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Management and Outcome of Prosthetic Patch Infection after Carotid Endarterectomy: A Single-centre Series and Systematic Review of the Literature

C.D. Mann*, M. McCarthy, A. Nasim, M. Bown, M. Dennis, R. Sayers, N. London, A.R. Naylor

Department of Vascular Surgery, Leicester Royal Infirmary, Infirmary Square, Leicester LE1 5WW, United Kingdom

WHAT THIS PAPER ADDS

- Prosthetic patch infection following carotid endarterectomy is a rare but feared complication. Although covered stents have emerged as an alternative treatment strategy, there is limited long term data regarding late reinfection. For now, patch excision and autologous reconstruction remains the 'gold standard' and can be performed with low rates of reinfection. Revascularisation with prosthetic material should be avoided.

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ABSTRACT

Objectives: Outcomes following prosthetic patch infection after carotid endarterectomy (CEA).
Methods: Retrospective audit and systematic review.
Results: 22 patients were treated between January 1992 and April 2012, 5 having undergone their original CEA at another institution. The commonest infecting organism was *Staphylococcus*. One patient was treated by antibiotic irrigation, one was stented, while 20 underwent debridement and patch excision plus; carotid ligation ($n = 3$), vein patching ($n = 3$) or vein bypass ($n = 14$). There was one peri-operative stroke, but no peri-operative deaths. There were no reinfections at a median follow-up of 54 months. A systematic review identified 123 patients with prosthetic patch infection in the world literature. Thirty-six (29%) presented <2 months, 78 (63%) presented >6 months after the original CEA. Seventy-nine of/87 patients (91%) with a positive culture yielded *Staphylococci* or *Streptococci*. Seventy-four patients were treated by patch excision and autologous reconstruction. Four (5%) developed reinfection <30 days, but later reinfections have been reported. Seven of nine patients (78%) undergoing prosthetic reconstruction either died or suffered reinfection. Five patients were treated with a covered stent, none developing reinfection (median followup 12 months).
Conclusion: Patch infection following CEA is rare. Few have undergone stenting and long term data are awaited. For now, patch excision and autologous reconstruction remains the 'gold standard'.

Introduction

Randomised trials have demonstrated that carotid endarterectomy (CEA) has a proven role in selected patients with symptomatic and asymptomatic carotid disease.[1–4] During traditional CEA, the arteriotomy can be closed primarily or be selectively/routinely patched. No randomised trials have compared routine with selective patching, but randomised trials have shown that a policy of routine patch closure (compared with routine primary closure) is associated with significant reductions in peri-operative stroke, early thrombosis, late restenosis and late stroke.[5,6] In systematic reviews, synthetic patches (PTFE, Dacron) conferred similar short and long-term benefits (compared with autologous vein) and offer the added benefits of being readily available, they avoid groin incisions, they preserve the long saphenous vein for future coronary or peripheral vascular reconstructions and have a lower incidence of early patch rupture.[7–9] However, and in common with vascular reconstructions elsewhere in the body, the main disadvantage with using prosthetic material is the risk of infection. Interestingly, while everyone has heard of prosthetic patch

* Corresponding author. Tel.:+44 7734203808.
E-mail address: chris.mann@doctors.org.uk (C.D. Mann).

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infection, after CEA, fewer than 80 cases had been reported in the world literature prior to the current report.[10]

The aim of this audit was to establish the prevalence, presentation, management and outcome of prosthetic patch infection in a large single-centre series, supplemented by a systematic review of the literature which identified 123 patients who have been treated for prosthetic patch infection.[10–38]

Materials and Methods

Between 1st January 1992 and 1st April 2012, 2215 patients underwent CEA in the Vascular Unit at the Leicester Royal Infirmary. Of these, 198 (8.9%) underwent vein patch closure (during the first half of the study period) and have been excluded from analyses, leaving 2017 patients who underwent prosthetic patch closure (Polyester = 2012, PTFE = 5). The Leicestershire, Northamptonshire and Rutland Research Ethics Committee advised that this study did not fall under the remit of the NHS Research Ethics Committee as it was audit/service evaluation. Eight of the 22 patients were included in an earlier report of patch infection published in 2002.[39]

Primary operative technique

All 2017 patients underwent CEA under normocarbic, normothermic general anaesthesia, using systemic heparinisation, intravenous prophylactic antibiotics, routine shunting, routine tacking and routine prosthetic patching. Between 1992 and 1995, three doses of intravenous Co-Amoxiclav (1.2 g) were administered for antibiotic prophylaxis starting immediately prior to induction of anaesthesia and 8 hourly thereafter. This policy changed to three doses of intravenous Cefuroxime (750 mg) and Metronidazole (500 mg) between 1995 and 2009, but reverted back to intravenous Co-Amoxiclav from 2010 to the current date. There is no evidence that changes in antibiotic prophylaxis influenced the incidence of patch infections. All patients with an accessible temporal window underwent routine intra-operative transcranial Doppler (TCD) monitoring of blood flow velocity in the middle cerebral artery (MCA) and completion angioscopy.

Post-operative care after original CEA

Patients were monitored in theatre recovery for 3–6 h. In an attempt to minimise early neck haematoma formation (which might predispose towards early wound infection), blood pressure was carefully monitored, although it was not until 2008 that written guidance on managing post CEA hypertension was provided.[40] Anyone with a clinical wound infection was treated with oral or intravenous antibiotics as appropriate. The majority of patients were discharged on day 3. Patients were reviewed in the outpatient clinic at 6 weeks and thereafter discharged with instructions to return if any new symptoms or wound complications developed. This audit therefore, only includes patients in whom a diagnosis of patch infection was suspected and who were then referred to the Vascular Unit for treatment. It is accepted that a small but indeterminate number may have suffered a late patch infection and not have been referred back.

Principles of managing carotid patch infections

Where ever possible, patients with suspected patch infection had routine bloods (haematology/biochemistry) and blood cultures taken, as well as microbiological swabs from any discharging wounds. All patients were started on parenteral antibiotics (Cephalosporin and Metronidazole until 2002, Co-amoxiclav thereafter), revised once cultures/sensitivities were available and continued for 6 weeks following surgery or until there was no clinical evidence of infection. Intravenous Vancomycin was used in cases of early infection (<60 days) to cover against MRSA. Where possible, surgery was delayed until cultures were available. Our policy was to perform Duplex ultrasound (unless contraindicated by massive haemorrhage) in order to exclude false aneurysm and to establish the patency of the internal (ICA), external (ECA) and common carotid arteries (CCA), as well as patch corrugation which has been recognised as a warning sign of patch infection.[41] In addition, CT angiography was increasingly performed during the 20 year audit period in order to evaluate the status and/or accessibility of the upper limits of the ICA. CT imaging was always performed in any patient in whom there was any question of having to access the distal ICA near the skullbase.

The operative technique was similar in most cases. Nineteen procedures were performed with intra-operative TCD monitoring. TCD monitoring was not available for the three emergency operations associated with patch rupture which took place either at weekends or out of hours during week days. The first step was to control the proximal CCA immediately above the clavicle. The original incision was then reopened preserving the hypoglossal and vagus nerves and the distal ICA exposed. On a practical note, it is not unusual to encounter considerable fibrosis around the upper limits of the original reconstruction. This increases the risk of inadvertent haemorrhage and cranial nerve injury. Accordingly, it is now our practice to insert a Pruitt Inahara shunt as soon as possible (proximal CCA clamp, shunt inserted through the partially exposed patch before distal dissection and exposure of the upper ICA is completed). An inflated Fogarty balloon catheter controls the ECA (Fig. 1). The presence of a functioning shunt ensures adequate brain perfusion, but more importantly it permits easier distal exposure because the shunt is now palpable (thus identifying the ICA in any fibrous tissue) and because the risk of sudden haemorrhage following any breach in the arterial wall is avoided. In these situations, we would normally position a segment of reversed saphenous vein over the distal limb of the Pruitt shunt prior to insertion so that a bypass can then be performed without shunt removal.

Where ever possible, arterial reconstruction (vein patch, vein bypass) was performed after complete debridement of infected

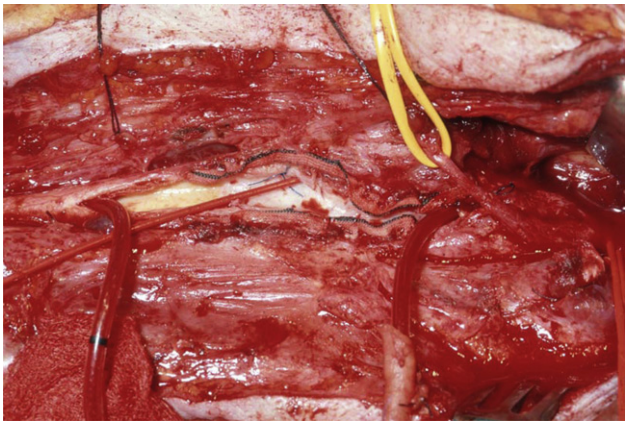

Figure 1. Using the 'high approach' to the upper carotid region,[42] the infected patch has been partially exposed but the distal ICA is enclosed in fibrous tissue. A shunt has been inserted through the exposed segment of patch and a Fogarty catheter controls the ECA origin. The shunt enables the surgeon to know where the distal ICA runs and enables more controlled distal exposure with no risk of major haemorrhage. A segment of reversed vein has been placed over the distal limb of the Pruitt shunt so that an interposition bypass can be performed without having to remove the shunt at a later time.

tissues and patch removal, unless the ICA was known to be occluded pre-operatively. Ligation was performed in cases of ICA occlusion and very occasionally in patients with a known MRSA infection provided good collateral flow had been demonstrated on TCD at the original operation (mean MCAV >20 cm/s). If pre-operative investigations suggested that access to the upper ICA might be challenging, a combined procedure was undertaken with an Ear Nose and Throat Surgeon using an operative technique permitting access to the skullbase which has been described elsewhere.[42] All wounds were closed over suction drains. Gentamicin-impregnated sponges (Gentacoll/Collatamp G®, Schering-Plough Ltd, Denmark) were placed around the reconstruction and debrided tissues to optimise eradication of infection.

Literature review

A systematic literature review was undertaken of patients with prosthetic patch infection following CEA up to 25th August 2011. Studies were identified by manual journal reviews (*European Journal of Vascular and Endovascular Surgery, Journal of Vascular Surgery, Annals of Vascular Surgery*), cross-referencing, and an electronic PUBMED search, using the search terms 'patch infection', prosthetic infection, 'carotid pseudoaneurysm'. Patients with suspected patch infection following vein patch angioplasty or after biological patching (eg bovine pericardium) were excluded from this review, as were reports of patients treated for pseudoaneurysms with no suspicion of infection or following primary closure. Thirty-three eligible studies were identified following a detailed literature review.[10–38,43–46] Four were subsequently excluded due to an inability to extract individual patient data and outcomes,[43–46] while the current paper supercedes an earlier study[39] The remaining 29 series (101 patients) were included in the systematic review,[10–38] along with the 22 patients from the current study. Cumulative analyses were performed using the Kaplan–Meier method on SPSS.

Results

Between January 1992 and 1st April 2012, 17/2017 patients (0.8%) undergoing their primary CEA at the Leicester Royal Infirmary with prosthetic patching were subsequently treated for patch infection. In addition, five other patients who underwent their primary CEA elsewhere were referred and treated for prosthetic patch infection at the Infirmary, giving a total of 22 patients in the series. Table 1 outlines their presentation and management. Six presented within 60 days of surgery, all of whom had suffered early wound complications after the primary operation (5 superficial wound infections, 1 deep wound abscess). Systemic antibiotics were started immediately upon presentation and were then revised based upon cultures and microbiology advice. Three patients (Cases 4, 6, & 15) presented with early patch rupture and massive haemorrhage and were taken immediately to theatre without further investigation. All other patients underwent urgent Duplex scanning to exclude/confirm a false aneurysm as well as establish the patency of the common, internal and external carotid arteries.

The commonest infecting organism was *Staphylococcus* ($n = 11$), including 4 cases of *MRSA*. There have been no cases of MRSA patch infection since 2004. In addition there were 3 coliform and 2 *pseudomonas* infections. The choice of redo-operation depended upon i) bacteriology; ii) the effect of carotid clamping on MCA velocity at the original procedure; iii) patency of the ICA; and iv) ease of distal access. The majority underwent debridement and patch excision, plus secondary reconstruction with either a vein patch ($n = 3$) or reversed saphenous vein bypass ($n = 14$). Three patients underwent debridement and patch excision plus carotid ligation. The latter patients had pre-operative Duplex ultrasound evidence of ICA occlusion and TCD evidence that the ECA did not significantly contribute to collateral flow. Two of the patients with ICA occlusion had *MRSA* infection.[47] One patient with an abscess culturing *β-haemolytic Streptococcus* was treated with debridement, on-table rifampicin irrigation, and post-operative benzylpenicillin irrigation because the original operation had involved a quite high distal ICA dissection. The remaining patient presented with a rapidly expanding false aneurysm, 14 years following his original operation. He had been treated elsewhere with a ruptured abdominal aortic aneurysm 12 months prior to presentation and intravenous jugular lines had been inserted on the side of the neck which subsequently developed a patch false aneurysm. It was assumed (though never microbiologically proven) that this probably represented a patch infection. Due to co-morbidities (the patient was now on home oxygen), this was treated by insertion of a covered stent and 6 weeks of oral antibiotic cover (Fig. 2).

Table 1
Presentation and management of carotid patch infections.

Case	Post-operative wound problem	Presentation	Interval	Primary management	Culture	Follow-up (months)	Reinfection
1	Wound infection	Abscess	10 days	Ligation	MRSA	133 (Died)	No
2	Wound infection	Abscess	13 days	V. patch	Coliforms	12	No
3	Abscess	Abscess	16 days	Antibiotic irrigation	β-haemolytic Strep	180	No
4	Wound infection	Patch rupture	24 days	V. bypass	Pseudomonas	79 (Died)	No
5	Wound infection	Abscess	27 days	V. bypass	Coag neg Staph	76	No
6	Wound infection	Patch rupture	7 weeks	V. bypass	MRSA	51 (Died)	No
7	Wound infection	False aneurysm	9 weeks	V. patch	Staph aureus	170 (Died)	No
8	None	Sinus	6 months	V. bypass	MRSA	39 (Died)	No
9	None	Sinus	9 months	V. bypass	No growth	111	No
10	None	Sinus	11 months	V. bypass	No growth	5	No
11	None	False aneurysm	12 months	V. patch	No growth	2.5	No
12	None	Sinus	13 months	V. bypass	Coag neg Staph	16 (Died)	No
13	None	Sinus	18 months	Ligation	Coliforms/MRSA	96 (Died)	No
14	None	Sinus	18 months	V. bypass	Coliforms	54	No
15	None	Patch rupture	23 months	V. bypass	Coag neg Staph	23 (Died)	No
16	None	Sinus	24 months	V. bypass	Pseudomonas	53	No
17	None	False aneurysm	35 months	V. bypass	Coag neg Staph	47	No
18	Haematoma – evacuated	Sinus	37 months	V. bypass	Coag neg Staph	139	No
19	None	Sinus	41 months	Ligation	Coag neg Staph	125	No
20	Wound infection	Abscess	45 months	V. bypass	No growth	102	No
21	None	False aneurysm	81 months	V. bypass	No growth	36	No
22	None	False aneurysm	164 months	Carotid stent	No cultures	29	No

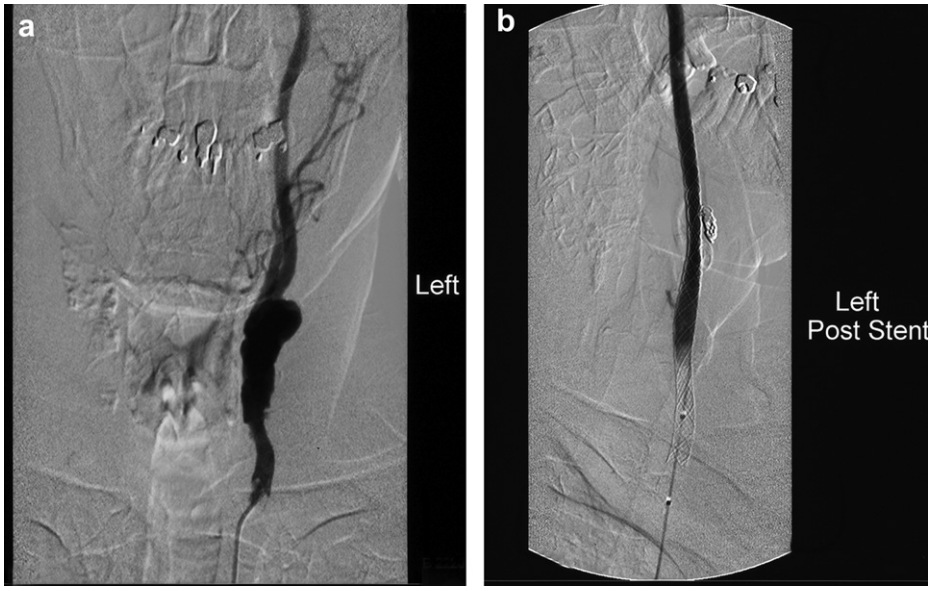

Figure 2. (a) Rapidly expanding false aneurysm 14 years after CEA. The patient had undergone repair of a ruptured AAA 12 months prior to presenting with the patch infection and had had emergency jugular lines inserted on the side of the patched CEA. It was assumed, though never proven, that this represented a patch infection. (b) False aneurysm excluded after insertion of a covered stent. Six weeks post-operatively, the stent was normal on duplex imaging. At three months, it had occluded (without symptoms).

Outcome

No patient was lost to follow-up (median 54 months; range 2.5–180). There were no peri-operative deaths. There was one post-operative stroke, giving a 30-day death/stroke rate of 4.5%. This patient (Case 6) presented with acute patch rupture and massive haemorrhage secondary to MRSA infection 7 weeks following surgery. At the original operation, dissection extended high into the neck (making control of the distal ICA extremely difficult following patch rupture) and he was noted to have very low MCA velocities (6 cm/s) during clamping, at the primary operation indicating that carotid ligation would not be tolerated. He therefore underwent a reversed saphenous vein bypass following fracture of the styloid process to facilitate distal access. Unfortunately he developed a dense hemiparesis following recovery from anaesthesia, presumably due to cerebral ischaemia during attempts to secure distal vascular access and shunt insertion. He did, however, recover significantly over the following 6 months and returned to independent living.

Nine patients (41%) suffered cranial nerve injuries, four persisting at 30 days. All patients have been followed up with clinical and Duplex ultrasound surveillance. During the course of follow-up, no patient has suffered a stroke or developed reinfection. Eight patients have died, but none as a consequence of stroke or reinfection. The patient in whom a covered stent was inserted was found to have occluded his stent (without symptoms) at 3 months (Fig. 2). The Duplex ultrasound scan at 6 weeks following stent insertion was normal.

Systematic review

Including the 22 patients from the current series, 123 prosthetic patch infections have been reported between 1962 and 2012.[10–38] Table 2 outlines the mode of presentation relative to the delay from CEA to onset of infection, while Table 3 reports how many of the patch infection patients reported early wound complications. After their primary operation. Thirty-six patients (29%) presented <2 months (usually reporting peri-operative wound complications) with infection/abscess and patch rupture predominating. Seventy eight patients (63%) presented after more than 6 months had elapsed, with chronic sinus and false aneurysms being the most common presentation. Interestingly, it was quite rare to find a history of post-operative wound infection or haematoma after the original operation in patients who presented late with their patch infection. Only two patients in the systematic review presented with ipsilateral stroke or TIA and there have been only 13 cases of acute patch rupture in the world literature.

Eighty-seven of 112 patients (78%) in whom the bacteriological results were reported in the constituent papers reported a positive culture. Of these positive cultures, 79 (91%) yielded *Staphylococci* or *Streptococci*, including 11 *MRSA* infections. The remaining isolates were enterobacter ($n = 4$), pseudomonas ($n = 3$), bacteroides ($n = 3$), coliforms ($n = 3$), proteus ($n = 2$), enterococcus ($n = 1$), and corynebacterium ($n = 1$).

Table 4 summarises the treatment strategies in the 123 patients in the systematic review. Most authors advocated patch excision (94/123 patients, (76%)), with the main debate being the optimal mode of revascularisation. Seventy-four were treated by debridement, patch excision and autologous reconstruction with either;

Table 2
Timing and mode of presentation of 123 patients treated for prosthetic carotid patch infections.

	Timing of prosthetic patch infection after CEA		
	<2 months	2–6 months	>6 months
Presentation			
Wound infection/abscess	20	1	12
Patch rupture	6	1	6
False aneurysm	3	2	22
Sinus discharge	6	3	28[a]
Sinus + false aneurysm	0	2	6
TIA/Stroke	1[b]	0	1[b]
Swelling	0	0	3[c]
Total	36	9	78

[a] 2 presented with TIA/Stroke as well.
[b] Paper did not specify any other presentation but reported positive blood cultures.
[c] All three cases from Knight et al.[10]

Table 3
Relationship between peri-operative wound complications and timing of prosthetic patch infection.

	Timing of prosthetic patch infection after CEA		
	<2 months	2–6 months	>6 months
Wound infection	19	3	1
Haematoma	7	0	2
Nothing	5	4	36
No data provided	5	2	39
Total	36	9	78

vein bypass ($n = 37$), vein patch ($n = 30$), arterial bypass ($n = 5$), or primary closure ($n = 2$). Six of these patients suffered peri-operative strokes, (two fatal) giving a 30-day death/stroke rate of 8.1%. Four survivors undergoing autologous reconstruction developed reinfection <60 days requiring further debridement and autologous venous reconstruction. After 60 days, no patient treated by autologous reconstruction developed a late reinfection.

Seven patients underwent debridement, patch excision and carotid ligation. One suffered a peri-operative stroke, but there were no cases of reinfection. Ten patients underwent prosthetic reconstruction following debridement and patch excision. Of these 7/9 (78%) either died in the peri-operative period or developed reinfection within 24 months.

In 29 patients (23.6%), the prosthetic patch was left *in-situ*. Twenty-three underwent debridement plus; abscess drainage ($n = 14$); muscle flap coverage ($n = 5$), or post-operative antibiotic irrigation ($n = 1$). In this heterogenous group of patients, there was one peri-operative stroke (4.3%), while three developed late reinfection (13%). One patient was treated with antibiotics alone, and was infection free at 24 months. An important subgroup are the five patients who were treated by insertion of a covered stent. To date, none who were treated by covered stent insertion have died or suffered a peri-operative stroke and none have developed a secondary infection of the stent (median follow-up 12 months). The Leicester patient occluded his covered stent at 3 months.

Overall infection-free survival is shown in Fig. 3a, with a 10-year infection-free survival rate of 78%. Fig. 3b compares cumulative infection-free survival in patients treated by patch excision and autologous reconstruction (patch, bypass, primary closure, ligation) compared with all other management strategies. The 10-year infection-free survival was significantly higher (93.5% vs. 47.1% ($p = 0.007$)) in patients undergoing autologous reconstruction.

Discussion

Despite everyone being aware of prosthetic patch infection following CEA, only 123 cases have now been reported in the world literature, suggesting that this complication is grossly under-reported. The prevalence of patch infection ranges from 0.4 to 1.8%.[10–38] The current series (the largest to date) reported a prevalence of 0.8% in 2017 patients. Some may view this figure as being too high, but there will always be a small risk of infection in any vascular procedure involving prosthetic material and a prevalence <1% is similar to the risk of vein patch rupture if the saphenous vein is harvested from the ankle.[48] It is, however, accepted that the vein rupture risk is less if the groin saphenous vein is used, but that will limit access to a potentially important autologous conduit in the future. Alternatives to prosthetic patching include eversion endarterectomy, bifurcation advancement or perhaps the use of biological patches. In Leicester, we have now made a decision to change to bovine pericardial patches (instead of polyester patches) because of their perceived resistance to infection (virtually no cases have been reported in the world literature) and because they are less prone to suture line bleeding. Our Unit routinely administers dual antiplatelet therapy (regular Aspirin plus 75 mg Clopidogrel the night before surgery[49,50]) in order to reduce post-operative thromboembolic complications, but this practice is associated with the potential for increasing the risk of neck haematomas (ie a potential risk factor for patch infection).

The timing of patch infection appears to follow a bimodal distribution, with one third presenting within two months of the original procedure (usually with a preceding wound complication), while two thirds present late (>6 months); usually with chronic sinuses or pseudoaneurysm formation. *Staphylococci* and *Streptococci* were the commonest offending pathogens in 90% of cases, and awareness of this can dictate selection of the optimal choice of antimicrobial therapy whilst awaiting definitive cultures.

In most centres, Duplex ultrasound is the first-line investigation. This enables visualisation of patch corrugation (recognised to be a warning sign of impending patch infection up to a year before overt clinical presentation[41]), the presence of deep space

Table 4
Review of management strategies and outcome of 123 patients treated for prosthetic carotid patch infection.

Procedure	*N*=	30d death	30d stroke	30d D/S	Reinfection in survivors (months)	No reinfection in survivors (months)	No data ($n =$)	
Debridement plus								
Post-op antibiotics	3	0	0	0	77	8,8		
Post-op antibiotic irrigation	1	0	0	0		180		
Muscle flap	5	0	1	1		3,12,24,45		
Abscess drainage	14	0	0	0	2,3	0,1,1,3,16,24,28,44,60,62,72,87		
No debridement plus								
Systemic/oral antibiotics	1	0	0	0		24		
Covered stent	5	0	0	0		3,12,12,32,29		
Debridement + patch excision +								
Carotid ligation	7	0	1	1		42,96,112,125,133	1	
Vein patch	30	1	3	3	1, 1, 2	2.5,3,6,6,6,8,12,12,12,12,16,16, 19,20,23,24,24,24,36,42,56,170	2	
Vein bypass	37	1	3	3	1	1,3,4,5,8,11,12,12,12,16,23,24,24, 26,36,39,44,45,47,51,53,54, 56,60, 62,76,79,102,111,139	2	
Prosthetic reconst	10	1	1	1	0.5,2,12,12,16,24	3,12	1	
Primary closure	2	0	0	0		3	1	1 SCM flap
Arterial bypass (native)	5	0	0	0			5	3 PM; 1 SCM flaps
Arterial bypass (allograft)	3	0	0	0			3	

SCM, sternocleidomastoid; PM, pectoralis major.

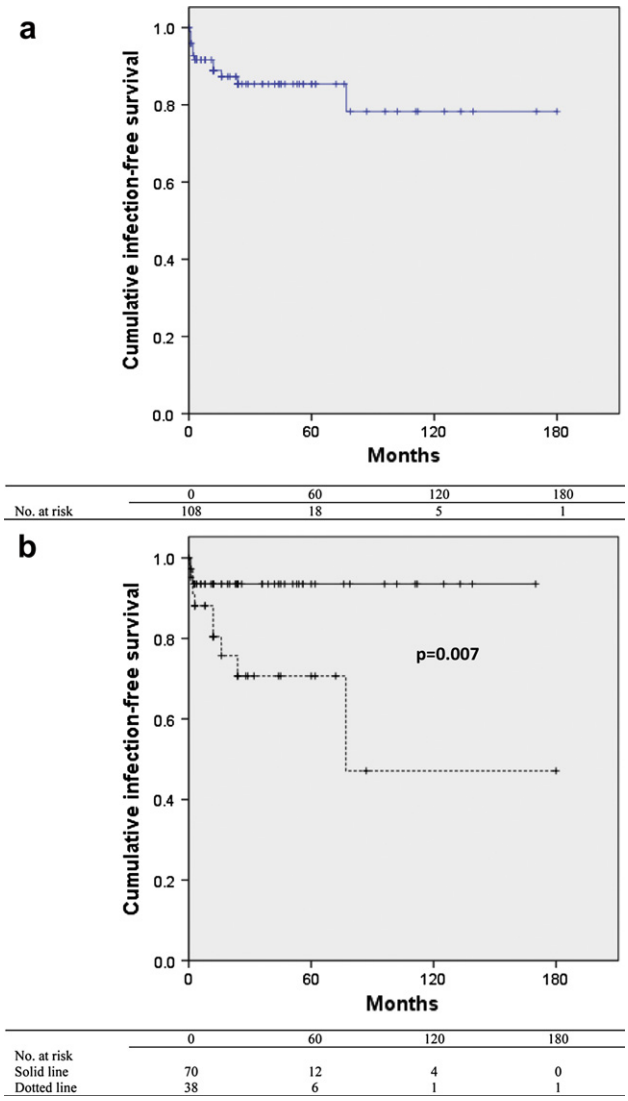

Figure 3. Infection-free survival following treatment of infected prosthetic carotid patch. (a) Overall infection-free survival. (b) Infection-free survival in patients treated with patch excision and autologous reconstruction or ligation (solid line) versus other methods (dotted line; $p = 0.007$ log-rank).

collections, as well as pseudoaneurysm formation. Cross-sectional imaging can help with evaluating the upper ICA, ease of access and help determine whether steps should be taken pre-operatively to plan an operation that may require access to the skull base.[42] Intra-arterial catheter digital subtraction angiography adds little unless it is a prelude to insertion of a covered stent or coil/detachable balloon.

The available evidence would suggest that surgical exploration along with debridement of all infected tissue and patch excision, combined with revascularisation is probably still the 'gold standard' management strategy for the majority of patients. Options for autologous reconstruction include vein patch closure or bypass, or bypass using an autologous arterial conduit (e.g. the superficial femoral artery[36]). Whilst concerns have been raised regarding the potential for catastrophic vein graft rupture in a contaminated wound, the literature does not to back this up. However, replacement with a prosthetic patch does carry a very high risk reinfection and should be avoided.[14,17,35]

Carotid ligation has historically been associated with a high risk of peri-operative stroke,[13] but (in the presence of MRSA infection) may be necessary due to the organism's virulence.[47] In the presence of a known ICA occlusion on Duplex ultrasound, ligation can be performed without undue concern. If ligation is to be considered intra-operatively, the surgeon should already have checked upon the likely status of the collateral circulation pre-operatively. For example, those patients who developed neurological symptoms during carotid clamping under locoregional anaesthesia at the primary operation will obviously not tolerate ligation. Similarly, patients with a mean MCA velocity <20 cm/s are also unlikely to tolerate ligation. In these situations, every attempt should be made to reconstruct the carotid artery. If necessary, seek expert assistance.

The systematic review included five patients who underwent treatment by insertion of a covered stent. This is an interesting subgroup as it may prove difficult to be sure that there was a proven infection (eg Case 22 in our series). Each of the five stented cases had good peri-operative outcomes (Table 4), but it remains to be seen whether late reinfection becomes a problem. To date, there has been insufficient follow-up data to address this issue (median 12 months) and it is hoped that centres with longer term experience of using covered stents to treat prosthetic patch infections will publish their experience. Because most centres prefer to publish 'good outcomes', it may be that the available literature on the role of covered stents is biased.

In summary, prosthetic patch infection following CEA is a rare, but under reported complication. In light of the fact that most authors prefer to publish the 'best results', it is highly likely that the pooled mortality and morbidity rates in Table 4 represent an underestimation of their true value in 'real world' practice. Accordingly, rigorous attention to preventive measures remains paramount. For now, patch excision and autologous reconstruction remains the 'gold standard'.

Conflict of Interest

None.

Funding

None.

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