Clinical-Radiographic Correlations in Cervical Spondylosis

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Cervical spondylosis is a progressive and disabling disease of later life characterized by a variable combination of upper extremity radiculopathy and predominantly motor myelopathy. The underlying pathologic process seems to begin with degeneration of cervical intervertebral discs, most commonly at the C4-5, C5-6 and C6-7 levels. Bony hypertrophic changes then develop as ridges or bars along the margins of the affected interspace. How these bony-ligamentous changes produce neurologic deficits has been, and remains, a subject of debate. In all probability the observed radiculopathy results either from direct, mechanical compression of individual nerve roots within their foramina of exit or from stretching of the nerve roots secondary to abnormal angulation of vertebral bodies adjacent to degenerated interspaces, or to a combination of both.

The mechanism of production of the myelopathy of cervical spondylosis remains more obscure. Simple mechanical compression of the spinal cord by hypertrophic bars anteriorly, or infoldings of the ligamentum flavum posteriorly, would be expected to produce predominantly sensory myelopathy. Kahn's ingenious theory implicating the dentate ligaments as stress transmitters has been badly shaken by anatomical studies showing that the tethering effect of these ligaments is restricted to rostro-caudal and lateral displacement of the cord, with anteroposterior movement being relatively unrestricted.

Considerable interest has also been devoted to the possible role played by vascular factors in the pathogenesis of the myelopathy of cervical spondylosis. Demonstrable thrombosis of the anterior spinal artery is rarely found, although one might expect this to be a common sequel if this vessel were compressed to a significant degree against underlying bony bars.

Breig, et al., have reported a microangiographic cadaver study of the circulation of the cervical spinal cord. They were struck by the predominantly transverse course of "regional arteries" (anterior sulcal arteries) within the central spinal cord. They noted that mechanical flattening of the cord tended to narrow the lumina of these vessels through stretching. Such cord distortion or flattening could be produced by cervical flexion, and was particularly pronounced in the presence of transverse spondylotic ridges. Furthermore, clinical observations clearly document deterioration in patients with spondylotic myelopathy coincident both upon cervical flexion and extension while the syndrome of acute traumatic central cord injury, to which spondylotics are peculiarly susceptible, characteristically results from a hyperextension injury. Radiographically, also, a complete obstruction to the cephalad flow of subarachnoid contrast medium during myelography can often be demonstrated only with the neck in dorsal extension.

Compression of radicular arteries, or veins, within the neural foramina has been a popular concept of the possible vascular pathogenesis of cervical spondylosis. Anatomical studies have demonstrated that the blood supply of the spinal cord is derived in greatest measure from a relatively few segmental vessels. In the cervical region two or more such dominant arteries enter the lower cervical segments in precisely the same area in which the bony-ligamentous changes of spondylosis are apt to be severe. The relatively greater sensitivity to hypoxia of anterior horn cells and posterolateral fiber columns would then explain the clinical specificity of the disease.
The exact mechanism of embarrassment of these radicular vessels is of itself a matter of dispute. The thesis of nerve root fibrosis, championed by Frykholm,\textsuperscript{12} is not widely accepted as a major factor in spondylotic myelopathy, though studies by Wilkinson\textsuperscript{20} and by Breig\textsuperscript{6} lend some support. In the presence of degenerative disease of intervertebral discs, presumed to be the initiating pathologic process in cervical spondylosis, some narrowing of the intervertebral foramina can usually also be demonstrated.\textsuperscript{10} The additional development of lateral bony spurs would then be expected to produce significant compression of nerve roots and their accompanying vessels.

Such bony changes should of course be readily and consistently visible by appropriate radiography. In fact, however, considerable disagreement exists as to the correlation of clinical and radiographic findings. Payne and Spillane\textsuperscript{18} have demonstrated radiographic changes typical of cervical spondylosis in patients with minimal or absent neurologic deficits. Some authors have declared the changes seen on plain roentgenograms, or even on myelography, to be of little or no value in estimating clinical severity of the disease.\textsuperscript{2,4,12,23} Nonetheless, several clinical-radiographic correlations have gained acceptance. It is generally conceded that patients with cervical spondylosis are more apt to have interspace narrowing and hypertrophic spurs and bars, though these changes may also be seen in people who do not exhibit clinical manifestations of spondylosis.\textsuperscript{17,18} In addition, patients with significant myelopathy generally have a decrease in the anteroposterior (AP) diameter of the cervical canal.\textsuperscript{6,9,13,16,18,26}

Thus, the predominant radiographic correlation of cervical spondylosis is with hypertrophic degenerative changes and decreased AP canal diameter, and would seem to support the notion of a purely mechanical compression of the spinal cord as the pathoanatomic basis of spondylotic myelopathy. That such a mechanism has been deemed unlikely in terms of the clinical features of the disease has been noted. Yet the clinically more popular notion of a vascular basis of pathogenesis has received little support from clinical-radiographic correlations. In an attempt to explore this apparent ambiguity, we have correlated the radiographic and clinical findings of a series of 54 patients with the clinically accepted diagnosis of cervical spondylosis who were seen at the Boston Veterans Administration Hospital between 1963 and 1965. Other patients, in whom plain x-rays and myelographic studies were inadequate for the purpose of this study, have been excluded.

Methods

A series of measurements were made at the four interspaces between C-3 and C-7, as demonstrated on plain radiographs and on cervical myelograms. Included were measurements of “pre-existing” sagittal canal diameter, height of posterior interbody spurs, “absolute” AP canal diameter (“pre-existing” sagittal diameter minus spur height), interpedicular width, width of cord shadow, and “sagittal cord diameter” (estimated by measuring from the anterior limit of the cord shadow within the column of contrast medium to the nearest point on the overlying laminae). The degree of foraminal encroachment and the degree of transverse interruption of the column of contrast medium (the severity of “myelographic bars”) were estimated semiquantitatively.

Results

Average measurements and correlation between findings on plain x-ray films and on myelograms have been presented in a separate report.\textsuperscript{24} Of special interest were the excellent correlations found between the height of posterior interbody spurs seen on plain films and the magnitude of transverse bars demonstrated on myelography; the lack of correlation found between the magnitude of transverse myelographic bars and various measurements of spinal cord widening was equally significant. It was also demonstrated that the diminished sagittal spinal canal diameter seen in cervical spondylosis was one dimension of a general decrease in canal size affecting both sagittal and transverse diameters.

The clinical-radiographic correlations in the present study were quantitated mathematically in terms of their correlation coefficient \textit{r} (a perfect correlation = 1.000) and its reliability factor \textit{P}. The correlation between the presence of foraminal en-
croachment demonstrated on oblique plain x-ray films and the clinical occurrence of objective radiculopathy compared with patients who complained only of subjective radicular pain was marginal \((r=0.335; P=0.04)\). Although patients with no objective signs of radiculopathy occasionally demonstrated significant foraminal encroachment, patients with radiculopathy almost always \((97\%)\) demonstrated at least some foraminal encroachment.

The existence of clinically evident myelopathy correlated fairly well with myelographically demonstrated transverse bars \((r=0.402; P=0.003)\), almost equally well with foraminal encroachment \((r=0.392; P=0.006)\), and somewhat less well with posterior interbody spurs demonstrated on plain x-rays \((r=0.268; P=0.05)\). A similar degree of correlation with myelopathy was found with measurements of minimal “pre-existing” sagittal canal diameters in each patient \((r=0.398; P=0.004)\), while the closest correlation of all was found with the “absolute” AP canal diameter, that is, the canal diameter minus the height of the posterior interbody spur at that interspace \((r=0.529; P=0.0002)\). Myelopathy was rarely seen in patients with “pre-existing” sagittal canal diameters greater than 16 mm or with “absolute” AP canal diameters greater than 14 mm. “Pre-existing” sagittal canal diameters less than 14 mm were most commonly associated with myelopathy, while no patient with an “absolute” AP canal diameter less than 8 mm escaped myelopathy.

Although narrowing of “pre-existing” sagittal canal diameter, “absolute” AP canal diameter and sagittal cord diameter were most severe in patients with myelopathy; they were also more severe in patients with objective radiculopathy than in patients with pain only. In contrast, the severity of foraminal encroachment, posterior interbody spurs and myelographic bars failed to show a consistent increase in severity in the radiculopathy group.

All patients with myelopathy were found to have significant abnormalities demonstrated on plain radiographs or involvement of at least one interspace on myelography, while the demonstration of significant abnormalities at all four interspaces was more often associated with myelopathy. Nonetheless the distribution of the number of levels involved was remarkably similar for those patients with myelopathy and those without.

Perhaps surprisingly, the poorest radiographic correlations of clinically evident myelopathy were found with measurements of maximum cord width per patient \((r=0.073; P=0.60)\) and with measurements of maximum increase in cord width at adjacent interspaces in each patient \((r=0.137; P=0.34)\). Despite these correlations, the following measurements were never exceeded except by patients who showed objective signs of clinical disease, either radiculopathy or myelopathy: cord width greater than 20 mm, change in cord width greater than 2 mm, canal width less than 22 mm, and cord/canal ratio greater than 90%. Although a considerably larger percentage of patients with myelopathy showed narrowing of canal width than did patients with radiculopathy, there was little difference between the two groups in measurements of absolute cord width or change in cord width. Thus, spinal cord flattening would seem to be a relatively unreliable radiographic concomitant of cervical spondylotic myelopathy.

**Conclusions**

As others have noted, the clinical features of radiculopathy and myelopathy of cervical spondylosis correlate imperfectly with the radiographic features of the disease. Yet, the apparent imperfections of these correlations is misleading. Although many people with radiographic findings typical of spondylosis fail to demonstrate the clinical features of involvement of the nervous system, typical radiographic findings almost invariably accompany neurologic involvement.\(^6\) This factor can be valuable in ruling out cervical spondylosis as an etiologic factor in obscure and atypical disease states.

This study has attempted to clarify the pathophysiology of the radiculopathy and myelopathy of cervical spondylosis by means of an analysis of radiographic data. A strictly mechanical, compressive etiology of observed radiculopathy was supported by finding a significant correlation with foraminal encroachment on oblique plain films. Radiographic evidence of foraminal encroachment was occasionally present without
clinical objective evidence of radiculopathy, but radiculopathy was almost never found without foraminal encroachment. Other reports have confirmed this finding.\textsuperscript{9,17}

The pathophysiology of spondylotic myelopathy remains more uncertain. Much pathological and physiological evidence supports a vascular basis, with vascular compression occurring most likely at a radicular level. If this indeed is the situation, one could expect to find a significant correlation between myelopathy and radiographically demonstrated foraminal encroachment. However, Crandall and Batzdorf\textsuperscript{9} failed to demonstrate foraminal osteophytes on plan x-rays in 36 of their 62 patients with spondylotic myelopathy, while Lees and Turner\textsuperscript{14} stated that none of their patients who presented symptoms of radiculopathy alone later developed myelopathy.

The present study did reveal a significant correlation between myelopathy and radiographically demonstrated foraminal encroachment, but it also demonstrated that clinically evident myelopathy correlated well with a narrowing of the “pre-existing” sagittal diameter of the cervical spinal canal. Furthermore, the closest clinical-radiographic correlation observed in this study was with narrowing of the “absolute” AP diameter of the canal. Similar correlations have been observed by others.\textsuperscript{5,9,20,21,25,30}

We feel that the correlation between myelopathy and foraminal encroachment may be fortuitous. The more significant correlation would seem to be with the presence of more medial hypertrophic changes, with the more lateral foraminal hypertrophic changes being secondary and less likely of pathophysiological significance.

Thus, while correlation of clinical myelopathy with foraminal encroachment is irrefutable and argues for a vascular basis of the myelopathy of cervical spondylosis, the far closer correlation with narrowing of the “absolute” AP canal diameter argues even more forcefully for a mechanical, compressive basis (with or without embarrasment of intrinsic cord vasculature), a thesis which has been held to be less attractive on clinical grounds.

We would like to suggest, however, one factor that has been little considered in prior studies of the pathophysiology of spondylotic myelopathy, namely, the shape of the cervical spinal canal. One major objection to the mechanical, compressive theory is the observation in pathologic studies in spondylotic myelopathy of a relative sparing of the anterior third of the spinal cord, even though this portion of the cord lies closest to the spondylotic bars.\textsuperscript{9,25} Another major objection to this theory has been the clinical observation that the resultant myelopathy is a predominantly motor one, with relative sparing of sensory tracts, especially the posterior columns. These objections are presumably based on a conception of the bony cervical canal as having roughly parallel anterior and posterior plane surfaces. That this is not the case has been emphasized in anatomic studies which clearly demonstrate the canal to be more nearly triangular in cross section, with steeply sloping dorsal laminae.

Thus a combination of anterior compression from bony ridges with posterior compression from the dorsal laminae or infoldings of the dura or ligamenta flava\textsuperscript{6} would result in maximum compression of dorsolateral portions of the spinal canal including the corticospinal tract (see Fig. 1). Further support for this concept of a combination of simultaneous dorsolateral and anteroposterior cord compression is provided by the correlation which we have demonstrated between sagittal canal diameter and interpedicral canal width: a canal narrowed in its AP diameter is likely also to be narrowed in width. This same phenomenon helps to explain the surprisingly poor correlation between myelopathy and spinal cord widening demonstrated myelographically. If cord compression were manifested in a unidirectional, anteroposterior fashion, widening of the cord would almost certainly result. But, if in addition to anteroposterior compression the spinal cord were subjected to bilateral posterolateral compression, the tendency for the cord to increase demonstrably in width would be sharply restricted.

Thus the spinal cord in cervical spondylosis is subjected to compression from, not two, but three directions, with the lines of force traversing the corticospinal tracts, the anterior horn cells, and the ventral thalamic decussation. Such a distribution of functional and structural derangement should result in spastic paraparesis, upper
extremity weakness and atrophy, and loss of pain and temperature perception in the upper extremities. It is precisely this combination of findings which typifies cervical spondylotic myelopathy clinically.\textsuperscript{9,14,17} The patient with a pre-existingly small cervical spinal canal who subsequently develops hypertrophic cervical spondylosis suffers repetitive trauma to his cervical spinal cord with each flexion and with each extension of his neck, as he alternately presses his cord anteriorly against the underlying spondylotic bars or in folds his dura and ligamenta flava into the dorsolateral aspect of his spinal cord, which has no room to escape by migrating forward. The restriction of this repetitive trauma either by a cervical brace or by adequate posterior or anterior surgical decompression undoubtedly underlies the good results achieved both by non-surgical and by surgical means.

![Diagram of cervical spinal canal and cord](image)

Fig. 1. Cross-sectional drawings of cervical spinal canal and cord. Upper: Normal. Lower: Cervical spondylosis with radiculopathy and myelopathy. Arrows emphasize triangular shape of canal, resulting in bilateral dorsolateral compression of cord.

Cervical Spondylosis

Summary

We have analyzed clinical-radiographic correlations in a series of patients with cervical spondylosis while studying the pathophysiology of the resultant radiculopathy and myelopathy. Our results support the concept of a purely mechanical, compressive etiology for spondylotic radiculopathy.

The etiology of spondylotic myelopathy has been less clear. Possible vascular factors have received much attention, but little supporting evidence is available. A mechanically compressive etiology has been less popular on clinical grounds, but received strong support in the present study. We have stressed the triangular shape of the cross section of the cervical spinal canal as an important factor in understanding both clinical and radiographic aspects of the disease.

References


