# 2

## LUMBAR DISC DEGENERATION

Ali Cetin SARIOGLU M.D., Tuncay KANER M.D., Cimen ELIAS M.D.

Vertebral degeneration is a process that also includes bony and ligamentous structures degeneration. Vertebralcorpus, facet articulations, intervertebral discs (IVD) and ligaments are affected structures. Concordantly degeneration related to vertebral column aging process and vertebral column degeneration disease must be distinguished. In clinical evaluation there are not any certain symptoms to distinguish aging process from degenerative disease. Nevertheless to understand vertebral column natural aging process as a systematic process, it is also important to understand lumbar disc degeneration pathophysiology. Biomechanical and biochemical many changes play role in IVD degeneration including intrinsic, extrinsic and genetic factors. Compression and torsional injuries of vertebral column, excessive pressure and congenital anomalies causes over pressure on IVD. In addition to these factors atherosclerosis, vascular disease, anemia, immobilization, diabetes and tobacco usage increases disk degeneration. Chronic low back pain mostly known and important reason is IVD degeneration.

As well-known degenerative disk disease is thought to be primary reason of instability in vertebral segments. Finally in degenerative disc disease segmental instability found to be responsible of low back pain and treatment arranged according to this results.

In this chapter lumbar disc degeneration etiology and pathophysiology will be discussed.

### 1. Etiologic Factors in Disk Degeneration

IVD height forms %20 of vertebral column height. IVD has steady structure that connected with end

plates of 2 adjacent vertebral segments and concurrently it absorbs excessive pressure. Gelatinous structure called Nucleus pulposes that surrounded byanulusfibrosus of IVD and anulusfibrosus itself is responsible of controlled mobilization of IVD. Anterior longitudinal and posterior longitudinal ligaments are strengthen disk spaces. ALL is tougher than PLL and adheres to vertebrae more tighter than that anulusfibrosusdoes. Disc fluid amount decreases gradually by aging. In newborns disc tissue is well vascularized while after 30 years there are almost no vascularization. Disc tissue is nourished and oxygenated by diffusion from cartilaginous structure of end plates. Normal IVD has isotropic structure. Nucleus resembles a bag filled with liquids, which transmits pressure equally to end plates. In addition to that excessive pressure that applied on anulus is transmitted to other sides by nucleus which resembles to tire of a car. By disc degeneration isotropic feature of disc changes and aberrational pressure distribution occurs. This mentioned irregular pressure distribution is believed to be main reason for disc degeneration and low back pain.

Pressure and traumas that affects discs thought to be reason of disc degeneration also compression and torsional injuries also increases disc degeneration.

In several researches indicates that physical activity increases disc degeneration however there are some other researches that indicate the oppsite way.

Many clnical trials which took many years indicate that spinal fusions increase the pressure over the disc below and amplifies degeneration hillibrandand friends completed a research which took ten years in this research %26 of patient with overpressure in these segments after fusion causes much more degeneration in time, in addition to this, degeneration can occur as a clinical case such as degenerative disc disease.

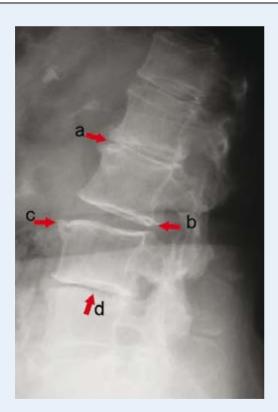
Age is an important parameter in disc degeneration in older age groups the percentage of patients with degenerated discs are higher. Smoking, physical activities, familial history, dibates mellitus, atherosclerosis, obesity and vascular diease increases this percentage just like age. unefficent disk nutrition is an important issue in disc degeneration. Disc nutrition is supplied by diffusion from cartilage tissue, it needs oxygen and glucose just like other cells in whole body structure. If nutrition is not sufficient lactic acid levels increases and pH levels decreases in the core of disc. Lower oxygen and pH levels rapid cell damage.

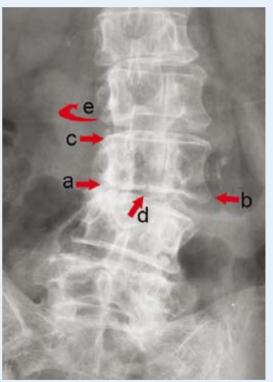
Genetics also found to be affective in all these degenerative risk factors. Vitamin D receptor genes Type 9 collagen genes matrix metalloproteinase genes are all related with ddiscdegeneration. In conclusion disc nutrition and mechanic pressure have the most massive effect on disc degeneration among all ethiologic factors.

#### 2. Pathogenesis

IVD degenerative changes includes stages below;

- Decreasing of disc height
- Irregularties in cartilogenous structure of end plates
- Sclerosis in end plates in disc space
- Ostheophyt formation





**Image2:** 68 years old woman patient has changes on direct lumbosacral AP and lateral X-rays in consequence of intervertebral disc degeneration.

**a-b)** Osteophyte formation **c)** Traction chipurs **d)** decrease intervertebral disc distance and cartilage deformations of end plate.

Pathologic degenerative changes in disc complex (vertebral corpus IVD and nucleus pulposes aging and post traumatic degenerative changes) begins with cartilogenous tissue degeneration of end plates. malnutrition in nucleus pulposes by fluid lost and changes in disc biochemistry is followed by degenerations.

By diffusion distruption glucose and oxygenation need is not sufficient.

Finally lactate accumulates in disk space and pH values decreases.

Under the acidic Phcircumstances, diffusion impairs by increase of cell destruction and decrease ofintervertebral disc liquid content. On the other hand, activations of proteinase and proteoglycan structure have massive changes, low pH degree give rise to that inflammation cells migration to vertebral disc. (32) Granulation tissue composes scar tissue, nucleus size decreases and annulus fibrosus wrinkles. Migration of scar tissue induce inner part of annulus fibrosus; fibrocartilagenous tissue, degeneration

and granulation tissue migration causes ruptures and also even protrusions. For the first time, Crock described "internal disc degeneration" (33). In defects that occurs as a result of fissures in cartilagenous plates may aslo induces internal herniations, that called "Schmorl nodules". (34)

The gas accumulation in the degenerated disk is called "vacuum phenomenon" (Figure 3). Mucoid degeneration formation results with instability. This cycle may even cause torsional instability and bulging of annulus fibrosus and may lead to further degeneration and sequels (8). At this stage, macromolecular disorders takes place instead of microcellular degeneration.

Degeneration of annulus structure results with torn in annulus, by this torn in the annulus, IVD fluid may exude and leads to pain in waist, legs and headaches. Low pH degree values as a result of necrotic ruptured discs slide to anterior and pain may occur in abdominal area (35). Degenerative discs is mostly unstable, causes low back pain originated from facet joint.





**Figure 3:** Fifty-year-old female patient, T1-weighted MR imaging, and vacuum gas accumulation due to degeneration at L4-L5 disc. The vacuum phenomenon is observed

In order to control pain associated with instability, lumbar spine hyperlordosis and facet articulation deadlock occurs spontaneously. This situation leads to hypertrophy of the posterior elements and spinal canal and foraminal stenosis (36,37) (Figure 4). Anatomically, the presence of annular fibers at an angle of 30 degrees in the range of the disk shows greater resistance to rotation only when anuler fibers are tighten. By decrease of disc space fibers are losen and permit rotational movements.

In degenerative disc disease; If disk space in height and distance decreases and depending on that relaxation of the annular fibers of the vertebral body occurs then around the long axis of rotation of the spine.

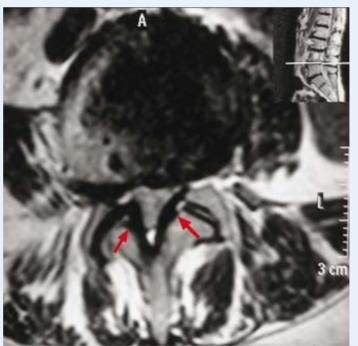
30-degree of angular orientation of the fibers of annulus fibrosus only prevents rotation of the annular fibers when it is stretched. As a result of narrowing of the distance of IVD fibers loosens and disc can easily rotate.

Thus, mechanical low back pain occurs depending on torsional instability. lumbarvertabra integrity disruption may results with discogenic pain. (8,38).

Instability associated with degeneration of the disc is described firstly by Knutsson (40). Frymoyer, has dived the instabilities into as a "primary segmental instability," and "secondary segmental instability". Frymoyer, mentions in his classification as the primary instability can be seen in patients with degenerative disc disease who has no previous surgery history and reported degenerative disc disease as the primary cause of instability (Figure 5). Pathology of discogenic pain and degenerative instability,

described by Kirkaldy-Willis and Farfan and segmental stability stated that minimal dysfunction could lead to major changes and degenerative process is explained in three phases:





**Figure 4:** Fifty-year-old female patient, sagittal and axial T2-weighted MR imaging sequences shows hypertrophy and spinal central and foramina stenosis associated with it.

#### 2. a. Stage of dysfunction

The characteristic features of this phase degeneration of nucleus pulposes, internal rupture of the disc, annular tears and facet joint degeneration forms.

#### 2. b. Stage of instability

Stage of instability includes posterior facet joint capsules and ligaments relaxation, reduction of height in disc space and segmental instability seems to increase.

#### 2. c. Stage of restabilization

At this stage, the prominent pathologies are increase of collagen in the disc, loss of disc elasticity, facet hypertrophy and osteophyte, fibrosus formation. In this last stage, foraminal and central spinal stenosis develop.

In the chronic instability classification Benzel defined degenerative disc disease as a result of disc degeneration, linked to "dysfunctional segmental motion"

Because, as mentioned, the biochemical and biomechanical processes that begin with excessive disc degeneration increase by disc degeneration with aging and increases the number of annular tears.

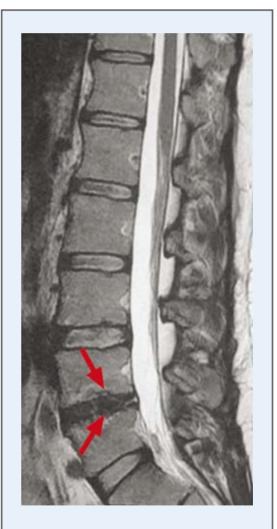
Beginning of the progression of intervertebral disc degeneration as a result of the disk space is breaking down the distance, in fact, a segment of the disk instability begins.

This is due to instability of disc degeneration. Frymoyer <sup>(4,5)</sup>, named as "the primary segmental instability," Kirkaldy-Willis <sup>(41)</sup>, called discogenic pain and degeneration processes described in the "phase instability"; Benzel <sup>(6)</sup> stated as degenerative disc disease, chronic instabilities classifying in "dysfunctional segmental motion".

By Benzel, the progression of disc degeneration as a result of the reducing in distance and end plates connected with the annular fibers at 30 degrees due to reduced resistance refers to "torsional instability" (8). Annular tears in the intervertebral disc spacecollapsing, facet joint degeneration beginning, and image with magnetic resonance imaging (MRI) which shows decrease in disk intensity as a black disc and consequences occurred by reduced translational deformity seen in dynamic X-rays; shows degenerative and chronic instability statement.

In this mentioned statement, protrusions of the disc also can be seen and described by Crock for the first time <sup>(33)</sup>, as a "internal disc destruction" which can be painful. At this point, pain disappeares by resting of patient and the pain increased, especially while standing and in motion. Recognition of this process is very important and treatments at this stage should be under consideration of instability.

With patients who do not respond to medical treatments, surgical approach could be done either by excising protruded disc or subtotal discectomy however it only increases low back pain and



**Figure 5:** Thirty-seven year-old female patient with sagittal sections in T2-weighted MR imaging; the L4-L5 degenerative disc disease that is responsible of instability.

segmental instability. Chronic instability, instability stage in the process of degeneration of the spinal segment, if left untreated can progress and end up with "restabilization stage" which is described by Kirkaldy-Willis. To prevent progression of disc degeneration and segmental instability some reactions occur as a result of instability (subperiosteal bone formation) and consequently osteophytes occur (8). Presence of osteophytes at this stage triggers the facet joints hypertrophy and fibrosis. With osteophyte formation disc degeneration process slows down and stabilize the instable segment (3,41). Progression of disc degeneration, facet joints and ligaments and thickening of the central or foraminal spinal stenosis may develop after the formation of osteophytes. In some cases the instability process proceed degenerative spondylolisthesis is developed. This is also another form of chronic instability and it named as "slow instability" ("glacial instability") by Benzel.

#### 3. Conclusion

Intervertebral disc degeneration, is a complex mechanisms which is trying to be defined in a certain situation. Despite of Biochemical, biomechanical, and investigation of the genetic causes, its mechanism is not completely understood. The etiologic factors that the disc nutritional disorders and mechanical

loading on the disc is considered to be the most important reasons. Lumbar disc herniations is common process in degenerative disc disease and does not occur in every case. Over the years, various researchers tried to explain the chronic and degenerative instability, has an important role in the process of degenerative vertebrae segment. Treatment approaches and surgical options should be planned accordingly and considering chronic instability. Instability phase is painful process that decrease quality of life in patients and it recovers by restabilistaion phase but still it takes long period of time with excessive pain. Medical treatment and exercise programs are insufficient in many cases, a selection of fusion surgery, although the effective stabilisation is effective choice, it has been serious surgery. Therefore, minimally invasive fusion systems are mainly chosen solution. However, dynamic stabilization without fusion solves these pathologies and its mortality and morbidity rate is very low. Considering fusion surgery complications with variable techniques, in future a posterior dynamic stabilization will take place of fusion surgery for treatment of chronic instability. to understand possibilities in Disc degeneration formation and prevention mechanisms and for disc regeneration mechanism there are many researches which gives hope to understand but still for fully understanding new researches are still needed

#### References

- Malinsky J: The ontogenetic development of nerve terminitions in the intervertebral discs of man. Acta Anat 38:96-102, 1959.
- 2- Çakmak A: Yaşlanan omurga- Lomber dejenerasyon. Türk Fiz Tıp Rehab Dergi 52 (Özel Ek A): A26-A31, 2006.
- 3- Erman T, Çetinalp NE; Lomber disk dejenerasyonu, rejenerasyon ve doğal Seyir. Koç K ed: Lomber dejeneratif disk hastalığı. Ankara, Buluş Matbaacılık, 37-50, 2008.
- 4- Frymoyer JW: Segmental instability. In Frymoyer JW, (ed): The adult spine. New York: Raven Press, 1991, 1873-1891.
- 5- Frymoyer SW, Selby DK: Segmental instability. Rationale for treatment. Spine 10:280-286, 1985.
- 6- Benzel EC: Stability and instability of the spine. In Biomechanics of spine stabilization. New York, Thime, Copyright by AANS, 29-43, 2001.
- 7- Martin MD, Boxell CM, Malone DG; Pathophysiology of lumbar disc degeneration: A review of the literature. Neurosurg Focus 13(2):1-6, 2002.
- 8- Benzel EC: Degenerative and inflammatory diseases of the spine. In Biomechanics of spine stabilization. New York, Thime, Copyright by AANS, 45-60, 2001.
- 9- Hukins DW: A simple model for the function of proteoglycans and collagen in the response to compression of the intervertebral disc. Proc R Soc Lond B Biol Sci 249:281285, 1992.
- 10- Mulholland RC, Sengupta DK: Rationale, principles and experimental evaluation of the concept of soft stabilization. Eur Spine J 11(2):S198-205, 2002.
- 11- Krag MH, Seroussi RE, Wilder DG, Pope MH: Internal displacement distrubution from in vitro loading of human thoracic and lumbar spinal motion segments: Experimental results and theorical predictions. Spine 12(10):1007, 1987.
- 12- Arnoczky SP, McDevitt CA: The meniscus: Structure function, repair and replacement. In Buckwalter JA, Einhorn TA, et al (eds): Orthopaedic basic science: Biology and bicomechanics off the musculoskeletal system. Rosemont, American Academy of Orthopaedic Surgeons, 2000, pp 557-566
- 13- Battie MC, Videman T: Lumbar disc degeneration epidemiologyp and genetic influences. Spine 29(23): 2679-2690, 2004.
- 14- Ong A, Anderson J, Roche J: A Pilot Study of the Prevalence of Lumbar disc degeneration in Elite Athletes

- with Lower Back Pain at the Sydney 2000 Olympic Games. Br J Sports Med. 37(3): 263-266, 2003.
- 15- Videman T, Sarna S, Battie MC, et al: The long-term effects of physical loading and exercise lifestyles on back-related symptomes, disability and spinal pathology among men. Spine 20(6):699-709, 1995.
- 16- Lehmann TR, Spratt KF, Tozzi JE, et al: Long-term follow-up of lower lumbar fusion patients. Spine 12(2):97-104, 1987.
- 17- Hilibrand AS, Carlson GB, Palumbo MA, et al: Radioculopathy and myelopathy at segments adjacent to the site of a previous anterior cervical arthrodesis. J Bone Joint Surg Am 81:519-528, 1999.
- 18- Miller JA, Schmatz C, Schultz AB: Lumbar disc degeneration: Correlation with age, sex, and spine level in 600 autopsy specimens. Spine 13:173-178, 1988.
- 19- Kalichman L, Hunter DJ: The genetics of intervertebral disc degeneration. Familial predisposition and heritability estimation. Joint Bone Spine 1-5: 2008.
- 20- Pye SR, Reid D, Adams J, et al: Influence of weight, body mass index and lifestyle factors on radiographic features of lumbar disc degeneration. Ann Rheum Dis 66:426-427, 2007.
- 21- Urban JPG, Roberts S: Degeneration of the intervertebral disc. Arthritis Res Ther 5(3):120-30, 2003.
- 22- Holm SH. Nutrition of the intervertebral disc. In: Weinstein JN, Wiesel SW, (ed). The lumbar spine. Philadelphia, W.B. Saunders Corp, 1990, pp 244-260.
- 23- Wallach CJ, Gilbertson LG, Kang JD; Gene therapy applications for intervertebral disc degeneration. Spine 28(15S):S93-S98, 2003.
- 24- Horner HA, Urban JP. 2001 Volvo Award Winner in Basic Science Studies: Effect of nutrient supply on the viability of cells from the nucleus pulposus of the intervertebral disc. Spine 26: 2543-49, 2001.
- 25- Ala-Kokko L: Genetic risc factors for lumbar disc disease. Ann Med 34:42-47, 2002.
- 26- Videman T, Leppavuori J, Kaprio J, et al: Intragenic polymorphism of the vitamin D receptor gene associated with intervertebral disc degeneration. Spine 23: 2477-2485, 1998.
- 27- Annunen S, Paassilta P, Lohiniva J, et al: An allele of COL9A2 associated with intervertebral disc disease. Science 285(5426): 409-412, 1999.
- 28- Takahashi M, Haro H, Wakabayashi Y, et al: The association of degeneration of the intervertebral disc with 5a/6a polymorphism in the promoter of the human matrix metalloproteinase -3 gene. J Bone Joint Surg Br 83(4):491-495, 2001.

- 29- Holm S: Pathophysiology of disc degeneration. Acta Orthop Scand Suppl 251:13-15, 1993.
- 30- Schmorl G, Junghanns H. The human spine in health and disease. (2<sup>nd</sup> ed), New York, Grune and Stratton, 1971.
- 31- Doita M, Kananati T, Harada T, et al: Immunohistologic study of the ruptured intervertebral disc of the lumbar spine. Spine 21(2):235-241, 1996.
- 32- Herkowitz HN, Dvorak J, Bel G, et al: The lumbar spine. (3<sup>rd</sup> ed), Philadelphia, Lippincott Williams and Wilkins, 2004.
- 33- Crock HV: Internal disc disruption, a challenge to disc prolapse fifty years on. Spine 11 (6):650-653, 1986.
- 34- Mayfield FH: Cervical spondylosis: A comparison of the anterior and posterior approaches. Clin Neurosurg 13:181-188,1965.
- 35- Fernstrom U: Intervertebral disc degeneration with abdominal pain. Acta Chir Scand 113(6):436-437, 1957.

- 36- Kirkaldy-Willis WH, Paine KW, Cauchoix J, McIvor G: Lumbar spinal stenosis. Clin Orthop 99:30-50, 1974.
- 37- Kirkaldy-Willis WH, McIvor GW: Spinal stenosis. Clin Orthop 115:2-3, 1976.
- 38- Schmidt TA, An HS, Lim TH, et al: The stiffness of lumbar spinal motion segments with a high-intensity zone in anulus fibrosus. Spine 23:2167-2173, 1998.
- 39- Haid RW, Dickman CA: Instrumentation and fusion for discogenic disease of the lumbosacral spine. Neurosurgery Clinics of North America 4(1):135-148, 1993.
- 40- Knutsson F: The instability associated with disc degeneration in the lumbar spine. Acta Radiol 25:593-609, 1944.
- 41- Kirkaldy-Willis WH, Farfan HF: Instability of the lumbar spine. Clinical Orthop Relat Res. 165:110-123, 1982.