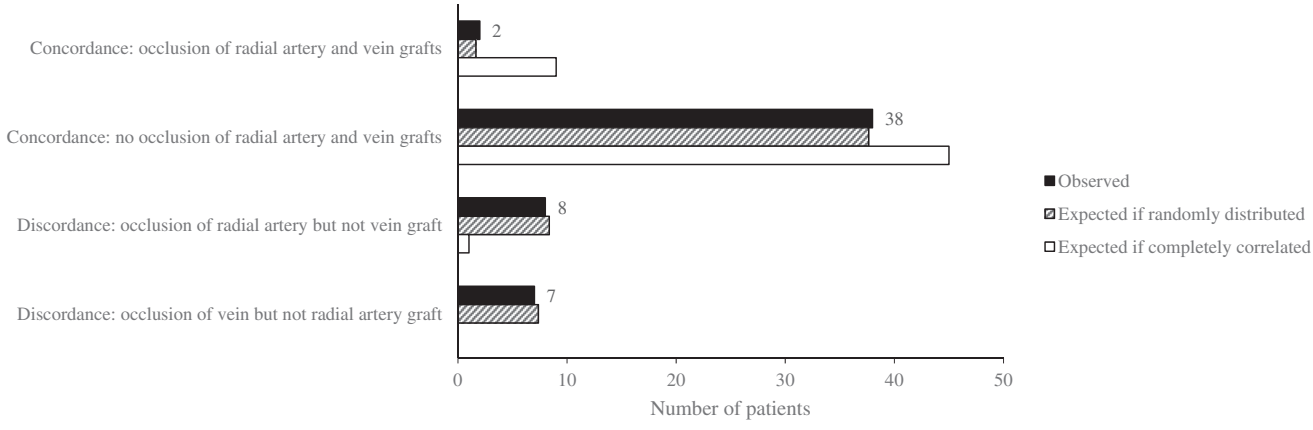

Figure 1. Independent long-term patency of radial artery and saphenous vein grafts. Angiographic results obtained solely for the study purpose are shown for patients who received combined radial artery and saphenous vein grafting during coronary bypass surgery 7.6–12.1 (mean 8.9) years before. The observed concordance and discordance of the radial artery and saphenous vein graft occlusions are shown together with the patterns expected if occlusion of these graft types were either entirely causatively linked (100% concordance) or resulted from completely independent processes (concordance and discordance distributed as predicted solely by the occlusion rates of each graft type). The observed pattern resembles that of a random distribution ($P = 1.0$ Fisher's exact test).

Table 1. Anthropometric and vascular parameters, biochemical profile, co-morbidity, smoking habits, and medication stratified according to long-term radial artery and saphenous vein graft status.

		Graft							
		Radial artery				Saphenous vein			
	Time relative to the index operation	No occlusion	Occlusion	OR (95 % CI)	*P*	No occlusion	Occlusion	OR (95 % CI)	*P*
N		62	14			46	9		
Gender (men, rate)		0.90	0.71	0.60 (0.35, 1.05)	.072	0.93	1.0	*	
Creatinine (μmol/L); mean (SD)	2–3 years postoperatively	83 (21)	84 (19)	1.09 (0.22, 5.49)	.92	83 (20)	80 (16)	0.62 (0.07, 5.55)	.67
	8–12 years postoperatively	85 (22)	87 (23)	1.17 (0.23, 5.99)	.85	85 (21)	77 (13)	0.28 (0.02, 3.49	.32
Microalbuminuria (mg/L); mean (SD)	8–12 years postoperatively	48 (119)	70 (176)	1.63 (0.33, 8.15)	.55	48 (110)	66 (121)	1.60 (0.19, 13.8)	.67
HbA1c (mmol/mol); mean (SD)	8–12 years postoperatively	6.2 (0.99)	6.6 (1.32)	1.31 (0.75, 2.30)	.35	6.3 (1.03)	6.1 (2.39)	0.86 (0.44, 1.68)	.67
High sensitivity C-reactive protein (mg/L); mean (SD)	8–12 years postoperatively	2.58 (2.06)	11.47 (15.2)	2.43 (1.20, 4.90)	.013	3.24 (5,09)	4.26 (5.84)	1.29 (0.47, 3.55)	.62
Adiponectin (mg/L); mean (SD)	8–12 years postoperatively	8.01 (3.59)	9.62 (3.85)	1.40 (0.79, 2.46)	.25	8.28 (4.24)	7.40 (3.9)	0.78 (0.30, 2.02)	.61
Low density lipoprotein (mmol/L); mean (SD)	8–12 years postoperatively	2.76 (0,52)	3.13 (1.68)	1.36 (0.79, 2.35)	.27	2.83 (0.82)	2.41 (0.68)	0.50 (0.17, 1.43)	.19
High density lipoprotein (mmol/L); mean (SD)	8–12 years postoperatively	1.30 (0.21)	1.40 (0.36)	1.30 (0.71, 2.38)	.40	1.31 (0.35)	1.24 (0.50)	0.83 (0.38, 1.84)	.65
Cholesterol (mmol/L); mean (SD)	8–12 years postoperatively	4.5 (0.5)	5.0 (1.9)	1.45 (0.84, 2.50)	.18	4.6 (0,9)	4.1 (0.7)	0.38 (0.12, 1.21)	.10
Triglycerides (mmol/L); mean (SD)	8–12 years postoperatively	1.6 (0.69)	1.7 (1.03)	1.07 (0.59, 1.96)	.81	1.6 (0.69)	1.6 (0.65)	0.99 (0.42, 2.30)	.98
Salicylate (rate)	At discharge	0.94	1.0	*		0.98	0.89	0.18 (0.01, 3.14)	.24
	2–3 years postoperatively	0.89	1.0	–[a]		0.89	0.89	–	
Non-salicylate platelet inhibitor (rate)	At discharge	0.032	0	–		0	0.22	–	
	2–3 years postoperatively	0.10	0	–		0.15	0	–	
Platelet inhibitor (rate)[b]	8–12 years postoperatively	0.84	1.0	–		0.85	1.0	–	
Latest documented postoperative platelet inhibitor use (days); mean (SD)		4508 (1382)	4173 (877)	0.75 (0.39, 1.47)	.41	4358 (1440)	4815 (990)	1.54 (0.60, 3.94)	.37
Statin (rate)	At discharge	0.90	0.93	1.39 (0.15, 12.6)	.77	0.87	1.0	*	
	2–3 years postoperatively	0.95	1.0	–		0.96	1.0	–	
	8–12 years postoperatively	0.84	0.93	–		0,85	1.0	–	
Latest documented postoperative statin use (days); mean (SD)		4253 (1472)	4042 (1511)	0.86 (0.47, 1.58)	.63	4087 (1576)	4815 (990)	1.88 (0.71, 4.92)	.20
Angiotensin converting enzyme inhibitor (rate)	At discharge	0.24	0	*		0.13	0.33	3.33 (0.65, 17.0)	.15
	2–3 years postoperatively	0.24	0.20	–		0.24	0.29	–	
	8–12 years postoperatively	0.19	0.14	–		0.17	0.22	–	
Angiotensin inhibitor (rate)	8–12 years postoperatively	0.27	0.43	*0.5 (0.15, 1.67)*	*.26*	0.24	0.22	–	
Calcium antagonist (rate)	At discharge	0.95	1.0	*		0.96	1.0	*	
	2–3 years postoperatively	0.39	0.33	–		0.45	0,25	–	
	8–12 years postoperatively	0.32	0.21	–		0.33	0.22	–	
Diabetes mellitus[c] (rate)	Ever	0.26	0.36	1.60 (0.47, 5.48)	.46	0.24	0.22	0.91 (0.16, 5.03)	.91
Hypertension[c] (rate)	Ever	0.77	0.43	0.22 (0.06, 0.74)	.014	0.70	0.56	0.55 (0.13, 2.35)	.42
Peripheral arterial disease[d] (rate)	At presentation	0.097	0.071	0.72 (0.08, 6.49)	.77	0.087	0	*	
Chronic obstructive pulmonary disease[e] (rate)	At presentation	0.032	0.071	2.31 (0.19, 27.4)	.51	0	0.11	*	
Flow mediated vasodilation (peripheral arterial tonometry ratio); mean (SD)	8–12 years postoperatively	1.79 (0.47)	1.72 (0.54)	0.87 (0.47, 1.61)	.66	1.79 (0.55)	1.79 (0.41)	1.00 (0.52, 1.92)	.99
Non-grafted radial artery diameter (mm) at rest; mean (SD)	8–12 years postoperatively	2.5 (0.6)	2.1 (0.5)	0.57 (0.30, 1.11)	.10	2.4 (0.65)	2.7 (0.47)	1.40 (0.70, 2.82)	.34
Non-grafted radial artery diameter (mm) after exercise; mean (SD)	8–12 years postoperatively	2.5 (1.0)	2.2 (0.67)	0.59 (0.25, 1.42)	.24	2.6 (1.2)	2.4 (0.50)	0.90 (0.43, 1.89)	.78
Brachial artery diameter (mm) at rest; mean (SD)	8–12 years postoperatively	4.6 (0.91)	4.2 (0.58)	0.65 (0.35, 1.23)	.18	4.5 (0.89)	4.4 (0.50)	0.91 (0.42, 1.93)	.80
Brachial artery diameter (mm) after exercise; mean (SD)	8–12 years postoperatively	4.8 (0.92)	4.4 (0.74)	0.63 (0.32, 1.24)	.19	4.7 (0.96)	4.4 (0.50)	0.71 (0.33, 1.56)	.40
Systolic blood pressure (mmHg); mean (SD)	8–12 years postoperatively	147 (20.9)	137 (25.0)	0.58 (0.29, 1.17)	.13	145 (21.2)	136 (23.3)	0.61 (0.26, 1.43)	.25
Diastolic blood pressure (mmHg); mean (SD)	8–12 years postoperatively	88 (14.7)	77 (15.1)	0.39 (0.18, 0.86)	.02	87 (16.6)	83 (14.1)	0.77 (0.35, 1.70)	.52
Tobacco smoker[f] (rate)	At presentation	0.34	0.43	*0.68 (0.21, 2.23)*	*.53*	0.30	0.56	–	
	2–3 years postoperatively	0.26	0.43	2.16 (0.65, 7.17)	.21	0.26	0.44	2.27 (0.52, 9.86)	.28
	8–12 years postoperatively	0.30	0.38	1.47 (0.42, 5.15)	.55	0.24	0.67	6.20 (1.30, 29.5)	.022
	Ever	0.73	0.57	*1.99 (0.6, 6.57)*	*.26*	0,67	0.89	–	
Body mass index (kg/m^2); mean (SD)	At presentation	28.4 (3.5)	27.1 (4.4)	0.68 (0.36, 1.29)	.24	28.0 (3.0)	28.5 (3.0)	1.24 (0.51, 3.01)	.63
Waist (cm)/height (cm) ratio; mean (SD)	8–12 years postoperatively	0.59 (0.08)	0.56 (0.09)	0.69 (0.39, 1.23)	.21	0.58 (0.07)	0.58 (0.08)	0.98 (0.46, 2.08)	.95

76 patients receiving a radial artery graft during coronary artery bypass surgery underwent direct angiography, vascular assessment and biochemical analyses 7.6–12.1 (mean 8.9) years postoperatively. Together with data for comorbidity, medication, and smoking habits, the results were stratified according to whether or not at least one bypass of the graft type indicated was occluded. Values shown are from a logistic regression analysis, except those in italics which were obtained by separate chi-square tests. SD: standard deviation, OR: odds ratio[g], CI: confidence interval, *: no estimate obtained by logistic regression.

[a]OR and P could not be calculated by the chi-square test due to $\geq$ 1 value $<$ 5 i the contingency table.

[b]Two patients were on dual platelet inhibition beyond 3 years after the index operation.

[c]Pharmacologically treated.

[d]Received treatment for limb ischemia at any time or a diagnosis of anuerysmal disease of the aorta or iliac vessels.

[e]Pharmacologically treated.

[f]On a daily basis.

[g]For continuous variables, OR are presented per SD.

Table 2. Factors independently associated with long-term radial artery or saphenous vein graft occlusion.

	Graft			
	Radial artery		Saphenous vein	
	OR (95 % CI)	*P*	OR (95 % CI)	*P*
Variable				
High sensitivity C-reactive protein	2.35 (1.24, 4.44)	.008		
Cholesterol			0.41 (0.12, 1.42)	.16
Hypertension ever treated	0.14 (0.03, 0.62)	.010		
Tobacco smoker 8–12 years postoperatively			6.02 (1.19, 30.6)	.030

A multivariable logistic regression analysis was performed in order to identify independent risk factors for graft occlusion as determined by direct angiography 7.6–12.1 (mean 8.9) years after coronary artery bypass surgery with a radial artery graft. The analyses were based on anthropometric, biochemical, and vascular assessments together with data for comorbidity, medication, and smoking habits (Table 1). Preoperative target vessel occlusion might influence graft patency [2] and was also included as a dichotomous variable. Those two variables with the highest level of significance in the univariable analysis were entered into the binary logistic regression models. OR: Odds ratios (ORs are per standard deviation for continuous variables) for the association between the parameter indicated and angiographically determined occlusion of at least one bypass of the type indicated among the 76 patients, of whom 55 also had at least one saphenous vein graft. 68 values were missing (1.9%).

putative link between chronic inflammation and radial artery graft failure.

We found radial artery graft patency to be better among patients treated for hypertension. This may be due to the properties of antihypertensive medication, being in accordance with the observations that calcium channel blockers apparently prevent radial artery graft failure [6] and ACE-inhibitors (prescribed mainly for hypertension) were independently associated with radial artery graft patency at the 2–3 years follow-up of the RABAGAST cohort [1]. Such effects have not generally been observed for vein grafts.

Our data corroborate the well-established link between saphenous vein graft failure to tobacco smoking [14]. More notable is the high vein graft longevity among our patients despite their unfavorable lipid profile. LDL drives graft degeneration which in turn is mitigated by statin therapy, most effectively when the plasma LDL concentration is extensively lowered [4,15,16]. The vast majority of our patients were long-time statin users and – although they may have fared even better with higher statin doses – they probably benefitted from statin effects besides lipid level reduction, such as inhibition of neointimal formation, smooth muscle proliferation, and inflammation [16,17].

In order to avoid potential confounders related to patient, treatment, and evaluation variables, the two graft types were compared in the same patient with the same method at the same time. Given the high follow-up rate and an evaluation protocol not driven by clinical events, selection bias should be minimal. Therefore, our finding that occlusion of the radial artery and vein grafts occurred independently is robust and consistent with the contention that the two graft types fail to dissimilar pathogenic mechanisms. Underpinning this notion is the unique sensitivity of radial artery grafts to the degree of target vessel stenosis [18] which was evident also in the RABAGAST trial; all arterial grafts anastomosed to a proximally occluded vessel maintained long-term patency [2]. Importantly, the data implicating specific predictors for graft occlusion should be interpreted cautiously as the number of observations is small. Moreover, we did neither measure the dimensions of bypassed vessels, since intraoperative probing might inflict endothelial damage nor quantify the stenoses, as fractional flow reserve measurements were not routinely available. A limitation of this study is that these target vessel properties might be differentially distributed among saphenous vein and radial artery targets and thus confound the relative patency rates.

Conclusion

The RABAGAST trial confirms and extends the understanding that radial artery and vein grafts are differentially susceptible to various mediators of graft occlusion. For both types of conduits, a defined set of risk factors are potential targets for preventive measures. Accordingly, ours and similar studies combined should ultimately guide graft selection and the design of secondary prevention regimens tailored to the individual patient.

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Disclosure statement

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