Restricted Diffusion in Spinal Cord Infarction Demonstrated by Magnetic Resonance Line Scan Diffusion Imaging

Raul G. Nogueira, MD; Rafael Ferreira, MD; P. Ellen Grant, MD; Stephan E. Maier, MD, PhD; Walter J. Koroshetz, MD; Ramon G. Gonzalez, MD, PhD; Kevin N. Sheth, MD

Background and Purpose—We report on the use of line scan diffusion magnetic resonance imaging in the evaluation of spinal cord infarctions.

Methods—Data on 19 patients with clinical findings consistent with spinal cord infarctions and abnormal findings on line scan diffusion imaging were reviewed. The Apparent Diffusion Coefficient (ADC) measurements for the normal spinal cord and for the areas of abnormality were calculated from trace ADC maps.

Results—Restricted diffusion was found in all 19 patients. Absolute ADC values in the ischemic area ranged between 395.4 and 575.8×10⁻⁶ mm²/s, with ADC ratios ranging between 39.4% and 57.4%.

Conclusions—Line scan diffusion imaging is technically feasible and appears to be a reliable method to diagnose spinal cord infarction in the acute setting. (Stroke. 2012;43:532-535.)

Key Words: spinal cord infarct ■ MRI ■ diffusion

Magnetic resonance imaging may be normal for hours to a few days after spinal cord infarction (SCI). Diffusion-weighted magnetic resonance imaging (MRI) has proven to be the most sensitive diagnostic tool in acute cerebral ischemia and has become a promising technique in the evaluation of acute spinal cord syndromes. We report our experience with the utilization of magnetic resonance (MR) line scan diffusion imaging (LDSI) for the evaluation of SCI.

Patients and Methods

Data on 19 patients with SCI and abnormal findings on LDSI were collected between February 2001 and April 2009. The data were independently reviewed by neurologists (R.G.N. and K.N.S.) to confirm accuracy of the diagnosis. Criteria for SCI diagnosis included an acute deficit; spinal cord imaging corresponding to spinal artery territory as described by Masson et al; and no other diagnosis after extensive work-up, including evaluation for autoimmune and hypercoagulable disorders, viral serologies and cerebrospinal fluid analysis, gadolinium-enhanced brain MRI, and evoked potentials (unless the etiology was clear based on the clinical scenario).

Description of the Imaging Technique and Analysis

MR examinations were performed on a Signa 1.5T imaging system (GEMS). The LDSI technique has been previously described. Images were obtained on either the sagittal or axial plane using a phased-array spine coil (GEMS) with the following typical sequence parameters: TR (repetition time; effective), 3960 ms; line-to-line TR, 90 ms; TE (echo time), 76 ms; field of view, 320×160 mm; rectangular acquisition matrix, 128×128 (lines); spatial in-plane resolution, 2.5×1.25 mm; section thickness, 3 mm; skip, 0 mm; b of 5 and 750 s/mm², with the maximum b value applied in 3 orthogonal directions. The receiver bandwidth was ±7.8 kHz. Total acquisition time without signal averaging (1NEX) was approximately 6 minutes, 33 seconds. In some cases, a smaller field of view was selected, and 3 or 4 averages were obtained to compensate for the lower signal-to-noise ratio at higher spatial resolution. LDSI postprocessing and image analysis were performed according to previous technical descriptions. The Apparent Diffusion Coefficient (ADC) measurements for the normal spinal cord and for the areas of infarction were calculated from trace ADC maps by using the average value of 4 to 7 distinct regions of interest (area, 0.03–0.06 cm²) positioned over the spinal cord, carefully avoiding the inclusion of cerebrospinal fluid. The ADC ratio was subsequently calculated based on the average ADC for the area of infarction and normal spinal cord.

Results

Relevant findings are summarized in Table. The mean patient age was 70±11 years. Fifty-three percent of the patients (10/19) were men. LDSI was obtained between 3 hours and 9 days from symptom onset. The clinical presentation included acute onset of bilateral motor-sensory deficits in all cases. Eighteen cases were thought to be related to occlusion of the...
anterior spinal artery and 1 case to occlusion of the posterior spinal artery. The etiology was postsurgical in 11 patients. One patient was thought to have fibrocartilaginous embolism syndrome. No definite etiology for the SCI was identified for 7 cases despite extensive evaluation as detailed above.

Three lesions were cervical (15.8%); 5 lesions were thoracic (26.3%); 4 lesions were thoracolumbar (21%); and 7 lesions were in the lumbar/conus region (36.8%). The average ADC was 1000.2 (±103) × 10⁻⁶ mm²/s for the normal spinal cord (range, 813.3–1188.2), and 487.5 (±47) × 10⁻⁶ mm²/s (ADC ratio, 49%) for the SCI lesions (range, 395.4–575.8). The average length of the abnormal restriction lesion was 62.6 ± 64.6 mm. Five patients had follow-up MRI with LSDI, with subsequent elevation in ADC values seen in all cases. Figure 1 and 2 illustrate cases involving the cervical cord and the conus region, respectively.

Discussion

SCI is a relatively rare disease, accounting for only about 1.2% of all strokes; however, SCI still represents one of the most common causes of acute noncompressive myelopathies. MRI evaluation is essential in the diagnosis of SCI. Abnormalities on T2-weighted sequences are seen in more than 90% of SCI

Table. Data Summary

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (Y)</th>
<th>Sex</th>
<th>Underlying Cause</th>
<th>Location</th>
<th>Lesion Length (mm)</th>
<th>ADC Average Infarct Core × 10⁻⁶ mm²/s</th>
<th>ADC Average Normal Cord × 10⁻⁶ mm²/s</th>
<th>ADC Ratio (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>M</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>36</td>
<td>473</td>
<td>971</td>
<td>48.8</td>
</tr>
<tr>
<td>2</td>
<td>78</td>
<td>F</td>
<td>No obvious cause</td>
<td>Conus</td>
<td>32</td>
<td>510</td>
<td>1026.8</td>
<td>49.7</td>
</tr>
<tr>
<td>3</td>
<td>76</td>
<td>M</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>36</td>
<td>561</td>
<td>977.5</td>
<td>57.44</td>
</tr>
<tr>
<td>4</td>
<td>68</td>
<td>M</td>
<td>Hypotension after hemiolectomy</td>
<td>T6-L1</td>
<td>206</td>
<td>452</td>
<td>1149.66</td>
<td>39.37</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>F</td>
<td>Acute congestive heart failure</td>
<td>T4-T7</td>
<td>25</td>
<td>534</td>
<td>984</td>
<td>54.28</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>F</td>
<td>No obvious cause</td>
<td>C3-C6</td>
<td>32</td>
<td>505</td>
<td>1096.14</td>
<td>46.13</td>
</tr>
<tr>
<td>7</td>
<td>74</td>
<td>F</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>3</td>
<td>431</td>
<td>960.2</td>
<td>44.92</td>
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<tr>
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<td>71</td>
<td>M</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>30</td>
<td>453</td>
<td>920.66</td>
<td>49.23</td>
</tr>
<tr>
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<td>M</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>22</td>
<td>508</td>
<td>902</td>
<td>56.36</td>
</tr>
<tr>
<td>10</td>
<td>81</td>
<td>M</td>
<td>Postoperative AAA</td>
<td>Conus</td>
<td>38</td>
<td>575</td>
<td>1188.2</td>
<td>48.45</td>
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<tr>
<td>11</td>
<td>48</td>
<td>M</td>
<td>Prior trauma and cord compression</td>
<td>C3-C5</td>
<td>34</td>
<td>492</td>
<td>870.5</td>
<td>56.59</td>
</tr>
<tr>
<td>12</td>
<td>75</td>
<td>F</td>
<td>Prior trauma and cord compression</td>
<td>C6-C7</td>
<td>17</td>
<td>531</td>
<td>1101</td>
<td>48.22</td>
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<tr>
<td>13</td>
<td>84</td>
<td>F</td>
<td>Postoperative AAA</td>
<td>T6-T9</td>
<td>52</td>
<td>462</td>
<td>920</td>
<td>50.21</td>
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<td>14</td>
<td>77</td>
<td>M</td>
<td>Postoperative coronary stent</td>
<td>T8-T12</td>
<td>100</td>
<td>477</td>
<td>982</td>
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<tr>
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<td>50</td>
<td>M</td>
<td>Repair of type B aortic dissection</td>
<td>T5-L1</td>
<td>227</td>
<td>447</td>
<td>1018</td>
<td>43.9</td>
</tr>
<tr>
<td>16</td>
<td>76</td>
<td>M</td>
<td>Postoperative esophageal CA</td>
<td>T8-T9</td>
<td>30</td>
<td>395</td>
<td>813.28</td>
<td>48.62</td>
</tr>
<tr>
<td>17</td>
<td>75</td>
<td>F</td>
<td>Fibrocartilaginous embolus</td>
<td>T7-T12</td>
<td>114</td>
<td>474</td>
<td>1123.28</td>
<td>42.26</td>
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<tr>
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<td>82</td>
<td>F</td>
<td>No obvious cause</td>
<td>T2-T4</td>
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<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
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<td>55</td>
<td>F</td>
<td>Radicular branch occlusion</td>
<td>T12-L1</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

ADC indicates Apparent Diffusion Coefficient; M, male; AAA, abdominal aortic aneurysm; F, female; CA, cancer; NA, data not available.

SCI is a relatively rare disease, accounting for only about 1.2% of all strokes; however, SCI still represents one of the most common causes of acute noncompressive myelopathies. MRI evaluation is essential in the diagnosis of SCI. Abnormalities on T2-weighted sequences are seen in more than 90% of SCI.
cases. However, these changes are not specific, and it is often difficult to distinguish SCI from other causes of acute noncompressive myelopathies based on MRI alone. Sagittal MR images of anterior spinal artery infarction usually demonstrate an isolated pencil-like area of T2-hyperintensity involving the centromedullary region, often encompassing more than 2 vertebral segments. In contrast, demyelinating lesions are usually smaller and tend to involve the lateral and posterior aspects of the cord. Because of the higher vulnerability of the gray matter to ischemia, axial T2-sequences may show bilateral hyperintensities that are mostly confined to the anterior horn area, leading to the typical snake eyes or owl’s eyes configuration.

One of the major limitations of current MRI techniques is the failure to reveal any abnormalities during the early phases of SCI. Diffusion-weighted imaging (DWI) has been established as the most sensitive modality for the diagnosis of acute cerebral ischemia and has the potential to become an important technique in the evaluation of spinal cord pathology. There are, however, several technical difficulties in obtaining adequate diffusion-weighted (DW) images of the human spinal cord including: motion artifacts caused by physiological movement of the spinal cord and surrounding structures (eg, cerebrospinal fluid pulsatile flow, swallowing, respiration, and cardiac motion), susceptibility artifacts caused by the presence of bone and cerebrospinal fluid interfaces, and the low signal-to-noise ratio caused by the small pixel dimensions required for appropriate visualization of the spinal cord. While single-shot echo-planar diffusion imaging offers adequate resistance to motion artifacts, in the spine it is plagued by severe distortion artifacts. However, with alternative, albeit slower, diffusion imaging techniques, like navigated pulsed-gradient multi-shot spin-echo imaging, navigated segmented echo-planar diffusion imaging, or LSDI, adequate DWI of the spinal cord is feasible.

Indeed, abnormal findings on DW images of the spinal cord have been shown in patients with tumors, radiation and spondylotic myelopathies, multiple sclerosis, epidermoid cyst, and myelitis. In addition, DW imaging using single-shot fast spin echo and echo-planar techniques has been successfully employed to demonstrate acute and subacute SCI. However, to the best of our knowledge, the use of LSDI to diagnose acute SCI has been reported only in a single previous case.

Our results using LSDI concur with the previous published ADC values for the normal spinal cord and SCI. A major limitation of our study was the inability to image patients during the hyperacute phase (first 3 hours after the ictus). Therefore, additional studies are needed to determine the sensitivity and specificity of LSDI for the detection of spinal cord ischemia in this setting. In addition, LSDI as a technique has some limitations. LSDI is not widely available and is relatively slow. This calls into question the signal-to-noise ratio advantage of LSDI over echo-planar imaging, specifically if signal-to-noise ratio is scan-time normalized.

In conclusion, DW imaging of the spinal cord is technically challenging, but is currently feasible. In our opinion, LSDI has several advantages over other DW imaging modalities and should be incorporated into the routine evaluation of acute noncompressive myelopathies.

Disclosures
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References


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Abstract

ラインスキャン拡散磁気共鳴画像によって示される脊髄梗塞の拡散制限

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背景および目的：脊髄梗塞の評価におけるラインスキャン拡散磁気共鳴画像の使用について報告する。

方法：脊髄梗塞と一致する臨床所見およびラインスキャン拡散画像上に異常所見を有する19例の患者のデータを評価した。正常な脊髄および異常領域の拡散係数（ADC）測定値をトレースADCマップから計算した。

結果：19例の患者全員に拡散制限が認められた。虚血領域のADC絶対値は395.4 ~ 575.8 × 10^-6 mm²/sの範囲であり、ADC比は39.4 ~ 57.4%の範囲であった。

結論：ラインスキャン拡散画像は技術的に可能であり、急性期に脊髄梗塞を診断するための信頼できる方法であると思われる。

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図1 急性四肢不全麻痺を呈する63歳女性。矢印は、矢状断T2(A)およびDWI(B)の高信号と、拡散制限と一致するADC(C)の低信号を示す。T2軸位断像(D)は、前部脊髄梗塞の特徴であるsnake-eye形に脊髄の前面に生じる高信号を示す。

注：ラインスキャン拡散画像法は他の拡散画像法に比して磁化率の効果やmotion artifactの影響を受けにくい。AJNR 2000; 21: 1344-1348参照。