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Letter to the Editor

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Commentary on "Classification(s) of Cervical Deformity"

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To the editor,

I read with interest the abovementioned article that focuses on analysing the various classifications of cervical deformity described in the literature.¹ As discussed by the authors, the classification of cervical deformity is essentially based on underlying aetiology, focus of the curve apex, regional and global spinal alignment. The authors have discussed their own classification scheme that focuses on assisting decisions regarding surgical management. The various classifications schemes, including the one described by Kaidi and Kim¹ are designed on radiological observations of subaxial cervical spinal configuration and include cervicothoracic curvatures, alignments and deformities.

It is surprising that discussion regarding status of craniovertebral junction stability has been entirely ignored in these classifications.² Our observation is that the existing classification schemes have focussed their analysis on physical deformations and radiological observations and not so much the issue of instability in the atlantoaxial and subaxial cervical spine.

The authors could have included our classification of cervical deformities in their reference list.² Our contention is that atlantoaxial and/or subaxial spinal instability is a nodal point of pathogenesis of several spinal deformities. Apart from range of torticollis and short neck that are directly related to atlantoaxial instability, deformities in the cervical spine encountered in a number of pathological conditions that include degenerative spinal disease have their origin in spinal instability that more frequently includes atlantoaxial instability.^{2,3} Understanding of the underlying unstable spinal segments and their stabilization forms the cornerstone of surgical treatment.

We have recently described a novel clinical entity of central or axial atlantoaxial instability (CAAD).⁴⁻⁷ In CAAD, the atlantoaxial instability cannot be identified on the basis of conventional validated radiological parameters that include abnormal alteration of atlantodental interval or any evidence of neural or dural compression by the odontoid process. In CAAD, instability is diagnosed on the basis of alignment of facets in the lateral profile imaging, telltale radiological evidences and is confirmed by direct manipulation of bones of the region during surgery. Chiari formation, basilar invagination, syringomyelia, Klippel-Feil abnormality, platybasia, bifid arches of atlas, os-odontoideum and a range of so-called 'pathological anomalies' have their origin in CAAD and constitutes its tell-tale evidences.⁸⁻¹⁰ Our articles on the subject have observed that all these abnormalities when present discretely or in cohort are secondary to atlantoaxial instability, are naturally protective or adaptive in their role and are potentially reversible following atlantoaxial stabilization. Considering this understanding, we prefer to call Chiari 'malformation' as Chiari 'formation' and craniovertebral 'anomalies' as craniovertebral 'alterations.^{11,12}

For the first time in the literature, we discussed the issue of 'vertical' spinal instability as a nodal point of pathogenesis of spinal spondylosis.¹³ Vertical spinal instability is essentially telescoping of the spinal segments related to weakness of the muscles involved primarily in the standing human posture.¹⁴ Such listhesis or instability is difficult if not impossible to identify on conventional radiological imaging. It was speculated that is not disc space reduction or disc degeneration but listhesis of the subaxial facets where the muscles of the nape of neck and back of spine are focussed that is the initial manifestation of spinal degeneration that initiates a cascade of secondary events that culminate into pathology of spinal degeneration. The so-called pathological entities in degenerative spinal disease that include reduction in the intervertebral disc space, bulging of the disc into the spinal canal, osteophyte formation, bulging of the intervertebral ligaments that ultimately result in reduction in the spinal and neural canal are secondary manifestation or tell-tale evidences of vertical spinal instability.¹² Cervical deformities that include kyphotic and scoliotic deformities are also secondary manifestation of vertical spinal instability. Our articles discuss that all the secondary alterations observed in spinal degeneration that include spinal deformity are protective or adaptive in their role and are potentially reversible following stabilization of the affected spinal segments.¹⁵

On the basis of presence of vertical spinal instability with or without atlantoaxial instability, we classified cervical deformities into 3 groups.² Type 1 cervical kyphosis was when kyphosis was associated with atlantoaxial instability that was diagnosed on the basis of increased atlantodental interval. Kyphotic deformity was considered to be secondary to atlantoaxial instability and atlantoaxial stabilization was identified to be the treatment. Type 2 cervical kyphosis was when there was subaxial vertical cervical spinal instability. The vertebrae involved in the entire curvature of kyphosis were considered to be unstable and all these segments needed stabilization. Type 3 cervical kyphosis was when both CAAD and vertical spinal inability were identified to be the cause of deformity. Atlantoaxial and subaxial cervical spinal stabilization of the affected spinal segments was the treatment.

Postlaminectomy cervical spinal deformity and alteration in spinal curvature in cases related to neurofibromatosis and other tumors are also related to spinal instability.^{16,17} Spinal stabilization is the treatment.

Our classification system of cervical spinal deformities is on the basis of identifying the unstable spinal segments. Essentially, we observe that instability is the cause and stabilization of the affected segments is the treatment of spinal deformities.

We believe that the authors will acknowledge these concepts and consider modification in their classification scheme.

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