Ossification of posterior longitudinal ligament and cervical spondylosis: Same cause - Same treatment

There has been an ongoing discussion for decades about the causal relationships between ossification of posterior longitudinal ligament (OPLL) and cervical spondylosis. Most authors believe that OPLL is a discrete clinical entity and it can be associated with cervical spondylosis in approximately 25% of cases.

Over several decades, the pathogenesis of cervical spondylosis has been convincingly related to primary disc degeneration, disc space reduction, and subsequent relentlessly progressive pathological processes such as osteophyte formation, ligamentum flavum hypertrophy, facetal retrolisthesis, and similar such events that eventually result in spinal and neural canal stenosis and related neurological symptoms and deficits. Decompression of the neural structures by removal of the offending bone and soft-tissue elements by anterior cervical route by single or multiple level corpectomy or by posterior route by laminectomy or foraminotomy or by laminoplasty is the accepted and widely practiced mode of surgical treatment. As multiple level bone removal has been identified to result in instability in the long-run, a host of stabilization procedures have been discussed.[1]

In general, the exact pathogenesis of OPLL has only been speculated as no definite or conclusive incriminating factor has been identified on the most instances. A number of genetic, dietary, environmental, and systemic issues have been identified to be the possible factors that lead to the formation of abnormal calcification/ossification of the posterior longitudinal ligament that encroach into the spinal canal, compress the spinal cord, and lead to progressive symptoms related to myelopathy.[2,3] Location of the ossification anterior to the spinal cord and posterior to single or multiple vertebral bodies make direct surgical resection a challenging and dangerous surgical issue. Some authors have identified that both dynamic and static processes come into play and produce symptoms.[4] As in cervical spondylosis, decompression from anterior or posterior spinal routes have been identified to result in satisfactory relief from symptoms. A number of surgical strategies have been described that focus on decompressing the compressed spinal cord. The need for stabilization of the spinal segments following bone and soft-tissue decompression continues to be a subject of intense clinical discussion.

In the year 2010, we identified that vertical instability that was focused at the point of fulcrum of spinal movements at facet joint was the nodal point of the pathogenesis of process of spinal degeneration. The weakness of the muscles related to their disuse or misuse or injury and standing human posture was incriminated to initiate and propel the process of degeneration.[5,6] On the basis of our experience, we identified that facetal distraction resulted in or has the potential to result in regression of all the known pathological spinal events such as osteophyte formation, ligamentum flavum hypertrophy, increase in the disc space height, and increase in the spinal canal and neural foraminal height.[5–8] As we progress in our clinical understanding, we realize that “only-fixation” of the involved spinal segments by transarticular technique of facetal fixation and the operation aimed at arthrodesis of the affected segments was the optimum and rational surgical treatment. It was observed that there was no need for any distraction or decompression of spinal bone or soft-tissue elements.

Our current understanding is that in several cases of multilevel cervical spinal degeneration, particularly those presenting with severe symptoms related to myelopathy have instability of the atlantoaxial joint.[10] Identification and treatment of atlantoaxial instability forms an important component of surgical treatment. Atlantoaxial instability might not be identified by conventionally described
radiological characters such as abnormal alteration of atlantodental interval on dynamic imaging or any evidence of dural or neural compression. We discussed the clinical entity of “axial” or “central” atlantoaxial instability. Such atlantoaxial instability is diagnosed on the basis of evaluation of alignment of facets of atlas and axis and direct observation of status of facets and the joint by physical assessment and evidence and manual manipulation of the bones of the region. The exact role of atlantoaxial instability in the initiation and progression of degenerative spondylotic changes in the subaxial spine remains to be identified. However, it is crucial to evaluate the status of the atlantoaxial joint especially in high cervical spondylosis and in cases where the neurological deficits are significant and disproportionate to the extent of spinal changes observed on imaging. The author is convinced that atlantoaxial instability is significantly common in cases with multiple levels spinal degeneration and is probably an underestimated and undertreated entity. Failure to treat atlantoaxial instability could be the cause of failure of surgical treatment.

It was realized that multiple segmental spinal instability was also the nodal and primary pathogenetic issue in cases with OPLL. Atlantoaxial instability was relatively more often associated than in cases with only cervical spondylosis. Atlantoaxial instability was not only frequent in high cervical OPLL but was also identified in low cervical OPLL. Atlantoaxial and subaxial spinal fixation essentially formed the basis of surgical treatment in these cases.

From our experience in treating both cervical spondylotic disease and OPLL, we identify that spinal instability is the common factor. OPLL seems to be a subsequent or delayed stage of processes affecting cervical spondylosis. Multiple segment spinal stabilization is the treatment both for cervical spondylosis and for OPLL. While the inclusion of the atlantoaxial join in the fixation process is mandatory in almost all cases with cervical OPLL, it is necessary in selected cases particularly where the neurological deficits are significantly severe and in cases with high cervical spinal degeneration.

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