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Original Article

Posterior atlantoaxial 'facetal' instability associated with cervical spondylotic disease

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Abstract

Aim: The association of single or multiple level cervical spondylotic disease with atlantoaxial instability is assessed. The implications of identifying and treating atlantoaxial instability in such an association are highlighted. **Materials and Methods:** The analysis is based on an experience with 11 patients treated during the period June 2013-June 2014. All patients had single or multilevel cervical spondylotic disease. The spinal canal compromise and evidence of cord compression was evident on imaging in the cervical subaxial spine and was related to disc bulges and osteophytic bars. There was no or relatively insignificant compression of the cervicomedullary cord by the odontoid process. There was no evidence of odontoid process-related instability on dynamic imaging. Apart from presence of features of cervical spondylosis, investigations and surgical exploration and direct manual handling of the facets revealed evidence of Type B (posterior) atlantoaxial'facetal' instability in all patients. Our 5-point clinical grading system and Japanese Orthopaedic Association (JOA) scores were used to monitor the patients both before and after surgery and at follow-up. Surgery involved both at lantoaxial and subaxial cervical fixation. During the average period of follow-up of 9 months (5-17 months), all patients showed remarkable and gratifying neurological recovery. **Conclusion:** We conclude that atlantoaxial facetal instability can be 'frequently' associated with cervical spondylosis and needs surgical stabilization. Our surgical outcome analysis suggests that missing or ignoring the presence of atlantoaxial facetal instability can be an important cause of suboptimal result or failure of surgery for cervical spondylotic myelopathy.

Key words: Atlantoaxial dislocation, cervical spondylosis, facetal instability, segmental arthrodesis

INTRODUCTION

We report our experience with 11 cases that were being investigated for and were identified to have cervical spondylotic disease-related myelopathy. Additional evaluation of radiological images revealed presence of atlantoaxial facetal instability. There was no spinal canal compromise or cord compression at the level of craniovertebral junction. There was no evidence of atlantoaxial dislocation when assessed using the conventional radiological parameters. However, there was atlantoaxial 'facetal' instability as per our recently discussed classification of instability in the region.[1] The treatment strategy in such cases is evaluated. The results of treatment are based on our preliminary observations, but it is clear that the subject of such intensity needs a double-blinded study with a scientifically set protocol.

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MATERIALS AND METHODS

During the period June 2013-June 2014, we surgically treated 33 cases having single or multiple level cervical spondylotic disease.

Of these, 11 patients were identified to have atlantoaxial facetal instability, and were analyzed. Patients having basilar invagination, traumatic or 'congenital' atlantoaxial dislocation, and other craniovertebral junction related bone fusions and anomalies as per conventionally described parameters were excluded from the study. All patients in the series were males and their ages ranged from 36 to 65 years (average 50 years). All patients presented with clinical symptoms and radiological findings suggestive of cervical 'spondylotic' myelopathy. Clinical grading system, used by us in the evaluation of craniovertebral junction instability related myelopathy[1] was used to monitor the patients both before and after surgery and at follow-up. Additionally, Japanese Orthopaedic Association (JOA)[2] was used to clinically evaluate the patients. Tables 1-4 show the clinical and radiological profile of the patients at the time of presentation and at follow-up assessment. All patients were investigated with plain radiographs, computed tomography (CT), and magnetic resonance imaging (MRI), both before and after operation and at follow-up. There was Type B atlantoaxial facetal dislocation in all patients [Figures 1 and 2]. In the sagittal profile, imaging of either MRI or CT, the maximal atlantoaxial facetal malalignment was between 15 and 30% in seven cases, 30 and 50% in three patients, and more than 50% in one patient. All patients underwent atlantoaxial segmental stabilization with the techniques described by us in 1988.[3,4] Subaxial transarticular fixation was done at two levels in two patients, at three levels in four patients, at four levels in four patients, and at five levels in one patient. The decision regarding the level/s of subaxial fixation was based on radiological imaging and observation of status of the facets on direct handling during surgery. Both atlantoaxial and cervical sub axial transfacetal screw fixation surgery involved wide opening of the joint, denuding of the articular cartilage, and introduction of bone graft chips within the joint. The screws used for atlantoaxial fixation were 2.8-3 mm in diameter and 28 mm in length. The atlas and axis screws were similar in dimensions. For subaxial spine transfacetal fixation, the screws used were 2.8 mm in diameter and were on an average 14 mm long. Additionally, bone graft pieces harvested from the iliac crest (seven patients) and bone pieces obtained by section of the spinous process at its base (all patients) were placed in the midline and over the facets, after appropriately preparing the host bone. All patients had immediate postoperative recovery in all their symptoms related to myelopathy and progressive clinical recovery. The degree of clinical improvement as monitored during follow-up assessment is shown in Tables 3 and 4. Postoperatively, the patients were placed in external cervical orthosis for a period of about 8weeks. The patients were mobilized following surgery, but were advised to avoid neck movements. Bone fusion within the facets and lamina could be assessed only after 6 months of surgery. As all patients were considerably disabled prior to surgery, they resumed their minor to moderate physical activities after about 8-week period.

DISCUSSION

Craniovertebral junction instability has been frequently identified to be associated with secondary effects on the cervical spine and even in the rest of the spine.[5,6] Short neck, bone fusions, spondylotic changes, hyperextension of the neck, and similar such alterations in the cervical spine have been associated with craniovertebral junction instability in general and basilar invagination in particular.[7] In the years 2004 and 2008, we

Table 1: Presenting clinical and radiological features

Sex	Number of patients
Male	11
Mean age years	50 (36-65) years
Level of involvement	
C1-2	11
C2-3	2
C3-4	7
C4-5	8
C5-6	8
C6-7	—
C7-T1	1
Number of levels fixed	
Two-level	2
Three-level	4
Four-level	4
Five-level	1

Table 2: Extent of atlantoaxial dislocation

Extent of dislocation (%)	Number of patients
15-30	7
30-50	3
>50	1

Table 3: Distribution as per our clinical grading system

Grade	Description	Number of patients (preoperative)	Number of patients (postoperative)
Grade 1	Independent and normally functioning	—	7
Grade 2	Walks on own but needs support/help to carry out routine household activities	1	3
Grade 3	Walks with minimal support and requires help to carry out household activities	3	1
Grade 4	Walks with heavy support and unable to carry out household activities	5	
Grade 5	Unable to walk and dependent for all activities	2	

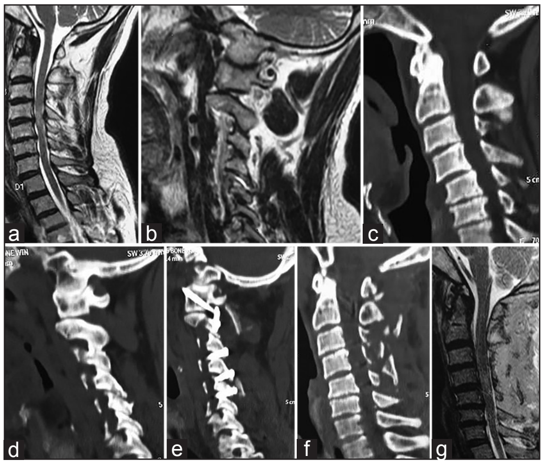

Figure 1: Images of a 65-year-old male patient. (a) T2-weighted MRI shows multilevel spondylotic changes. (b) MR image with slice through the facets. It shows posterior atlantoaxial facetal dislocation. (c) CT showing the cervical spine and the degenerative changes. (d) CT with slice through the facets showing posterior atlantoaxial facetal dislocation. (e) Postoperative scan showing atlantoaxial facetal alignment. Atlantoaxial and subaxial instrumentation can be seen. (f) Postoperative CT. Bone graft pieces can be seen in the midline. (g) Postoperative MRI. MRI = Magnetic resonance imaging, CT = Computed tomography

Table 4: Pre- and postoperative clinical assessment as per JOA scoring system

JOA score	Preoperative (no. of patients)	Postoperative (no. of patients)
<7	4	
8-12	6	4
13-15	1	6
16-17	—	1

JOA = Japanese orthopaedic association

reported our experience with high cervical disc/s related cord compression and its association with basilar invagination and atlantoaxial instability, and stressed upon the significance of primarily addressing the instability at craniovertebral junction.[8,9] In the year 2009, we identified reversibility of soft tissue and bone alterations in the craniovertebral junction and the spine following atlantoaxial fixation surgery.[7] The study concluded that the craniovertebral junction instability is the primary phenomenon and the craniovertebral and cervical morphological structural alterations are secondary effects. We identified the possibility of increase in the neck length towards normalcy and recovery of torticollis, and hypothesized the potential of reversibility of spondylotic changes and bone fusions following atlantoaxial stabilization.

Primary craniovertebral junction region degenerative changes and related instability has only rarely been reported.[10-12] However, recent studies have identified craniovertebral junction to be frequently involved with degenerative changes and is a common cause of cervical pain and myelopathy. Degenerative changes in the craniovertebral junction have been associated with reduction in atlantoaxial joint space, facetal instability, and

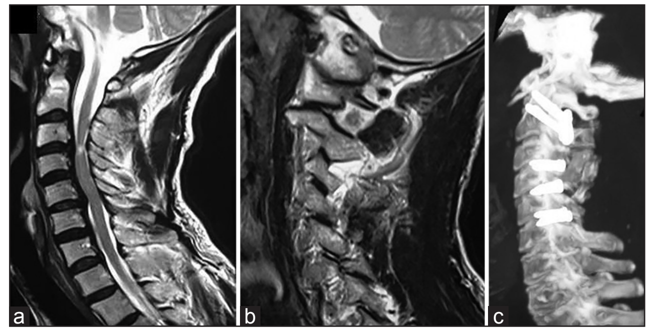

Figure 2: Images of a 52-year-old male patient. (a) T2-weighted MRI shows multilevel cervical spondylosis. (b) MRI slice through the facets showing posterior atlantoaxial dislocation. (c) CT reconstruction image showing the implants in atlantoaxial and subaxial regions

osteophyte formation in and around the facets, apical ligament and other ligaments, and in the retro-odontoid space.[6,12] Such degenerative changes suggest the possible presence of instability in the region that needs surgical treatment.

Association of cervical spondylosis with atlantoaxial or craniovertebral degenerative changes or degeneration-related instability has only infrequently been discussed in the literature.[5,6,12,13] The craniovertebral segment of cervical spine is largely excluded or ignored from the umbrella of spinal spondylosis. In the present report, we identified presence of atlantoaxial facetal instability in association with cervical subaxial single or multilevel degenerative spondylotic changes. There was no evidence of spinal canal compromise or cord compression at the level of craniovertebral junction. Dynamic imaging did not reveal any odontoid process-related instability or cord compression. Although no morphological studies are available that discuss alignment of the facets of atlas and axis during dynamic movements, it appears that some degree of facetal malalignment can be considered to be within the spectrum of normal variation. We recently reported an alternative classification of atlantoaxial facetal dislocation.[1] In this classification we identified 'posterior' or 'reverse' facetal instability (type B atlantoaxial facetal instability), wherein the facet of atlas is dislocated posterior to facet of axis as against the more common and previously identified form of Type A dislocation, wherein the facet of atlas is dislocated anterior to facet of axis. Despite the facets being in alignment in Type C atlantoaxial facetal alignment, the region was unstable as presence of basilar invagination was always associated with instability. Instability in such cases was defined as being 'central' or 'axial' and was identified during direct surgical handling of the atlantoaxial facets. Such posterior or reverse (Type B) and central (Type C) instability have not been identified earlier in the literature. The absence of an increase in atlantodental interval in such cases (Type B and C instability) or any evidence of direct compression of the neural structures by the odontoid process despite the presence of facetal instability made identification of instability by conventional imaging difficult or impossible. We recently presented our experience with Type B and C facetal instability that were identified in association with chronic instability of craniovertebral junction. It was identified that such chronic atlantoaxial instability led to longstanding morphological soft tissue and bone malformations. Such chronic alterations can include short neck, torticollis, bone fusions, platybasia, and a host of other malformations that are frequently associated with basilar invagination in general and Group B basilar invagination in particular.[7] We included Chiari malformations and syringomyelias, a continuum of the same series of alterations.[14] It was proposed that in such cases, it is necessary to perform atlantoaxial stabilization surgery and all the morphological structural changes that are secondary to primary instability have the potential for reversal. Atlantoaxial stabilization resulted in neurological recovery, despite the fact that no direct compression of the neural structures at the level of craniovertebral junction was obvious on imaging. As neural indentation or compromise is not early or marked in such cases, chronicity is the hallmark of such forms of dislocation.

In our presented cases, there was evidence of presence of Type B atlantoaxialfacetal dislocation in all cases. Our 25 year experience of directly handling the facets of atlas and axis for atlantoaxial stabilization in over 1,200 cases, clearly showed evidence of atlantoaxial instability in these cases. As in the cases with basilar invagination, there was no evidence of direct compression of the craniocervical cord by the odontoid process. However, there was clear evidence of relatively 'moderate' spondylotic changes and evidence of neural compression in the subaxial cervical cord.[15-19] The symptoms were relatively longstanding and the neurological disability and extent of myelopathy were more marked and disproportionate to the extent of spondylotic changes. The cervical spondylotic changes were more frequent in higher subaxial cervical spine than in the lower cervical spine that is more frequent site of general spondylotic disease. We could identify presence of atlantoaxial facetal instability in 11 presented cases during the relatively short duration of our study. These patients constituted 33% of total multilevel spondylotic myelopathy cases treated during the period. It is unclear if atlantoaxial instability was an association, a cause or an effect of cervical spondylosis. It may be that the atlantoaxial instability is the primary event that is manifested by secondary alterations in the cervical spine in the form of cervical spondylosis. Or it may be that there is generalized or multisegmental facetal instability in some cases with cervical spondylotic myelopathy that could be identified at the atlantaxial facetal level due to their relatively large size of the facets and their horizontal positioning. Muscle weakness related to their abuse or disuse and the consequent vertical instability of the spinal segments as a defining pathogeneticissue of cervical spondylosis has been discussed by us earlier.[20-22] In essence, it appears that Type B atlantoaxial facetal instability that is frequently the cause of basilar invagination can also be the cause or an association of cervical spondylosis. On the basis of this hypothesis, it appears that as atlantoaxial fixation forms the treatment of basilar invagination, it can also form the treatment of selected cases of cervical spondylosis.[23] In the presented series, we resorted to atlantoaxial stabilization and in addition performed single or multilevel cervical arthrodesis as per our policy of treatment of cervical spondylosis.[13] The number of segments of subaxial spinal vertebrae that were fixed depended on the level of spinal compression observed on imaging and also of the facetal instability seen on direct observation during surgery.[13,20-22] The subaxial spinal fixation was done by the transarticular screw fixation technique. We achieved remarkable and 'unusually' gratifying clinical outcome in all cases. However, it is clear that a larger experience by a number of groups of surgeons is mandatory to conclusively assess the significance of association of atlantoaxial instability with cervical spondylosis. Due to the fact that such association of atlantoaxial instability and cervical spondylosis has not been reported earlier and the relative rarity of such coexistence, it was difficult to muster sufficient confidence in performing only atlantoaxial stabilization. As atlantoaxial fixation was done in addition to stabilization of the cervical spine, the exact contribution of atlantoaxial stabilization in the positive neurological outcome is difficult to assess. However, the extent of neurological improvement observed in the cases appeared significantly greater than in the cases where the treatment is focused on subaxial cervical cord compression.

To analyze the exact significance of atlantoaxial instability in cases with cervical spondylosis, a blinded study with scientifically designed considerations is mandatory. As there was no associated radiological or even clinical evidence of craniocervical cord compression, it could well be that presence of facetal instability as identified was an incidental observation. However, direct handling of the facets during surgery confirmed the presence of instability. On the basis of our encouraging experience, it appears that atlantoaxial facetal instability must be investigated in all cases of cervical spondylosis, particularly when the symptoms are disproportionate to the extent and degree of compression of the cervical cord related to spondylosis. High subaxial cervical spondylosis needs special focus on the atlantoaxial instability in general and facetal malalignment in particular. It needs to be assessed if only atlantoaxial stabilization will suffice in such cases. It is also necessary to assess if the instability of the spine is more generalized in cases with cervical spondylosis than what is observed on radiological imaging that focuses on status of neural structures. More importantly, dynamic relationships of the facets needs to be evaluated on the basis of radiological and clinical studies based on normal population to evaluate the extent of facetal malalignment that could be considered within the range of normal variation.

CONCLUSION

From our experience and remarkably gratifying and satisfactory clinical outcome in all cases, it appears that atlantoaxial instability can be a frequent association with cervical spondylotic myelopathy. Addressing atlantoaxial instability may be of paramount importance to achieve clinical success.

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