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 renowned 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).

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 of view, he denied fever and local signs of infection (skin changes, erythema) were absent. Leukocytes count (7 850 cells/mm³) was in the normal range (4.3–11 000 cells/mm³), 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. Prooperative 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).

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[5]. 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[6], 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[44], 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 Ahuja 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 manevers, 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
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 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.

Table 1
CEA–related carotid pseudoaneurysm treated with stent–graft.

<table>
<thead>
<tr>
<th>Author, yr</th>
<th>Cases</th>
<th>Age</th>
<th>Gender</th>
<th>Intervention</th>
<th>Onset</th>
<th>Signs/ symptoms</th>
<th>Sizing</th>
<th>Infection</th>
<th>Stent–graft</th>
<th>Devices</th>
<th>Filter</th>
<th>Complication</th>
<th>LOS (days)</th>
<th>Therapy</th>
<th>Follow–up (months)</th>
<th>Outcome</th>
<th>Priaty</th>
<th>Shrinkage</th>
<th>Recurrence</th>
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<td>May, 1997</td>
<td>1</td>
<td>70</td>
<td>M</td>
<td>right CEA + 264</td>
<td>neck swelling</td>
<td>3 0 x no</td>
<td>Passage® 1</td>
<td>no minor stroke</td>
<td>7</td>
<td>Warfarin</td>
<td>alive</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
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<td>Lin, 2003</td>
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<td>74</td>
<td>M</td>
<td>right CEA + 24</td>
<td>neck pain + skin changes</td>
<td>5 0 x no</td>
<td>Wallgraft® 1</td>
<td>no uneventful</td>
<td>n.r.</td>
<td>n.r.</td>
<td>18</td>
<td>alive</td>
<td>yes</td>
<td>yes</td>
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<td>Hett, 2003</td>
<td>1</td>
<td>80</td>
<td>M</td>
<td>right CEA + 30</td>
<td>neck pain + skin changes</td>
<td>42 no</td>
<td>Wallgraft® 2</td>
<td>no AMI (1st 6 pop)</td>
<td>0</td>
<td>dead</td>
<td>8</td>
<td>alive</td>
<td>yes</td>
<td>no</td>
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<td>Terramani, 2005</td>
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<td>82</td>
<td>M</td>
<td>right CEA + 1 dacron patch</td>
<td>expanding mass</td>
<td>44 x no</td>
<td>Wallgraft® 2</td>
<td>no hematoma</td>
<td>n.r.</td>
<td>8</td>
<td>yes</td>
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<td>1</td>
<td>72</td>
<td>M</td>
<td>right CEA + 48</td>
<td>expanding mass</td>
<td>60 no</td>
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<td>n.r.</td>
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<td>alive</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
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<td>79</td>
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<td>TIA</td>
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<td>n.r.</td>
<td>Clopidogrel 12 + ASA</td>
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<td>yes</td>
<td>yes</td>
<td>no</td>
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<td>McCready, 2004</td>
<td>1</td>
<td>58</td>
<td>M</td>
<td>right CEA + Surgisis</td>
<td>neck fullness</td>
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<td>82</td>
<td>F</td>
<td>right CEA + 24</td>
<td>headache + expanding mass</td>
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<td>Wallgraft® + ECA coils</td>
<td>no uneventful</td>
<td>2</td>
<td>n.r.</td>
<td>6</td>
<td>alive</td>
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<td>Martin, 2005</td>
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<td>68</td>
<td>M</td>
<td>right CEA + 204</td>
<td>expanding mass</td>
<td>60 x no</td>
<td>Vialah® 2</td>
<td>no uneventful</td>
<td>n.r.</td>
<td>Clopidogrel 3 + ASA</td>
<td>alive</td>
<td>yes</td>
<td>nz.</td>
<td>no</td>
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<td>Aleja, 2007</td>
<td>2</td>
<td>70</td>
<td>M</td>
<td>left CEA + 240</td>
<td>expanding mass</td>
<td>63 x no</td>
<td>Vialah® 3</td>
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<td>n.r.</td>
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<td>yes</td>
<td>yes</td>
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<td>86</td>
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<td>expanding mass</td>
<td>23 x yes</td>
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<td>n.r.</td>
<td>12</td>
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<td>Briguori, 2007</td>
<td>1</td>
<td>76</td>
<td>M</td>
<td>left CEA + 7</td>
<td>follow-up finding</td>
<td>15 x no</td>
<td>Florec® 1</td>
<td>no uneventful</td>
<td>2</td>
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<td>3</td>
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<td>87</td>
<td>M</td>
<td>right CEA + 12</td>
<td>neck pain + bunion</td>
<td>80 x no</td>
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<td>Troutman, 2010</td>
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<td>66</td>
<td>M</td>
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<td>Present, 2011</td>
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<td>76</td>
<td>M</td>
<td>left CEA + 300</td>
<td>asymptomatic</td>
<td>35 x no</td>
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<td>no ama ischemia 6 (1st pop)</td>
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n.r. = not reported; CEA = carotid endarterectomy; LOS = length of stay; TIA = transitory ischemic attack; ASA = acetlysalicilic acid.
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.

**References**


