CT myelography with intramedullary enhancement in cervical spondylosis

YOSHINOBU IWASAKI, M.D., HIROSHI ABE, M.D., TOYOHIKO ISU, M.D., AND KAZUO MIYASAKA, M.D.

Departments of Neurological Surgery and Radiology, Hokkaido University School of Medicine, Sapporo, Japan

The authors describe seven cases of cervical spondylosis in which small high-density areas were detected in the spinal cord on delayed computerized tomographic (CT) myelography. These high-density areas are believed to represent cavities or areas of cystic necrosis. In all seven cases the cervical spinal canal was narrow, and the spondylosis was located at multiple levels, causing a so-called "pincer effect." On the CT scans the high-density areas resembled fried eggs in the gray matter. These areas were localized near the abnormal cervical discs. In two cases in which the Brown-Séquard syndrome was noted, the symptoms could be attributed to the morphology of the high-density area on the affected side of the cord. Following decompressive surgery, most of the symptoms improved except for numbness of the upper extremities and motor weakness of hands.

KEY WORDS: cervical spondylosis • computerized tomography myelography • intramedullary cavitation • spinal cord compression

Morphological changes in the spinal cord in cases of cervical disc disease have been identified more rapidly since the recent advances in neuroradiological diagnosis. However, these observations have related to extramedullary abnormalities or deformities of the spinal cord by compression: there are no neuroradiological studies of intramedullary changes. In seven recent cases of cervical spondylosis undergoing preoperative delayed computerized tomography (CT) myelography, we have identified areas of enhancement that appeared to be cavitated intramedullary lesions of the cervical spinal cord. Although the cases are few, certain similarities can be found in the neurological symptoms, radiological findings, and mechanism of formation of intramedullary cavities in this condition. This paper presents our findings.

Summary of Cases

Patient Population

The series included four men and three women, ranging in age from 51 to 68 years (average 59 years). All of the patients had cervical spondylosis with no acute disc herniations. Five to 18 hours after intrathecal injection of 10 to 13 ml metrizamide (220 mg I/ml), CT scans of the spine were obtained in all patients using sections 4-mm thick.

Neurological Findings

Neurological symptoms began with numbness or motor weakness of the hands and fingers in four cases, weakness of the lower extremities in two cases, and urinary disturbance in one case. None of the patients had a history of trauma. On admission, all seven patients had motor weakness of their hands and fingers and peripheral hypesthesia of the upper extremities (Table 1). There were no other significant differences from other patients with cervical spondylosis as far as the neurological findings were concerned.

Radiological Findings

Table 2 summarizes the preoperative neuroradiological findings. In all cases there was cervical spondylosis with a narrow spinal canal, as well as findings of spinal cord compression by osteophytes and/or disc protrusions at more than two levels from C3-4 to C6-7. In addition, posterior spinal cord compression from the thickening or indenting of the ligamenta flava during extension of the spine, the so-called "pincer effect," was detected in one case at a single level and in six cases in multiple levels. Areas of high-contrast were detected in the spinal cord by delayed CT myelography, and consisted of two small round or elliptical shapes in the gray matter, one on the right and one on the left side. These
TABLE 1
Summary of clinical data in the seven cases in this series

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Initial Symptoms</th>
<th>Duration*</th>
<th>Neurological Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 55</td>
<td>numbness of lt fingers</td>
<td>5 yrs</td>
<td>spasticity &amp; hyperreflexia of both legs, sensory disturbance of palms</td>
</tr>
<tr>
<td>2</td>
<td>F, 65</td>
<td>weakness of rt hand</td>
<td>5 mos</td>
<td>tetraparesis (rt &gt; lt), generalized hyperreflexia, sensory disturbance below T-4 on lt</td>
</tr>
<tr>
<td>3</td>
<td>M, 52</td>
<td>numbness of rt extremities</td>
<td>2 yrs</td>
<td>lt hemiparesis, hyperreflexia of both legs, sensory disturbance below T-4 on rt</td>
</tr>
<tr>
<td>4</td>
<td>M, 51</td>
<td>weakness of rt leg</td>
<td>6 mos</td>
<td>motor weakness of rt hand, spasticity &amp; hyperreflexia of legs</td>
</tr>
<tr>
<td>5</td>
<td>M, 68</td>
<td>urinary disturbance</td>
<td>5 yrs</td>
<td>muscle atrophy of arms, tetraparesis, sensory disturbance in C5-6 area</td>
</tr>
<tr>
<td>6</td>
<td>F, 61</td>
<td>numbness of fingers</td>
<td>8 yrs</td>
<td>rt hemiparesis, generalized hyperreflexia (rt &gt; lt), sensory disturbance below C-5</td>
</tr>
<tr>
<td>7</td>
<td>F, 67</td>
<td>numbness of fingers</td>
<td>5 yrs</td>
<td>motor weakness of rt hand, generalized hyperreflexia, sensory disturbance of fingers</td>
</tr>
</tbody>
</table>

*Time from onset of symptoms to admission.

TABLE 2
Preoperative neuroradiological findings in this series

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Level of Disc Disease</th>
<th>Level of Pincer Effect</th>
<th>Extent of Syrinx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C4-5, C5-6</td>
<td>C4-5</td>
<td>C4-5 (2 levels)</td>
</tr>
<tr>
<td>2</td>
<td>C3-4, C4-5, C5-6</td>
<td>C5-6</td>
<td>C-5 (1 level)</td>
</tr>
<tr>
<td>3</td>
<td>C4-5, C5-6</td>
<td>C4-5, C5-6</td>
<td>C5-6 (2 levels)</td>
</tr>
<tr>
<td>4</td>
<td>C4-5, C5-6, C6-7</td>
<td>C4-5, C5-6, C6-7</td>
<td>C-5 (1 level)</td>
</tr>
<tr>
<td>5</td>
<td>C3-4, C4-5, C5-6, C6-7</td>
<td>C3-4, C4-5, C5-6, C6-7</td>
<td>C3-5 (3 levels)</td>
</tr>
<tr>
<td>6</td>
<td>C4-5, C6-7</td>
<td>C3-4, C5-6, C6-7</td>
<td>C4 (1 level)</td>
</tr>
<tr>
<td>7</td>
<td>C4-5, C5-6, C6-7</td>
<td>C4-5, C5-6, C6-7</td>
<td>C4-5 (2 levels)</td>
</tr>
</tbody>
</table>

High-density areas were localized within two segmental levels. In two patients (Cases 2 and 3) with a modified Brown-Séquard syndrome, there was no unilateral compression of the spinal cord by the extradural lesion. However, on the side corresponding to the cortical spinal tract symptoms, the density of the enhancement was higher, and one patient had atrophy on that side.

Surgical Procedures and Results

Three patients underwent spinal fusion via the anterior approach. Two patients were subjected to laminectomy, and two patients had both fusion and laminectomy. Postoperatively, the myelopathy improved in all cases. Motor weakness and numbness of the hands and fingers, while somewhat better, persisted in most patients. Postoperative delayed CT myelography, performed in five patients, disclosed a high-density area in the spinal cord in three patients similar to that seen before operation, but this could not be seen in the other two cases.

Illustrative Cases

Case 1

This 55-year-old man had noted a sensation of numbness of the left hand and fingers for 5 years. For 1 year before admission, he had noted hypesthesia of the hands and fingers and a tight feeling in the lower right extremity. Neurological examination showed spastic paraparesis with increased deep tendon reflexes in the lower extremities and moderate hypesthesia in both palms.

Myelography revealed anterior spinal cord compression by osteophytes and disc herniation at the C4-5 and C5-6 levels. There was also posterior compression at the C4-5 level, which was intensified during extension of the spine. Computerized tomography, performed 5 hours after instillation of metrizamide revealed bilateral high-contrast areas like two fried eggs in the gray matter at C-4 and C-5 (Fig. 1 left). Repeat CT performed 21 hours after metrizamide infusion showed no additional high-density areas. The patient underwent anterior spinal fusion at the C4-5 and C5-6 levels.

Postoperatively, the paresis of both legs and hypalgesia of the right palm disappeared, but the hypesthesia of the left palm was unchanged. One month after surgery, the high-density area was not visible on CT scanning 5 hours after metrizamide injection (the same period between myelography and CT scanning as preoperatively), but at 8 hours the CT scan clearly showed the high-density areas (Fig. 1 right).

Case 3

This 52-year-old man had noted numbness and ataxia for 2 years on the right side below the T-4 level. Neurological examination on admission showed motor weakness of the left extremities, muscular atrophy of the upper left extremity, increased deep tendon reflexes of the lower extremities, and hypealgesia below the T-4 level on the right (Brown-Séquard syndrome). Myelography showed the pincer effect at the C4-5 and C5-6 levels. Delayed CT, conducted 5 hours after myelography, demonstrated two high-density areas in the spinal cord from C-5 to C-6. One of the high-density areas seemed to occupy most of the left side of the cord, suggesting atrophy, while the high-density area on the right side was mostly within the gray matter and the white matter seemed still to be in good condition (Fig. 2).

The patient was treated with both anterior fusion and laminectomy. During laminectomy, a small myelotomy was performed at the left posterior root entry zone, and a small cavitation was verified at the C-5...
CT myelography for cervical spondylosis

level. After the operation, there was moderate improvement in both the motor weakness of the lower left extremity and the right hemisensory disturbance.

Discussion

The development of high-resolution CT scanning and aqueous contrast material has aided in studying the pathological changes in the spinal cord associated with cervical disc disease. A CT scan can show the degree and direction of spinal cord compression by the osteophytes and discs, the degree of deformation or atrophy of the spinal cord, and whether or not there is a pincer effect on extension of the spine. However, all reported findings concerned extramedullary changes and have not included intramedullary lesions. Studies of intramedullary lesions of the cord have been confined to animal experiments and postmortem observations. If intramedullary pathological changes could be identified by clinical investigation, the findings could be very useful in determining therapy and in studying pathophysiological mechanisms.

In recent years, clinical diagnosis of syringomyelia, an important pathological change in the spinal cord, has been established safely with myelography and delayed CT scanning. In our seven patients with cervical spondylosis studied by CT myelography, we observed high-contrast areas similar to those noted in cases of syringomyelia. We believe that these intramedullary high-density areas may actually represent cavities in the cord. However, this question cannot be settled for the present since no postmortem findings are available in our seven cases.

Similar to cases of syringomyelia, the high-contrast areas in our patients were localized within the cord and could not be detected except by delayed CT scanning. We postulate, however, that these areas represent either a single cavity, a mass of microcavities, or cystic necrosis. Indeed, in our Case 3 a cavity was confirmed by operation, and autopsy findings in several other cases of cervical spondylosis have shown a cavity or necrosis extending over a wide area in the gray matter. Animal experiments have revealed that various non-reversible changes, such as cavitation or necrosis in the cord (especially in the gray matter), can be caused by spinal cord ischemia or compression. Accordingly, it is logical to believe that the high-density areas seen on delayed CT myelography represent nonreversible pathological changes, such as cavitation or cystic necrosis in the gray matter.

In two of five cases so studied, the high-density areas were not found on delayed postoperative scans. We believe that this is due to one of the following causes: 1) a change in the time required for the contrast material to enter the cavity secondary to a change in cerebrospinal fluid flow caused by the operation; 2) obstruction of the route to the cavity; or 3) the collapse of the cavity itself. This may explain the situation in Case 1, in which the high-density area was not detected by CT.
Using the same dose of metrizamide and time delay as before the operation, but it did appear after a greater time delay.

In order to conclude that a cavity is caused by cervical spondylolysis, differentiation from other diseases that are associated with cavitation in the cord is required. Thus, cervical spondylolysis is excluded as the etiology in patients older than middle age with complications of acute herniated disc disease, those with symptoms related to syringomyelia, and those with syringomyelia after traumatic injury of the cord. In our cases we conclude that cavitation was probably caused by cervical spondylolysis, because: 1) none of the patients had a traumatic injury, an Arnold-Chiari malformation or other malformations in the foramen magnum, or evidence of arachnoiditis; 2) the cavities were localized within the two spinal levels where the cervical intervertebral disc disorders were situated; and 3) most of the patients' symptoms were improved by extradural resection.

In general, static compression, dynamic compression, and cord ischemia are considered to be the factors that cause symptoms of cervical spondylolysis.11,1 These factors may also apply to cavitation, and include continuous long-term cord compression over various levels, repeated minor trauma from the pincer effect due to frequent cervical movements during daily activities, and compromise of intramedullary blood circulation because of deformation by cord compression.

In spite of the fact that clinical symptoms caused by this cavitation were not as marked in our seven patients as in cases of syringomyelia, numbness and motor weakness in the hands and fingers were observed in most of the patients before operation. Even after operation these symptoms improved very little compared to other symptoms. This suggests that some patients with apparent radiculopathy may have intramedullary damage.

In the future, more cases with specific symptoms, such as a modified Brown-Séquard syndrome, will be needed to study the relationship between clinical symptoms and intramedullary pathological changes. The means by which aqueous contrast material passes into the cord in the cases of cervical spondylolysis is not known; it does not enter via the central canal, as is reported to be the case with syringomyelia. The possibility of transmedullary passage is supported by the localization of the high-density areas and the cavity at the levels of the cervical disc disorder.

References

8. Mail WGP, Druckman R: The pathology of spinal cord lesions and their relation to clinical features in protrusion of cervical intervertebral discs. (A report of four cases.) Brain 76:70-91, 1953