MR Findings of Transverse Myelitis and Its Clinical Correlation

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Purpose: The purpose of this study is to correlate the MR findings with clinical stage and clinical outcome, and to describe the evolutilonal changes of abnormal MR findings of transverse myelitis.

Materials and Methods: Medical records and spinal MR images of 23 patients with both clinical and radiological diagnosis of transverse myelitis were retrospectively reviewed. MR findings were correlated with clinical stages including interval between MR imaging and full development of clinical symptoms, and compared with the clinical outcome.

Results: Diffuse high signal intensity of the spinal cord on T2-weighted image with mild cord bulging (67%) and focal contrast enhancement of the cord (75%) were observed within the first four weeks after full development of clinical symptoms. The findings decreased in extent or vanished later than four weeks on either initial or follow-up MR images. Most patients with either cord atrophy or focal hemorrhage within the cord lesion had poor clinical outcome.

Conclusion: The MR findings of transverse myelitis are nonspecific, which may be seen in a variety of diseases. Serial MRIs, especially follow-up examination over at least one month after full development of clinical symptoms are useful in the diagnosis of transverse myelitis and predicting its prognosis.

Index Words: Spinal cord, MR, Spinal cord, infection

INTRODUCTION

Transverse myelitis is an idiopathic inflammatory or demyelinating syndrome of the spinal cord involving both halves of the cord, often over a considerable length, and appearing without any history of previous neurological disease (1). The most common features are weakness of the lower extremities and bilateral sensory loss, usually with a well-defined sensory level. It may progress to urinary and fecal incontinence (1, 2).

Magnetic resonance (MR) imaging is the modality of choice for detecting pathological processes of the spinal cord (3-6). To our knowledge, only a few reports of MR imaging of transverse myelitis, mostly of acute variety, have appeared in the literature. Those reports described conflicting findings with respect to the signal intensity of the spinal cord (7-11).

The purpose of this study was to describe the MR findings of the spinal cord lesion and to correlate the MR findings with clinical stage and outcome in patients with transverse myelitis.

MATERIALS and METHODS

Medical records and spinal MR images of 23 patients with clinical diagnosis of transverse myelitis and who had abnormal signal intensity in the spinal cord with no or slight cord bulging on MR imaging, were retrospectively reviewed.

Criteria for the diagnosis of transverse myelitis were based on: (1) the development of loss of sensory and motor function; (2) spinal transverse segmental level of sensory disturbance (patients with Brown-Sequard syndrome were excluded); (3) no history of antecedent neurologic disease (e.g., multiple sclerosis) or other known neurologic disease including trauma, metastases, and encephalitis; (4) no clinical or radiologic
evidence of spinal cord compression. Twenty-three patients fulfilled these criteria, consisting of 15 males and eight females, 25—74 years old (mean 54.6 years). The patients who had an intramedullary mass with increased size on follow-up MR imaging were excluded. Clinical follow-up varied from 2 to 36 months (mean 14.6 months).

Five patients had viral or bacterial infection preceding the onset of transverse myelitis by 4 to 27 days. Of these five patients, three had upper respiratory infection (possibly viral), and one each had herpes zoster and acute gastroenteritis. In one patient who subsequently developed optic neuritis, multiple sclerosis (MS) evolved out of acute transverse myelitis (Fig. 1). Another patient with acute symptoms had systemic lupus erythematosus (SLE). Etiology of the disease was not known in the remaining 16 patients.

The patients were divided into three groups according to the patterns of development of symptoms: acute variety (n=12), in which symptoms develop rapidly and reach their peak of severity within days; the subacute variety (n=9), in which the disease evolves over a period of 2 to 6 weeks; and the chronic variety (n=2), in which more than 6 weeks elapse between the onset and the full development of the clinical symptoms.

All patients were imaged on a 2.0 T superconducting unit using spin-echo pulse sequence. T1-weighted sagittal images were obtained with a repetition time (TR) of 500—800 ms and an echo time (TE) of 30 ms. Proton density- and T2-weighted sagittal and axial images were obtained with a TR of 2500 ms and TEs of 30 and 80 ms, respectively. In 15 patients, T1-weighted axial and sagittal images were obtained after intravenous injection of gadopentetate dimeglumine (Magnevist® Schering, Germany) 0.1 mmol/kg. The matrix number was 180—256 × 256 for sagittal images and 180—210 × 256 for axial images. FOV was 256 mm for sagittal images and 180—210 mm for axial images. The slice thickness/gap was 3 mm/1 mm for sagittal images and 7 mm/2 mm for axial images. The interval between MR imaging and the full development of symptoms were variable (Table 1). Nine follow-up MR examinations were performed in eight patients. The periods of follow-up varied from 1 to 25 months (mean 11.2 months).

The MR images were reviewed regarding the location, extent, signal intensity, and contrast enhancement pattern of the cord lesion, presence or absence of cord bulging, and serial change of cord abnormalities on the follow-up MR images. These MR findings were correlated with clinical stages including interval of time between MR imaging and full development of clinical symptoms, and with clinical outcome.

**RESULTS**

The clinical stages, MR findings of initial and follow-up studies, and clinical outcome of the 23 patients are summarized in Table 1. The spinal cord was slightly enlarged (Fig. 1 and 2) in 14 patients, within normal range in eight patients, and slightly atrophic in the remaining one patient (Fig. 3a). Enlargement of the cord was frequently seen within four weeks after full development of symptoms (12 of 18 patients). The initial T2-weighted MR images showed high signal intensity within the spinal cord in all 23 patients. On T1-weighted images, the signal intensity was slightly hypointense or isointense relative to normal spinal cord. The lesions were variable in location, but mainly located at low- and mid-thoracic levels (18 patients). The length of the lesions depicted on T2-weighted sagittal images were variable, ranging from two to ten vertebral hei-

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**Fig. 1.** MR images of a 42-year-old man with multiple sclerosis which was evolved out of acute transverse myelitis.

a-c. Three weeks after full development of symptoms, T2-weighted sagittal image (a) shows diffuse high signal intensity (arrow heads) in the mildly enlarged cord at C3-5 level. Post-contrast sagittal (b) and axial (c) images show focal patchy enhancement (arrow) in posterior portion of cervical cord at C4 level.
On T2-weighted axial images, the high signal intensity was usually centrally located within the cord, and involved over two thirds of transverse surface of the cord in 17 patients (Fig. 2b), and less than two third in six patients. It was homogenous and round-shaped in all patients. In five of nine patients involving lower thoracic spinal cord, the lesions extended caudally through the conus medullaris, and showed "button hole" or "owl's eyes" appearance at the conus medullaris (Fig. 3b). A focal area of low signal intensity within the lesions on both T1 and T2-weighted images suggested hemorrhage (Fig. 4) was observed in two patients. Contrast enhancement of the lesion was noted in 11 of 15 patients and was usually seen within four weeks after full development of clinical symptoms (9 of 12 patients). It was focal patchy in appearance in seven (Fig. 1) and small round in four patients.

On follow-up MR images obtained in eight patients, the spinal cord which had been slightly enlarged on initial MR image was normalized between one and 12 months in five patients (Fig. 2), and became atrophic at 14 and 25 months in two patients (Fig. 4). In the remain-

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Table 1. Summary of MR Findings and Clinical Outcome

<table>
<thead>
<tr>
<th>Patient No./Age(Sex)</th>
<th>Variety/Etiology</th>
<th>Location</th>
<th>Interval *</th>
<th>MR Finding</th>
<th>CE **</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SI on T2WI/Cord Size/Hemorrhage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/57/F 57/F</td>
<td>Acute/Post-infectious</td>
<td>T8-L1</td>
<td>1day</td>
<td>high/enlarged/+</td>
<td>-</td>
<td>poor (paraplegia, neurogenic bladder)</td>
</tr>
<tr>
<td>2/37/F 37/F</td>
<td>Acute/Unknown</td>
<td>T9-T11</td>
<td>3day</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>normalized</td>
</tr>
<tr>
<td>3/58/F 58/F</td>
<td>Acute/Unknown</td>
<td>T10-L1</td>
<td>5day</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (weakness)</td>
</tr>
<tr>
<td>4/64/F 64/F</td>
<td>Acute/Post-infectious</td>
<td>T4-T8</td>
<td>5day</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (weakness)</td>
</tr>
<tr>
<td>5/46/F 46/F</td>
<td>Acute/Unknown</td>
<td>T4-T8</td>
<td>6day</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>poor (paraplegia, neurogenic bladder)</td>
</tr>
<tr>
<td>6/63/M 63/M</td>
<td>Acute/Unknown</td>
<td>T10-L1</td>
<td>11day</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (weakness)</td>
</tr>
<tr>
<td>7/31/F 31/F</td>
<td>Acute/SLE</td>
<td>T7-T12</td>
<td>1wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>8/65/M 65/M</td>
<td>Subacute/Unknown</td>
<td>T2-T3</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (weakness)</td>
</tr>
<tr>
<td>9/67/M 67/M</td>
<td>Subacute/Unknown</td>
<td>T4-T7</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>10/35/M 35/M</td>
<td>Subacute/Unknown</td>
<td>C5-C6</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>11/45/F 45/F</td>
<td>Subacute/Unknown</td>
<td>T6-T8</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>12/50/M 50/M</td>
<td>Subacute/Unknown</td>
<td>T4-L1</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>13/70/M 70/M</td>
<td>subacute/Unknown</td>
<td>T7-T10</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>+</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>14/28/M 28/M</td>
<td>Acute/Post-infectious</td>
<td>T8-T9</td>
<td>2wk</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>expired d/t pneumonia</td>
</tr>
<tr>
<td>15/55/M 55/M</td>
<td>Subacute/Post-infectious</td>
<td>C2-C3</td>
<td>3wk</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>16/40/M 40/M</td>
<td>Chronic/Unknown</td>
<td>T5-T6</td>
<td>3wk</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>17/42/M 42/M</td>
<td>Acute/MS</td>
<td>C3-C5</td>
<td>3wk</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (numbness)</td>
</tr>
<tr>
<td>18/74/F 74/F</td>
<td>Acute/Unknown</td>
<td>T10-L1</td>
<td>3wk</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (dysesthesia)</td>
</tr>
<tr>
<td>19/37/M 37/M</td>
<td>Subacute/Unknown</td>
<td>T7-T9</td>
<td>2mo</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>fair (weakness, dysesthesia)</td>
</tr>
<tr>
<td>20/54/M 54/M</td>
<td>Subacute/Unknown</td>
<td>T6-T10</td>
<td>3mo</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>poor (paresthesia)</td>
</tr>
<tr>
<td>21/56/M 56/M</td>
<td>Chronic/Unknown</td>
<td>T3-T4</td>
<td>4mo</td>
<td>high/enlarged/-</td>
<td>-</td>
<td>good (paresthesia)</td>
</tr>
<tr>
<td>22/27/M 27/M</td>
<td>Acute/Post-infectious</td>
<td>T9-L1</td>
<td>7mo</td>
<td>high/mild atrophy/-</td>
<td>-</td>
<td>poor (parelegia)</td>
</tr>
<tr>
<td>23/59/M 59/M</td>
<td>Subacute/Unknown</td>
<td>T9-L1</td>
<td>11mo</td>
<td>high/mild atrophy/-</td>
<td>-</td>
<td>fair (weekness, paresthesia)</td>
</tr>
</tbody>
</table>

Sl = signal intensity, T2WI = T2-weighted image
* interval of time between MR and full development of symptoms
** CE = contrast enhancement: + enhanced, - not-enhanced
ing patient, a cord of normal size was not changed. The high signal intensity of the cord decreased in extent between one and 13 months in six patients, and disappeared at 24 and 25 months in two patients (Fig. 2).

The clinical outcome of the present series was variable: normalized in one; good (minimal neurologic sign) in 13; fair (weakness, incontinence) in four; poor (paralysis or wheelchair-dependent) in four; expired due to pneumonia in one patient. Poor clinical outcome with severe residual dysfunction was associated with MR findings of cord atrophy and/or focal hemorrhage in three of four patients (Table 1).

**DISCUSSION**

Transverse myelitis is an uncommon but not rare condition occurring in all age groups. The annual incidence has been estimated at one per 1.34 million population in a report (12).

Only few previous reports discussed the MR signal characteristics of acute transverse myelitis (7-11). Those papers emphasized diffuse cord enlargement but described conflicting signal characteristics of the cord on long TR sequence. Merine et al. (10) reported the lesion was isointense relative to normal cord on a long TR sequence in both of their two cases. However, diffuse increased signal intensity on long TR sequence was the most common finding in the other reports (7-9, 11). Contrast enhancement of the lesion was also documented. In the present series, all patients showed diffuse increased intensity of the cord on long TR sequence. However, MR findings of cord enlargement and contrast enhancement were variable (Table 1). A focal lesion of low signal intensity on both T1- and T2-weighted images in our two cases presumably indicates acute or chronic petechial hemorrhage (Fig. 4), which has not been described previously in transverse myelitis. Although the cause of hemorrhage is not
known in transverse myelitis, vascular insult might be attributed to it.

Serial changes of the cord abnormalities have been reported in a few cases (7, 9). Those reports described serial change of abnormal findings in acute transverse myelitis, but provided no details in relation to the interval of time between MR study and full development of clinical symptoms. In our cases, diffuse involvement of the cord, cord enlargement, and focal contrast enhancement were mostly observed within the first four weeks after full development of clinical symptoms, regardless of clinical variety, and they decreased in extent or vanished later than four weeks on either initial or follow-up MR images. This change is similar to that seen in spinal cord ischemia (13). It is interesting to note that these changes of MR findings correlate invariably with the interval of time between MR study and full development of symptoms, though they were variable with the interval of time after symptom onset.

The etiology of transverse myelitis remains largely unknown, though many well documented associations exist and include viral illness (14-16), vaccinations (17), MS (12), vascular insufficiency (18), and SLE (11). Preceding infection was noted in one-third of the
patients with acute transverse myelitis in Hoffman's series (19). Berman et al (12) found that postinfectious transverse myelitis was more common in individuals under age 40. Paine and Byers (18) found a dissociation between pain and other sensory modalities in patients with acute transverse myelitis. Dissociated sensory loss is usually attributed to a vascular cause. Sensory deficits of high and middle thoracic levels were more common in acute transverse myelitis without prior infection. Since blood supply is more limited at high and middle thoracic cord levels, their findings would be compatible with a vascular cause in patients without infection (18). Another support for vascular etiology in some cases of acute transverse myelitis is that clamping of the aorta or dissecting aneurysm may cause a clinical picture of acute transverse myelitis (20). In this series, the temporal evolution of MR findings similar to that of a cord ischemia, mainly affecting thoracic levels, and old age of patients suggest that the etiology of unidentified transverse myelitis, which was the largest group of our series, might be vascular insufficiency.

In general, it seems not difficult to differentiate transverse myelitis from neoplasm, even though diffuse high signal intensity on a long TR sequence, cord enlargement, and contrast enhancement were nonspecific findings that may result from a variety of diseases such as ischemia, demyelinating disease, and tumor (6, 21). In view of the MR findings in the present series, a small focal or patch enhancement within the much larger lesions of high intensity on T2-weighted images is in favor of transverse myelitis, whereas the more pronounced larger enhancement within the enlarged cord suggests cord tumor. Vanishing or diminution of these findings on the follow-up MR images is also helpful in differentiation from the neoplasm.

In a report regarding the prognosis of transverse myelitis (1), rapid onset of disability and back pain were often associated with poor outcome. Our study suggests that cord atrophy and focal hemorrhage on MR images are related to poor prognosis.

In conclusion, even though MR findings of transverse myelitis are nonspecific, they correlate well with clinical stage and outcome. Serial MRI, especially follow-up MRI over at least one month after full development of clinical symptoms are useful for diagnosis of transverse myelitis and predicting its prognosis.

REFERENCES

횡단성 척추염의 자기공명영상 소견 및 임상소견과의 연관성

서울대학교 의과대학 방사선과학교실
김재승·장기현·나동규·한문희·최흥곤·김지혜

목적: 횡단성 척추염의 자기공명영상소견을 임상적 경과 및 예후와 비교하고 시간적 전개에 따른 자기공명영상소견의 변화를 기술하고자 한다.

대상 및 방법: 임상적으로 횡단성 척추염으로 진단된 환자 중 자기공명영상소견에 이상을 보였던 23명의 자기공명영상과 임상소견을 후향적으로 검토하여 자기공명영상소견을 임상적 증상이 완전히 발현한 시기로부터의 기간과 연관지어 분석하였다. 환자의 예후와도 비교하였다.

결과: T2강조 자기공명영상에서 척수의 확장을(67%)이나 국소적인 조영증강(75%)을 동반한 척수내 미만성의 고신호강도가 임상적 증상의 완전 발현 후 한달이내에서 관찰되었고 한달 이후에는 병변이 소실되거나 그 정도가 감소되었다. 척수의 위축이나 국소적인 출혈이 있던 환자는 예후가 나빴다.

결론: 횡단성 척추염의 자기공명영상소견은 다른 척수질환에서도 나타나는 비특이적인 것이지만 임상적 증상이 완전히 발현한 시기로부터 한달 이후에 추적 자기공명영상을 시행하는 것은 횡단성척추염의 진단과 그 예후를 예측하는데 유용하다.
본회 창립 50주년 및 X선발견 100주년 기념학술대회 사전등록 및 초록 제출 안내

본회 창립 50주년 및 X선발견 100주년 기념학술대회를 개최함에 있어 사전등록, 초록제출 마감, 전시작품 규격, 호텔 예약등을 아래와 같이 안내하오니 업무에 참고하시기 바람니다.

- 아래 -
1) 대회일시: 1995년 9월 26일(화) - 9월 30일(토) 까지
2) 장소: 쉐라톤 워커힐 호텔 컨벤션센터
3) 사전등록: 1995년 6월 30일(금) 까지

| 등록비 | 사전등록('95. 6. 30 이전) | 80,000원 | 40,000원 |
|-----------------|-----------------|-----------------|
| 현장등록('95. 6. 30 이후) | 100,000원 | 50,000원 |

송금처: 지회 및 검진협회 지부소속 회원은 검진협회 지부를 통하여
일괄 납부하며 주시면 감사하겠습니다. 송금구좌는

외환은행 양재동지점 (224-18 08032 2) 예금주 서정호 입니다.

4) 초록마감: 1995년 4월 29일(토)까지 (구연, 전시포함)

제출처: 본학회사무국
구면 및 전시에 제출하는 초록형식은 다음과 같이 통일하며, 이러한 형식으로 구성되지 않은 초록은 접수될 수 없음을 알려드립니다. 접수한 초록은 학술위원회 심사과정을 거쳐 제목여부가 결정되나 기일 엄수하시고 아래 사항을 주지하시어 제출하여 주시기 바랍니다.

① 초록의 구성은 다음 4개 항목이 그 부제와 함께 명기되어야 하며 전체의 용량은200단어 내외로하여 프린터로 출력한 견본과 함께 Computer Diskette(아래한글)으로 제출
② 목 적: 연구의 배경 및 목적을 기술
③ 대상 및 방법: 중요 부분은 자세히 기술
④ 결 과: 관찰된 결과의 기술
⑤ 결 론: 결과로부터 도출된 결론을 기술

제출초록 전면에 “구연”, “전시”를 구별 표시하며, 구면의 경우 구면 예정자를 반드시 표기할 것.

① 본아미 구분을 반드시 표기한 것.
② 전시작품 규격 및 제출에 관하여 금년도는 Backboard type만 가능하며 판넬 1 space의 크기를 가로 80cm × 세로 100cm로 제작하고 부득이 널던 space가 필요하면 가로 80cm × 세로 150cm로 제작하여 원칙적으로 1 space로 제작합니다. 또한 판넬은 두께 5mm 이하의 하드보드 및 스쳐로플을 사용하고 액자같은 테두(나무나 금속품) 붙이지 말것. Illuminating type은 사용하지 않으며 판넬크기가 종전과 변동이 있으니 숙지하시기 바랍니다.

5) 호텔예약마감: 1995년 6월 30일(금) 까지
학술대회 기간중 호텔예약을 이용하실 분은 6월 30일까지 쉐라톤 워커힐 호텔로 직접 예약하시기 바람요. 제실료는 정상가격에서 41% 할인된 100,000원입니다.(부가세, 봉사료 별도) 예약처는 쉐라톤 워커힐 (02) 453-0131