A Combined Approach to Treatment of the Dissecting Middle Cerebral Artery Fusiform Aneurysm

A Case Report

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Summary

A fusiform aneurysm in the terminal M1 middle cerebral artery (MCA) segment was treated by a construction of a high-flow arterial extracranial-intracranial (EC-IC) bypass. Due to severe bypass vasospasms, local vasodilating agents together with percutaneous angioplasty and stent implantation were applied, but failed due to subsequent bypass occlusion. To remedy this complication a new bypass was created from a segment of the saphenous vein, followed by MCA aneurysm embolization and parent artery occlusion. One year after the surgery, the venous bypass remains patent and the aneurysm occluded, with the patient fully active, without any neurological sequelae.

Introduction

Detection of a fusiform dissecting aneurysm of the middle cerebral artery at the bifurcation brings concerns about its treatment. Intracranial fusiform aneurysms can be divided into two clinically different subtypes: acute dissecting aneurysms and chronic fusiform or dolichoectatic aneurysms^{1,2}. Of these two, the natural history and growth mechanism of chronic fusiform aneurysms remains unknown. We present the case of a 29-year-old woman with a large dissecting fusiform aneurysm in the depth

of the left Sylvian fissure where the sequence of interventions led to documented improvement through microsurgical implantation of a non-constrictive, venous vascular graft.

Case Report/Technique

Case description. A 29-year-old woman was initially examined by an otiatrist for complaints of pain in the left auriculotemporal area. Middle ear inflammation was excluded and the patient was referred to our centre for computed tomography examination. This revealed a globular calcified formation in the area of the left Sylvian fissure. Subsequent magnetic resonance angiography showed an ectasia of the terminal M1 segment of the left MCA. Digital subtraction angiography subsequently confirmed a dissecting fusiform aneurysm 25 x 24 x 20 mm of size, with a tubular ectasia of the terminal M1 segment behind the upper temporal artery and aneurysmatic pouch in the area of the left MCA division. The ectatic M1 segment partially surrounded the aneurysmal sac and divided it into a larger, ovoid-shape upper part, and a smaller, funnel-shaped formation, with the MCA protruding out from the lower pole (Figure 1A, B).

The family history was unremarkable and no major risk factors for aneurysm were identified except for the recurrent ipsilateral ear infec-

tions. As an infant the patient had been operated for the left middle ear inflammation, which relapsed after the surgery. This was stabilized only after antro-mastoidectomy at the age of ten. Culture of the left ear smear obtained perioperatively at that time revealed pyogenic *Staphylococcus aureus*. It is plausible that this infection or accompanying surgical injury could have contributed to the development of the dissecting fusiform aneurysm of the left MCA division.

Treatment plan

At presentation, the treatment options for the existing condition were confounded by the complex morphology, size and localisation of the aneurysmal sac. A survey of the relevant literature suggested that up to 80% of the patients with untreated “unclippable” large and giant dissecting aneurysms die or become severely disabled within five years from diagnosis, due to growth or rupture of the lesion¹. This makes the intervention both necessary and relatively urgent.

While an endovascular intervention involving bridging the aneurysm with a stent would have been the simplest option, this was thought to be unsafe, due to the shape of the lesion including: the aneurysmal ectasia originating 2 mm behind the lateral lenticulostriate arteries, significant stenosis at the entry to the aneurysmal sac and the pronounced lumen angulation (Figure 1 C,D). For these reasons we decided to apply a combined treatment including construction of a high-flow EC-IC bypass, connecting the insular segment of the left M2-MCA branch to the external carotid artery, with subsequent fusiform aneurysm embolization and terminal-MCA occlusion.

Intervention

For the construction of the high-flow bypass graft, the left radial artery segment was harvested with the intracranial end-to-side anastomosis placed onto the strong insular branch of the MCA trunk and the extracranial end connected to the prepared stump of the external carotid artery (ECA). Perioperative arterial bypass flow was monitored using the Doppler flow pencil probe (Transonic HT331 1.2-4.8MHz).

Immediately after the bypass construction, carotid angiography revealed massive arterial vasospasm at the intracranial portion, as well as

in the extracranial part of the arterial bypass graft, even though the arterial graft was prepared in a heparin bath and predilated with heparinised physiological saline syringe pressure. Spasms disappeared following systemic and local administration of vasodilating agents (*Isosorbide dinitrate* intraarterially, 10 ml in the course of 15 minutes and *Nimodipine* 15 ml intra-arterially in continuous infusion). Thereafter, we proceeded with the aneurysm embolization using the detachable coils Cerecyte-*Presidio* (Micrus Endovascular Corp., San Jose, Ca, USA) 6 pcs – 134 mm and GDC Vortex (Boston Scientific Corp., Fremont, Ca USA) 3.5 x 20 mm and 3 x 20 mm fibered coils.

Once again, following the aneurysm and MCA occlusion, massive vasospasm was detected on the follow up angiography in the extracranial part of the bypass graft. Due to this complication, isosorbide dinitrate was re-administered, followed by balloon angioplasty dilatation of the vasospastic portions and implantation of two Vision Coronary stents (Abbott Vascular, Illinois, USA) 3.5 x 20 mm and 3 x 20 mm into the stenosed areas. Subsequent angiography revealed good perfusion of the insular branch and the left cortical arteries.

The patient recovered from anaesthesia without any neurological deficit, but 16 hours after the procedure she developed Glasgow Coma Scale 7pts - sopor state and right side central hemiparesis. CT scan excluded intracranial haemorrhage, or ischemia. The carotid angiography again showed severe vasospasms of the bypass graft. (Figure 1E,F), which was corrected by a balloon angioplasty dilatation and further local intra-arterial spasmolytic nitrate administration, resulting in resolution of the hemiparesis.

Non-constrictive venous bypass graft

As the recurrence of the vasospasm was worrisome we decided to construct a second bypass graft to support the brain circulation using a small saphenous vein segment. We reasoned that a less muscular structure of this implant would relieve the scale of vasoconstructive events and the associated risk. A hollow opening in the temporal bone for the vein fixation was created and the upper end of the venous graft was connected end-to-side onto the intracranial part of the arterial anastomosis. The lower end of the bypass was connected to the stump of the left ECA below the primary anas-

Figure 1 A) Fusiform dissecting aneurysm (arrow) at the bifurcation of the left MCA in anteroposterior and lateral projections. Stenosis is apparent before the aneurysmal sac (entry side of dissection). Bilobular pouch of the aneurysm and branching of M2 – MCA segment from the tubularly widened terminal M1 – MCA part. B) Lateral projection of the aneurysmal sac (arrow). C) 3D-three-dimensional digital rotational angiography with the aneurysmal pouch of the terminal MCA segment reconstruction. Significant stenosis marked with an arrow is visible behind the upper temporal artery origin. D) Drawing of the fusiform dissecting aneurysm with the arrows indicating stenosis before the aneurysm and at the terminal M1 MCA segment angulation.

tomosis (Figure 1 G,H). Interestingly, the flow in the first arterial bypass disappeared after completion of the second venous graft. Post-operative angiography revealed sufficient flow via both the venous and distal intracranial part of the arterial bypass graft, feeding well the insular and core branches.

The patient was discharged from the hospital on the seventh post-operative day without any apparent neurological and cognitive alterations. During the course of the 12-month fol-

low up period the patient has been in a good physical and mental health and fully active, without any neurological symptoms. Periodical colour coded duplex Doppler flowmetry showed a sufficient blood flow with normal spectral analysis (Figure 1I,J).

Discussion

It was only in the last decade, that clinical trials have brought to light new findings concern-

Figure 1 E,F) DSA of the dysfunctional arterial EC-IC high-flow bypass with severe stenosis (arrow) in the intracranial section following an application of isosorbide dinitrate (Isoket), nimodipin (Nimotop) and balloon angioplasty. There is restraint blood flow through the arterial bypass graft (arrow) at the level of diffuse vasospasm. G) DSA taken at 3 post-operative days showing functional venous EC-IC high-flow bypass which connects the intracranial part of the arterial graft (arrow) with the embolized left MCA aneurysm. Therapeutic occlusion of the left MCA in the terminal M1 segment is also visible. H) Angiogram of the functional venous high-flow bypass in the lateral projection. There is an intracranial anastomosis connected to the previous arterial bypass. Good blood flow (arrow) is visible throughout the insular and cortical branches, with corresponding quick venous drainage via the cerebral sinuses.

ing the pathogenesis of fusiform aneurysms¹. According to Nakatomi et Al, the development of intracranial fusiform aneurysms occurs in four stages². The pathology of fusiform aneurysms includes the presence of a fibrous tissue on the outer wall and vascularized hyperplastic intima, but both the internal elastic membrane and tunica media layer are missing. Such a

weakened wall of these aneurysms easily ruptures and may cause death.

We believe that the formation of a fusiform aneurysm in our case could be related to a bacterial infection, causing a chronic recurrent middle ear inflammation lasting for about ten years. While the literature to this effect does not exist it is conceivable that such a chronic

inflammatory process may have affected the bone and also the wall of the MCA, causing a gradual development of a large dolichoectatic dissecting fusiform aneurysm. In our case, the aneurysm was classified as "type 3" according to Anne Osborn's classification of the fusiform aneurysms³.

In general, the recommended methods of treatment commonly related to saccular aneurysms suggest clipping and coiling procedures, which, however, are not suitable for dissecting fusiform aneurysms in the majority of clinical cases. One approach to correct such aneurysms is to bridge their lumens with a remodelling stent and aneurysm embolization. However, it is a risky and technically often ineffective corrective method with this type of aneurysms^{4,6}. The use of a muscle layer or polymers to strengthen the dissecting aneurysm wall would have been difficult in this case. Another possible treatment alternative is a resection of the affected MCA area and placement of an intracranial arterial bypass graft⁷. However, this procedure may be associated with a difficulty to construct the arterial anastomosis, composed of a thicker wall of the original artery. Taking all these factors into consideration, we chose a combined microsurgical and endovascular corrective approach, i.e. to ensure a sufficient flow to the MCA territory with an EC-IC bypass anastomosis, followed by parent artery occlusion and aneurysm embolization using platinum detachable coils. This intervention was hampered in our case by recurrent bypass graft vasospasms. It is well known that arterial bypass grafts are prone to vasospasms even if properly harvested, stored and manipulated during the surgical procedure. In our case this recurred in spite of graft predilatation and subsequent pharmacological and angioplastic intervention. It should be considered that the commonly used predilatation of the graft with pressurised physiological saline solution may result in overstraining of the muscular layer and even its disruption. Indeed, such a damaged wall may expose tissue factor in disrupted tunica media, causing thrombin generation, which in turn may contribute to vasospasms via endothelin released from the endothelium or other mechanisms^{8,9}.

The vasospasms of the arterial bypass graft in our patient were finally resolved only by construction of a second EC-IC bypass graft, using a small saphenous vein segment. Due to

Figure 1 I) Duplex Doppler - normal blood flow through the venous bypass graft at cervical portion. Normal spectral flow analysis of the lumen of 2.8 mm of internal diameter. J) Normal spectral blood flow in the terminal bypass section, as well as in the M2 MCA branches, while the embolized MCA aneurysm remains without flow.

poorly developed tunica media (responsible for vasoactivity), the lumen of the venous bypass graft was patent, thus supplying sufficient blood flow to the left MCA. A similar surgical procedure was also reported in literature for the treatment of fusiform aneurysms amenable neither to clipping nor to endovascular intervention^{10,11,12}.

The sufficient blood flow through the venous graft was periodically measured throughout the six-month follow-up after the procedure with colour coded Doppler flow imaging. The creation of a hollow in the temporal bone retroauricularly ensures the correct positioning of the

graft and protects it against possible graft wall traumatization or compression. Construction of the venous EC-IC high-flow bypass together with aneurysm embolization and parent artery occlusion is an alternative and novel approach for the treatment of these complicated fusiform aneurysms. The use of the venous graft segment for the second bypass construction was effective and safe. This may suggest that the non-

constrictive nature of venous grafts could be more advantageous than the more traditional arterial grafts.

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