Diaphragmatic paralysis caused by cervical spondylosis

Case report

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The authors describe a rare case of diaphragmatic paralysis caused by cervical spondylosis. A 64-year-old man presented with dyspnea as well as cervical radicular pain and left-sided upper-extremity motor weakness. Chest radiography revealed elevation of both sides of the diaphragm. All symptoms were ameliorated immediately after cervical laminoplasty, and spirometry revealed improvement of ventilatory function 6 months after surgery. Cervical spondylosis should be considered a factor that can cause respiratory dysfunction.

KEY WORDS • diaphragmatic paralysis • spondylosis • laminoplasty • cervical spine

Diaphragmatic paralysis, which is familiar to chest physicians, usually results from infiltration of lung cancer, surgery-induced injury of the phrenic nerve, infection or trauma, or neurological diseases such as amyotrophic lateral sclerosis. Cervical spondylosis, which is a common disease, can impair phrenic nerve function by compressing the C-4 neuromere and/or nerve. Dyspnea, however, is rarely observed in patients with cervical spondylosis, and only a few cases of diaphragmatic paralysis caused by cervical spondylosis have been reported. In this report, we describe a case in which respiratory disturbance was caused by cervical spondylosis and was ameliorated by cervical laminoplasty, and we discuss the importance of cervical spondylosis as the factor causative of respiratory dysfunction.

Case Report

Presentation and Examination. This 64-year-old man with a 15-year history of atrial fibrillation and hypertension experienced sudden-onset nuchal pain and dyspnea when he bent his neck forward. Cervical spondylosis and left hemidiaphragmatic paresis were diagnosed at a nearby medical clinic. Despite a 3-month course of medication, the cervical pain was exacerbated and spread over the shoulders bilaterally. Dyspnea was also aggravated and became so severe that it disturbed the patient’s sleep. He began to experience motor weakness of the left arm 4 months after the onset of the aforementioned symptoms and was referred to our hospital. On physical examination, the breath sounds over the lower lobe of the left lung were diminished. Neurological examination demonstrated motor weakness of muscles supplied by the right C-5 neuromere and the left C5–7 neuromeres, radicular pain in the left C-4 dermatome, hypesthesia of the left C-5 and C-6 dermatomes, and bilateral lower-extremity hyperactive deep tendon reflexes.

A chest radiograph obtained after a maximal inspiration revealed an elevation of the diaphragm, especially on the left side (see Fig. 1B), although a review of a chest radiograph obtained 2 years previously indicated no abnormality (Fig. 1A). Computerized tomography scanning demonstrated no mediastinum and thorax abnormality. Ventilatory function was evaluated using spirometry (Table 1). Vital capacity was 2.25 L (66.6% of predicted), TV was 0.51 L, FVC was 1.95 L (57.7% of predicted), and FEV1.0% was 68.2% (100.4% of predicted). Cervical radiography demonstrated narrowing of the intervertebral disc spaces and malalignment of the cervical spine. Myelography revealed poor filling of contrast material in the subarachnoid space at C3–4, where instability was prominent (Fig. 2). Postmyelography CT scanning also revealed C3–4 compression of the spinal cord (Fig. 3A). Magnetic resonance imaging also demonstrated compression of the spinal cord due to a herniated disc and a thickened ligamentum flavum at C3–4 (Fig. 4). In addition, the subarachnoid space anterior to the spinal cord was diminished along the kyphotic VBs from C4–5 to C6–7, indicating anterior osteophyte-induced compression of the spinal cord at these levels.

Operation. The patient underwent an expansive open-
Diaphragmatic paralysis caused by cervical spondylosis

Two years after the surgery, chest radiography revealed good sleep. Spirometry revealed improvement in ventilatory function. VC was 2.41 L (73.9% of predicted), TV was 0.76 L, FVC was 2.26 L (69.3% of predicted), and FEV1.0% was 67.7% (100.3% of predicted) (Table 1).

Postoperative Course. One week after the operation, our patient’s neck and shoulder pain had subsided and motor weakness of the deltoid muscles improved. Left biceps brachii and brachioradialis muscles exhibited a full recovery. The lower-extremity deep tendon reflexes also returned to normal. Interestingly, the patient felt gradual decrease of dyspnea, which had been his chief complaint, although chest radiography and spirometry demonstrated no remarkable changes at that time. Six weeks postoperatively, he felt physically relaxed during respiration and enjoyed good sleep. Spirometry revealed improvement in ventilation: VC was 2.41 L (73.9% of predicted), TV was 0.76 L, FVC was 2.26 L (69.3% of predicted), and FEV1.0% was 67.7% (100.3% of predicted) (Table 1). Two years after the surgery, chest radiography revealed good expansion of the both lungs; however, the expansion of the left lung was judged to be incomplete because the position of the left diaphragm was higher than that of the right diaphragm, and this finding had been reversed 2 years before surgery (Fig. 1A and C).

Discussion

Association of Diaphragmatic Paresis With Cervical Spondylosis

There has been no report in which authors have examined the function of the diaphragm in patients with cervical spondylosis. Indeed, it is actually impossible to conduct electrophysiological studies of the phrenic nerves and the diaphragm. In our case, there was no direct evidence showing that the diaphragmatic paresis was caused by cervical spondylosis; however, several lines of indirect evidence indicate that this is correct. First, causative factors such as mediastinum tumors, infection, trauma, and other neurological diseases were excluded by physical, neurological, and radiological/neuroimaging examinations. Second, dyspnea was ameliorated in conjunction with neurological symptoms of deltoid muscle motor weakness after laminoplasty-related decompression of the spinal cord and the nerve roots. Third, VC, TV, and FVC improved after surgery. Although FEV1.0% was unchanged during the postoperative course, all the functions associated with diaphragmatic movements were improved. This result coincided with those reported in previous studies showing that VC and FVC, but not FEV1.0%, were lower in the cases of chronic cervical myelopathy.

The phrenic nerve of one side does not control the motion of the contralateral diaphragm because the tension of one side of the diaphragm is not well transmitted to the other because of the posterior indentation made by the spinal column and aorta. Therefore, the unilateral failure of the phrenic nerve results in the elevation of the ipsilateral diaphragm. Unilateral diaphragmatic paresis can be caused by the interruption of any pathway from the spinal cord to the diaphragm. Piehler, et al., reported that hemidiaphragmatic paresis was caused by infiltration of bronchial cancer in one third of 105 cases, whereas in another one third it was due to accidental injury during thoracic or neck surgery. Trauma, infection, and neurological diseases accounted for the remaining cases. Therefore, a great majority of cases of diaphragmatic paresis are caused by the direct damage to the phrenic nerve; in such instances, the diaphragmatic paresis is unilateral and is evident on chest radiography. The diaphragmatic paresis produced by cervical spondylosis, however, may be bilateral and may be missed on chest radiography if the diaphragmatic paresis of one

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<tr>
<th>Ventilatory Aspect</th>
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<tr>
<td>VC (L)</td>
<td>2.25</td>
<td>2.41</td>
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<tr>
<td>TV (L)</td>
<td>0.51</td>
<td>0.76</td>
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<tr>
<td>FVC (L)</td>
<td>1.95</td>
<td>2.26</td>
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<td>FEV1.0% (%)</td>
<td>68.2</td>
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* Spirometry showed improvements in VC, TV, and FVC, which are the indicators of restrictive pulmonary function, but not in FEV1.0%, which is the indicator of occlusive pulmonary function, suggesting the improvement of the diaphragmatic paralysis.

J. Neurosurg: Spine / Volume 2 / May, 2005

605
The side is at the same level as that of the contralateral side. In our case, it was uncertain whether right-sided diaphragmatic paresis was present before surgery (Fig. 1A and B) because the radiographic finding of diaphragm elevation required the patient to inhale. The comparisons of positions of the right and left sides of the diaphragm, however, clarified that the left-sided diaphragmatic paresis was present before surgery and was ameliorated 2 years after surgery (Fig. 1B and C).

**Anatomical Considerations**

Inspiratory neurons are concentrated in nuclear columns within the pontine and medullary tegmentum. Axons that arise in the inspiratory neurons descend just lateral to the ventral horns of the C-1 through C-3 neuromeres and terminate on the nuclear columns of cells that consist of motor neurons innervating the diaphragm. Keswani and Hollinshead reported that these nuclear columns, from which the phrenic nerves arise, exist in the most medial part of the gray matter of the cervical ventral horn extending from the C-3 to the C-5 neuromere. The C-4 nerve root mainly supplies nerve fibers of the phrenic nerve whereas the C-3 and C-5 nerve roots contribute minimally. There is a discrepancy between the level of the neuromere and that of the VB. For example, the C3–4 disc space corresponds to the C-5 neuromere. Consequently, impairment of phrenic nerve function is caused by C-4 neuromere compression at the C2–3 disc space (segmental myelopathy) or to the anterior C-4 nerve root at C3–4 (radiculopathy). In our case, there was no noticeable spin-

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**FIG. 2.** Flexion (A) and extension (B) myelograms revealing poor filling of contrast material in the subarachnoid space from C3–4 to C5–6 and significant instability at C3–4.

**FIG. 3.** Postmyelography CT scans revealing compression of the C3–4 cord before surgery (A) and the well-expanded spinal canal after surgery (B). Note that the nerve roots were observed within the well-expanded subarachnoid space after surgery (arrows).
Diaphragmatic paralysis caused by cervical spondylosis

al cord compression at the C2–3 disc space where the C-4 neuromere is thought to exist. Because radicular pain was observed in the left C-4 dermatome, it is reasonable to speculate that the anterior C-4 root compression at the C3–4 disc space caused the diaphragmatic paresis. After spinal canal enlargement, which resolved the nerve root compression within the spinal canal, the radicular pain and diaphragmatic paresis were ameliorated.

There has been only one paper in which authors have reported on diaphragmatic paresis caused by cervical spondylosis. In that report the patient experienced shortness of breath for 3 months and left hemidiaphragmatic paresis caused by cervical spondylosis, which was severe at the level of the C3–4 disc space, was diagnosed. Six weeks after a C2–6 laminectomy, the left hemidiaphragmatic paresis and dyspnea improved. Findings in this case and ours indicate that the C3–4 lesion is a causative factor of diaphragmatic paresis. This is in accordance with the aforementioned anatomical considerations.

Cervical Spondylosis as a Cause of Respiratory Failure

Respiratory failure after the acute spinal cord injury above C3–4 is well documented; however, cervical spondylosis is not recognized as a factor causative of respiratory failure. This may be due to the fact that a minority of cases of cervical spondylosis involve the upper cervical spine. In addition, little attention has been paid to subtle changes of respiratory function. In recent studies, however, clinicians have reported mild improvement of pulmonary function after cervical laminoplasty. Because the spinal cord plays important roles in controlling respiration, cervical spondylosis may affect respiration and may be an important causative factor of respiratory failure. Further studies are required to clarify this point.

Conclusions

Although there is a possibility that the phrenic nerve palsy may result from upper cervical lesions, the association of cervical spondylosis with pulmonary function has attracted little attention. In this report, a case of cervical spondylosis causing diaphragmatic paresis is discussed. An awareness of this association could promote a correct diagnosis and proper treatment of pulmonary dysfunction.

References


Manuscript received November 5, 2004. Accepted in final form February 9, 2005.
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