Fracture of a carotid stent and restenosis of common carotid artery

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In recent years we have observed dynamic growth in the amount of carotid stenting (CAS) as an alternative to surgical endarterectomy (CEA) [1].

We present a case of stent fracture of a balloon-expanding stent in a patient who underwent stenting for stenoses of the common and internal left carotid arteries.

A 56-year-old hypertensive woman was admitted to our institution in March 2007 due to symptomatic recurrent left carotid artery stenosis, manifested by headaches, dizziness, and transient visual disorders. In 1998, she experienced a stroke, after which she underwent a bilateral carotid endarterectomy. On admission, the patient reported periodic headaches and dizziness, transient visual disorders, and persistent aphasia. Additionally, she complained of some chest pain on exertion (class 3 CCS). Neurological examination revealed discrete symptoms of left-sided paresis, pyramidal signs on the right side, and a small motor aphasia. The patient was on permanent treatment with aspirin, clopidogrel, β-blockers, angiotensin-converting enzymes inhibitors, calcium channel blockers, diuretics and statins. Nuclear magnetic resonance (NMR) performed in ambulatory conditions did not indicate central nervous system damage. An ultrasound scan revealed a high-grade stenosis in the left internal carotid artery (LICA) – restenosis after CEA, with no significant flow disturbances in the right internal carotid artery (RICA).

On angiography, the LICA stenosis was confirmed, but also a second critical stenosis was diagnosed at the ostium of the left common carotid artery (LCCA, Figure 1).

A decision for carotid stenting was made, and this was performed on March 27, 2007. A balloon-expandable, stainless-steel Genesis stent (Cordis), 8 mm × 24 mm, was implanted (10 atm – 30 s) at the ostium of the LCCA. There was not much resistance in dilating the stenotic segment.

At this moment we decided to stop the procedure (Figure 2), and to perform the stenting of the LICA in the second stage, a couple of weeks later. The patient was discharged from the hospital with no complications after 2 days. Combined antiplatelet therapy was recommended, with 75 mg clopidogrel, for 4 weeks, in combination with 75 mg aspirin as permanent therapy.

On July 8, 2007 the patient was readmitted with the intent of treating the LICA stenosis. However, selective left carotid angiography revealed an

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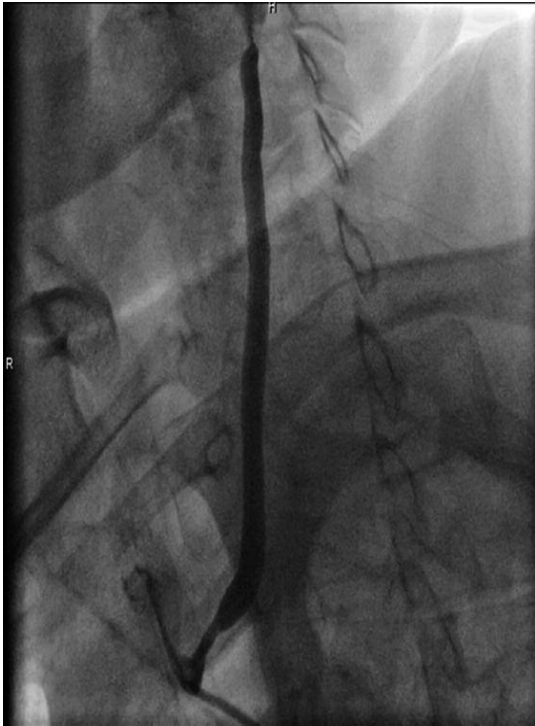
Figure 1. High-grade stenosis at the ostium and bifurcation of left common carotid artery

Figure 2. Angiographic result after stenting the ostium of the left common carotid artery
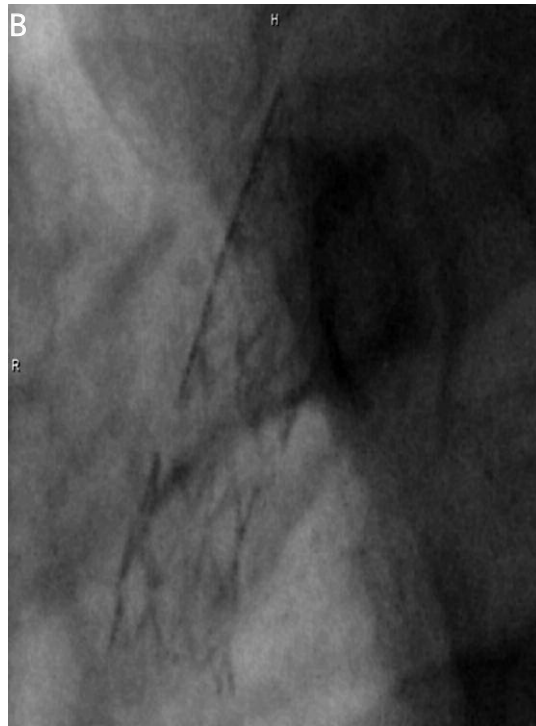
Figure 3. A – In-stent restenosis after stenting the ostium of the left common carotid artery. B – Stent fracture at the ostium of the left common carotid artery

in-stent restenosis within the previously implanted stent at the ostium of the LCCA. Furthermore, a fracture in the mid part of the stent was diagnosed with complete separation of the stent fragments. The fracture correlated with the area of restenosis (Figures 3 A and 3 B).

After surgical consultation it was decided that the patient would be treated with repeated percu-
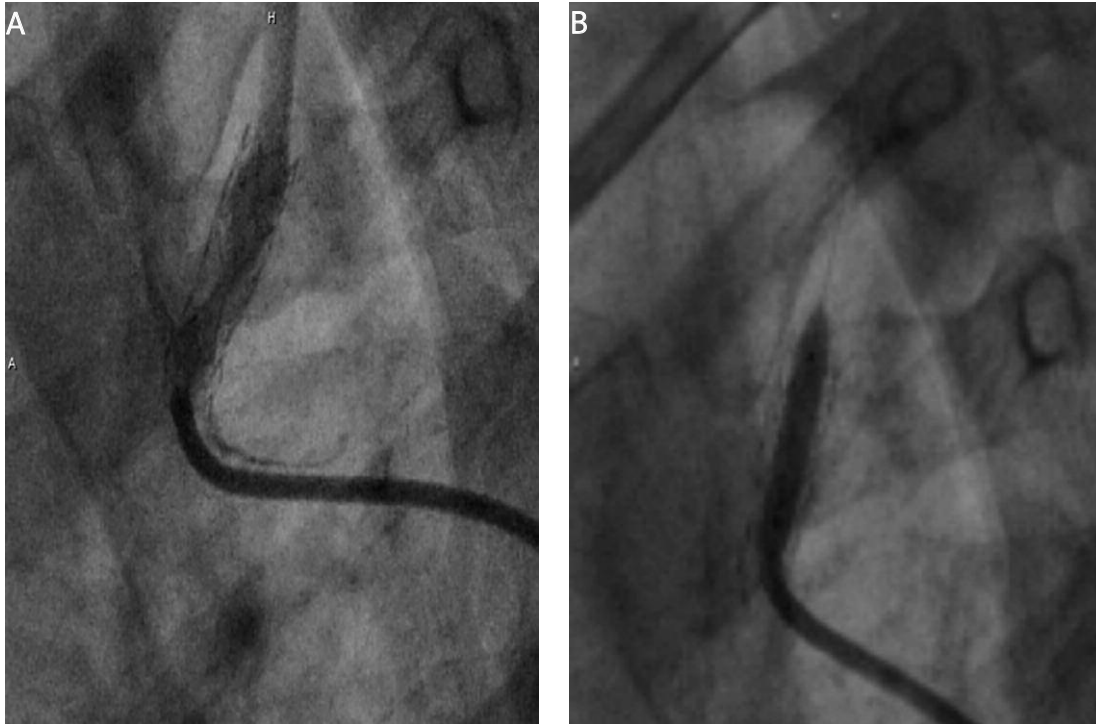
Figure 4 A, B. Balloon angioplasty of the left common carotid artery ostium

taneous intervention. The procedure was performed on August 2, 2007. This time we decided to treat both segments of the left carotid artery. The LCCA was approached using a Judkins right 8F guiding catheter. Then a cerebral protection device, EmboShield 5.5-7.0 mm (Abbott Vascular), was positioned in a distal part of the LICA. The distal lesion was pre-dilated (12 atm – 15 s) using an XTRM-WAY 4.0/15 mm balloon (Blue Medical Devices B.V.). The proximal lesion was predilated with the same balloon and a pressure of 10 atm. Subsequently, a Viatrac 5.5/15 mm balloon (Abbott Vascular) was positioned within the ostium of the LCCA and inflated twice with a pressure of 10 atm for 15 s. Then, a self-expanding, nitinol AccuLink stent (Abbott Vascular) 10/30 mm was positioned and opened in the distal lesion. The stent was post-dilated with the same Viatrac balloon and a pressure of 10 atm for 15 s. An optimal angiographic result was obtained, with a residual stenosis of less than 20% for both treated segments (Figures 4 A, B). The post-procedural course was uneventful, and the patient was discharged from the hospital the day after the procedure. Double antiplatelet therapy was recommended for 2 months.

In March 2010, a control angiography was performed, which revealed 40% stenosis at the ostium of the left common carotid artery (Figure 5).

In peripheral vessels, stent fractures are quite common after femoral and popliteal artery stenting. Only a few cases of carotid stent fracture have been reported and almost all of them concern self-expan-

Figure 5. The 40% stenosis at the ostium of the left common carotid artery

dable stents implanted in the region of the bifurcation of the common carotid artery [1, 2]. The main reason for stent fracture in this region may be the exposure of the device to different directional forces. In the neck, a significant rotational stress is put on a carotid stent as a result of movement around the atlanto-axial pivot joint. Also, flexional/extensional stresses are created by movements of the cervical

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vertebral joints. There are some additional risk factors such as calcification and angulation of the internal carotid artery [3]. A rare cause of stent fracture may be neck trauma [4]. Some studies show that internal carotid artery (ICA) stent fractures may be quite common. Regular surveillance with plain radiography in addition to duplex ultrasonography recognizes fracture in 1.9% to 29% of implants [3, 5]. Fracture of a stent may cause restenosis, except for areas of relatively large vessel diameter [6]. In a series of 14 consecutive patients with stent fracture, in-stent restenosis occurred in three of them (21%) [5].

So far, only one case of balloon-expandable stent fracture in the common carotid artery (CCA) has been reported [7]. In this case however, the event was not associated with in-stent restenosis. The explanation may be the fact that the proximal segments of this artery are partially protected by the wall of the chest, and no rapid movement, stretch or compression of the artery in this region is possible. Nevertheless, in our case the stent was implanted at the most proximal part of the CCA, at its origin from the aorta. Also unique is the fact that the fracture occurred very early, within three months after implantation. Certainly the fracture must have contributed to the in-stent restenosis, with significant narrowing of the vessel in the fractured area. The stent was widely and symmetrically opened with a pressure of 10 atm, which suggests that the vessel was not severely calcified.

Another important issue is the treatment of in-stent restenosis of the carotid arteries. Single reports suggest an endovascular approach with the use of another type of stent, while some others suggest stent-graft implantation or surgical treatment with stent removal, and possible accompanying endarterectomy as needed [3, 8-10].

In our case, a difficult and unfavorable localization of the stent (the ostium of the common carotid artery) was a substantial contraindication to surgical treatment. Out of multiple options for endovascular treatment we chose the simplest one – balloon angioplasty. The decision was made not only because of the simplicity of this technique, but also because of the need for additional stenting of the ICA, which we wanted to perform in a single-stage procedure.

The effect of angioplasty in a broken stent by means of a smaller-diameter balloon using similar pressure remains unknown due to the impossibility of angiographic verification. It cannot be ruled out that the episode of transient ischemic attack and passing left-sided paresis could be related to the increased area of restenosis of the proximal part of the brachiocephalic trunk.

Administration of antiplatelet agents is an essential component of care before the carotid stenting procedure. Most available data on adjunctive antiplatelet therapy and angioplasty pertain to the treat-ment of coronary occlusive disease. Performing the stenting procedure in our patient, we lacked sufficient literature evidence and recommendations for adjunctive therapy. Therefore clopidogrel was administered empirically for 5 days before the procedure, and this therapy was continued for 4 weeks thereafter. One can exclude, however, that either aspirin or clopidogrel could be responsible for in-stent restenosis and stent fracture. In view of our present experience, we would continue this antiplatelet therapy even longer, 3 to 6 months after the procedure.

The neurological improvement (mostly in terms of speech) after 16 months of observation of the patient treated by balloon angioplasty, however, suggests that the blood circulation in the central nervous system during this period has not worsened. This is confirmed by the ultrasound results, which showed no significant differences in the speeds of flow in both carotid arteries.

Control angiography would reveal the actual status of the vessel but unfortunately the patient declined this procedure. In light of our experience there still remains an open question as to the causes of rapid stent fracture with restenosis development and the choice of therapy in such cases.

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