CASE SERIES

Endovascular therapy of extracranial carotid artery pseudoaneurysms: case series and literature review

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ABSTRACT

Background Experience with endovascular therapy of extracranial carotid artery pseudoaneurysm (ECAP) has been growing, and various results suggest it as a suitable treatment option. We present a consecutive case series of patients with ECAPs treated with endovascular therapy, and a pertinent literature review.

Methods A prospectively maintained database of neuroendovascular procedures was retrospectively reviewed for cases of ECAP treated between January 2007 and December 2012. The primary outcome of interest was incidence of periprocedural (within 30 days) neurologic and cardiopulmonary complications. PubMed was searched for relevant endovascular studies from 2000 to 2012 for the review.

Results In our series, 12 patients with 14 ECAPs received stents, with or without coils. No perioperative neurologic or cardiopulmonary complications occurred. Median duration of clinical follow-up was 6.25 months (range 0–50 months), and median duration of imaging follow-up was 6.25 months (range 0–50 months), with eight patients asymptomatic and three showing improved symptoms (one patient with two lesions was lost to follow-up). Literature review revealed an overall primary outcome of 8.6% and no procedure related deaths.

Conclusions Endovascular treatment of ECAP is safe and effective at improving symptoms. There were no perioperative or permanent neurologic sequelae in the 11 patients with follow-up, and all symptomatology related to ECAP improved or resolved with treatment.

INTRODUCTION

Extracranial carotid artery pseudoaneurysm (ECAP) is an infrequent lesion most commonly traumatic, spontaneous, or iatrogenic in origin. Iatrogenic lesions develop as complications of procedures (ie, post-carotid endarterectomy (CEA) or misplaced central venous catheters). Spontaneous lesions are often associated with spontaneous dissection of the carotid artery and/or fibromuscular dysplasia (FMD). Other less frequent causes include vasculitis (ie, Behçet's disease) and infection. Within an unresolved ECAP, thrombi may form and subsequently embolize, causing transient or permanent ischemic events,[1–4] thus most agree that ECAPs require treatment. Treating ECAPs with anticoagulation or antiplatelets alone has been reported to have a low risk of ischemic events[5] but is not effective at resolving lesions, and thus a long term risk of embolization is a possibility. If symptoms persist despite medical management, endovascular or surgical treatment is indicated to eliminate the ECAP as a source.[6–10] Another indication for invasive therapy is an enlarging ECAP because of the risk of carotid blowout. Overall complication rates after endovascular therapy are difficult to discern from many small case series with varying results.

We report a contemporary series of symptomatic ECAPs treated with endovascular therapy, and review the literature.

MATERIALS AND METHODS

Chart review

The local institutional review board approved the collection of data for the completion of this retrospective study.

We searched the prospectively maintained database of neuroendovascular procedures at our hospital for consecutive cases of ECAP treatment between January 2007 and December 2012. Indication for treatment was either transient ischemic attack (TIA) or stroke, or intractable symptoms that did not resolve with medical management (eg, neck pain or pulsatile mass). Pseudoaneurysm was defined angiographically as abnormal dilation of the carotid artery greater than or equal to twice the diameter of the vessel. This finding signifies either breach of the carotid artery intima and media resulting in subadventitial collection of blood or breach of all three arterial layers forming a hematoma that tamponades the artery.

Demographic and medical data were retrospectively reviewed, including age, gender, clinical presentation, ECAP etiology, indication for procedure, and antiplatelet or anticoagulant therapy. Traumatic lesions were further divided as acute or remote, with acute lesions defined as treatment of the ECAP within 30 days of the traumatic event and remote lesions defined as treatment >30 days after the event. ECAPs were deemed iatrogenic if an intervention obviously resulted in creation of the lesion. Spontaneous ECAPs were defined as any lesion without a history of trauma, iatrogenic event, or other obvious etiology. Mycotic lesions were defined as those ECAPs that developed secondary to infection and were not otherwise accounted for by another cause (ie, ECAPs secondary to CEA that were found to be infected were labeled as iatrogenic in nature).

Anatomic data of the lesions were reviewed, including location and size of the pseudoaneurysm (at its widest diameter×at the widest diameter perpendicular to this), and presence of dissection and/or FMD. Dissection was defined angiographically by the presence of a stenosis, double lumen, or an intimal flap. Anatomic locations of the lesions were

distributed by type using the classification system suggested by Attigah *et al*[11] (table 1).

Procedural details were noted, including coils and type of stenting device used, and intraprocedural and postprocedural complications, both clinically and radiographically. Follow-up data were obtained via detailed review of the medical record. Perioperative complications included were any events involving the access site, causing neurologic symptoms, or causing cardiopulmonary or renal compromise, whether transient or permanent, during the 30 days after the procedure. The primary outcome of interest was periprocedural neurologic and cardiopulmonary complications.

Procedure

Patients who underwent endovascular therapy for ECAPs were pretreated with dual antiplatelet therapy (typically, clopidogrel and aspirin). Stenting was performed in a biplane angiographic suite. The procedure was performed under conscious sedation after administration of fentanyl and midazolam. Standard femoral artery access was obtained via a mural puncture technique. After femoral artery access was secured, heparin was administered to a resultant activated coagulation time of 250–300 s. The artery containing the lesion was catheterized using a 6 F guide catheter over a 0.035 inch guidewire with fluoroscopic visualization and roadmap guidance if necessary. After anatomic details of the lesion were confirmed with angiography, a self-expanding stent was selected with a nominal diameter <1 mm larger than the diameter of the non-diseased carotid artery just proximal to the pseudoaneurysm. The lesion was crossed with a steerable 0.014 inch microwire. Generally, embolic protection was not employed as the risk of thromboembolism of these lesions is low in our experience. The stent was then delivered in position covering the ostium of the pseudoaneurysm over the microwire and deployed to cross the lesion. Coils were used at the neurosurgeon's discretion to accelerate obliteration, and were typically delivered through a 0.0165 inch inner diameter microcatheter after stenting by accessing the aneurysm through the free cell area of the stent.

Postoperatively, patients were monitored on a telemetry unit with hourly neurological examinations for a minimum of 12 h. Routine postoperative care included a carotid ultrasound study within 24 h of stent placement. This study was compared with the results of follow-up ultrasonography performed at 1 month, 6 month, and 12 month intervals thereafter to assess for in-stent stenosis. Additional follow-up studies, including invasive or noninvasive angiography, were performed at the discretion of the attending surgeon (the most recent study performed was recorded, along with the length of follow-up; if follow-up was not recorded, it was considered 0 months). Patients were placed on dual antiplatelet therapy postoperatively (typically, clopidogrel 75 mg daily for 1–6 months and aspirin 325 mg daily indefinitely).

Table 1 Classification of extracranial carotid artery pseudoaneurysms*

Type	Morphology and anatomic location
I	Short aneurysm of ICA
II	Long aneurysm of ICA
III	Involving proximal ICA and carotid bifurcation
IV	Involving both ICA and CCA
V	Isolated to CCA

*Scheme adapted from Attigah *et al*.[11]
CCA, common carotid artery; ICA, intracranial carotid artery.

Literature review

A review of the English language literature was performed for ECAPs treated with endovascular therapy since the year 2000, by searching PubMed for 'pseudoaneurysm' and 'carotid' and 'endovascular' or 'stent' (the year 2000 was chosen arbitrarily with a twofold intent of including enough subjects to decrease the effects of outliers and including data relevant to the current technology of endovascular therapy). A total of 141 articles were retrieved for full review. In addition, the bibliographies of the retrieved articles were cross referenced to obtain 64 additional articles that were retrieved for full review. Articles were excluded from our analysis for the following reasons: the pseudoaneurysm involved the intracranial carotid artery, external carotid artery, or subclavian artery; a combination of open surgery and endovascular treatment of the carotid artery was performed; endovascular therapy using the open carotid artery approach was performed; lesions related to head and neck cancer (HNC); perioperative outcome was not reported; or a single case was reported. Case reports of two or more subjects in total were included even if only one ECAP was being reported (the reason for this was twofold: most case series of ECAP included other causes of carotid artery aneurysms in the series and because case reports of a single subject often reported extreme anomalies not representative of the majority of ECAPs encountered in practice). The primary outcome of interest was periprocedural (within 30 days) neurologic and cardiopulmonary complications.

RESULTS

Twelve patients with 14 ECAPs were treated endovascularly at our hospital (table 2). Mean patient age was 51.6 years (range 26.9–68.5 years) and there was a slight preponderance of men (58%). The ratio of left to right lesions was 1:1. Eleven lesions were type I (short aneurysm involving the internal carotid artery (ICA)), two were type III (aneurysm involving the proximal ICA and carotid bifurcation), and one was type V (common carotid artery). The lesions were either spontaneous or traumatic in etiology. Of the spontaneous lesions, 50% showed FMD and 75% had associated dissections. Two of five traumatic lesions were acute; three were remote. Indications for treatment included stroke or TIA in four (33%), intractable neck pain and/or headache in three (25%), pulsatile neck mass in three (25%), and Horner's syndrome in two (17%).

No major complications occurred, but two patients had minor perioperative complications. Patient No 9 was found at an outside hospital to have a right femoral artery access site hematoma and deep vein thrombosis on postoperative day 4, which was treated with oral anticoagulation. No neurologic or cardiopulmonary sequelae occurred, and 50 months after the procedure, the patient remained free of symptoms related to the endovascular procedure. Patient No 2 was noted to have a small, non-flow limiting common carotid artery dissection during routine ultrasonography after the procedure, which was confirmed by digital subtraction angiography (figure 1). No intervention was performed; the patient remained asymptomatic for 13 months, and follow-up imaging at that time showed resolution of the dissection.

Median duration of clinical follow-up was 6.25 months (range 0–50 months). Eleven of 12 patients had clinical follow-up, with eight patients (72.7%) asymptomatic and three others (27.3%) showing improved symptomatology. Patient No

Table 2 Case series demographics, treatment, and follow up

Pt No	Age (years)	Presentation	Etiology	Type	Side	Dissection/ FMD	Size (mm), shape	Stent (mm)/coil (No)	30 day complications	Imaging follow-up	Months	Clinical follow-up	Months	Postoperative anticoagulation*
1	Mid 60s	Horner's syndrome	Spontaneous	I	L	Y/Y	7×2, saccular	Xpert 5×30, 1 coil in PA	0	CTA: no residual, patent	13.0	Asymptomatic	8.0	Clopidogrel 75 mg indefinitely, ASA 325 mg indefinitely
2	Mid 50s	Amaurosis fugax	Spontaneous	I	R	Y/Y	11×5, saccular	Xpert 5×40	POD 0 small, asymptomatic, non-flow-limiting R CCA dissection	MRA: no residual, patent	13.0	Asymptomatic	13.0	Dipyridamole 100 mg every 6 h for 13 mos, clopidogrel 75 mg indefinitely
3	Early 30s	CVA—hemiparesis	Trauma (blunt)	I	R	Y/N	20×11, saccular	Xpert 4×40, 2 coils in PA	0	MRA: small residual, patent	11.5	LLE weakness improved; LUE weakness resolved	12.0	Clopidogrel 75 mg for 6 mos, ASA 325 mg indefinitely
4	Late 60s	Horner's syndrome, 3 weeks of headaches	Spontaneous	I	L	Y/N	17×8, saccular	Xpert 5×40	0	MRA: small residual, patent	6.0	Miosis resolved, persistent L ptosis	6.0	Clopidogrel 75 mg for 3 mos,† ASA 325 mg indefinitely
5	Early 40s	Neck pain	Spontaneous	I	R	Y/N	15×5, fusiform	Xpert 5×30	0	CTA: no residual, patent	5.0	Asymptomatic	6.0	Clopidogrel 75 mg for 6 mos, ASA 325 mg indefinitely
6	Late 50s	Headache, hand numbness/ paresthesia	Spontaneous	I	L	Y/N	12×6, fusiform	Xpert 5×40	0	CTA: small residual, patent	3.0	Asymptomatic	1.0	Clopidogrel 75 mg for 3 mos, ASA 325 mg indefinitely
7	Mid 40s	Severe L neck and temporal head pain	Spontaneous	I	L	Y/Y	14×8, fusiform	Xpert 4×40	0	CTA: no residual, patent	1.0	Asymptomatic	1.0	Clopidogrel 75 mg for 3 mos, ASA 81 mg indefinitely
8	Mid 20s	Pulsatile neck mass	Trauma (penetrating)	III	L	N/N	10×8, saccular	Xact 79×30	0	None	none	None	none	Not recorded
8	Mid 20s	See above (same patient)	Trauma (penetrating)	III	L	N/N	25×5, saccular	Xact 79×40	0	None	none	None	none	Not recorded
9	Early 50s	TIAs, headache	Trauma (blunt)	I	R	Y/N	14×8, saccular	Precise 6×40	POD 4 R groin (access site) hematoma and DVT found at outside hospital	Ultrasound: no residual, patent	50.0	Suffered head trauma years after endovascular therapy that caused headache, transient diplopia	50.0	Clopidogrel 75 mg for 1 mo, ASA 325 mg indefinitely (warfarin transiently for DVT)
9	Early 50s	See above (same patient)	Trauma (blunt)	I	R	Y/N	13×5, saccular	Precise 6×30	See above (same patient)	Ultrasound: no residual, patent	50.0	See above (same patient)	50.0	See above (same patient)
10	Late 50s	Headache	Spontaneous	I	R	N/Y	12×4, saccular	Wallstent 6×22	0	DSA: no residual, patent	6.5	Asymptomatic	6.5	Clopidogrel 75 mg for 1 mo, ASA 81 mg indefinitely
11	Early 50s	Pulsatile neck mass	Trauma	V	R	Y/N	120×25, fusiform	Wallstent 10×37 (×2), 10×31, 8×29, balloon angioplasty	0	DSA: no residual, patent	9.0	Asymptomatic	9.0	Clopidogrel 75 mg for 1 mo, ASA 325 mg indefinitely
12	Mid 60s	Pulsatile neck mass	Spontaneous	I	L	N/N	30×27, saccular	Wallstent 10×37 (×2), 10×31	0	DSA: residual 6×5 mm, patent	5.5	Asymptomatic	1.0	Warfarin 5 mg alternating with 3.2 mg and ASA 325 mg indefinitely, prasugrel 5 mg

Stent manufacturers: Precise, Cordis (Bridgewater, New Jersey, USA); Wallstent, Boston Scientific (Natick, Massachusetts, USA); Xact and Xpert, Abbott Vascular (Santa Clara, California, USA).
*Medications are once daily unless specified otherwise.
†Non-compliant with clopidogrel.
ASA, acetylsalicylic acid (aspirin); CCA, common carotid artery; CTA, CT angiography; CVA, cerebrovascular accident; DSA, digital subtraction angiography; DVT, deep vein thrombosis; FMD, fibromuscular dysplasia; L, left; LLE, left lower extremity; LUE, left upper extremity; MRA, MR angiography; PA, pseudoaneurysm; POD, postoperative day; R, right; TIA, transient ischemic attack.

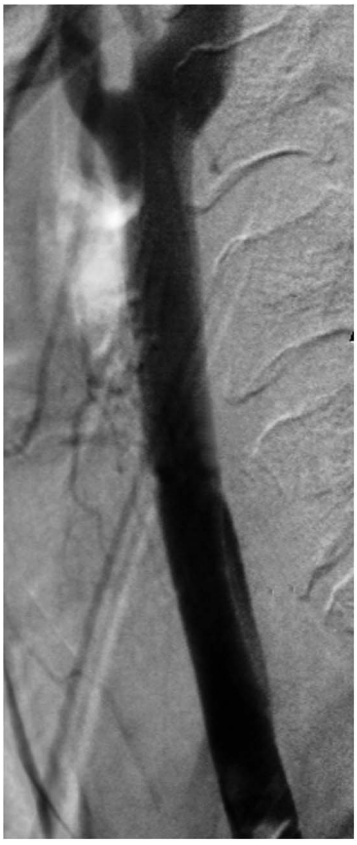

Figure 1 Patient No 2. Digital subtraction angiogram shows a non-flow limiting dissection of the right common carotid artery found immediately postprocedure. The patient was asymptomatic, and there was no evidence of residual dissection on the follow-up MR angiogram obtained 13 months later (not shown).

3's left upper extremity weakness resolved and left lower extremity weakness improved. Patient No 4's miosis resolved despite persistent ptosis. Patient No 9's paresthesias and headache improved, but the ptosis persisted. Patient No 8 was lost to follow-up.

Median duration of follow-up imaging was 6.25 months (range 0–50 months). Seven of 12 (58.3%) ECAPs were completely obliterated (figure 2); five (41.7%) were markedly decreased in size. All stents in patients who underwent follow-up imaging remained patent, with no cases of in-stent stenosis.

The stent used most commonly was the Xpert (Abbott Vascular, Santa Clara, California, USA) in seven patients (seven lesions). Other stents used were the Wallstent (Boston Scientific, Natick, Massachusetts, USA) in three patients (three lesions), Precise (Cordis, Bridgewater, New Jersey, USA) in one patient (two lesions), and Xact (Abbott Vascular) in one patient (two lesions). Multiple stents were used in four patients (including patient Nos 8 and 9, each of whom had two lesions) using an overlapping method (figure 3). Coils were used with a stent in two patients (figure 4).

Literature review

A total of 27 published studies describing endovascular therapy of 128 ECAPs in 118 patients met the criteria for inclusion in our analysis in addition to our current study (table 3).[9 12–36] The average age of the patients was 41.1 years among the 100 patients in those articles reporting age. Men totaled 60.4% of 91 patients in whom the sex was reported. There was a left to right vessel ratio of 1:1 in 80 lesions in articles reporting laterality. A total of 64 of 100 patients with presentations reported were symptomatic before receiving endovascular therapy. Most lesions were types I and V (table 4) based on the classification of Attigah *et al*.[11] Carotid artery dissection was reported in 51.6% of the lesions. Iatrogenic lesions were post-CEA in 11 ECAPs, secondary to surgical injury in three ECAPs, central venous catheter trauma in two ECAPs, and fine needle aspiration in one ECAP. The primary outcome (rate of perioperative neurologic and cardiopulmonary complications) was 8.6% (table 5). This was accounted for by a 7% stroke or TIA rate and a 1.6% mortality rate. Both deaths were unrelated to ECAP treatment, one

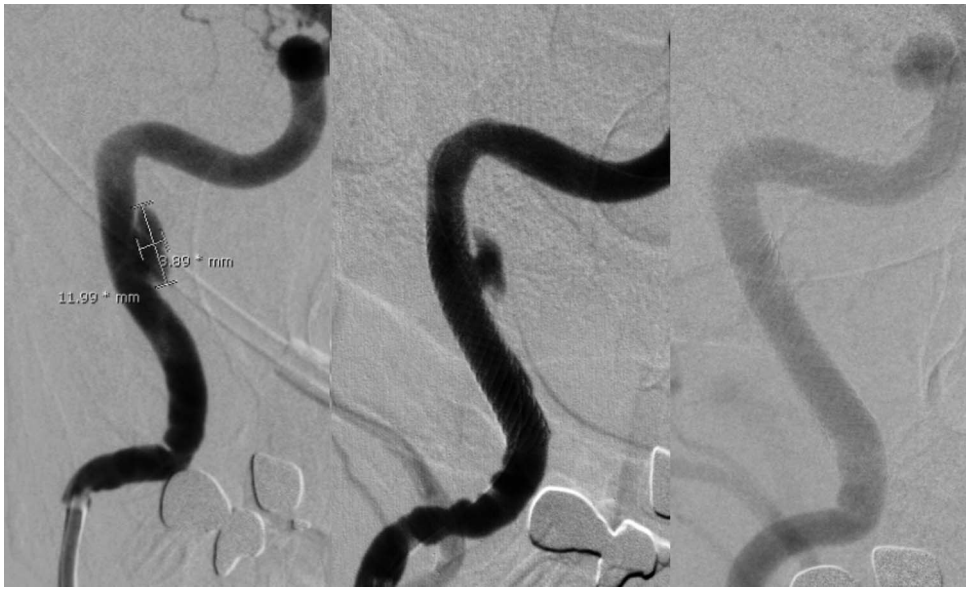

Figure 2 Patient No 10. Digital subtraction angiograms of a right extracranial internal carotid artery pseudoaneurysm (type 1). Left: Before endovascular treatment. Middle: Immediately after deploying a 6×22 mm Wallstent (Boston Scientific, Natick, Massachusetts, USA). Note the initial persistence of the pseudoaneurysm. Right: No residual pseudoaneurysm 6.5 months after endovascular treatment.

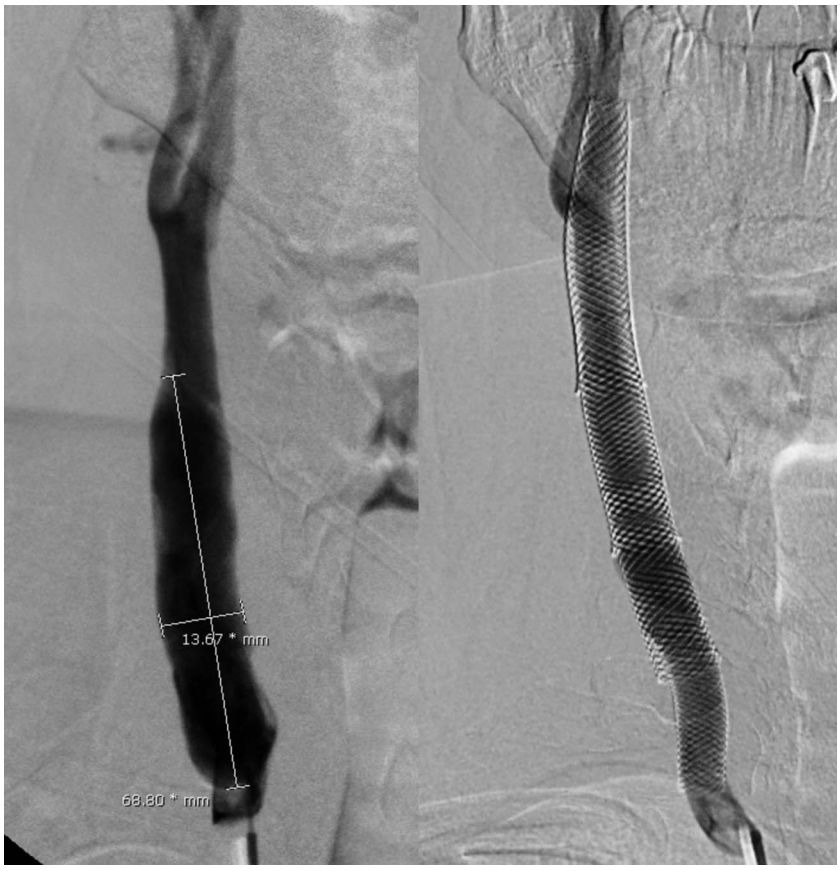

Figure 3 Patient No 11. Digital subtraction angiograms of a right common carotid artery fusiform pseudoaneurysm (type V). Left: Before endovascular treatment. Right: Immediately after deploying four Wallstents (Boston Scientific, Natick, Massachusetts, USA): two 10×37 mm; one 10×31 mm; and one 8×29 mm, using an overlapping technique. Balloon angioplasty was also used in this case.

being the result of a large pre-existing stroke[20] and the other secondary to multiple trauma.[25]

The four most commonly used stents were the Wallstent in nine studies, Wallgraft (Boston Scientific) in six studies, Smart stent (Cordis) in six studies, and Precise stent (Cordis) in five studies. In 13 studies, coils were used to obliterate 15 ECAPs, two external carotid arteries, one ICA, and one fistula.

Post-stenting anticoagulation or antiplatelet regimens were reported in 21 of 27 studies. Most used dual antiplatelet therapy with aspirin and either clopidogrel (76.2%) or ticlopidine (9.5%). One study used aspirin monotherapy and another used clopidogrel monotherapy. Two studies used warfarin.

Follow-up imaging occurred for a mean of 7.3 months (range 0–46 months) and clinical follow-up occurred for a mean of

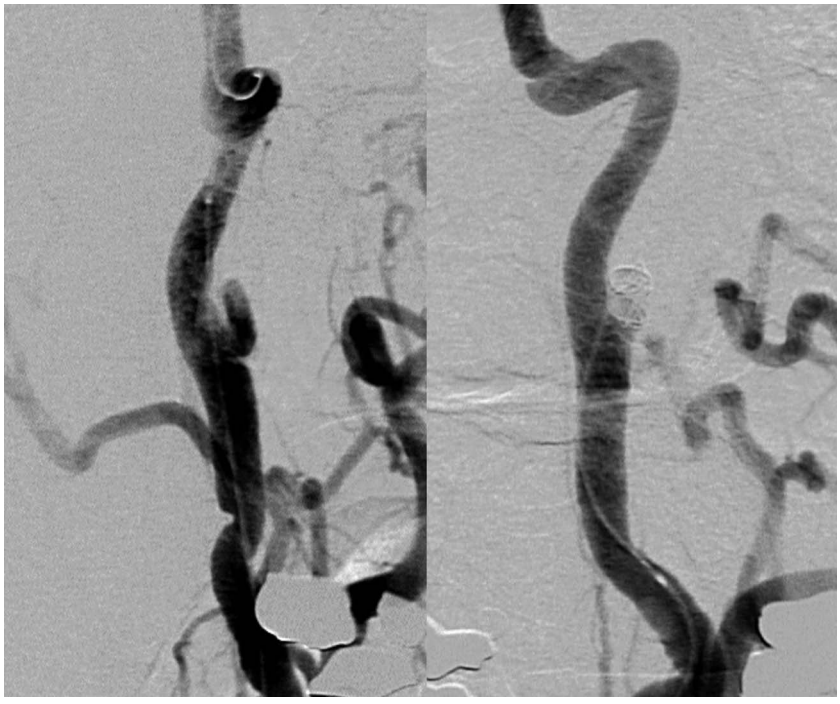

Figure 4 Patient No 1. Digital subtraction angiograms of a left common carotid artery pseudoaneurysm (type 1). Left: Prior to endovascular treatment. Right: Immediately after deploying a 5×30 mm Xpert stent (Abbott Vascular, Santa Clara, California, USA) and inserting one coil into the pseudoaneurysm.

Table 3 Review of the literature—cases of extracranial carotid artery pseudoaneurysm treated with endovascular therapy

Author, year	Patients (n)	ECAP (n)	Etiology (n)	Stent/balloon devices used	Coils/NBCA and location (n)	Post-stent anticoagulation regimen	Primary outcome*	Median clinical F/U (months)	Median imaging F/U (months)
Seward *et al* 2014	12	14	8 spontaneous, 6 trauma	Xpert, Precise, Wallstent	2 coils—PA	Clopidogrel, ASA	0	16.6	11.2
Hassan *et al* 2012	3	4	4 spontaneous	Precise, Neuroform	0	Clopidogrel, ASA	1 TIA	NR	4
Kim *et al* 2012	2	2	1 vasculitis (Behçet's), 1 spontaneous	Jostent	1 coil—ECA	NR	1 TIA	NR	5
Singhal *et al* 2009	1	1	1 iatrogenic (FNA)	None	1 coil—ICA	NR	0	24	NR
Berne *et al* 2008	3	3	3 trauma	Smart, Precise	1 coil—PA	Clopidogrel, ASA	1 CVA	0	6
Stephen *et al* 2008	1	1	1 mycotic	Covered stent—dipped in rifampicin	0	ASA	0	6	6
Gupta *et al* 2008	1	1	1 iatrogenic (pCEA)	Wallgraft	0	Clopidogrel, ASA	0	3	3
Yi *et al* 2008	5	5	5 trauma	Fluency, iCast, Viabahn, Driver, Smart	0	Clopidogrel, ASA	1 death (due to multiple trauma)	0.5	0.5
Szopinski *et al* 2005	2	2	1 trauma, 1 iatrogenic (pCEA)	Jostent	0	NR	0	NR	NR
Joo *et al* 2005	4	4	4 trauma	Jostent, Wallstent, Precise	1 coil—PA	Clopidogrel, ASA	0	9	9
Kadkhodayan *et al* 2005	15	20	5 spontaneous, 3 iatrogenic (2 pCEA, 1 CVC), 12 trauma	Wallstent, Smart, Precise	3 coils—PA	Clopidogrel, ASA	1 TIA	14.5	5.6
Cothren *et al* 2005	23	23	23 trauma	Magic Wallstent	0	NR	3 CVA	NR	Median NR (mean 2.4)
Layton *et al* 2004	1	1	1 trauma	PTFE covered stent	1 coil—ECA 1 coil—fistula	Clopidogrel, ASA	0	NR	8
McCready *et al* 2004	4	4	4 iatrogenic (4 pCEA)	Wallgraft	0	Clopidogrel, ASA	2 TIA	NR	0
Saket *et al* 2004	4	4	1 trauma (blunt), 1 vasculitis (Behcet's), 2 iatrogenic (cervical disc surgery, PM placement)	Palmaz with ePTFE (custom fabricated), Hemobahn, Jostent, Smart, Viabahn	0	NR	1 death (due to pre-existing CVA)	7.25	7
ul Haq *et al* 2004	2	2	2 trauma	Jostent	0	Clopidogrel or ticlopidine, ASA	0	NR	4.5
Wyers and Powell 2004	1	1	1 spontaneous	Wallgraft	1 coil—ECA	Clopidogrel, ASA	1 CVA	NR	0.5
Nicholson *et al* 2004	1	1	1 iatrogenic (CVC)	Wallgraft	0	None	0	NR	NR
Kubaska *et al* 2003	1	1	1 iatrogenic (pCEA)	Wallgraft	0	NR	0	NR	6
Lesley *et al* 2003	1	1	1 trauma	NIR, Smart	0	Clopidogrel, ASA	0	NR	17
Amar *et al* 2002	1	1	1 trauma	Wallgraft	0	Clopidogrel	0	NR	NR
Albuquerque *et al* 2002	4	4	2 spontaneous, 2 trauma	Wallstent	0	Antiplatelet	0	NR	11
Redekop *et al* 2001	2	2	2 trauma	Palmaz vein covered, PTFE covered stent	0	Clopidogrel &/or ASA	0	7.5	3
Assali *et al* 2001	2	2	1 trauma, 1 spontaneous	Wallstent	1 coil—PA	Clopidogrel, ASA	0	4.5	7.5
Bush *et al* 2001	5	5	3 trauma, 2 iatrogenic (2 pCEA)	Wallstent, Smart	5 coils—PA	Warfarin, ASA, clopidogrel	0	8	5
Malek *et al* 2000	3	3	2 spontaneous, 1 trauma	Wallstent, Magic Wallstent	2 coils—PA	Clopidogrel or ticlopidine, ASA	0	17	6
Coldwell *et al* 2000	14	16	16 trauma	Wallstent, Palmaz (balloon expandable)	0	Warfarin, ASA	0	Median NR (mean 6)	Median NR (mean 16)

*Primary outcome, perioperative (within 30 days) neurologic or cardiopulmonary complications.
ASA, acetylsalicylic acid (aspirin); CVA, cerebrovascular accident; CVC, central venous catheter; ECA, external carotid artery; ECAP, extracranial carotid artery pseudoaneurysm; ePTFE, polytetrafluoroethylene (proprietary); FNA, fine needle aspiration; F/U, follow up; ICA, internal carotid artery; NBCA, n-butyl cyanoacrylate; NR, not reported; PA, pseudoaneurysm; pCEA, post-carotid endarterectomy; PM, pacemaker; PTFE, polytetrafluoroethylene; TIA, transient ischemic attack.

Table 4 Review of the literature—location based on types listed in table 1

Type	No of lesions*
I	27
II	2
III	5
IV	1
V	16
ICA (insufficient data to classify)	50

*Location was reported for 101 of 128 lesions.
ICA, internal cerebral artery.

Table 6 Review of the literature—follow-up results >1 month postprocedure

Outcome	No of patients or lesions	Per cent
New symptoms on clinical evaluation	3/72 patients	4.2
Residual symptoms	9/61 patients	14.8
Stenosis/occlusion	10/97 ECAP	10.3
Pseudoaneurysm completely obliterated	84/89 ECAP	94.4
Return to operating room	3/105 patients	2.9

ECAP, extracranial carotid artery pseudoaneurysm.

6 months (range 0–50 months). Complete obliteration of ECAPs occurred in 94.4% of lesions. Stenosis or occlusion of stents occurred in 5.3% of lesions[12 17 32 33] (table 6). Further endovascular therapy was required in three of 105 patients (2.9%) (table 6), consisting of placement of an additional stent[31 36] and angioplasty of the left common femoral artery for decreased pulses postoperatively.[12]

Complications that occurred >30 days postoperatively included three TIAs.[27 32] Residual symptoms were reported in nine of 61 patients (14.8%), but all except one showed improvement from the preoperative presentation[12 16 22 31 35] (table 6).

DISCUSSION

Our series of 14 cases of ECAP treated with endovascular stenting demonstrates the safety of this approach for symptomatic traumatic and spontaneous lesions, with no perioperative neurologic or cardiopulmonary complications, and no deaths. Endovascular treatment also proved clinically efficacious as each of the 11 patients with follow-up demonstrated marked symptomatic improvement, although improvement in patient No 3 was likely due to the natural history of stroke recovery rather than an effect of treatment. Similar results with lesions of similar etiologies have been reported by Kadkhodayan *et al*[12] and Albuquerque *et al*,[28] with perioperative neurologic or cardiopulmonary complication rates of 5% and 0%, respectively. Follow-up imaging in our series showed that all pseudoaneurysms were either markedly decreased in size or completely obliterated, theoretically reducing the risk of thromboemboli. Kadkhodayan *et al*[12] and Albuquerque *et al*[28] reported complete obliteration of all lesions.

At our institution, we treat asymptomatic lesions with anticoagulants and/or antiplatelets. Invasive therapy is only pursued if ischemic events or intractable symptoms develop, or if on follow-up enlargement of the ECAP is noted. Unfortunately, we do not have data to discuss the results of conservative management at our institution, although the results in the literature of conservatively treated ECAP are positive.[34]

Review of the literature reveals a 7% rate of perioperative (within 30 days) ischemic events with endovascular therapy. Over long term follow-up, three additional minor ischemic events were reported. Findings were similar across all etiologies, except for vasculitis, which had a higher complication rate but too small of a cohort (two patients) to discern the validity of this finding. All lesions were either significantly decreased in size or completely obliterated (94.4%). The majority of in-stent stenosis or occlusion reported was the result of using anticoagulation rather than antiplatelet therapy postoperatively.[34] Symptoms resolved (85.2%) or improved in all patients but one. Non-disabling symptomatic sequelae occurred in 4.2% of patients.

In comparison, management of symptomatic ECAPs with anticoagulant and/or antiplatelet agents alone has shown some good results. Guillon *et al*[37] reported no ischemic events of symptomatic ECAPs secondary to arterial dissection treated with warfarin and aspirin. However, it was less effective at resolving symptoms (43.7%) than endovascular therapy (85.2%). On follow-up, 95% of lesions persisted, carrying a long term risk of embolization. Is this risk of embolization lower or greater than the risk of complications from surgical or endovascular therapy? Unfortunately, there is a lack of sufficient data to answer this question. If symptomatic relief is an important goal of treatment, it appears more likely to be achieved with endovascular therapy versus medical management.

It is important to distinguish ECAP in our review from those caused by surgical and/or radiation therapy of HNC (ie, carotid blowout syndrome). Lesions related to HNC have a very high perioperative neurologic complication and mortality rate (48.6% and 30%, respectively, on review of the literature since 2000).[12 15 24 25 36 38–40] The poor outcome in HNC lesions is explained by their emergent presentation and terminal nature of the illness.

Table 5 Review of the literature—primary outcome

Etiology	ECAP (No of lesions)	Primary outcome	Per cent	CVA/TIA	Per cent	Mortality*	Per cent
Trauma	85	6	7.1	5	5.9	1	1.2
Spontaneous	24	1	4.2	1	4.2	0	0
Iatrogenic	16	3	18.8	2	12.5	1	6.3
Vasculitis	2	1	50.0	1	50.0	0	0
Mycotic	1	0	0	0	0	0	0
Total	128	11	8.6	9	7.0	2	1.6

*Mortality within 30 days postoperatively.
CVA, cerebrovascular accident; ECAP, extracranial carotid artery pseudoaneurysm; TIA, transient ischemic attack.

Many different types of stents were used, and no difference was noticed in outcome according to particular stent types. For high cervical lesions in our series, we used the Xpert stent, which to our knowledge has not been reported for the treatment of ECAP, although it has been reported in the treatment of a vertebral artery pseudoaneurysm.[41] This device appears to be another option in the endovascular armamentarium for treating ECAP.

Limitations of our study include its retrospective nature and exclusivity of using patients from a neuroendovascular database, which may have excluded patients assigned to conservative treatment that did not have an angiogram (as mentioned above, all patients with ischemic events or intractable symptoms had endovascular therapy). Patients treated with open surgery (although no cases could be recalled) were also not recorded. Limitations of the review included confinement of all data to small case series. Despite the presence of periprocedural data in all of the articles, many did not report long term clinical follow-up evaluation, thus weakening our clinical conclusions.

CONCLUSION

Our series suggests that endovascular treatment of ECAPs is safe and effective at improving symptoms related to ECAPs. No permanent neurological sequelae were noted in patients with available follow-up, and radiographic improvement was noted in all patients.

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Competing interests EIL has shareholder/ownership interests in Intratech Medical Ltd, Mynx/Access Closure, and Blockade Medical LLC. He serves as a principal investigator for the Covidien US SWIFT PRIME trials. He receives compensation from Abbott for carotid training for physicians.

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