Ultrasound Findings in Spontaneous Extracranial Vertebral Artery Dissection

Matthias Sturzenegger, MD; Heinrich P. Mattle, MD; Anita Rivoir, MD; Frank Rihs, MD; Cornelia Schmid, MTA

Background and Purpose: In this study we analyzed the value of ultrasound examination for diagnosis of vertebral artery dissection.

Methods: The vertebrobasilar arterial system was assessed in 14 patients using transcranial and extracranial pulsed-wave Doppler and duplex sonography.

Results: The dissections were verified by angiography (in 1 patient), magnetic resonance imaging (in 5), or both (in 8). The dissected segments were atlantoaxial (V-3) in 6, V-3 and intertransverse (V-2) in 3, V-3 and intracrani (V-4) in 3, and V-2 in 2 patients. Extracranial and transcranial Doppler examination of the atlas loop, involved in 12 patients, showed absent flow signal in 5, low bidirectional flow signal in 1, and poststenotic low flow blood velocities in 3 patients. Seven of these patients had high-grade stenosis or occlusion. The stenotic segment with increased flow signal could be identified directly in 2 patients. Duplex examination of the intertransverse segment confirmed absent flow in 4 patients, making technically insufficient examination unlikely. In the 2 patients with directly detected stenosis, duplex examination showed low flow velocities before the stenosis. The combined use of extracranial and transcranial Doppler and duplex sonography increases the diagnostic yield to detect vertebral artery pathology. If any abnormal sonographic finding was considered, the yield was 86%; relying only on definitively abnormal findings (absent flow signal, severely reduced vertebral artery blood flow velocities, no diastolic flow, bidirectional flow, and a stenosis signal), the yield was 64%.

Conclusions: In most cases, there is no pathognomonic ultrasound finding for vertebral artery dissection. However, if a patient presents with suggestive symptoms, ultrasound may corroborate the clinical suspicion and aid in the decision regarding early anticoagulant treatment. A definite diagnosis can be made noninvasively when magnetic resonance imaging demonstrates hematoma in the vessel wall. Angiography yields additional information such as nature of underlying vascular disease, site and extent of dissection, intracranial extension, and presence of pseudoaneurysm. (Stroke. 1993;24:1910-1921.)

Key Words • angiography • dissection • Doppler • ultrasonics • vertebral artery

Dissections of cervicocerebral arteries have been increasingly recognized during the last 10 years as a cause of head and neck pain associated with transient ischemic attacks or strokes, especially in young patients.1-8 Internal carotid artery (ICA) dissections seem to be much more frequent than vertebrobasilar dissections (VAD).9 However, our experience with 14 patients diagnosed during a 2½-year period exceeds the cited incidence of 1 to 3 cases per year in large referral-based hospitals.4,7,10,11 It is therefore possible that VAD is underestimated.

The prominent clinical features of VAD are severe ipsilateral neck and occipital head pain and vertebrobasilar ischemia. Distal artery-to-artery embolism from local thrombus at the stenotic segment12,13 or anterograde progression of thrombosis may cause vertebrobasilar infarction leading to permanent disability or death. Therefore, most authors recommend immediate anticoagulation with heparin in extracranial VAD to prevent progressive clot propagation, embolism, and complete vessel occlusion. In many patients, there is a delay between dissection signaled by severe neck and head pain and the symptoms caused by the cerebral ischemia. Early diagnosis, therefore, is mandatory. When VAD is suspected clinically, the final diagnosis relies on angiography or magnetic resonance imaging (MRI) or both.1,7,14 Ultrasound examination was recently shown to be a sensitive and reliable tool in early diagnosis of ICA dissection.15-17 Its role in VAD has yet to be studied.2 There is only one larger series analyzing ultrasound findings in 10 patients, with multiple sites of VAD in 7 patients and bilateral VAD in 5,18 VAD was verified by angiography in 9 patients, which may not be reliable in differentiation from atherosclerotic occlusion (5 of 21 dissection sites) and proximal (V-1 segment) stenosis (7 of 21 dissection sites). Only one dissection was corroborated by MRI.18

In this report, we analyzed the ultrasound findings of the vertebrobasilar system in 14 patients with spontaneous extracranial VAD verified by angiography or MRI to define its role for diagnosis and management of VAD.

Subjects and Methods

Fourteen consecutive patients (7 women and 7 men; mean age, 45 years [range, 34 to 67 years]) with extracranial VAD proved by angiography or MRI or both
were analyzed. They were all admitted within 2.5 years (1990 through 1992). The patients were specifically interviewed regarding (1) vascular risk factors (high blood pressure, diabetes, hypercholesterolemia, smoking, use of oral contraceptives); (2) previous neck or head trauma, unusual neck movements, head position, sports activities, and strenuous exercises in the period before VAD; (3) warning symptoms (head or neck pain, vertebrobasilar transient ischemic attack); and (4) actual (presenting) symptoms (headache and neck pain, symptoms of vertebrobasilar ischemia) (Table 1). All patients had complete blood cell count, sedimentation rate, serum protein electrophoresis, profile of blood glucose, cholesterol, and lipid electrophoresis, coagulation studies (platelet count, prothrombin time, antidiolipin antibodies), liver enzymes, serum creatinine, Lyme and lues serology, electrocardiogram, chest x-ray, x-ray of cervical spine in four planes, echocardiogram, extracranial and transcranial pulsed-wave Doppler sonography, and extracranial Doppler duplex sonography. The ultrasound investigator was not involved in the patients’ management, and in the 6 patients who had imaging procedures before ultrasound examination, he was blinded to these results. Twelve patients had cerebral computed tomographic scan, and 8 had lumbar puncture on the day of admission. Nine patients had selective cerebral catheter angiography, 13 had MRI scans, 8 patients had both MRI scans and angiography, 1 had angiography only, and 5 had MRI only (Table 3). Arterial stenosis and mural hematoma were judged according to location, extent, regularity, and degree. The location of the arterial changes was defined according to standard criteria:9,20 V-1 (prevertebral) segment: from origin of subclavian artery to entry of transverse foramen; V-2 (intertransverse) segment: course in the costotransverse canal C-6 to C-2; V-3 (atlantoaxial) segment: C-2 transverse process to occipital foramen; V-4 (infratraquebral) segment: dural entry to junction with contralateral vertebral artery (VA). All patients except two (patients 8 and 14) had clinical and ultrasonographic follow-up every 2 to 3 months for at least 6 months (Table 1). Ten patients had follow-up MRI scans. None had control angiography.

Duplex Examination

A high-resolution, real-time duplex ultrasound system (Diasonics SPA 1000) was used, consisting of a 10-MHz echotomography imaging probe and a 4.5-MHz pulsed-wave Doppler probe with real-time gray scale fast-Fourier transform Doppler spectrum analysis. The extracranial carotid system (common carotid artery [CCA], bifurcation, ICA, and external carotid artery [ECA]) and the VA (before entering the transverse foramina [V-1 segment] and in the intertransverse segment [V-2]) were imaged on both sides, and blood flow velocities (BFV) were recorded.21-25 B-mode resolution was not adequate enough in all patients to allow reliable measurements of VA diameters. To analyze BFV, the C-5/C-6 intertransverse segments were used.

Extracranial Doppler Examination

The technique for extracranial Doppler examination has been described in detail previously.26 We used the same device as for transcranial Doppler recordings, described below. The carotid systems (CCA, ECA, ICA), the subclavian artery in the supraclavicular region, and VA at the atlas segment (V-3) and through the occipital foramen (V-4 segment) were examined by continuous insonation on both sides. For VA examination the probe was positioned medial to the mastoid process (lateral suboccipital approach).27-30 Doppler signals at an insonation depth between 3 and 7 cm were allocated to the VA.26 Supratrochlear and ophthalmic arteries and carotid siphons were measured by a transorbital approach.

Transcranial Doppler Examination

The technique for transcranial Doppler recordings has been described in detail by Aaslid.21 A microprocessor-controlled directional pulsed-wave Doppler device operating at 2 MHz was used for all transcranial Doppler ultrasound examinations (TC 2000, EME, Uerlingen, Germany). Middle cerebral, anterior cerebral, posterior cerebral artery, and ICA bifurcation were analyzed at different depths by a transtemporal approach and intracranial VA and basilar artery through the occipital foramen. The VA was insonated from the lateral suboccipital approach. The basilar artery was examined from a median suboccipital probe position at an insonation depth of 7.5 cm or more.26,29,30,32,33

Analysis of Ultrasound Findings

Maximal systolic and diastolic and time mean BFV were measured in each vessel. Pulsatility index (PI) was calculated according to the following formula: 

PI = (Systolic BFV - Diastolic BFV) / Mean BFV.26

Eighty normal control subjects with the same age and sex distribution as the patients were examined the same way. Their values served as reference and are presented in Table 2.25 The BFV and PI of the affected and unaffected VA and of basilar artery of patients were compared with those of the control subjects. In regard to criteria, a side difference of mean or diastolic BFV exceeding 12 cm/s persisting on repeated measurements was considered potentially abnormal ("weak" abnormal sign). This cutoff value was chosen from the results of the side differences measured in the control subjects (average +2 SD) (Table 2). This sign is considered weak abnormal because hypoplasia may be the cause. Absent flow signal, severely reduced VA BFV, no diastolic flow, bidirectional flow, and a stenosis signal were considered definitively abnormal ("hard" abnormal sign).

Cerebral Angiography

Selective vertebral arteriograms were obtained in 9 patients. Catheterizations were performed by the percutaneous femoral approach; 6F polyethylene catheters and Iopamidol as contrast agent were used. Maximal stenosis was classified into three grades: slight, moderate (more than 50% diameter reduction) (Fig 1), and high grade (more than 80% diameter reduction).

Magnetic Resonance Imaging

Five-millimeter imaging contiguous axial and sagittal sections of the head and neck using T1- and double-echo T2-weighted spin-echo sequences were acquired using a General Electric Signa 1.5-T imaging system. Most patients also had images using a fat-suppression technique (chemical shift imaging method45) (Fig 2c). Stenosis was classified measuring the maximal degree of luminal (flow void) narrowing at the dissection site. No
Table 1. Clinical Findings

<table>
<thead>
<tr>
<th>Pt/ Age, y/Sex</th>
<th>Preceding “Trauma”</th>
<th>Vascular Risk Factors</th>
<th>Warning Symptoms</th>
<th>Presenting Symptoms</th>
<th>Neurological Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/39/M</td>
<td>Sleeping in car with oblique head position</td>
<td>Migraine</td>
<td>Pain 8 d before stroke</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; photophobia, nausea, vomiting</td>
<td>Minimal pyramidal signs right side</td>
</tr>
<tr>
<td>2/38/F</td>
<td>Jogging</td>
<td>Smoking, migraine</td>
<td>None</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting, tinnitus, hypoacusis</td>
<td>V, VII, VIII, IX right, nystagmus, right ataxia</td>
</tr>
<tr>
<td>3/48/F</td>
<td>Blow to the ear</td>
<td>Smoking</td>
<td>None</td>
<td>None</td>
<td>No vertebrobasilar ischemia (aphasia and right hemiparesis)</td>
</tr>
<tr>
<td>4/42/F</td>
<td>Skiing</td>
<td>Hypertension</td>
<td>Pain 3 wk before stroke</td>
<td>Moderate intermittent ipsilateral occipital headache; vertigo, diplopia, dysarthria, nausea, vomiting</td>
<td>IX, XII left, nystagmus, left Horner, right ataxia and hemiparesis</td>
</tr>
<tr>
<td>5/48/F</td>
<td>None</td>
<td>None</td>
<td>Pain 2 wk before stroke</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting</td>
<td>V, IX, XII left, right ataxia and slight hemiparesis</td>
</tr>
<tr>
<td>6/41/M</td>
<td>Cervical manipulation</td>
<td>None</td>
<td>None</td>
<td>Mild persistent ipsilateral neck pain; vertigo, nausea, diplopia, dysphagia</td>
<td>V, IX, X right, nystagmus, right Horner, left sensory hemisindrome, right ataxia (Wallenberg)</td>
</tr>
<tr>
<td>7/46/M</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Severe intermittent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting, dysphagia, diplopia</td>
<td>V, IX left, left Horner, left ataxia, sensory hemisindrome (Wallenberg)</td>
</tr>
<tr>
<td>8/39/M</td>
<td>Cervical discectomy (C-5/C-6); cervical fusion C-6/C-7</td>
<td>None</td>
<td>None</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting, dysphagia</td>
<td>Left hyperreflexia</td>
</tr>
<tr>
<td>9/40/M</td>
<td>Cervical manipulation; head turning while parking the car</td>
<td>None</td>
<td>Neck pain 4 wk before stroke</td>
<td>Moderate persistent midline neck pain; vertigo, nausea, vomiting</td>
<td>Mild right ataxia</td>
</tr>
<tr>
<td>10/44/F</td>
<td>Head turning while parking the car</td>
<td>None</td>
<td>Neck pain 4 wk before stroke</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting, dysarthria, sensory, singultus, diplopia</td>
<td>VII, IX, X left, left Horner, nystagmus, left ataxia, right dissociated sensory hemisindrome (Wallenberg)</td>
</tr>
<tr>
<td>11/67/F</td>
<td>None</td>
<td>Hypertension</td>
<td>Recurrent vertebrobasilar TIA, no pain</td>
<td>No pain; vertigo, dysarthria, oral paresthesia, right hemidysesthesia</td>
<td>Gait ataxia, nystagmus</td>
</tr>
<tr>
<td>12/45/F</td>
<td>None</td>
<td>Migraine</td>
<td>Pain 2 wk before stroke</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; nausea, vomiting, hemianopia, dysarthria, oral paresthesia (vertebrobasilar TIA)</td>
<td>None</td>
</tr>
<tr>
<td>13/56/M</td>
<td>None</td>
<td>None</td>
<td>2 episodes of neck pain and vertigo 3/2 wk ago</td>
<td>Severe persistent ipsilateral neck pain and occipital headache; vertigo, nausea, vomiting, dysarthria</td>
<td>Left ataxia</td>
</tr>
<tr>
<td>14/34/M</td>
<td>Forcefully pulling tight diving suit over head</td>
<td>None</td>
<td>Neck pain 3 wk before stroke</td>
<td>Moderate ipsilateral neck pain; vertigo, nausea, vomiting, dysphagia, paresthesia</td>
<td>VII, IX, X right, right Horner, right ataxia, left dissociated sensory hemisindrome (Wallenberg)</td>
</tr>
</tbody>
</table>

Pt indicates patient; TIA, transient ischemic attack.

Flow void was considered high-grade stenosis or occlusion. Criteria for mural hematoma were thickened vessel wall with hyperintense signal on T1- and T2-weighted images (Fig 2).

Statistical analysis was performed using simple regression analysis and nonparametric tests such as the Wilcoxon signed-rank test and the Mann-Whitney U test.
Nine patients had warning symptoms preceding established neurological deficit: intense neck pain or occipital headache in 8 and vertebralbasilar transient ischemic attack in 2. Twelve patients presented with severe headache, 12 with neurological deficits, and 1 with a history of vertebralbasilar transient ischemic attack. Four patients had a Wallenberg syndrome. Twelve patients were treated with anticoagulants and 1 with aspirin. One patient had no treatment. Seven patients had persistent neurological deficits, 4 of disabling degree. The clinical findings at the initial examination are reported in Table 1.

**Laboratory Findings**

Thirteen patients had echocardiography, which was normal in 11, showed left ventricular enlargement in 1, and mitral valve prolapse in another patient. Eight patients had cerebrospinal fluid analysis, with normal results in all. Blood analyses were normal in all.

**Neuroradiology Findings**

Cerebral computed tomographic scan on the admission day was performed in 12 patients. There was no brain-stem or subarachnoid hemorrhage.

Angiography was performed in 9 patients. It showed high-grade stenosis of the VA in 5 and moderate stenosis in 4 patients (Fig 1). Six had an irregular stenosis, which is considered typical for VAD (Fig 1). One patient had a megadolichoectatic basilar artery, and 1 had VA coiling.

Thirteen patients had MRI of the brain and neck vessels. Our findings included cerebellar and brain-stem infarction in 4 patients, isolated cerebellar infarction (all posterior inferior cerebellar artery territory) in 4, isolated brain-stem infarction in 4, and no signal changes in the brain parenchyma in 1 patient (Table 3).

Magnetic resonance imaging of the neck vessels showed narrowing of the vessel lumen (flow void) at the dissection level in all patients. Minimal or absent flow void, interpreted as high-grade stenosis or occlusion, was seen in 6 patients. A mural hematoma, most often small and crescent, was identified in 12 patients as hyperintense signal on both T1- and T2-weighted images (Fig 2). Because of its small extent, mural hemorrhage could be best identified and distinguished from adjacent fatty tissue with fat-suppression images (Fig 2e). In the 1 patient without visible mural hematoma, the delay from first symptoms to MRI was 6 weeks (Table 3).

Combining MRI and angiographic findings, VAD affected the V-3 segment in 6 patients, the V-3 and V-2 segment in 3, the V-3 and V-4 segment in 3, and the V-2 segment in 2. In summary, the V-3 segment was affected in 12, the V-2 segment in 5, and the V-4 segment in 3 patients (Table 3).

**Ultrasound Findings**

No arterial flow signal could be recorded on Doppler examination of the VA at the atlas loop and intracranially (extracranial/transcranial Doppler) in 5 patients with high-grade stenosis of the V-3 segment and in 1 with moderate stenosis of the V-2 segment. Low BFV when compared with the contralateral side was found in 2 patients with high-grade stenosis of the V-3 segment and in another with high-grade stenosis of the V-2 segment. Compensatory high BFV on the contralateral side was found in 4 patients with a high-grade stenosis of the V-3

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**Table 1. Continued**

<table>
<thead>
<tr>
<th>Follow-up, mo</th>
<th>Treatment</th>
<th>Evolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>3/6</td>
<td>None</td>
<td>Complete remission</td>
</tr>
<tr>
<td>4/12/18</td>
<td>Heparin, coumarin 1 y</td>
<td>Minimal right ataxia</td>
</tr>
<tr>
<td>3/6/12/24</td>
<td>Heparin, coumarin 1 y</td>
<td>Minimal right ataxia</td>
</tr>
<tr>
<td>3/6</td>
<td>Coumarin 7 mo</td>
<td>Complete remission</td>
</tr>
<tr>
<td>3/6</td>
<td>Heparin, coumarin 1 mo (hematuria); aspirin</td>
<td>Persistent diplopia and disabling ataxia</td>
</tr>
<tr>
<td>4/6</td>
<td>Heparin, coumarin 6 mo; aspirin</td>
<td>Persistent disabling diplopia, mild ataxia, cognitive impairment</td>
</tr>
<tr>
<td>1/3/5</td>
<td>Heparin, coumarin 9 mo; aspirin</td>
<td>Complete remission</td>
</tr>
<tr>
<td>3/12</td>
<td>Aspirin</td>
<td>Complete remission</td>
</tr>
<tr>
<td>3/6</td>
<td>Heparin, coumarin</td>
<td>Persistent Horner, mild dysarthria and diplopia, disabling ataxia</td>
</tr>
<tr>
<td>2/6</td>
<td>Heparin, coumarin</td>
<td>Complete remission</td>
</tr>
<tr>
<td>2/6</td>
<td>Heparin, coumarin</td>
<td>Recurrent pain and vertigo, no signs</td>
</tr>
<tr>
<td>2/6</td>
<td>Heparin, coumarin</td>
<td>Minimal ataxia</td>
</tr>
<tr>
<td>2</td>
<td>Heparin, coumarin</td>
<td>Horner, sensory impairment</td>
</tr>
</tbody>
</table>

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**Results**

**Clinical Findings**

All patients had unilateral VAD. One patient had an additional dissection of the ipsilateral ICA (patient 3) and 1 patient of the contralateral ICA (patient 12). There was a history of preceding minor trauma in 9 patients, and 6 had one or more vascular risk factors.
TABLE 2. Doppler Findings in Control Subjects (n=80)

<table>
<thead>
<tr>
<th></th>
<th>ECD/TCD Suboccipital</th>
<th></th>
<th>Duplex Cervical</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BFV</td>
<td>PI</td>
<td>BFV</td>
<td>PI</td>
</tr>
<tr>
<td>Left VA</td>
<td>29±7</td>
<td>0.8±0.18</td>
<td>30±7</td>
<td>1.1±0.27</td>
</tr>
<tr>
<td>Right VA</td>
<td>28±7</td>
<td>0.8±0.15</td>
<td>29±6</td>
<td>1.2±0.3</td>
</tr>
<tr>
<td>Mean side difference</td>
<td>4.2±4.1</td>
<td>0.15±0.15</td>
<td>3.2±3.5</td>
<td>0.21±0.23</td>
</tr>
</tbody>
</table>

Values are mean±SD. Values of blood flow velocity (BFV) are time mean values. ECD/TCD suboccipital indicates extracranial and transcranial Doppler examination via suboccipital approach (V-3 and V-4 segment); duplex cervical, Doppler and B-mode examination at midcervical (intertransverse) segment; PI, pulsatility index; and VA, vertebral artery. Left and right VA were examined in control subjects, dissected and nondissected VA in patients.

segment and in another with a high-grade stenosis of the V-2 segment. The stenotic segment with increased BFV could be detected in only 2 patients, who had moderate stenosis of the V-3 segment (Tables 3 and 4).

Duplex examination of the midcervical intertransverse (V-2) segments in those patients without detectable Doppler signal at the atlas loop demonstrated absent flow signal or high-resistance flow profile without diastolic flow (Fig 3). Thus, the Doppler finding was corroborated as pathological and not due to technical problems. In the 2 patients in whom extracranial Doppler detected the stenosis directly at the atlas loop, duplex helped to distinguish this high BFV from compensatory high BFV due to contralateral stenosis by demonstrating low BFV in the prestenotic segment ipsilaterally.

Two patients had normal ultrasound findings: 1 with a moderate stenosis of the V-2 segment and 1 with a moderate stenosis of the V-3 and V-4 segment (Tables 3 and 4). All 5 patients with increased BFV on one side had contralateral high-grade stenosis or occlusion. No
patient had any signs of atherosclerotic disease in the vertebrobasilar or carotid artery system.

Relying on any (weak and hard) abnormal finding, the diagnostic yields were as follows: extracranial/transcranial Doppler, 71%; duplex, 79%; both together, 86%. When only definitively (hard) abnormal findings such as absent flow signal, severely reduced VA BFV, no diastolic flow, bidirectional flow, and a stenosis signal were considered, the yields were 57%, 50%, and 64%, respectively. A specificity cannot be calculated because there is no pathognomonic ultrasound finding for VAD. There were no false-positive ultrasound findings in the patient group studied. The two patients with normal examinations had only mod-

**Fig 2.** Magnetic resonance images at 1.5 T (patient 6). Sagittal paramedian (a), coronal (b), and axial (c and d) T1-weighted (repetition time, 500 milliseconds; echo time, 18 milliseconds) sections demonstrate crescent hyperintense signal (wall hematoma) (arrows) surrounding the lumen (signal void) of right vertebral artery at the atlas loop (V-3 segment) (a,b,c) and in the subarachnoid (V-4) segment (d). Fat-suppression technique (e) allows definite differentiation from fatty tissue surrounding the vertebral artery and also showing hyperintense signal on T1-weighted sequences.
TABLE 3. Results of Investigations

<table>
<thead>
<tr>
<th>Pt</th>
<th>Delay</th>
<th>Findings</th>
<th>Delay</th>
<th>Vessels</th>
<th>Findings</th>
<th>Brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10 d</td>
<td>Delayed antegrade filling left VA, moderate stenosis at C-3/C-2/ataxis (V-2, V-3), partial thrombosis intracranial segment (V-4), megadolicho basilar artery</td>
<td>Not done</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>2</td>
<td>4 d</td>
<td>Coiling right VA at C-4/C-5 level, high-grade stenosis before coiling, right PICA not filling; hypoplastic left VA, stenosis at C-1/C-2 level (V-3) (old dissection?)</td>
<td>7 d</td>
<td>Narrowed lumen (flow void) right VA at C-5 level, small crescent mural hematoma**, small caliber of left VA</td>
<td>Right cerebellar (PICA) and pontomesencephalic infarction</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>31 d</td>
<td>Long distance irregular high-grade stenosis left VA (hypoplasia?): (flame-shaped occlusion left ICA)</td>
<td>25 d</td>
<td>Crescent mural hematoma* of left VA (and left ICA), no flow void</td>
<td>No infarction in verteobasilar territory (MCA infarction left)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>11 d</td>
<td>Irregular high-grade stenosis left VA at C-1/C-2; left PICA not filling</td>
<td>18 d</td>
<td>Mural hematoma* of left VA from C-2 level to intracranial junction with right VA, no flow void</td>
<td>Infarction of left ventral medulla oblongata</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>24 d</td>
<td>Small left VA ending in PICA (hypoplasia?); irregular moderate stenosis at C-1/C-2, normal right VA</td>
<td>6 wk</td>
<td>Narrowed lumen (flow void) left VA, marked at C-1/C-2 level; no mural hematoma*</td>
<td>Left cerebellar (PICA) and medulla oblongata infarction</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>6 h</td>
<td>Irregular moderate stenosis right VA at C-1 and intracranially, stenosis of right PICA branch</td>
<td>1 d</td>
<td>Mural hematoma* of right VA from C-2 level to intracranial junction with left VA</td>
<td>Right cerebellar (PICA) and medulla oblongata infarction</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>17 d</td>
<td>Irregular moderate stenosis left VA at C-1 and intracranially</td>
<td>16 d</td>
<td>Mural hematoma* of left VA from C-2 level to intracranial junction with right VA</td>
<td>Infarction of left dorsolateral medulla oblongata</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Not done</td>
<td>...</td>
<td>4 d</td>
<td>Mural hematoma* of left VA at C-1/C-2 level, no flow void of left VA</td>
<td>Left cerebellar (PICA) infarction</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Not done</td>
<td>...</td>
<td>28 d</td>
<td>Mural hematoma* at level of C-4 to C-6 vertebral body</td>
<td>Right cerebellar (PICA) infarction</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Not done</td>
<td>...</td>
<td>3 d</td>
<td>Mural hematoma* of left VA at C-1/C-2 level, no flow void of left VA</td>
<td>Left cerebellar (PICA) and medulla oblongata infarction</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Not done</td>
<td>...</td>
<td>10 wk</td>
<td>Narrowed lumen of right VA at C-1/C-2 level; small mural hematoma* (fat suppression)</td>
<td>Left dorsal paramedian pontomesencephalic infarction</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>20 d</td>
<td>Short high-grade stenosis left VA at C-1 (additional moderate stenosis and coiling left ICA in its high cervical segment)</td>
<td>13 d</td>
<td>Mural hematoma* of left VA at C-1/C-2 level, no flow void (mural hematoma of right ICA)</td>
<td>Small left cerebellar (PICA) infarction</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Not done</td>
<td>...</td>
<td>2 d</td>
<td>Mural hematoma* and thickened wall of left VA at C-1/C-2 level, no flow void</td>
<td>Left cerebellar (PICA) infarction</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>20 d</td>
<td>Delayed antegrade filling right VA, short irregular high-grade stenosis right VA at C-1/C-2</td>
<td>21 d</td>
<td>Mural hematoma* and thickened wall of right VA at C-1/C-2 level</td>
<td>Infarction of right dorsolateral medulla oblongata</td>
<td></td>
</tr>
</tbody>
</table>

Pt indicates patient; Delay, interval from first symptoms; MRI, magnetic resonance imaging; ECD, extracranial Doppler sonography (insonation of both vertebral arteries [VA] by lateral suboccipital approach at the atlas segment); TCD, transcranial Doppler sonography (insonation of deep VA and basilar artery [BA] by median suboccipital approach); Duplex, imaging (B-mode) and insonation of both VA in the intertransverse segment (V-2) on both sides; PICA, posterior inferior cerebellar artery; ICA, internal carotid artery; MCA, middle cerebral artery; BFV, blood flow velocity; ipsilateral, dissected vertebral artery; and stump-flow signal, no diastolic flow.

*Hematoma is identified on spin-echo magnetic resonance sequences as a hyperintense, often crescent, signal on T1- and T2-weighted sections.

erate stenosis. All patients with high-grade stenosis or occlusions had hard abnormal findings.

The time mean BFV obtained with suboccipital Doppler insonation of both V-4 segments showed higher values on the nondissected side than on the dissected side (Table 5). Comparison with control subjects indicates that this was due to increased BFV of the nondissected VA. This difference was not significant for
the group as a whole (P=.09, compared with P=.19 in control subjects). However, the correlation between right and left time mean VA BFV in control subjects (r=.64, F=57.6, P=.0001) was lost in patients (r=.18, F=0.45, P=.52). The side differences of time mean BFV in the patients (dissected versus nondissected side) compared with control subjects (left versus right side) was higher (P=.01).

The PI also showed a correlation between right and left in control subjects (r=.43, F=18.6, P=.0001), but there was no correlation between dissected and nondissected side in patients (r=.07, F=0.06, P=.82). The side difference of PI in patients compared with control subjects was significant (P=.03).

Duplex examination of the intertransverse segment (V-2) showed lower BFV of the dissected VA compared with the nondissected side (P=.002) (Table 5). BFV in control subjects was the same in both VAs (P=.3). In control subjects there was a correlation between time mean BFV of left and right VA (r=.42, F=9.6, P=.003).
Vertebral artery dissection in most reported series 
or case reports causes verteobasilar stroke (13 of our 
14 patients) with persistent (7 of 14) and often 
disabling (4 of 14) neurological deficits. VAD may occur 
asymptomatically and be detected by chance.5-7-9 It may 
be present with headache only,1,3,38 or it may cause various 
transient or persistent syndromes of verteobasilar 
ischemia such as tinnitus, amnesia,48 Horner’s syn-
drome, vestibular and cerebellar syndromes,1,7 lateral 
medullary syndrome (50% in the reported series),1,2,7 or 
even locked-in syndrome.7 Headache is not present in 
all symptomatic patients (2 of our 14 patients).1,2,35 The 
reported clinical features may be biased because of 
invasive investigation (angiography) of severe cases with 
stroke only. Benign forms and the most severe cases 
may escape detection when additional investigations are 
not performed.

In at least half of the patients, head and neck pain 
and, more rarely, verteobasilar transient ischemic 
attack precede the onset of stroke as a warning symp-
tom heralding early nonocclusive dissection (9 of our 14 
patients).1,2,39 Usually the pain has an acute sharp 
quality, never previously experienced, and severe or 
unbearable intensity. Mostly it is located in the lateral 
occiput and neck on the dissection side.1,3,7,40 The 
recognition and correct interpretation of these warning 
symptoms may offer the possibility of stroke prevention 
by anticoagulant therapy.

Diagnosis cannot be made reliably on clinical grounds 
alone, especially in the early phase. Embolic or ather-
 thrombotic VA occlusion or intracranial dissection with 
subsequent subarachnoid hemorrhage may cause similar 
pain41,42 and also verteobasilar ischemia. Angiog-
raphy, at present still considered the gold standard for 
definite diagnosis, is not without risk36,37 and has severe 
limitations. Pathognomonic findings such as a double 
lumen or an intimal flap are found only exception-
ally.43-45 A long-distance stenosis may be mistaken for a 
hypoplasia,46 and when the vessel is occluded, the 
etiology of the occlusion may not be recognized.

For this reason a noninvasive screening method is 
desirable that allows the selective use of angiography33 
or, as our patients demonstrate, of MRI. MRI seems to 
become the method of choice to detect even small mural 
hematomas.47,48 While ultrasound proved to be a sensi-
tive and reliable tool in the noninvasive diagnosis of 
carotid artery dissection,14-16 its role in VAD is not 
established.2 There are only a few case reports dealing 
with different ultrasound techniques used in a few 
patients and applying different diagnostic crite-
ria.2,5,18,49-53 The only larger series analyzing ultrasound 
findings in 10 patients verified VAD by angiography, 
which has the above-mentioned limitations.18 Five had 
VA occlusion and 5 had V-1 segment stenosis, both of 
which conditions cannot reliably be differentiated from 
those of atherosclerotic origin without demonstration of 
mural hematoma on MRI. Only limited parameters 
such as high resistance signal and absent flow were 
cluded. The diagnostic yield of transcranial Doppler 
was strikingly low (2 of 10 abnormal results) despite 
location of 13 of 21 dissections in the C-1/C-2 and distal 
VA segment.

In general, not much credit is given to ultrasono-
graphic investigations of the VAs for several reasons:
**TABLE 4. Summary of Ultrasound Findings**

<table>
<thead>
<tr>
<th>Dissection features</th>
<th>ECD/TCD, Suboccipital</th>
<th>Duplex Sonography, Midcervical</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-grade stenosis/occlusion, V-3 (n=7) (Pts 3, 4, 8, 10, 12, 13, 14)</td>
<td>No signal ipsilateral (5) Low BFV* ipsilateral (1) Bidirectional flow (1) High BFV* contralateral (4) Stenosis (0)</td>
<td>No signal ipsilateral (4) Low BFV* ipsilateral (2) No diastolic flow (2) High BFV* contralateral (4) Stenosis (0)</td>
</tr>
<tr>
<td>Moderate stenosis, V-3 (n=4) (Pts 1, 6, 7, 11)</td>
<td>Stenosis* (2) Normal (2)</td>
<td>Low BFV* ipsilateral (2) High pulsatility (1) Normal (1)</td>
</tr>
<tr>
<td>High-grade stenosis, V-2 (n=1) (Pt 2)</td>
<td>Normal</td>
<td>Low BFV* ipsilateral</td>
</tr>
<tr>
<td>Moderate stenosis, V-2 (n=2) (Pts 5, 9)</td>
<td>Low BFV* ipsilateral and high BFV* contralateral (1) Normal (1)</td>
<td>No signal ipsilateral (1) Normal (1)</td>
</tr>
</tbody>
</table>

Diagnostic criteria

<table>
<thead>
<tr>
<th>Diagnostic yield</th>
<th>ECD/TCD</th>
<th>Duplex</th>
<th>Combination of both methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any (&quot;weak&quot; or &quot;hard&quot;) abnormal sign</td>
<td>10/14 (71%)</td>
<td>11/14 (79%)</td>
<td>12/14 (66%)</td>
</tr>
<tr>
<td>Definitively (&quot;hard&quot;) abnormal sign (absent flow signal, bidirectional flow, no diastolic flow, stenosis* signal)</td>
<td>8/14 (57%)</td>
<td>7/14 (50%)</td>
<td>9/14 (64%)</td>
</tr>
</tbody>
</table>

ECD indicates extracranial Doppler; TCD, transcranial Doppler; and Pt, patient.

*Stenosis is defined as blood flow velocity (BFV) > mean + 2 SD of control subjects with broadened frequency spectrum and audible turbulence; high BFV, > mean + 2 SD of control subjects; and low BFV, < mean + 2 SD of control subjects.

First, it is more difficult to detect VA than carotid pathology using Doppler and duplex sonography because of the deep location of the VAs covered by sound-absorbent structures, frequent anatomic variations, extensive collateral anastomotic network at several levels, and the small caliber of the VAs,19,20,54. Second, surgical interventions in the vertebrobasilar system are still a matter of great debate, and vascular anatomy and stroke mechanism suggest the need for medical interventions. Third, the hemodynamics of vertebrobasilar arterial diseases are different from those of the carotid system.27,28,55,56.

Examination of the VA origin has a high failure rate both with Doppler27,28,32,55 and duplex21,25,57,58 sonographic techniques. While atherosclerotic stenosis of the VA is most frequent at its origin,9,59-63 dissection most frequently affects the atlantoaxial (V-3) segment: 12 of our 14 patients and up to 90% of the cases in the literature.1,2,5,18,46,64 This may be explained by the greatest mobility of the VA at this segment, making it vulnerable to trauma.1,2,20,64,65 Sensitivity of Doppler ultrasound in skilled hands to detect occlusive disease of distal VA and basilar arteries seems higher.29,32,66 However, detection of a stenosis of less than 60% to 80% reduction in diameter is usually not possible.

For these reasons we used a combination of extracranial and transcranial pulsed-wave Doppler sonography to examine the atlantoaxial (V-3) and the intracranial (V-4) segments, and we used duplex sonography for examination of the (prelesional) intertransverse (V-2) segment at the C-5/C-6 level.

We could not detect any pathognomonic finding or combination of findings using these ultrasound methods. The dissection itself could not be visualized, and in the case of direct stenosis detection (high BFV) or indirect signs of stenosis (severely reduced or absent ipsilateral VA BFV, increased PI, increased contralateral BFV), no further information about its cause could be obtained.

However, in the rare case when dissection affects the pretransverse (V-1) or low cervical intertransverse (V-2) segment directly accessible to duplex scanning, Touboul et al30 reported quite distinct findings: increased arterial diameter, decreased pulsatility, and intravascular echos in addition to hemodynamic signs of stenosis or occlusion. Hoffmann et al38 reported seven dissections of the V-1 segment, two of them occluded, and seven dissections of the V-2 segment. However, no data on B-mode findings were given.

Nevertheless, the yield to detect any abnormality or side difference in the hemodynamic parameters (BFV, PI) was 86% and was 64% when only definitively (hard) abnormal findings were considered (Table 4). It is obvious that the two ultrasound methods (extracranial and transcranial Doppler examination of the atlantoaxial and intracranial segment, and duplex examination of the intertransverse segment) are complementary to each other (Table 4). Their combined application provided several advantages: higher diagnostic yield, help in differentiating findings from technical failure and hypoplasia, and help in determination of the pathological side in the case of side differences. The value of duplex sonography is first to confirm an absent, low, or high flow signal found with continuous-wave or pulsed-wave Doppler. In the series of Hoffmann et al,18 duplex was abnormal in 8 of 10 patients, showing high resistance signal in 6, no flow in 1, and reversed flow in 1 patient. Second, it helps to differentiate between hypoplasia or aplasia and stenosis or occlusion in the case of a low or absent flow signal, respectively, by measuring the diameter of the vessels.25,50,66,67 Reliable analysis of high cervical V-2 and V-3 segments is not possible. This shortcoming may be overcome in the future by color-coded duplex sonography and higher resolution of the B-mode scan.51,68,69

Discrepancies between ultrasound findings and those of angiography or MRI concerning degree of stenosis...
Table 5. Doppler Findings in Patients (n=14)

<table>
<thead>
<tr>
<th></th>
<th>ECD/TCD Suboccipital</th>
<th>Duplex Cervical</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BFV</td>
<td>PI</td>
</tr>
<tr>
<td>Dissected VA</td>
<td>20±24</td>
<td>0.5±0.4</td>
</tr>
<tr>
<td>Nondissected VA</td>
<td>36±11</td>
<td>0.76±0.14</td>
</tr>
<tr>
<td>Mean side difference</td>
<td>14±28</td>
<td>0.3±0.45</td>
</tr>
</tbody>
</table>

Values are mean±SD. Values of blood flow velocity (BFV) are time mean values. ECD/TCD suboccipital indicates extracranial and transcranial Doppler examination via suboccipital approach (V-3 and V-4 segment); duplex cervical, Doppler and B-mode examination at midcervical (intertransverse) segment; PI, pulsatility index; and VA, vertebral artery. Left and right VA were examined in control subjects, dissected and nondissected VA in patients.

References

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Ultrasound findings in spontaneous extracranial vertebral artery dissection.
M Sturzenegger, H P Mattle, A Rivoir, F Rihs and C Schmid

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