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Clinical Article

Management of spontaneous haemorrhagic intracranial vertebrobasilar dissection: review of 21 consecutive cases

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Summary

Introduction. Haemorrhagic intracranial vertebrobasilar dissection is an uncommon cause of nontraumatic subarachnoid haemorrhage (SAH) and accounts for only 1–10% of non-traumatic SAH. Treatment in the acute phase is considered to be essential because of the high risk of rebleeding and the consequent unfavourable outcome. However, the location, the potential for involvement of eloquent vessels and the histopathological characteristics of the vessel wall make treatment demanding from both a technical and anatomical point of view. We report our experience in the management of this disease.

Patients and treatments. From 1989 to June 2006, we managed 21 patients with spontaneous haemorrhagic dissection located in the intracranial vertebrobasilar system, 13 patients were treated using an endovascular approach, 1 by surgical clipping, and 7 were managed conservatively.

Results. Among the 13 patients treated endovascularly, 7 underwent proximal occlusion, 4 underwent parent artery embolization at the site of dissection, and 2 underwent endovascular trapping. Severe, treatment-related complications due to dislodgement of the thrombus during the procedure occurred in 1 patient, who then died from brainstem ischaemia. One patient died from severe pneumonia and one patient was left disabled from vasospastic ischaemia resulting from severe initial SAH. The remaining 10 patients had satisfactory outcomes: none rebled after treatment and when discharged they had Karnovsky scores of 80–100.

Of the 7 conservatively treated patients, three died of rebleeding and four were discharged with Karnovsky scores of 50–100. One patient, who was treated surgically, was discharged with a Karnovsky of 90.

Conclusion. The high rate of rebleeding and consequent mortality among the patients treated conservatively argues for treatment in the acute phase. Treatment should be guided by each patient's angiomorphology, clinical condition and the experience of the neurosurgical/neuroradiological team. Options include endovascular or surgical trapping of the dissection and proximal occlusion and embolisation of the parent artery at the site of the dissection.

Keywords: Vertebro-basilary dissection; SAH; proximal occlusion; embolisation.

Introduction

Spontaneous haemorrhagic intracranial dissection is an uncommon disease. Since Yonas *et al*. [39] described the pathological and radiographic features of intracranial dissecting aneurysms as a cause for subarachnoid haemorrhage (SAH), it has been increasingly recognised as a cause of SAH with an unfavourable prognosis and a

high rate of rebleeding. Some 1–10% of all intracranial non-traumatic SAH are caused by ruptured intracranial dissection, and in children, the rate may be even higher [20].

The majority of haemorrhagic intracranial arterial dissection is located in the posterior circulation most likely reflecting it's structure since histological studies [30] have shown that the intradural vertebral artery has a thin media and adventitia with fewer elastic fibers, so dissections of the intradural vertebral artery are prone to result in SAH, in contrast to dissections of other vessels [2, 29]. Methods of treatment include conservative management, endovascular exclusion of the lesion or surgical intervention.

Conservative treatment is generally not the first choice for acutely ruptured dissections if other options are feasible. Endovascular treatments comprise of parent artery occlusion, proximal arterial occlusion, endovascular trapping, and stent implantation (with or without additional coiling). Surgical options for acute haemorrhagic intracranial dissection include trapping, Hunterian ligation, bleb clipping, and wrapping.

Between 1989 and June 2006, 21 patients with spontaneous haemorrhagic intracranial vertebrobasilar dissection were treated in our institution. Those included in this series had a spontaneous haemorrhagic intracranial vertebrobasilar dissection. Dissections located in peripheral vessels of the posterior fossa, such as posteroinferior cerebellar artery (PICA) were not included nor were dissections presenting with ischaemic embolic events. We also excluded traumatic vertebrobasilar dissection, posterior circulation dissection located on other peripheral arteries, PCA (posterior cerebral artery), and partially thrombosed aneurysms (classified as sub-acute or chronic dissections) [16] with or without haemorrhage, because their natural history and treatment strategy varies significantly from acute haemorrhagic dissections. We realise that some vertebral-PICA origin aneurysms with berry appearance may have corresponded to true dissection processes yet these where treated with intra luminal coiling like all other bifurcation berry aneurysms in other locations and were therefore also not included in this series.

The treatment strategies used, in relation to anatomy and clinical results, are reported and discussed in the light of the pertinent literature.

Patients and methods

From 1989 to June 2006, 505 patients with a ruptured intradural aneurysm were entered into the Neurovascular Data Bank of Bicêtre, of these, 68 (13.5%) had a lesion in the posterior fossa. Twenty-one patients with a ruptured dissection in the intracranial vertebrobasilar trunk, including the upper basilar artery, (4% or 31%) are reported. Subarachnoid haemorrhage (SAH) was verified by CT scan and/or lumbar puncture.

This retrospective series included 11 male and 10 female patients, whose age ranged from 6 to 67 years (mean 42.9). Presenting symptoms were headaches, disturbance in consciousness, convulsion, and cranial nerves palsy. In 9 patients, headaches preceded a sudden deterioration of the clinical state by 13 h to 4 days. Four patients experienced cardiopulmonary arrest after their clinical deterioration. Three patients were admitted in spring, 8 in summer, 4 in autumn, and 2 in winter.

Dissection with systemic disease was included in this series; the diagnosis of which was based on relevant familial and personal history. There was a history of hypertension in 4 patients (patients 10, 16, 18 and 20), spontaneous hematuria in 1 (patient 2), and other patients had familial immune deficiency with fungal infection (patient 5), lupus erythematodes and chronic phlebitis (patient 16, for whom over-anticoagulation was established by laboratory examination after haemorrhage), neurofibromatosis type II (patient 17), and lower limb venous varices (patient 7). One patient had a family history of spontaneous SAH without evidence of an associated aneurysm.

The patient's condition on admission was evaluated according to the Glasgow coma scale (GCS) and the Hunt-Hess (H&H) grading system, as set out in Table 1.

All patients underwent 4-vessel digital subtraction angiography. The diagnosis of dissection was based on angiographic signs, i.e. fusiform dilatation, the ''pearl-and-string''-sign, ectasias distant to an arterial bifurcation, stenosis with a ring-like appearance proximal to a fusiform ectasia, and irregular luminal stenosis.

Endovascular treatment was chosen as the first option and performed depending on the patient's status, location of the dissection, anatomy of related vessels, and the possible collateral circulation. Endovascular treatment was performed in all patients directly after the diagnostic angiography was carried out, i.e. in most instances in the acute phase. The intervention was performed under general anaesthesia, a balloon occlusion test occlusion with angiographic analysis was performed when needed. Evaluation of the collateral circulation was based on meticulous arterial and venous anatomic analysis under general anesthesia rather than relying on

Table 1. *Features of 21 patients with haemorrhagic vertebro-basilar dissection, management and outcome*

Patient number	Age/ sex	Relevant history	Presenting symptoms	GCS HHG	Location of dissection	Angiographic features	Management	Results (Karnovsky) time (months)
1	39/F	EDS?	SDHACD convulsion	5 4	V4, distal to PICA, bilateral ICAs	pearl and string	proximal occlusion	90 12
2	59/M	–	headache CPA	10 3	V4, no evident PICA	fusiform	PAO	40 0.5
3	36/M	–		15 2	V4, distal to PICA	fusiform	conservative treatment	90 90
4	6/M	CCMCI	headache vomit	15 2	from bilateral V4 to whole BA, ending in right P1, left ICA	fusiform	conservative treatment	die of rupture of left MCA dissection months later
5	15/M	–	SDHACD convulsion	15 2	upper 1/3 of BA	fusiform	proximal occlusion	30 9
6	58/F	LEVV	SDHACD hemiplegia dysarthria	15 2	middle and upper 1/3 junction of BA	fusiform	PAO	100 4
7	56/M	–	SDHACD CPA	5 4	V4, distal to PICA	fusiform	conservative treatment	die of rebleeding
8	56/F	FHSAH	headache	7 3	V4, distal to PICA	fusiform	proximal occlusion	die of pulmonary infection
9	38/F	–	SDHACD photophobia	15 2	V4, PICA involved	stenosis	proximal occlusion	90 22
10	46/F	H&O	convulsion	14 2	V4, PICA involved	fusiform	proximal occlusion	90 1.5
11	64/M	–	SDHACD	15 2	V4, PICA involved	fusiform	conservative treatment	100 7
12	46/M	–	SDHACD convulsion	6 4	V4, distal to PICA	pearl and string sign	PAO	die of complication
13	42/M	–	headache	6 4	V4, PICA involved	fusiform	conservative treatment	die of rebleeding
14	15/F	–		10 3	lower 1/3 of BA	pearl and string sign	surgical clipping	90 4
15	40/F	LE	headache hemiplegia	12 3	left V4, proximal to PICA. Right V3 dissection	fusiform	conservative treatment	good condition when discharged. No follow up
16	43/M	H&O	headache	15 1	middle and lower 1/3 of BA	fusiform	conservative treatment	50 41
17	15/F	NFII	hemiparesis dysphagia CPA	15 3	middle and upper 1/3 of BA	fusiform	PAO	back to school dysphagia improved
18	67/M	H&O	headache	14 2	V4, PICA involved	fusiform	proximal occlusion	100 18
19	53/F	FMD	headache 1 day	15 1	distal to PICA	fusiform	trapping with fibered coils	100 14
20	60/M	H&O	headache	15 1	distal to PICA extracranial origin of PICA	fusiform	proximal occlusion with fibered coils	90 12
21	47/F	FMD	headache vertigo	14 2	distal to PICA	pearl and string sign	trapping of the dissection with fibered coils	80 12

EDS Ehlers–Danlos syndrome, *LEVE* lower extremity venous varix, *H&O* hypertension and obesity, *SDHACD* sudden deterioration of headache after certain duration, *NFII* neurofibromatosis type II, *PAO* parent artery occlusion, *CCMCI* chronic cutaneous mucosal candidiosis infection, *CPA* cardiopulmonary arrest, *FHSAH* family history of SAH, *LE* lupus erythematosus, *FMD* fibromuscular displasia.

patient's cooperation in the acute phase of SAH, where pictures are often of a poorer quality.

Detachable balloons (GVB 16, Alpine Scientific company) and bare or fibered electrolytically detachable coils (GDC, Boston Scientific, Fremont, USA) were used for arterial occlusion. When the dissection had been secured by endovascular embolisation, standard management of the SAH was carried on. Heparinisation was

continued for 1–2 days depending on the risk of ischemia in relation to clot extension distal to the occluded segment and the need to perform ventricular shunting. Aspirin was administrated following intervention and until the first angiographic control study 3 months later.

Results

Location and angiographic finding

Among 21 vertebrobasilar system dissections, 16 displayed regular or irregular fusiform dilation, 4 had a ''pearl-and-string'' sign, and one patient showed only a stenosis.

The basilar artery was involved in six patients: in one of these the dissection extended from both vertebral arteries to the basilar artery, in the other five patients only the basilar artery was involved.

In vertebral artery dissections without basilar involvement, the dissection was proximal to the posterior inferior cerebellar artery in 1 of 15 patients, distal to it in eight and in the other five the dissection involved the origin of the PICA. In one patient, the posterior inferior cerebellar artery could not be identified. The angiographic signs and the location of the dissection are listed in Table 1. Vasospasm was demonstrated (remote from the dissected area) in 4 of the 21 patients.

Conservative management

Seven patients were treated conservatively. Among these, 3 patients (patients 3, 4, and 16) were referred to our unit in the postacute stage (3–6 weeks after the haemorrhage occurred), conservative treatment was therefore elected. Each of these three patients was in a good clinical state (GCS 15, H&H 1 or 2) and was

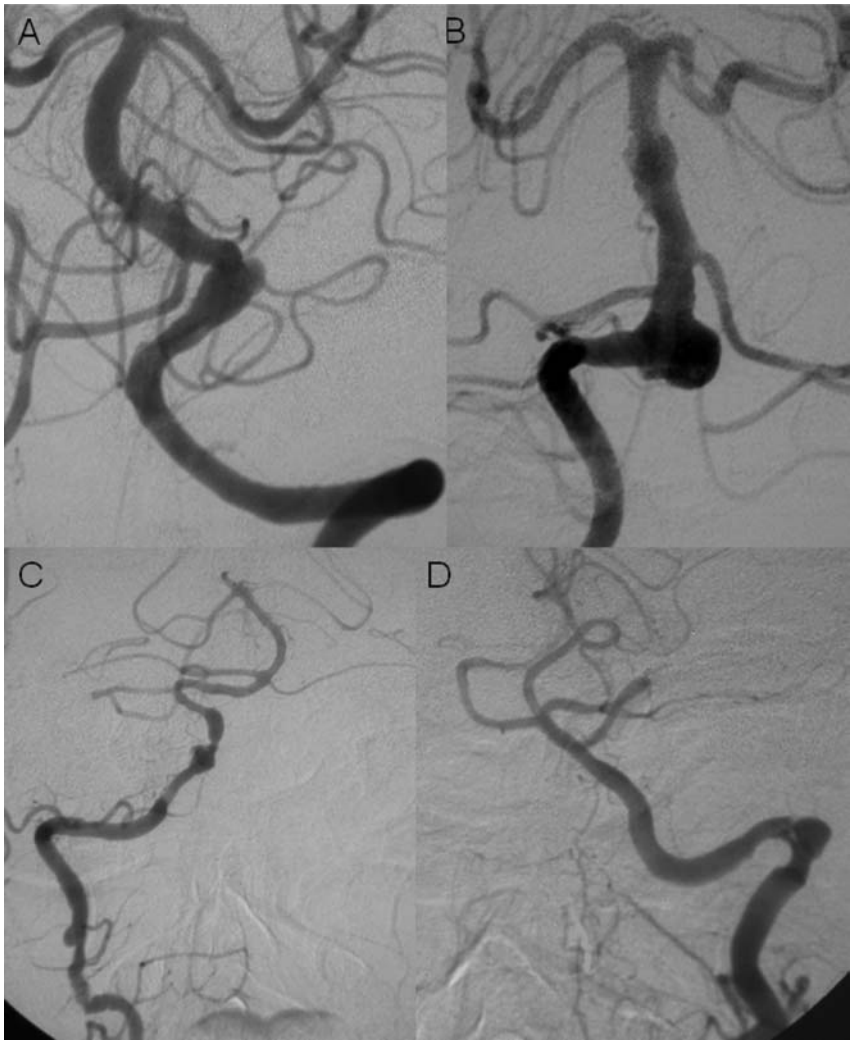


Fig. 1. Representative angiograms of three different patients showing dissections that developed on either a normal artery (*frame A*), or on an impaired arterial segment (*frame B*), or on pathological vessels (*frames C and D*). *Frame A*: Patient 8. A–P projection showed a vertebral dissection that developed on an ostensibly normal vessel. *Frame B*: Patient 16. A–P projection showed a basilar artery dissection that developed on a segmental dysplastic basilar artery. *Frames C and D*: Patient 15. Right (C) and left (D) vertebral artery injection demonstrated multiple and bilateral vertebral artery dissections located on pathological vessels. The patient had a history of lupus erythematosus

discharged from our unit for further follow-up. One patient (patient 4) died 16 months after the initial event following dissection of a different vessel, the middle cerebral artery. Postmortem histology of the intracranial vessels was obtained and showed a thickened vessel wall without inflammatory infiltrations. The elastic lamina had been destroyed and only rare elastic fragments remained. Follow-up of the remaining two patients showed in one (patient 3) stable morphology after 7.5 years, and slight asymptomatic enlargement of the occluded vertebral artery distal to the origin of the posterior inferior cerebellar branch at 39 months in the other (patient 16).

In two of the seven patients treated conservatively, endovascular or surgical treatments were deemed not feasible: Both (patients 11 and 13) had a dissecting aneurysm in their vertebral artery involving the origin of a dominant posterior inferior cerebellar artery supplying the brain stem. In one patient, the segment distal to the origin of the posterior inferior cerebellar artery was occluded, while in the other, the severity of the SAH and concomitant vasospasm was considered very liable to compromise severely the collateral capabilities. Occlusion of the vertebral artery would have put each patient at considerable risk of major ischemia of the posterior fossa and was therefore not performed. One of these two patients, initially in a good clinical state, had made a complete recovery to normal life at 7 months follow-up. The other patient who was initially in a poor clinical state (GCS 6, H&H 4), rebled on the seventh day following the initial event and died.

The remaining 2 of these 7 patients were treated conservatively for the following reasons: One (patient 7) was in a poor clinical status (GCS 5 with persistent bradycardia), and was therefore not treated as an emergency. He rebled during his admission and died. The other patient (patient 15), in a good clinical state on admission (GCS 15, H&H 1) had an associated iatrogenic over-anticoagulation, given for lupus erythematosis; medical correction was the chosen treatment, and she was discharged after 3 weeks in a good clinical condition and subsequently lost to follow-up.

Endovascular treatment

Endovascular treatment for intradural dissection

Endovascular treatment was used in 13 patients. In 11, occlusion of a parent artery was performed either at the origin of the dissected segment (4 patients) or proximally in the vertebral artery (7 patients) and in 2 endovascular trapping was achieved. No stent was used in our series. The treatment choices are summarized in Table 2.

Ten intradural vertebral dissections were treated endovascularly. Eight were distal to the origin of the posterior inferior cerebellar artery, six of these eight patients were treated with occlusion of the parent artery while endovascular trapping was performed in the remaining two.

Each of the two patients treated by endovascular trapping of the dissection was in a good clinical condition (patient 19, GCS 15, HH 1, patient 21 GCS 14 and H&H 2) each made an excellent recovery and went back to normal life (follow-up: 14 and 12 months).

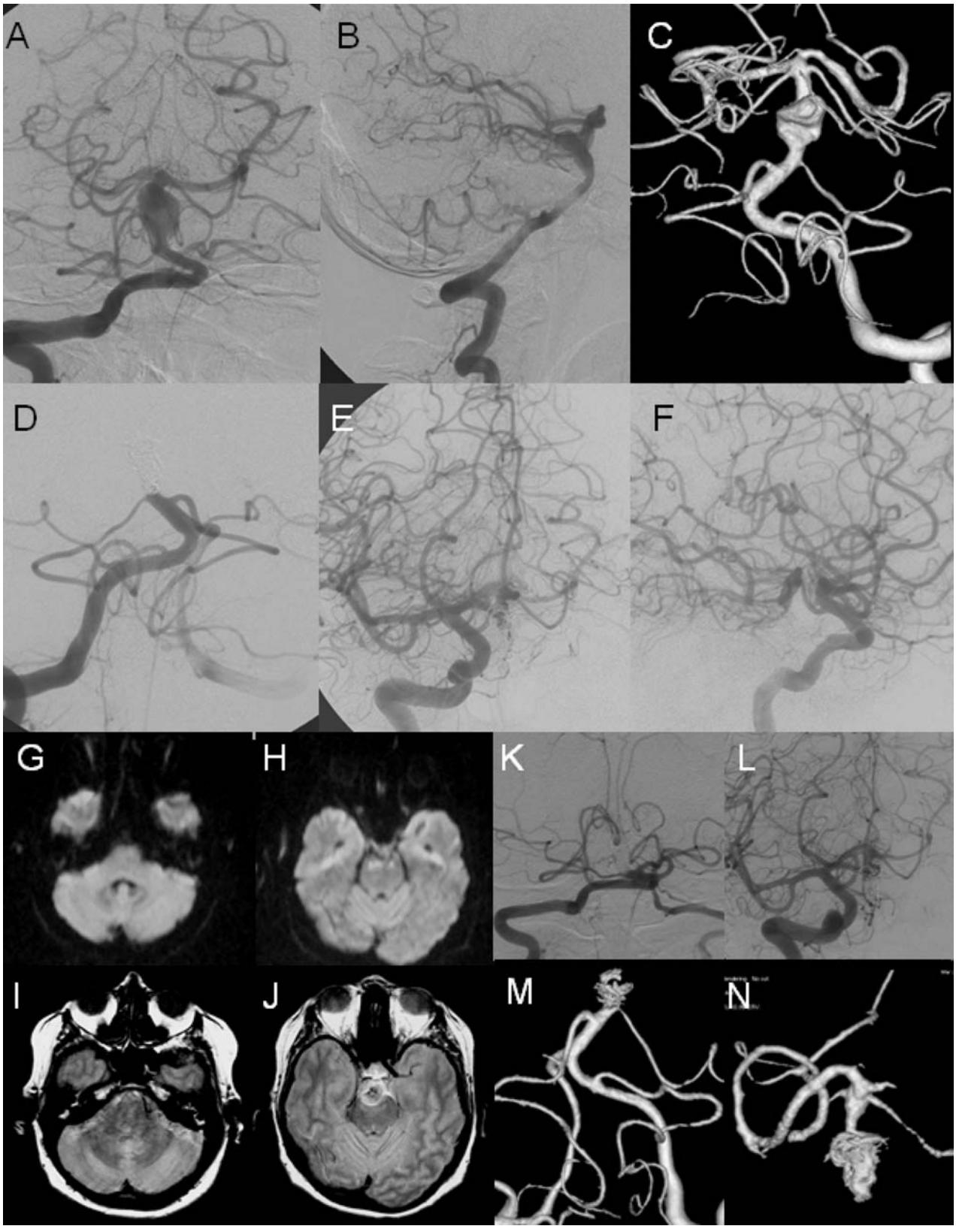
In one patient (patient 12, GCS 6, HH 4), treated with parent artery occlusion thrombembolic complication occurred during the intervention and the patient died from brainstem ischemia. One additional patient (patient 8) died from severe pneumonia. One patient in whom the posterior inferior cerebellar artery could not be demonstrated and who had associated severe vasospasm (patient 2, GCS 10, H&H 3) experienced multiple infarctions caused by the vasospasm resulting from his initial bleed. He was discharged from the hospital with a Karnovsky score of 40.

The remaining patients had returned to their normal activity when followed up 12 months following the initial event, one of whom had been in a poor clinical state on admission (patient #1 GCS 5, H&H 4). Follow up angiography at 3 months showed that the posterior inferior cerebellar artery was patent in each of the two remaining patients (patients 1 and 20). One (patient 20) in

Table 2. *Location of dissection and management*

	Basilar artery	Proximal to PICA	PICA origin involved	Distal to PICA origin	No evident PICA
Proximal occlusion	1		3	3	0
Parent artery embolisation	2	0	0	1	1
Endovascular trapping	0	0	0	2	0
Surgical clipping	1	0	0	0	0
Conservative management	2 (1 with bilateral VA dissection)	1	2	2	0

PICA Posterior inferior cerebellar artery.


A
B
C
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F
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H
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L
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J
M
N

whom occlusion had been performed proximal to the origin of the posterior inferior cerebellar artery, showed complete remodeling of the dissected segment.

In 3 patients, the dissection involved the origin of the posterior inferior cerebellar artery (patients 9, 10 and 18). Each was treated by proximal occlusion alone and they had a good recovery (Karnovsky score 100, patient 18) and 90 (normal activity, minor signs, patients 9 and 10) on follow-up 1.5–22 months after their initial event. Follow-up angiography showed that their posterior inferior cerebellar arteries were patent and that the dissected vessel had remodeled to a normal vessel.

Endovascular treatment for dissection of the basilar artery

In three patients treated endovascularly, the dissection was confined to the basilar artery. One (patient 5) with a fusiform dissection involving the upper 1/3 of the basilar artery was first treated with pseudoaneurysm embolisation in another unit. The patient was transferred to our hospital and the dissection reruptured during diagnostic angiography. Immediate proximal occlusion of both vertebral arteries had to be performed. On clinical follow-up 9 months later, the patient was severely disabled with multiple brain stem infarctions. The other two patients (patients 6 and 17) had a fusiform dissection in the junction of the upper and middle 1/3 of the basilar artery. Each dissection was treated by occlusion of the parent artery. Follow-up MRI in the next week showed thrombosis in the aneurysm in each patient; no parenchymal ischaemia was found. Angiographic follow-up after 4 months showed complete disappearance of the aneurysm in one patient who is symptom-free and back to work. The other patient returned to school with improved dysphagia 1 month after intervention.

Surgical treatment

One patient (patient 14) was treated surgically: a dissecting aneurysm in the lower third of the basilar artery involving the origin of right dominant AICA could not be treated via an endovascular approach, therefore, surgical bleb clipping was attempted. Angiography 10 days and 3 months after the operation showed exclusion of the bleb and stable fusiform appearance. Clinically the patient recovered completely.

Discussion

In this article we have described our experience in the management of patients with an acutely ruptured dissection in the vertebrobasilar system. As can be seen from the differing strategies, general guidelines for this disease cannot be given, and treatment has to be individually tailored to the patient's angiomorphology, their clinical status and the experience of the treating neuroradiological/neurosurgical team.

Epidemiology and pathomechanism

In this series, ruptured vertebrobasilar trunk dissection accounted for 4.2% of intracranial aneurysmal SAH and 30.9% of posterior fossa aneurysmal SAH. There was no gender dominance in this series.

Although syphilis, fibromuscular dysplasia, collagen disease, and trauma are known to be associated with dissection, the pathogenesis of most dissections is still unclear. It's worthy of mention that there was a spring-summer dominance of onset in this series. Recent infection has been reported to be a risk factor for SAH by contributing to the formation and rupture of aneurysms [18]. Furthermore, cervical dissections, which tend to cause ischemic events, were found to have autumnal predominance and a possible association with infection was proposed [32]. It is interesting to note the difference of susceptive season between ischemic and haemorrhagic dissections. In South Africa, haemorrhagic dissections are noted from late September to March (corresponding to Spring and Summer in the northern hemisphere) (A. Taylor personal communication).

Mizutani *et al*. [24] pointed out that the sudden disruption of the internal elastic lamina with subsequent

Fig. 2. Patient 6: A 58-year-old lady presented with sudden onset of headache which relieved spontaneously on the same day. However, headaches recurred after 2 days and the patient subsequently lost consciousness. Neurological examination showed a right hemiplegia and dysarthria. CT scan demonstrated diffuse SAH. *Frames A–C*: Angiography showed a fusiform aneurysm located at the junction between the upper and middle thirds of the basilar artery. The irregular posterior surface indicated intraluminal thrombosis anterior to the brain stem. No brain stem perforating vessels were opacified at the level of the dissection. *Frames D–F*: Angiography after embolisation showed preservation of both anterior inferior cerebellar arteries and adequate collateral circulation of the upper part of the basilar artery from the right internal carotid artery. The most distal portion of the dissection was still partially opacified. *Frames G–J*: MRI (DWI, *frames G and H*) and proton density images (I, J) five days after endovascular therapy showed thrombosis in the aneurysm, there were some patchy areas of parenchymal ischemia in the territory of the brain stem perforators. *Frames K–N*: An angiogram 4 months later showing total obliteration of the aneurysm and normal parenchymal opacification

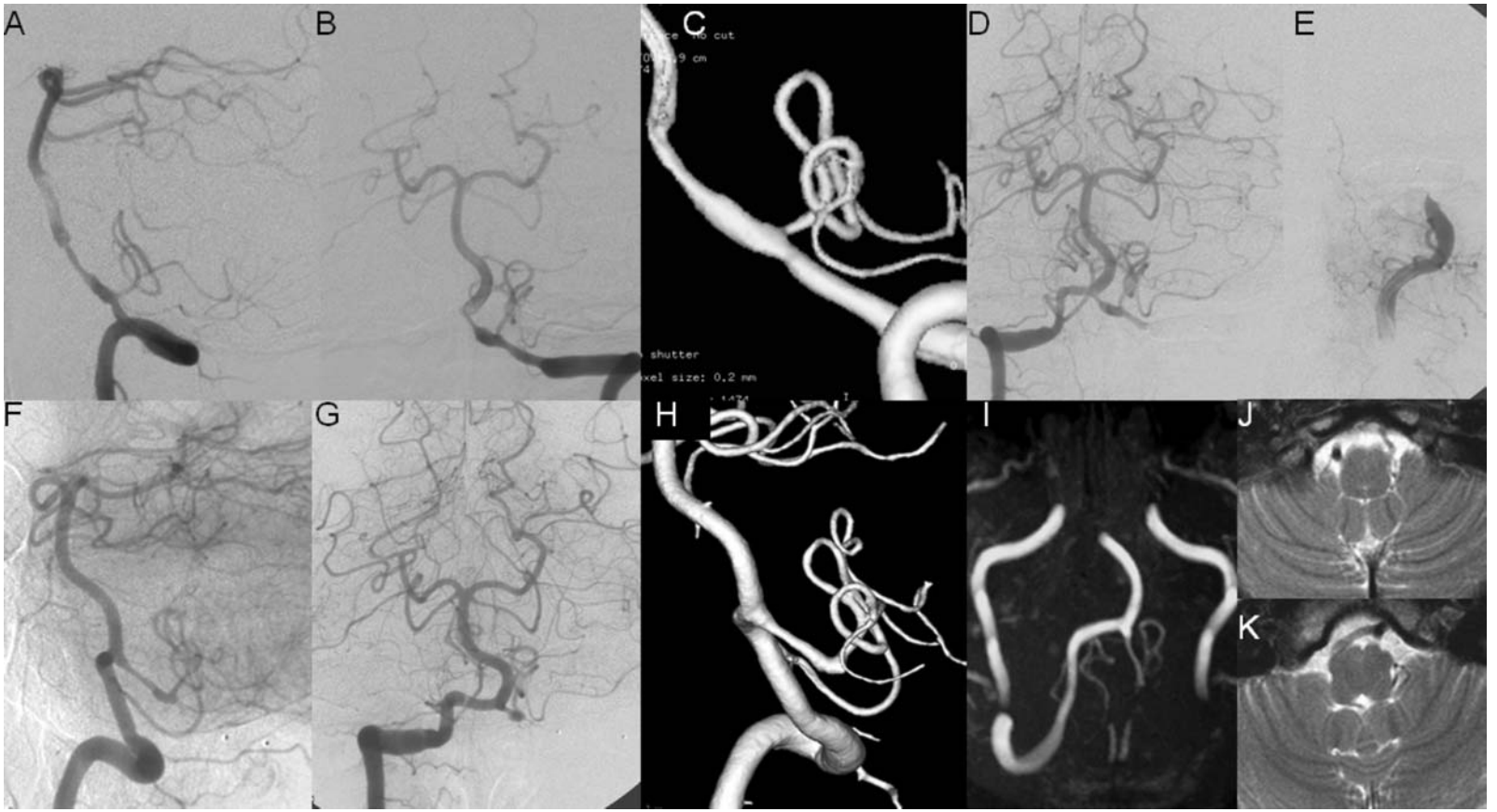

Fig. 3. Patient 9: A 38-year-old female presented with head and neck pain, which worsened over the course of two days and was accompanied by nausea and photophobia. Cranial CT showed SAH. *Frames A–C*: Angiography showed irregular stenosis in the left V4 segment of the codominant left vertebral artery with involvement of the origin of the left posterior inferior cerebellar artery. *Frames D–E*: Proximal occlusion was performed at the level of C1. Right vertebral artery injection after detachable balloon occlusion of the healthy vessel segment proximal to the dissection showing opacification of the left posterior inferior cerebellar artery through the contralateral vertebral artery. Although the dissection was still opacified, there was no recurrent bleeding. *Frames F–H*: Control angiography at 10 months demonstrating remodelling of the segment between the posterior inferior cerebellar artery and the vertebro-basilar junction while the caecum of the dissection is not opacified anymore. *Frames I–K*: MRI at 22 months showing a normal contour of the vessels and no abnormal signals either in the brain tissue or the vessel wall. The patient had a good clinical outcome, with a Karnovsky score of 90

penetration of circulating blood into the media is the primary mechanism underlying the development of cerebral dissecting aneurysms. The histopathological results of the single instance where an autopsy specimen was available in this series is in accord with this concept. Although we found no evidence of an inflammatory infiltrate in the vessel wall, the elastic lamina had been destroyed and only a few elastic fragments remained. All dissections included in this series led to transmural rupture and SAH. The most frequent angiographic finding in this series was of a regular or irregular fusiform dilatation. However, ruptured dissections may not have an obvious ''aneurysmal'' dilatation, and stenotic vessel segments can also lead to haemorrhagic events. That is why we used the term ''haemorrhagic dissection'' rather than ''haemorrhagic dissecting aneurysm'' in this manuscript.

Natural history

The choice of treatment and its timing continue to be controversial. Acutely ruptured dissections are unstable and have a tendency to rebleed. The rebleeding rate has been reported to be as high as 71.4% in a group of 42 untreated patients [24]. The mortality rate of these rebleeds was high, being 46.7% in this series. As a rule, the shorter the time since the initial haemorrhage, the higher the risk of rebleeding in the acute phase. In the study conducted by Mizutani, 70% of rebleeds occurred within the first 24 h after the initial SAH and 80% in the next week. Fortunately, as the time passes, the risk of rebleeding decreases considerably. Yamaura *et al.* [35] proposed that a ruptured dissecting aneurysm enters into a healing stage approximately 1 month after the initial SAH. In Mizutani's series, only 10% of rebleeding occurred more than one month after the initial haemorrhage. In their discussion of the histopathology of the healing response following dissection, Mizutani *et al*., proposed that vessel wall repair is completed after the neointima covers the entire area of the arterial wall. This repair occurs from the disrupted ends of the media toward the ruptured portion. As they pointed out, this healing mechanism may be delayed under several circumstances such when there is an extensive defect of the aneurysmal wall in the ruptured portion (i.e. large aneurysms), aneurysms with abundant thrombus in the ruptured portion (since neointima may appear along with retraction of the thrombus), or aneurysms in which the media is completely separated from the adventitia. This vessel wall reaction, however, seems to be unpredictable. In our institution, conservative management was adopted when the patient was referred to us 3 weeks after initial haemorrhage and there was no evidence of progression in the angiogram. None of these patients rebled.

In our series, patients in a poor clinical status tended to rebleed. This tendency has already been reported for ruptured saccular aneurysms [9, 33]. In a large series of 1076 cases of ruptured intracranial saccular aneurysm, Rosenorn found a significantly higher rate of rebleeding in patients in poor clinical condition when compared to those in a good clinical state [28]. The classical view is that late recurrence does not occur at the same site so that haemorrhagic dissection is an expression of an acute process in the vessel wall that can be followed by repair and spontaneous healing. Sometimes the damaged vessel wall heals spontaneously, at others the assistance of endovascular occlusion is needed.

Patients with a spontaneous dissection may have an underlying systemic vessel wall disease, and have multiple dissections [21, 41]. In this circumstance, increased haemodynamic stress following unilateral occlusion of a vertebral artery can result in the development of a new dissection [10, 17] although this was not encountered in our series.

Treatment options

The aim of treatment in the acute phase is to secure the patient from rehaemorrhage, balancing between the twin risks of bleeding and healing. Among the various surgical approaches referred to in the introduction, merely to wrap the dissected segment is believed to be insufficient but surgical clipping of the bleb or endovascular pseudoaneurysm embolisation risks disrupting the aneurysm during operation. Following this type of intervention, there is a continuing risk that the dissection will regrow or rerupture [1].

Endovascular occlusion of the parent artery

A saccular aneurysm will persist unless treated by surgical clipping or endovascular embolisation. In contrast, reflecting the intrinsic mechanism of healing, a dissection can resolve spontaneously [26]. The aims of treatment are, first, to reduce the ''hemodynamic stress'' on the vessel wall that could produce rerupture and, second, to provide a suitable environment for healing. In our experience, as reported here, both goals could be achieved by eliminating or reversing the flow within the dissection through sacrificing the parent artery close to or even far proximal to the dissection. If the dissection is not

completely excluded from the antegrade arterial circulation following proximal occlusion, the potential for rebleeding still exists [14, 38]. Rebleeding can be anticipated when the dissection cavity increases in size after proximal occlusion [3, 11]. Rebleeding from a ''caecum-like'' dissection usually occurs within several hours after proximal occlusion, presumably as result of haemodynamic changes in its lumen [37]. Despite these concerns, in our series, no patient who had been treated by occlusion (on site or proximal) of the parent vessel rebled subsequently; even if the full-length or the proximal part of the dissected lumen became a caecum after proximal occlusion, follow-up control angiography showed that the stump had thrombosed. In those patients where flow was still visible through the full-length or distal part of the dissected lumen after proximal occlusion, follow-up showed that the dissection had remodeled to a normal luminal structure, and no perforator ischemia was noted. Although recanalisation after occlusion of the parent artery has been reported [27, 31], this did not occur in our experience.

Endovascular trapping

Because histopathology shows that the rupture point is in close proximity to the entrance into the dissection [25], an option in treatment is double catheterization and simultaneous embolisation of the proximal and distal portions of the dissection [12]. Endovascular trapping does not cross the dissected segment (which should be avoided). However, ischemia may occur in the territory of brainstem perforators arising from the healthy vessel distal to the dissected portion that will be occluded. We do not think, therefore, that trapping is superior to occlusion of the proximal parent vessel. This is because the theoretical benefits (exclusion of the diseased vessel segment) are outweighed by the risk of occluding brain stem perforators in the occluded distal healthy vessel. We found that proximal occlusion alone prevented rebleeding in all patients.

Intracranial stenting

Stent implantation has the advantage of preserving the patency of the parent vessel and remodeling blood flow [8]. Nevertheless, there are unnecessary difficulties and hazards. These are that the stent has to be navigated through the dissection, inducing the risk of further dissecting the wall, that uncovered stents that do not fully exclude the dissection from the circulation may lead to early rebleeding, and that stent-assisted coiling of the dissection (as well as coiling of the dissection itself) might cause rupture of the dissection or lead to recanalisation if the coil migrates through the dissected vessel wall, so not completely securing the situation [22]. Finally, the use of stent grafts in the intracranial circulation is experimental [15], and likely to occlude perforators to the brain stem and even may induce a secondarily symptomatic excessive neointimal proliferation. This technique was not considered in this group of patients.

Morbidity of endovascular techniques

Of the 21 patients we report, 2 died and 1 was left with disability. This is consistent with 2 other, larger series with mortalities of 13.8–28.6% and morbidities of 17.2–19.0% reported by Yuki and Anxionnat [2, 40].

The main factors contributing to disability and death are a poor clinical state at admission and severe vasospasm caused by the initial SAH. The results favour the use of endovascular treatment as the first option for haemorrhagic vertebrobasilar dissection.

Ischemia induced by embolisation is a major concern. Anatomical research [23] has shown that perforators can arise from the distal vertebral artery, especially on the nondominant side and from the vertebrobasilar artery at a site approximately 14 mm proximal and 16 mm distal to the vertebrobasilar junction. As a rule, the more distal the origin of the posterior inferior cerebellar artery, the more likely it is to give rise to perforators [19, 7]. The length of occlusion should, therefore, be as short as possible. This might be achieved more easily with the use of detachable fibered coils. When thrombosis occurs in the abnormal segment of the vessel, it is highly likely that many of the perforators involved have already been occluded by thrombus at the time of diagnosis [34, 5].

Extensive thrombosis with subsequent occlusion of perforators is another possible complication [6, 2], but this did not occur in our experience.

During occlusion of a proximal artery, coil protrusion into the dissected vessel segment should be avoided in order to minimize the risk of aneurysm rupture. Another concern is migration of thrombus from the proximal vessel segment during coiling. In our experience, proper heparinisation is usually sufficient to avoid this complication, nevertheless, it occurred in one patient with a poor initial grade. As when treating a berry aneurysm, we start heparinisation after the first coil is placed and continue anticoagulation for 1–2 days followed by aspirin for 3 months until angiography has been repeated.

Table 3. *Summary of findings in larger reports of the management of vertebrobasilar dissection*

Report	Number of cases	Technique adopted	Technique-related complication in % (number of patients)	Overall morbidity in % (number of patients)	Overall mortality in % (number of patients)
Chifumi *et al.* [4]	24	surgery	LCNP: 76.5 (13/17) LTS: 26.3 (5/19) RC: 21.1 (4/19)	76.5 (transient) 26.7 (persistent)	13.3 (2/15 according to available data)
Yuki *et al.* [40]	29	endovascular	3.4 (1/29)	13.8 (4/29)	17.2 (5/29)
Anxionnat *et al.* [2]	21	endovascular	8.3 (1/12)	28.6 (6/21)	19.0 (4/21)
Present	21	endovascular	7.7 (1/13)	14.3 (3/21)	23.8 (5/21)

LCNP Lower cranial nerve palsy, *LTS* long tract signs, *RC* respiratory complications.

The 2 deaths in our series were not related to choice of treatment; one thromboembolic complication occurred as a result of low grade dissection at an early stage of coil deployment.

Surgery

Proximal occlusion and surgical trapping of the dissecting segment are reasonable options in the treatment of haemorrhagic vertebrobasilar dissection. Rehaemorrhage after surgical proximal occlusion has been reported [3, 13], whereas trapping excludes the dissection from the circulation thus eliminating the risk of rebleeding which has not been reported after surgical trapping for vertebrobasilar dissection. However, the location of dissection makes the surgical approach technically demanding with a high risk of cranial nerve damage. Rigorous comparison between surgical and endovascular treatment is difficult, because most reports of surgical treatment include less than 10 patients and their features are not comparable. Nevertheless, the findings in representative surgical [4] and endovascular [36, 40] series are summarized in Table 3. The incidence of up to 76% of lower cranial nerve palsy and of 21% of respiratory complications after surgical treatment favors an endovascular approach as the primary choice in the treatment of haemorrhagic dissection of the vertebral or basilar arteries.

Conclusions

Arterial dissections are acute lesions that can heal spontaneously; lowering the hemodynamic stress by complete exclusion or reversal of flow seems to be sufficient to induce the healing process. Occlusion of the parent artery is an effective way to achieve this with a low morbidity and mortality. Treatment should be carried out as soon as possible in an acute haemorrhagic dissection, however, after 3 weeks to 1 month, conservative management is recommended if the abnormal lumen is not increasing. After permanent occlusion of a vessel, we recommend the use of heparin and aspirin until the first follow-up angiogram.

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Comment

Zhao *et al.* report on a retrospective series of 21 patients harbouring subarachnoid haemorrhage from vertebral artery dissections. The disease itself is serious and the outcome of patients with this type of bleeding is in general poor. However, due to a high rate of early re-bleeding and mortality associated with re-bleeding, in the initial stage of the disease an acute initiation of therapy appears mandatory.

Different strategies of treatment – endovascular, typically with parent artery occlusion, conservative or surgical clipping – are demonstrated and show that the disease is so rare, that guidelines cannot be given and treatment recommendation is limited to ''thorough investigation of the patients' angiomorphology'' with individual choice of treatment. In general, typically endovascular treatment is the treatment of choice in hemorrhagic intracranial vertebrobasilar dissection.

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