



INSTRUCTIONAL LECTURES & PANEL PRESENTATIONS

**CERVICAL SPONDYLOTIC MYELOPATHY.
ITS CLINICAL MANIFESTATIONS AND SPINAL CORD PATHOLOGY****Kazuo Yonenobu, M.D., D.M.Sc.***Institution(s):**Vice-director, National Osaka-Minami Hospital**Clinical Professor, Department of Orthopaedic Surgery, Osaka University Medical School*

Cervical spondylosis is one of the most common disorders that all people over middle age. Problems brought by this condition have become more serious to societies where aged population is expanding. Clinical manifestations of cervical spondylosis are quite diverse and schematically these can be classified into discopathy, radiculopathy, myelopathy and miscellaneous conditions. Among them, myelopathy is relatively rare but disability of myelopathic patients is more severe and permanent.

Clinical features of cervical spondylotic myelopathy (CSM) are composition of symptoms of segmental and long tract impairment. Segmental symptoms are ambiguous to radicular symptoms and thus the symptoms are sometimes regarded as radicular origin, and diagnosed to be radiculomyelopathy erroneously. In Japan we have many patients with CSM, probably due to developmental spinal canal size. Experientially, most of patients with CSM have only combination of long tract and segmental signs, and the diagnosis of CSM is easy by neurological and radiological examinations. On the other hand, we have experienced patients having muscular weakness and atrophy of the upper extremity without obvious sensory deficit of the upper extremity, that is vague to lower motor neuron diseases⁽²⁻³⁾. Sobue named this

condition cervical spondylotic amyotrophy (CSA). Anyway, both conditions are myelopathic. What changes of the spinal cord occur in these conditions?

Since Key described encroachments on the spinal cord by ventral ridges of disc origin in 1838, the condition has been studied from various viewpoints. Although several pathological studies on the spinal cord were done, little was known about relationship between clinical manifestations and spinal cord pathology of CSM. We carried out a clinicopathological study to elucidate the relationship⁽²⁾. We found that severity of pathological changes was paralleled to severity of myelopathic symptoms, and the severity of pathological changes could be represented with a simple morphometric parameter, compression ration (=anteroposterior diameter of the spinal cord divided by transverse diameter).

Development of computed tomography (CT) allows us to observe a cross-sectional shape of the spinal cord and to measure its area. We studied relationship between a transverse area of the cord at maximally compressed level and various clinical parameters including severity of myelopathy evaluated with the scale proposed by the Japanese Orthopaedic Association (JOA

score), duration of disease and surgical results⁽⁴⁻⁵⁾ and found the transverse area at maximally compressed level significantly correlated with surgical results. Namely, the transverse area is the most significant indicator of viability of the spinal cord in CSM.

Our previous pathological study on CSM reveals that an initial change of the spinal cord is demyelination of the white matter. When demyelination of the white matter advances, the gray matter is involved and shows cystic changes. Namely, the white matter reduces the size more than the gray matter. On the other hand, severity of pathologic change is correlated with severity of myelopathy. So, positive correlation between the transverse area of the cord and the severity of myelopathy before surgery could be speculated. However, significance of the correlation was not high and a correlation between the transverse area and severity of myelopathy after surgery was statistically positive. We think that the difference of results between from the pathological study and the clinical study is due to the difference of subjects, that is, in the former study the subjects are in the end stage of CSM, while in the latter study, the subjects are in the initial or advanced stage of CSM.

With appearance of magnetic resonance imaging (MRI) following CT, we can obtain clues of internal changes of the cord as well as external forms of the cord in various sections. The most recognizable change was signal change of the cord (high in T2 weighted and low in T1 weighted image). We also investigated relationship between the signal change of the cord and the clinical parameters that were analyzed in the CT study. We could not find significant correlation between the high signal intensity area (HSIA) and surgical results. A significant positive parameter correlated to multisegmental

HSIA was muscle atrophy of the upper extremity. The HSIA represents cavity formation in the gray matter and thus in CSM that long-tract dysfunction is a main feature of disease, the HSIA cannot be an indicator of severity of myelopathy or a predictor of surgical results. When damage of the gray is extensive, usually the white matter is also damaged severely. In another word, the multisegmental HSIA indicates probable extensive damage of the white matter and thus, can be a predictor of surgical results.

Another type of myelopathy is cervical spondylotic amyotrophy (CSA). Characteristics of CSA are muscle atrophy of the upper extremity, which means predominant involvement of the gray matter. There are two types of CSA; proximal and distal types. The proximal type of CSA is characterized by weakness or atrophy of the deltoid, biceps and other shoulder girdle muscles. The distal type represents muscle weakness and atrophy of the hand and forearm. In both types sensory deficit of the upper extremity is usually absent or mild compared to severe motor dysfunction of the upper extremity. No sign and symptom of long-tract involvement except for exaggerated tendon reflexes is detected. Although these clinical features are delusive to lower motor neuron diseases, this should be differentiated because these are treatable surgically. Multiple-level spondylotic protrusions and developmental narrow spinal canal are believed to be etiological in the distal type, although etiology of the proximal type is still controversial.

Conclusively, clinical manifestations of CSM are diverse and some patients with CSM show signs and symptoms mimicking intrinsic neurological diseases. However, in most cases, diagnosis of CSM is feasible with careful history taking, minute neurological examination, appropriate radiological examination and wide knowledge on this condition.

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