Background: Occlusive disease of the posterior circulation represents a heterogeneous group of strokes that differ in etiology, clinical presentation, and prognosis. Computed tomography provides suboptimal visualization of posterior-circulation infarcts. Anatomic definition of traditional magnetic resonance imaging sequences has been used for clinicoradiologic correlation in patients with posterior-circulation disease. These studies focused on the subacute rather than the acute phase of ischemia. Lesion volumes on diffusion-weighted imaging (DWI) and perfusion imaging were found to have a good correlation with 24-hour National Institutes of Health stroke scale (NIHSS) score in ischemia of the anterior circulation. Correlation between NIHSS score and lesion volume in posterior-circulation infarcts is unknown.

Objectives: To investigate whether DWI is useful for clinicoradiologic correlation of posterior-circulation ischemia within 24 hours after symptom onset and whether NIHSS score correlates with lesion volumes in patients with posterior-circulation stroke.

Patients and Methods: In a database analysis of 631 patients with stroke from June 26, 1996, to July 30, 1999, 115 patients (18%) had symptoms of posterior-circulation ischemia by imaging and clinical criteria. Among these 115, we included all patients (n=40) who underwent DWI within 24 hours from symptom onset (mean, 9.7±7.1 hours). All 40 patients also underwent magnetic resonance angiography and T2-weighted imaging. Seventy-five did not meet inclusion criteria: in 45, magnetic resonance imaging was performed more than 24 hours after symptom onset; 12 did not have DWI; in 11 patients, symptoms resolved within 24 hours; 6 had hemorrhages; and 1 had a border zone infarct.

Results: An acute lesion on DWI corresponding to the patient's symptoms was detected in all 40 patients, 16 (40%) of whom had detectable acute lesions on T2-weighted images. The lesions on DWI were larger in 11 of the 16 patients with positive T2-weighted images. Acute lesion volume did not correlate with NIHSS score (n=40; r=0.30; P=.06, Spearman rank) also when DWI lesion volumes were divided by cause and territory.

Conclusions: Diffusion-weighted imaging is more effective than T2-weighted imaging in patients with acute posterior-circulation strokes. The DWI lesion volume did not significantly correlate with NIHSS score, suggesting that NIHSS is more weighted toward anterior-circulation stroke symptoms.

Arch Neurol. 2001;58:621-628
PATIENTS AND METHODS

PATIENTS

Patient data were collected from the stroke database of Beth Israel Deaconess Medical Center, Boston, Mass. Data for this study were obtained retrospectively from June 26, 1996, to June 30, 1998 (356 patients), and prospectively from July 1, 1998, to July 30, 1999 (275 patients). We included only patients whose symptoms lasted more than 24 hours. The time of onset of the stroke was taken from the time the patient was last seen at baseline neurologic status, including patients who awakened with the deficit. All patients were examined acutely by a stroke fellow (I.L. or R.H.L.) and a stroke attending physician (C.C., S.W., or L.R.C.). The diagnosis of stroke in the distribution of the posterior circulation was based on clinical and radiologic examination by a stroke fellow (I.L. or R.H.L.) and a stroke attending physician (C.C., S.W., or L.R.C.). The NIHSS score was recorded acutely by a clinician certified in the administration of the scale at the time the MR imaging was performed. For all patients, MR imaging and NIHSS were performed shortly after the patient was admitted to the department and before any acute treatment was started.

Patients were included in the analysis regardless of therapy, since inclusion based on treatment was not the aim of the study. The multisequence MR imaging protocol must have included at least DWI, T2WI, MRA and must have been performed as a single session within 24 hours after symptom onset.

DATA ACQUISITION

The MR imaging studies were performed on a 1.5-T scanner (Siemens Vision; Siemens Medical System, Erlangen, Germany). Scans were obtained as part of the patients’ diagnostic evaluation. To minimize the effect of diffusion anisotropy along the white matter fiber tracts, 2 diffusion gradients with 2 b values (0 and 1000 s/mm²) were applied on 3 axes (x, y, and z). A DWI trace image was then calculated online by averaging the signal intensities obtained with the strongest diffusion gradient (b=1000 s/mm²) applied on the 3 axes.

The pulse sequences and typical variables for DWI, T1-weighted images, T2WI, fluid attenuation inversion recovery, and MRA have been described in detail elsewhere.20

VOLUMETRIC ASSESSMENT OF LESION SIZE

All data processing was done with Advanced Visualization Systems Software (AVS, Waltham, Mass) running on a computer workstation (Hewlett-Packard, Hopkins, Minn). Volumes were measured on the image of maximum contrast (ie, DWI trace with the highest b value) between lesion and normal brain regions. The DWI and T2WI volumes and the vascular lesions on MRA were recorded by 1 experienced blinded observer. The DWI and T2WI volumes were derived by measuring the lesion size twice per measurement and recording the average measure. One investigator blinded to the clinical symptoms measured the lesion volume twice, and the values were averaged. The intraobserver reliability for this investigator was previously assessed in a series of 110 patients (r>0.95).

Volumes for the regions of interest were computed by multiplying the measured area per slice by section thickness. Since there was no interslice gap, these volumes are good estimates of the full extent of the true region of interest.

CLINICORADIOLOGIC CORRELATION

The lesion sites were divided into 3 main vascular territories according to the New England Medical Center Posterior Circulation Stroke Registry.19 (1) Proximal intracranial posterior-circulation territory includes the areas supplied by the intracranial vertebral arteries, the medulla oblongata, and the cerebellar territories supplied by the posterior inferior cerebellar artery. (2) Middle intracranial posterior-circulation territory includes the portion of the brainstem supplied by the basilar artery up to its superior cerebellar artery branches, the pons, and the portion of the cerebellum supplied by the anterior inferior cerebellar artery (AICA). (3) Distal intracranial posterior-circulation territory includes all of the territories supplied by the rostral basilar artery, the superior cerebellar artery, the posterior cerebral arteries (PCAs), and their branches (including thalamus, occipital, and temporal lobes).

STATISTICAL ANALYSIS

We compared NIHSS score vs DWI lesion volumes by Spearman rank correlation test with the use of a computerized statistical package (Statview Version 6.1.2; SAS Institute Inc, Cary, NC) and the Bonferroni correction for multiple comparisons. Comparisons were made for all DWI lesion volumes matched with the corresponding NIHSS score. We also performed correlation between NIHSS score and the DWI lesion volume subdivided by territory according to the New England Medical Center classification (proximal, middle, and distal) and subdivided by cause subtype (ie, small vessel branch, embolic [cardiac and artery-to-artery]).

Data are given as mean±SD unless otherwise indicated.

of the adenosine triphosphate–dependent sodium/potassium pumps, (2) increase in glutamate tissue concentration, and (3) size of infarcted tissue on histologic examination.10,13–15

The National Institutes of Health stroke scale (NIHSS) is easy to administer and widely used in patients with acute stroke in all vascular territories.1,16 The volume of lesions on DWI correlates with NIHSS in patients with anterior-circulation strokes.17 Data indicating the utility of DWI in acute stroke have been acquired mostly in patients with anterior-circulation ischemia.3,4,15,17–19 No studies to date, to our knowledge, have specifically addressed the capability of DWI in the imaging of patients with acute posterior-circulation strokes. In addition, the relationship between lesion volumes and NIHSS has not been studied for posterior-circulation infarcts.

Therefore, we investigated whether DWI signal changes and vascular lesions by magnetic resonance angiography (MRA) corresponded to clinical findings in pa-
patients with posterior-circulation ischemia imaged within 24 hours from the onset of symptoms. In the same patients, we also correlated acute lesion volume on DWI and NIHSS scores.

RESULTS

PATIENTS

Among 631 patients with symptoms of stroke, 115 (18%) had a final discharge diagnosis of stroke within the posterior circulation. Seventy-five patients were excluded from the analysis: 43 because of the time of imaging (MR imaging was performed more than 24 hours from symptom onset); 12 because DWI was not performed; 11 because the symptoms resolved within 24 hours; 6 because of hemorrhages on brain imaging; and 1 because of a border zone infarct in the territory of the middle cerebral artery–PCA. Forty patients were included in the analysis. Mean age was 66.2±14.2 years (range, 30-92 years); there were 30 men and 10 women. The mean and SD of the time of symptom to scan was 9.7±7.1 hours. The median of the time from symptom onset to scan was 8.7 hours (interquartile range, 4.3-15.8 hours). Fifteen patients underwent imaging before 6 hours.

IMAGING

An area of abnormality that corresponded to the patients’ symptoms and signs was shown in all 40 patients on the DWI images and on 16 (40%) of the T2WI scans. The acute lesion volume was greater (>30% difference, ie, twice the SD of the measurement) in the DWI images than in the T2WI images in 11 of 16 patients with positive findings on T2WI (Table). The DWI lesions were also correctly identified in all 40 patients by 2 investigators blinded to the patients’ clinical findings. These lesions matched the acute neurologic signs. None of the patients had acute simultaneous anterior-circulation lesions. In 19 patients, T2WI showed other lesions that did not correspond to the patient’s acute symptoms. These lesions were consistent with old infarcts.

CLINICORADIOLOGIC CORRELATION

Acute DWI signal changes and intracranial lesions on MRA corresponded anatomically to well-described stroke syndromes. Location of the DWI and intracranial MRA lesions for all the patients is summarized in Table and Figure 1. The documentation of the vascular lesion by MRA provided insight into stroke mechanisms. Nineteen patients had strokes most likely caused by penetrating and branch artery disease. These strokes were mostly in the middle and distal territories. The average lesion volume for these strokes was less than 1 cm³ (0.56±0.28 cm³) (Table). They were all located in the pons and thalamus. One patient had a stroke at the pontomedullary junction. Twenty-one patients had ischemia most likely caused by thromboembolism involving the territories of the large intracranial posterior-circulation arteries.

These had a large variation in volume, from 0.17 cm³ in a patient with an anterior medullary syndrome (Figure 2) to 28.0 cm³ in a left occipital lobe infarct caused by a left PCA occlusion.

Intracranial MRA, performed as part of the multisequence MR imaging protocol, showed a large-artery lesion in 16 patients. Six had intracranial vertebral artery lesions, 7 had basilar artery lesions (Figure 3) (1 was an aneurysm), 2 had PCA lesions, and 1 patient had a PCA and intracranial vertebral artery lesion. Five had multifocal arterial disease. Nineteen either were normal or showed minor plaque disease.

The assessment of stroke mechanism was based on the results of further evaluation that included extracranial artery imaging that consisted of aortic arch and origin of the vertebral artery by MRA or Doppler imaging. Echocardiography and additional laboratory evaluation were performed when indicated. Regarding stroke mechanisms, 21 patients had ischemia probably caused by thromboembolism mostly involving either proximal or distal territories. The result of the evaluation subsequent to the acute MR imaging study showed that 17 patients most likely had either cardioembolic or artery-to-artery embolism from the extracranial portion of the vertebral artery or from the aortic arch. These infarctions were generally in the proximal or distal territories. Nineteen patients had strokes most likely caused by penetrating and branch artery disease. These strokes were usually in the middle and distal territories (pons and thalamus).

DWI AND NIHSS SCORE

In our patients, acute lesion size on DWI did not significantly correlate with NIHSS score (n=40; r=0.30; P=.06) with the use of Spearman rank correlation with the Bonferroni correction for multiple comparisons. The correlations between NIHSS score and New England Medical Center vascular territory and between NIHSS score and different subtypes of strokes were not statistically significant (Figure 4).

COMMENT

In the present series, a multisequence MR imaging protocol including DWI and MRA provided imaging of lesion sites in all patients with acute posterior-circulation disease studied so far. The T2WI underestimated the acute lesion volume present on the acute DWI within the first 24 hours. In our patients, DWI lesion volume did not significantly correlate with NIHSS score. Most DWI studies have focused on patients with anterior-circulation infarcts.3,4,13,17-19 Regarding small subcortical infarctions, Singer et al21 reported that DWI had 94% accuracy in the diagnosis of such strokes in both anterior- and posterior-circulation territories imaged between 7 hours and 4 days from symptom onset. The authors reported that DWI failed to show the presence of acute ischemia in only 4 of 39 patients. Diffusion anisotropy may be a limitation of DWI in the detection of small infarcts occurring in areas particularly rich in white matter fiber tracts.21,22
<table>
<thead>
<tr>
<th>Patient No./ Sex/Age, y</th>
<th>Stroke Syndromes</th>
<th>Lesion on DWI</th>
<th>Vascular Territory</th>
<th>IC</th>
<th>MRA</th>
<th>Lesion Volume, cm³</th>
<th>NIHSS Score</th>
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<td>B VA dissection</td>
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<td>Normal</td>
<td>8.80</td>
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</table>

*DWI indicates diffusion-weighted imaging; MRA, magnetic resonance angiography; T2WI, T2-weighted imaging; NIHSS, National Institutes of Health stroke scale; IC, intracranial; H, hemiparesis; R, right; VA, vertebral artery; L, left; lat, lateral; med, medial; B, bilateral; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery; BA, basilar artery; PMH, pure motor hemiparesis; AH, ataxic hemiparesis; INO, internuclear ophthalmoplegia; 1/2, 1/2 syndrome; III, III cranial nerve palsy; PS, pure sensory; thalamo-genic, thalamo-genulate; HS, hemisensory; LOC, loss of consciousness; SCA, superior cerebellar artery; HH, homonymous hemianopia; and PCA, posterior cerebral artery.
Diffusion tensor MR imaging has been proposed to correct the anisotropy-induced error in calculation of the apparent diffusion coefficient of water. Because of the time constraints of acute stroke evaluation, such analysis was not performed in the present study. However, a DWI trace image was calculated online by averaging the signal intensities obtained with the strongest diffusion gradient (b=1000 s/mm²) applied on the 3 axes. This provides a rotationally invariant image (trace) that has fewer anisotropy artifacts. In our patients, DWI trace images detected acute lesions (as small as 0.2 cm³) in our patients with posterior-circulation stroke. Lesion volume on DWI was less than 1 cm³ in 26 patients and less than 0.5 cm³ in 19. In another series, Ay et al²⁴ used DWI in 782 consecutive patients with strokelike deficits in all territories. Among 782 patients, 17 had acute DWI-negative ischemia, 5 had transient ischemic attacks, 2 had prolonged reversible deficits, 3 (who had abnormalities on perfusion-weighted images) subsequently developed evidence of infarction, and 7 had lacunes (3 of which were later shown to be in the brainstem). Although the authors did not report whether they used DWI trace images in their analysis, these numbers are remarkably small (7 of 782) and, together with our data, suggest that DWI is reliable in the

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Figure 1. Location of the lesions on diffusion-weighted imaging and magnetic resonance angiography for all 40 patients. Proximal territory (patients 1-9) included 7 medullary and 2 cerebellar infarcts. Middle territories (patients 10-21) included 11 pontine and 1 pontomesencephalic infarcts. Distal territories (patients 22-40) included 1 superior cerebellar artery territory infarct and 7 thalamic, 4 occipital, and 2 temporal and occipital lobe infarcts. Five patients had multiple acute lesions throughout the brainstem and occipital lobe because of basilar artery occlusion.
evaluation of very small subcortical strokes. In 19 patients, T2WI showed other lesions that did not correspond to the patient’s acute symptoms. Those lesions were consistent with old infarctions. The data are in agreement with DWI and ADC signal intensity changes observed in time course analysis in experimental ischemia and in patients with stroke. These observations may be useful in both routine clinical evaluation and for development of new treatments, since multiple old infarctions are common, especially in patients with penetrating and branch artery disease. As recently demonstrated by Oliveira-Filho et al., DWI can detect new acute lesions that are otherwise undetected by T2WI or fluid attenuation inversion recovery and differentiate them from old infarctions.

In our patients, the initial interpretation of DWI with intracranial MRA corresponded with the results of further evaluation regarding stroke lesions, vascular lesions, and stroke mechanisms. Traditional MR imaging sequences such as T2WI have been used for clinicoangiographic correlation studies of the subacute to chronic phase of posterior-circulation strokes. In the present series, T2WI underestimated the lesion volume present on the acute DWI. Therefore, DWI may combine the anatomic definition of MR imaging with high sensitivity for hyperacute ischemia. The sensitivity of MRA in intracranial disease compared with conventional angiography has not been well studied. With this limitation, in the present series, the combination of DWI and MRA seemed to be able to document brain and vascular lesions in the acute setting. In the present study, decisions regarding further investigations and management were made shortly after the patient arrived in the emergency department. Although a larger study assessing the cost-effectiveness of such an approach is required, our preliminary observation indicates that an acute multisequence MR imaging study may be able to provide acute information on brain and vascular abnormalities that may prompt targeted evaluation and treatment.

The DWI and perfusion imaging lesion volumes, obtained within 6.5 hours of symptom onset, were found to have a good correlation with 24-hour NIHSS score and 7-day neurologic outcome in patients with anterior-circulation infarcts. Investigators posit
that DWI and perfusion-weighted imaging together with stroke scales might be used to screen and observe candidates for acute thrombolysis and drug trials. However, while stroke scales provide an effective assessment of patient symptoms, all have some disadvantage. Most impairment scales, such as the Scandinavian Stroke Scale, the Canadian Stroke Scale, and the NIHSS, are weighted toward motor and sensory symptoms more than hemianopia and cranial nerve deficits. In addition, most scales have not been validated in patients with posterior-circulation stroke. Because of the limitations in CT and clinical scales, patients with posterior-circulation stroke are often excluded from drug trials. In the present series, acute lesion size on DWI did not correlate with acute NIHSS score. The lack of correlation could be due to either the small number of patients studied or a discrepancy (particularly present in posterior-circulation disease) between infarct size and loss of function. In fact, relatively large infarct in the occipital cortex might only cause a hemianopia, whereas small (<1 cm³) pontine or midbrain infarction can cause severe deficits. Therefore, although NIHSS represents an easy-to-administer and widely validated scale, it seems more useful in patients with anterior-circulation ischemia.

In the present series, because the diagnosis of posterior-circulation stroke was based in part on the result of the DWI, the specificity and sensitivity of DWI and T2WI were not assessed. However, because of the current limitations in clinical scales and in CT imaging, the lesion size definition obtained by the use of DWI may be a useful tool in clinical routine evaluation and clinicoradiologic correlation studies in acute posterior-circulation ischemia. The utility of DWI and MRA to shorten the diagnostic evaluation and selection of treatment of acute posterior-circulation strokes is promising and needs to be studied in a larger cohort of patients.

Figure 3. Images in a 79-year-old man with hypertension, coronary artery disease, and intermittent atrial fibrillation who was found unresponsive on the floor. He had appeared normal a few minutes earlier. On examination he was in respiratory distress, was comatose, had pinpoint pupils, and showed decerebrate posturing. A through E, Diffusion-weighted imaging (DWI) trace images (acquired 2 hours after the onset) show multiple hyperintense areas scattered from the pons to the midbrain and in the left occipital cortex. J, T2-weighted image (T2WI) shows poorly defined hyperintensity in the left occipital cortex. K, Magnetic resonance angiogram (MRA) shows basilar artery occlusion.
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REFERENCES