Case Series

Endovascular and Surgical Management of Vertebral Artery Dissecting Aneurysms Presenting With Subarachnoid Haemorrhage: Medium-term Experience

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Abstract

Introduction: Dissecting aneurysms of the vertebral artery are associated with a high incidence of rebleeding and mortality if untreated. Current endovascular alternatives to surgery are being evaluated. Materials and Methods: We conducted a retrospective review of our experience with endovascular as well as surgical treatments of dissecting vertebral artery aneurysms in 6 patients. Three patients were treated with a combination of coils in the proximal artery and aneurysm sac; 1 with a combination of coils and balloon; and 2 with aneurysm clipping surgery. All patients were treated with the primary intention of coil deposition within the aneurysm sac and proximal vertebral artery. No proximal flow arrest was used. Anticoagulation was given during and after the procedure. Results: Technical success was achieved in all patients, and there was no post-procedural neurological deficit. Follow-up for a mean of 40 months (range, 7 to 64) confirmed the clinical stability of symptoms, as well as the cessation of flow to the aneurysm. Conclusions: Endovascular treatment of vertebral artery dissections is a viable alternative to surgery in the appropriate setting. It has the benefit of being minimally invasive, allowing concomitant balloon testing as well as retaining the option to convert to surgery.

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Key words: Balloon occlusion, Embolisation, Intracranial haemorrhages, Vertebral artery dissections

Introduction

Dissecting aneurysms of the vertebral artery often present with subarachnoid haemorrhage.[1] A high mortality occurs with rupture.[2] Currently, the treatment options for vertebral artery dissecting aneurysms presenting with subarachnoid haemorrhage (SAH) are still controversial and include trapping of the aneurysms with or without bypass as well as proximal occlusion, performed either traditionally via open surgery or through endovascular means. The retrospective results and clinical follow-up of endovascular therapy with coils in 3 patients, a combination of coils and balloons in 1 and surgery in 2 patients are presented.

Materials and Methods

This is a retrospective study of 6 consecutive patients presenting with SAH from vertebral artery dissecting aneurysms. All patients were treated during the acute stage of the disease with the primary intention of depositing coils within the aneurysm and the parent artery. None of the aneurysms were of traumatic aetiology. The diagnosis of subarachnoid haemorrhage was made on non-enhanced computed tomographic (CT) scans of the brain in all patients. The dissecting aneurysms were diagnosed on 4-vessel angiogram or magnetic resonance imaging (MRI)/angiography. Test balloon occlusion was performed in 1 patient to assess tolerance for permanent parent vessel occlusion.

Technical Protocol

Initial diagnostic angiograms were obtained via the transfemoral approach with 5 Fr Headhunter (Cordis Europa NV, Johnson & Johnson, Netherlands) catheters. Selective catheterisation was achieved with a Fastracker 18MX or Excelsior microcatheter (Target Therapeutics, Boston

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Scientific, MN, USA). The modality of treatment was decided after consensus between the referring neurosurgeon and the interventional radiologist was reached. Informed consent was obtained prior to treatment. A combination of Guglielmi detachable coil (GDC) 10 and GDC 18 (Boston Scientific, MN, USA) and complex helical fibred coils 18 (Target Therapeutics, Boston Scientific, MN, USA) were employed to achieve occlusion within the aneurysm sac as well as within a short segment of proximal artery. No proximal flow protection was employed in our endovascularly treated patients. In 1 patient (patient 1), the mode of treatment was converted to open surgery after a failed attempt at endovascular therapy. In another (patient 2), the coils were supplemented with the use of a detachable silicone balloon (Target Therapeutics, Boston Scientific, MN, USA).

Results

A summary of our patient characteristics is presented in Table 1. All of our patients did not experience rebleeding after admission. One patient presented to us during an episode of rebleeding but had no subsequent rebleeding after admission. Balloon test occlusion was performed and tolerated in 1 patient, in the others a test occlusion was deemed unnecessary due to the presence of a contralateral dominant or co-dominant vertebral artery. Our patients did not experience any deterioration in neurological function or rebleeding after treatment. Follow up periods ranged from 7 to 64 months with a mean of 40 months. Three patients made complete recoveries and 3 continued to have residual neurological deficit. Treatment options and outcomes are summarised in Table 2.

Table 1. Patient Characteristics

Patient no.	Age/sex	Location	Side	WFNS grade	Angiogram findings
1	45/M	Post-PICA	R	4	Fusiform
2	45/M	Pre-PICA	R	2	Fusiform
3	59/F	Post-PICA	L	3	Fusiform
4	47/M	Pre-PICA	R	4	Fusiform
5	42/M	Distal VA (absent R PICA)	R	2	Fusiform
6	42/M	VA-PICA	L	3	Fusiform

L: left; PICA: posterior inferior cerebellar artery; R: right; VA: vertebral artery; WFNS: World Federation of Neurosurgeons

Table 2. Treatment Interval and Rebleeding

Patient no.	Treatment	Interval until (days)	Pre-treatment rebleeding	Balloon test	Post-treatment rebleeding
1	Proximal clipping	33	N	N	N
2	Coiling	2	N	N	N
3	Coiling	26	Y (day 5)	Y	N
4	Coiling	7	N	N	N
5	Coiling	2	N	N	N
6	Proximal clipping	120	N	N	N

Patient 1

A 45-year-old Chinese male with no medical history of note lost consciousness while playing jackpot. On arrival, his neurological status was a World Federation of Neurosurgeons (WFNS) grade of 4. He had conjugate gaze deviation to the right. A non-enhanced CT of the head revealed subarachnoid blood in the basal cisterns and cerebral oedema. An urgent 4-vessel angiogram showed an 11.7 x 6 mm-fusiform aneurysm in the post-posterior inferior cerebellar artery (PICA) segment of the right vertebral artery. An attempt at endovascular occlusion was made but unfortunately, a 360-degree, irregular, atherosclerotic tortuous loop was found in the proximal vertebral artery, complicating catheter navigation to the aneurysm with kinking of the microcatheter. Conversion to post-PICA proximal artery clipping was performed 33 days from presentation. A check angiogram 7 months later revealed occlusion of the post-PICA right vertebral artery, with distal retrograde filling from the vertebro-basilar junction. The aneurysm was not seen. The patient recovered uneventfully, and at follow-up 64 months later could walk with the aid of a walking stick.

Patient 2

A 45-year-old Chinese male presented with a “thunderclap” headache, vomiting and drowsiness. On arrival, his GCS was 13 (WFNS Grade 2) and he had a transient right sixth nerve palsy. A non-enhanced CT of the head revealed a small amount of blood in the third and fourth ventricles, with moderate obstructive hydrocephalus. Angiography performed the next day revealed an intradural right pre-PICA fusiform aneurysm measuring 15 x 6.5 mm. Coil embolisation 2 days from presentation involved a total of 15 GDC and 11 fibred coils to achieve endosaccular and parent vessel occlusion. However, despite tight packing, slow antegrade flow was still present. Complete occlusion was finally achieved with a detachable balloon just proximal to the coil. The left vertebral artery showed good antegrade flow into the posterior circulation with preservation of normal branches. There was no interval change in the patient’s neurological status. An MRI 10 days later showed complete occlusion of the right distal vertebral artery. At the last outpatient appointment 22 months later, the patient demonstrated full recovery with no residual diplopia.

Patient 3

A 59-year-old Malay lady presented with sudden loss of consciousness preceded by headache, diplopia and vomiting while she was in the Middle East. She recovered and returned to Singapore 5 days later, with a severe headache and neck stiffness. On admission, she was found to have neck stiffness, left sixth nerve and right eighth nerve palsies as well as bilateral lower limb weakness (WFNS 3). A non-enhanced CT of the head revealed a subarachnoid haemorrhage with intraventricular extension, but no hydrocephalus. Four-vessel angiography the next day revealed a fetal origin of the right posterior cerebral artery, dominant left vertebral artery, with a post-PICA fusiform dilatation of the left vertebral artery near the PICA origin. Parent artery and endosaccular coiling was performed after successful test occlusion 26 days from initial presentation, with preservation of the left PICA. A check angiogram performed at the end of the procedure showed filling of the left posterior cerebral artery from the anterior circulation with a small amount of reflux to the basilar tip. She was well on follow-up 54 months after her first admission, with only residual bilateral lower limb paraesthesia.

Patient 4

A 47-year-old Chinese male presented with Grade 4 WFNS subarachnoid haemorrhage. He was intubated, and angiogram revealed a pre-PICA fusiform dilatation of the right vertebral artery. Endosaccular and proximal artery coiling was performed on day 7 of admission. On follow-up 55 months later, he had residual loss of lower limb function, but was independent in activities of daily living.

Patient 5

A 42-year-old Chinese male with no past medical history of note presented with a sudden-onset severe headache, loss of consciousness, a 15-minute episode of generalised tonic-clonic seizure, and urinary incontinence. On arrival, his GCS was 14 (WFNS 2). He complained of neck stiffness, but no localising neurological sign was elicited. His blood pressure (135/80 mm Hg), pulse (83 bpm) and blood glucose levels and clotting profile were within normal limits. An urgent non-contrast enhanced CT of the brain revealed extensive subarachnoid haemorrhage with intracranial extension (Fig. 1a) and early obstructive hydrocephalus (not shown). An angiogram performed the next day revealed a dissecting aneurysm located in the distal right vertebral artery 1 cm proximal to the vertebro-basilar junction. The left PICA was dominant and an AICA/PICA variant was seen on the right (Figs. 1b and 1c). The patient returned the next day (2 days post-admission) for endosaccular embolisation of the dissection. Seven GDC coils of 18 and 10 calibre and 7 fibred coils were used to obtain complete occlusion of the vertebral artery. A subclavian angiogram at the end of the procedure demonstrated good perfusion of the basilar system from the left vertebral artery with normal opacification of the intracranial vessels of the posterior circulation. There was no contralateral filling of the aneurysm (Figs. 1d and 1e).

The patient recovered, with no recorded episode of symptomatic rebleeding. A follow-up MRI/MRA scan of the brain 6 months later revealed thrombosis of the terminal right vertebral artery, with patency of the left vertebro-basilar system and the rest of the circle of Willis. No infarct or haemorrhage was identified. He remained well at the review at 38 months, with no neurological deficit.

Patient 6

A 42-year-old Indonesian male with a history of non-insulin-dependent diabetes mellitus (on diet control) presented with a subarachnoid haemorrhage involving the basal cisterns diagnosed on CT in his home country. He recovered after a month in hospital and came to Singapore electively for further management. He had difficulty walking, with motor weakness elicited in both lower limbs (WFNS 3). Admission blood tests were unremarkable, with a normal clotting profile and full blood count. An MRI/MRA revealed a left posterior inferior cerebellar artery aneurysm.

A 4-vessel angiogram was performed, showing a fusiform aneurysm arising just distal to and involving the junction of the left PICA and vertebral artery (Figs. 2a and 2b). The patient underwent surgical clipping 4 months after his presentation. A left suboccipital craniotomy and clipping with reinforcement from two 9-mm clips distal to the left PICA was performed. He made an uneventful recovery.

A postoperative angiogram performed 7 months later did not reveal opacification of the aneurysm. There was good opacification of the left PICA artery, which also supplied the right PICA territory (Figs. 2c and 2d). The right vertebral artery and its branches were opacified, with no reflux into the terminal left vertebral artery seen.

Discussion

There are 2 forms of dissecting vertebral artery aneurysms: intra- and extradural aneurysms. Extradural dissections are usually managed medically with anticoagulation and may present with embolic events related to the ipsilateral PICA or basilar territories. Only in medically refractory cases is surgical or endovascular treatment employed to occlude or isolate the aneurysm.

In contrast, dissecting aneurysms (perhaps more accurately termed aneurysms of dissection) of the intradural vertebral arteries often present with subarachnoid haemorrhage.[1] A second episode of bleeding (rebleeding) is common and deadly. Rebleeding rates were estimated at 71% of cases by Mizutani et al,[2] with subsequent re-rupture of the aneurysms in 57%. A high mortality was associated

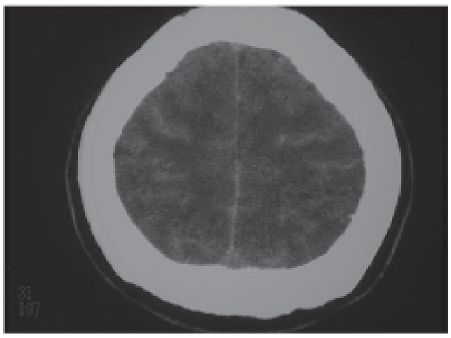

Fig. 1a. 42-year-old Chinese male with headache. Unenhanced axial CT demonstrates blood in the subarachnoid space.

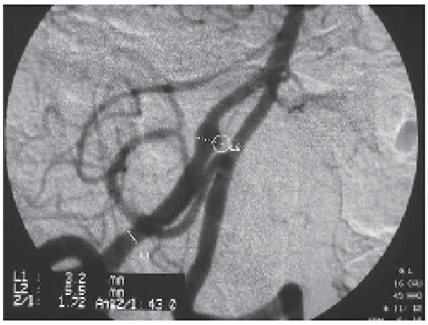

Fig. 1b. 42-year-old Chinese male with headache. Oblique projection of catheter angiogram of the posterior circulation demonstrates dissecting aneurysm located in the distal right vertebral artery 1 cm proximal to the vertebro-basilar junction. The left PICA was dominant and an AICA/PICA variant was seen on the right.

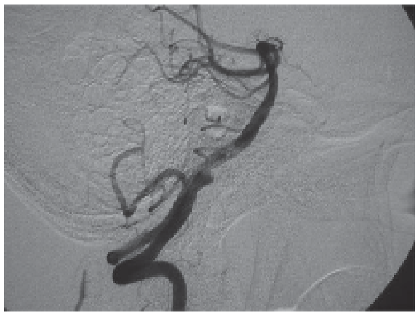

Fig. 1c. 42-year-old Chinese male with headache. Lateral projection of catheter angiogram of the posterior circulation confirms the presence of a dissecting aneurysm.

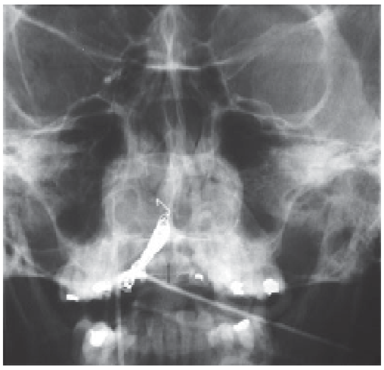

Fig. 1d. 42-year-old Chinese male with headache. Unsubtracted frontal projection demonstrates coils within the aneurysm.

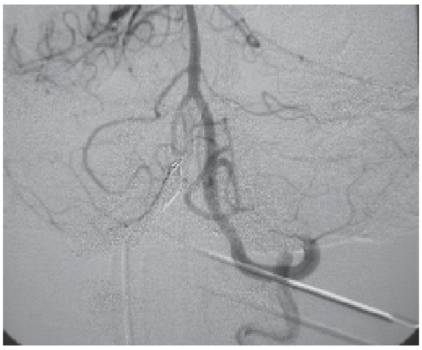

Fig. 1e. 42-year-old Chinese male with headache. Repeat angiogram demonstrates no retrograde filling of the aneurysm via a contralateral vertebral artery angiogram.

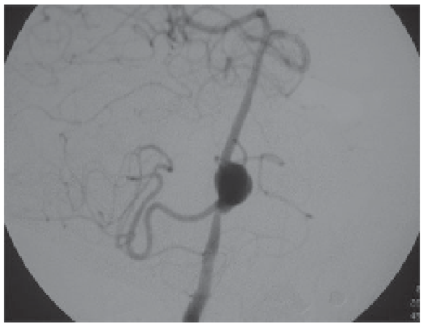

Fig. 2a. 42-year-old Indonesian male with subarachnoid haemorrhage. Frontal projection of selective right vertebral artery catheter angiogram demonstrates a saccular aneurysm arising from the right VA-PICA junction.

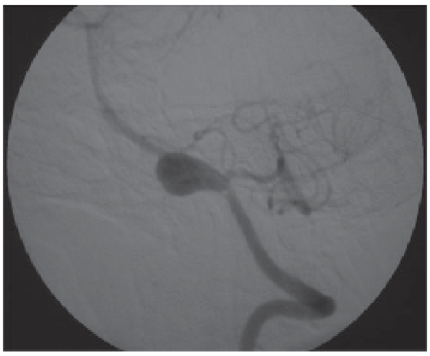

Fig. 2b. 42-year-old Indonesian male with subarachnoid haemorrhage. Lateral projection of selective right vertebral artery catheter angiogram demonstrates a saccular aneurysm arising from the right vertebral artery-PICA junction.

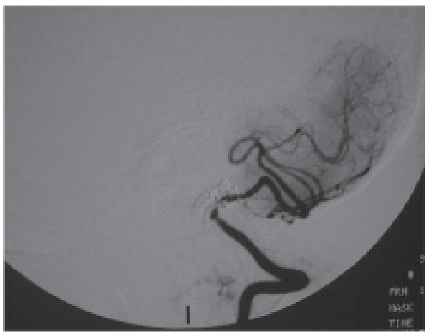

Fig. 2c. 42-year-old Indonesian male with subarachnoid haemorrhage. Lateral projection of post-surgical clipping right vertebral artery catheter angiogram confirms obliteration of the aneurysm.

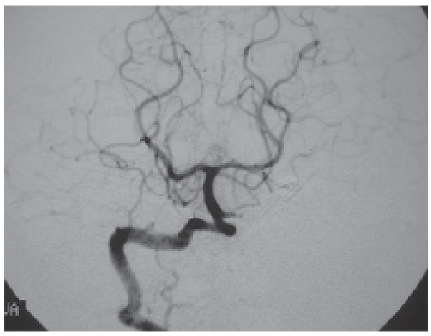

Fig. 2d. 42-year-old Indonesian male with subarachnoid haemorrhage. Lateral projection of post-surgical clipping right vertebral artery catheter angiogram confirms obliteration of the aneurysm. Note absence of right PICA opacification.

with aneurysm re-rupture (46.7%), compared to 8% without re-rupture.[2] The mortality figures are likely to be underestimated since subarachnoid haemorrhage around the brainstem can induce cardiopulmonary arrest,[3] resulting in under-reporting of the actual number of cases involved.

Vertebral artery dissection occurs more commonly in males between 35 and 50 years of age. There is no established association with atherosclerosis or hypertension.[3,4]

The clinical presentation of dissecting aneurysms includes persistent severe neck pain, headache and sixth to tenth cranial nerve palsies.[1] Transient coma or loss of consciousness (1 to 24 hours) occurs in 26% to 33% of patients.[2,3]

Vertebral artery dissecting aneurysms are classified according to their location along the vertebrobasilar axis, either pre-PICA, post-PICA, or involving the PICA origin. With dissections involving 1 vertebral artery only, the aneurysm is located distal to the posterior inferior cerebellar artery origin (post-PICA) in 61% of cases. Those involving the PICA origin occur in 8% to 20%, and pre-PICA, in 14% of these patients.[2,3] Some studies[2,3] suggest a tendency for involvement of the right vertebral artery.

Radiological investigations usually start with an unenhanced CT scan, which may reveal blood in the fourth ventricle or basal cisterns. When CT is negative, lumbar puncture is mandatory in the correct clinical setting and may reveal evidence of xanthochromia.

Magnetic resonance angiography is also able to detect the presence of acute SAH on the FLAIR (fluid attenuated inversion recovery) sequence[5] as well as demonstrate the aneurysm itself. Findings include visualisation of a double lumen and intimal flap, intramural haematoma and haematoma in the parent artery.[6]

Catheter angiography is usually the next investigative step. The diagnostic angiographic features of a dissecting aneurysm include: a fusiform dilatation associated with proximal and/or distal narrowing, the “pearl and string” sign and retention of intramural contrast in the venous phase. A linear defect giving rise to a double lumen appearance is pathognomic. The diagnosis is confirmed surgically by visualisation of an intramural haematoma.

Certain pitfalls are associated with angiography during the acute presentation. It may not reveal a fusiform aneurysm that blends with background atherosclerosis. In addition, overlap of the aneurysm with a branch of the anterior circulation on a single radiographic projection may be misinterpreted as a negative finding.[6] The use of rotational angiography and 3D-angiography may reduce the incidence of these misdiagnoses. Therefore, a high index of suspicion and repeat angiography are indicated in the face of a negative first angiogram.[3,7]

The timing of repeat angiography depends on clinical judgement and on the adequacy of the initial angiogram. If non-filling of a particular vertebral artery is detected on direct cannulation, attempts should be made to look for vascular reconstitution from other branches of the external circulation.[8] If the vertebral circulation is insufficient, the carotid contribution to the basilar artery assumes increased importance.

Dissecting vertebral artery aneurysms can be treated with either surgical or endovascular techniques. The optimal method of treatment remains highly individualised to the case at hand, and the lack of standard protocols for treatment is partly due to the relative rarity of this condition.

Surgical Treatment

Early surgery during an acute episode of SAH in posterior fossa aneurysms is associated with significant mortality of up to 30%,[2,9] but this is counterbalanced by the catastrophic outcome of re-rupture. Early surgery has also been advocated to relieve the pressure of a haematoma, if present.

Dissecting aneurysms are, by their nature, not amenable to neck clipping and isolation. This is because they have no aneurysm neck over which a clip may be easily applied. Hence, proximal (Hunterian) occlusion, or trapping is employed provided that the contralateral vertebral artery is of adequate capacity to supply the basilar artery distally. Surgical trapping is very effective in preventing further episodes of rebleeding. It is assumed that once a clip can be securely applied to trap or proximally occlude the aneurysm, thrombosis is achieved and the aneurysm will regress. Yamaura et al[4] reported good or moderate recovery in 15 of 19 operated cases of vertebral artery dissection, with no cases of recurrent haemorrhage. The remainder suffered severe disability but these patients had presented with severe clinical disability (Hunt and Hess grade V) in the first place.

Surgical treatment is complicated by a number of factors. As the majority of dissecting vertebral artery aneurysms arise post-PICA and are often located near the midline, surgical treatment can be technically difficult.[10] It may often be complicated by lower cranial nerve palsies.[7] Trapping surgery is more complicated than proximal clip occlusion because of the amount of retraction necessary on the brainstem and the limited visualisation of the distal end of the aneurysm with this approach.

Positioning of the clip relative to the PICA merits discussion. Yamaura et al[4] reported 3 cases of postoperative lateral medullary syndrome in 19 patients with dissecting vertebral artery aneurysms treated with proximal clipping and application of the clip distal to the PICA origin. This is noteworthy as it is a result of occlusion of brainstem perforating arteries rather than occlusion of the PICA alone. They proposed that when the PICA is a major supplier of blood to the medulla, the PICA should remain patent proximal to the clip. When the vertebral artery is the main supplier (through perforating branches), clip occlusion should be performed proximal to the PICA origin so that collateral perfusion is preserved.[4]

We did not detect any episodes of symptomatic brainstem ischaemic episodes in our surgically treated patients (n = 2).

Endovascular Treatment

Endovascular treatment is divided into proximal parent vessel occlusion (sacrifice), and endosaccular packing. A variety of materials may be used. They can be simply divided into detachable balloons or coils. The major difference between balloon and coil deployment lies in the rapidity of inducing thrombosis. When a balloon is deployed, flow dynamics are altered immediately, sometimes with dramatic effect. Balloons can be deployed in relatively large vessels. The main disadvantages associated with balloons include the relatively large size of delivery systems (usually 8 or 9 French), as well as the inadvertent release and migration of the balloon, resulting in improper placement.

With coils, however, achievement of thrombosis takes time. The consequence is a theoretical chance of distal emboli during coiling, which gave rise to the concept of proximal protection with a temporarily inflated balloon (see below). Coils may be employed for both proximal vessel occlusion or endosaccular packing. They can also be delivered using much smaller systems (5/6 Fr guide catheter systems).

The use of temporary proximal flow arrest using a nondetachable balloon (proximal protection) has been suggested to reduce the risk of distal emboli during coil deployment for proximal artery occlusion.[3] However, Kurata et al[3] did not employ proximal protection (flow-arrest techniques) in their series of 24 patients. The reason for not doing so was to avoid inadvertent distal coil migration when restarting blood flow on deflation of the protecting balloon. Instead, they assessed coil fixation by intermittent angiography.

The need for proximal protection was shown to be unnecessary with proper size selection of the first coil (33% to 50% larger diameter than that of the parent artery) and systemic intra-procedural anticoagulation.[11] All of our patients who underwent endosaccular packing were treated without proximal flow arrest, but they received systemic anticoagulation. No symptomatic embolic episodes occurred during or after the procedures.

In treating aneurysms of vertebral artery dissection, 2 basic approaches may be employed: proximal vessel occlusion or endosaccular packing (internal trapping) with sacrifice of the vessel. The haemodynamic effects of flow alteration from parent vessel occlusion either with a balloon or surgical clipping are similar if the embolic agent (balloon or coil) is deployed just proximal to the aneurysm.[12] The other technique, endosaccular packing, uses mainly coils. Here, the "sac" of the aneurysm as well as the adjacent parent vessel are filled with coils to achieve thrombosis.

Parent vessel occlusion, however, may not completely prevent subsequent rupture as there may still be persistent blood flow from the contralateral vertebral artery in a retrograde manner or antegrade filling from a patent thyrocervical trunk. The risk of rebleeding becomes higher if the aneurysm is further from the site of occlusion,[10] due to the time required for distal progression of thrombosis. In addition, the use of balloon occlusion carries the risk of obliteration of brainstem perforators, often located between 14 mm proximal and 16 mm distal to the vertebrobasilar union,[13] as the length of artery obliterated by the balloon is slightly more than in surgical clipping. In a study of 5 patients treated with balloon proximal parent vessel occlusion, Tsukahara et al[12] reported rebleeding in 2 patients. Four of their 5 patients did not demonstrate resolution of their aneurysms at follow-up (19 to 48 months) due to postulated collateral circulation filling. For proximal parent vessel occlusion, Halbach et al[14] stipulated the need to deploy the balloon or coil at, or just proximal, to the lesion in order to avoid incomplete thrombosis. If the length of artery permits, a combination of both endosaccular and parent vessel coiling can be used to achieve complete occlusion.

In endosaccular occlusion, coils are deposited within the aneurysm "sac" itself. The term may be misleading since most dissecting vertebral artery aneurysms are fusiform rather than saccular (like berry aneurysms). Endosaccular occlusion therefore occludes the parent vessel as well. Endosaccular occlusion became feasible mainly with the development of the GDC system. Relying on electrolytic detachment, this elegant system allows the coil to be manoeuvred into the optimal position and then deployed by electrolysis without mechanical movement within the fragile aneurysm. Furthermore, softer GDCs can be made more compact and densely packed when deployed. Therefore, the system permits the shortest segment occlusion of the dissected site.[10] The coils are deployed within the true lumen, collapsing the dissection and inducing thrombosis. Soft GDC coils also prevent trauma to the fragile aneurysm walls. GDC coils are made of platinum, which makes them MRI compatible. The thrombogenic effect of the coils is difficult to predict, necessitating packing with many coils within an aneurysm.

In 1 of our patients, endosaccular coiling alone was not able to stop the blood flow despite the deposition of 15 GDCs and 11 fibred coils within a large and dominant vertebral artery. The patient received intra-procedural anticoagulation. Finally, proximal balloon occlusion was employed to achieve cessation of flow. This case illustrates the point that in evaluating endovascular treatment, allowance should also be made for adequate length of the parent vessel, should the need for simultaneous endosaccular and proximal balloon occlusion arise.

Kurata et al[3] published their findings in a series of 24 ruptured dissecting vertebral artery aneurysms. Endosaccular embolisation was performed within 4 days of onset of symptoms. They experienced no complications with coil embolisation. Radiologic investigation showed complete occlusion of the dissection and patency of the unaffected artery at a mean follow-up of 9 months.[3] We report similar results over our 40-month follow-up period.

Bilateral Dissecting Vertebral Artery Aneurysms

We have thus far dealt with dissecting aneurysms of a single vertebral artery. In cases of bilateral dissection, the preferred modality of treatment has not been clearly established due to the rarity of this condition. Mizutani et al[2] described 1 patient with bilateral vertebral artery ruptured dissecting aneurysms managed conservatively, and 3 others treated surgically with proximal clipping and trapping. One patient treated with proximal clipping had severe disability, and the other 3 made complete recoveries (including the 1 patient managed conservatively because of spontaneous occlusion of the symptomatic side followed by healing of the contralateral side angiographically 3 years later). Kurata et al[3] reported 1 case of bilateral dissection where only 1 side was treated with coil embolisation initially. Bleeding due to progression of the contralateral dissection could not be prevented. The authors concluded that in patients with bilateral dissection or, incidentally, hypoplastic contralateral VAs, prior bypass surgery or stent placement should be considered.

Dissections Involving the PICA

Another difficult scenario arises with dissections involving the PICA. The difficulty arises because the contralateral vertebral artery may perfuse the PICA via rich posterior circulation collaterals. This continued perfusion may delay the onset of thrombosis. Proximal occlusion of these aneurysms may result in delayed rupture because of continued contralateral vertebral artery perfusion. The alternative treatment, which is trapping, means sacrificing the PICA, with the consequence of a cerebellar infarct. In particular, dissecting aneurysms involving the PICA within 10 mm of the midline and more than 13 mm from the clivus to the aneurysm are associated with serious morbidity.[4] Mizutani et al[2] described their experience in 7 such patients, 3 of whom were treated with trapping, and 4 with proximal clipping alone. Two patients in the proximal clipping group suffered subsequent re-rupture of aneurysms, and 1 demonstrated continual filling of the aneurysm by the contralateral vertebral artery. The other patient recovered completely. Patients treated with trapping fared worse, with 2 patients suffering severe disability. However, 1 patient with PICA involvement as well as bilateral aneurysms fared better due to an abundant posterior collateral circulation. Yamaura et al[10] reported on a case of a ruptured right PICA-vertebral artery junction aneurysm treated with GDC, resulting in an "excellent" outcome.

More recently, Yasui et al[15] reported 2 patients with vertebral artery dissections involving the PICA treated surgically. Clipping of the PICA beyond the choroidal point (the site at which the PIA produces small rami to the anterior aspect of the tonsil and choroid plexus of the fourth ventricle) can be undertaken in theory, because important brainstem and deep cerebellar nuclei perforators arise proximally. Ideally, trapping of the aneurysm with PICA reconstruction should be performed, but there is a risk of brainstem infarct if the anastomosis fails. These outcomes cannot be predicted by preoperative angiograms, and the ideal treatment has yet to be established. Iihara et al[16] proposed a treatment algorithm for such aneurysms. When the diagnosis is made, endovascular proximal occlusion of the vertebral artery can be performed. If the other intact vertebral artery is hypoplastic or absent, balloon test occlusion of the affected parent vertebral artery should be performed under neurophysiological monitoring. Once the patient survives the acute stage of SAH, and recovers consciousness, repeat angiography may be performed to look for contralateral perfusion of the aneurysm. If there is continued filling, test balloon occlusion is performed via the contralateral vertebral artery, and sited distal to the aneurysm. This is to test if endosaccular trapping may be safely performed. Ipsilateral external carotid arteriography is also performed to look for collateral perfusion of the PICA territory (since it will be obliterated in endosaccular coiling). If the patient tolerates a 20-minute occlusion, endosaccular coiling is performed. Otherwise, a bypass procedure will need to be performed. They reported an overall treatment morbidity of 16.7% with this algorithm, and it remains to be seen if it will enter mainstream practice.[16]

Hypoplastic or Aplastic Contralateral Vertebral Artery

The decision to treat aneurysms of the dominant vertebral artery is complicated by the presence of a hypoplastic or aplastic contralateral vertebral artery. In this case, preoperative balloon test occlusion may be useful in evaluating brainstem perfusion, as measured by somatosensory and brainstem-evoked potentials. Balloon test occlusion cannot be carried out at the same time as surgical treatment, which is an advantage for endovascular treatment. Unfortunately, there are disadvantages associated with balloon test occlusion, including the increased risk of bleeding.[7] Furthermore, successful tolerance of the test does not predict delayed brainstem infarcts caused by thrombosis of perforating arteries.[10] Therefore, in such cases surgical control or prior stenting may be considered as the preferred treatment modality.

Disadvantages of Endovascular Treatment

Several disadvantages are inherent in the endovascular technique. Access to the site of aneurysm may be difficult in tortuous arteries. In our study, we had a patient with a 360-degree loop in his vertebral artery, which made access to the dissection site particularly difficult despite numerous attempts by the neuroradiologist. In the end, the patient underwent proximal artery clipping. In addition, the endovascular approach to atherosclerotic arteries can also be hazardous and difficult as these vessels are tortuous, and the risk of dislodging a thrombus is significant. Furthermore, haematoma cannot be evacuated with an endovascular approach.

Conclusion

Endovascular treatment is a fast-developing field. A new approach to treating these dissections with stents and a combination of coils looks promising. The idea is to place a stent across the neck of the aneurysm to act as scaffolding for coil deposition within the sac of the aneurysm. Lylyk et al[17] published their results in 8 consecutive cases, with complete occlusion in 7 patients. Follow-up with angiography and clinical examination for a mean of 13.1 months (range, 4 to 42 months) showed no deterioration or rebleeding. The technique has the benefit of preserving parent vessel patency while occluding only the aneurysm. The authors encountered several difficulties, including flexibility, trackability and radiopacity of the stents. In addition, porous stents were used, and the problems of neointimal hyperplasia and increased thrombogenicity have not been fully evaluated in these cases yet. Ideally, a stent-graft can be employed, but designs commercially available at the time of writing are not yet flexible enough.

Citing difficulties of inserting coils between the struts of the porous stents, Benndorf et al[18] suggest using double stent placement (one within the other) to narrow the pores between the stents, hence altering blood flow to the aneurysm. The flow alteration is postulated to accelerate thrombosis within the dissection. Certainly, it will be interesting to follow the development of these devices.

We report no significant complications, rebleeding or severe disability with surgical and endovascular treatments in our cohort of patients. The presenting grade of haemorrhage correlated with final outcome, regardless of treatment modality (Table 3). It may be that the high degree of mortality associated with this condition results in a selection bias in favour of patients with a better prognosis anyway. Furthermore, this study is limited by the small number of cases due to the rarity of this condition. We are also limited by the biases inherent in a retrospective review.

Table 3. Presentation Grade, Treatment Modality and Outcomes

Presentation grade (WFNS)	Treatment modality	Outcome
4	Surgery	MD
4	Coil	MD
3	Coil	MD
3	Coil/balloon	CR
2	Coil	CR
2	Surgery	CR

CR: complete recovery; MD: moderate disability but independent in activities of daily living; SD: severe disability

1: Glasgow Coma Scale (GCS) 15; 2: GCS 13-14, no motor deficit; 3: GCS 13-14, with motor deficit; 4: GCS 7-12; 5: GCS 3-6

The final choice of treatment depends on several factors: the patient's anaesthetic risk, the location of dissection, amount of purchase on the parent vessel available for endosaccular and proximal balloon occlusion, involvement of the PICA, dominance or hypoplasia of the contralateral artery, and feasibility of catheter navigation. We feel that endovascular treatment should be considered first as the procedure can be performed with less morbidity and is efficacious. If the dissection is not amenable to endovascular treatment, the information gained from the angiography will aid in surgical planning.

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REFERENCES

1. Yamaura A, Watanabe Y, Saeki N. Dissecting aneurysms of the intracranial vertebral artery. J Neurosurg 1990;72:183-8.
2. Mizutani T, Aruga T, Kirino T, Miki Y, Saito I, Tsuchida T. Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. Neurosurgery 1995;36:905-13.
3. Kurata A, Ohmomo T, Miyasaka Y, Fujii K, Kan S, Kitahara T. Coil embolisation for the treatment of ruptured dissecting vertebral aneurysms. AJNR Am J Neuroradiol 2001;22:11-8.
4. Yamaura A. Diagnosis and treatment of vertebral aneurysms. J Neurosurg 1988;69:345-9.
5. Wiesmann M, Mayer TE, Yousry I, Medele R, Hamann GF, Bruckmann H. Detection of hyperacute subarachnoid hemorrhage of the brain by using magnetic resonance imaging. J Neurosurg 2002;96:684-9.

6. Andoh T, Shirakami S, Nakashima T, Nishimura Y, Sakai N, Yamada H, et al. Clinical analysis of a series of vertebral aneurysm cases. Neurosurgery 1992;31:987-93.
7. Nohjoh T, Houkin K, Takahashi A, Abe H. Ruptured dissecting vertebral artery aneurysm detected by repeated angiography: case report. Neurosurgery 1995;36:180-3.
8. Pritz MB. Evaluation and treatment of aneurysms of the vertebral artery: different strategies for different lesions. Neurosurgery 1991;29:247-56.
9. Peerless SJ, Hernesniemi J, Gutman FB, Drake CG. Early surgery for ruptured vertebrobasilar aneurysms. J Neurosurg 1994;80:643-9.
10. Yamaura I, Tani E, Yokota M, Nakano A, Fukami M, Kaba K, et al. Endovascular treatment of ruptured dissecting aneurysms aimed at occlusion of the dissected site by using Guglielmi detachable coils. J Neurosurg 1999;90:853-6.
11. Barr JD, Lemley TJ. Endovascular arterial occlusion accomplished using microcoils deployed with and without proximal flow arrest: results in 19 patients. AJNR Am J Neuroradiol 1999;20:1452-6.
12. Tsukahara T, Wada H, Satake K, Yaoita H, Takahashi A. Proximal balloon occlusion for dissecting vertebral aneurysms accompanied by subarachnoid hemorrhage. Neurosurgery 1995;36:914-20.
13. Mahmood A, Dujovny M, Torhe M, Dragovic L, Ausman JI. Microvascular anatomy of foramen caecum medulla oblongatae. J Neurosurg 1991;75:299-304.
14. Halbach VV, Higashida RT, Dowd CF, Fraser KW, Smith TP, Teitelbaum GP, et al. Endovascular treatment of vertebral artery dissections and pseudoaneurysms. J Neurosurg 1993;79:183-91.
15. Yasui T, Komiyama M, Nishikawa M, Nakajima H. Subarachnoid hemorrhage from vertebral artery dissecting aneurysms involving the origin of the posteroinferior cerebellar artery: report of two cases and review of the literature. Neurosurgery 2000;46:196-201.
16. Iihara K, Sakai N, Murao K, Sakai H, Higashi T, Kogure S, et al. Dissecting aneurysms of the vertebral artery: a management strategy. J Neurosurg 2002;97:259-67.
17. Lylyk P, Cohen JE, Ceratto R, Ferrario A, Miranda C. Combined endovascular treatment of dissecting vertebral artery aneurysms by using stents and coils. J Neurosurg 2001;94:427-32.
18. Benndorf G, Herbon U, Sollman WP, Campi A. Treatment of a ruptured dissecting vertebral artery aneurysm with double stent placement: case report. AJNR Am J Neuroradiol 2001;22:1844-8.