Dissection of the Extracranial Vertebral Artery: Clinical Findings and Early Noninvasive Diagnosis in 24 Patients

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ABSTRACT

Dissection of cervical arteries causes ischemic stroke in young adults. This reports the clinical, ultrasonographic, and neuroradiological findings in 24 patients with 28 vertebral artery dissections in the neck (4 occurring bilaterally). In 20 patients (83%), the dissection was temporally related to trauma. No patients had an underlying vascular disease, for example, atherosclerosis or fibromuscular dysplasia. In all, the major initial manifestation was pain in the occipital or neck region. The next most common symptoms were vertigo and nausea (in 17 patients). Clinical manifestations were vertebrobasilar transient ischemic attack (TIA) (5 patients: in 2 patients vestibulocerebellar TIA, in 1 patient visual TIA, in 1 patient motor TIA, and in 1 patient brain stem TIA with perioral paresthesia), cerebellar infarction (10 patients, in 4 patients bilateral), brainstem infarction (5 patients), posterior cerebral artery territory infarction (1 patient), and multiple vertebrobasilar ischemic lesions (3 patients). Typical angiographic findings were irregular narrowing of the vessel lumen or a tapering stenosis with distal occlusion. Magnetic resonance imaging showed a thickened vessel wall with hematoma signal at the site of the dissection. Duplex color-flow imaging was valuable for the early diagnosis of extracranial vertebral artery dissection and for follow-up examinations. The distal V1- and the proximal V2-segment (at the level of C6 vertebra) was the most frequent localization of dissections (in 43%). The outcome was favorable except for 2 patients with basilar artery occlusion. Embolism to the basilar artery may be avoided by early administration of anticoagulants.

Key words: Vertebral artery, dissection, duplex scanning, ultrasonics, noninvasive diagnosis.

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Dissection of extracranial vertebral arteries can be either spontaneous or associated with minor or significant neck trauma, for example, sport activities and chiropractic manipulation.[1,2] Spontaneous dissection can be related to an underlying disorder such as fibromuscular dysplasia, hypertension, atherosclerosis, migraine, arteritis, or Marfan's disease.[3-5]

Before the introduction of newer imaging techniques, angiography was long considered mandatory for diagnosis. Noninvasive techniques–magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and duplex color flow imaging–are becoming more reliable, facilitating the diagnosis. The value of MRI, MRA, and duplex ultrasonography in detection of cervical artery dissection has been described.[6-8] There are also reports of clinical and angiographic findings in vertebral artery dissection.[9] In the last few years, reports on ultrasonographic findings in cervical artery dissection have appeared increasingly in the literature.[10]

Vertebral artery dissection is one of the most common causes of posterior circulation ischemia in young patients without risk factors for atherosclerotic arterial disease. This reports clinical, ultrasonographic, and neuroradiologic findings in 24 patients with 28 dissections (4 bilateral) of the extracranial vertebral artery. Additionally, the aim of the study is to evaluate the contribution of the sonographic methods to the early diagnosis of vertebral artery dissection and in follow-up examinations. Appropriate management is discussed.

Subjects and Methods

Over a 4-year period, 24 patients (10 men and 14 women) aged 18 to 60 years (mean age 39.1 years) with extracranial vertebral artery dissection, referred routinely to our ultrasound laboratory, were followed (Table 1).

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Table 1. Clinical and Sonographic Findings in 24 Patients With Extracranial Vertebral Artery Dissection (28 Dissections)

Case-Patient Initials	Age	Sex	Risk Factors	*Activity (Neck Trauma)	Neck/Head Pain	Main Clinical Symptoms	Ischemic Signs CCT/MRI	Localization of Extracranial Sonographic Findings
1-BK	45	F	None	Dubious	+++	Nausea, vertigo, nystagmus	Vertebrobasilar TIA	Origin V0 and proximal V1 segment
2-MO	42	M	Hypertension	Breast stroke swimming	+	Nausea, vomitus, ataxia	Bilateral cerebellar infarction	Bilateral distal V1 and proximal V2 segments
3-BJ	57	M	Hypertension	Collapse, neck injury	+	Ataxia, nystagmus	Right cerebellar infarction	Distal V1 and proximal V2 segments
4-HA	52	F	Hormone therapy, hypercholesterine	Gymnastics	++	Left homonymous miahemianopia	Right occipital infarction	Distal V1 and proximal V2 segments V1 – Kinking
5-OH	60	M	Hypertension, migraine, smoking	Dubious	+	Vertigo, left V, VIII, IX Palsy	Brain stem ischemia	Origin V0 and proximal V1 segment
6-MM	25	M	None	Ski accident with neck injury	++	Vertigo, right hemiparesis	Vertebrobasilar TIA	Distal V1 and proximal V2 segments
7-SC	43	F	Obesity	Bicycle accident	+++	Nystagmus, right hemi-hypesthesia	Brain stem ischemia	Origin V0 and proximal V1 segment
8-BK	53	M	Hypertension	Dubious	++	Vertigo, blurred vision, ataxia	Vertebrobasilar TIA	Origin V0 and proximal V1 segment
9-JP	39	M	None	Car accident, polytrauma, neck injury	+	Tetraparesis, brain stem symptoms	Brain stem infarction, cerebral contusion	Distal V1 and proximal V2 segments
10-EP	25	F	Oral contraceptives	Car accident, neck trauma	++	Vertigo, ataxia, vomitus	Right cerebellar infarction	Distal V1 and proximal V2 segments
11-NI	32	F	None	Chiropractic manipulation	+++	Vertigo, ataxia, vomitus, nystagmus	Bilateral cerebellar infarction	Bilateral distal V1 and proximal V2 segments
12-HA	39	M	None	Chiropractic manipulation	+++	Ataxia, vertigo, nausea	Left cerebellar infarction	Distal V1 and proximal V2 segments
13-MG	18	F	None	Trauma, chiropractic manipulation	+++	Ataxia, locked-in syndrome, tetraparesis	Brain stem infarction	V3 segment
14-VS	25	M	None	Jogging, neck injury	++	Perioral paresthesia, vertigo	Vertebrobasilar TIA	Distal V2 segment
15-SG	43	F	Left-handed person, migraine	Dubious	++	Vertigo, ataxia, vomitus, nystagmus	Left cerebellar infarction	Origin V0 and proximal V1 segment
16-MT	28	M	None	Snow board accident, neck trauma	++	Dysarthria, right hemiparesis, hemianopia	Brain stem and occipital infarction	Distal V1 and proximal V2 segments
17-EM	36	F	None	Bike accident	+++	Left Wallenberg syndrome	Cerebellar and brain stem infarction	Distal V1 and proximal V2 segments
18-KE	55	F	Smoking	Physical activity	+++	Vertigo, right hemi-hypesthesia, hemianopia	Left occipital and thalamus infarction	Distal V1 and proximal V2 segments
19-TC	52	F	None	Car accident	+++	Vertigo, left hemiparesis,	Brain stem infarction	Origin V0 and proximal V1 segment
20-WF	37	M	None	Physical activity	++	Vertigo, ataxia, nystagmus	Bilateral cerebellar infarction	Bilateral distal V1 and proximal V2 segments
21-KN	38	F	Migraine	Neck injury	+++	Vertigo, blurred vision, nystagmus	Left cerebellar infarction	Distal V1 and proximal V2 segments
22-FM	19	M	None	Disco “head banging”	++	Vertigo, ataxia, nystagmus	Bilateral cerebellar infarction	Bilateral distal V1 and proximal V2 segments
23-RJ	39	F	Migraine	Dental treatment	+++	Vertigo, ataxia, nystagmus	Left cerebellar infarction	V3 segment
24-AN	37	F	Migraine	Neck injury	+++	Vertigo, ataxia, nystagmus	Vertebrobasilar TIA	Distal V2 segment

CCT = CT scan (cranial computed tomography), MRI = magnetic resonance imaging, TIA = transient ischemic attack.
Plus signs indicate intensity of neck/head pain: slight (+), moderate (++), severe (+++).
*Activity = physical activity (or neck trauma) before the onset of the acute symptoms.

In all patients, the onset of symptoms was abrupt. Three men and 1 woman had bilateral vertebral dissection. In 20 patients (83%), the dissection was temporally related to minor or significant trauma. In 6, the dissection occurred after physical effort or sports activities such as jogging, swimming, and gymnastics. In 9, neck trauma due to car or bike accidents and skiing or snow board accidents preceded the acute ischemic symptoms in the vertebrobasilar territory. In 3, dissection occurred during or immediately after chiropractic manipulation. In 1 patient, prolonged dental treatment, that is, root canal therapy, preceded by 1 day the onset of a cerebellar infarction due to a dissection (patient number 23). One 19-year-old patient (no 22) was admitted to the hospital with a bilateral cerebellar infarction caused by a bilateral vertebral artery dissection after he had spent several hours in a disco dancing with a continuous rhythmical movement of the head ("head banging"). In only 4 patients was the temporal relation between dissection and potential trauma questionable. However, 1 of these patients with left vertebral artery dissection was an active, left-handed tennis player (Fig 1).

Risk factors for stroke were present in 9 patients: hypertension (n = 4), hypercholesterinemia (n = 1), oral contraceptive or hormone therapy (n = 2), smoking (n = 2), and obesity (n = 1). Atherosclerotic lesions in the carotid arteries were diagnosed in 1 patient; however, the plaques were small and nonstenotic. Five patients had migraine headache disorder. None had an underlying vascular disorder such as fibromuscular dysplasia, Marfan's disease, or arteritis.

A complete neurovascular ultrasonographic study, including continuous Doppler examination, conventional transcranial Doppler (TCD Medasonics, 2 MHz transducer, Medilab GmbH, Würzburg, Germany, and DWL Elektronische Systeme GmbH, Sipplingen, Germany) and extra- and transcranial color-coded duplex ultrasonography, was carried out on all patients. Color-coded duplex ultrasonography was performed during the first 2 years of the study using the Acuson 128 XP 10 system (Acuson, Mountain View, CA), equipped with a linear-array transducer with a 7.0 MHz imaging frequency and 5 MHz pulsed-wave Doppler frequency and with a sector transducer with 2 MHz imaging frequency for the transcranial imaging. During the second 2 years of the study, transcranial examination was performed with an Acuson Seqouia^{TM} 512 ultrasound system (Siemens Medical Solutions, Nürnberg, Germany). The vertebral arteries were imaged in both transverse and longitudinal planes from origin V0 up to the V3 segment at the atlas loop as previously described.[11,12] The ultrasonographic results were recorded by a Sony Mavigraph UP-5000 P color video printer and on a magnetic optical disc (Sony Deutschland GmbH, Köln, Germany). An MRI of the brain was performed on all patients, digital subtraction angiography (DSA) on 15 patients, and MRA on 19 patients.

The inclusion criteria of this study were based on a combination of the typical history, severe initial head and neck pain, clinical symptoms related to the vertebrobasilar system, ultrasonographic study, and any evidence of dissection with DSA, MRI, or MRA. The purpose of the study was to evaluate the sonographic findings and to describe them in relation to findings obtained through other imaging methods in the context of the patients' typical history.

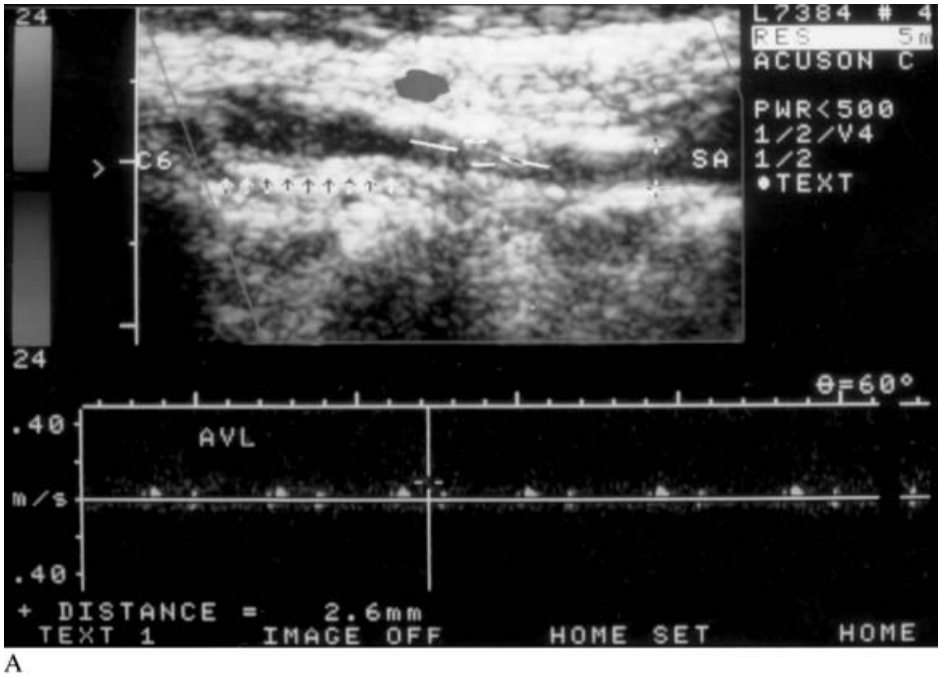
Results

In all patients, the major initial manifestation was pain in the occipital or neck region, which developed immediately or within several hours after the presumed traumatic insult that provoked the dissection. The most commonly reported symptoms were vertigo and nausea. Additionally, we observed a variety of symptoms related to cerebellar, brainstem, and occipital lobe ischemia; cerebellar symptoms were predominant in 11 patients (45.8%).

The time interval between the onset of the local symptoms (pain in the occipital or neck region) and onset of focal neurological symptoms or stroke varied between a few hours and as much as 4 months. In 2 patients, the onset of neurological symptoms arose immediately after chiropractic "treatment." In 6 patients, the focal neurological symptoms occurred within the first 12 hours after the presumed traumatic event. In the majority of the patients (n = 12), the onset of stroke was within the first 14 days. In the 4 other patients, the onset of neurological deficits lagged by 1 to 4 months.

MRA (n = 19) and DSA (n = 15) confirmed the ultrasonographic diagnosis in all patients. In 2 patients with reproducible sonographic findings, angiography was not performed. Irregular narrowing and/or tapered stenosis progressing to occlusion were the most frequent findings. Two pseudoaneurysms were observed in 1 patient, in another patient, kinks in the V1 segment were detected. In 6 patients, angiography revealed occlusion of the vertebral artery at the origin.

CT or MRI showed unilateral cerebellar infarction in 6 patients (with an occlusion of the ipsilateral posterior inferior cerebellar artery (PICA) in 4 of them), bilateral cerebellar infarction in 4 patients, posterior cerebral artery territory infarction in 1 patient, and brain stem infarction in 5 patients. Of these last 5, 3 showed involvement of the proximal brain stem circulation territory, 1 of the middle,



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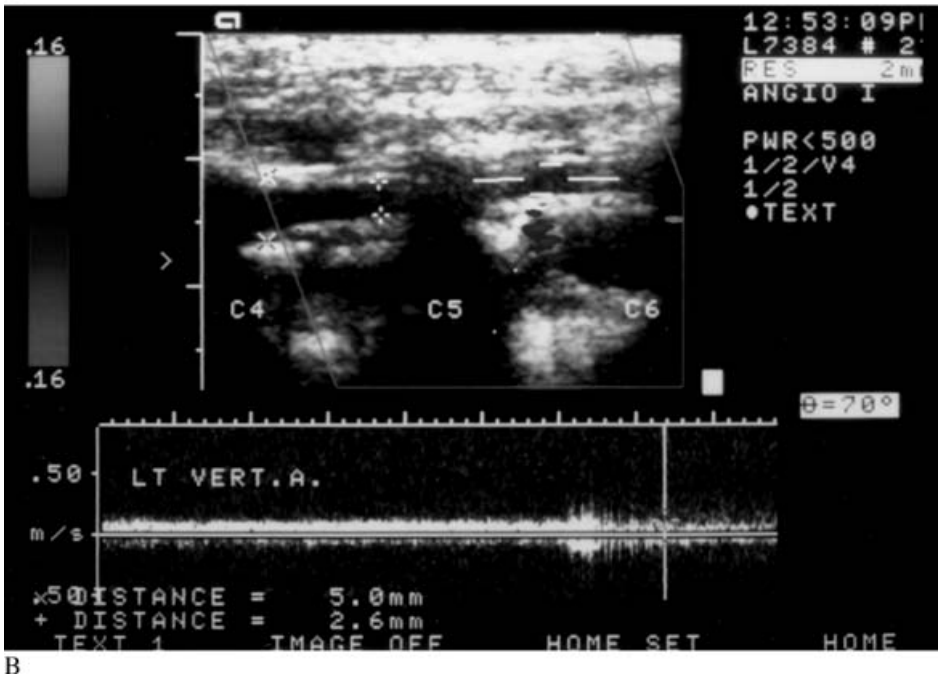

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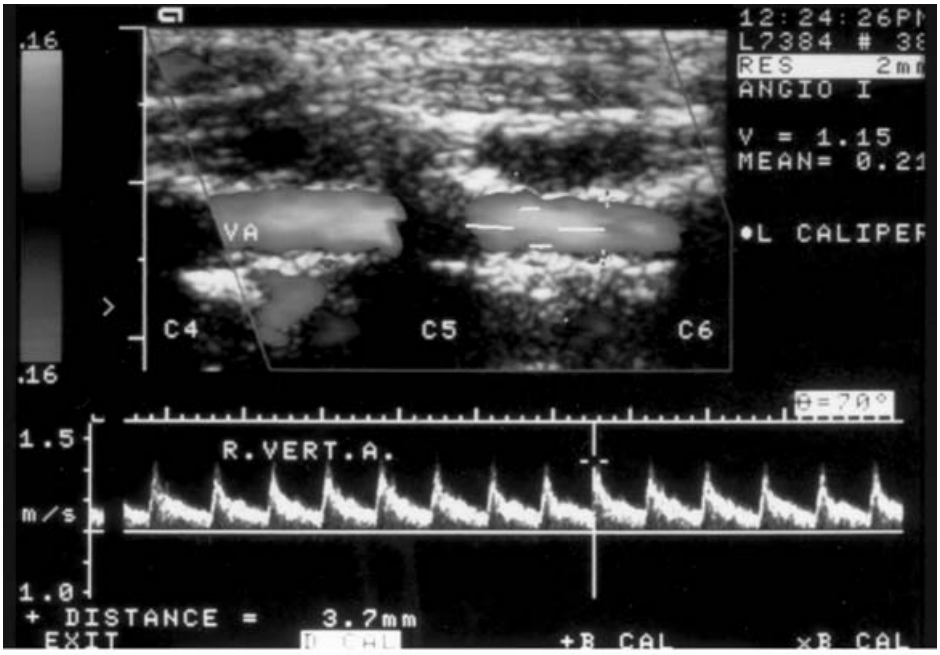
Fig 1. Findings in a 43-year-old woman with a dissection of the left vertebral artery. (A temporal relation between the dissection and a minor trauma was not certain. This patient was an active left-handed tennis player and suffered from migraine.) (A) Ultrasonographic image of the V0/V1 segment of the left vertebral artery (1). Diameter of the vessel at the origin from the subclavian artery (2, SA) is 2.6 mm. In the distal V1 segment, at the entrance of the vertebral artery into the transverse foramen of the C6 vertebra (3), a dilation of the lumen (4) with irregular vessel wall is shown. No blood flow can be detected in the lumen of the artery. (B) View of the V2 segment of the left vertebral artery. The longitudinal course of the artery is interrupted by transverse processes of the C4, C5, and C6 vertebrae causing acoustic shadowing. Diameter of the artery is 2.6 mm. No color-coding in the lumen is a very typical sign of a recent occlusion located more proximally (C4, C5, C6 indicate transverse processes).

and 1 of the distal brain stem territories (classification according to Caplan scheme).[13] In 3 patients, multiple vertebrobasilar ischemic lesions were present. The variable distribution of the ischemic lesions strongly suggests an arterio-arterial source of the embolism. Interestingly, where a unilateral ischemic lesion was in the



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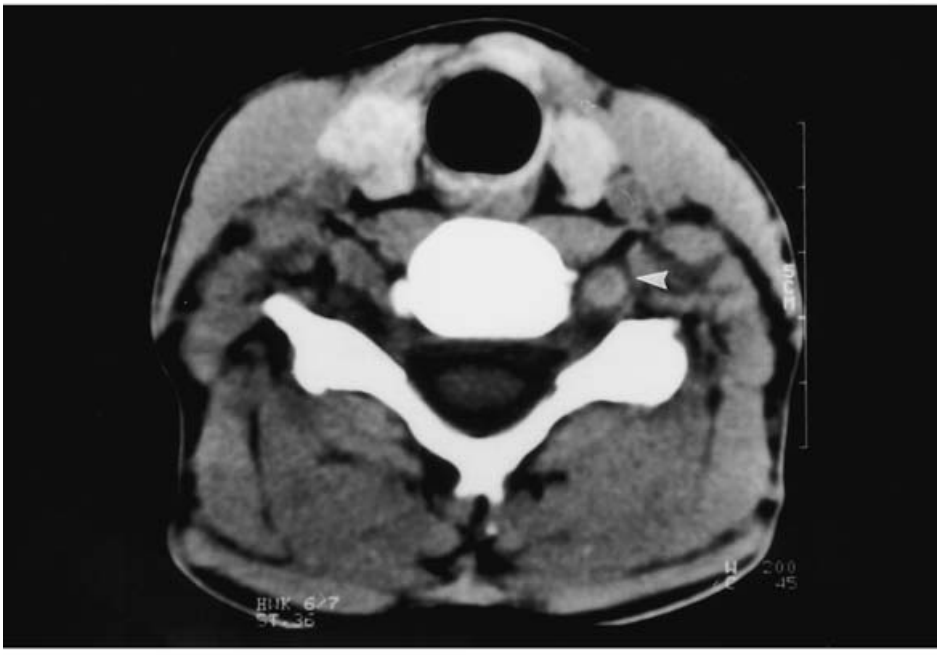

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Fig 1. (C) View of the V2 segment of the right vertebral artery (with the use of the same transducer position as on the left side) showing a normal contralateral vertebral artery in the midcervical course. Diameter of the artery is 3.7 mm. (D) Admission CT-scan at the level of C6/C7 vertebrae shows the signal intense enlarged lumen of the left vertebral artery in the area of the dissection.

posterior circulation, the potential source of embolism in all cases emanated from the ipsilateral extracranial vertebral artery. CT scan and MRI were normal in 5 patients (Table 2).

TCD results were abnormal in 2 patients with basilar artery occlusion (nos 9 and 13). Using suboccipital insonation, we found a high-resistance flow pattern with decreased diastolic flow or even without a diastolic flow component at a depth of 65 mm, indicating an obstruction more distally.

In 18 patients, a dissection was primarily detected by duplex color-flow imaging. Six patients, previously diagnosed by angiography, were referred to our ultrasound laboratory for follow-up color-flow examination.

Table 2. MRI Findings in 24 Patients With Extracranial Vertebral Artery Dissection (28 Dissections)

	n
Unilateral cerebellar infarction	6
Bilateral cerebellar infarction	4
Brain stem infarction	5
Posterior cerebral artery territory infarction	1
Multiple vertebrobasilar ischemic lesions	3
Normal findings	5
Total	24

Table 3. Localization of Extracranial Sonographic Findings in 24 Patients With Extracranial Vertebral Artery Dissection (28 Dissections)

	n
Origin V0 and proximal V1 segments	6 (21.4%)
Distal V1 and proximal V2 segments	18 (64.3%)
Distal V2 segment	2 (7.15%)
V3 segment	2 (7.15%)
Total	28 (100%)

Subgroup at the level of the C6 vertebra: n = 12 (43%).

Ultrasonographic abnormalities in the extracranial course of the vertebral artery were observed in all patients (Table 3). The findings varied depending on mechanism and on site of dissection.[14,15]

In our patients, the distal V1 segment and the proximal V2 segment was the most frequent localization of dissections (18 dissections) (Fig 1). In a subgroup of 12 cases (43%), the vertebral artery was dissected at the point of entry into the transverse foramen of the C6 vertebra (Fig 1A). Intramural hematoma, irregularities of the vessel wall, dissecting membrane, tapering stenosis, true and false lumen, intravascular echoes, localized arterial dilation with decreased pulsatility, and pseudoaneurysm formation could be visualized in this region.

In 2 patients, the artery was affected in the distal V2 segment. At the level of the C3 vertebra, a pseudoaneurysm formation could be imaged in 1 patient.

In 6 patients, the dissection was found at the origin or in the proximal V1 segment. In 4 patients, the vertebral artery was occluded, whereby rostral to the occlusion, a vessel lumen without blood flow was found.

In 2 patients, the dissection was located at the atlas loop. The ultrasound diagnosis was based on indirect signs: within normally configurated arteries in V1 and V2 segments, a high-resistance flow pattern was detected (Fig 2).

In follow-up examinations, the abnormal ultrasonographic findings regressed in 20 dissections (71.4%). Two vertebral artery occlusions recanalized.

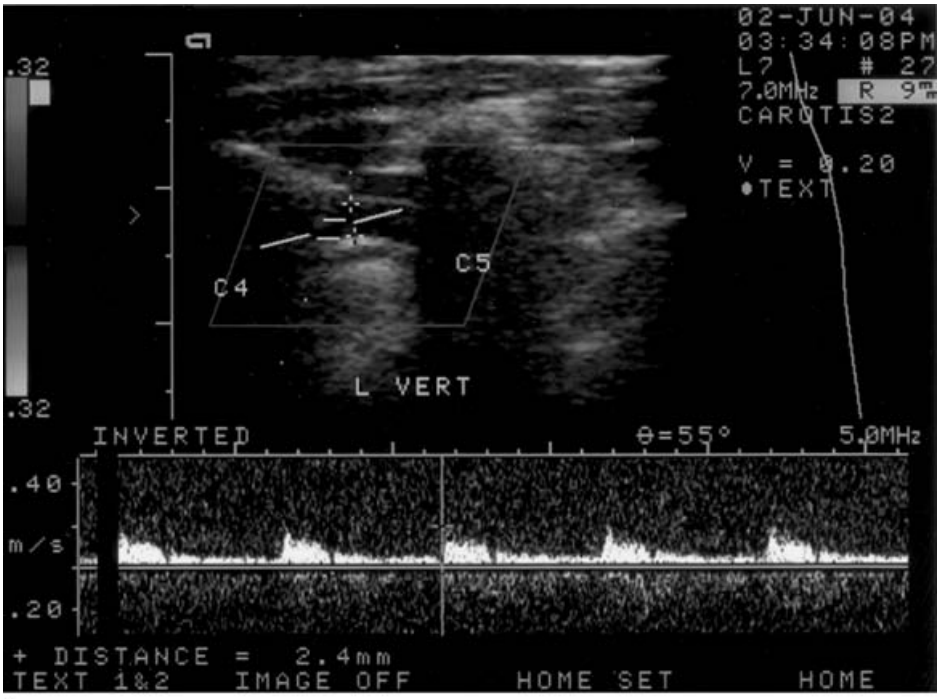
The outcome was favorable except for 2 patients with basilar artery occlusion. Patient number 9 had severe neurological and neuropsychological deficits due to additional brain trauma caused by a car accident. In patient number 13, sudden deterioration resulting in a locked-in syndrome occurred after a basilar artery occlusion, probably due to intra-arterial embolization from the dissected region. Unfortunately, in this patient, local intra-arterial lysis failed. Sixteen patients were discharged without neurological findings, whereas 6 had slight neurological deficits.

Discussion

The mean age (39.1 years) of our patients confirms that dissection tends to affect younger individuals. Only a few of them had important risk factors. Atherosclerosis or other underlying vascular disorders were etiologically unimportant. A history of migraine, reported in other studies, may have played a role only in 2 cases of spontaneous dissection. A study concerning carotid artery redundancy concludes that internal carotid artery redundancy and dissection are significantly associated.[16] Accordingly, the relationship between vertebral kinks and dissection is possible in patient number 4. In the majority of our patients (83%), however, dissection of the extracranial artery was related to minor or significant trauma or chiropractic neck manipulation.[17]

Early recognition of vertebral artery dissection is important, particularly in patients with only local symptoms, or minor ischemic symptoms, for which the administration of either antiplatelet agents or anticoagulation can be considered to potentially reduce the risk of first-time–or recurrent and potentially more severe–posterior circulation infarction.[18] Even the first clinical symptoms can already be suggestive of a dissection. In all our patients, the first symptoms (occipital headache, neck pain, and vertigo) were very abrupt. The time interval between the onset of these local symptoms and onset of focal neurological symptoms or stroke was very variable (between several hours and up to 4 months); therefore, an exact statistical analysis based on these data is not possible. Nevertheless, it is clinically quite crucial to know the window of opportunity for recognizing local symptoms caused by dissection and the time in which intervention may be possible to prevent the development of cerebral ischemic symptoms.

The subsequent symptoms can present slowly or suddenly according to the extent of injury to the vessel: Local thrombosis at the site of a dissected intima may cause transient vertebrobasilar symptoms due to microembolism or even major complications such as locked-in syndrome on



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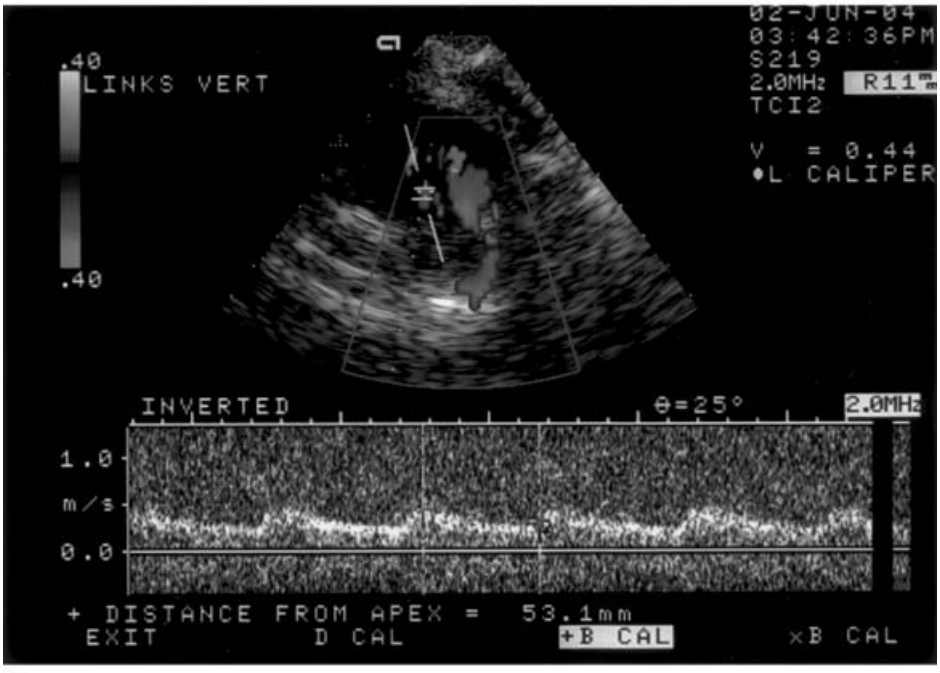

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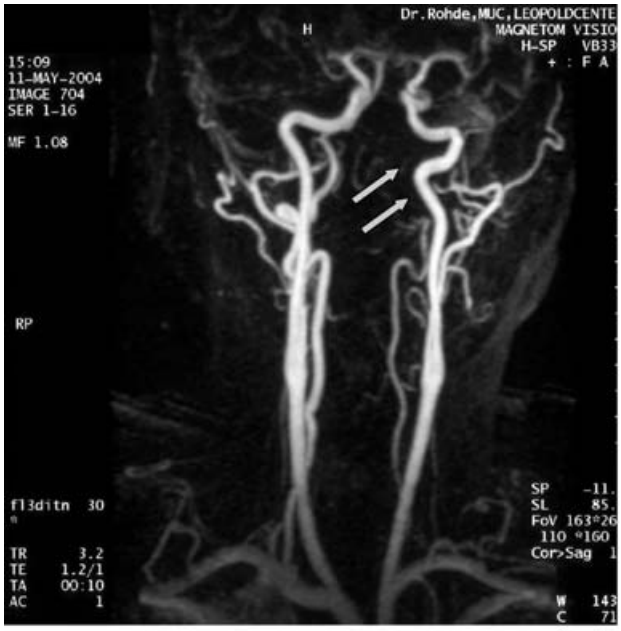
Fig 2. Findings in a 39-year-old woman with a dissection of the left vertebral artery in a V3 segment, which occurred 1 day after prolonged dental treatment. (A) High-resistance flow pattern in the V2 segment of the left vertebral artery, recorded between the C4 and C6 cervical vertebrae. The decreased end diastolic blood flow velocity indicates indirectly an obstruction more distally. (C4, C5 indicate acoustic shadowing caused by transverse processes). (B) Color-coded transcranial duplex sonography using suboccipital insonation shows a good flow in the right vertebral artery and in the basilar artery. The flow signal in the left vertebral artery is difficult to obtain, the blood flow velocity is decreased, pulsatility is lower as recorded extracranially—due to reduced blood flow distally from the flow obstruction—located in V3 segment.

account of occlusion of the basilar artery. To avoid this serious complication, an early administration of anticoagulants or antiplatelet agents is very important. In 1 patient (no 13) who was admitted to our hospital several days after the onset of the first symptoms as a consequence of chiropractic manipulation, appropriate treatment could



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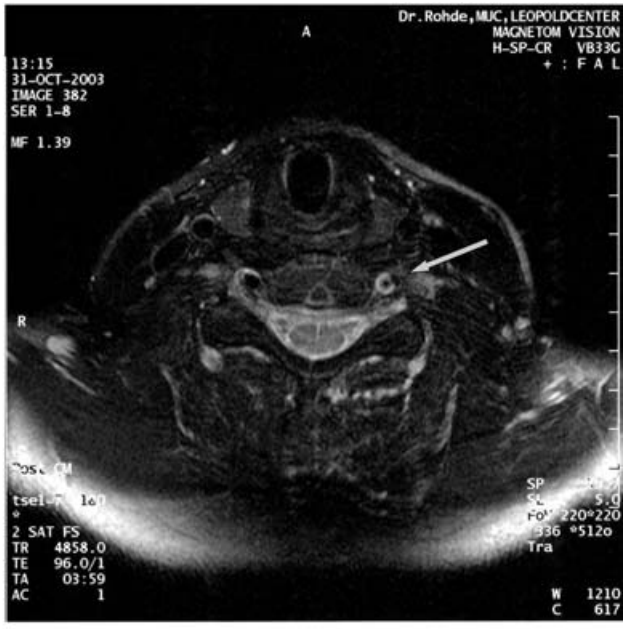

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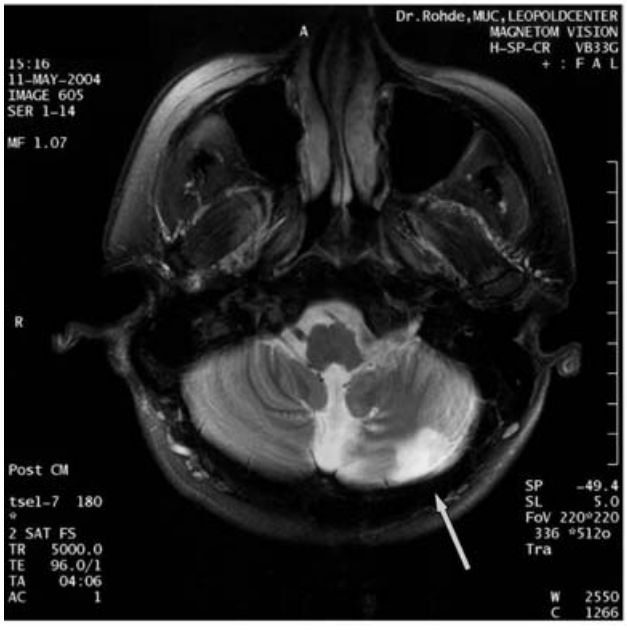

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Fig 2. (C) Magnetic resonance angiography findings of a reduced flow in the V3/V4 segment of the left vertebral artery (arrow). (D) The intramural hematoma at the site of the dissection can be easily detected using magnetic resonance imaging with a fat-suppression technique. (E) Left cerebellar infarction caused by flow obstruction in the left vertebral artery. (MRI, MRA findings: Courtesy Dr. Rohde/Dr. Sollfrank, Neuroradiology, Munich, with permission).

not be started in the early stage of the disease. This led to sudden deterioration, resulting in brain stem infarction with locked-in syndrome. In another patient (no 9) with brain stem infarction admitted to the hospital after a severe car accident, the administration of anticoagulants was not possible due to subdural hematoma and multiple traumas. Various reports underline the benefit of anticoagulants in the early management of a dissection.[19] In our study, all patients who were treated with anticoagulants had a favorable outcome. However, up until now there is still no randomized trial evidence comparing anticoagulation with antiplatelet therapy in patients with dissection.[20]

Recently developed noninvasive techniques allow complications as described in patient number 13 to be avoided. The first step should be (depending, of course, on local availability) a neurovascular examination using

TCD and duplex ultrasonography.[21] We have described the diagnostic potential of the color-flow duplex ultrasonography in the evaluation of extracranial vertebral artery dissection in another study.[15,22] Where a dissection of the imagable segments (V0 to distal V2 segments) of the vertebral artery has occurred, a direct visualization of the vessel pathology can confirm the diagnosis. As in the previous study, the most significant and most frequent pathological findings could be observed in the distal V1 segment, at the entrance of the vertebral artery into the transverse foramen of the C6 vertebra–in 43% of dissections (Fig 1A). At the junction of V1 and V2–where the "free" prevertebral course of the artery continues in the canal of the transverse foramina of C6 to C2 vertebrae–the artery is possibly exposed to the greatest mechanical injury.[23] This is of particular significance, because especially this region can be diagnosed very easily using duplex sonography. If through such previous pinpointing, the examiner is aware of the possible location of pathological changes in this region, the chances of false-negative findings, that is, of overlooking a dissection, can be reduced. In contrast, in a dissection of V3 and/or V4 segments, the vessel lesion cannot be visualized directly, owing to methodical problems–here the typical indirect signs with high-resistance flow characteristics diagnosed in the midcervical course indicate an obstruction located more distally.[24,25] However, these examinations require an experienced examiner.

The next diagnostic procedures should be MRI and/or the dynamic CT scan. In a study including 15 patients, Zuber showed that MR imaging can directly reveal an intramural hematoma whose signal intensity varies with the age of the hematoma.[26] Sturzenegger underlined the advantages of the MRI fat-suppression technique in detecting a thickened vessel with intramural hematoma.[19]

Although MRA still has some limitations concerning the estimation of the degree of stenosis and is not sensitive enough to recognize subtle changes in the vessel wall (eg, in fibromuscular disease), it has, as a screening procedure in combination with other noninvasive techniques, an essential place in the diagnostic spectrum.[27,28] Contrast angiography may no longer be routinely necessary for confirming dissection and should be performed only in selected cases. Nevertheless, based on our follow-up study of 24 patients, it is not possible to make an exact statistical analysis of the advantages of the noninvasive techniques we described over angiography. Only a multicenter study could throw more light on this subject.

In conclusion, using noninvasive techniques, the diagnosis of a dissection of the extracranial vertebral artery poses no problems, providing the examiner is familiar with this disease. The early diagnosis of dissection is important so that the appropriate treatment can be started and complications, resulting from embolism to the basilar artery, can be avoided.

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