Spontaneous Intradural Vertebral Artery Dissection: A Single-Center Experience and Review of the Literature

Hasan Kocaeli, M.D.,¹ Chiraz Chaalala, M.D.,¹ Norberto Andaluz, M.D.,^{3,4} and Mario Zuccarello, M.D.^{1,2}

ABSTRACT

Objectives: To define the natural history of spontaneous intracranial vertebral artery dissections (VADs) and to review current treatment strategies. **Material and methods:** We searched the MEDLINE database for all existing English and French literature on VADs through January 2008. Keywords employed were intradural/intracranial vertebral artery dissection, vertebral artery dissection, and vertebral artery dissection treatment. We also reviewed our series of patients with spontaneous VAD treated in the past 5 years. Data were collected, categorized, and analyzed. **Results:** In our sample of 457 patients, men were more frequently affected than women, and the mean age was 51.8 years. The majority of patients (79%) presented with subarachnoid hemorrhage (SAH). We experienced a high incidence (37%) of recurrent SAH, particularly within the first 24 hours after SAH first occurred. Angiographic fusiform dilatation and pearl-and-string lesions were the most common finding. Patients who presented with SAH fared worse than those who presented with ischemia. **Conclusions:** Due to a high rate of recurrent bleeding, we concluded that early treatment by either surgical or endovascular route is indicated in patients who present with SAH secondary to spontaneous intradural VADs. Treatment decisions should take into account the site and type of dissection, vertebral artery dominance, and involvement of posterior inferior cerebellar artery.

KEYWORDS: Vertebral artery, arterial dissection, intradural vertebral artery, vertebral artery dissection, vertebral artery dissection treatment

Spontaneous vertebral artery dissection (VAD) is a rare condition that occurs in relatively young patients who present with subarachnoid

hemorrhage (SAH) or ischemic symptoms.^{1,2} The etiology of spontaneous VAD remains unknown, and multiple predisposing factors (i.e., arterial

¹Department of Neurosurgery, University of Cincinnati College of Medicine, ²The Neuroscience Institute and Mayfield Clinic, Cincinnati, Ohio; ³Department of Neurosurgery, University of South Florida; ⁴James A. Haley Veterans Hospital, Tampa, Florida.

Address for correspondence and reprint requests: Mario Zuccarello, M.D., c/o Norberto Andaluz, M.D. Department of Neurosurgery, University of South Florida, 13,000 Bruce B. Downs Blvd. ML112, Tampa, FL 33612 (e-mail: nandaluz@health.

usf.edu).

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Received: April 25, 2008. Accepted: September 10, 2008. Published online: January 9, 2009.

DOI 10.1055/s-0028-1114296. ISSN 1531-5010.

hypertension, fibromuscular dysplasia, and collagen abnormalities) have been incriminated in its pathophysiology.^{1,3-6} Unlike traumatic VAD, spontaneous VAD can have an unfavorable outcome, especially in patients presenting with intradural VAD and SAH.²

Intradural VADs represent 3 to 5% of clinical and autopsy series of patients with SAH. In one clinical series, 28% of vertebrobasilar system aneurysms were of a dissecting nature.⁷ Spontaneous VAD is most commonly observed in the V2 and V3 segments of the vertebral artery (VA); only 11% of spontaneous VADs occur in the V4 (intracranial) segment of the VA.⁴ The natural history of spontaneous VAD remains unclear, and its treatment is controversial. Despite the lack of general guidelines for the treatment of spontaneous VAD, it is common practice to treat aggressively patients who present with hemorrhage, and to treat conservatively those who present with ischemic symptoms.^{2,8-14} However, Yamada et al have reported favorable outcomes in patients with SAH treated conservatively.¹⁴ In an attempt to define the natural history of spontaneous VAD, and to propose appropriate treatment strategies for such lesions, we performed a comprehensive review of the literature and analyzed our clinical series of patients treated for VAD at our institution in the past 5 years.

MATERIAL AND METHODS

A query of the Medline database was conducted for articles in the English and French literature published before January 2008 using the following keywords: intradural/intracranial vertebral artery dissection, vertebral artery dissection, and vertebral artery dissection treatment. The criteria for our analysis included: clinical or autopsy series with five or more patients with unilateral spontaneous VAD of the intradural, intracranial (V4) segment of the VA for whom individual data on at least clinical presentation and outcome were present. Case reports, series with less than five patients, patients

with bilateral VAD, extension of dissection into the basilar artery, traumatic VAD, and extracranial VAD were excluded. Using the same methodology, charts of patients treated for spontaneous intradural VADs at the University of Cincinnati between January 2003 and January 2008 were retrospectively reviewed and collected in a table. Tables containing the following patient information (when available) were built: age, gender, comorbidities, clinical presentation, angiographic findings, site of dissection in relation to the posterior inferior cerebellar artery (PICA), side of the dissection, relationship to vertebral dominance, recurrence of symptoms or rebleeding, treatment, and outcome.

Clinical symptoms were classified in one of the following categories: (1) SAH; (2) ischemia, if the patient presented with transient ischemic attack (TIA) or stroke; (3) other, if the patient presented with pain (including neck pain or headache without hemorrhage), or if the dissection was diagnosed incidentally. Subarachnoid hemorrhage was graded according to the Hunt and Hess grading scale, based on the information provided in each report. Angiographic findings were classified as follows: (1) pearl-and-string lesion, if the dissection consisted of a succession of aneurysmal dilatations and vessel constrictions; (2) string lesion, if the dissection consisted only of a constriction; (3) fusiform aneurysm, if the dissected segment was dilated in a diffuse way; (4) saccular aneurysm, if the dilatation was focal and had a neck; (5) double lumen; and (6) arterial occlusion. Based on the multiple outcome measures described in the clinical series reviewed, outcomes were classified as:

- Good recovery if the patient had a Glasgow Outcome Score (GOS) of 4 to 5, a modified Rankin score (mRS) of 0 to 2, or a Karnofsky score of 80 to 100
- Moderate disability if the patient had a GOS of 3, an mRS of 3, or a Karnofsky score of 50 to 70
- Severe disability if the patient had a GOS of 2, an mRS of 4 to 5, or a Karnofsky score of 10 to 40
- Death

Table 1 Summary of Clinical Data, Findings, Treatment, and Outcomes on Eight Patients with Spontaneous Vertebral Artery Dissection*

Age, Gender	Risk Factors	Clinical Presentation	Angiographic Findings	Site of Dissection	Side of Dissection	VA Dominance	Recurrent Symptoms	Treatment	Outcome
46, female	HTN, cocaine	SAH, HH 3	Fusiform aneurysm	PICA	Right	Right	No	Proximal clipping	Death
34, male	None	Ischemia	Fusiform aneurysm	Distal to PICA	Left	Right	No	Proximal coil occlusion	VA Moderate disability
38, female	None	SAH, HH 3	Pearl-and-string lesion	Unknown	Left	Right	No	Proximal coil occlusion	VA Good recovery
45, female	Tobacco, cocaine	SAH, HH 3	String lesion	Extra- and intracranial	Right	Codominance	No	Proximal clip occlusion VA + OA-PICA bypass	Good recovery
49, male	HTN, tobacco, cocaine	SAH, HH 3	Fusiform aneurysm	PICA	Right	Codominance	No	Proximal clip occlusion	Good recovery
52, female	None	SAH, HH 3	Fusiform aneurysm	Distal to PICA	Right	Left	No	VA segment trapping	Good recovery
57, female	HTN	SAH, HH 2	Fusiform aneurysm	Distal to PICA	Right	Codominance	No	Proximal clip occlusion	Good recovery
42, female	None	Ischemia	Fusiform aneurysm	PICA	Left	Codominance	No	Coil occlusion VA	Moderate disability

*Patients were treated over 5-year period at the Department of Neurosurgery, University of Cincinnati.
VA, vertebral artery; HTN, arterial hypertension; SAH, subarachnoid hemorrhage; HH, Hunt and Hess grade; PICA, posterior inferior cerebellar artery; OA, occipital artery.

RESULTS

Using the search strategy described previously, 30 studies including 457 patients were obtained for review.^{2,3,6,9-12,14-35} Our clinical series consisted of eight patients, whose characteristics are summarized in Table 1. An illustrative case is presented in Fig. 1.

Patient Characteristics

Data on patient age were available in 28 of 30 series (443 patients). Overall mean age at presentation was 51.8 years. For patients who presented with SAH or with "other symptoms," mean age was 52.4 years; for those presenting with ischemic symptoms, mean age was 50.2 years. Gender data, available in all but one series, revealed that VAD occurred in 61% of men and 39% of women. Among risk factors, analysis of data available in only seven studies (95 patients) revealed a 38% prevalence of arterial hypertension. Fibromuscular dysplasia, often cited in the pathophysiology of dissections, was noted in only four patients.

Clinical Presentation

The most frequent presentation of spontaneous VAD was SAH (79%), followed by ischemia (16%) and other symptoms (5%). Of patients who presented with SAH, 37% experienced recurrent SAH (which occurred most commonly within 24 hours of initial SAH), and 0.3% had subsequent ischemic symptoms. Among those who presented with ischemia, 6% had recurrent ischemic events, and 1.5% suffered concomitant SAH. In this series, 1.5% of patients presented with both SAH and ischemic symptoms at onset. Some of those patients classified as SAH had a history of previous neck pain or headache and were included in the SAH group because it was impossible to rule out SAH at onset, given the fact that they did not receive diagnostic computed tomography (CT) scan on symptom onset.

Figure 1 Illustrative case: A 49-year-old man who had a history of hypertension, tobacco, and recent cocaine consumption presented with a history of headache 4 days before his admission. His neurological examination was normal. (A) Admission computerized tomography scan revealed subarachnoid hemorrhage in the perimedullary cistern. (B) Axial T1-weighted magnetic resonance scan demonstrates a large, fusiform, right vertebral artery (VA) with hyperintense signal representing intramural hematoma. (C) Angiogram depicts a right fusiform dissecting aneurysm incorporating the origin of the right posterior inferior cerebellar artery (PICA). (D) An occipital artery-to-PICA bypass was performed followed by (E) clip occlusion of the VA just proximal to the aneurysm. (F) Left VA injection shows good filling of the basilar artery. The patient did well after surgery and has been neurologically stable for 2 years.

Angiographic Findings

Reports on individual angiographic findings were available in 26 of the 30 series analyzed, representing a total of 365 patients. Findings are summarized in Table 2 and Fig. 2. Fusiform dilatation and pearl-and-string lesions were the most common findings in this series, representing 63.2% and 24.1%, respectively. For patients who presented with SAH, the most frequently reported angiographic findings were fusiform aneurysm (70.8%), and pearl-and-string lesion (24.5%). In the group of patients who presented with ischemia, angiographic findings

were more evenly distributed, the most common finding being the string lesion (30.5%; Table 2). The relation between the angiographic findings and outcomes is summarized in Fig. 3.

Twenty-three studies documented the anatomical side of spontaneous VAD. The right side was affected more often (54.8% of cases) than the left side (45.2% of cases). Three series reported on the location of the affected vertebral artery in relation to vertebral dominance. The dominant side was affected in 23% of cases, and the vertebral codominance was present in 52% of patients with VAD.

Table 2 Summary of Angiographic Findings Distribution and their Clinical Presentation in 365 Patients with Spontaneous Vertebral Artery Dissection

	Fusiform Aneurysm	Pearl-and-String Lesion	Occlusion	String Lesion	Double Lumen	Saccular Aneurysm	Negative
SAH	199	69	1	10	0	1	1
Ischemia	17	13	7	18	2	2	0
Other	15	6	3	0	1	0	0
Total	231	88	11	28	3	3	1

SAH, subarachnoid hemorrhage.

Site of dissection was mentioned in 22 series (324 patients). Dissection was distal to the PICA in 50.4% of cases, proximal to PICA in 29.3% of cases, and at the origin of PICA in 20.3% of cases; PICA on the side of dissection was absent in 6% of cases.

Treatment

The majority of patients who presented with SAH were treated with either surgical or endovascular procedures,^{2,6,8,11,13} whereas most of the patients who presented with ischemia received medical

Figure 2 Angiographic findings in 457 patients with spontaneous vertebral artery dissections. ISCH, ischemia; SAH, subarachnoid hemorrhage; N, negative; SA, saccular aneurysm; DL, double lumen; S, string lesion; P&S, pearl-and-string lesion; FA, fusiform aneurysm. **Figure 3** Percentage of outcome distribution for patients with vertebral artery dissection categorized according to angiographic findings. SD&D, severe disability and death; MD, moderate disability; GR, good recovery.

Figure 4 Percentage of outcome distribution for patients with vertebral artery dissection categorized according to clinical presentation. SD&D, severe disability and death; MD, moderate disability; GR, good recovery; SAH, subarachnoid hemorrhage.

therapy.^{8,10,14} A variety of surgical and endovascular options and combinations were used, including: proximal vertebral artery occlusion of the vertebral artery by clip placement or endovascular methods, bleb clipping, surgical or endovascular vertebral artery trapping, aneurysm wrapping, and parent vessel stenting.^{2,6,11,13,20,23,25,27-29,33,35} Medical treatment consisted primarily of blood pressure control and antiplatelet therapy. Intravenous heparin or subcutaneous low molecular weight heparin were employed in some cases.^{2,9,11,14,20,33,34,36}

Outcome

Patients who presented with SAH fared worse than those who presented with ischemia (Fig. 4). Outcomes in SAH patients correlated with their

Figure 5 Percentage of outcome distribution for patients with vertebral artery dissection categorized according to angiographic location of spontaneous vertebral artery dissection in relation to the posterior inferior cerebellar artery (PICA). SD&D, severe disability and death; MD, moderate disability; GR, good recovery.

Hunt and Hess grades at presentation. Recurrent SAH was also associated with worse outcomes; 65 (14.2%) of the patients who died suffered recurrent SAH. The only death in non-SAH patients occurred in the ischemia group and was unrelated to VAD. Outcomes based on angiographic location of spontaneous VAD in relation to PICA origin were available for 295 patients in this series (Fig. 5).

DISCUSSION

Spontaneous VAD is a rare disease for which treatment guidelines are nonexistent.^{2,6,28} Therapeutic strategies reported in the literature, which rely on clinicians' personal experience, range from aggressive surgical and/or endovascular modalities to medical therapy, and even include no treatment at all.^{2,9,11,14,20,33,34,36}

The results of our analysis of 457 patients with spontaneous VAD reported in the literature in French and English revealed that average age at presentation was 51.2 years, and men were affected more often than women (1.5:1 ratio). The right side was affected in more than half of the patients. Relationship of spontaneous VAD to vertebral artery dominance has been previously suggested.^{14,23,27} However, in our series this did not seem to be the case because codominance was present in the majority of patients. Although only a few series investigated and reported on the occurrence of minor trauma, the fact that more than half of the VA dissections were distal to the PICA—and, therefore, away from the tethered segment of the VA at its penetration through the dura mater into the posterior fossa—raises the theory of plausible spontaneous VAD without concomitant trauma. Although many risk factors have been proposed in the pathophysiology of intracranial dissections (i.e., arterial hypertension, fibromuscular dysplasia, polycystic kidney disease, defects in the internal elastic lamina, fibrous intimal thickening, mucoid-substance accumulation in the vessel

wall, cystic medial degeneration, migraine, moyamoya, homocystinuria, atherosclerosis, syphilis),⁵ the most frequently reported risk factor in the selected series was arterial hypertension. Whether arterial hypertension plays a key role in the development of spontaneous VAD or it represents a precipitating factor that leads to dissection in a weakened vessel remains uncertain.^{4,5}

Spontaneous VAD may present clinically in a variety of ways including SAH, ischemic stroke, headache, or neck pain.⁴ The most frequent clinical presentation in our series was by far SAH (79% of patients), a finding consistent with that of other large series.^{2,11,23} However, this finding could represent a publication, or selection, bias because many asymptomatic patients may go undiagnosed and never seek medical attention. In our analysis, some of the patients classified in the SAH group had neck pain or headache before their hemorrhage and it was difficult to establish whether these symptoms were related to intramural dissection or sentinel hemorrhage. Clinical presentation with SAH was strongly associated with worse outcomes when compared with the other groups. As with SAH of aneurysmal origin, the higher the Hunt and Hess grade at presentation, the worse the outcome. Recurrent bleeding was a serious and often fatal consequence of VAD presenting with SAH, occurring in more than one third of those patients, and typically it occurred within the first 24 hours after SAH first appeared. Recurrent bleeding from spontaneous VAD was probably underestimated because only cases where it was proved either by CT scan or autopsy were included. In patients presenting with ischemia, bleeding or recurrent symptoms seemed to be the exception.

The origin of spontaneous VADs involving the V4 segment is multifactorial and cases associated with trauma, hypertension, and fibromuscular dysplasia have been reported.⁴ The V4 region represents the most proximal part of the intracranial VA and is likely subject to shearing forces associated with head motion. Histological changes that occur as the VA transitions from extra- to intracranial may also predispose the segment to dissection. At

this level, the thickness of the tunica media and the adventitia tapers as the vessel pierces the dura.¹⁶

Two mechanisms have been proposed to account for the clinical presentations of spontaneous VAD.^{16,37} For VADs presenting with ischemia, it has been postulated that dissection between the internal elastic lamina (IEL) and the media^{16,37} pushes the IEL toward the vessel lumen, resulting in narrowing or occlusion. On the other hand, dissection within the tunica media or adventitia, followed by a disruption of the entire vessel wall, has been theorized as the mechanism leading to SAH.

In this review, most of the patients who presented with recurrent SAH and subsequently died had either pearl-and-string lesions (70.8%) or fusiform aneurysms (25.4%) on angiography. However, the occurrence of such lesions was not necessarily associated with poor outcomes or recurrent SAH. In fact, some of the patients who presented with such lesions received no treatment due to the following reasons: benign presentation,^{8,9,12,26} poor clinical condition on arrival or after recurrent bleeding,^{2,10,14,18,27} absence of collateral circulation,^{10,14} or delayed diagnosis.¹⁴ Long-term follow-up for most of those patients is missing. In a series of 42 patients, Mizutani et al found no correlation between the type of angiographic dissection and subsequent rupture, although the angiographic findings in those patients were not discussed.² In a later report, the authors classified nonatherosclerotic dissecting VA aneurysms into four types, based on data obtained from a series of 85 patients, some of which had a histopathological correlation. The types they defined were:

- Type-1: Dissecting VA aneurysms corresponded to an acute disruption of the IEL. Patients who presented with this type of lesion were more prone to SAH and recurrent bleeding.
- Type-2: Dissecting VA aneurysms correlated with a fragmented IEL with a compensating, thickened intima without intraluminal thrombus. Patients harboring this type of lesion are thought to have a benign course.

- Type-3: Dissecting VA aneurysms were enlarging dolichoectatic dissecting aneurysms characterized by fragmentation of the IEL, thickening of the intima, and organized intraluminal thrombus, placing patients at risk for mass effect on the brainstem and SAH.
- Type-4: Dissecting VA aneurysms were saccular aneurysms unrelated to the branching points, characterized by areas with minimally disrupted IEL without intimal thickening, and a high risk of rupture.²⁴

In 2001, Mizutani et al classified dissecting aneurysms into either entry-only or entry-exit type aneurysms. In the first type, blood enters the pseudolumen without any exit route, resulting in a potentially unstable aneurysm; whereas in the second type, blood enters then exits the pseudolumen.³⁸ This theory might explain why some patients with the same angiographic lesion could have different presentations and outcomes. Whether the origin of the dissection is an intramural bleed with rupture of vasa vasorum or a defect of the wall's elastic tissue with consequent penetration of blood flow from the true lumen into the false lumen might be another explanation for the differences in clinical behavior.³⁹ Histological differences in the wall's structure are another reason why VADs do not heal following a similar pattern on serial angiographic follow-up.^{1,8,10,26,36,40–42}

Since the late 1990s, the treatment of spontaneous VAD appears to be changing in parallel to the progress in endovascular technology. Due to the high rate of surgical morbidity, which is mostly related to lower cranial nerve injury, endovascular treatment in the form of proximal parent artery occlusion, endovascular trapping, and/or intracranial stenting has become the first line of treatment reported in most series.^{6,10,22,23,31,33,34} Spontaneous VAD presenting with ischemia can usually be treated conservatively. Although it appears controversial, one series reported that proximal parent artery occlusion alone prevented recurrent bleeding in 100% of cases.⁶ When hypoplastic contralateral VA is encountered or the origin of the PICA is

involved, either bypass surgery or stent placement to preserve the affected artery is necessary.²³ Blood pressure control represents an important treatment aspect, not only to prevent recurrent bleeding in the acute phase, but also during follow-up to promote healing in conservatively treated patients. Finally, it should be emphasized that in cases in which spontaneous vertebral artery dissection is clinically suspected, repeat angiogram must be considered because the first angiogram might be negative.

The current literature review constitutes only a collection of the available data and its conclusions should not be grounds to modify clinical practice because of selection and publication biases. The analyses and observations made only take into account the subset of patients for whom particular information of interest was available. However, this information provides some insight into the epidemiology and natural history of the disease, as well as the large need for treatment guidelines.

CONCLUSIONS

The question of whether VAD should be treated aggressively or medically still remains unanswered. The only outcome predictor identified in our study was initial clinical presentation, which appears to be related to the severity of arterial wall damage. Treatment strategies should rely not only on clinical grounds, but also on a combination diagnostic imaging methods, such as angiogram and magnetic resonance imaging, to understand the severity of the dissection.

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