Atlantoaxial and subaxial cervical spinal fixation: Can it revolutionize surgical treatment of cervical myelopathy related to Ossified posterior longitudinal ligament?

Ossification of posterior longitudinal ligament (OPLL) can lead to relentlessly progressive symptoms of cervical myelopathy and crippling neurological symptoms and deficits. The presence of ossification anterior to the bulk of cervical cord that is positioned in a relatively compact manner in the spinal canal presents a complex therapeutic challenge. Although the abnormal ossification can sometimes be "soft" and brittle, often it can be firm and bony. It can be densely adherent to the dura and can even transgress the dura and directly indent or invade into the spinal cord neural tissue. The OPLL is known for its relentless and progressively growing character. The natural history of untreated OPLL has been evaluated, and most studies have identified that the ossification grows in dimensions over time and symptoms are progressive. OPLL frequently presents itself at an advanced stage when it occupies a significant dimension of the canal. The "pathological" bony intrusion results in compromise of the space for cervical spinal cord and the patients present with symptoms related to progressively increasing cervical myelopathy. OPLL can extend both vertically and transversely making surgical exposure of its entire extent a formidable surgical issue. Surgical attempts to directly remove the ossification by multilevel corpectomy or to indirectly widen the spinal canal dimension by wide decompressive laminectomy or laminoplasty have both been associated with severe neurological deterioration. The possibility of such devastating postoperative neurological deficits has made cervical OPLL a surgeon's nightmare. In general, the treatment of OPLL is riddled with controversies and opinions, and the search for the optimum form of treatment continues.

Although identified worldwide, literature survey suggests OPLL is more prevalent in Asian countries. Although several theories have been put forth with regard to the pathogenesis of OPLL, the exact hypothesis is only at the speculation level.[1-3] It does appear that OPLL is a complex and multifactorial disease process that combines environmental, genetic, metabolic, and degeneration related or unrelated mechanical factors. It has been observed that there is an increased prevalence of OPLL in patients with hypoparathyroidism, acromegaly, and diabetes.[4] OPLL has been identified in association with other musculoskeletal diseases such as diffuse idiopathic skeletal hyperostosis, ankylosing spondylitis, and spondyloarthropathy.[5,6]

The pathogenetic relationship of OPLL with cervical spondylotic myelopathy is a subject of intense analysis. It remains unclear whether OPLL is only an advanced variant of cervical spondylosis. Epstein reported that more than 25% of patients having cervical spondylosis have some degree of OPLL. It is unclear if OPLL simulates osteophytes in its pathogenesis or if OPLL is an advanced version of osteophyte formation.[7,8]

The role of instability in the development and progression of the OPLL and in maturation of clinical symptoms is not entirely clear. Some investigators point out that the mechanical stress induces a biochemical response that leads ultimately to osteogenic induction in the ligament cells and formation of OPLL.[9-11]

OPLL is classified into the local, segmental, continuous, and mixed types based on imaging findings. Considering that the ossification extends over affected spinal segments and frequently courses through the mobile transition zone, the general opinion is that ossification by itself participates in adding to the strength of spinal stabilization. Although instability has been considered an issue in the management of OPLL, it is more related to instability that possibly

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accrues after the wide bone decompression that is generally necessary to remove the entire extent of OPLL or to widen the spinal canal dimensions. Issues of kyphosis and spinal instability have been discussed on several occasions after anterior and posterior decompressive surgery.[12]

The stark presence of intraspinal ossified mass and deformation of the spinal cord has diverted the focus of understanding to cord compression and to reduced spinal canal volume. We recently proposed that the nodal point of the pathogenesis of both cervical spondylosis and OPLL is spinal instability.[13-15] Our multiple recent articles have identified that "vertical spinal instability" that is manifested at the level of facets[16] is primarily related to pathogenesis of both degenerative spondylosis and OPLL. As we mature further into analysis of the subject, we realize that atlantoaxial instability is frequently, if not always, associated with multisegmental cervical spondylotic degeneration and with cervical OPLL.[17,18] Literature search suggests that discussion on the subject of cervical spondylosis and of OPLL is limited to subaxial cervical spine. The role of atlantoaxial joint that is most mobile joint of the body and is most prone for instability is underestimated and ignored from the discussion of pathogenesis and treatment. Understanding of the fact that atlantoaxial and spinal instability is the defining factor in the pathogenesis of OPLL and in the development of symptoms related to myelopathy has the potential of revolutionizing the treatment modalities.

The lateral location of the subaxial facets and their oblique profile makes identification of instability difficult or impossible even with modern computer-based imaging. Experience in the handling of facets and identifying the presence of instability by physical observation of weak or open articular cavity and direct manipulation and handling of the bones of the region can assist in determining the presence of instability.

Identification of the fact that there can be atlantoaxial instability even in the absence of radiological evidence of instability on dynamic imaging has expanded the scope of understanding of pathology of the region and in instituting a correct form of treatment. Identification of atlantoaxial instability has been traditionally done by criterion of the presence of abnormal atlantodental interval on dynamic imaging. However, understanding of the fact that there can be atlantoaxial instability without atlantodental interval affection and without any radiological evidence of dural tube or neural compression has a defining influence in the treatment of a number of clinical entities such as basilar invagination, Chiari malformation, syringomyelia, cervical spondylosis, Hirayama's disease, and OPLL among others.[19-22] We labeled such atlantoaxial instability as central or axial atlantoaxial instability.[23] Atlantoaxial instability was diagnosed on the basis of facetal malalignment or by direct bone manual manipulations during surgery.[23] Evaluations were done on sagittal imaging of CT scan with the head in neutral position and the cuts that passed through the atlantoaxial facets. In type 1 atlantoaxial facetal instability, the facet of atlas was dislocated anterior to the facet of axis. In type 2 atlantoaxial facetal instability, the facet of atlas was dislocated posterior to the facet of axis. In type 3 atlantoaxial facetal instability, the facets are normally aligned and instability in such cases is diagnosed on the basis of manual manipulation of bones during surgery. While in type 1 atlantoaxial facetal instability, atlantodental interval is abnormally increased, such an increase is not the hallmark of type 2 and 3 atlantoaxial facetal instability. In types 2 and 3, odontoid process-related compression of dura/subarachnoid space or neural structures is not the primary feature. The clinical presenting symptoms in cases with central or axial atlantoaxial instability are longstanding and chronic and relatively subtle. Musculoskeletal morphological features such as short neck/bone fusions, platybasia, and similar such features and neural responses like syringomyelia and Chiari 1 malformation that are indicative of secondary and protective responses of the body are more predominant in such cases.[24] Our analysis suggests that chronic central and axial atlantoaxial instability is a major or a prime partner in the development of multilevel spinal instability that is observed in cervical spondylotic degeneration and in OPLL.[17,18,22]

The treatment aimed at stabilization of the spinal segments resulted in remarkable clinical recovery. In our initial experience, we stabilized only subaxial spine, the levels of fixation essentially depended on the levels of extension of OPLL and observation of instability of spinal segments during operation. We achieved gratifying clinical outcome.[13] As we graduated further in the field, we realized that atlantoaxial instability is frequently associated or can even be a prime point of genesis of OPLL that extend superiorly up to the C2 vertebral level.[18] From this observation, we realized that subaxial fixation levels should be extended superiorly to involve the atlantoaxial joint whenever the ossification extends up to or above the second cervical level. However, it is currently becoming obvious that atlantoaxial joint should be included in the multi-spinal level fixation construct in all cases of cervical OPLL, even when it involves only lower of mid-cervical levels.

No bone or soft tissue resection was done. Our operation was aimed at arthrodesis of the involved or unstable spinal

segments. The articular cartilage of the facet joint was widely denuded before the surgery for fixation. Subaxial spinal fixation was done by transarticular method of fixation[25] and atlantoaxial fixation was done by the technique described by us in 1988.[26,27] Bone graft was jammed into the articular space adjoining the screw. In addition, bone graft was overlaid on the appropriately prepared host bone of laminae and arch of atlas. For this purpose, bone graft was harvested either from the iliac crest or from the spinous processes, or both of these graft materials were simultaneously used. The interspinous process and interlaminar ligaments were widely removed to limit any movements that could affect bone fusion.

Dramatic postoperative recovery following only multi-level spinal fixation that included atlantoaxial joint confirms the validity of the hypothesis. The improvement occurs in the immediate postoperative phase, and the recovery is sustained and progressive. Our 35-year long experience with various forms of treatment for OPLL indicates that the quality, extent and durability of recovery from symptoms as is obtained by multi-level spinal fixation is far superior to procedures that involve bone removal and decompression. The improvement can be classified into the term "magical." Spinal stabilization with the techniques deployed by us is remarkably simple and quick and safe. The improvement seen in our cases where there was no attempt at decompression of the cord by bone, or soft tissue resection confirms the fact that it is not the cord compression or cord deformation that is the cause of symptoms, it is repeated microtrauma related to the instability that causes symptoms.[28] The fact that only stabilization result in recovery from symptoms is suggestive of the fact that instability is the prime and nodal point of pathogenesis of OPLL.[22] The extent of the contribution of atlantoaxial instability in the entire process of multi-level spinal instability remains to be assessed but seems to be primary. Our experience suggests that OPLL and osteophytes related to degenerative spinal disease have a common pathogenetic origin and are Nature's protective efforts to tackle issues related to spinal instability. As discussed earlier in case with osteophytes, it seems that stabilization of unstable spinal segments has a potential of spontaneous resorption of OPLL.[22]

Atul Goel

Department of Neurosurgery, KEM Hospital and Seth GS Medical College, Parel, Mumbai, Maharashtra, India

Address for correspondence: Prof. Atul Goel, Department of Neurosurgery, KEM Hospital and Seth GS Medical College, Parel, Mumbai - 400 012, Maharashtra, India.
E-mail: atulgoel62@hotmail.com

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