Vertebral Artery Dissection: Presenting Findings and Predictors of Outcome

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Marcel Arnold, MD; Marie Germaine Bousser; Gregor Fahrni, MS; Urs Fischer, MD; Dimitrios Georgiadis, MD; Joubin Gandjour, MD; David Benninger, MD; Matthias Sturzenegger, MD; Heinrich P. Mattle, MD; Ralf W. Baumgartner, MD

Background and Purpose—Few data exist about clinical, radiologic findings, clinical outcome, and its predictors in patients with spontaneous vertebral artery dissection (sVAD).

Methods—Clinical characteristics, imaging findings, 3-month outcomes, and its predictors were investigated in consecutive patients with sVAD.

Results—One hundred sixty-nine patients with 195 sVAD were identified. Brain ischemia occurred in 131 patients (77%; ischemic stroke, n=114, 67%; transient ischemic attack, n=17, 10%). Three patients with ischemic stroke showed also signs of subarachnoid hemorrhage (SAH); 3 (2%) had SAH without ischemia. The 134 patients with brain ischemia or SAH had head and/or neck pain in 118 (88%) and pulsatile tinnitus in seven (5%) patients. The remaining 35 patients (21%) had isolated head and/or neck pain in 21 (12%) cases, asymptomatic sVAD in 13 (8%), and cervical radiculopathy in one case (1%). Location of sVAD was more often in the pars transversaria (V2; 35%) or atlas loop (V3; 34%) than in the prevertebral (V1; 20%) or intracranial (V4; 11%) segment ($P=0.0001$). Outcome was favorable (modified Rankin scale score 0 or 1) in 88 (82%) of 107 ischemic stroke patients with follow up. Two (2%) patients died. Low baseline National Institutes of Health Stroke Scale score ($P<0.0001$) and younger age ($P=0.007$) were independent predictors of favorable outcome.

Conclusions—sVAD is predominantly located in the pars transversaria (V2) or the atlas loop (V3). Most patients show posterior circulation ischemia. Favorable outcome is observed in most ischemic strokes and independently predicted by low National Institutes of Health Stroke Scale score and younger age. **(*Stroke*. 2006;37:2499-2503.)**

Key Words: dissection ■ MRI ■ outcome ■ stroke

Spontaneous dissection of the vertebral artery (sVAD) is a potentially disabling and probably underdiagnosed cause of stroke mainly affecting young adults.[1,2] Few small-scale series have reported the clinical manifestations of sVAD, which are thought to be occipital headache or neck pain, or both, and posterior circulation ischemia.[3–8] Two studies have also analyzed the clinical outcome and factors predicting clinical outcome.[6,9]

The aim of this study is to determine the presenting clinical and vascular imaging findings as well as clinical outcome and its predictors in a large series of patients with sVAD.

Methods

The prospectively collected data of all patients presenting with first-ever sVAD at three tertiary care centers (Lariboisière Paris, Zurich, and Berne) from January 1997 through June 2005 were reviewed. Patients were pooled from prospective databases of patients with spontaneous cervical artery dissection (sCAD). Of a total of 537 patients with sCAD, 144 (27%) had sVAD alone, 25 (5%) sVAD and spontaneous internal carotid artery dissection (sICAD), and 368 (68%) sICAD. All patients with at least one sVAD were included in the study. VAD were categorized as spontaneous when occurring spontaneously or associated with a minor trauma.[10] Seventeen patients with a major head or neck trauma (car accident or sports trauma) causing blunt vertebral artery injury were excluded from the study. Ischemic deficits were classified according to their duration as stroke (>24 hours) or transient ischemic attack (TIA; ≤24 hours).

Investigations

Risk factors for ischemic stroke and sCAD were diagnosed as follows. Hypertension was defined as a history of antihypertensive treatment or a history of hypertension (systolic blood pressure [BP] >160 mm Hg, diastolic BP >90 mm Hg, or both) until September 2000.[11] The new World Health Organization criteria for diagnosis of hypertension (systolic BP >140 mm Hg, diastolic BP >85 mm Hg, or both) were used since October 2000.[12] Hypercholesterolemia was defined as total cholesterol value >5.2 mmol/L.[11] All patients had a neurologic examination, including the determination of stroke severity by the National Institute of Health Stroke Scale (NIHSS), a physical

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From the Department of Neurology (M.A., M.G.B.), Assistance Publique, Hôpitaux de Paris, Lariboisière, Paris, France; the Department of Neurology (M.A., U.F., M.S., H.P.M.), University Hospital Bern, Bern, Switzerland; and the Department of Neurology (G.F., D.G., J.G., D.B., R.W.B.), University Hospital Zurich, Zurich, Switzerland.

Correspondence to Ralf W. Baumgartner, MD, Department of Neurology, University Hospital, Frauenklinikstrasse 26, 8091 Zürich, Switzerland. E-mail ralf.baumgartner@usz.ch

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examination, routine blood examinations, an electrocardiogram, and an assessment of the cerebral arteries by angiography and/or MRI of the neck as mentioned previously. Patients with ischemic stroke had also cranial CT, MRI, or both. Lumbar puncture was performed only in patients with clinical suspicion of subarachnoid hemorrhage (SAH) or intracranial extension of sVAD on magnetic resonance angiography (MRA) or digital subtraction angiography (DSA).

Angiographic, Cervical MRI, and Cerebral CT and MRI Studies

Only patients with diagnosis of sVAD confirmed by MRI with T1 fat-suppression technique (wall hematoma), DSA, or both were included. Angiographic criteria consisted of string sign, pseudoaneurysm, or intimal flap. Occlusive forms diagnosed by DSA, which did not show in addition at least one of the aforementioned three typical angiographic findings, were confirmed by MRI detection of the wall hematoma.[7,13] The site of sVAD was defined by DSA, MRA, and/or cervical MRI. The presence of fibromuscular dysplasia was assessed only in patients who underwent DSA.

All radiologic findings including the confirmation of sVAD diagnosis were assessed by M.A. in Paris and G.F., M.A., and R.W.B. in Bern and Zurich by consensus reading.

Thrombolytic and Antithrombotic Therapy

Five patients with stroke with basilar artery occlusion were treated with local intraarterial thrombolysis, and another four patients with stroke with intravenous thrombolysis. The usual treatment included intravenous heparin followed by oral warfarin with a target international normalized ratio of 2.5 (range, 2.0–3.0) for 3 to 6 months (n=97). In contrast, patients who showed at brain imaging hemorrhagic transformation or large infarcts according to the judgment of the treating physician were treated with 100 to 300 mg oral aspirin per day during the first 7 to 14 days (n=32). Subsequently, the treating physician decided whether the patients were left on aspirin (n=15) or the platelet inhibitor had to be replaced by oral warfarin (n=17) for 3 to 6 months. Patients with intracranial sVAD or intracranial extension of sVAD were treated with 300 mg aspirin per day for 3 to 6 months (n=34). If 3-month ultrasound follow up or MRA showed complete recanalization of the vertebral artery, antithrombotic therapy was stopped. Otherwise, antithrombotic therapy was maintained until 6-month follow up. In patients with complete recanalization after 6 months, antithrombotic therapy was stopped. In case of residual vertebral artery stenosis or occlusion and in cases with fibromuscular dysplasia, 100 to 300 mg oral aspirin daily was prescribed. Six patients with SAH resulting from intracranial sVAD did not receive any antithrombotic treatment.

Clinical Follow Up

Clinical follow-up information of patients with ischemic stroke was obtained through neurologic examination (n=100) or a structured telephone interview (n=7) by a neurologist after 3 months. The modified Rankin Scale score (mRS) was recorded in all patients.[14] Seven patients were lost to follow up.

Statistical Analysis

Statistical analysis was performed with SPSS 10 for Macintosh statistical software (SPSS Inc). For comparison of outcome, we divided patients into two groups with favorable (mRS score 0–1) and nonfavorable (mRS score 2–6) outcome. The χ^2 test was performed for crosstabulation. Mann-Whitney test was used for noncategorical variables. The following variables were analyzed: age, sex, treating hospital, history of minor trauma, time interval from symptom onset to diagnosis, baseline NIHSS score, smoking, hypertension, diabetes mellitus, hypercholesterolemia, history of migraine, presence of multiple dissections, location of sVAD, vertebral artery occlusion at angiography, thrombolysis, and antithrombotic treatment. For comparison of the type of antithrombotic treatment, we divided patients into three groups as follows: (1) aspirin only, (2) heparin followed by warfarin, and (3) aspirin administered for at least 7 days followed by warfarin. Then, logistic regression analysis with a forward stepwise method was performed to determine the independent association of favorable outcome with other clinical and radiologic factors. The cutoff in the univariate analyses as requirement for inclusion in the multivariate analyses was $P<0.25$.

Results

Presenting Characteristics and Clinical Findings

There were 195 sVAD in 169 patients (mean age, 43±9; median, 43; range, 21–69 years). Seventy-nine (47%) were female and 90 (53%) male ($P=0.716$). Patients were recruited in the University Hospitals of Paris Lariboisière (n=89 [53%]), Zurich (n=46 [27%]), and Bern (n=34 [20%]). Seventy-six (45%) of 169 patients presented with left, 67 (40%) with right, and 26 (15%) with bilateral sVAD. In 25 patients (15%), 21 of them with unilateral sVAD and four with bilateral sVAD, an additional sICAD was detected. Median time interval from symptom onset to diagnosis was 4 days (range, 2 hours to 88 days). The presenting characteristics are summarized in Table 1. One patient with unilateral sVAD had a known Ehlers Danlos syndrome type IV; another patient with a bilateral sVAD had Marfan syndrome. None of the patients had a family history of cervical artery dissection.

Clinical symptoms occurred in 156 (92%) of 169 patients with sVAD. The remaining 13 patients (8%) had asymptomatic sVAD, which accompanied symptomatic sICAD. Presenting clinical symptoms were ischemic stroke in 114 (67%), TIA in 17 (10%), and occipital head and/or neck pain alone in 21 (12%) patients, SAH without ischemia in three (2%) patients, and a sensorimotor cervical radiculopathy C5/C6 in one patient (1%). Three of the patients with ischemic stroke showed also signs of SAH on brain imaging. A total of 118 (88%) of 134 patients with ischemic or hemorrhagic symptoms had also occipital head and/or neck pain and seven patients (5%) a pulsatile tinnitus. Median NIHSS score on admission in patients with ischemic stroke was 3 (range, 1–35). Of the 114 patients with ischemic stroke, 15 (13%) had a TIA before stroke. Median time interval from TIA to stroke onset was 1 day (range, 1 hour to 17 days). No patient showed symptoms and signs of spinal cord ischemia.

Presenting Angiographic and Cervical MRI Findings

Angiography (DSA and/or MRA) and cervical MRI was performed in 130 of 169 patients, angiography alone in 27

TABLE 1. Presenting Characteristics in 169 Patients With 195 Spontaneous Vertebral Artery Dissections

Characteristics	No. of Patients (%)
Current smoking	62/169 (37)
Hypertension	36/169 (21)
Diabetes mellitus	3/169 (2)
Hypercholesterolemia*	89/135 (66)
Family history of stroke	29/162 (18)
Oral contraceptives	19/60 (32)
History of migraine	49/166 (30)
History of minor neck trauma	27/169 (16)

*Mean cholesterol values±SD (median, range) were 5.49±1.02 (5.54, 2.41–7.85) mmol/L.

patients, and cervical MRI alone in 12 patients. Thus, a total of 157 patients with 182 sVAD underwent angiography (MRA alone, 96 patients; DSA alone, 25 patients; MRA and DSA, 36 patients) and 142 patients cervical MRI.

MRA or DSA showed vertebral artery occlusion in 70 (38%), vertebral artery stenosis in 102 (56%), and an aneurysm and stenosis in 10 (6%) of 182 sVAD. Cervical MRI of 142 patients revealed a mural hematoma in 143 of 157 VADs (91%). Seven of 61 patients (11%) who underwent DSA showed fibromuscular dysplasia.

Of a total of 195 dissections, 21 were located intracranially and 174 extracranially, 19 of them with extension to the intracranial segment. The proximal beginning of sVAD was located in the prevertebral (V1) segment in 40 (20%), the pars transversaria (V2) in 68 (35%), the atlas loop (V3) in 66 (34%), and the intracranial (V4) segment in 21 (11%) sVAD ($P=0.0001$).

Clinical Outcome and Predictors of Clinical Outcome in Patients With Ischemic Stroke

Follow-up examinations were obtained in 107 of 114 (94%) patients; seven were lost to follow up. The mRS score was 0 in 26 (24%) patients, 1 in 62 (58%) patients, 2 in 10 (9%) patients, 3 in two (2%) patients, 4 in three (3%) patients, 5 in two (2%) patients, and two (2%) patients were dead. On univariate analysis, low NIHSS score ($P<0.0001$) and younger age ($P=0.043$) were predictors of a favorable clinical outcome (Table 2). After multiple regression analysis, low NIHSS score on admission ($P<0.0001$) and age ($P=0.007$) remained independent predictors of a favorable clinical outcome. Sex, the treating hospital, history of minor trauma, time interval from symptom onset to diagnosis, vascular risk factors, a history of migraine, the presence of multiple dissections, location of sVAD, vertebral artery occlusion at angiography, thrombolysis, and the type of antithrombotic treatment were not independently associated with clinical outcome (Table 2).

Discussion

This is the first large series analyzing presenting clinical, cerebrovascular, and brain imaging findings as well as clinical outcome and its predictors in patients with sVAD.

Previous smaller series reported conflicting results on sex predominance in patients with sVAD with percentages of male patients ranging from 16 to 60%.[1,5,15] In this study, there were no significant differences in sex prevalence. The percentage of men was 54% and is similar as in previous large studies of cervical artery dissection reporting 48 to 55% male patients.[11,16,17]

The frequency of hypercholesterolemia was higher in this than in previous studies of cervical artery dissection or sICAD.[11,18] However, the results of these studies are not entirely comparable because of different populations. Because of the lack of a control group, it remains unclear whether the higher prevalence of hypercholesterolemia observed in this study is the result of chance or whether hypercholesterolemia is a risk factor for sVAD.

The majority of our patients presented with symptoms and signs of posterior circulation ischemia, consisting mainly of stroke, occipital headache or neck pain, or both, which is in accordance with the results of previous series.[6,17,19] Ischemic stroke was preceded by at least one TIA in 13% of the patients with latencies between stroke and TIA of up to 17 days. This group is of particular interest because the stroke might have been prevented by immediate recognition of the symptoms and insaturation of an antithrombotic treatment. Twelve percent of the patients presented with headache and/or neck pain as the only symptom of sVAD and 8% had asymptomatic sVAD with simultaneous sICAD. This higher percentage of patients with pain as the only symptom of sVAD compared with previous series is probably the result of the fact that all three participating centers perform immediate imaging of the cervical arteries in any case of unusual head or neck pain.[1]

Only six of 40 patients with primary intracranial location or intracranial extension of sVAD experienced a SAH; three of them presented also with ischemic stroke, a finding that has not been reported in previous series.[20] The low number of SAH might be the result of selection bias, because patients with suspicion of SAH are usually admitted to the departments of neurosurgery.[21] The fact that SAH and ischemic stroke may occur in the same patients has to be kept in mind in clinical practice, and therefore we perform lumbar puncture also in patients with ischemic stroke with intracranial extension of sVAD or severe and sudden headache.

Fibromuscular dysplasia (FMD) was observed in 11% of the present patients with sVAD who underwent DSA. This rate is similar to the 5 to 18% FMD rate reported in patients with spontaneous cervical artery dissection.[16,18,22] The rate of FMD may be overestimated, because in case of suspicion of FMD, DSA may be more likely to be performed.

Different studies have reported conflicting results with respect to the anatomic location of sVAD. According to a small US study, approximately two thirds of sVAD occurred in the V3 segment.[1] Other authors reported no predilection of location in sVAD.[8,23] In our study, we assumed that intimal laceration occurred in the proximal part of dissection and that the mural hematoma would extend in the same direction as the bloodstream that is in the cranial direction. Thus, in dissections extending over several vertebral artery segments, just the proximal segment was used for defining the location. Using this approach, sVAD occurred significantly more often in the V2 or V3 segment than in the V1 or V4 segment. Arterial imaging showed intracranial extension of sVAD in 19 of 174 primary extracranially located sVAD. The possible intracranial extension of sVAD should be considered before the beginning of an anticoagulant treatment.

Clinical outcome was favorable (mRS 0–1) in the majority of patients and mortality was low. These results are in accordance with the results of smaller series reporting about patients with sVAD.[1,4,24] and sICAD.[25–27]

Low baseline NIHSS score and younger age were independent predictors of a favorable outcome. In contrast to previous series, intracranial location of sVAD and bilateral sVAD did not predict an unfavorable clinical outcome in our study.[6,9]

Our study is limited by its retrospective analysis of prospectively collected data. Another drawback of this and

TABLE 2. Predictors of Clinical Outcome After 3 Months in 107 Patients With Ischemic Stroke

Characteristics	Favorable Outcome (mRS 0–1) Patients, n (%)	Nonfavorable Outcome (mRS ≥2) Patients, n (%)	*P* Univariate Analysis (OR; 95% CI)	*P* Regression Analysis (OR, 95% CI)
Sex				
Female	39/46 (85)	7/46 (15)	0.55	
Male	49/61 (80)	12/61 (20)	(0.73; 0.26–2.04)	
Age	Continuous variable	Continuous variable	0.043	0.007
	(Mann-Whitney test)	(Mann-Whitney test)		(1.12; 1.03–1.21)
Treating hospital				
Bern	17/24 (71)	7/24 (29)		
Zurich	29/34 (85)	5/34 (15)	0.25	
Paris	42/49 (86)	7/49 (14)		
History of minor trauma				
Yes	15/18 (83)	3/18 (17)	0.89	
No	73/89 (82)	16/89 (18)	(0.91; 0.24–3.52)	
Latency to diagnosis	Continuous variable	Continuous variable	0.15	
	(Mann-Whitney test)	(Mann-Whitney test)		
Baseline National Institutes of Health Stroke Scale score	Noncategorical variable	Noncategorical variable	<0.0001	<0.0001
Smoking				
Yes	33/43 (77)	10/43 (23)	0.22	
No	55/64 (86)	9/64 (14)	(1.85; 0.68–5.02)	
Hypertension				
Yes	21/27 (78)	6/27 (22)	0.40	
No	67/79 (85)	12/79 (15)	(1.6; 0.53–4.77)	
Diabetes mellitus				
Yes	3/3 (100)	0/3 (0)	0.41	
No	85/104 (82)	19/104 (18)	(0.96; 0.93–1.01)	
Hypercholesterolemia				
Yes	43/51 (84)	8/51 (16)	0.90	
No	30/36 (83)	6/36 (17)	(0.93; 0.29–2.95)	
History of migraine				
Yes	21/23 (91)	2/23 (9)	0.19	
No	66/83 (80)	17/83 (20)	(0.37; 0.08–1.73)	
Multiple dissections				
Yes	16/21 (76)	5/21 (24)	0.42	
No	72/86 (84)	14/86 (16)	(1.61; 0.51–5.11)	
Location of spontaneous vertebral artery dissection				
V1 segment	17/24 (71)	7/24 (29)		
V2 segment	20/26 (77)	2/24 (23)	0.22	
V3 segment	34/39 (87)	5/39 (13)		
V4 segment	17/18 (94)	1/18 (6)		
Vertebral artery occlusion				
Yes	39/45 (87)	6/45 (13)	0.31	
No	49/62 (89)	13/62 (11)	(0.58; 0.20–1.66)	
Thrombolysis				
Yes	5/9 (56)	4/9 (44)	0.03	
No	83/98 (85)	15/98 (15)	(4.43; 1.06–18.40)	
Antithrombotic treatment				
Aspirin	27/33 (82)	6/33 (18)		
Aspirin/warfarin	10/13 (77)	3/13 (23)	0.766	
Heparin/warfarin	48/58 (83)	10/58 (17)		

Aspirin/warfarin indicates aspirin administered for at least 7 days followed by warfarin; heparin/warfarin, heparin followed by warfarin.

other studies performed in patients with sVAD is that some cases presenting with occipital or neck pain alone may not have been diagnosed, because they were not investigated using angiography and cervical MRI. Patients were included over a long time period with different examination techniques. However, sVAD is a rare disease, and we thus had to collect patients over a long period of time to obtain a sufficient sample size. The number of intracranial (V4) sVAD is probably underestimated in this study, because patients with intracranial sVAD causing SAH are sometimes hospitalized in the departments of neurosurgery and thus not assessed in our dissection registry. Finally, extension of sVAD to the V4 segment may have been missed in some patients because lumbar puncture, which may detect SAH, was not performed systematically.

In conclusion, sVAD was observed most frequently in the V2 or V3 segment. The majority of patients present with posterior circulation ischemia. Clinical outcome was favorable in most patients. Low NIHSS score on admission and younger age were independent predictors of a favorable clinical outcome.

Disclosures

None.

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