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Editorial

Central or axial atlantoaxial instability: Expanding understanding of craniovertebral junction

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The atlantoaxial joint is the most mobile joint of the body. Because of a large array of movements that originate from here, there is a larger possibility of the movements becoming abnormal, resulting in instability of the region. Conventionally, atlantoaxial instability has been diagnosed on the basis of abnormal alterations in the atlantodental interval and by radiological evidences of indentation of the odontoid process into the cervicomedullary cord.[1,2] The fact that there can be atlantoaxial instability without the evidence of odontoid process-related cord compression appears to be a beginning of a new era in the understanding of the subject. Atlantoaxial instability can be symptomatic even when there is no clear evidence of compression of the cord by the odontoid process. Instability of the atlantoaxial vertebrae can be in the presence of or even in the absence of facetal malalignment. Atlantoaxial instability, apart from being anteroposterior or horizontal, can be axial, central, or vertical as well. Identification of the later form of atlantoaxial instability widens the scope of evaluation of the craniovertebral junction and offers an opportunity for its therapeutic exploitation.

As we become mature in our understanding of the craniovertebral junction pathology, we realize that the instability of the most mobile joint of the body can be associated with or may be the prime pathogenetic issue in a number of common clinical entities such as cervical spondylosis, ossified posterior longitudinal ligament, cervical kyphotic and scoliotic deformities, Chiari malformation, syringomyelia, and basilar invagination.[3-5] In majority of these situations, the atlantoaxial instability is not anteroposterior but is central or axial.[6,7] Ignoring "central" atlantoaxial instability in such cases can be a major reason of failure of treatment.

In cases where there is compression or indentation of the neural structures, the symptoms are relatively acute. When there is instability in the absence of direct neural compression by the odontoid process, or the instability is central or axial, the symptoms are longstanding or chronic in nature. Majority of musculoskeletal and neural responses in such cases are protective in nature, designed to protect the neural structures and stall or delay neurological deficits.[8,9]

The facets of atlas and axis are like rectangular blocks placed like bricks, one over the other.[10] This profile alignment is in variance with the rest of the spine, where the facets are aligned in an oblique or angled fashion. While the movements at the atlantoaxial facet joint are circumferentially transverse, the movements are in the form of telescoping in the subaxial facets. The facetal malalignment is relatively easily identified in the atlantoaxial joint rather than in the subaxial spine due to this structural difference. Due to the brick-like arrangement, the facets of atlas and axis can sustain the weight-bearing function, despite the instability. On the other hand, the facets can be unstable, despite being in alignment. We call such instability as central or axial instability.

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We classified atlantoaxial "facetal" instability into three types.[6] In Type 1 instability, the facet of atlas is dislocated anterior to the facet of axis. Type 1 facetal malalignment simulates the alignment of vertebral bodies in lumbosacral spondylolisthesis.[11] The atlantoaxial instability in such cases is anteroposterior or horizontal. In Type 2 instability, the facet of atlas is dislocated posterior to the facet of axis, and the malalignment simulates retrolisthesis of the facets. In Type 3, the facets are in alignment, but manual handling of bones during surgery can identify the presence of instability. The atlantoaxial instability in Type 2 and 3 is central or axial in nature.

Type 1 facetal instability is associated with an increase in the atlantodental space and indentation of the odontoid process into the craniocervical spinal cord. The symptoms are relatively acute in such cases. This type of atlantoaxial instability is associated with the more frequent forms of atlantoaxial dislocations where the symptoms are relatively acute. Posttraumatic atlantoaxial dislocations and Group A basilar invagination is usually associated with Type 1 atlantoaxial facetal instability.

In Type 2 and Type 3 facetal instability, the odontoid process does not indent into the cord and the atlantodental interval is not increased. The cerebrospinal fluid spaces around the cord and posterior to the odontoid process can be entirely normal in these cases. As the neural deformation and compromise is not acute, the disease process is longstanding and chronic in nature. The musculoskeletal and neural deformities are more predominant in these subgroups. The symptoms are not related to direct neural compression, but seem to be related to secondary and protective bodily responses that are initiated to stall or delay the symptoms related to neural compression.

We recently identified the premier role of instability in the pathogenesis of degenerative spondylosis of the spine. Muscle weakness related to disuse or abuse and lifelong-standing human position has destabilizing effects on the spine.[12-15] Facetal overriding in the subaxial spine results in a "telescoping" effect. The vertical instability of the spine as a result of telescoping of the vertebra results in a cascade of secondary effects that cumulate to result in spinal and root canal stenosis and subsequent related symptoms.[16] Ligamentous buckling, that includes ligamentum flavum and posterior longitudinal ligaments and associated osteophyte formation and disc space reduction, appear to be secondary effects of vertical spinal instability.[17] The net effect of instability is the reduction in the spinal and root canal dimensions. We discussed the surgical treatment for degenerative spine and evaluated the validity of only fixation as the philosophical treatment modality.[18] The atlantoaxial region is generally excluded from the umbrella of degenerative spinal disease. Craniovertebral junction has been considered immune to degenerative diseases. We have recently realized that atlantoaxial instability can be frequently associated with degenerative spinal disease.[4,5] Degeneration-related instability could primarily involve the atlantoaxial joint.[19] Degeneration of the cervical spine can be associated with degenerative involvement of the atlantoaxial joint. It may be possible as well that degenerative affection of the atlantoaxial joint may be the primary event in the development of subaxial spinal degeneration. The presence of abnormal ossifications in the vicinity of the atlantoaxial joint or around the odontoid process can be indicators of instability.[19] Buckling of the posterior longitudinal ligament or ossifications posterior to or around the odontoid process can be indicators of atlantoaxial instability.[20] In cases with degenerative spine, the facetal instability is more commonly of Type 2 or of Type 3.

We recently identified instability as the major or a sole pathogenetic factor for ossification of the posterior longitudinal ligament (OPLL) and its consequent devastating symptoms.[21] Stabilization of the involved facets was proposed as treatment for this vexed clinical problem. We identified the presence of atlantoaxial instability in a number of cases with OPLL, particularly those that extended in the high cervical spine. We identified the presence of Type 2 or Type 3 atlantoaxial facetal instability in such cases. As our experience is growing in the field, we realize that ignoring the presence of central or axial atlantoaxial instability can be a major cause of treatment failure.

Type 2 and Type 3 facetal instability is associated with chronic instability as seen in Group B basilar invagination. The odontoid process may not directly indent into the spinal cord, but several musculoskeletal and neural alterations and clinical manifestations are direct consequence of instability. This fact can be seen by analyzing the postoperative clinical recovery following atlantoaxial stabilization that can be remarkable and in the immediate postoperative period. We recently evaluated cases with Chiari 1 malformation and identified the presence of Type 2 and 3 facetal instability.[3] We speculated that Chiari 1 malformation is secondary to or a result of Nature's protective measure in response to atlantoaxial instability. We philosophized that Chiari 1 malformation is like Nature's airbag that is placed in position in response to atlantoaxial instability and is an attempt to prevent pinching of critical neural structures between bones.[22] Marked clinical improvement following surgery aimed at atlantoaxial stabilization points toward the validity of the concept.

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