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 intracranial (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 blood flow 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 vertebral artery 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 underrecognized.

The prominent clinical features of VAD are severe ipsilateral neck and occipital head pain and vertebrobasilar ischemia. Distal artery-to-artery embolism from local thrombosis at the stenotic segment12,13 or antegrade 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 2½ 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, anticardiolipin 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 (intracranial) 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 (Diasomics 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 recordings 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 sinuses were measured by a transorbital approach.

**Transcranial Doppler Examination**

The technique for transcranial Doppler recordings has been described in detail by Aaslid.31 A microprocessor-controlled directional pulsed-wave Doppler device operating at 2 MHz was used for all transcranial Doppler ultrasound examinations (TC 2000, EME, Ueberlingen, 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 method24) (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, singulitus, diplopia</td>
<td>VII, IX, X left, left Horner, 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, dysarthria, 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

Statistical analysis was performed using simple regression analysis and nonparametric tests such as the Wilcoxon signed-rank test and the Mann-Whitney U test.
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></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>Minimal right ataxia</td>
</tr>
<tr>
<td>3/6</td>
<td>Heparin, coumarin 6 mo; 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>

Nine patients had warning symptoms preceding established neurological deficit: intense neck pain or occipital headache in 8 and vertebrobasilar transient ischemic attack in 2. Twelve patients presented with severe headache, 12 with neurological deficits, and 1 with a history of vertebrobasilar 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 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

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>Duplex Cervical</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BFV</td>
<td>PI</td>
</tr>
<tr>
<td>Left VA</td>
<td>29±7</td>
<td>0.8±0.18</td>
</tr>
<tr>
<td>Right VA</td>
<td>28±7</td>
<td>0.8±0.15</td>
</tr>
<tr>
<td>Mean side difference</td>
<td>4.2±4.1</td>
<td>0.15±0.15</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 Dopper 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-
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></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>10 d</td>
<td>Delayed antegrade filling left VA, moderate stenosis at C-3/C-2/atlas (V-2, V-3), partial thrombosis intracranial segment (V-4), megaladicho 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 pontomedullar 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 vertebrobasilar 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 colling 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.

cerate 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
<table>
<thead>
<tr>
<th>Delay</th>
<th>ECD/TCD</th>
<th>Duplex</th>
<th>Ultrasound Validation</th>
</tr>
</thead>
</table>
| 10 d  | VA ipsilateral: stenosis
       | VA contralateral: normal
       | VA contralateral: normal | Stenosis directly detected, side difference of BFV |
| 1 d   | VA ipsilateral: normal
       | VA contralateral: normal
       | VA contralateral: high BFV   | Side difference of BFV, indicating stenosis or hypoplasia |
| 27 d  | VA ipsilateral: no signal
       | VA contralateral: high BFV
       | VA contralateral: high BFV   | No signal indicating occlusion or high-grade stenosis, contralateral compensatory high BFV |
| 4 d   | VA ipsilateral: no signal
       | VA contralateral: high BFV
       | VA contralateral: high BFV   | No signal indicating occlusion or high-grade stenosis, contralateral compensatory high BFV |
| 18 d  | VA ipsilateral: low BFV
       | VA contralateral: high BFV
       | VA contralateral: normal    | Side difference of BFV indicating stenosis or hypoplasia |
| 9 h   | VA ipsilateral: normal
       | VA contralateral: normal
       | Both sides normal, symmetric BFV | Pathology missed |
| 18 d  | VA ipsilateral: stenosis
       | VA contralateral: normal
       | VA contralateral: normal    | Stenosis directly detected, prestenotic low BFV and high pulsatility |
| 6 d   | VA ipsilateral: no signal
       | VA contralateral: normal
       | VA contralateral: normal    | No signal indicating occlusion, high-grade stenosis |
| 5 d   | VA ipsilateral: normal
       | VA contralateral: normal
       | VA contralateral: normal    | Pathology missed |
| 3 d   | VA ipsilateral: no signal
       | VA contralateral: normal
       | VA contralateral: normal    | No signal indicating occlusion, high-grade stenosis |
| 6 wk  | VA ipsilateral: normal
       | VA contralateral: normal
       | VA contralateral: normal    | High pulsatility indicating distal stenosis |
| 14 d  | VA ipsilateral: low BFV
       | VA contralateral: high BFV
       | VA contralateral: high BFV   | Side difference of BFV, indicating stenosis or hypoplasia |
| 4 d   | VA ipsilateral: low bidirectional (oscillating) BFV
       | VA contralateral: high BFV
       | VA contralateral: high BFV   | No signal indicating occlusion, high-grade stenosis |
| 20 d  | VA ipsilateral: no signal
       | VA contralateral: normal
       | VA contralateral: normal    | No signal indicating occlusion, high-grade stenosis |

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 and case reports causes vertebrobasilar stroke (13 of our 14 patients)\textsuperscript{1,2,4-7,35} with persistent (7 of 14) and often disabling (4 of 14) neurological deficits. VAD may occur asymptomatically and be detected by chance,\textsuperscript{5,7,9} it may present with headache only,\textsuperscript{1,3,30} or it may cause various transient or persistent syndromes of vertebrobasilar ischemia such as tinnitus, amnesia,\textsuperscript{38} Horner’s syndrome, vestibular and cerebellar syndromes,\textsuperscript{1,7} lateral medullary syndrome (50% in the reported series),\textsuperscript{1,2,7} or even locked-in syndrome.\textsuperscript{7} Headache is not present in all symptomatic patients (2 of our 14 patients).\textsuperscript{1,2,25} 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, vertebrobasilar transient ischemic attack precede the onset of stroke as a warning symptom heralding early nonocclusive dissection (9 of our 14 patients).\textsuperscript{1,2,29} 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.\textsuperscript{1,3,40} 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 atherothrombotic VA occlusion or intracranial dissection with subsequent subarachnoid hemorrhage may cause similar pain\textsuperscript{41,42} and also vertebrobasilar ischemia. Angiography, at present still considered the gold standard for definite diagnosis, is not without risk\textsuperscript{36,37} and has severe limitations. Pathognomonic findings such as a double lumen or an intimal flap are found only exceptionally.\textsuperscript{43-45} A long-distance stenosis may be mistaken for a hypoplasia,\textsuperscript{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 angiography\textsuperscript{33} or, as our patients demonstrate, of MRI. MRI seems to become the method of choice to detect even small mural hematomas.\textsuperscript{47,48} While ultrasound proved to be a sensitive and reliable tool in the noninvasive diagnosis of carotid artery dissection,\textsuperscript{14-16} its role in VAD is not established.\textsuperscript{2} There are only a few case reports dealing with different ultrasound techniques used in a few patients and applying different diagnostic criteria.\textsuperscript{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.\textsuperscript{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 included. 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 ultrasonographic investigations of the VAs for several reasons:

**Fig 3.** Duplex ultrasound findings of a patient with high-grade stenosis of the left V-3 vertebral artery segment. The intertransverse segment between the transverse processes of C-6 and C-5 vertebral bodies (arrows) are insonnated on both sides (L indicates left side; r, right side). There is a high resistance flow profile with reversed flow in the early diastole and low blood flow velocities on the left side (top). On the unaffected right side (bottom) the flow profile is normal, but the blood flow velocities are high.

This correlation was lost in patients ($r=.01$, $F=.001$, $P=.97$). Side differences of BFV were significantly higher in patients than in control subjects ($P=.0001$).

In the patients with measurable BFV, there was an increased PI of the affected VA compared with the nonaffected side ($P=.05$) or to control subjects, indicating distal high resistance. Correlation between left and right side in control subjects for PI was high ($r=.47$, $F=18.9$, $P=.0001$). In patients the correlation was lost ($r=.03$, $F=0.01$, $P=.92$).

**Discussion**

Vertebral artery dissection is considered to be rare.\textsuperscript{3,5,7,35} Our findings in 14 patients observed within 2½ years suggest that its incidence may be underestimated.\textsuperscript{2,3} One reason is that the clinical features, although quite typical, are not familiar to many physicians: in not one of our 14 patients was the diagnosis of VAD suspected by the referring physicians. Patients are referred because of headache or vertebrobasilar ischemia. Another reason might be the common opinion that diagnosis of VAD is possible only by angiography, a procedure that is not readily performed because of its risks.\textsuperscript{36,37}
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 (“weak” or “hard”) abnormal sign</td>
<td>10/14 (71%)</td>
<td>11/14 (79%)</td>
<td>12/14 (66%)</td>
</tr>
<tr>
<td>Definitively (“hard”) 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 pathologies using Doppler and duplex sonography because of the deep location of the VAs covered by sound-absorbing structures, frequent anatomic variations, extensive collateral anastomotic network at several levels, and the small caliber of the VAs. 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 supplementary 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, 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. 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.

Discrepancies between ultrasound findings and those of angiography or MRI concerning degree of stenosis...
(eg, no Doppler signal with a moderate stenosis) may be explained by the delay between the examinations or by difficult grading of the stenosis in the imaging procedures due to very short or irregular stenosis or vessel superposition (Fig 1). Exact determination of percent diameter reduction of the vessel lumina (flow void) using axial magnetic resonance sections proved difficult as well. There may be several reasons for this, such as the tiny dimensions of the VA segments compared with the voxel size, partial volume averaging when the vessel is imaged obliquely, or the highly complicated effects of flowing blood on signal in spin-echo images (Fig 2c and 2d). Normal vertebrobasilar ultrasound findings do not exclude VAD that causes only moderate stenosis. In the case of suggestive symptoms and signs, a normal ultrasound result should not delay anticoagulant treatment with heparin after brain-stem hemorrhage and subarachnoid hemorrhage have been excluded with computed tomographic scan and lumbar puncture when appropriate. We did not observe any adverse effect of anticoagulants in our series and are not aware of any reported deterioration due to anticoagulant treatment in patients with extracranial VAD. However, it must be mentioned that anticoagulation is not of proven benefit, i.e., there is no prospective randomized study available. Dissection is a dynamic process that may cause rapid lumen obliteration but also early recanalization or fluctuating or slowly progressive mural hematoma. This means that hemodynamic compromise and, as a result, ultrasound findings may greatly vary. If ultrasound examination is unrevealing, diagnosis can be confirmed by MRI (Fig 2). MRI and magnetic resonance angiography may become the method of choice for diagnosis of VAD. Angiography may still be needed in certain cases. When intracranial dissection is suspected, eg, in a patient with a stiff neck due to subarachnoid hemorrhage, the causative pseudoaneurysm may still be better delineated with conventional angiography than with MRI. Experience with magnetic resonance angiography is still too limited to allow any conclusion. Sequential ultrasound examination can show recanalization or normalization of blood flow and thus proves helpful in determining the duration of anticoagulant treatment.

In conclusion, combined Doppler and duplex ultrasound examination is a sensitive screening technique in VAD but cannot replace diagnostic confirmation by imaging procedures such as MRI.

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