

Editorial

Atlantoaxial instability associated with single or multi-level cervical spondylotic myelopathy

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Single or multi-level cervical spondylotic disease is commonly encountered spinal ailment. The clinical entity has been under extensive discussion for over a century. A number of treatment protocols have been advocated to address the issue. Disc degeneration or disc space reduction has been the most accepted pathological event that has been incriminated to be the starting point that initiates a cascade of pathological events that ultimately lead to development of single or multiple level myelopathy. Disc space reduction and loss of its water content have been related to “old” age or to disuse or misuse of the spine. A number of secondary pathological events subsequently evolve that are both anterior and posterior to the spinal cord and even circumferential to it. Osteophyte formation and ligamentary hypertrophy result in the spinal canal and intervertebral neural foraminal compromise and consequently in symptoms of myelopathy and/or radiculopathy. The entity has been considered to be “stable” and the aim of surgery is essentially to “decompress” the neural structures by removing the compressing elements of protruded disc, osteophytes, ligaments, and bone indentations. Instability is generally considered to be an issue, that is, a consequence of the surgical treatment that involves removal of soft tissues and bones during the process of decompression of the spinal cord and the nerve root.

Goel discussed the issue of vertical instability related to the inadequate or suboptimal functioning of the muscles of the spine as the primary nodal point of the genesis of the entire process of spinal degeneration.^[1-3] The instability is related to a consequence

of long-term or life-long standing human posture. The instability of the spine is manifested at the site of movements of the spine, namely the facets. Vertical instability or telescoping of the spinal segments and not the disc degeneration seems to be the primary event in spinal degeneration. All other events such as ligamentous buckling (identified as pathological hypertrophy), osteophyte formation (related to or a consequence of buckling of the posterior longitudinal ligaments), and reduction of the disc space are secondary events and are related to “telescoping” of the vertebral segments.^[4] The lateral location of the facets makes identification of instability using conventional radiography and even the modern computer-based imaging difficult or impossible. On the other hand, identification of disc space reduction, osteophyte formation, hypertrophy of ligamentum flavum, and even spinal cord changes are relatively easy. As the instability or its evidence was not identified, the entire thrust of pathogenesis has been historically laid on the disc degeneration and disc space reduction. Identification of the fact that facet instability and their overriding is the primary issue in spinal degeneration has the potential of revolutionizing the treatment strategies that are conventionally used. Essentially, the theory suggests that stabilization of the spine is necessary in cases with degenerative spinal disease and “decompression” of the canal is not a rational form of treatment.^[3] Goel recently advocated that “only fixation” of the affected spinal segments is the rational

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and effective treatment of spinal degeneration.^[5-7] Removal of bones — corpectomy from anterior and laminectomy from posterior, ligaments, osteophytes, and discs is not necessary and can only be counter-effective in the long run.

The craniovertebral junction is generally excluded from the umbrella of degenerative diseases. In cases with multi-level spinal spondylotic disease, the discussion generally ends at C3-4 and rarely at C2-3 levels. As we mature in our understanding in spinal degeneration, we have realized that craniovertebral junction is an equal if not the senior partner or a contributor to spinal degeneration.^[8,9] Degenerative changes are relatively frequent at the craniovertebral junction.^[8] Craniovertebral region degeneration is essentially observed in the region of its most mobile segment at the atlantoaxial facet joint.^[8] Occipitoatlantal joint degeneration is seldom observed, as the movements at this site in normal life are only limited, and stability is the hallmark of this joint. Like in the rest of spine, the essential element in craniovertebral junction degeneration is instability. Instability at the atlantoaxial joint is the primary point of pathogenesis, and all other features are secondary in nature and are probably protective in function. The degeneration at the craniovertebral junction is manifested by reduced atlantoaxial joint space that results in superior and posterior migration of the odontoid process resulting in generally subtle, but sometimes-severe basilar invagination and relatively “fixed” or only subtly mobile atlantoaxial dislocation.^[10] Perifacetal and peri-odontoid calcifications are evidence of “secondary” osteophyte formation and seem to be an attempt of nature to provide stability to the region.^[11,12] Osteophyte formation in this case and in cases with spinal degeneration at other sites is equivalent to peri-dental crust formation that is indicative of instability of the tooth and seems to be an event that attempts to stabilize the tooth-gum junction. Identification of features of degeneration around the facets and the odontoid process signals the presence of instability of the atlantoaxial joint. The frequently recognized entity of retro-odontoid calcification/ossification simulates osteophyte formation and is suggestive of atlantoaxial instability.^[11]

While more frequently and commonly in atlantoaxial dislocation or instability the facet of atlas dislocates anterior to the facet of axis (Type A facet instability), we recently identified posterior dislocation of facet of atlas in relationship with the facet of axis (Type B facet instability).^[13] We identified that even when the facets are in alignment there can be instability at the atlantoaxial facet joint (Type C facet instability). Apart from radiological characteristics, identification of instability of the facets should also be based on analysis of clinical parameters. On some occasions, the instability can be identified only during direct manual handling of the atlantoaxial bones during surgery. While Type A atlantoaxial facet instability is usually an acute phenomenon, Type B and Type C facet instability are generally associated with long-standing or chronic dislocations such as those associated with Group B basilar invagination and cases having Chiari malformation.^[14] Atlantoaxial dislocations associated with cervical spinal degeneration are usually long-standing in nature and are more frequently of Type B or Type C.^[9]

While facet instability at the subaxial spine is difficult to diagnose on conventional imaging, identification of atlantoaxial facet instability can be relatively straightforward as the facets of atlas and axis are rectangular and box or brick-like and are placed one above the other.^[15-17] It is crucial to realize that facet instability is the primary issue in atlantoaxial instability and in the subaxial instability related degeneration. The more mobile the region is, more are the chances that there will be affection or degeneration of its function. Atlantoaxial facet instability and its “degeneration” can be more frequent than the instability elsewhere in the spine. Diagnosis of instability on the basis of clinical evaluation and radiological parameters can be crucial and can provide an opportunity for treatment and symptomatic relief.

Craniovertebral or atlantoaxial degeneration can be a primary and isolated event. We had identified earlier that chronic atlantoaxial dislocations could be associated with short neck and torticollis. The shortening of neck and several musculoskeletal alterations in such a situation are not a primary phenomenon but can be secondary and naturally protective in its function.^[18] The shortening of neck can result from a reduction in the disc space, osteophyte formation, and can have other features that are generally associated with spondylotic spinal degeneration. In very long duration instability, bone fusions (Klippel-Feil abnormality) can be observed, more frequently at occipitoatlantal joint and C2-3 vertebrae. The spondylotic cervical spinal changes are secondary to primary atlantoaxial instability. Treatment of atlantoaxial instability is necessary in such cases, and the secondary alterations have the potential to spontaneously resolve. We hypothesized that there is a potential for osteophytes to resolve and bone fusions to un-fuse following surgical stabilization of the atlantoaxial instability.^[19]

In cases with single or multi-level cervical spinal degeneration, it is mandatory to assess and identify the presence of atlantoaxial instability. If the presenting neurological deficits are more or unusually pronounced and significant when related to the extent of spinal degeneration seen on radiology, atlantoaxial instability can certainly be considered and evaluated. Ignoring the presence of atlantoaxial instability, particularly in multi-level spinal degeneration, can lead to incomplete or a failed surgical procedure. We recently identified that in a significant percentage of cases with multi-level cervical spinal degeneration there is atlantoaxial instability.^[9] It is not yet clear to us if the atlantoaxial instability is the primary or an associated event in these cases. Accordingly, it is not possible to theorize if only atlantoaxial fixation would suffice in such cases.

We suggested that “only fixation” of the affected spinal segments is the optimum form of treatment for spinal degeneration.^[5-7,20] This is based on our evaluation and understanding that instability at the facets is the primary event in spinal degeneration. It is still unclear if the instability at multiple cervical levels always or only sometimes includes atlantoaxial instability. However, it is clear that atlantoaxial instability is frequently associated with multi-

level spinal degeneration. Stabilization of the atlantoaxial facet joint can result in remarkable neurological recovery.^[9,21-23]

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