Diffusion-Weighted Magnetic Resonance Imaging in Symptomatic Vertebrobasilar Atherosclerosis and Dissection

Sebastian Koch, MD; Murtaza Amir, MD; Alejandro A. Rabinstein, MD; Yolanda Reyes-Iglesias, MD; Jose G. Romano, MD; Alejandro Forteza, MD

Background: Acute multiple brain infarction (AMBI) pattern on diffusion-weighted imaging (DWI) is associated with arterial and cardiac sources of embolism. The DWI characteristics of patients with stroke due to vertebrobasilar arterial dissection and atherosclerotic disease have not been reported in detail.

Objective: To describe the DWI stroke patterns in patients with posterior circulation occlusive disease to determine mechanisms of ischemia.

Design: Retrospective analysis of infarct patterns in patients with symptomatic vertebrobasilar disease.

Setting: Large community-based teaching hospital.

Patients: Patients admitted with stroke due to vertebrobasilar disease were identified retrospectively. Patients were included if DWI was obtained within 7 days of symptom onset.

Main Outcome Measure: Infarct patterns were analyzed according to established templates of vascular territories.

Results: Eleven patients with vertebral dissection and 39 patients with atherothrombosis were identified. An AMBI pattern was present in 8 (72%) of 11 patients with arterial dissections and 25 (64%) of 39 patients with atherosclerotic disease ($P = .48$). Distal embolism to the terminal branches of the basilar artery occurred with equal frequency in both groups and was found in half of all cases. Isolated thalamic infarction did not occur. Pontine infarction was noted in 2 (18%) of 11 patients with dissections and 18 (46%) of 39 patients with atherosclerosis ($P = .09$). Cerebellar border zone involvement was found in 14 (36%) of 39 patients with atherosclerosis and 4 (37%) of 11 patients with dissections ($P = .6$).

Conclusions: Large arterial disease is frequently associated with AMBI in the posterior circulation. The incidence of AMBI was comparable to that reported in the anterior circulation. This DWI study supports the importance of embolism as the main mechanism of infarction in patients with vertebrobasilar occlusive disease. On the basis of our experience, large-vessel vertebrobasilar disease rarely causes isolated small-vessel thalamic infarction.

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the study if DWI was obtained within 7 days from symptom onset. We reviewed the medical records and radiologic data (catheter cerebral angiogram, neck and brain magnetic resonance angiogram, and neck MRI with fat-suppressed images) of all eligible patients to confirm the diagnosis of vertebrobasilar atherothrombosis or dissection. Patients were diagnosed as having vertebrobasilar dissection on clinical grounds (young age, presence of neck pain, relative absence of cardiovascular risk factors) and typical imaging findings, including angiographic appearance (ie, presence of tapered stenosis or occlusion, string and pearl sign, dissecting aneurysms and double lumen, or intramural hematoma on axial MRI). Lesion location at the V3 segment was also considered suggestive of dissection. Atherothrombotic infarction was diagnosed on the basis of advanced age, presence of cardiovascular risk factors, and angiographic findings of occlusive disease in locations characteristically affected by atherosclerosis (ie, vertebral origin, immediate pre– and post–posterior inferior cerebellar artery origin, vertebrobasilar junction, and midbasilar artery), particularly if these occurred in the setting of additional extracranial and intracranial atherosclerotic involvement of the contralateral vertebral artery and anterior circulation. Patients with penetrating injury to the neck as a cause of dissection were excluded. Patients with high-risk cardiac sources of embolism (as defined by Trial of ORG10172 in Acute Stroke Treatment criteria, or aortic sources of embolism were excluded. Medium- and low-risk cardiac sources of embolism were included if angiographic location was consistent with dissection or atherosclerosis.

All patients underwent comprehensive clinical MRI. The DWI was performed on 1.5-T magnetic resonance systems (Phillips, Infinion, or Eclipse) with echo planar imaging. Diffusion gradients were applied in 3 orthogonal directions to yield isotropic DWI with the following parameters: repetition time/echo time, 6202/103.1 milliseconds; field of view, 240 mm; matrix, 100 × 100 mm. Apparent diffusion coefficient maps were calculated and compared with DWI abnormalities to eliminate T2 shine-through.

The DWI studies were reviewed for infarct patterns. Vascular territories were determined according to established vascular maps.10-12 Infarctions were examined for the presence of AMBI and involvement of different vascular territories. The AMBI was defined as 2 separate DWI-positive lesions within either the same or different vascular territories. An AMBI pattern was also determined if the infarct involved 2 noncontiguous areas within the distribution of the same artery (for example, midbrain and occipital lobe infarction) unless complete territorial infarction had occurred. We examined the frequency of distal embolism to the terminal branches of the basilar artery by noting infarctions in the territory of the posterior cerebral, superior cerebellar, and thalamic perforating branches. Cerebellar border zone involvement was assessed according to previously established criteria.10 All DWIs were independently interpreted by 2 observers blinded to the clinical data (S.K. and A.A.R.). In cases of discrepancy, a consensus reading was obtained. The local institutional review board approved the study design. We used the Fisher exact test to compare categorical variables, and the level of significance was established at P<.05.

RESULTS

A total of 63 patients with symptomatic vertebrobasilar disease were identified. Thirteen patients were excluded for the following reasons: DWI was performed more than 7 days from symptom onset (n=6), contraindications to MRI were present (n=2), no MRI was performed (n=4), and DWI showed no lesions despite persistent stroke symptoms (n=1). Of the remaining 50 patients, 39 were diagnosed as having atherosclerotic disease and 11 patients had arterial dissection. Mean age of the groups was 65 and 49 years, respectively. In the atherosclerotic group, 85% had a history of hypertension, 23% had diabetes, and 33% had dyslipidemia. Patients with dissection had a history of hypertension in 45%, diabetes in 27%, and dyslipidemia in 54% of cases. Cerebral angiography was performed in 82% of patients, with the remainder undergoing magnetic resonance angiography. All patients had an electrocardiogram on admission, and echocardiography (either transesophageal or transthoracic) was performed in 86%. Echocardiography showed medium-risk embolic sources in 7 patients (patent foramen ovale in 4, segmental wall motion abnormalities in 2, and mild global ventricular hypokinesis in 1). The DWI was obtained a mean of 58 hours (range, 5-144 hours) after symptom onset. Median time to imaging was 48 hours, and 14 patients underwent imaging within 24 hours.

Symptoms on presentation, vascular lesion site, and DWI patterns are illustrated in the Figure. The vascular lesion was proximal to the origin of the posterior inferior cerebellar artery in 16 (41%) of 39 patients with atherosclerosis and in all cases of dissection. In the atherosclerosis group, 15 (38%) had basilar artery or vertebrobasilar (unilateral or bilateral) junction stenosis or occlusion, 7 (18%) had bilateral vertebral disease, and 17 (44%) had unilateral vertebral stenosis or occlusion. Vessel occlusion was present in 7 (64%) of 11 patients with arterial dissection. In the atherosclerosis group, 17 patients (44%) had at least 1 vertebral occlusion and 7 (18%) had basilar occlusion.

An AMBI was present in 8 (72%) of 11 arterial dissections and 25 (64%) of 39 patients with atherosclerotic disease and did not differ statistically between the 2 groups (P=.48). Distal embolism to the terminal branches of the basilar artery occurred with equal frequency and was found in 55% of dissections and 54% of patients with atherosclerosis. The thalamus was involved in 1 (9%) of 11 patients with dissection and 6 (15%) of 39 patients with atherothrombosis. Isolated thalamic infarction was not observed in any patient. The mean±SD number of lesions was 4.6±3.9 in patients with dissection and 3.1±2.1 in those with atherosclerosis (P=.08). Pontine infarction occurred in 2 (18%) of 11 patients with dissection and 18 (46%) of 39 patients with atherothrombosis (P=.09).

Cerebellar infarction was present in 9 (82%) of 11 patients with arterial dissection and 29 (74%) of 39 patients with atherosclerosis (P=.47). Cortical cerebellar involvement was observed in 8 (73%) of 11 arterial dissections and 11 (28%) of 39 patients with atherosclerotic infarction (P=.01). Cerebellar border zone involvement was found in 14 (36%) of 39 patients with atherosclerosis and 4 (37%) of 11 patients with dissection (P=.6). No isolated cerebellar border zone infarction occurred (ie, in every case of cerebellar border zone involvement, there was concomitant infarction in the brainstem, thalamus, occipital lobe, or cerebellar cortex).
### Basilar Lesion

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms</th>
<th>Angiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>50/M; L hemiparesis; midbasilar stenosis 50%</td>
<td>68/M; dysarthria, L hemiparesis; midbasilar stenosis</td>
<td>19</td>
</tr>
<tr>
<td>58/F; vertigo, dysphagia, vertigo, R hemiparesis; midbasilar occlusion</td>
<td>67/M; dysarthria, L hemiparesis, vertigo</td>
<td>36</td>
</tr>
<tr>
<td>59/F; L hemiparesis; proximal basilar occlusion</td>
<td>R hemiparesis, midbasilar occlusion</td>
<td></td>
</tr>
<tr>
<td>57/M; neck pain, vertigo; R V3 tapering occlusion</td>
<td>46/M; ataxia, dysarthria, ataxia; R V3 occlusion</td>
<td>45</td>
</tr>
<tr>
<td>51/M; vomiting, ataxia, facial numbness; V4 occlusion</td>
<td>40/M; visual difficulties, seizure; R V5 tapering occlusion</td>
<td>46</td>
</tr>
<tr>
<td>51/M; vomiting, ataxia, facial numbness; V4 occlusion</td>
<td>47/M; headache, vertigo; L V3 occlusion</td>
<td>47</td>
</tr>
<tr>
<td>50/M; vertigo, dysphagia, vertigo, R hemiparesis; midbasilar occlusion</td>
<td>40/F; neck pain, vertigo, dysarthria, diplopia; R V1-V4 irregular stenosis</td>
<td>48</td>
</tr>
<tr>
<td>43/F; R hemianopsia; L V3 80% stenosis</td>
<td>38/M; headache, vertigo, ataxia; L V1 90% stenosis</td>
<td>50</td>
</tr>
</tbody>
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### Vertebral Origin Lesion

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms</th>
<th>Angiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>51/M; vomiting, ataxia, facial numbness; V4 occlusion post-PICA</td>
<td>59/M; L hemiparesis, dysphagia, R hemiparesis and numbness; midbasilar occlusion</td>
<td>19</td>
</tr>
<tr>
<td>58/F; vertigo, dysphagia, R hemiparesis and numbness; R V4 90% stenosis post-PICA</td>
<td>67/M; dysarthria, L hemiparesis, vertigo</td>
<td>36</td>
</tr>
<tr>
<td>65/M; ataxia, dysarthria, L hemiparesis; L V4 occlusion pre-PICA; R V4 90% stenosis post-PICA</td>
<td>R hemiparesis, midbasilar occlusion</td>
<td></td>
</tr>
<tr>
<td>58/F; vertigo, dysphagia, R hemiparesis; L V4 70% stenosis post-PICA</td>
<td>38/M; headache, vertigo, ataxia; L V1 90% stenosis</td>
<td>50</td>
</tr>
</tbody>
</table>

### Vertebral Dissection

<table>
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<th>Symptoms</th>
<th>Angiographic Findings</th>
</tr>
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<tbody>
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<td>51/M; vomiting, ataxia, facial numbness; V4 occlusion pre-PICA</td>
<td>59/M; L hemiparesis, dysphagia, R hemiparesis and numbness; midbasilar occlusion</td>
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<tr>
<td>65/M; ataxia, dysarthria, L hemiparesis; L V4 occlusion pre-PICA; R V4 90% stenosis post-PICA</td>
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<td>58/F; vertigo, dysphagia, R hemiparesis; L V4 70% stenosis post-PICA</td>
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<td>50</td>
</tr>
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**Figure.** Diffusion-weighted imaging infarct patterns. Data are presented as age in years/sex; symptoms; and angiographic findings by lesion. VBJ indicates vertebrobasilar junction; PICA, posterior inferior cerebellar artery; R, right; and L, left.

**COMMENT**

We found that most patients with symptomatic vertebrobasilar large-vessel disease present with an AMBI pattern on DWI. No significant differences were apparent in the AMBI pattern between patients with dissection and atherosclerosis. Increased cortical cerebellar involvement was noted in dissection. Isolated thalamic infarcts were not seen in either group.

The AMBI pattern has been found in 29% of consecutive patients with stroke of all subtypes. Cardioembolism and large-vessel disease have been consistently associated with a higher incidence of AMBI. The AMBI pattern is seen in as many as 83% of patients with extra-
cranial carotid atherosclerotic disease. In strokes due to carotid dissection, 71% of patients had this pattern of infarction.14

Intra-arterial embolism is an important mechanism of stroke in posterior circulation occlusive disease. Previous studies of posterior circulation strokes have relied on conventional MRI and computed tomography, which are not as sensitive as DWI in detecting small ischemic lesions, and have enrolled patients with all stroke etiologies. In a series of 236 patients with posterior circulation infarction (37% with large-vessel disease) evaluated with conventional MRI, 27 (11%) had AMBI, defined as contrast-enhancing lesions. In 22 patients with posterior circulation stroke and AMBI pattern on DWI, most (68%) had large-vessel disease. In the New England Medical Center Posterior Circulation Stroke Registry, verteobasilar disease was the most frequent mechanism of infarction in patients with strokes in multiple arterial territories. A recent study described DWI characteristics of infratentorial strokes in 22 patients and found a higher lesion burden in patients with cardioembolic stroke than noted in the present study of large-vessel stroke (8 vs 3.4).

Small deep cerebellar infarcts have been associated with large-vessel occlusive disease and may be due to hemodynamic mechanisms. With the increased sensitivity of DWI for small ischemic lesions, we demonstrate that border zone infarcts do not occur in isolation but are always associated with acute cortical cerebellar, brainstem, or supratentorial lesions. This suggests that embolism is the major mechanism of infarction in large-vessel vertebrobasilar occlusive disease.

We found no case of isolated thalamic small-vessel infarct in patients with proximal vertebrobasilar occlusive disease. In the anterior circulation, extracranial carotid disease may not be associated with lacunar infarction even though this issue remains unsettled. We believe that our data show that posterior circulation occlusive disease rarely results in isolated thalamic small-vessel infarction.

In summary, our study shows that large-artery disease is frequently associated with AMBI in the posterior circulation. The incidence of the AMBI pattern is comparable to that reported in the anterior circulation. We found no significant differences in the AMBI pattern between dissection and atherosclerotic stroke. Isolated small-vessel thalamic infarction is associated only rarely with large-vessel vertebrobasilar disease.

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Author Contributions: Study concept and design: Koch, Rabinstein, and Forteza. Acquisition of data: Koch, Amir, Rabinstein, and Reyes-Iglesias. Analysis and interpretation of data: Koch, Amir, Rabinstein, Reyes-Iglesias, and Romano. Drafting of the manuscript: Koch. Critical revision of the manuscript for important intellectual content: Koch, Amir, Rabinstein, Reyes-Iglesias, Romano, and Forteza. Administrative, technical, and material support: Amir, Reyes-Iglesias, and Forteza. Study supervision: Koch, Romano, and Forteza.

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REFERENCES