Vertebrobasilar Dissection with Pseudo-aneurysm or Subarachnoid Hemorrhage: Intracranial Stenting as the Only Treatment

A Report of Three Cases

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Summary

Vertebrobasilar dissection may cause ischaemia or subarachnoid haemorrhage and can pose a significant treatment challenge. Endovascular treatment using stents alone has been described but there are few reports of its clinical application. We here report our experiences from three cases of vertebrobasilar dissection and pseudo-aneurysm or subarachnoid hemorrhage treated with stents alone.

Introduction

Intradural dissection of the vertebral artery is an unusual cause of subarachnoid hemorrhage¹ with significant mortality of 19% – 83%. Posterior fossa ischaemia and infarct are also possible sequelae due to thrombotic emboli or progressive luminal stenosis. Numerous congenital diseases, such as Ehler-Danlos syndrome Type IV, α_1 -antitrypsin deficiency or hyperhomocysteinaemia increase the likelihood of dissection, as does trauma². A defect in the internal elastic lamina is thought to be the weak point which results in the underlying tunica media dissecting under the influence of arterial pressure. If the dissection traverses the vessel wall it may cause a subarachnoid hemorrhage or create a thin-walled pseudo-aneurysm that may rupture at a later stage. Once the vessel has ruptured, it is prone to re-rupture³.

Endovascular reconstruction using stents alone has recently been proposed for the treatment of intradural vertebral artery dissection⁴. We here report our experience from three such cases.

Case 1

A 49-year-old manual labourer presented with sudden severe headache. He had otherwise been healthy prior to admission. At presentation his Glasgow Coma Score (GCS) was 13, however his condition quickly deteriorated to GCS 3 (Hunt and Hess 5) before an urgent computed tomography (CT) scan could be arranged. This disclosed a large subarachnoid hemorrhage. A ventricular drain was inserted and the patient was transferred to our tertiary care unit.

The following day the patient had recovered to GCS 14. Conventional and CT angiography revealed a tortuous basilar artery for which he received symptomatic treatment only (Figure 1). He remained an inpatient.

Fourteen days later a significant deterioration of the patient's condition (GCS 6) heralded a likely third subarachnoid hemorrhage. Repeat CT angiography showed unequivocal basilar dilatation and pseudo-aneurysm of the basilar artery (Figure 2). With the patient under general anaesthesia two Neuroform 4.5 mm x 20 mm stents (Boston Scientific, USA) and one Wingspan 4.5 mm x 15 mm stent (Boston Sci-

Figure 1 Anteroposterior CT angiogram at admittance showed only a slight basilar artery dilatation.

Figure 2 Anteroposterior CT angiogram 15-days after symptom onset showed severe basilar artery dilatation and pseudo-aneurysm formation.

entific, USA) were deployed in the basilar artery. Abciximab (18 mg bolus followed by overnight infusion) and heparin (bolus of 5000 units) were used for anticoagulation during the procedure. As the patient was intolerant to aspirin, clopidigrel 75 mg daily was the only drug used for continuing anticoagulation during the following eight weeks.

The patient was discharged from hospital five weeks after the treatment. At discharge he

was mobile, eating and drinking and oriented in time, place and person. A moderate dysphasia persisted. Within a few months the basilar artery regained a near normal configuration (Figure 3). It is now two years since the intervention and although his condition has continued to improve slowly, he is prevented from returning to his previous employment by diplopia and vertigo. A ventriculoperitoneal shunt remains in situ.

Case 2

A 46-year-old male nurse presented with left-sided weakness, confusion and difficulty swallowing. CT angiography the following day showed bilateral extracranial internal carotid artery (ICA) dissections and an aneurysm in the C1 segment of the left internal carotid artery as well as a probable old occlusion of the right vertebral artery. The left vertebral artery showed evidence of prior extracranial and acute intracranial dissection with a narrow remaining lumen and intramural pseudo-aneurysm. The posterior fossa was to some extent also supplied by a narrow left posterior communicating artery. Magnetic resonance imaging (MRI) showed infarcts in the medulla oblongata, pons, the occipital lobes and cerebellum. Nevertheless, the patient recovered well and was able to walk with assistance two days after symptom onset. He was started on dicumarol. Twelve days after admission the pa-

Figure 3 Anteroposterior angiogram of the right vertebral artery 1-year after treatment with stents alone shows aneurysm resolution and remodelling of the parent artery.

tient's condition worsened significantly and he was transferred to our tertiary care unit. He had developed a hemiplegia with a positive Babinski sign on his left side as well as nystagmus. The left pupil was fixed. Repeat MRI showed an additional right pontine infarction. His condition deteriorated progressively over the coming hours with dysphagia, aphasia and almost complete left-sided hemiplegia. The patient was eventually unable to maintain an open airway and was intubated.

Pre-intervention angiography showed progression of the intramural dissection of the distal left vertebral artery with extension into the basilar artery (Figure 4) and a vertebral artery pseudo-aneurysm. Under general anaesthesia a 37 mm Enterprise stent (Cordis Neurovascular Inc., USA) was deployed over the dissection, with immediate restoration of flow to the vertebrobasilar system. However, the intramural cavity remained open. It was also noted that the dissection of the left ICA had progressed, but without affecting flow in the vessel. After the procedure the patient was given 75 mg clopidigrel daily for three months. He also received 75 mg aspirin for six months. Fifteen days after the treatment the patient returned to the referring hospital.

It is now three months since treatment (Figure 5). The pseudo-aneurysm is present but stable. The patient is still receiving neurorehabilitation as an inpatient and continues to improve. He walks with a stick and has a left hemiparesis and visual field defect. His swallowing and speech is mostly intact.

Case 3

A retired 66-year-old man arrived at our unit unconscious with GCS 6. He was known to suffer from type II diabetes mellitus and hypertension. A CT scan showed a large subarachnoid hemorrhage and a ventricular drain was immediately inserted. Subsequent angiography suggested the source of the bleed to be dissection of the basilar artery (Figure 6). Due to the patient's poor condition treatment of the dissection was postponed.

Over the following four days the patient improved to GCS 10 and thus we made an attempt to secure the bleeding point. With the patient under general anaesthesia two stents (Neuroform 4.5 mm x 20 mm, Neuroform 4.5 mm x 15 mm) were deployed in the basilar artery using the 'stent within a stent' technique.

Figure 4 Lateral angiography showing left vertebral artery dissection and pseudo-aneurysm.

During the procedure the patient received a bolus of 20mg abciximab and 4000 units of heparin, followed by an infusion of 600 units of heparin. Continuing anticoagulation, starting the day after the procedure, was clopidigrel 75

Figure 5 Lateral CT angiography 3-months after treatment shows the stent in place. The pseudo-aneurysm is still present though slightly reduced in size.

Figure 6 Angiography before intervention shows a dissected basilar artery with a pseudo-aneurysm.

mg daily and aspirin 75 mg.

Due to his severe subarachnoid hemorrhage the patient's condition slowly deteriorated and ten days after the procedure he fell unconscious. Several CT angiograms showed a stable aneurysm and there was no evidence of rebleed (Figure 7). The patient was discharged to secondary care three weeks later and quietly passed away a few days later. Clinically there

Figure 7 The patient's condition deteriorated 10-days after intervention, CT-angiography however showed a slight reduction in aneurysm size and no evidence of rebleed.

was no suspicion of rebleed. Unfortunately it was not possible to carry out a post-mortem.

Discussion

Patients presenting with subarachnoid hemorrhage following vertebrobasilar dissection may be at up to 70% risk of rebleed in the first 24 hours and so urgent treatment is required to reduce mortality⁵. Experience with the Neuroform stent suggested that it could be used alone to treat at least small dissecting aneurysms or pseudo-aneurysms⁶.

Although the treatment of ruptured and unruptured vertebrobasilar dissections using a 'sole stenting' technique has been described in the literature¹, there are still relatively few accounts of its clinical application. It is a minimally invasive approach which may be considered when other treatments, such as parent vessel occlusion or aneurysm coiling with or without balloon remodelling or stenting are impossible or dangerous. Compared to balloon mounted stents, a self-expanding stent reduces the risk of arterial rupture during stent deployment. However, there is the risk that catheter manipulation or stent placement might cause further dissection, vessel occlusion or perforation. Cannulation or stenting the false lumen is another potential hazard.

Flexible, self-expanding stents like for example the Neuroform, Wingspan or Enterprise systems may work in several ways to treat cerebral dissections and aneurysms. Firstly, they exert a radial force which stabilizes the vessel wall and compresses dissected layers to maintain a lumen. Secondly, the stent attenuates flow vortices⁷ giving rise to stagnation and the formation of stable aneurysmal thrombus.

Thirdly, the stent mesh serves as a matrix for neointimal formation, a process which is thought to begin about two weeks after implant⁸. Finally, stents have been shown to reduce wall shear stress (WSS) to a desirable level. Both high and low WSS are implicated in detrimental vascular remodelling, aneurysm growth and aneurysm rupture⁹.

These concepts have been applied in two new stents recently introduced to the market. The Pipeline (Chestnut Medical, Menlo Park, CA, USA) and the Silk (Balt, Montmorency, France) stents are intended as monotherapy for aneurysms in the cerebral vasculature. The main benefit of these systems is a dense stent

mesh which diverts flow more effectively. In animal models, Pipeline completely or nearly completely occluded 88% of aneurysms, flow to branch vessels was preserved and neointimal hyperplasia decreased over time to 15% at six months¹⁰. There are only two published cases of Pipeline implanted in humans; both gave good results at up to one year follow-up¹¹.

Anticoagulation is routinely used for all endovascular interventions to reduce the risk of thromboembolism. Furthermore, as stents are inherently thrombogenic dual antiplatelet therapy is a prerequisite. Despite these measures none of the patients described here bled or rebled after treatment.

Interestingly, the patient that did not tolerate aspirin and received clopidogrel alone did not develop in-stent thrombosis.

Conclusions

In conclusion, vertebrobasilar dissection with pseudo-aneurysm or subarachnoid hemorrhage may be treated successfully with stents alone, albeit experience with this technique is very limited. If possible proven techniques like stent assisted coiling or balloon remodelling should be used.

Experience with the new generation of stents will tell whether this practice continues to be the case.

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