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OPLL: Not Decompression but Fixation as a Form of Surgical Treatment

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Ossification of the posterior longitudinal ligament, its occupation of space in the spinal canal and subsequent symptoms related to spinal cord compression has continued to remain a challenging therapeutic issue. The surgical treatment remains debated as the primary cause of the pathogenesis of OPLL remains unidentified. Our recent observation that atlantoaxial and subaxial multisegmental spinal instability is the nodal point of pathogenesis of both degenerative cervical spondylosis and OPLL has the potential of revolutionizing the treatment [1-6].

OPLL is identified throughout the world, but the reports from Asia outnumber those from other continents. Dietary, environmental and physical body constitution related factors, apart from host of other possible etiological factors have been suggested to be the possible causes. In general, the patients harboring OPLL are marginally or significantly obese and have a relatively sedate life style. We recently hypothesized the factor of multisegmentalspinal instability that includes atlantoaxial instability in majority of patients as the primary issue that is responsible for genesis and growth of OPLL [1-6]. The instability is of long-standing and persistent in nature and is subtle in character. We identified old fractures of the odontoid process having subtle instability presenting with OPLL in the adjoining segments. Also we have identified OPLL in cases with basilar invagination with longstanding instability.

Ossification of the posterior longitudinal ligament and increase in the bone content of the spinal column has been associated with an increased stability of the spine. The surgery essentially involves efforts that increase the space for compressed spinal cord so that it has space to 'breathe' comfortably [7,8]. Although direct surgical removal of the OPLL has been uniformly accepted to be the optimum and relevant surgical strategy, wide removal of OPLL in its vertical and transverse extents is technically a formidable issue. Location of the firm and bony mass anterior to the spinal cord that is housed in a compact bone enclosure and frequent transgression of the OPLL of the dura and even invasion into the neural structures makes surgical exposure and direct surgical removal possible only in a minority of cases. In general, anterior or posterior spinal canal decompression is the choice of treatment. Anterior decompression is done by either complete or partial corpectomy and posterior decompression is achieved by laminectomy or laminoplasty procedures. The indications, validity and extent of anterior or posterior decompression vary with each treating surgeon. Surgical experience and comfort level of treating surgeon with anterior or posterior approach usually dictates the approach selection.

Instability of the spine as a primary issue in the pathogenesis of OPLL has not been discussed earlier. Some authors have identified instability as a dynamic component and spinal cord compression by the OPLL as the static component of various presenting symptoms and suggest that addressing both these components is essential for recovery in neurological symptoms. Decompression of the compressed spinal cord is the aim of all surgical procedures. Several surgeons observe that such extensive bone removal is wrought with possibility of delayed spinal instability and recommend multiple level spinal fixation along with decompressive procedures.

The twin surgery of decompression and fixation generally form the treatment of OPLL. Despite the strategy of anterior or posterior approach, surgery on OPLL is wrought with potential of devastating complications that range from cerebrospinal fluid leakage to partial or even complete motor deficit in all four limbs. Such a complication in a conscious and otherwise awake patient can be the most horrifying complication in spine surgery for the patient, for the relatives and for the surgeon.

In the year 2010 we identified that primary or nodal point of pathogenesis of cervical spondylosis is multisegmental spinal instability [9-12]. It was discussed that vertical instability at the facets is the point of genesis of degenerative spinal disorders [13]. Standing human posture and misuse or disuse of extensor muscles of the spine can lead to spinal instability. Accordingly, we treated cases with single

or multiple level cervical and lumbar degenerative spinal disease presenting with symptoms of radiculopathy or myelopathy with 'only fixation' as a modality of treatment [14]. We subsequently identified that OPLL is also a consequence of spinal instability. It was identified that like osteophyte formation was considered to be a secondary phenomenon of spinal instability, OPLL was also a phenomenon that was secondary to spinal instability. Accordingly, the treatment of OPLL was focused on the primary pathogenetic issue of instability rather than the consequences or secondary effects of instability that could even have a protective function [1-6]. As it was identified that any form of decompression or even removal of osteophytes is not only not essential but also counter-effective in cases with degenerative spinal disease, decompression by removal of bone elements or removal of OPLL was observed to be counter-effective in the treatment. The important and probably crucial observation was that it is not neural deformation or compression that is the cause of symptoms in such cases, but it is repeated micro-injury secondary to subtle spinal instability that is culprit [15]. Essentially, instability is a more important cause of symptoms than the radiological appearance of compression.'Only fixation' of spinal segments was seen to be remarkably safe and resulted in gratifying clinical outcome [16].

As we matured in our clinical understanding and subtleties of biomechanics of the spine, we identified that atlantoaxial instability can be associated with a majority of cases of multisegmental instability that is present in cervical spondylosis or in cases with OPLL [2,3,5]. The instability of the spinal segments can be observed by direct visualization of the facets. The fact that facets are never fused in cases with OPLL despite the ossification that is observed to traverse multiple segments is indicative of their continued activity or 'hyper activity'. The instability of the facets can be radiologically observed at the level of facets of C1 and C2 that are large and rectangular box-like and have a more horizontal profile. It was identified that atlantoaxial instability in most of these cases is of Goel type 2 atlantoaxial facetal instability meaning thereby that in neutral head position the facet of atlas was dislocated posterior to the facet of axis, or of Goel type 3 atlantoaxial facetal instability wherein the facets were in alignment and instability was diagnosed only on the basis of direct bone manipulations during surgery [17]. Goel type 1 instability, wherein the facet of atlas is dislocated anterior to the facet of axis is seldom associated. Goel types 2 and 3 were labeled as central or axial atlantoaxial instability as alteration of atlantodental interval and dural or neural compression by the odontoid process were not the hallmarks. As neural compression is not early or prominent, the neurological symptoms in such cases are of longstanding duration or chronic in nature. We identified that such central or axial atlantoaxial instability was associated with chronically unstable clinical situation that is identified in cases with Chiari malformation, syringomyelia, basilar invagination, retroodontoid ligamentous hypertrophy and Hirayama disease [18-23]. It was also identified that central or axial atlantoaxial instability is a frequent association with cases with multisegmental cervical spinal degeneration and with OPLL. Our current observation is that atlantoaxial fixation and multisegmental spinal fixation is mandatory in most cases with cervical OPLL. As has been observed in cases with osteophytes in spinal degeneration, it appears that only fixation can result in spontaneous resorption or disappearance of OPLL after multi-segmental fixation.

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