

Pathophysiology of lumbar disc degeneration: a review of the literature

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FROM ABSTRACT

Lumbar disc degeneration occurs because of a variety of factors and results in a multitude of conditions.

Alterations in the vertebral endplate cause loss of disc nutrition and disc degeneration.

Aging, apoptosis, abnormalities in collagen, vascular ingrowth, loads placed on the disc, and abnormal proteoglycan all contribute to disc degeneration.

Some forms of disc degeneration lead to loss of height of the motion segment with concomitant changes in biomechanics of the segment.

Disc herniation with radiculopathy and chronic discogenic pain are the result of this degenerative process.

Abbreviations used in this paper:

ALL = anterior longitudinal ligament
PGE = prostaglandin E
PLL = posterior longitudinal ligament
VB = vertebral body

THESE AUTHORS ALSO NOTE:

Lumbar disc degeneration is common.

ANATOMY OF THE INTERVERTEBRAL DISC

The bands of the anulus are arranged in a specific pattern to resist forces placed on the lumbar spine.

The anulus is connected to the cartilaginous endplate and to the vertebral body by Sharpy fibers.

“The ALL and PLL further strengthen the disc space.”

The ALL is stronger than the PLL, and resists extension forces. The ALL is

innervated.

The PLL resists flexion forces. The PLL is richly innervated by nociceptors to the sinuvertebral nerve.

"The PLL strongly attaches to the annulus fibrosus, and frequently is torn in cases of free fragment disc herniation."

"A meningeal branch of the spinal nerve, better known as the recurrent sinuvertebral nerve, innervates the area around the disc space."

The sinuvertebral nerve has 2 sources:

- (1) A sensory branch.
- (2) "Afferent innervation to the sinuvertebral nerve arises via the rami communicantes from multiple superior and inferior dorsal root ganglia."

"In both human and animal studies, the outer anular regions are innervated, but the inner regions and nucleus pulposus are not innervated."

"Degenerated human lumbar discs have been shown to contain more nerve tissue and to be more vascular than normal discs."

PROTEOGLYCAN AND WATER CONTENT

"The strength of the lumbar disc is related to the fluid and proteoglycan content of the disc."

Proteoglycan is a molecule composed of a protein attached to a sugar.

Proteoglycans are hydrophilic and therefore bind water.

Proteoglycans are also known as glycosaminoglycans and include such structures as chondroitin and collagen.

Proteoglycans draw in water through osmosis into the nucleus pulposus.

Disc proteoglycans decrease with age, and therefore so does disc water content.

Disc hydration is also reduced with applied spinal loads, producing degenerative changes.

Disc degeneration may also have a genetic basis.

VASCULARITY OF THE DISC

The disc receives most of its nutrition by diffusion.

Disc nutrition is “facilitated by spinal motion.” **[IMPORTANT]**

Disc nutrition is supplied via the capillary beds of the cartilaginous VB endplate.

These capillary beds are from the distal branches of the interosseous arteries supplying the VB.

Vascular and lymphatic supplies are present in the anulus of patients who are up to 20 years old, but not afterwards.

Changes in disc vascular supply cause disc degeneration.

[IMPORTANT: autonomic concomitants]

COLLAGEN DISTRIBUTION

“Collagen is a widely distributed proteoglycan in the body.”

With age, disc collagen suffers from oxidative stress, increasing degenerative disease. **[Take Your Antioxidants]**

Collagen cross-links are important in the mechanical stability of the disc, and these cross-links also decrease with age.

ROLE OF APOPTOSIS

“Apoptosis is programmed cell death and occurs in both normal and pathological states.”

Apoptosis occurs at a higher rate in cases of free disc fragments compared with contained disc herniations, which is why free disc fragments have an increased rate of of resorption and remodeling of the extruded material.

MECHANICAL STRESS AND INFLAMMATORY COMPONENTS

“Disc degeneration is related to mechanical stresses.”

“Degeneration may begin in early adulthood and may change the disc in such a way that herniation is imminent.”

“Disease in either the facet joints or the disc is not reliably correlated with patient age.” **[IMPORTANT]**

Studies show that the range of motion of a degenerated disc is decreased.

Degenerated discs have reduced ability to withstand applied stresses.

“Degenerated discs have higher than normal concentrations of fibronectin, which may be elevated as part of the response to injury.” **[Fibrosis Of Repair]**

Applied disc stress may decrease the expression of Type II collagen and result in disorganization of the annulus fibrosus. **[IMPORTANT:Subluxation and the internal disc derangement of Clarence Gonstead, DC]**

The herniated disc induces an inflammatory response.

Importantly, “the presence of inflammation in patients does correlate with a better postoperative outcome when compared with those with herniated discs showing no inflammation.” **[WOW, one might question the use of anti-inflammatory drugs on potential surgical patients].**

“The painful disc may be the result of the effect of PGE-2, and, in fact, a positive straight-leg raise result has been correlated with the amount of PGE-2.” **[REMEMBER, PGE-2 is the pro-inflammatory eicosanoid formed from the omega-6 fatty acid arachidonic acid].**

DISC HERNIATION

The “symptoms of lumbar disc disease are the result of either herniation of the nucleus pulposus through a mechanically weak annulus fibrosus or from tearing of the annulus itself.”

“This can lead to radiculopathy from nerve root compression or an inflammatory process affecting nerve roots or the spinal cord.”

TERMINOLOGY:

- (1) A disc bulge is a symmetrical extension of the disc beyond the endplates.
- (2) A protrusion is a focal area of extension still attached to the disc.
- (3) An extruded fragment is one that is no longer connected to the disc.
- (4) A sequestered fragment is contained within the PLL.

CONCLUSIONS

Lumbar disc herniation is most probably because of a combination of longstanding degeneration and a subsequent change in the ability of the disc to resist applied stress. **[IMPORTANT]**

“Discs that are degenerated show abnormal vascularity, and abnormal distribution of collagen and collagen cross-links.”

“Radicular pain is often associated with disc herniation, which may be due, in part, to an inflammatory response to the portion of nucleus that has been extruded.”

This inflammation is characterized by a release of PGE-2.

KEY POINTS FROM DAN MURPHY

- (1) The intervertebral disc is richly innervated.
- (2) Degenerated discs have more nerve innervation than normal discs.
- (3) The integrity of the disc is dependent upon its proteoglycan content because proteoglycans bind water.
- (4) Disc proteoglycans can suffer from oxidative stress, increasing degeneration.
[AGAIN, Take Your Antioxidants]
- (5) The disc is avascular after age 20 years.
- (6) Proteoglycans draw water through osmosis into the nucleus pulposus through the cartilaginous vertebral endplates.
- (7) Therefore, disc nutrition requires appropriate spinal motion.
[Consistent with our understanding of the subluxations complex].
- (8) The range of motion of degenerated discs is reduced.
- (9) Degenerated discs are more fibrotic.
- (10) The vertebral body cartilaginous endplate is supplied by capillary beds.
- (11) Vascular compromise **[Including subluxations driven sympathetic autonomic concomitants]** will cause disc degeneration.
- (12) Altered mechanical stress **[subluxation complex]** results in disorganization of the annulus fibrosus.
- (13) Herniated disc induces an inflammatory response by releasing PGE-2, a pro-inflammatory eicosanoid formed from the omega-6 fatty acid arachidonic acid.
- (14) This inflammation results in local disc pain and radiculopathy.