Is disc herniation “secondary” to spinal instability? Is it a protective natural response?

The greatest medical discovery in the last 100 years is of its profound ignorance. Lewis Thomas and Lyall Watson

How little we know of a marvel called “intervertebral” disc.[1] The realization of one’s cerebral limitations ushers in a sense of humility that begets a sense of wonder that spawns a reverie.[1]

Standing on two legs is a special facet of human life. This unique lifestyle feature differentiates humans from all other animals. The predominance of extensor muscles located in the back of spine and nape of neck facilitates human standing posture and executes activities of bending, running, jumping, and performing acts of life. However, weakness of these muscles due to their disuse, misuse, or injury can lead to acute or chronic telescoping of the spinal segments or “vertical spinal instability.”[2] Recent concept is that it is not the age-related reduction in the water content of the disc or disc disease, but it is muscle weakness-related vertical spinal instability that initiates and propels the process of spinal “degeneration.”[3] Listhesis or telescoping of the facets that form the fulcrum of all spinal movements is the initial consequence of muscle incompetence and all other recognized and the so-called “pathological” events are secondary and probably “protective” natural maneuvers. Our earlier studies have identified that disc space reduction, disc bulges, buckling of posterior longitudinal ligament and ligamentum flavum, and osteophyte formation are processes consequent to vertical spinal instability.[3‑9] Neck pain, spasm of the muscles of nape of neck or back, and similar such symptoms are an attempt by nature to avoid excessive movements and potential worsening of instability or neural consequences. Vertical spinal instability, muscle spasms, and secondary “pathological” consequences result in reduction in the vertical height of the spinal column and consequent reduction in the dimensions of spinal canal and of intervertebral spinal neural foramina.

Chronic spinal instability as a result of gradual, long-standing, and progressive weakness of muscle provides an opportunity for natural protective processes to develop and mature. Such processes include chronic neck pain, muscle spasm, reduction in the intervertebral spaces leading to buckling of intervertebral ligaments such as posterior longitudinal ligament and ligamentum flavum, disc space reduction, bulging of disc into the spinal canal, osteophyte formation, and similar such so-called pathological events. Symptoms of neck and shoulder pain and tingling paresthesiae in the hands and fingers and eventually motor weakness seem to be natural attempts that limit the neck movements and subsequently body movements. Such instability is more often focused at the point of fulcrum of all spinal movements at the facet articulation. Due to lateral location of the facets, oblique profile, and location away from neural structures, identification of instability even on modern computer-based imaging is difficult if not impossible.[10] Radiologically identifiable structural instability occurs only as a delayed phenomenon. The very fact that surgery that involves “only-fixation” of the involved spinal segments results in immediate postoperative relief from symptoms is a testimony to the concept.[3‑9,11] Essentially, all the known “pathological” features of spinal degeneration can regress or disappear following such surgery. Our recent studies have identified eventual reduction in the size of osteophytes following a surgical procedure that involves only spinal stabilization.[12]

Recent evidence suggests that acute disc herniation is either a result or a cause of spinal instability. Injury to the back as a result of lifting of heavyweight or sudden jerk has been recognized as initiating factor that results in acute disc herniation “secondary” to spinal instability? Is it a protective natural response? J Craniovert Jun Spine 2021:12:213-5.
herniation. Injury to the annulus fibrosus with or without acute compression of the disc pulp is the probable cause of disc herniation. The clear radiological observation of herniated disc, evidence of neural compression, and presence of related neural symptoms have historically blamed such disc herniation as the culprit and the entire management protocol is focused on disc herniation.

Conventionally and universally, restraining back movements by using external arthrodesis and “complete” rest have been identified to result in relief from symptoms. If such treatment is successful, it is generally recognized that direct surgical resection of the herniated disc can be avoided or is not necessary. It is well known that the long-term outcome of herniated disc is its resorption. It is possible that conservative nonsurgical treatment by stopping spinal movements results in healing of the torn spinal ligaments/muscles and spinal stability is restored.

However, “complete” bed rest is a difficult aim to achieve. With external arthrodesis with the help of cervical collar or lumbar belts, complete blockage of all spinal movements is difficult and the slightest movement can result in an acute exaggeration of pain. We recently proposed that “only fixation” of the involved spinal segments without any manipulation of the herniated disc might be the treatment.[3,11] We preferred transarticular screw fixation, wherein the fixation is focused at the site of fulcrum of all movements.[13] Such treatment can be considered to be “internal” arthrodesis and firm stabilization of the spinal segments, a procedure that any kind of external arthrodesis cannot match. Our remarkable clinical results following such treatment confirm the efficacy of such treatment. “All” symptoms that include radiating pain/paresthesiae or weakness have the potential to resolve in the immediate or early postoperative phase.

This observation that the symptoms resolve on spinal fixation is suggestive of the fact that the “abnormal” or “excessive” movement is the cause of symptoms and symptoms resolve as soon as such movements are stopped. It is unclear if the movements can result in compression of the disc bulge or exaggeration of disc herniation and resultant compression. The question is if disc herniation is a cause or effect of spinal instability and if it has a natural protective function. Like all other so-called “pathological” events such as osteophyte formation, buckling of the intervertebral ligaments, reduction in the disc space, herniation, protrusion of the intervertebral disc can also be a protective natural response aimed to counter spinal instability. The pain as a result of muscle/ligamentous injury and that related to muscle spasm is protective in nature and like elsewhere in the body aims to avoid or restrict spinal movements related to instability and provide an opportunity and environment for healing and spontaneous fusion/stabilization. Herniation of the disc and its related symptoms such as radiating pain, tingling paresthesiae, and weakness could also be natural phenomena aimed to restrict the individual to have complete bed rest and avoid any movements such that spinal stability can be restored as early as it is possible. Once local tissues heal, the related symptoms soon wane and the process of resorption of the herniated disc is initiated. We have observed immediate postoperative resolution of all symptoms followed instrumented fixation of the affected spinal segment. Like Chiari formation and syringomyelia are natural self-deformative and self-destructive protective processes in the event of atlantoaxial instability,[14] disc herniation can be a natural protective root “compressive” response in the event of spinal segmental instability. The natural protection is ultimately aimed for the preservation of movement and life.

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