Editorial

Is evidence of bone "formation" and "fusion" in the spinal segment an evidence of segmental spinal instability?

Chronic or longstanding spinal instability induces a range of naturally "protective" maneuvers; all focused to stall, delay or avoid neurological manifestations or deficits and in the long run are helpful for human function and life. Chronic or longstanding facetal instability related to weakness of the muscles that partake in standing human posture is the point of genesis of a number of clinical issues in the craniovertebral junction and in the subaxial spine. The major bulk of the muscles has fulcrum of their activity at the facetal articulation. The muscle weakness is expressed at the facetal articulation in the form of manifest or potential listhesis.[1] Chronic instability is usually subtle and may not be identified on dynamic radiological imaging. Chronic instability is usually of "central" or "axial" variety in the atlantoaxial joint and is of "vertical" variety in the subaxial spine.[1-4] In the subaxial spine, spinal degeneration or spinal spondylosis is a manifestation of chronic vertical spinal instability.[1] Such spinal degeneration can be at a single level or multiple levels and in the cervical spine is frequently associated with atlantoaxial instability.[5] The clinical manifestations of chronic instability are generally relentlessly progressive and ultimately disabling. Understanding the fact that acute or chronic instability is the cause of radiculopathy and/or myelopathy can have significant clinical implications.

Our earlier articles identify a variety of neural, spinal structural, and musculoskeletal alterations initiated and propelled by chronic or longstanding atlantoaxial instability. Basilar invagination, Chiari formation, Klippel-Feil alterations, assimilation of atlas, C2-3 fusion, platybasia, retro-odontoid pseudotumor, retro-odontoid pannus, and several other generally considered pathological clinical entities are in fact secondary natural response to atlantoaxial instability, are protective in their function and are potentially reversible following atlantoaxial stabilization.[6-9] In the presence of chronic atlantoaxial instability, the alterations can apparently be self-destructive like syringomyelia, usually are in close vicinity of site of instability such as assimilation of atlas, C2-3 fusion, os-odontoideum, and bifid arches of atlas and axis and can even have remote structural manifestations such as dorsal kyphoscoliosis.[9-13] All these listed entities are radiologically evident, appear to be pathologically neural compressive and are generally treated as primary disorder by surgery, i.e., "decompressive" in nature. Our concept is that atlantoaxial instability may not be evident on radiological imaging but can be identified by its secondary manifestations. The treatment is essentially directed at the primary or the nodal point of pathogenesis. Atlantoaxial fixation rather than any form of decompressive surgery is necessary and optimal form of surgical treatment (give that image of craniovertebral junction osteophytes/bone formation at spinal ligament).

Disc space reduction, disc bulge into the spinal canal, osteophyte formation, buckling of the interspinal ligaments that include posterior longitudinal ligament and ligamentum flavum all leading to reduction in the spinal canal and neural foraminal dimensions or in spinal canal "stenosis" are secondary to vertical instability, are protective in their function and are potentially reversible following stabilization of the affected spinal segments. All these listed manifestations present an image of neural compression and "decompression" of the compressing elements has been identified to be the surgical treatment. In contrast, on the basis of the concept that instability is the nodal point of pathogenesis, we have recommended only stabilization or fixation as a mode of surgical treatment.[14-19] "Decompression" by resection of bone and soft tissue elements in an unstable spinal situation can have negative clinical implications.

Access this article online	
Website: www.jcvjs.com	Quick Response Code
DOI: 10.4103/jcvjs.jcvjs_139_22	

Submitted: 13-Nov-22 **Accepted:** 18-Nov-22
Published: 07-Dec-22

How to cite this article: Goel A. Is evidence of bone "formation" and "fusion" in the spinal segment an evidence of segmental spinal instability?. J Craniovert Jun Spine 2022;13:365-7.

Evidence of bone fusion in a spinal segment include osteophyte formation in the inter-vertebral bones more commonly posterior and anterior to the vertebral bodies. Other evidence of bone fusion are along the facetal articulation and in the region of the disc space. In our opinion, ossification of posterior longitudinal ligament (OPLL) is also secondary to spinal instability, has a naturally protective function and is indicative of spinal instability.

The presence of "abnormal" bone fragments (osteophytes, bone fusions, and OPLL), excessive fluid content (syringomyelia or external syringomyelia), or "abnormal" fat content as in Hirayama disease are indicators of unstable spinal segment/s and indicate the need for spinal stabilization.[20-23]

On magnetic resonance imaging, the "abnormal" bone formation in general and osteophytes, in particular, appear as neural compressive and direct resection and decompression of the neural structures is the universally accepted treatment. Our several related articles suggest that "abnormal" bone formation and "abnormal" fusion are secondary to chronic or longstanding spinal instability, have a neural "protective" function and are reversible following surgery that involves spinal stabilization.[21] Bone fusion along the facets is often flimsy and weak. The formed bone is usually of soft, fragile, and gritty nature and can be relatively easily broken. Once the bone bridge is removed the abnormal mobility of the facetal articulation can be appreciated. Identification of the levels of spinal instability and stabilization is the treatment. Any kind of direct resection of the osteophytes or indirect decompression by laminectomy or laminoplasty can have negative connotations.

Longstanding instability ultimately leads to vertebral segmental fusion. It is crucial to identify if bone fusion is "complete" or final or is "incomplete" and in formative stages. While complete fusion "might" be ignored, incomplete fusion is an evidence of local spinal instability and when the patient has corroborative neurological symptoms he/she will need stabilization. The identification of unstable spinal segments is the key issue. Apart from clinical parameters and radiological guides, direct visual inspection and manual manipulation of the bones of adjoining facetal articulation will determine the spinal levels that need stabilization.

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