Vertical spinal instability and cervical spondylosis: Is it focal or multisegmental?

Standing human posture and disuse or misuse of spinal muscles leads to "vertical" spinal instability that is manifested at the facets.[1] Listhesis of superior facet over the inferior facet is the nodal point of the pathogenesis of degenerative spinal spondylosis. It was speculated that it is not the reduction in the water content of the disc or disc space reduction that is the primary factor that initiates the process of spinal degeneration, but it is facetal overriding as a result of instability.[1] The posteriorly located spinal muscles initiate and conduct all movements of the spine. We had earlier discussed that odontoid process and the intervertebral discs are the brain of all movements and paraspinal muscles that focus on the facets are the brawn of these movements.[2] Discs and odontoid process are like opera conductors that regulate all movements without playing any music by themselves.[2] The fulcrum of movements is focused at the facets. Anterior spinal muscles are thin and "curtain-like" and participate only marginally in movements. Instability of the spine is manifested at the facets and results in vertical reduction in spinal height. The intervertebral ligaments that include ligamentum flavum and posterior longitudinal ligaments buckle as a result. Ligamentum flavum is not hypertrophied or pathological, but it is buckled as a result of vertical height reduction. Similarly, posterior longitudinal ligament buckles and initiates the process of osteophyte formation. Disc height reduction does not seem to be a primarily pathological or age-related effect but is a response of vertical height reduction of the spinal segment. The sum effect is reduction in the dimensions of the spinal and root canal. Essentially, listhesis or overriding of the facets is the primary event, and all other effects are its consequences or are secondary in nature.

The facets being lateral and with an oblique profile are poorly visualized even on modern imaging.[3] The facetal overriding and instability cannot be appropriately assessed or evaluated by dynamic imaging. On the other hand, the secondary consequences of the reduction of the spinal and neural canal and compressive effects of posterior longitudinal ligament and ligamentum flavum are vividly visualized on imaging. Cord changes can also frequently be visualized. Essentially, the primary event of facetal instability is not radiologically evaluated, but its effects and consequences are relatively well seen. The "compressive" effects on the cord are so prominent and evident that the entire focus of surgical treatment is concentrated on "decompression" of the cord and provide it with a wide and elaborate "breathing" space. Consequently, the focus of treatment is on the effects of the problem and not toward its cause. The treatment focused on instability has now been identified to be the primary target. Facet distraction-arthrodesis or only facetal fixation is currently the pillar of treatment of degenerative spinal disease.[4-8] The secondary morphological effects on the spine have the potential to regress, once the primary problem is rectified.[9]

The spinal cord and roots have remarkable resilience and ability to tolerate compression and deformation. In long-standing or chronic situations, the spinal cord can get atrophied or develop "self-destructive" neural changes to prevent or delay the development of neural deficits. More than compression or deformation, it is subtle and repeated microtrauma or injury to the spinal cord as a result of instability that is the cause of neurological deficits or symptoms.

Muscle weakness and its related spinal instability is usually a more generalized phenomenon rather than being focal. Spinal degeneration is usually multisegmental. Clinical parameters and radiological investigations can guide toward the site of spinal instability. However, it is impossible to identify the unstable spinal segments only on the basis of analysis of

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radiologically affected segments. In the absence of reliable radiological guides, direct visual inspection of spinal segments in the vicinity of radiologically evident-affected regions manifested by osteophyte and ligamentum flavum-related sites of cord compression is probably the most effective way to identify and treat unstable spinal segments.[3]

Visual inspection of the facets and manual assessment of their abnormal mobility are probably an ideal method to identify the unstable spinal segments. Spinal instability can be present even in the absence of radiological evidence of cord compression. "Open" facetal articulation, abnormal bone alignments and movements, osteophyte formation over the facets, and similar such features are evidence of instability. Identification of instability on the basis of direct visual inspection needs experience in facetal handling.

Atlantoaxial joint is the most mobile joint of the body. Its special morphological character that allows it circumferential movements also subjects it to increased possibility of developing instability. Degenerative spinal changes that are a result of instability are most likely to develop at the atlantoaxial facetal articulation. Identification and treatment of atlantoaxial instability are the most crucial issue in the success of surgery in degenerative spinal disease.[10-12] Atlantoaxial instability may be present even in the absence of any radiological evidence of instability or feature of spinal cord compression. Radiological evidence of facetal malalignment and direct visual inspection can lead to the identification of atlantoaxial instability. We have labeled such instability as central or axial atlantoaxial instability.[13,14]

In general, it is crucial to be aware of the presence of spinal instability despite the absence of its demonstration that is based on radiological evidence of cord compression. Inability to recognize the presence of instability in segments adjacent to those seen on radiological imaging is probably the most important cause of development of "adjacent segment disease" as a delayed consequence of treatment of cervical spondylosis. Direct surgical identification of instability is more easily possible during the posterior surgical approach. Exploration of the segments adjacent to those segments that are identified to have known evidence of spinal degeneration and cord compression may be crucial to comprehensibly treat spinal degeneration and to avoid delayed adjacent segment disease. Being aware of the fact that atlantoaxial instability may be present in the absence of radiological evidence is crucial.

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