Imaging in the Diagnosis and Follow-up Evaluation of Vertebral Artery Dissection

Chien-Jung Lu, MD, Yu Sun, MD, Jiann-Shing Jeng, MD, Kou-Mou Huang, MD, Bao-Show Hwang, Win-Hwan Lin, Rong-Chi Chen, MD, Ping-Keung Yip, MD

The purpose of this report is to discuss the value of ultrasonographic examination in the diagnosis and follow-up evaluation of vertebral artery dissection. We collected data on 8 patients with 11 pathologic vessels: 9 were affected intracranially and 6 were affected extracranially. Four vessels were affected in both intracranial and extracranial segments. Extracranial color-flow duplex sonography could detect abnormalities in every extracranial vertebral artery dissection. Most abnormal findings were non-specific, including severely reduced flow, absence of flow, and absence of diastolic flow. A specific finding (intramural hematoma) was noted in one artery. Abnormal transcranial color-coded sonographic findings included absence of flow, reduced velocity and reversed flow direction. Three intracranial dissecting arteries showed normal findings on transcranial color-coded sonography. Ultrasonographic follow-up study revealed evidence of improvement; this was noted almost exclusively in the extracranial segments of the vertebral artery but infrequently in the intracranial segment. Extracranial color-flow duplex sonography is sensitive in the detection of extracranial vertebral artery dissection, both in initial diagnosis and in follow-up evaluation. Key words: Dissection, vertebral artery; Vertebral artery; Extracranial color-flow duplex sonography; Transcranial color-coded sonography.

ABBREVIATIONS
VAD, Vertebral artery dissection; SAH, Subarachnoid hemorrhage; MR, Magnetic resonance; ECDS, Extracranial color-flow duplex sonography; TCCS, Transcranial color-coded sonography; PICA, Posterior inferior cerebellar artery; SCA, Superior cerebellar artery

Vertebral artery dissection has been considered an important cause of stroke in the posterior circulation, particularly in the young adults. It may result in a variety of clinical manifestations. VAD can be asymptomatic and noted incidentally. It can occur spontaneously or after trauma. It may affect the vertebral artery intracranially or extracranially. Occipital pain and brainstem ischemic stroke are the most common findings in VAD. When VAD occurs intracranially, it can also result in SAH. Clinical diagnosis of VAD was at times difficult and usually had to be confirmed by angiography. At present, MR imaging and MR angiography are often applied in patients with VAD and have been accepted as alternative methods in diagnosis. Ultrasonographic examination has been reported to be another convenient and noninvasive method in diagnosing VAD. It can provide both anatomic and hemodynamic informa-
tion about the intracranial and extracranial segments of the vertebral artery. In this report, we present our experience in the diagnosis and follow-up evaluation of VAD with special reference to the ultrasonographic findings. On the basis of these findings, the role of the ultrasonographic examination is discussed.

SUBJECTS AND METHODS

Between 1997 and 1998 12 patients with young stroke in the posterior circulation or with occlusion of the vertebral artery had been admitted to neurologic and neurosurgical wards and were suspected to have VAD. Their cases all were reviewed retrospectively regarding clinical manifestations and neuroradiologic and sonographic findings. Angiography was the standard examination for the diagnosis. If the patient did not accept angiography, MR imaging or MR angiography could be used as the alternative to prove diagnosis. We identified eight patients (three women and five men; mean age, 37 years; range, 24 to 53 years) with VAD to analyze their ultrasonographic findings.

Clinical Assessment

The clinical assessment included recording the clinical presentation, evaluation of treatment, and observation of outcome. The following elements needed special inquiry in taking history: (1) vascular risk factors,19 including hypertension, diabetes mellitus, hyperlipidemia, smoking, and use of oral contraceptives; patients with these vascular risk factors had to be evaluated carefully, and patients with a previous history of atherosclerosis in the carotid artery were excluded from this study; (2) previous head or neck trauma and cervical chiropractic manipulation; (3) warning symptoms, including occipital headache, neck pain, and vertebrobasilar transient ischemic attack; (4) presenting symptoms, and (5) type of stroke syndrome (SAH or brainstem or cerebellar infarct).

Neuroradiology

Arterial pathology was evaluated by the location of lesion, severity of stenosis, and regularity of the lumen. The location of the lesion was defined according to standard criteria.20,21 Maximal stenosis was classified into three grades: (1) mild stenosis (less than 50% diameter reduction of the lumen), (2) severe stenosis (greater than 50% reduction), and (3) occlusion.

Cerebral Angiography

Selective cerebral angiograms were obtained in six patients. The lesions were classified into (1) those located in the extracranial segment, (2) those located in the intracranial segment, (3) those located in both the intracranial and the extracranial segments, and (4) those with nonvisualization of the vessel. The specific angiographic findings of VAD included the following: (1) intimal flap, (2) double lumen, and (3) pseudoaneurysm. The string sign (tapered stenosis with distal occlusion) was considered a suggestive finding of VAD.

MR Imaging

Head MR images were obtained in six patients. MR imaging was used mainly for demonstrating the lesion of infarct. Parenchymal lesions with decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images were defined as those with ischemic change. When the patient did not accept angiography, head MR imaging could be used as an alternative method to demonstrate the pathology of the vessel. Loss of flow void was considered to represent high-grade stenosis or occlusion of vessel. The specific MR imaging finding was intramural hematoma in thickened vessel wall with abnormal signal intensity on T1-weighted and T2-weighted images.

MR Angiography

MR angiograms were obtained in four patients. The degree of stenosis was classified the same as that mentioned previously. Nonvisualization of the vessel on MR angiography was considered to represent high-grade stenosis or occlusion. As in MR imaging, MR angiography could be used as an alternative diagnostic method if the patient did not accept angiography. Only demonstration of pseudoaneurysm was considered to be a specific criterion for the diagnosis of VAD.

Ultrasonographic Examination

All patients underwent ECDS and seven had TCCS examinations. Four patients had ECDS follow-up evaluation (from 1 month to 24 months), and five had TCCS follow-up evaluation (from 1 month to 24 months).

ECDS

ECDS was performed using either an Aloka SSD-3000 system (Aloka, Wallingford, CT) or a Diasonics
Clinical Findings

One patient had a history of preceding cervical chiropractic manipulation, but no one had had major trauma. None of the patients had vascular risk factors. Headache was noted in six patients, among whom three had headache preceding the onset of the neurologic deficits. Stroke syndrome could be identified in seven patients: Wallenberg syndrome in two, cerebellar infarct in four (in the territory of the PICA in two and of the SCA in two), and SAH in one. In one patient the stroke syndrome was undetermined because no corresponding lesion was noted in cerebral hemisphere or brainstem. Three patients received antiplatelet agents, one with anticoagulant and two with initial anticoagulant then antiplatelet therapy. Two patients had no treatment: one had SAH (bilateral intracranial VAD) and the other had a left cerebellar stroke that progressed rapidly to brainstem failure. Seven patients improved gradually with no recurrent stroke.

Neuroradiology Findings

The results of neuroradiologic investigations are listed in Table 1. Selective angiograms were obtained in six patients: three patients had unilateral VAD and the other three had bilateral VAD. The extracranial segment was affected in five arteries and intracranial segment in four. An intimal flap was noted in one artery, pseudoaneurysm in three arteries, string sign in one artery, and tapered stenosis at V-4 (intracranial portion from dural entry to junction with contralateral vertebral artery) in two arteries. Tapered occlusion was noted at V-1 (from the origin of the artery to the entry of the transverse foramen) in one artery and at C2 (from the C2 transverse process to the foramen magnum) in one artery. MR imaging of the head was performed in six patients. Intramural hematoma was noted in one artery (at V-4). The stroke lesion was located in the lateral medulla in one patient, in the lateral medulla and cerebellum in one patient, and in the cerebellum in three patients (one in the PICA and two in the SCA). MR angiography was performed in four patients. Six pathologic lesions of the vertebral arteries were noted: pseudoaneurysm in one, severe stenosis with pseudoaneurysm in one, and nonvisualization of the vessel in four.
Combining the MR imaging, MR angiography, and angiography findings, 11 pathologic arteries were identified. Five patients had unilateral VAD, and three had bilateral VAD. No associated carotid artery dissection was noted. Nine vertebral arteries were affected intracranially and six were affected extracranially. Four arteries were affected in both the intracranial and the extracranial segments.

### Ultrasonographic Findings

The ultrasonographic findings are listed in Table 1. All patients’ bilateral extracranial vertebral arteries were examined. Intramural hematoma was noted in one artery whose Doppler signal remained normal. Absence of flow signal was recorded on ECDS examination of four vertebral arteries whose neuroradiologic findings showed total occlusion, occlusion at V-1, occlusion at V-4, and intramural hematoma at V-4 (by MR imaging), respectively. Severe reduced blood flow was noted in three vessels. Absence of diastolic flow was noted in one artery. Normal ECDS findings were noted in two arteries with intracranial VAD. ECDS showed definitely abnormal findings in every extracranial VAD. The positive rate was 100% (6 of 6 vessels) in detecting abnormality of the extracranial VAD using ECDS. However, specific findings were infrequent (1 of 6 vessels).

TCCS examination was performed in 7 patients with 10 affected vessels. Absence of flow was noted in four intracranial arteries, reduced velocity in one vessel, and reversed flow direction in one vessel. Eight intracranial dissecting vessels were examined. Normal TCCS findings were noted in three cases of intracranial VAD and in one case of extracranial VAD. The positive rate was 63% (5 of 8 vessels) in detecting flow abnormality of intracranial VAD using TCCS.

ECDS follow-up evaluation was performed in four patients with seven VADs (Table 2). Hematoma was resolved in one vessel. Improvement in vertebral flow was noted in three arteries with extracranial VAD. These findings were observed only in patients

### Table 1: Results of Investigations in 8 Patients with VAD

<table>
<thead>
<tr>
<th>No./Age/Sex</th>
<th>Angiography</th>
<th>MR Angiography</th>
<th>MR Imaging</th>
<th>ECDS</th>
<th>TCCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/24/M</td>
<td>None</td>
<td>LVA: Nonvisualization</td>
<td>Left cerebellar ischemia, SCA</td>
<td>LVA: Absence of flow</td>
<td>LVA: Absence of flow</td>
</tr>
<tr>
<td>2/41/F</td>
<td>LVA: Occlusion at V-0; RVA: Mild stenosis at V-1 and V-2</td>
<td>None</td>
<td>Left cerebellar ischemia, PICA</td>
<td>LVA: Absence of flow; RVA: Intramural hematoma</td>
<td>LVA: Reversed flow; RVA: Within normal limits</td>
</tr>
<tr>
<td>3/32/M</td>
<td>LVA: String sing, occlusion at V-3; RVA: Tapered occlusion at V-1</td>
<td>LVA: Nonvisualization; RVA: Nonvisualization</td>
<td>Right lateral medullary ischemia</td>
<td>LVA: Severely reduced flow; RVA: Absence of flow</td>
<td>LVA: Absence of flow</td>
</tr>
<tr>
<td>4/28/M</td>
<td>LVA: Tapered occlusion at V-3</td>
<td>LVA: Nonvisualization</td>
<td>Left medullary and cerebellar ischemia</td>
<td>LVA: Absence of diastolic flow</td>
<td>LVA: Absence of flow</td>
</tr>
<tr>
<td>5/41/M</td>
<td>LVA: Mild stenosis irregular surface with pseudoaneurysm at V-4; RVA: Intimal flap and severe tapered stenosis with pseudoaneurysm at V-4</td>
<td>LVA: Pseudoaneurysm at V-4; RVA: Severe stenosis with pseudoaneurysm at V-4</td>
<td>None*</td>
<td>LVA: Within normal limits; RVA: Severely reduced flow</td>
<td>LVA: Within normal limits; RVA: Reduced velocity</td>
</tr>
<tr>
<td>6/53/F</td>
<td>LVA: Irregular stenosis with pseudoaneurysm at V-4</td>
<td>None</td>
<td>No corresponding lesion</td>
<td>LVA: Within normal limits</td>
<td>LVA: Within normal limits</td>
</tr>
<tr>
<td>7/28/M</td>
<td>LVA: Tapered stenosis at V-4</td>
<td>None</td>
<td>None†</td>
<td>LVA: Severely reduced flow</td>
<td>None</td>
</tr>
<tr>
<td>8/50/F</td>
<td>None</td>
<td>None</td>
<td>Right cerebellar ischemia, SCA (RVA: mural hematoma at V-4)</td>
<td>RVA: Absence of flow</td>
<td>RVA: Within normal limits</td>
</tr>
</tbody>
</table>

*Brain CT showed subarachnoid hemorrhage. †Brain CT showed left cerebellar infarct, PICA territory.

LVA, Left vertebral artery; RVA, Right vertebral artery; V-0, origin of vertebral artery; V-1, from the origin of the artery to entry of transverse foramen; V-3, from the C-2 transverse process to the foramen magnum; V-4, intracranial portion from dural entry to junction with contralateral vertebral artery.
with longer intervals of follow-up after the ictus (i.e., at least 3 months in this series). ECDS was sensitive in detecting the resolution of extracranial VAD (4 of 5 vessels). TCCS follow-up evaluation was performed in five patients with eight VADs. Most vessels had no significant change. Mild improvement of flow was noted only in one vessel.  

Case Reports

Patient 2 (Fig. 1)

This patient was a 41 year old woman who had had cervical chiropractic manipulation about 1 month before the onset of symptoms. She had occipital headache and accepted head MR imaging examination. MR imaging disclosed cerebellar infarct in the territory of the left PICA. Angiography showed irregular stenosis in the right vertebral artery and total occlusion on the left side. VAD was suspected. On the ECDS examination, an intramural hematoma was found in the V-1 segment. The presence of VAD was confirmed. The follow-up ECDS examination showed that the intramural hematoma was resolved 6 months later.

Patient 4 (Fig. 2)

The patient was a 28 year old man who had left Wallenberg syndrome. Head MR imaging demonstrated infarct located in the left lateral medulla and the left cerebellum (in the territory of the left PICA). The left intracranial vertebral artery was not visualized in MR angiography. Angiography showed occlusion of the left vertebral artery at the V-3 segment. ECDS did not reveal any significant abnormal findings on B-mode imaging. However, Doppler signals revealed absence of diastolic flow in the left extracranial vertebral artery. TCCS detected normal Doppler signals in the right intracranial vertebral artery and basilar artery but no flow in the left intracranial vertebral artery.

DISCUSSION

Although a so-called “typical” clinical presentation could suggest the occurrence of VAD, this diagnosis sometimes could not be made reliably by clinical symptoms alone. It requires confirmation by neuro-radiologic findings. Angiography has been the gold standard for the diagnosis for a long time, but it has three major shortcomings.

First, the findings are not always specific. Intimal flap and double lumen are considered to be specific angiographic findings but are found only infrequently. The presence of a pseudoaneurysm is also a pathognomonic finding. A less specific finding is long segment of irregular stenosis, or string sign. Occlusion of the vertebral artery is considered the least specific abnormal finding.

Second, angiography is an invasive procedure. Many clinicians hesitate to submit patients to this examination even though obvious neurologic symptoms are present. It seems to be necessary to find a convenient method to make an early diagnosis, because early recognition of VAD may offer the possibility of stroke prevention by administration of an anticoagulant.23
**Figure 1** Angiography and ECDS of the vertebral artery, patient 2. A, Angiography of the right vertebral artery shows irregular stenosis in the extracranial segment (arrow), including V-1 and V-2. B, A hypoechoic lesion within dilated vessel wall at V-1 segment (arrow). An intramural hematoma was considered. C, Six month follow-up examination. The intramural hematoma has disappeared (arrow). ECDS clearly demonstrated the resolution of the intramural hematoma.

**Figure 2** Patient 4. A, Head MR imaging using T2-weighting shows increased signal intensity in the left lateral medulla (arrowhead) and the left cerebellum (arrow). B, Angiography of the left vertebral artery shows occlusion at V-3 segment (arrow). C, ECDS demonstrates high resistance flow at the V-1 segment. This case illustrates the most frequent manifestations of VAD.
Third, it is not convenient for frequent follow-up evaluation. At the present time, with the advances in noninvasive examinations, MR imaging and MR angiography can also offer valuable information in diagnosing VAD.\textsuperscript{12} The MR imaging features of VAD have been reported in several previous studies.\textsuperscript{9–12} An eccentric signal void surrounded by a semilunar area of abnormal signal intensity in a dilated vessel wall is considered characteristic for the diagnosis of VAD.\textsuperscript{12} A circular region of increased signal intensity surrounding the vessel, thrombus within the lumen without luminal dilation, and nonvisualization of the vessel were considered less specific for the diagnosis.\textsuperscript{10} The validity of MR angiography also has been studied.\textsuperscript{2,24} Double lumen and pseudoaneurysm have been reported to be specific MR angiographic findings for diagnosis.\textsuperscript{24} Poor visualization or nonvisualization of the vessel was less specific. The diagnosis cannot be made on the basis of a small lumen because of physiologic variations in the diameter of the vertebral artery. Although MR imaging and MR angiography are reliable and noninvasive methods in the diagnosis and follow-up evaluation of VAD, they are expensive.

Touboul and coworkers\textsuperscript{16} reported the extracranial duplex sonographic findings of VAD, including association of local dilatation of artery diameter with hemodynamic signs of stenosis or occlusion and decreased pulsatility and increased intravascular echoes in an enlarged segment. Hoffmann and colleagues\textsuperscript{17} and Sturzenegger and coauthors\textsuperscript{18} also had proposed some definitely abnormal Doppler sonographic findings. However, none of these definitely abnormal features was pathognomonic of VAD. Bartels and Flugel\textsuperscript{13} reported the ECDS findings of extracranial VAD. Typical features were irregular stenosis, dissecting membrane with true and false lumen, localized increase in diameter of the artery, pseudoaneurysm, intramural hematoma, and tapered stenosis with distal occlusion. These authors concluded that extracranial VAD could be diagnosed by ECDS imaging.

As a matter of fact, the ultrasonographic features of VAD are determined by two major factors: the site of dissection and the severity of stenosis caused by VAD. If VAD affects the V-1 or proximal V-2 segment, ECDS examination may show one or more specific findings. As the specific findings appear, the efficacy of ECDS in the diagnosis is no less than that of angiography, MR imaging, or MR angiography. However, if the VAD affects the V-3 or V-4 segment, it is more difficult to obtain these specific findings even when Doppler signal shows evidence of definite abnormality. Because most patients whom Bartels and Flugel collected had extracranial VAD, it may be easier to find specific abnormalities by ECDS. However, in our experience, so-called “specific findings” were infrequently noted. ECDS is useful in diagnosis of VAD, but maybe not so good as Bartels and Flugel reported.\textsuperscript{13}

ECDS was sensitive but rarely specific. In our report, ECDS could show definitely abnormal findings in every extracranial VAD. However, these ECDS findings were often nonspecific, including absence of flow, severely reduced flow, and absence of diastolic flow. Specific abnormal findings were noted infrequently. Only one, intramural hematoma, was noted in one artery (patient 2). To detect specific findings, we suggest as thorough an examination of the vertebral artery as possible, not just the routine single image usually obtained as part of the carotid artery evaluation. The vertebral artery should be examined segment by segment, from its origin to the most distal part that can be imaged, to see the typical findings that have been reported.\textsuperscript{13,16–18} Among these findings, intramural hematoma, intimal flap, pseudoaneurysm, and double lumen could be considered to be specific findings for the diagnosis of VAD. Irregular stenosis and tapered stenosis are less specific. However, VAD can still be diagnosed if the stenotic artery has an eccentric residual lumen with focal or successive segmental increase in diameter.\textsuperscript{25}

ECDS is also useful in follow-up evaluation. As shown for patient 2, ECDS clearly demonstrated the resolution of the intramural hematoma. In patient 1, head MR imaging, MR angiography, ECDS, and TCCS showed occlusion of the left vertebral artery. This patient had no vascular risk factors, coagulation disorders, or other pathologic vascular condition. VAD was suspected. He had follow-up ultrasonography and neck MR imaging 2 years later. The ECDS showed recanalization of the previously occlusive vessel but neck MR imaging still showed occlusion of the left vertebral artery. VAD was diagnosed when ECDS demonstrated resolution of the occlusive vessel.

TCCS could not give findings that were as informative as those of ECDS. In some previously published reports, Doppler ultrasonography was found to be a sensitive method for the detection of pathologic conditions of the vertebral artery.\textsuperscript{15,26} However, most of our TCCS findings were also nonspecific. Definitively abnormal findings included absence of flow, reduced velocity, and reversed direction of flow. So far, TCCS can provide hemodynamic information but no specific findings for the diagnosis of intracranial VAD. In patient 5, whose angiography showed irregular stenosis with pseudoaneurysm in
bilateral intracranial vertebral arteries, TCCS examination could not show the existence of these pseudoaneurysms. In this patient with bilateral VADs, Doppler signal showed reduced velocity in the intracranial right vertebral artery and normal findings on the left side. As shown in this patient, TCCS was less sensitive.

On the basis of the aforementioned results and review of literature, we found that ECDS is sensitive in the initial diagnosis to detect the flow abnormalities in patients with extracranial VAD, but specific findings are infrequent. TCCS also is valuable but is less sensitive in diagnosing intracranial VAD. On long-term follow-up evaluation, ultrasonographic examinations have shown to be good and convenient enough to demonstrate recanalization, which is considered to be indirect evidence of arterial dissection. ECDS examination is reliable in showing resolution of extracranial VAD. Moreover, the evolution of sonographic changes is more valuable in the extracranial ECDS examination than in the TCCS. In conclusion, for definite diagnosis of VAD, angiography and MR imaging may be more suitable for initial diagnosis because specific findings of ECDS were found infrequently. However, ultrasonographic examination is necessary in uncertain or difficult case from the beginning because it is convenient and sensitive in follow-up evaluation.

REFERENCES