Degenerative arthritis of the craniovertebral junction

A review of related publications in the literature suggests that cervical spinal degeneration has more often been identified and surgically treated in the lower cervical spine, less frequently in the upper cervical spine, and only rarely in the craniovertebral junction. Our observations identify that degenerative changes in the craniovertebral junction are relatively frequent and are probably underdiagnosed and undertreated.

Our recent articles relate the term “degeneration” to “instability.” “Vertical” spinal instability (VSI) related to weakness of muscles that partake in standing human position and facilitate major motor activities is the nodal point of genesis, propulsion, and propagation of process of degeneration. This understanding is in variance with the existing concept that disc space reduction or “old” age-related loss of water content of the disc form the basis of spinal degeneration. VSI results in telescoping of the spinal segments and initially or primarily leads to listhesis of rostral facet over caudal facet, a process termed as retrolisthesis of facets.

All the known so-called “pathological” alterations in spinal degeneration such as buckling of the intervertebral ligaments that include ligamentum flavum and posterior longitudinal ligament, osteophyte formation, and disc space reduction that are ultimately associated with reduction in the spinal canal and intervertebral foraminal height are secondary alterations in the face of VSI. Our studies in the craniovertebral junction and spine conclude that all the “secondary” issues related to spinal instability have a natural protective role and more importantly are reversible following stabilization of the affected spinal segment.

The relevant concept is that more mobile a spinal segment, more likely it is to develop instability and spinal degeneration. Lower cervical spinal segments (C4-5, C5-6, and C6-7) and lower lumbar spinal segments (L4-5 and L5-S1) have been more often associated with degeneration. These “junctional” spinal segments are the more mobile regions of the spine and are consequently more likely to develop instability. Our concept is that it is not neural compression or deformation, but it is spinal instability that is the cause of symptoms. Consequently, there can be symptoms without any radiological evidence of neural compression.

Both clinical features and radiological guides direct toward the unstable spinal segments. Identification of instability in segments adjoining those suggested by clinical and radiological guides can be done by manual manipulation of bones during surgery. Ignoring such instability appears to be a cause for the so-called “adjacent segment disease” often encountered in cases operated for degenerative spinal issues.

The process of spinal “degeneration” or spinal “instability” is more often chronic or longstanding in nature. Secondary natural alterations are more prominent in cases where the instability is subtle. In cases where the secondary natural processes are prominent, the symptoms are relatively minor or subtle and longstanding but are relentlessly progressive. It appears that once the instability begins, the process of spinal degeneration becomes progressive. However, if exercising and muscle strengthening therapy can restore the effectiveness of the affected muscles, the instability can probably be reversed.

Stability and mobility are hallmarks of the craniovertebral junction. The occipitoatlantal joint is the most stable, and the atlantoaxial joint is the most mobile joint of the body. Degenerative alteration in the occipitoatlantal joint is uncommon, and it is probably related to the strength of the joint and to the limited range of movements. Considering the wide-ranging and circumferential mobility in the atlantoaxial articulation, the probability of complex instability or degeneration is highest when compared to the rest of the spinal segments.

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Probably related to longstanding and subtle nature of instability, a range of natural “protective” measures, and the presence of significant buffer of cerebrospinal fluid in the vicinity, manifestation by clinical symptoms and neurological deficits can be delayed. Pain and chronic spasms of the neck muscles, both natural processes aim to limit the range of the neck movements and prevent or delay the impact of instability. Reduction in the atlantoaxial joint space, buckling of the posterior longitudinal ligament, retroodontoid thickening of ligaments or pseudotumor formation, para-articular osteophyte formation, ossification in the vicinity of the ligaments of the region (apical and transverse), facet bone destruction or hypertrophy, and several similar events are indicators of the presence of atlantoaxial instability, are secondary in nature, are naturally protective and have the potential of reversal following atlantoaxial stabilization.\[1\] [Figures 1-5].

Chronic atlantoaxial instability can be associated with short neck, short head, short spine, torticollis, dorsal spinal kyphoscoliosis, basilar invagination, Chiari formation, syringomyelia, Klippel-Feil abnormality, platybasia, C2-C3 fusion, assimilation of the atlas, bifid arches of the atlas and axis, and a range of other secondary alteration.\[7-16\] Subaxial spinal instability is frequently observed in cases with chronic atlantoaxial instability that is identified to be “degenerative” in nature.\[17\] The presence of disc bulges at the C2-C3 spinal segment is a frequent observation and is more often a secondary alteration to primary atlantoaxial instability.\[18\] Disc bulges indenting into the spinal cord at the lower cervical spinal segments can be observed. It is critical to evaluate if the changes in the subaxial cervical spine are secondary events related to atlantoaxial instability or are primary in nature.

On the basis of alignment of the facets of atlas and axis on lateral profile imaging and direct observation of instability by manual manipulation of bones of the region, we classified
atlantoaxial instability into three types. Type 1 atlantoaxial instability is when the facet of atlas is dislocated anterior to the facet of axis. In such cases, atlantodental interval is increased, and there is neural and dural compression by the odontoid process. In such cases, atlantoaxial instability is more often of acute or relatively acute nature, symptoms are more pronounced and secondary natural alterations are not frequent or prominent. Type 2 atlantoaxial instability is when the facet of atlas is dislocated posterior to the facet of axis. Type 3 atlantoaxial instability is when the facets are in alignment. In both Types 2 and 3 atlantoaxial instability, atlantodental interval may not be abnormally altered, and there might not be any neural or dural compression by the odontoid process. Such types of atlantoaxial instability are labeled as “central” or “axial” atlantoaxial instability (CAAD). CAAD is identified by telltale clinical and radiological evidence and by the identification of musculoskeletal and neural alterations and is confirmed by direct manipulation of bones during surgery. Degenerative alterations in the craniovertebral junction are usually associated with CAAD. Symptoms are “longstanding” and secondary alterations in the craniovertebral junction and subaxial cervical spine can be prominent. Compression of the dural or neural tube is not the hallmark. Identification of the presence of atlantoaxial instability and its appropriate treatment can result in dramatic clinical recovery.

For the sake of understanding of the participant and for formulating a surgical strategy, we divided the craniovertebral junction degeneration into three types.

Type 1 atlantoaxial degeneration: the instability in such cases is essentially limited to the atlantoaxial joint. There can be telltale evidence of instability in the form of reduction in the joint space, osteophyte formation, retroodontoid “pseudotumor, buckling of the atlantoaxial ligaments, osteophyte formation in the region, facetal destruction or hypertrophy, evidence of pseudo-fusion of the atlantoaxial facetal articulation, and similar such alterations. The affection of subaxial spinal segments is not prominent. Surgical treatment in such cases is atlantoaxial fixation with the aim to achieve atlantoaxial arthrodesis.

Type 2 atlantoaxial degeneration: in such cases, in addition to atlantoaxial instability and its consequences, there is secondary affection of subaxial spinal segments. More often C2-C3 disc bulges and evidence of adjoining spinal cord compression. The surgical treatment in such cases is atlantoaxial stabilization. Inclusion of the subaxial cervical spine in the fixation construct is not necessary.

Type 3 atlantoaxial degeneration: In patients with Type 3 atlantoaxial degeneration, there is multisegmental subaxial spinal degeneration. The patients are generally disabled and might have evidence of severe myelopathy. The surgical treatment in such cases is atlantoaxial and multisegmental subaxial spinal fixation. More recently, we identified a modified technique of atlantoaxial fixation that included the section of muscles attached to C2 spinous process and trans-articular C2-C3 fixation. The technique retained the rotatory movements executed by muscles attached to the transverse process of atlas and obliterated flexion-extension movements executed by the muscles attached to the C2-spinous process. We have advocated that all these patients need “only-fixation” of the affected spinal segments and despite radiological evidence of spinal cord compression, “decompression” by the resection of bone and soft tissues can have negative consequences.

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