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The Effect of Patching on Reducing Restenosis in the Carotid Revascularization Endarterectomy Versus Stenting Trial

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Abstract

Background and purpose—The purpose is to determine whether patching during carotid endarterectomy (CEA) affects the perioperative and long-term risks of restenosis, stroke, death, and MI as compared to primary closure.

Methods—We identified all patients who were randomized and underwent CEA in CREST. CEA patients who received a patch were compared to patients who underwent CEA with primary closure without a patch. We compared peri-procedural and 4-year event rates, 2-year restenosis rates, and rates of reoperation between the two groups. We further analyzed results by surgeon specialty.

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Results—There were 1,151 patients who underwent CEA (753 (65%) with patch; 329 (29%) with primary closure). We excluded 44 patients who underwent eversion CEA and 25 patients missing CEA data (5%). Patch use differed by surgeon specialty: 89% of vascular surgeons, 6% of neurosurgeons, and 76% of thoracic surgeons patched. Comparing patients who received a patch versus those who did not, there was a significant reduction in the two-year risk of restenosis, and this persisted after adjustment by surgeon specialty (HR 0.35, 95% CI 0.16–0.74, P=.006). There were no significant differences in the rates of periprocedural stroke and death (HR 1.58, 95% CI 0.33–7.58, P=.57), in immediate re-operation (HR 0.6, 95% CI 0.16–2.27, P=.45), or in the four-year risk of ipsilateral stroke (HR 1.23, 95% CI 0.42–3.63, P=.71).

Conclusions—Patch closure in CEA is associated with reduction in restenosis though it is not associated with improved clinical outcomes. Thus, more widespread use of patching should be considered to improve long-term durability.

Clinical Trial Registration—http://clinicaltrials.gov/show/NCT00004732

Keywords

carotid artery narrowing; carotid artery stenosis; carotid endarterectomy; vascular closure patches

Introduction

Carotid endarterectomy (CEA) is a well-established intervention for the prevention of stroke in patients with symptomatic and asymptomatic significant carotid artery stenosis.[1–5] This operation is durable and has low rates of morbidity and mortality.[6,7] It is, however, associated with a low but significant rate of restenosis of 5–15% due to intimal hyperplasia or progression of atherosclerotic disease, with the attendant significant treatment challenges.[8–10] One of the factors potentially involved in the pathogenesis of recurrent carotid stenosis after CEA has been postulated to be the type of closure after endarterectomy.

Several retrospective studies have examined the role of primary closure of the carotid artery versus patch angioplasty in the development of carotid restenosis. These analyses have suggested that patch angioplasty is associated with a lower risk of restenosis and post-operative stroke.[11–15] However, other investigators have shown no difference in the rate of restenosis after CEA with primary closure or angioplasty.[16] Small prospective randomized trials by AbuRahma et al have indicated that patch angioplasty closure is associated with a lower risk of post-operative stroke and restenosis.[17–19] One trial randomized patients who underwent bilateral CEAs to receive patch angioplasty on one side and primary closure on the other side. This study showed that patch endarterectomy was associated with significantly less post-operative neurologic complications, stroke, and recurrent stenosis.[17]

However, overall evidence in support of the superiority of patch angioplasty versus primary closure after CEA is limited. A recent meta-analysis of trials comparing the two methods concluded that patch angioplasty may reduce the rate of restenosis, and potentially reduce the risk of postoperative ipsilateral stroke, with a non-significant trend towards reduction in mortality.[20]

We thus sought to perform a secondary analysis of a rigorous prospective randomized controlled trial comparing carotid endarterectomy versus stenting (Carotid Revascularization Endarterectomy versus Stenting Trial, or CREST).[21] The close long-term follow up of patients enrolled in this trial allowed us to analyze the rates of restenosis, peri-procedural stroke, death, major adverse events, and reoperation in patients who underwent primary closure versus patch angioplasty after CEA.

Materials and methods

The study design and primary results of CREST have been reported previously.[21,22] Briefly, 2502 patients were enrolled in 117 clinical centers in the USA and Canada between December 21, 2000, and July 18, 2008. The protocol was approved by the ethics and institutional review committees of all study institutions, and informed consent was obtained from all participants. Eligible symptomatic patients had a transient ischemic attack, amaurosis fugax, or a minor non-disabling stroke within 180 days of enrollment, and an ipsilateral carotid stenosis of 50% or greater by angiography; 70% or greater by ultrasound criteria; or 70% or greater by computed tomography, magnetic resonance angiography, or digital subtraction angiography when stenosis by ultrasound was 50–69%. Asymptomatic patients were eligible if they had a stenosis of 60% or greater by angiography; 70% or greater by ultrasound criteria; or 80% or greater by computed tomography, magnetic resonance angiography, or digital subtraction angiography when stenosis by ultrasound was 50–69%.

Patients were randomly assigned to carotid endarterectomy or carotid artery stenting within 2 weeks of planned procedure. Stroke and myocardial infarction were adjudicated by specialty committees masked to treatment assignment. All other outcomes were assessed by investigators unmasked to treatment allocation.

We included patients who were randomized and underwent CEA in CREST. Carotid endarterectomy was performed according to standard techniques on the basis of individual preferences of 477 surgeons. We individually reviewed all operative notes of primary closure CEA and excluded those patients who underwent eversion CEA. To ensure consistency in follow up timepoints, we included patients who underwent CEA within 30 days of randomization and analyzed only patients who received their assigned treatments.

Restenosis was identified using duplex ultrasound as described previously.[23] Duplex ultrasound was performed at baseline and at 1, 6, 12, 24, and 48 months after revascularization. These studies were undertaken at CREST-certified clinical center vascular laboratories with a standardized protocol that stipulated 16 doppler waveform samples at every examination.[23] All ultrasound images and doppler waveforms were analyzed at a central facility, the Ultrasound Core Laboratory at the University of Washington Ultrasound Reading Centre (URC).

We compared peri-procedural and 4-year event rates and 2-year restenosis rates, in addition to rates of reoperation between the two groups. The endpoints in this analysis were the primary endpoint of any stroke, death, or MI within 30 days of procedure and ipsilateral

stroke within 4 years of randomization. Periprocedural (within 30 days of procedure) and 4-year event rates; post-operative (up to 30 days) return to the operating room; and restenosis rate at two years were assessed. Restenosis was defined as 70% or greater diameter-reducing stenosis based on elevated peak systolic velocity of 3.0 m/s or greater on duplex ultrasound. Analysis of the frequency of high-grade restenosis and occlusion was a prespecified secondary analysis of the CREST protocol. The decision to use 3.0 m/s as the definition for restenosis was also made before unblinding of the restenosis data.

Event rates were calculated by Kaplan-Meier survival estimates. Treatment differences were assessed using logistic regression and proportional hazard models adjusting first for symptomatic status and then also for board specialty of operator. In the full CREST cohort including both CAS and CEA patients, it was determined that age and symptomatic status were the most important factors to adjust for in the analysis. As this analysis only included CEA patients, we tested to see if both age and symptomatic status were associatied with the outcomes. We determined that symptomatic status, but not age, was associated with the outcome and that inclusion or exclusion of age had almost no effect on the magnitude of the hazard ratio. As the number of events was somewhat small, and as age had little effect when included, we chose to only adjust for symptomatic status. Analyses were done with SAS (version 9.2).

Results

A total of 1,151 patients underwent CEA in CREST. We excluded 44 patients who underwent eversion CEA and 25 patients missing CEA data. We analyzed the outcomes in 1,082 patients, of whom 753 (70%) patients underwent CEA with patch angioplasty and 329 (30%) had CEA with primary closure. There were no significant differences between the two groups with respect to age, sex, comorbidities, and operative time (Table 1). Patients in the primary closure group were more likely to be white (96% vs. 93%, P=.04), symptomatic (66% vs. 49%, P<0.0001), and receive general as opposed to regional anesthesia (97% vs. 89%, P<.0001). Surgeons were more likely to shunt patients in the patch group (65% vs. 47%, P<.0001) (Table 1). There was a strong association between surgeon specialty and the use of patch: 89% of vascular surgeons and 76% of thoracic surgeons patched, while only 6% of neurosurgeons did so (Table 2). The type of patch used included 466 (62%) synthetic Hemashield Dacron patch, 217 (29%) bovine pericardial patch, 42 (6%) saphenous vein graft, 24 (3%) neck vein graft, and 4 (0.5%) unspecified.

Fifty-two patients had restenosis, of whom 27 (52%) were symptomatic and 25 (48%) were asymptomatic at baseline; in follow-up, 5 of these patients had a stroke after identification of the restenosis. Two-year restenosis rates differed significantly between the patch versus no patch groups. Restenosis was less frequent in the patch cohort when analysis was adjusted for symptomatic status (HR 0.26, 95% CI 0.14–0.45, P<.0001). This difference persisted after adjustment for surgeon specialty, with patch use strongly associated with a reduction in the risk of two-year restenosis (HR 0.35, 95% CI 0.016–0.74, P=.006) (Table 3). Of the 1042 patients in this analysis, 30 (2.9%) underwent a reintervention involving the target site or the target vessel. Of the 30 patients who underwent reintervention, 18 (60%) had restenosis, and 12 (40%) did not have restenosis (p<0.0001). Reintervention for the latter

patients may have taken place because of moderate stenosis that did not meet the protocol definition (peak systolic velocity of 3.0 m/s or greater on duplex ultrasound) and/or because of worrisome features of the plaque.No data was censored based on intervention for restenosis.

The primary end point of the CREST trial was not significantly different between the two groups (Table 4). In addition, after adjusting for symptomatic status, there were also no differences between the two groups in the rates of the primary endpoint (HR 0.74, 95% CI 0.41–1.35), as well as MI (HR 1.46, 95% CI 0.58–3.68). The rate of stroke was significantly lower in the patch cohort (HR 0.35, 95% CI 0.15–0.82, P=.02) after adjustment for symptomatic status, but was not different from the no-patch cohort after adjustment for surgeon specialty (HR 1.58, 95% CI 0.33–7.58, P=.57). The two groups were also not different in the outcomes of stroke and death (HR 1.58, 95% CI 0.33–7.58, P=.57) or the four-year risk of stroke (HR 1.23, 95% CI 0.42–3.63, P=.71) (Table 4).

Analysis of returns to the operating room revealed that, when adjusted for symptomatic status, the risk of reoperation within 30 days of CEA was significantly lower in patients who underwent patch angioplasty as compared to primary closure after CEA (HR 0.33, 95% CI 0.14–0.79, P=.02) (Table 5). However, this difference dissipated upon adjustment for surgeon specialty (HR 0.56, 95% CI 0.15–2.09, P=.39) (Table 5). The main reason for return was bleeding, which occurred in 19 of the 21 patients. One outcome thus remained significantly different after adjustment for surgeon specialty, a 65% reduction in the two-year risk of restenosis with the use of patch (HR 0.35, 95% CI 0.16–0.74, P<.01) (Table 3).

Discussion

Restenosis after CEA occurs in 5–15% of patients and presents challenges for treatment by either open surgical or endovascular approaches.[8–10] If left untreated, significant restenosis may become symptomatic.[23] Technical aspects of CEA have been examined as potential risk factors for the development of restenosis. Specifically, controversy exists regarding the optimal type of carotid artery closure after endarterectomy. Some studies have indicated superiority of patch angioplasty over primary closure in reducing restenosis, but data have been limited due to small study sizes.[20]

Arguments against the routine use of patching in CEA raise the risks of introducing a foreign body and thus creating the possibility for infection. If an autologous vein patch is used, the argument against it focuses on the possibility of development of degenerative pseudoaneurysms secondary to the presence of arterial pressure in a thin-walled vein. Potential disadvantages of using a patch is longer carotid occlusion time, longer time to achieving hemostasis and thus prolonged operative times potentially raising the risk of perioperative complications such as stroke.[24]

We performed a secondary analysis of CREST, the largest prospective randomized trial with rigorous follow up comparing CEA versus carotid artery stenting (CAS) in symptomatic and asymptomatic patients with significant carotid artery stenosis.[21] Previous analysis of restenosis after CEA versus CAS in CREST showed a similar restenosis rate of 6% for both

procedures.[23] However, patients with restenosis had a four-fold increase in the risk of ipsilateral stroke.[22] We show that, when compared to primary closure, patch angioplasty is associated with a significantly lower risk of two-year restenosis. The risks of periprocedural stroke and death, immediate reoperation, and stroke at four years post-operatively were lower with patch angioplasty when analysis was adjusted for age and symptomatic status. However, when adjusted for surgeon board specialty, these differences became no longer significant. Notably, the use of patch varied widely with surgeon specialty: most vascular and thoracic surgeons patched, while most neurosurgeons did not. CREST did not track infection or pseudoaneurysm formation, but the operative times were not longer when patch was used.

Another argument against patching concerns the fact that existing evidence in favor of patching is based on dated studies that were performed at a time prior to the widely prevalent use of aspirin and statins. Had those patients been under the most optimal medical management, perhaps patching would not be necessary for prevention of restenosis. Modern medical management of atherosclerosis was implemented in all CREST patients, including the use of statins, as well as control of diabetes and hypertension. All patients undergoing CEA were on aspirin for at least 5 days prior to the procedure, and continued on the medication indefinitely postoperatively. The use of statins was not mandatory in CREST. With the best currently available medical management in both groups, the use of patching during CEA is still associated with a decreased risk of restenosis.

Several prospective randomized trials have addressed the question of whether patching is superior to primary closure in CEA, with the limitation of low patient numbers. A Cochrane review of prospective randomized trials comparing patching versus primary closure in CEA has shown that patching was associated with a statistically significant reduction in the risk ipsilateral stroke at 30 days and one year; perioperative arterial occlusion; return to the operating room; any stroke at one year; and occlusion or restenosis greater than 50% at one year. Patching was also associated with a non-significant reduction in overall stroke, and in combined stroke and death. There were no statistically significant differences in arterial rupture or hemorrhage, rate of local infection, or cranial nerve palsy between the two groups; as well as no pseudoaneurysm formation at one year in 1,141 arteries. Although the authors note that the quality of the studies reviewed was not uniformly exceptional, the statistically significant differences found in the reviewed studies agree with our data.[20]

Other systematic reviews of randomized clinical trials comparing the effects of carotid patch angioplasty to primary closure in carotid endarterectomy have noted superiority of patching to primary closure in reducing the risks of perioperative and long term stroke and death, perioperative arterial occlusion, and restenosis in long term follow up[25]; ipsilateral stroke during the perioperative period and on long-term follow-up; acute arterial occlusion, long-term restenosis, and death.[26] Another comprehensive review identified 1,281 operations and concluded that patch angioplasty was associated with a reduction in the risk of perioperative stroke, stroke on long term follow up, and restenosis.[27] All of these studies emphasized limitations to their recommendations for patching over primary closure due to the small number of events, significant losses to follow up, and poor trial methodology and quality of the data.

Our work is in agreement with prior trials comparing patching versus primary closure in CEA in the reduced risk of restenosis with the use of a patch.[20] Most restenoses occur in the first two years after CEA,[28] and our follow up thus captures most restenoses. Known risk factors for restenosis include diabetes mellitus, hyperlipidemia, cigarette smoking, and female gender.[23] The clinical significance of reduced restenosis is historically not entirely clear. The rate of symptomatic restenosis is known to be lower than the overall rate of restenosis.[28] Notably, in a recent CREST analysis, patients with restenosis had significantly higher risk of stroke.[23] The results of this study suggest that restenosis is clinically significant in that it places the patient at a higher risk for stroke. In our study, the lower rates of restenosis with patching did not directly correlate to a reduction in perioperative and four-year stroke, as the decrease in stroke rates lost significance when analyses was adjusted for surgeon specialty.

This study does have limitations in that it is not a randomized prospective trial designed to compare patching versus primary closure a priori, and there was no secondary randomization to the type of closure after CEA. We also cannot comment on the risk of infection in this study. Longer follow up will allow for assessment of the risk of pseudoaneurysm formation.

Summary

In conclusion, our secondary analysis of CREST data supports the use of patch angioplasty for closure of arteriotomy in carotid endarterectomy. More widespread use of patching should be considered due to the clear association of patch closure with reduction in the risk of restenosis, and thus with superior long-term durability.

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Table 1

Patient characteristics

	Patch (n=753) N (%)	No Patch (n=329) N (%)	p-value
Age, years (mean ± SD)	69.2 ± 8.4	68.6 ± 9.3	0.29
Sex: male	496 (65.9)	234 (71.0)	0.09
Race: white	698 (92.7)	316 (96.1)	0.04
Presentation: symptomatic	369 (49.0)	216 (65.7)	<0.0001
Comorbidity			
Hypertension	653 (86.7)	275 (83.8)	0.21
Diabetes mellitus	232 (30.8)	107 (32.6)	0.56
Dyslipidemia	637 (84.9)	285 (87.2)	0.34
Current tobacco smoker	198 (26.8)	84 (25.7)	0.72
Prior cardiovascular disease or CABG	346 (47.3)	131 (42.0)	0.11
Left-sided carotid lesion	403 (53.5)	164 (49.9)	0.27
Procedural factors			
General (vs Block) Anesthesia	671 (89.2)	319 (97.3)	<0.0001
Shunt used	471 (64.7)	148 (47.1)	<0.0001
Procedure time*, min	173.3 ±62.9	171.9 ± 51.5	0.70

* Procedure time is missing on all patients who received Block anesthesia

Table 2

Carotid endarterectomy technique by surgeon board specialty

Specialty	CEA N (%)	Patch N (%)	Primary N (%)
Vascular surgeons	702 (65)	628 (89)	74 (11)
Neurosurgeons	233 (22)	14 (6)	219 (94)
Thoracic surgeons	142 (13)	108 (76)	34 (24)

* Board specialty was unavailable on 5 patients (3 patch; 2 no patch)

Table 3

Two-year restenosis rates after CEA with patch versus no patch

	Patch # events (rate ± SEM)	No Patch # events (rate ± SEM)	Hazard Ratio for Patch vs No Patch (95% CI)*	P-value	Hazard Ration (95% CI)†	P-value
Restenosis	20 (3.1±0.7)	32 (10.7±1.8)	0.27 (0.15,0.48)	<0.0001	0.35 (0.16,0.74)	0.006

*adjusted for symptomatic status

†adjusted for symptomatic status and surgeon board specialty

46 patients (13 no patch; 33 patch) are not included in the restenosis endpoint as they did not have ultrasounds read by the ultrasound core laboratory. 4 patients with restenosis information (2 patch; 2 no patch) did not have information on board specialty.

Table 4

Periprocedural and 4-year outcomes

	Patch # events (rate ± SEM)	No Patch # events (rate ± SEM)	Hazard Ratio for Patch vs No Patch (95% CI)*	P-value	Hazard Ratio for Patch vs No Patch (95% CI)†	P-value
Peri-procedural endpoints						
MI	19 (2.5±0.6)	6 (1.8±0.7)	1.46 (0.58,3.68)	0.43	1.07 (0.28, 3.99)	0.93
Stroke	9 (1.2±0.4)	13 (4.0±1.1)	0.35 (0.15,0.82)	0.02	1.58 (0.33, 7.58)	0.57
Stroke and death	9 (1.2±0.4)	13 (4.0±1.1)	0.35 (0.15,0.82)	0.02	1.58 (0.33,7.58)	0.57
Primary endpoint	28 (3.7±0.7)	18 (5.5±1.3)	0.74 (0.41,1.35)	0.33	1.26 (0.45, 3.56)	0.66
Four-year endpoints						
Stroke	22 (3.5±0.8)	20 (6.6±1.5)	0.54 (0.29,0.99)	0.047	1.23 (0.42,3.63)	0.71
Stroke and death	22 (3.5±0.8)	20 (6.6±1.5)	0.54 (0.29,0.99)	0.046	1.23 (0.42,3.63)	0.71
Primary endpoint	40 (5.9±0.9)	25 (8.1±1.6)	0.76 (0.46, 1.26)	0.29	1.13 (0.49, 2.63)	0.08

*adjusted for symptomatic status

†adjusted for symptomatic status and surgeon board specialty

Table 5

Return to the operating room in 30 day postoperative period

	Patch (n=752) N (%)	No Patch (n=327) N (%)	Odds Ratio for Patch vs. No Patch (95% CI)*	P-value	Odds Ratio for Patch vs. No Patch (95% CI)†	P-value
Return to operating room	9 (1.2%)	12 (3.7%)	0.29 (0.12, 0.71)	0.007	0.51 (0.13, 1.95)	0.32
Reason for return						
Stroke and thrombosis	0	1				
Other	1	0				
Bleeding	8	11				

*adjusted for symptomatic status

†adjusted for symptomatic status and surgeon board specialty