

Editorial

Spinal fixation as treatment of ossified posterior longitudinal ligament

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Ossified posterior longitudinal ligament is a relatively rare, but a crippling disorder. More importantly, the lesion is relatively difficult and potentially “dangerous” to treat surgically. This is more because the exact pathogenesis of the disease process is unclear. Multiple modalities of treatment have been recommended, but none has been uniformly accepted or is considered to be the ideal or “gold standard.” Anterior or posterior surgery, laminectomy or laminoplasty, fixation or no fixation and several such issues have plagued the minds of the surgeons who are courageous enough or sufficiently experienced to undertake treatment of ossified posterior longitudinal ligament (OPLL). Moreover, the bone firm ligaments located posterior to the vertebral bodies and anterior to the spinal cord are relatively difficult to approach, to widely expose and to radically resect. Adhesions to the dura and frequently observed deep indentations of the ossified ligaments into the spinal cord can make the issue of spinal cord injury and cerebrospinal fluid leakage relevant. The complications of surgery can be devastating to the patient and can even be life-threatening. No surgeon who is dealing with this issue can say with confidence that he has got the final answer to the problem, and the results of his proposed treatment are uniformly satisfying. The extent of the OPLL, in the vertical, transverse and antero-posterior dimensions has implications in decisions regarding the nature of the surgery and its outcome.

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 a 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 lifestyle. We recently hypothesized the factor

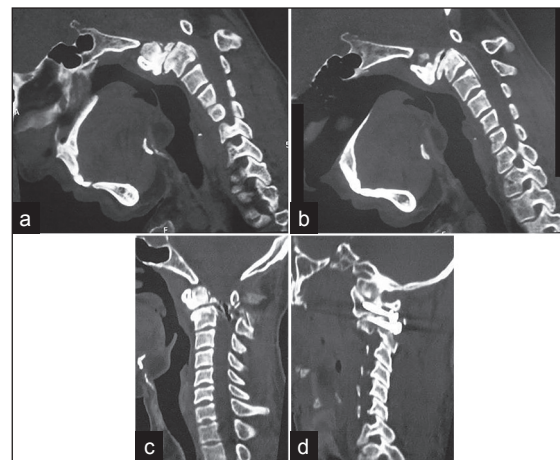


Figure 1: (a) Sagittal image of computed tomography scan of a 32-year-old male patient. He suffered road traffic accident 10 years prior to treatment. The symptoms of quadriparesis increased over the period of time. The image shows chronic odontoid fracture. (b) Lateral sagittal image showing ossified posterior longitudinal ligament at C2-C3 level. (c) Postoperative image showing distraction, reduction and fixation of the atlantoaxial joint. (d) Images showing the implant in the facets

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Figure 2: (a) Images of a 30-year-old man. Computed tomography scan showing os odontoideum and basilar invagination. The features suggest long-term instability. Ossified posterior longitudinal ligament at the level of C2-C3 can be seen. (b) Postoperative images showing distraction, reduction and fixation of the atlantoaxial joint

of spinal instability as the primary issue that is responsible for genesis and growth of OPLL. Long-standing subtle and persistent spinal instability was identified to be the principle or the only initiating and causative factor.^[1] We identified old fractures of the odontoid process having subtle instability presenting with OPLL in the adjoining segments [Figure 1]. Also, we have identified OPLL in cases with basilar invagination with long-standing instability [Figure 2].

OPLL is generally associated with a stable or more than normally stable disorder. Bone formation along the ligaments is considered to be an additionally stabilizing factor. As the lesion is considered to be “stable,” the treatment basically and conventionally involved decompression of the cord, removal of the OPLL or widening of the spinal canal size by removing bone anterior or posterior to the cord. Instability as an issue in the treatment of OPLL is generally discussed as a consequence of treatment that involves anterior corpectomy or posterior laminectomy or laminoplasty. The surgical procedures that involve stabilization are essentially done to stabilize the spine and to prevent the destabilizing effects of the “decompressing” surgery. The twin operative strategy that involves decompression and fixation, either by anterior or by posterior surgical route has been generally adopted. However, despite the surgical experience and expertise of the surgeon, such a surgical procedure and the maneuver of resection of the OPLL can be tedious, time-consuming and potentially hazardous. We had recommended oblique corpectomies for exposure and resection of the OPLL and had suggested that such a conservative surgical procedure does not affect the stability of the spine, and additional stabilization procedures can be avoided.^[2,3]

We analyzed our concept that instability is the primary issue and the defining phenomenon in the pathogenesis of clinical symptoms related to OPLL. We had recently discussed that vertical instability at the facets is the point of the genesis of

degenerative spinal disorders.^[4] Standing human posture and misuse or disuse of extensor muscles of the spine can lead to spinal instability. Similarly, we hypothesize that subtle and long-standing instability at the facets could be the initiating point for the development of OPLL.^[1] The role of instability in the development and progression of the OPLL and in the maturation of clinical symptoms will have to be assessed on the basis of a larger experience. 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-segment is indicative of their continued 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. Instability of the facets of C1-C2 is more often of Goel Type B facet instability, wherein, on lateral imaging, the facet of the atlas is displaced posterior to the facet of the axis. Such atlantoaxial facet instability is frequently observed in long length OPLL that extend over multiple-segments.^[5,6] Although some authors have identified an association of cervical spondylosis with OPLL, the exact pathogenetic relationship between spondylosis and OPLL is unclear. It is impossible to conclude that OPLL is an advanced form of cervical spondylosis or is a delayed manifestation of spinal degeneration.

We identified that only fixation of the multiple affected segments could form the most effective and rational form of surgical treatment.^[7] Imaging features of multiple-segment OPLL and cord compression can present a grotesque clinical situation. However, we have realized that the neural tissues in general and spinal cord, in particular, can tolerate significant compression when the pathology is long-standing and develops over a period of time. However, instability and repeated micro-injuries can result in cord damage and in related symptoms. Essentially, instability is a more important cause of symptoms than the radiological appearance of compression.^[8] Such features of resilience of the spinal cord can be observed in cases with syringomyelia and with benign extramedullary tumors when the cord can be seen to be a thin filament, but the patient may be neurologically remarkably stable.

We have resorted to facet fixation as the modality of treatment, and find it the most suitable and stable form of fixation of the spine.^[1,6,9-12] Atlantoaxial fixation is done using a lateral mass plate and screw fixation technique, as described by us in 1988.^[13,14] For subaxial fixation, we identified the transarticular method of fixation as the most stable form of fixation technique.^[15] Whenever OPLL extends till or above the level of C3, C1-C2 fixation appears to be mandatory. The number of levels of fixations is determined by the extent of the OPLL. However, we have realized that it may sometimes be better to fix the spinal segment beyond the spinal extent of OPLL. A remarkable clinical improvement was observed in our patients with this method of treatment. Longer follow-up will be mandatory to confirm that only fixation can be the best form of treatment for OPLL. However, our initial

results are encouraging. Moreover, if the fixation of the spinal segments fails as a form of treatment or if there are delayed symptoms; decompression of the bone can be performed at a later stage, without worrying about the issue of stability. The clinical outcome in our patients suggests that only fixation of the spinal segments can be a simple, safe and rationale form of treatment for the complex pathology of OPLL. Despite our enthusiasm due to gratifying clinical results, a long-term assessment of these patients will be mandatory to assess if stabilization assists in reversing only the dynamic component of the disease process or it also participates in affecting the growth characteristics or growth potential of OPLL.

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