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Vertebrobasilar artery dissection: current practice

椎骼基底的動脈解剖：現行的處理方法

Nine patients with vertebrobasilar artery dissections who presented with neurological symptoms or subarachnoid haemorrhage were identified and treated in the Neurosurgical Unit at the Prince of Wales Hospital in Hong Kong. An account of these patients is given and their treatment described. This paper further reviews selected literature to outline the concepts and appropriate management of vertebrobasilar artery dissections.

香港威爾斯親王醫院神經外科組對九名呈現神經徵兆或蛛網膜下出血的椎骼基底動脈夾層動脈瘤患者，進行了診斷和治療。本文敍述了這些患者的情況及相應之治療，並進一步總結了所選的文獻，以略述椎骼基底動脈夾層動脈瘤的概念和適當的處理措施。

Introduction

Vertebral artery dissections (VADs) are increasingly recognised as a cause of subarachnoid haemorrhage (SAH) or cerebrovascular ischaemia. These dissections may occur in the extracranial or intracranial segments of the vertebral artery; dissections in the latter may extend into the basilar artery. Vertebral artery dissections, which are potentially fatal, have no clearly stratified treatment protocols in the literature. With the introduction of endovascular treatment modalities in the management of cerebrovascular disease, however, several options have become available to physicians in the treatment of VADs. We report a series of patients with VADs and use these cases to illustrate the different treatment modalities available in contemporary neurovascular medicine.

Key words:
Aneurysm, dissecting;
Basilar artery;
Subarachnoid hemorrhage;
Vertebral artery

關鍵詞：
動脈瘤，夾層動脈瘤；
基底動脈；
蛛網膜下出血；
椎動脈

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Case series

Case 1

A 54-year-old hypertensive man experienced a sudden onset of severe headache and neck pain. On admission, the patient was fully conscious and demonstrated no focal neurological deficit. A computed tomography (CT) scan of the brain revealed SAH. A digital subtraction angiogram (DSA) revealed a right-sided intracranial fusiform vertebral artery aneurysm. The patient subsequently underwent endovascular obliteration of the lesion by complete occlusion at the site of pathology using six Guglielmi detachable coils (GDCs). A repeat DSA 6 months later revealed complete occlusion of the lesion. The patient made a full recovery.

Case 2

A 54-year-old man experienced a sudden severe headache and was admitted to hospital fully conscious and with no focal neurological deficit. A CT scan of the brain revealed SAH and intraventricular haemorrhage. A DSA demonstrated a right-sided intracranial fusiform vertebral artery aneurysm just proximal to the origin of the posterior inferior cerebellar artery (PICA). The patient underwent endovascular occlusion of the lesion at the site of pathology using nine GDCs, resulting in complete occlusion and leaving the PICA intact, as evident from backflow during a contralateral vertebral artery injection. The patient made a full recovery but refused follow-up angiography.

Case 3

A 63-year-old diabetic and hypertensive man was admitted to hospital after a sudden collapse. He was confused and had mild right hemiparesis. A CT scan of the brain revealed SAH and hydrocephalus that required a ventriculostomy. An initial DSA was not helpful in establishing the cause of SAH, as the patient was restless and only limited images could be acquired. The patient subsequently had a ventriculoperitoneal shunt inserted, followed by a second DSA, which demonstrated a left-sided intracranial fusiform vertebral artery aneurysm. Because the DSA suggested that the origin of the PICA was involved in the aneurysm, a surgical trapping procedure was performed. During the operation, a very small PICA was encountered and preserved. The patient made a good recovery and is independent for daily activities.

Case 4

A 43-year-old man presented to hospital after a first-time generalised convulsion. A CT scan of the brain revealed SAH. A DSA revealed a right-sided intracranial fusiform vertebral artery aneurysm just proximal to the PICA. Endovascular occlusion of the aneurysm was performed using nine GDCs detached at the site of pathology. A contralateral vertebral artery injection demonstrated good cross flow into the right PICA. The patient made a good recovery and a repeat DSA several months later revealed persistent complete occlusion.

Case 5

A 58-year-old hypertensive man presented to hospital with sudden onset of left hemiplegia. A CT scan and magnetic resonance imaging (MRI) of the brain demonstrated a pontine infarct and ectatic left vertebral and basilar arteries with haemorrhage in the wall of these vessels. A DSA confirmed vertebrobasilar dolichoectasia. The clinical and radiological features suggested a diagnosis of vertebrobasilar dissection. In hospital, the patient experienced a sudden deterioration in level of consciousness, which was thought to result from an extension of the dissection, as repeat brain scanning revealed no additional haemorrhage. To prevent further dissection, while preserving flow through the basilar artery, endovascular stenting of the affected segment was performed using three coronary artery stents. The procedure was uncomplicated and technically successful, but the patient did not regain consciousness and died during the admission.

Case 6

A 35-year-old woman presented to hospital with a 2-week history of moderately severe neck pain radiating to the occipital region. This did not resolve with the use of routine analgesics. There were no other symptoms or signs. Magnetic resonance imaging of the neck revealed haemorrhage in the wall of the left vertebral artery (Fig 1), and a magnetic resonance angiogram (MRA) demonstrated an irregular area in the left extracranial vertebral artery at cervical level C2 to C3 (Fig 2). The clinical and radiological features were thought to represent an extracranial VAD (ECVAD) and the patient received anticoagulation therapy for 3 months to prevent ischaemic complications. A follow-up MRA demonstrated resolution of the lesion and anticoagulation therapy was discontinued.

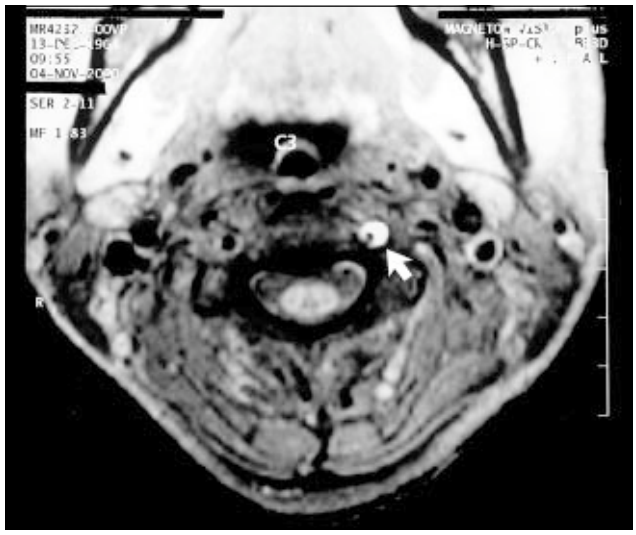


Fig 1. Spin-echo T1-weighted fat suppression axial magnetic resonance imaging at the C3 level, showing a haematoma in the wall of the left vertebral artery and narrowing of the lumen (white arrow)

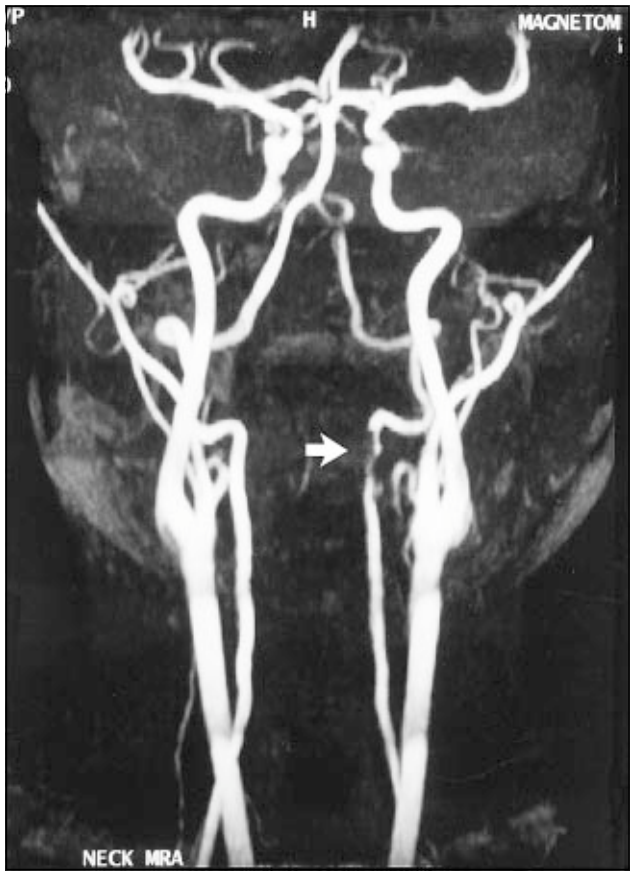


Fig 2. Three-dimensional time-of-flight magnetic resonance angiogram showing narrowing of the left vertebral artery at C2/C3 level (white arrow)

Case 7

A 55-year-old man presented to hospital having collapsed while playing chess in a park. On examination he was dull

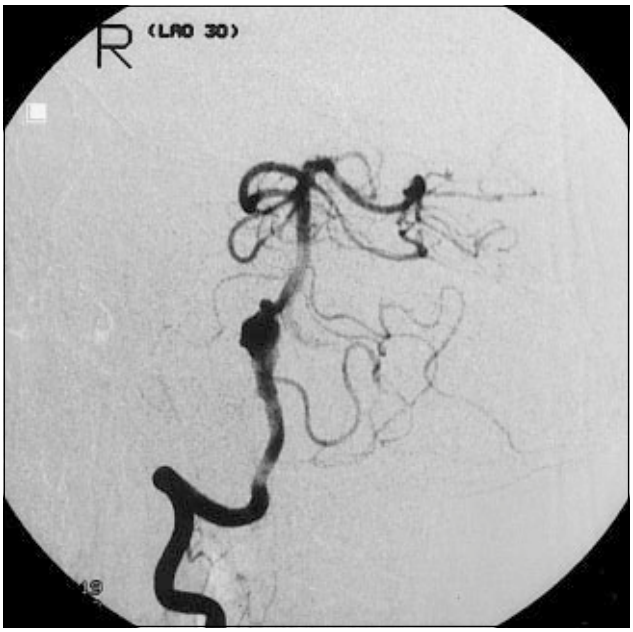


Fig 3. Digital subtraction angiogram of the right vertebral artery demonstrating a fusiform dissecting aneurysm distal to the posterior inferior cerebellar artery, but proximal to the vertebrobasilar junction

but conscious, and had no clinical features other than neck stiffness. A CT scan of the brain revealed diffuse SAH, which was prominent in the posterior fossa. A DSA revealed a right-sided intracranial fusiform vertebral artery aneurysm distal to the PICA, but not extending into the basilar artery (Fig 3). Endovascular occlusion of the aneurysm was performed using seven GDCs detached at the site of pathology, but sparing the PICA. Contralateral vertebral injection revealed good filling of the posterior circulation (Fig 4). The patient made a good recovery. Subsequently, follow-up angiography revealed persistent complete occlusion of the aneurysm.

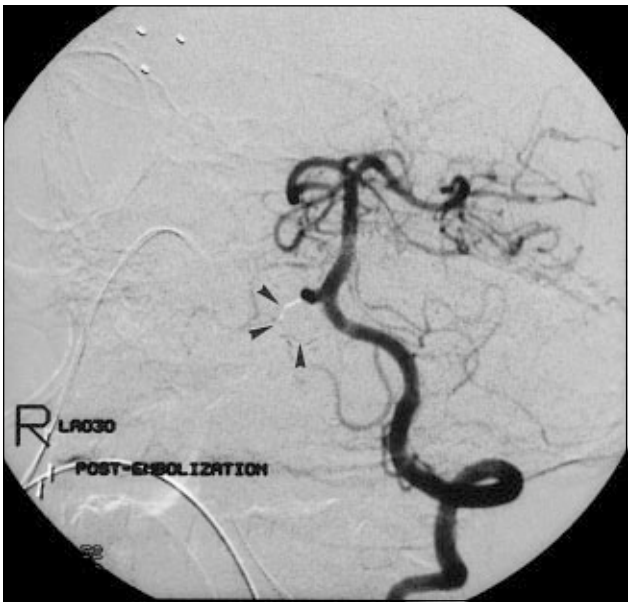


Fig 4. Digital subtraction angiogram of the left vertebral artery after embolisation, demonstrating the coil mass (arrow heads) and good filling of the posterior circulation

Case 8

A 50-year-old man presented with an SAH, localised on DSA to an intracranial dissection of the vertebral artery, just proximal to the PICA. Endovascular occlusion with seven GDCs was performed, sparing the PICA as demonstrated on contralateral vertebral artery angiography. This patient is still awaiting follow-up angiography.

Case 9

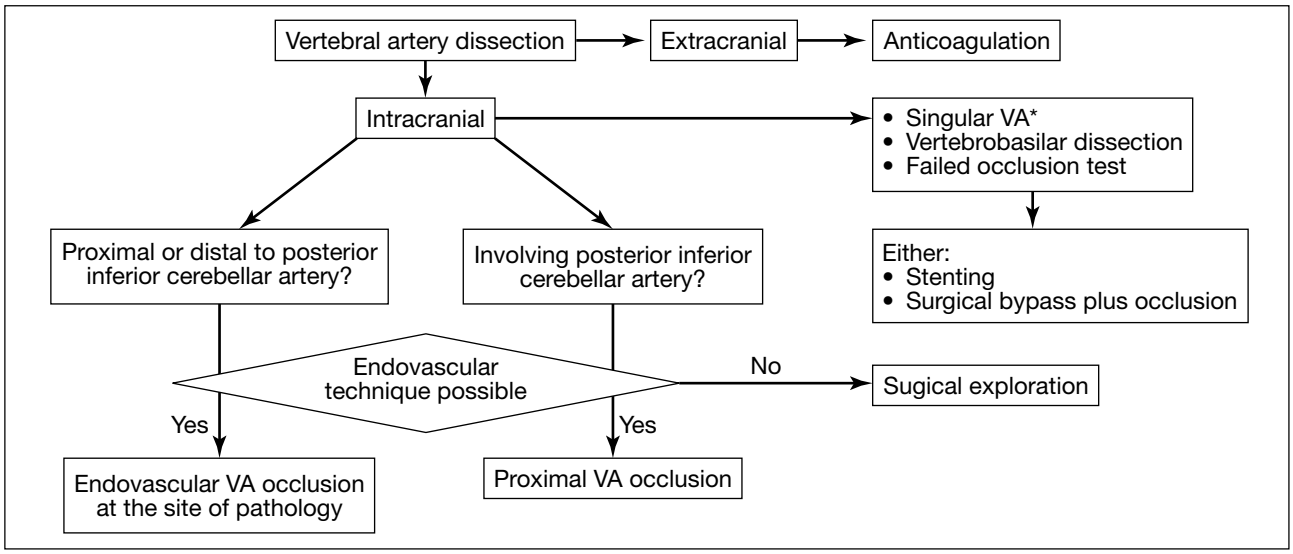
A 16-year-old girl presented with sudden onset of dizziness and vomiting. A CT scan revealed bilateral cerebellar infarcts. A DSA revealed a right-sided extracranial dissection of the vertebral artery at the C1 to C2 level, with absent filling of the peripheral right posterior cerebral artery, the right superior cerebellar artery, and the right PICA. The patient was commenced on anticoagulation therapy but due to clinical deterioration and cerebellar swelling, subsequently underwent posterior fossa decompression and infarct resection. She made a slow but steady recovery following surgery, and currently is moderately disabled but independent for daily activities. After 3 months of anticoagulation therapy, a repeat DSA revealed thrombosis of the right vertebral artery, but a completely normal posterior circulation following left vertebral artery injection. Anticoagulation therapy was subsequently discontinued.

Discussion

Vertebral artery dissections may occur in extracranial and intracranial segments of the vertebral artery.[1] Extracranial VADs are far more common than their intracranial counterparts and generally have a good prognosis.[2-4] Patients with ECVADs often present with neck pain and occipital headache.[1,2,4,5] If neurological symptoms and signs do not accompany these features, a dissection is often not suspected as a possible cause of the pain. The patient may subsequently experience the complication of vertebrobasilar ischaemia, which may occasionally be the presenting feature of a dissection. Sporting activities, chiropractic manoeuvres, and neck injuries were considered linked to the dissection in 53% of patients in one series.[2] Vertebral artery dissections may be bilateral in several patients, with a reported incidence of between 30% and 60%.[6]

Although ultrasonic methods can be used to diagnose VADs,[1] MRI accompanied by an MRA, and confirmed by a DSA, is usually required to establish the diagnosis. On MRI of the neck and posterior fossa, a T1-weighted image may show an increased diameter of the artery and a hyperintense signal surrounding a narrowed arterial lumen.[2,3,5,6] An MRA may show a narrowed lumen in the pathological segment. In 1999, Hosoya et al[6] reported the use of three-dimensional spoiled gradient recalled acquisition images as a useful tool in diagnosing VADs.

Magnetic resonance imaging and MRA are currently preferred non-invasive tools for establishing the diagnosis of VADs, but conventional angiographic techniques remain



* VA vertebral artery

Fig 5. Suggested algorithm for the management of vertebrobasilar dissections

the gold standard. A DSA may demonstrate several abnormalities, including one or more of the following: segmental arterial narrowing (string sign), segmental dilation (pearl sign), frank aneurysmal dilation, a double lumen (intimal flap), or a tapered occlusion.[2,3,5,6]

The treatment of ECVADs has, to the best of our knowledge, not been evaluated in a randomised controlled trial.[4] A proposed algorithm for treatment is found in Fig 5. Most authors recommend some form of anticoagulation for up to 3 months, or longer if follow-up radiological investigations reveal incomplete resolution of the disease process.[3] Suggested treatments are anticoagulation with heparin and warfarin, anticoagulation with a low molecular weight heparin, or antiplatelet therapy with agents such as aspirin or clopidogrel.

Intracranial VADs (ICVADs) can occur from the confluence of the vertebral arteries (ie where they join to form the basilar artery) to the entry of the cervical vertebral artery through the dura mater at the level of the foramen magnum. In some cases, dissections may extend distally to involve the basilar artery itself. In patients with ICVADs, the pathological process may extend into the subadventitia and lead to rupture of the vessel into the subarachnoid space and SAH. Intracranial VADs are much less common than their extracranial counterparts and, because of the risk of rebleeding, have a poorer prognosis.

The natural history of ICVADs has been described to some extent. Berger and Wilson[7] reviewed the literature published on these lesions between 1924 and 1983 and found 36 case reports. Thirty (83%) patients died within weeks of presentation. In patients who present with SAH, early rebleeding is common. Yamaura et al[8] described a sudden deterioration in seven of 21 patients, five of whom had had a second SAH. This risk of rebleeding has been confirmed by several other authors.[9-11] In a series of 17 patients with unruptured ICVADs monitored with serial angiography during conservative treatment, most cases resolved without the need for surgical intervention, however.[12] One patient underwent proximal artery occlusion (PAO) after follow-up angiography demonstrated aneurysmal enlargement.

Before the advent of endovascular treatment modalities, direct surgical approaches were the mainstay of management for ICVADs. The surgical procedures described have varied from PAO or trapping to procedures that try to strengthen or reinforce the vessel wall (eg base clipping or wrapping). A pertinent consideration is that if the dissection or aneurysm is not completely excluded from the circulation, the patient may experience a recurrent haemorrhage. Three important case series have been reported involving a total of 57 patients treated for ICVADS.[8,9,13] Patients treated with surgical procedures mostly underwent PAO, but several other procedures such as trapping or wrapping were performed.

Surgical trapping of ICVADs allows complete isolation of the diseased segment of the artery from the rest of the circulation by placing a vascular clip proximal and distal to the pathological process, thus removing the risk of a second haemorrhage. Trapping of ICVADs can also effectively be achieved by endovascular techniques. Initially, literature reports about the endovascular treatment of VADs focused on PAO.

Tsukahara et al[14] reported five cases of ICVADs managed with endovascular PAO. At follow-up angiography, four of five patients had residual aneurysms, although none had bled during the follow-up period. Halbach et al[15] reported a series of patients with ECVADs or ICVADs successfully treated by endovascular occlusion at, or just proximal to, the dissection site. One patient underwent a vertebral artery 'saving' procedure, with occlusion of the dissected lumen only.

Occlusion of the entire dissected segment of the vertebral artery is associated with the lowest probability of a second haemorrhage from the dissection. In contrast, after PAO, a second haemorrhage may occur from a more distal site because of backfilling from the contralateral vertebral artery. Yamaura et al[16] reported a series of six patients with complete occlusion achieved using GDCs at the site of pathology. No incidents of rebleeding occurred after treatment. More recently, Kurata et al[17] reported 24 patients, 18 of whom underwent endovascular coil placement at the site of pathology. Six patients did not undergo embolisation, and five of these died of repeat haemorrhage. Dissection of the vertebral artery may extend into the basilar artery. This is a difficult management problem, as a basilar artery dissection cannot be dealt with adequately by unilateral vertebral PAO. Moreover, surgery in this anatomical region is technically demanding and is often limited to supporting the diseased vessel with wrapping. If the patient can tolerate an occlusion test of the proximal basilar artery, thus relying on the posterior communicating arteries to perfuse the posterior circulation, permanent basilar PAO or bilateral vertebral artery occlusion may be performed. Basilar PAO can be performed surgically, as described by Amin-Hanjani et al,[18] or by endovascular techniques. Bilateral vertebral artery occlusion can easily be achieved using endovascular techniques.[19]

Using a surgical procedure to achieve an occlusion has the theoretical advantage of allowing visualisation of small perforators originating from the vertebral and basilar arteries; such perforators can then be carefully preserved. Damage to, or occlusion of, these perforators may cause significant brainstem injury. Using endovascular techniques rather than PAO to achieve an occlusion requires a longer segment of artery. This could lead potentially to occlusion of perforators, although in reality, the dissection has probably already achieved this effect.

If a patient is unable to tolerate PAO, for whatever reason, an artery 'saving' procedure is required, and was attempted in the current patient with a vertebrobasilar dissection by stenting. The use of intravascular stents in the intracranial posterior circulation is not yet common practice but has been reported. Higashida et al[20] reported the use of a stent, combined with endovascular coils, to successfully occlude a ruptured fusiform basilar artery aneurysm. Lylyk et al[21] recently reported nine cases of fusiform and dissecting vertebral artery aneurysms successfully managed with endovascular stents plus detachable coils. The stents commonly used in the treatment of coronary artery disease appear suitable for intracranial vessels. Occasionally, dissecting aneurysms can be occluded with detachable coils, as in the treatment of narrow-necked, saccular aneurysms, by detaching coils into the dissected segment, thus preserving flow through the parent artery.[15]

Dissection of the PICA represents a unique problem, and several cases have been reported in the literature.[22,23] Clinically, SAH or brainstem ischaemia are common features. Radiologically, the dissection is often found in the proximal segment of the PICA. Again, endovascular occlusion or surgical trapping of the diseased segment should be considered in patients presenting with ischaemia, and is mandatory in patients presenting with SAH. Balloon occlusion of the PICA has the advantage of indicating whether the artery can be permanently occluded, although this procedure may be difficult to perform. If the patient fails an occlusion test, some form of bypass procedure will be required before occlusion of the PICA. Such a bypass may involve an occipital artery-to-PICA anastomosis, or a PICA-to-PICA anastomosis.[22]

Lastly, the relationship between the PICA and the pathological segment of the vertebral artery is pertinent. Kitanaka et al[13] reported that of 25 VADs, two were proximal to the PICA, three involved the PICA, 16 were distal to the PICA, and in four cases, the PICA could not be identified radiologically. Patients in whom the PICA is involved in the dissection process pose a difficult problem. Sacrifice of the PICA could result in brainstem and cerebellar ischaemia. Yasui et al[23] reported 10 cases in which various techniques were used to circumvent this problem. These techniques ranged from PAO only, using micro-flow Doppler to establish backflow into the PICA, to technically demanding procedures such as PICA-bypass surgery, and PICA re-implantation.[24]

Conclusion

Vertebral artery dissections are increasingly being identified as the cause of vertebrobasilar ischaemia or SAH. These dissections are most readily diagnosed by having a high index of suspicion in patients with the relevant clinical signs and by appropriate investigation with MRI, an MRA, and/or a DSA. Extracranial VADs should be treated with anticoagulation. Intracranial VADs should not be treated with anticoagulants but managed by occlusion or stenting of the diseased segment of the artery. Various surgical and endovascular techniques are available. Ultimately, each patient requires a management plan taking into consideration co-morbidity, anatomical location of the pathology, and the techniques and expertise available in the institution concerned.

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