Original Article

Defining role of atlantoaxial and subaxial spinal instability in the pathogenesis of cervical spinal degeneration: Experience with "only-fixation" without any decompression as treatment in 374 cases over 10 years

ABSTRACT

Aim: The authors analyze their published work and update their experience with 374 cases of cervical radiculopathy and/or myelopathy related to spinal degeneration that includes ossification of the posterior longitudinal ligament (OPLL). The role of atlantoaxial and subaxial spinal instability as the nodal point of pathogenesis and focused target of surgical treatment is analyzed.

Materials and Methods: During the period from June 2012 to November 2022, 374 patients presented with acute or chronic symptoms related to radiculopathy and/or myelopathy that were attributed to degenerative cervical spondylotic changes or due to OPLL. There were 339 males and 35 females, and their ages ranged from 39 to 77 years (average 62 years). All patients were treated for subaxial spinal stabilization by Camille's transarticular technique with the aim of arthrodesis of the treated segments. Atlantoaxial stabilization was done in 128 cases by adopting direct atlantoaxial fixation in 55 cases or a modified technique of indirect atlantoaxial fixation in 73 patients. Decompression by laminectomy, laminoplasty, corpectomy, discoidectomy, osteophyte resection, or manipulation of OPLL was not done in any case. Standard monitoring parameters, video recordings, and patient self-assessment scores formed the basis of clinical evaluation.

Results: During the follow-up period that ranged from 3 to 125 months (average: 59 months), all patients had clinical improvement. Of 130 patients who had clinical evidences of severe myelopathy and were either wheelchair or bed bound, 116 patients walked aided (23 patients), or unaided (93 patients) at the last follow-up. One patient in the series was operated on 24 months after the first surgery by anterior cervical route for "adjacent segment" disc herniation. No other patient in the entire series needed any kind of repeat or additional surgery for persistent, recurrent, increased, or additional related symptoms. None of the screws at any level backed out or broke. There were no implant-related infections. Spontaneous regression of the size of osteophytes was observed in 259 patients where a postoperative imaging was possible after at least 12 months of surgery.

Conclusions: Our successful experience with only spinal fixation without any kind of "decompression" identifies the defining role of "instability" in the pathogenesis of spinal degeneration and its related symptoms. OPLL appears to be a secondary manifestation of chronic or longstanding spinal instability.

Keywords: Intervertebral disc, myelopathy, ossification of posterior longitudinal ligament, spondylosis

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Submitted: 24-Jan-24 **Accepted:** 26-Jan-24
Published: 13-Mar-24

Access this article online	
Website: www.jcvjs.com	**Quick Response Code**
DOI: 10.4103/jcvjs.jcvjs_11_24	

How to cite this article: Goel A, Vutha R, Shah A, Prasad A, Shukla AK, Maheshwari S. Defining role of atlantoaxial and subaxial spinal instability in the pathogenesis of cervical spinal degeneration: Experience with "only-fixation" without any decompression as treatment in 374 cases over 10 years. J Craniovert Jun Spine 2024;15:74-82.

INTRODUCTION

For several decades, “pathological” and “old” age-related “degenerative” alterations in the intervertebral disc that include reduction in its “water content” and mild-to-severe disc herniation have been implicated as a primary factor that initiates a cascade of events that culminate into the genesis of spinal degeneration.[1-4] Ossification of the posterior longitudinal ligament (OPLL) and its related myelopathy have generally been identified to be a discrete clinical entity.

In the year 2010, we introduced a novel viewpoint regarding the pathogenesis of degenerative spondylotic disease and accordingly proposed an alternative treatment format.[5-8] This concept hypothesized that “vertical” spinal instability is the primary or nodal pathogenetic issue in the initiation, development, and progression of degenerative spinal disease.[5,6,9]

Standing posture has unique implications for human spine. While extension of the spine is an active muscular movement that facilitates standing/sitting posture, flexion is essentially a passive movement. A large majority of the muscle bulk that works on the spinal column is located in the nape of the neck or back of the spine and has fulcrum of their activity that is focused on the facets and facetal articulation. Only a minority of muscles work around the intervertebral disc or the vertebral body. Our articles on the subject speculate that intervertebral disc (and odontoid process) is the brain of spinal movement, while brawn is the muscles.[10] Disc (and odontoid process) function like an opera conductor who regulates the entire orchestra without holding any instrument in his hands.[10] Acute or longstanding weakness of the muscles of the nape of the neck can be due to injury, misuse or disuse, and lack of their proper care by appropriate and “full” use. The impact of weakness of the muscles is primarily at the facetal articulation that becomes unstable, joint space is reduced, and there is listhesis of inferior facet of rostral vertebra over the superior facet of caudal vertebra or there is “vertical” spinal instability.[9] The net effect of instability is “telescoping” of the spinal segments. Due to their oblique profile and lateral location of the facets, a distance away from neural structures even modern computer-based dynamic images may not show clearly the abnormalities of alignment or instability of the facet joint.[11] Unlike the flat orientation of facets of atlantoaxial joint wherein the dislocation is horizontal, due to oblique orientation of the facets in the cervical and dorsal spine and a more vertical orientation in the lumbar spine, the dislocation is not horizontal, but it is vertical or oblique when observed from a profile view. The vertical facetal instability is radiologically manifested by several “secondary” alterations that include reduction in the intervertebral and interfacetal spaces, reduction in disc height, bulging of intervertebral discs into the spinal canal, buckling of the intervertebral ligaments, osteophyte formation, and evidences of bone fusion in the vicinity of vertebral bodies and facets.[12-14] These alterations result in a reduction in spinal canal or neural foraminal height or in spinal or neural canal “stenosis.”

Abnormal increase of atlantodental interval and odontoid process-related dural or neural compression on dynamic imaging is the only validated radiological parameter to diagnose atlantoaxial instability. We introduced a novel concept that there can be atlantoaxial instability even in the absence of abnormal alteration of atlantodental interval or any evidence of neural or dural compression. The nature of alignment of facets of the atlas and axis on lateral profile imaging with the head in neutral position, telltale clinical evidences, and direct intraoperative observations confirmed atlantoaxial instability.[15] We labeled such atlantoaxial instability as central or axial atlantoaxial instability (CAAD). Our publications on the subject discuss the implications of identification and treatment of CAAD in clinical entities such as Chiari formation, basilar invagination, syringomyelia, Klippel–Feil abnormality, assimilation of atlas, bifid arch of atlas, and os odontoideum.[16,17] Our further studies identified an association of CAAD with cervical spinal degeneration, myelopathy related to OPLL, and Hirayama disease.[16,17]

Essentially, there can be atlantoaxial and subaxial spinal instability and its related consequences without any radiological demonstration by validated parameters or direct evidences of abnormal bone movements or neural compression. The phenomenon of CAAD and of vertical spinal instability related to spinal degeneration and its consequent symptoms and musculoskeletal, neural, and spinal structural alterations are generally subtle, longstanding, and relentlessly progressive and ultimately disabling. Our articles on the subject identify all the “secondary” structural alterations in CAAD and in vertical instability in the subaxial spine as naturally protective or adaptive, indicative of the presence of spinal instability and potentially reversible following stabilization.[18]

The present article summarizes observations in our earlier published articles that identify vertical spinal instability as a defining factor in spinal degeneration and updates our clinical material.[18-34] The association of atlantoaxial instability in cases with cervical spinal degeneration and in cases having OPLL has not been comprehensibly analyzed in the literature. The potential clinical implications of these novel concepts motivate us to present our updated experience on the subject.

MATERIALS AND METHODS

During the period June 2012–November 2022, 374 patients having clinical and radiological evidence of single- or multiple-level spinal degeneration-related radiculopathy and/or myelopathy were surgically treated in the neurosurgery departments of the authors. Patients with myelopathy related to OPLL were included. Informed consent was taken before surgical treatment from all patients. All clinical tests and surgical procedures were conducted according to the principles of the Declaration of Helsinki. The analysis includes a review of published articles and updates the case material till November 2022. The study is based on a retrospective analysis of consecutively treated cases. All articles and editorials detailing our observations have been published in indexed scientific data resources that included PubMed and Medline.[26,35-55] All cases with radiologically obvious instability or manifest spinal listhesis identified on dynamic imaging were excluded.

There were 339 men and 35 women with ages ranging from 39 to 77 years (mean age: 62 years). In addition to the standard and validated parameters of Japanese Orthopedic Association score and Visual Analog Scale, Goel's clinical grading scale and patient self-assessment parameters were used to assess the clinical condition both before and after surgery.[21] In addition, video recordings before and after surgery were used to assess and confirm the outcome. All patients underwent dynamic plain radiography and computed tomography scan with the head in flexion, extension, and neutral position and magnetic resonance imaging (MRI). Table 1 summarizes the epidemiological and radiological information.

Identification of the levels of spinal segments that needed fixation formed a crucial therapeutic issue.[12] Radiological evidence of instability was suggested by the presence of disc space reduction, disc bulge or protrusion, and disc herniation or prolapse, osteophytes, evidences of bone fusion, cysts adjoining the facets, buckling of the ligamentum flavum, spinal deformities, and several such known issues associated with spinal degeneration. Altered cord signals in the presence or absence of corresponding cord compressive agent indicated instability.[35,49] Direct physical observation of the facets, open articular cavity or abnormal "water-content" in the facetal articulation, osteophytes in the vicinity of facets, evidence of facetal listhesis and excessive or abnormal movements on manual manipulation of the spinous process, and other exposed bones in the surgical field formed crucial direct evidence of spinal segmental instability. Essentially, it was observed that there could be spinal instability and related clinical symptoms, even in the absence of any obvious radiological markers.

Table 1: Epidemiologic and radiological features

Clinical/radiological finding	Previous studies	New Cohort	Total number of patients
Sex			
Male	282	57	339
Female	30	5	374
Number of levels fixed			
1	16	-	16
2	33	7	40
3	74	12	86
4	113	21	134
5	55	20	75
6	21	2	23
Group B CAAD (atlantoaxial and subaxial fixation)	94	34	128
Traditional C1–C2 fixation	55	-	55
Alternative technique of atlantoaxial fixation	39	34	73
Only subaxial fixation	218	28	246

CAAD – Central or axial atlantoaxial instability

Atlantoaxial instability was identified on the basis of our recently described classification.[15] The alignment of the facets was assessed on lateral profile imaging with the head in the neutral position. Type 1 atlantoaxial instability was when the facet of atlas was dislocated anterior to the facet of the axis. Type 2 atlantoaxial instability was when the facet of the atlas was dislocated posterior to the facet of the axis. Type 3 atlantoaxial instability was when the facets of the atlas and axis were in alignment. In both Types 2 and 3, there might not be any abnormal alteration in atlantodental interval or any dural or neural compression by the odontoid process. Instability in such cases is identified by the presence of telltale clinical and radiological evidences and is confirmed by observation of instability on direct manual manipulation of the bones of the region. Types 2 and 3 are labeled as CAAD.[16,17] Patients having Type 1 atlantoaxial instability were excluded from the analysis.

CAAD was grouped into three types. Group A CAAD was when it was associated with alterations related to craniovertebral junction such as basilar invagination, Chiari formation, syringomyelia, and platybasia. Group B CAAD was when it was associated with subaxial spinal issues such as spinal degeneration, OPLL, and Hirayama disease. Group C CAAD was when it was identified as a discrete clinical entity and was not associated with any other musculoskeletal or neural alterations. Cases with Group A and C CAAD were not included in the study and have been discussed elsewhere.[56,57] From Group B CAAD, clinical cases with Hirayama disease have been excluded.[58]

Surgical treatment

The aim of surgery in all cases was segmental spinal fixation that eventually resulted in arthrodesis. No decompression was done that involved resection of the vertebral body, laminae, disc, ligaments, osteophytes, and OPLL. Table 1 shows the number of spinal segments that were treated.

The transarticular screw technique described by Roy-Camille *et al*. in 1972 was deployed for subaxial spinal segmental fixation [Figures 1-3].[59] The joint cavity was widely opened, and screwing motion of a suitably sized osteotome-denuded articular cartilage and small bone chips were introduced into the articular cavity before screw insertion. Monoaxial titanium screws that measured 12–14 mm in length and 2.6 mm in width were used. In 45 facetal articulations, two screws were deployed at each facet to provide double-insurance fixation, a technique described by us earlier.[7,8,32] Intra-articular spacers (Goel facet spacers) were used in addition to transarticular screws to stabilize 44 spinal facets (18 patients).[5-8] Patients where only intra-articular facet distraction with spacer was used (without transarticular fixation at any level) are not included and have been discussed elsewhere.[5-8,21]

In the initial 55 cases, atlantoaxial fixation was done using the technique described by us in 1994.[60,61] In the subsequent 73 cases, atlantoaxial fixation was done using an alternative technique that involved the sectioning of muscles attached to the C2 spinous process and insertion of C2–3 transarticular screws.[62] The technique retained rotatory movements that were initiated and conducted by the muscles attached to the transverse process of the atlas and blocked all movements of the odontoid process.

Following instrumentation, all muscles and soft tissues attached to spinous processes and laminae were sharply cut. The exposed bone of the posterior surface of the laminae and facets were decorticated to make the environment suitable as the host bone. The spinous process of the treated spinal segments was cut at their base, was shredded in small pieces, and was used as bone graft material. Whenever the number of treated spinal segments exceeded three, bone graft was additionally harvested from the iliac crest.

RESULTS

The follow-up period ranged from 3 to 125 months. Table 2 depicts a summary of clinical assessments that were done in the immediate postoperative period, after 3 months of surgery, and at the last follow-up. Apart from authors, two independent neurosurgeon assessors conducted the clinical evaluations. The assessments were done on the basis of clinical data and analysis of video recordings both before surgery and at follow-up. A brief questionnaire in the patient's own vernacular language was used to develop the patient satisfaction score scale [Table 3].

All patients "improved" from their major presenting clinical symptoms in the "immediate" postoperative period. During the follow-up period that ranged from 3 to 125 months, all patients have shown clinical improvement. The extent of clinical recovery is shown in Table 2. Self-assessment and patient satisfaction score indicated that 92.2% of patients were "highly satisfied" with their clinical outcome. Out of 130 patients who were brought to the hospital on a wheelchair for treatment, 116 patients walked independently with (23 patients) or without (93 patients) any external support at follow-up. There was no surgery-related neural or vascular complication. One patient needed adjacent level anterior surgery that involved discoidectomy. No other patient needed any kind of surgical treatment or reoperation for persistent, recurrent, or exacerbated symptoms. There were no cases of implant failure or infections that forced aggressive wound treatment.

Arthrodesis of the treated segment was considered successful when the clinical improvement persisted, bone fusion could be observed across the facetal articulation, and there was no relative movement of any spinal component on dynamic imaging. With these basic parameters, bone fusion was considered to be successful in all the treated surgical levels. Several patients mentioned about reduction of neck movements and the related disability, but none actually complained about it. Neck rotation was preserved significantly in cases where atlantoaxial fixation was done with the alternative C2–3 fixation technique when compared to patients undergoing direct atlantoaxial fixation. The extent of neck movement restriction was not calculated. Delayed postoperative imaging after at least 12 months of surgery showed a reduction of dural compression opposite the level of osteophyte and spinal disc bulge at all the treated spinal levels in all patients. On visual impression, the extent of reduction in the disc-osteophyte-ligament complex was minimum (<25%) in 31 cases, moderate (25%–50%) in 41 of cases, good (50%–75%) in 62% of cases, and complete (100%) in 125 cases out of 259 cases in whom delayed imaging was available.[33] Partial or complete resorption of the herniated/prolapsed or extruded intervertebral disc was observed in 24 out of 33 cases where the MRI was done after 6 months of surgery [Figures 1-3].

Table 2: Pre- and post-operative clinical grades of the patients

Clinical grading	Preoperative (374 patients)	Postoperative (at last follow-up - 374 patients)
Goel's clinical grading		
Grade 1 – Independent and normally functioning	5	180
Grade 2 – Walks on own but needs support/help to carry out routine household activities	86	101
Grade 3 – Walks with minimal support and requires help to carry out household activities	153	53
Grade 4 – Walks with heavy support and unable to carry out household activities	84	29
Grade 5 – Unable to walk and dependent for all activities	46	11
JOA score		
<7	132	14
8–12	173	74
13–15	69	113
16–17	-	173
VAS score		
Neck pain	4–9 (6.9)	0–1 (0.3)
Arm pain	3–9 (8.1)	0–1 (0.6)

JOA - Japanese Orthopedic Association; VAS - Visual Analog Scale

Table 3: Patient Satisfaction Index

Parameter	Score=0 (not satisfied)	Score=1 (minimally satisfied)	Score=2 (satisfied)	Score=3 (remarkably satisfied)
Are you happy with the operation?	-	2	27	345
Are you relieved of sensory symptoms?	-	-	61	313
Can you make your fist/hand grip better?	-	-	9	365
Can you move your shoulders better?	-	-	23	351
Can you walk better?	-	2	14	358

Imaging was done after 12 months in 34 cases having OPLL. There was a marginal but definite reduction in the anteroposterior and vertical length of OPLL in three cases. In six cases, the quality of the OPLL changed, and it became "less dense" on T2-weighted MRI image. In none of the cases did the size of OPLL increase on follow-up.

DISCUSSION

Cervical spinal radiculopathy/myelopathy related to single or multiple-segment spinal degeneration is a common clinical condition, particularly in the elderly. Published data suggest that in the United States, approximately 4/100,000 of the population older than 55 years develop symptoms related to spinal spondylosis.[63] Surgical treatment has been advocated in 1.6/100,000 population. Similar figures are not available from India. Considering the clinical and financial implications on the society in general and to the affected individual in particular, it is prudent that the scientific evaluation of the subject is perpetual and any fresh or novel thought is appropriately analyzed, reviewed, and encouraged.

In 2010, on the basis of the concept that vertical spinal instability-related facetal listhesis is the primary event in spinal degeneration, we discussed the technique of facetal distraction using intra-articular spacers that aimed to restore the spinal alignment and stabilize the affected spinal segment and ultimately affected arthrodesis.[5-8] The technique resulted in spinal segmental stability and provided an indirect decompression of neural structures. For the first time in the literature, direct decompression of compressed neural structures as a mode of surgical treatment was entirely avoided. We adopted such treatment for single- and multiple-level cervical spinal degeneration-related radiculopathy and/or myelopathy.[7] A similar treatment of facetal distraction-arthrodesis was adopted for lumbar spinal degeneration that culminated into lumbar canal stenosis.[8]

As we matured in our understanding of the subject, we realized that vertical spinal instability is the nodal point of the pathogenesis of the entire spectrum of spinal degeneration and the so-called "pathological" radiological features observed are secondary, naturally protective or adaptive, and potentially reversible, we treated all our patients by "only-fixation" and the procedure was aimed at arthrodesis of the affected spinal segment/s.[9,19-21] All the patients in the presented series were treated by only stabilization without any direct or indirect decompression by the resection of laminae, vertebral body, intervertebral disc, osteophytes, ligamentum flavum, or manipulation of OPLL.

Figure 1: Images of a 71-year-old male patient. (a) T2-weighted magnetic resonance imaging (MRI) showing multisegmental spinal degeneration. Buckling posterior longitudinal ligaments and ligamentum flavum are seen, more prominently at C3–4, 4–5, and 5–6 levels, (b) sagittal image of computed tomography (CT) scan showing multiple segment disc space reduction and osteophytes, (c) sagittal image with the cut passing through facets. Abnormalities in facetal alignment, irregular articular surfaces, and osteophytes formation around the articulations can be seen, (d) three-dimensional reconstruction of CT scan showing listhesis of the facets that signal vertical spinal instability, (e) MRI after 9 months of surgery showing reduction of compression by posterior longitudinal ligament and by ligamentum flavum, (f) CT scan reconstruction showing C2–3, C3–4, C4–5, and C5–6 transarticular screw fixation. There is no bone decompression

The current trend of treatment is based on the concept of decompression of compressed neural structures. Some authors relate the clinical symptoms to static and dynamic factors. While the static factors are visibly neural compressive in nature, dynamic factor of instability has been implicated by few authors in the literature.[29-31,64] Stabilization following decompression is essentially suggested as resection of bone and ligamentous structures has been identified to result in delayed spinal instability. Our treatment protocol addresses only the dynamic factor as it was considered to be primary or nodal and the only pathogenetic factor of the entire degenerative process.[18-55] Static factors were not directly or indirectly addressed. We resorted to such treatment for single-level disc herniation-related radiculopathy or multiple-level spinal degeneration resulting in spinal canal "stenosis" related radiculopathy/myelopathy.[18-55] Our studies have identified intervertebral disc herniation to be a direct consequence of acute spinal instability or that the instability of the spinal segment/s is a result of disc herniation.[65] It was observed that it was not neural deformation or compression, but it was instability-related abnormal micromovements at the facets that formed the basis of neurological symptoms.[27]

The key issue in the surgical treatment is to identify the spinal segments that were unstable and needed stabilization. Apart from clinical and radiological guides, direct intraoperative observation of the status of the facets and manual manipulation of bones of the region assisted in identifying and treating unstable spinal segments. It was observed that apart from vertical spinal instability related to the subaxial spine, instability of the most mobile atlantoaxial joint was a "frequent" association in multisegmental spinal degeneration and particularly in patients presenting with symptoms related to "moderate" or "severe" myelopathy and in cases with "high" cervical spinal degeneration.[21,24,34] Our literature search did not locate significant data that depicted the association of atlantoaxial instability in cases with cervical spinal degeneration. CAAD was identified and treated in 128 (34.2%) cases.

Camille's technique of transarticular screw fixation was identified to be simple, strong, safe, and quick.[59,66] Transarticular screws traversed the firm part of the spinal vertebra and stabilized the fulcrum point of spinal movements and provided a "zero-movement" situation that was most conducive for bone fusion. Atlantoaxial stabilization was done with the techniques described by us.[60-62] No decompression by resection of any part of bone, osteophyte, ligaments, or disc was done.

Surgical treatment of myelopathy related to OPLL forms one of the more complex issues in spine surgery. In general, OPLL is presumed to be an outcome of several possible factors that include congenital, environmental, familial, or dietary issues. For the first time in the literature, we observed that OPLL is entirely an outcome of chronic or long-term spinal instability and spinal stabilization is the treatment.[28-31] It was observed that decompression of the neural structures by resection of bone; soft tissues or OPLL was unnecessary. It appeared that atlantoaxial instability is more often associated with subaxial spinal instability in patients presenting with myelopathy related to OPLL.[29-31]

Analysis of articles published in the literature and review of clinical material discussed in the presented article; it appears that the patient cohort treated by us had "significantly" more profound clinical and neurological symptoms and related deficits. Furthermore, the duration of clinical symptoms was longer when compared to similar studies. This appeared

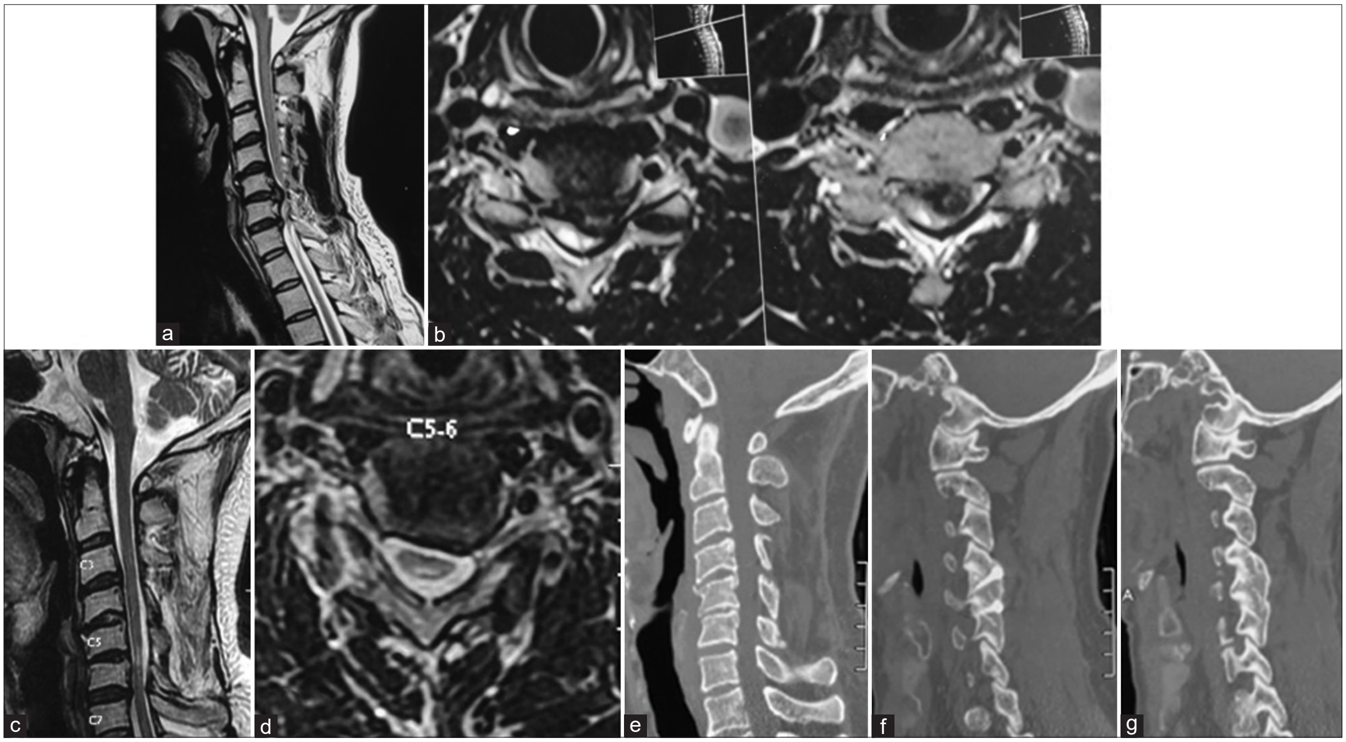

Figure 2: Images of a 46-year-old male patient. (a) Sagittal T2-weighted magnetic resonance imaging (MRI) cervical spine showing degenerative spinal changes in the form of large osteophytes, more prominently at C5–6 and C6–7 levels, (b) axial image of MRI showing large unilateral osteophyte/disc herniation, (c) postoperative MRI after 12 months of surgery showing a reduction in the size of osteophyte/disc herniation. (d) Postoperative axial MRI showing reduction in the size of osteophyte/disc herniation, (e) postoperative sagittal computed tomography (CT) scan showing no midline bone resection in the form of laminectomy, (f) postoperative CT scan cut through the facets showing transarticular screws, (g) CT scan cut showing arthrodesis of the treated segments

to be related primarily to the relatively poor and illiterate patient population treated by the authors in their charitable hospital/s that provides free medical treatment. Ignoring early symptoms and difficulty in easy and quick accessibility of specialized medical care were obvious reasons.

Postoperative moderate to severe and crippling neurological complications have been frequently identified following surgery that involves decompression by either anterior or posterior approaches, particularly in cases where multiple levels are treated. Mild-to-severe neurological deficits have ranged from 30% to 80% in the major reported series on the subject.[67,68] Our 100% improvement rate following treatment that involved only fixation indicates the safety and effectiveness of fixation as a mode of treatment and the futility of decompression. The difference in neck movement restriction and its related disability when compared to that resulting from conventional decompression and fixation techniques was not scientifically assessed. However, none of the patients actually complained about this disability.

Resorption of the herniated or bulging disc and osteophytes, increase in fluid content of the disc, unbuckling of the posterior longitudinal ligament and ligamentum flavum, and radiologically observed spontaneous neural decompression are indicative of the secondary nature of the so-called "pathological" factors in spinal degeneration.[33,69]

Although the complete resolution of OPLL was not radiologically observed in any case, probably related to the relatively short duration of follow-up assessment, change in its architecture, and probably its consistency was observed in 9 out of 34 cases where imaging was possible after at least 12 months of surgery.

The presented experience has the potential to influence the surgical treatment of degenerative spinal disease. All midline anterior and posterior decompressive surgical options remain available in cases where the clinical recovery is not satisfactory.

Needless to mention, scientific clinical studies based on blinded patient cohorts is necessary to accept the validity of the proposed surgical concepts. The senior author has several years' experience of treating patients having degenerative spine-related issues by anterior and/or posterior decompression. The clinical results of treatment by "only-fixation" seem to be far superior, quick, and lasting in nature.

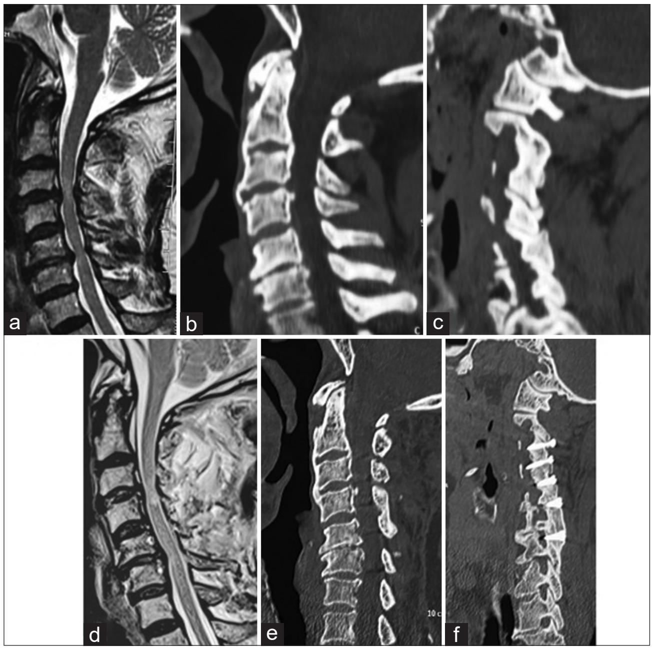

Figure 3: Images of a 56-year-old male patient. (a) Sagittal image of T2-weighted magnetic resonance imaging (MRI) showing evidences of spinal degeneration in the form of osteophytes at multiple segments and evidence of cord compression, (b) sagittal image of computed tomography (CT) scan showing osteophytes, ossification of posterior longitudinal ligament, and disc space reduction at multiple levels. Degeneration of spinal segments extends beyond the levels of compression seen on MRI, (c) CT scan cut passing through the facets showing irregularity of facetal articulation, osteophyte formation along the edges of facets, evidences of bone fusion and reduction in articular spaces, (d) postoperative MRI after 6 months of surgery. Reduction in compression of the spinal cord can be observed, (e) postoperative CT scan showing no midline bone decompression. Reduction in the size of osteophytes and thickness of ossification of the posterior longitudinal ligament can be observed, (f) Postoperative CT scan showing transarticular screw fixation and arthrodesis of the spinal segments

CONCLUSIONS

The concept that spinal instability is the nodal point of the pathogenesis of spinal degeneration has defining implication in the surgical treatment.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Lebl DR, Bono CM. Update on the diagnosis and management of cervical spondylotic myelopathy. J Am Acad Orthop Surg 2015;23:648-60.
2. Shedid D, Benzel EC. Cervical spondylosis anatomy: Pathophysiology and biomechanics. Neurosurgery 2007;60:S7-13.
3. Furlan JC, Kalsi-Ryan S, Kailaya-Vasan A, Massicotte EM, Fehlings MG. Functional and clinical outcomes following surgical treatment in patients with cervical spondylotic myelopathy: A prospective study of 81 cases. J Neurosurg Spine 2011;14:348-55.
4. Fehlings MG, Tetreault LA, Wilson JR, Skelly AC. Cervical spondylotic myelopathy: Current state of the art and future directions. Spine (Phila Pa 1976) 2013;38:S1-8.
5. Goel A. Facet distraction spacers for treatment of degenerative disease of the spine: Rationale and an alternative hypothesis of spinal degeneration. J Craniovertebr Junction Spine 2010;1:65-6.
6. Goel A. Facet distraction-arthrodesis technique: Can it revolutionize spinal stabilization methods? J Craniovertebr Junction Spine 2011;2:1-2.
7. Goel A, Shah A. Facetal distraction as treatment for single- and multilevel cervical spondylotic radiculopathy and myelopathy: A preliminary report. J Neurosurg Spine 2011;14:689-96.
8. Goel A, Shah A, Jadhav M, Nama S. Distraction of facets with intraarticular spacers as treatment for lumbar canal stenosis: Report on a preliminary experience with 21 cases. J Neurosurg Spine 2013;19:672-7.
9. Goel A. Vertical facetal instability: Is it the point of genesis of spinal spondylotic disease? J Craniovertebr Junction Spine 2015;6:47-8.
10. Goel A. Odontoid process and intervertebral disc: Do they have the same function? J Craniovertebr Junction Spine 2020;11:59-60.
11. Goel A. Beyond radiological imaging: Direct observation and manual physical evaluation of spinal instability. J Craniovertebr Junction Spine 2017;8:88-90.
12. Goel A. Indicators of spinal instability in degenerative spinal disease. J Craniovertebr Junction Spine 2020;11:155-6.
13. Goel A. Degenerative cervical myelopathy. Neurospine 2019;16:793-5.
14. Goel A. Is evidence of bone "formation" and "fusion" in the spinal segment an evidence of segmental spinal instability? J Craniovertebr Junction Spine 2022;13:365-7.
15. Goel A. Goel's classification of atlantoaxial "facetal" dislocation. J Craniovertebr Junction Spine 2014;5:3-8.
16. Goel A. Central or axial atlantoaxial instability: Expanding understanding of craniovertebral junction. J Craniovertebr Junction Spine 2016;7:1-3.
17. Goel A. A review of a new clinical entity of 'central atlantoaxial instability': Expanding horizons of craniovertebral junction surgery. Neurospine 2019;16:186-94.
18. Goel A. Role of subaxial spinal and atlantoaxial instability in multisegmental cervical spondylotic myelopathy. Acta Neurochir Suppl 2019;125:71-8.
19. Goel A. 'Only fixation' as rationale treatment for spinal canal stenosis. J Craniovertebr Junction Spine 2011;2:55-6.
20. Goel A. Only fixation for cervical spondylosis: Report of early results with a preliminary experience with 6 cases. J Craniovertebr Junction Spine 2013;4:64-8.
21. Goel A, Dandpat S, Shah A, Rai S, Vutha R. Muscle weakness-related spinal instability is the cause of cervical spinal degeneration and spinal stabilization is the treatment: An experience with 215 cases surgically treated over 7 years. World Neurosurg 2020;140:614-21.
22. Goel A, Dharurkar P, Shah A, Gore S, More S, Ranjan S. Only spinal fixation as treatment of prolapsed cervical intervertebral disc in patients presenting with myelopathy. J Craniovertebr Junction Spine 2017;8:305-10.
23. Goel A, Dharurkar P, Shah A, Gore S, Bakale N, Vaja T. Facetal fixation arthrodesis as treatment of cervical radiculopathy. World Neurosurg 2019;121:e875-81.
24. Goel A, Vutha R, Shah A, Patil A, Dhar A, Prasad A. Cervical spondylosis in patients presenting with "severe" myelopathy: Analysis of treatment by multisegmental spinal fixation – A case series. J Craniovertebr Junction Spine 2019;10:144-51.
25. Goel A. Is it necessary to resect osteophytes in degenerative spondylotic myelopathy? J Craniovertebr Junction Spine 2013;4:1-2.
26. Goel A. Is instability the nodal point of pathogenesis for both cervical spondylotic myelopathy and ossified posterior longitudinal ligament? Neurol India 2016;64:837-8.
27. Goel A. Not neural deformation or compression but instability is the cause of symptoms in degenerative spinal disease. J Craniovertebr Junction Spine 2014;5:141-2.

28. Goel A, Nadkarni T, Shah A, Rai S, Rangarajan V, Kulkarni A. Is only stabilization the ideal treatment for ossified posterior longitudinal ligament? Report of early results with a preliminary experience in 14 patients. World Neurosurg 2015;84:813-9.
29. Goel A. Is atlantoaxial instability the cause of "high" cervical ossified posterior longitudinal ligament? Analysis on the basis of surgical treatment of seven patients. J Craniovertebr Junction Spine 2016;7:20-5.
30. Goel A. Ossification of the posterior longitudinal ligament: Analysis of the role of craniovertebral and spinal instability. Acta Neurochir Suppl 2019;125:63-70.
31. Goel A, Grasso G, Shah A, Rai S, Dandpat S, Vaja T, *et al.* "Only spinal fixation" as surgical treatment of cervical myelopathy related to ossified posterior longitudinal ligament: Review of 52 cases. World Neurosurg 2020;140:556-63.
32. Goel A, Biswas C, Shah A, Rai S, Dandpat S, Bhambere S. Report of an eight-year experience with Camille's transarticular technique of cervical spinal stabilization. J Clin Neurosci 2022;95:9-19.
33. Goel A, Vaja T, Shah A, Rai S, Dandpat S, Vutha R, *et al.* Outcome of osteophytes after only-fixation as treatment for multilevel cervical spondylosis – A minimum of 12 months follow-up. World Neurosurg 2021;146:e876-87.
34. Goel A. Posterior atlantoaxial 'facetal' instability associated with cervical spondylotic disease. J Craniovertebr Junction Spine 2015;6:51-5.
35. Goel A. Letter to the editor. Cervical spondylotic myelopathy. J Neurosurg Spine 2019;20:1-2.
36. Goel A. Like anterior transoral decompression, will anterior cervical spine surgery find space in history books? J Craniovertebr Junction Spine 2020;11:1-3.
37. Goel A. Letter to the editor. Recurrent disc herniation and spinal instability. J Neurosurg Spine 2021;34:819-20.
38. Goel A. When is inclusion of C2 vertebra in the fixation construct necessary in cases with multi-level spinal degeneration? J Craniovertebr Junction Spine 2020;11:249-51.
39. Goel A. Degenerative arthritis of the craniovertebral junction. J Craniovertebr Junction Spine 2021;12:323-6.
40. Goel A. Basilar invagination, spinal "degeneration," and "lumbosacral" spondylolisthesis: Instability is the cause and stabilization is the treatment. J Craniovertebr Junction Spine 2021;12:327-8.
41. Goel A. Significance of atlantoaxial and subaxial spinal instability in cervical spinal spondylosis: Commentary on "clinical impact and correlations of odontoid parameters following multilevel posterior cervical fusion surgery". Neurospine 2022;19:924-6.
42. Goel A. Degenerative spinal disease: Evaluating the role of instability. Spine Res 2016;2:2.
43. Goel A. Atlantoaxial instability associated with single or multi-level cervical spondylotic myelopathy. J Craniovertebr Junction Spine 2015;6:141-3.
44. Goel A. Can decompressive laminectomy for degenerative spondylotic lumbar and cervical canal stenosis become historical? J Craniovertebr Junction Spine 2015;6:144-6.
45. Goel A. Only fixation for lumbar canal stenosis: Report of an experience with seven cases. J Craniovertebr Junction Spine 2014;5:15-9.
46. Goel A. Spinal fixation as treatment of ossified posterior longitudinal ligament. J Craniovert Jun Spine 2015;6:99-101.
47. Goel A. Letter: Lumbar fusion for degenerative disease: A systematic review and meta-analysis. Neurosurgery 2017;81:E64-5.
48. Goel A. Vertical spinal instability and cervical spondylosis: Is it focal or multisegmental? J Craniovertebr Junction Spine 2017;8:163-4.
49. Goel A. Is focal spinal cord "atrophy" an evidence of chronic spinal instability? J Craniovertebr Junction Spine 2017;8:295-6.
50. Goel A. Cervical facet joint degeneration. Neurospine 2022;19:847-50.
51. Goel A. Is the symptom of cervical or lumbar radiculopathy an evidence of spinal instability? J Craniovertebr Junction Spine 2018;9:81-2.
52. Goel A. Cervical fusion as a protective response to craniovertebral junction instability: A novel concept. Neurospine 2018;15:323-8.
53. Goel A. From "only decompression" to "only fixation:" A century-long journey of surgical treatment for spinal spondylosis. J Craniovertebr Junction Spine 2018;9:219-20.
54. Goel A, Ranjan S, Shah A, Patil A, Vutha R. Lumbar canal stenosis: Analyzing the role of stabilization and the futility of decompression as treatment. Neurosurg Focus 2019;46:E7.
55. Goel A. Is the term degenerative "spinal canal stenosis" a misnomer? J Craniovertebr Junction Spine 2019;10:75-6.
56. Goel A, Dhar A, Shah A, Jadhav D, Bakale N, Vaja T, *et al.* Central or axial atlantoaxial dislocation as a cause of cervical myelopathy: A report of outcome of 5 cases treated by atlantoaxial stabilization. World Neurosurg 2019;121:e908-16.
57. Shah A, Vutha R, Prasad A, Goel A. Central or axial atlantoaxial dislocation and craniovertebral junction alterations: A review of 393 patients treated over 12 years. Neurosurg Focus 2023;54:E13.
58. Goel A, Dhar A, Shah A. Multilevel spinal stabilization as a treatment for hirayama disease: Report of an experience with five cases. World Neurosurg 2017;99:186-91.
59. Roy-Camille R, Saillant G, Laville C, Benazet JP. Treatment of lower cervical spinal injuries – C3 to C7. Spine (Phila Pa 1976) 1992;17:S442-6.
60. Goel A, Laheri V. Plate and screw fixation for atlanto-axial subluxation. Acta Neurochir (Wien) 1994;129:47-53.
61. Goel A, Desai KI, Muzumdar DP. Atlantoaxial fixation using plate and screw method: A report of 160 treated patients. Neurosurgery 2002;51:1351-6.
62. Goel A, Patil A, Shah A, More S, Vutha R, Ranjan S. Alternative technique of C1-2-3 stabilization-sectioning of muscles attached to C2 spinous process and C2-3 fixation. J Craniovertebr Junction Spine 2020;11:193-7.
63. Lee GP, Ahir B, Chaudhry N, Engelhard HH. Cervical spondylotic myelopathy: An updated review. Neurosurg Cases Rev 2021;4:56.
64. Matsunaga S, Kukita M, Hayashi K, Shinkura R, Koriyama C, Sakou T, *et al.* Pathogenesis of myelopathy in patients with ossification of the posterior longitudinal ligament. J Neurosurg 2002;96:168-72.
65. Goel A. Is disc herniation "secondary" to spinal instability? Is it a protective natural response? J Craniovertebr Junction Spine 2021;12:213-5.
66. Goel A. Camille's transarticular technique of spinal fixation: An underused surgical technique. J Craniovertebr Junction Spine 2019;10:197-8.
67. Leckie S, Yoon ST, Isaacs R, Radcliff K, Fessler R, Haid R Jr., *et al.* Perioperative complications of cervical spine surgery: Analysis of a prospectively gathered database through the association for collaborative spinal research. Global Spine J 2016;6:640-9.
68. Wewel JT, Brahimaj BC, Kasliwal MK, Traynelis VC. Perioperative complications with multilevel anterior and posterior cervical decompression and fusion. J Neurosurg Spine 2019;20:1-6
69. Goel A, Shah A, Patni N, Ramdasi R. Immediate postoperative reversal of disc herniation following facetal distraction-fixation surgery: Report of 4 cases. World Neurosurg 2016;94:339-44.