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Stent-graft repair of carotid endarterectomy-related pseudoaneurysm

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ABSTRACT

Objective: Carotid endarterectomy-related pseudoaneurysms are rare lesions challenging to be treated with conventional open surgery. Endovascular stent-graft has been rarely adopted with encouraging results. We present a case of a huge carotid pseudoaneurysm treated with a stent-graft and managed with a stent-graft and reviewed the available literature on the management and results of this technique for these challenging lesions.

1. Introduction

Carotid pseudoaneurysms (CP) are infrequent lesions[1]. Etiology includes previous carotid surgery, trauma, infection, cancer or immune disorders[2,3]. Carotid redo conventional surgery in the previously operated neck, as well as after radiation therapy, are both technically demanding and prone to increased rates of complications especially for stroke, nerve damage and wound infection even in renown centres[4].

In these circumstances, endovascular techniques developed as a valid alternative; periodically, encouraging results have been reported in several case reports especially for carotid endarterectomy related (CEA-related) pseudoaneurysms[5-18].

We present the case of a post-endarterectomy huge CP managed with stent-graft (SG).

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2. Case report

He is a 76-year-old male; medical history was remarkable for tobacco abuse and hypertension, radiation and thyroidectomy for cancer, and a left carotid endarterectomy (CEA) plus dacron patch closure for a transient ischemic attack (TIA) performed twenty-five years before the actual admission. Over the years, he did not receive regular follow-up examination with echo-color-Doppler, but a recent carotid ultrasound showed a huge CP as well as an 80% re-stenosis at the proximal aspect of the internal carotid artery. A computed-tomography angiography was performed, showing a type 1 aortic arch with a bovine configuration, and confirmed the presence of a 35 mm × 40 mm CP with no blood extravasations or radiologic signs of infection (Figure 1). From a clinical point o view, he denied fever and local signs of infection (skin changes, erythema) were absent. Leukocytes count (7 850 cells/mm^3) was in the normal range (4.3-11 000 cells/mm^3), and blood cultures were negative for bacteria: therefore, we excluded the diagnosis of an infected patch and concluded

for a pseudoaneurysm dilation. Open repair was particularly challenging because of the previous repeated neck surgery as well as radiation therapy. We proposed an endovascular repair, and the informed consent was obtained. In the angiographic suite, under intravenous sedation and local anaesthesia a percutaneous right femoral artery approach was attempted first. The patient was given 70 IU/kg of heparin intravenously prior to manipulation of the arch vessels, and activated clotting time was maintained >275 s. Selective left carotid hiring was not successful from the transfemoral route; hence, we decided to use a percutaneous right brachial to better address the bovine configuration of the aortic arch. A direct cervical incision was not thought to be reasonable because of the presence of the extensive cervical scar as well as previous radiation treatment. An hydrophilic stiff-angled guidewire (Radiofocus®-Terumo Corp.; Somerset-NJ; USA) was used to obtain access to the external carotid artery; a long multipurpose catheter (Brite Tip®-Cordis J&J-Waterloo; Belgium) allowed a wire exchange for a 0.035-inch wire (SuperCore® - Guidant; Santa Clara®-CA; USA). An 8Fr for 65 cm long sheath (Avanti®-Cordis J&J-Waterloo; Belgium) was advanced just proximal to the common carotid artery stenosis and a cerebral protection device (Epi Filter EZ® -Boston Scientific; Natick-MS; USA) was positioned. Two heparin-bonded ePTFE SGs (Viabahn®-W.L. Gore & Ass.; Flagstaff-AZ; USA) were deployed using the telescope technique (distal first: 6 mm × 5 cm, then proximal 8 mm × 10 cm) and gently post-dilated to correct the focal stenosis at the common carotid artery. Final angiogram with cerebral study confirmed the complete exclusion of the CP with efficient flow into the internal carotid artery (Figure 2). Brachial and femoral accesses were closed with hand compression. The postoperative course was complicated: an expanding hematoma was observed at both percutaneous accesses. Conventional repair was required: a reversed saphenous vein graft was used to repair a dissecting flap of the brachial artery, whereas a primary suture was used to correct the defect of the femoral artery. The subsequent postoperative course was uneventful; he was discharged on day 6th postoperatively, and clopidogrel 75 mg (Plavix® - Bristol-Myers Squibb/Sanofi Pharmaceuticals; New York -NY; USA) plus acetylsalicylic acid 100 mg (Cardioaspirin ®-Bayer; Milano-IT) were started on a daily basis, *ad infinitum*. He was last seen 12 months after the procedure, asymptomatic: both the echo-color-Doppler and the CT-A showed the persistent exclusion of the CP, the shrinkage (7 mm) of the sac, the patency of the SGs without signs of endoleak or edge stenoses.

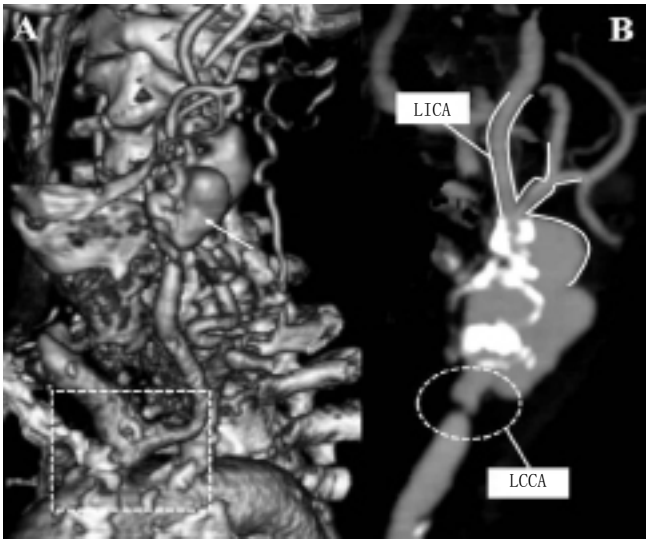

Figure 1. Preoperative CT-A: bovine arch configuration (*A*, *square*) and pseudoaneurysm (*arrow*) involving the whole patch. Multiplanar reconstruction (B) detailing the stenosis (*ring*) at the proximal aspect of the patch on the common carotid, and the take-off of the external carotid artery (*lines*).

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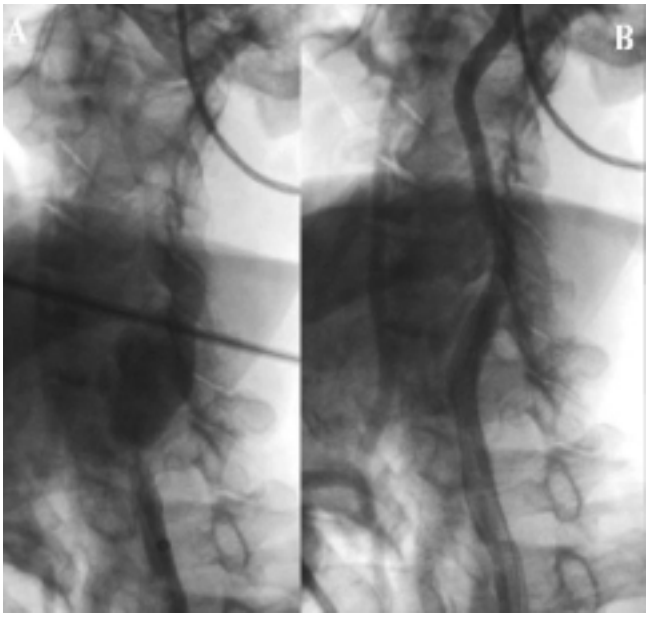

Figure 2. Intraoperative angiography: selective study (A) of the pseudoaneurysm, and final control after SG deployment (B) with the complete exclusion of the lesion.

3. Discussion

Carotid endarterectomy-related pseudoaneurysm is rare complication (less than 1% of all CEA) but the most frequently reported aneurysm of the extracranial carotid arteries[2,3,5]. Although the formation of the CP after patch angioplasty has been reported twice as common compared to primary closure, others have found a 0.33% incidence after patch angioplasty vs. 0.25% following primary closure,

a difference that is not statistically significant[1]. The most extensive series of CEA-related CPs has been published by El-Sabrout and Cooley[3] who reported a 57% incidence out of their 67 cases: in their data, patch closure was the dominant method of reconstruction.

The cause of our CP was undetermined. The pathogenesis of non infected CEA-related CPs may have different pathogeneses: rupture or dilatation of patch material, partial disruption of the suture line due to infection, or mechanical stress[3]. A recent review of Bond *et al*[19]. concluded that up to now there is no evidence to support the advantage of vein over synthetic patch material after CEA, but if synthetic material is selected, the available data appears to show benefits from PTFE as opposed to Dacron material. Because it occurred 25-years after the initial surgery, structural dilation of the entire Dacron patch have been considered a plausible cause. In the present brief review of CPs managed with SGs, we were unable to identify the type of reconstruction in 13% of the cases: nevertheless, in the reminder cases, we observed that 47% developed over a Dacron patch and 20% after vein patch (e.g. having superinfection in one case each), and no ePTFE patch dilation was reported.

Clinical onset of the CEA-related CPs is very heterogeneous in terms of delay from the original intervention: out of the 6/14 (43%) cases that developed within the first year, mean delay was 4 months vs. the mean 12 years of those presenting in the long run. Regardless the time of onset, an expanding pulsatile mass, together with related compressive symptoms, was the leading sign for medical alertness in both groups; cerebrovascular symptoms was observed in 1 case only.

Just cerebrovascular symptoms probably from thrombosis or embolism and the potential for possible rupture or fistulization should be considered reasonable indication for repair[1-5]. Before the advent of endovascular techniques, conventional surgery was the only modality to treat these lesions[2-4]. Even in the most renown centers, operative morbidity and mortality remained high: considering only the CPs, El-Sabrout *et al*[2]. reported and overall incidence of early mortality/major stroke of 10.8%, and only 13 (35.1%) patients remained asymptomatic without further interventions and showed no recurrence. Similarly, Hertzer *et al*[4]. had stroke, graft thrombosis, and a high incidence of cranial nerve injury (44%). More recently, endovascular repair has gained wide interest: the benefits of this approach include the avoidance of a hostile operative field, lack of risk of nerve injury, and accessibility of the high internal carotid artery[2]. Stent-graft repair of the pseudoaneurysm was weighted as the best approach for managing our case, because we had a large pseudoaneurysm that extended to the base of the neck requiring difficult proximal and distal vessels control. Even stent-grafting is not free of complication: in our review, we identified 1 (6.6%) death as a consequence of an acute myocardial infarction that was not possible to treat due to patient refusal, 2 (14.3%) access complications that required surgery, and further 2 (14.3%) cerebrovascular accidents that had no sequelae. However, to tell the truth, all the patients who were discharged alive are still doing well with no recurrent lesions (Table 1).

Carotid artery stenting is generally performed without cerebral protection for CEA-related re-stenosis or closure complication: the main source of embolization have been removed and re-stenoses are mainly an hyperplastic process[20,21]. We believe that in case of a CP distal embolization can still remain a threatening concern: in fact, sac thrombosis may generate emboli. This is the main reason why we used a protective filter; despite this, we noted that in the 15 cases we compiled in this brief review, filter protection has not been used ever. Filter protection should be a matter of debate: Martin *et al*[14]. clearly stated that unlike atherosclerotic occlusive disease, treatment of an aneurysm did not appear to cause an immediate intraoperative threat because a well-organized thrombus should carry less risk of distal embolization. Also McCready *et al*[12]. did not use filter to protect against embolization but after having had cerebrovascular complications their conclusive suggestion was to support cerebral protection devices with large CPs, even if were not able to substantiate recommendation. One potential alternative technique to prevent peripheral embolization could be a direct carotid artery access to circumvent the need to manipulate catheters through the aortic arch or filter devices. This approach has been suggested by Ahujia *et al*[15]. who described this approach to be more advantageous because the proximal aspect of the common carotid can be clamped during the stenting maneuvers, thereby minimizing the risk of embolization.

Endovascular treatments for CEA-related CPs have been already described with stent: Bush *et al*[22]. treated 2 cases of CP with stenting plus coils placed into the sac via the interstices. Alarmingly, a TIA was recorded few days after the procedure, specifically after a re-stenting procedure to correct an extremity stenosis. No details have been offered to the readers about that; it is difficult to think about a coil dislodgment, rather the friable thrombus inside the CP may have protruded into the endoluminal surface and may have been the source of emboli. The use of a SG could prevent either the risks of imprecise placement of coils or thrombotic embolization into the cerebral circulation. In addition, SG could also prevent a potential injury caused by a bare-metal stent: even if anecdotal, 2 cases of CP have been reported following carotid stenting.

Different devices have been reported in literature to be used: the advent of self-expanding device was an improvement over balloon-expandable because of their

Table 1

CEA-related carotid pseudoaneurysm treated with stent-graft.

Author, yr	Cases (*n*)	Age (year)	Gender	Intervention	Onset (months)	Signs/ symptoms	Sizing (mm)	Infection	Stent-graft	Devices (number)	Filter	Complication (type)	LOS (days)	Therapy (type)	Follow-up (months)	Outcome	Patency	Shrinkage	Recurrency
May, 1997	1	70	M	right CEA + n.r.	264	neck swelling	30 × 30	no	Passager®	1	no	minor stroke	7	Warfarin	6	alive	yes	yes	no
Lin, 2003	1	74	M	right CEA + pericardial patch	24	neck pain + expanding mass	50 × 60	no	Wallgraft®	1	no	uneventful	n.r.	n.r.	18	alive	yes	yes	
Hertz, 2003	1	80	M	right CEA + pericardial patch	30	neck pain + skin changes	42	no	Wallgraft®	2	no	AMI (1st p.op.)	6		0	dead			
Terramani, 2003	1	82	M	right CEA + dacron patch	1	expanding mass	41 × 36	no	Wallgraft®	2	no	hematoma	n.r.	n.r.	8	alive	yes	yes	no
Gupta K, 2004	1	72	M	right CEA + dacron patch	48	expanding mass	60 × 40	no	Viabahn®	1	no	uneventful	n.r.	Clopidogrel + ASA	6	alive	yes	yes	no
Baril, 2004	1	79	F	right CEA + dacron patch	132	TIA	n.r.	yes	Viabahn®	1	no	uneventful	n.r.	Clopidogrel + ASA	12	alive	yes	yes	no
McCready, 2004	1	58	M	right CEA + Surgisis patch	1	neck fullness	n.r.	no	Wallgraft®	2	no	TIA	n.r.	Clopidogrel	12	alive	no	yes	no
Mousa, 2005	1	82	F	right CEA + dacron patch	24	headache+ expanding mass	35 × 30	no	Wallgraft® + ECA coils	1	no	uneventful	2	n.r.	6	alive	yes	yes	no
Martin, 2005	1	68	M	right CEA + vein patch	204	expanding mass	60 × 25	no	Viabahn® + ECA coils	2	no	uneventful	n.r.	Clopidogrel + ASA	3	alive	yes	n.r.	no
Ahuja, 2007	2	70	M	left CEA + vein patch	240	expanding mass	63 × 53	no	Viabahn®	3	no	uneventful	n.r.	Clopidogrel + ASA	18	alive	yes	yes	no
		86	F	left CEA + vein patch	2	expanding mass	23 × 19	yes	Viabahn®	2	no	uneventful	n.r.	n.r.	12	alive	yes	n.r.	no
Briguori, 2007	1	76	M	left CEA + dacron patch	7	follow-up finding	15 × 10	no	Fluency®	1	no	uneventful	2	n.r.	3	alive	yes	n.r.	no
Gupta R, 2008	1	87	M	right CEA + n.r.	12	neck pain + hoarness	80 × 60	no	Wallgraft®	1	no	uneventful	n.r.	ASA	3	alive	yes	yes	no
Troutman, 2010	1	66	M	left CEA + dacron patch	3	neck pain + expanding mass	20 × 20	no	Wallgraft®	2	no	uneventful	1	Clopidogrel	108	alive	yes	yes	no
Present, 2011	1	76	M	left CEA + dacron patch	300	asymptomatic	35 × 40	no	Viabahn®	2	no	arm ischemia (1st p.op.)	6	Clopidogrel + ASA	12	alive	yes	yes	no
Total	15	758			86113										1627				

n.r. = not reported; CEA = carotid endarterectomy; LOS = length of stay; TIA = transitory ischemic attack; ASA = acetylsalicilic acid.

flexibility in conforming to tortuous segments, and because they easily accommodate varying diameters of the carotid arteries, especially at the transition from the common to the internal carotid[5,23]. In addition, a self-expanding stent is safer than a balloon-mounted stent because of potential arterial injury resulting from high balloon inflation pressures needed for stent deployment. The porosity and inflammatory nature of the Dacron covered SGs made it not ideal: Terramani *et al*[9]. reported the need to drain a persisting hematoma causing compressive symptoms fed by the porosity of the SG. In order to avoid this drawback, we preferred to use a more recent ePTFE device that have been optimized with technical and chemical aspects: the contoured edge as well as the heparin-bonded inner surface of the new Viabahn® was exploited to have better hemodynamic into the distal internal carotid artery and to prevent against stent re-stenosis.

Another interesting technical aspect to be discussed could be the need to exclude the external carotid artery. It has been suggested that if the external carotid artery arises in the vicinity of the pseudoaneurysm, it could be necessary to first embolize it with coils prior to SG placement, otherwise it will become a source of endoleak which may lead to subsequent pseudoaneurysm expansion[7,13,14]. This procedure could be offset by the risks of sequelae that include facial weakness and exertional pain associated with chewing. Reviewing the 15 cases we have identified we agree with most of them that did not perform preventive external carotid embolization.

Anatomical details of the external carotid artery take-off was not always specified: nevertheless, in all the 12 cases that did not perform embolization and used direct SG simply, no recurrency has been detected at a mean follow-up of 1 year and a half. Even if long-term results in the published literature is still lacking, simply because these devices have only been available in the past few years, SG management for CEA-related CPs seems to be a viable and effective treatment option with better results.

Conflict of interest statement

We declare that we have no conflict of interest.

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