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Letter to the Editor

Cervical Facet Joint Degeneration

Corresponding Author
Atul Goel
https://orcid.org/0000-0001-5224-6414

Department of Neurosurgery, Lilavati Hospital and Research Center, Bandra, Mumbai, India
Email: atulgoel62@hotmail.com

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Atul Goel[1,2,3]

[1]Department of Neurosurgery, Lilavati Hospital and Research Center, Bandra, Mumbai, India
[2]Department of Neurosurgery, R.N Cooper Hospital and Medical College, Mumbai, India
[3]Department of Neurosurgery, Bombay Hospital Institute of Medical Sciences, Mumbai, India

To the editor,

I read with interest the article by Okamoto et al.,[1] as it directly relates to our clinical interests. The article is focused on observations of facet joint degeneration and its relationship with degenerative cervical myelopathy. Unfortunately, the authors did not include our several publications on the subject in their reference list.[2-8] I wish to update this subject, particularly as regards the clinical implications of facet joint degeneration. The observations of the authors regarding facet joint degeneration reinforce our hypotheses on the subject.[2-8] Although we agree with the authors that the evaluation of facets is less debated and that are only a few relevant reports, we contest their statement that the relationship between facet joint degeneration and degenerative spinal disease is unknown.[2-11]

Mobility and stability are essential elements of life. Human beings are additionally burdened by their unique natural gift of a posture standing on 2 legs. The major bulk of human muscles is located on the extensor compartment of the body, or on its "back," and cater to movements that facilitate sitting, standing, and running. However, relatively few strands of muscles are located in the flexor or anterior compartment of the body, flexion movement being essentially of a passive nature. The activity of all major extensor muscles is focused on the facetal articulation, which forms the point of fulcrum of all movements. In essence, the activity of no major muscle group is focused on the disc or the odontoid process, or in other words, the disc or the odontoid process does not form a fulcrum point of movement. Our articles have discussed the role of the disc and the odontoid process in human movements. We philosophized that both the disc and odontoid process are like opera conductors who regulate all music without holding any instrument in their hands.[12] While muscles are the brawn, the disc (and odontoid process) is the brain of all movements.

We hypothesized that the weakness of muscles related to their disuse, abuse, or injury forms the basis of all spinal instability and deformities.[2-11] As the facets are the focal point of activity of spinal muscles and their movements, muscle incompetence has its initial impact on the facets and their articulation. We identified for the first time in the literature that such muscle weakness leads to telescoping or listhesis of the facets of spinal segments and labeled it as "vertical" spinal instability.[9] We hypothesized that vertical facetal instability is the primary issue in spinal degeneration and reduction in the disc space, bulging of the disc into spinal canal, buckling of intervertebral ligaments (including the posterior longitudinal ligament and ligamentum flavum), osteophyte formation, facetal and vertebral body fusion, and all the other known so-called "pathological" entities that lead to reduction in the spinal and neural canal dimensions are secondary natural responses.[7-11] Essentially, we observed that it is not disc fluid reduction or disc degeneration that is the primary point of inertia of

spinal degeneration, but instead vertical spinal instability.[9-11] In their study that focused on the pathology of facets, while evaluating the listhesis of vertebral bodies, the authors could have evaluated the listhesis of the facets even if it was of a subtle nature. Acute muscle weakness-related spinal instability can lead to disc herniation and listhesis of the spinal segment and acute clinical symptoms, usually in the form of radiculopathy.[13-17] The authors referred to this group of patients as the "rapid progression group." In contrast, chronic and longstanding instability leads to chronic secondary alterations and multisegmental cervical and lumbar canal stenosis and subtle and relentlessly progressive symptoms.[7,18,19] The authors referred to this group of patients as the "slow progression group."

The authors correctly mentioned that the lateral location of the facetal articulation, which is away from spinal neural structures, makes the identification of instability difficult or impossible. The authors used high-definition imaging to identify the various types of facetal degeneration, which include joint space narrowing, articular surface irregularity, facet joint opening, cyst formation, and ankylosing changes. As we speculated earlier,[2-11,20] the authors identified facet articulation changes in all patients with degenerative spinal disease. It can be added to the authors' observations that all these facetal changes are secondary to vertical spinal instability, which originates from muscle weakness.

The authors mentioned their observations of facetal alterations in asymptomatic patients and in the adjoining segments, even when this was not corroborated with parallel clinical symptoms and radiological changes. In our articles on the subject,[2-8,20] we have discussed this issue and identified spinal instability by clinical and radiological guides and by direct confirmation by the manual manipulation of bones. We have resorted to spinal fixation even when there was no radiological evidence of degeneration.[2-8] Understanding the fact that chronic muscle weakness is usually not segmental, but is often multisegmental,[8] and segments adjacent to those evident radiological guides can be unstable can avoid the commonly encountered issue of "adjacent segment disease."[8]

We observed that the atlantoaxial facetal joint, which is the most mobile joint of the body, is most susceptible to instability.[21-24] The subject of craniovertebral junction "degeneration" has been seldom discussed in the literature.[25] Our experience suggests that atlantoaxial instability can be present either discretely or can frequently be associated with multisegmental cervical spinal degeneration, more often in patients who present with symptoms related to severe myleopathy.[19] Atlantoaxial instability is usually of the central or axial variety and is chronic in nature. As discussed in our articles, atlantoaxial instability can be difficult or impossible to diagnose on radiological assessment of dynamic imaging and has to be diagnosed on the basis of tell-tale evidence.[26] All the facetal changes discussed by the authors can be starkly observed in the craniovertebral junction facets in the scenario of degeneration.[25] In addition, we discussed the presence of retro-odontoid pseudotumors in terms of their relationship with atlantoaxial instability.[27] We are convinced that ignoring atlantoaxial instability in such cases can lead to surgical failure. It is unfortunate that the authors have ignored the evaluation of atlantoaxial facets in their study.

In 2011, we identified facet distraction and fixation-arthrodesis (both cervical and lumbar) by deploying the specially designed "Goel facet spacer" as treatment for single-segmental and multisegmental spinal degeneration-related radiculopathy and/or myelopathy.[2-5] The treatment resulted in secondary spinal decompression. Our article was the first in the literature to mention that "decompression" by the removal of parts of bones, soft tissues, and osteophytes can be avoided in cases of spinal degeneration.

As we mature further, we realize that instability is the cause and stabilization is the treatment for spinal degeneration. Our multiple articles on the subject discuss this issue.[2-8,19] We have observed that ossification of the posterior longitudinal ligament (OPLL) is also a consequence of spinal instability, and only stabilization and not decompression is the treatment.[28-31] Although the authors evaluated the facetal articulation in cases of OPLL, they did not clearly delineate their specific pathological features.

We resort to the Camille technique of transarticular fixation and find it strong, simple, and safe; more importantly, it focuses on the fulcrum point of movements.[32,33] Using the strongest part of the spinal segment provides a base for strong screw purchase, firm stabilization, and a reliable opportunity for arthrodesis.

Essentially, muscle weakness-related facet degeneration leads to instability, and spinal stabilization is the treatment. All secondary alterations, such as osteophyte formation and ligamentum flavum buckling, are secondary, protective, and potentially reversible.[8] Compression of neural structures is always secondary to instability and decompression by removal of bone/soft tissues in an unstable spinal situation can be counter-effective.[2-11]

Conflict of Interest: The author has nothing to disclose.

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