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Review Article

Minimal Invasive Approaches to Lumbar Degenerative Stenosis and Degenerative Disc Disease

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Introduction

Spinal degeneration occurs because of primary or secondary spondyloarthritis and disc degeneration [1]. Some authors associate the onset of degeneration involving intervertebral disc and facet joints with the arthritis of facet joint while it is generally agreed that it occurs as a result of disc degeneration [1,2]. Disc structure starts degenerating in the third decade, water loss occurs in nucleus pulposus, disc height decreases with annular tears, facet joint distance gets longer, spine is inclined to abnormal movement and instability and get vulnerable to traumas because of ligament laxity. Because of repeated traumas combined with inflammatory factors, cartilage gets thinner, annular tears are enlarged, facet joint synovitis develops, cartilage is destructed and osteophytes develop. Posterior movement of disc structures causes stenosis in the spinal canal, hypertrophy develops in facet joints and lighamentum gets thicker. The result is degenerative stenosis [1-3].

Disc Degeneration

Intervertebral disc, which is one of the primary causes of the onset of spinal degeneration, has a vascular structure. It is composed of chondrocytes and fibroblast-like cells in extracellular matrix. Disc includes two main regions, which are nucleus pulposus (NP) and Annulus Fibrosis (AF). Chondroblasts and type-2 collagen are organized mainly in the gelatinous structure of NP I in the inner space of the disc while the basic structure of AF, which is lamellar, mainly consists of fibroblasts surrounded by type-1 collagen. Both cell groups synthesize the appropriate matrix they are in. In the lower and upper regions of the disc (endplates), there are chondrocyte cells that synthesize hyaline cartilage surrounded by a thin cortical bone [1].

Disc degeneration is characterized by NP dehydration and clefting that may turn into fractures in the endplate in addition to AF tear. Annulus loses its collagen fibril organization due to degeneration resulting in myxomatous degeneration. The number of lamellae increases, cell distribution is disturbed and clusters are formed. NP loses its water content and height, cavities are formed and it enlarges towards the posteriolateral side. Endplate degeneration is associated with subchondral sclerosis and calcification of hyaline cartilage. They all because

thinning of the disc, which loses its elasticity; as a result, nucleus and annular regions cannot be distinguished. Disc herniations can be classified as containment of nucleus inside annular ligament (Contained- NP) and protrusion of the material outside the disc (noncontained-NP). In non-contained herniations, annular tear is usually too large. Degeneration is advanced [1].

At cellular level, disc degenerastion starts with the increased cellular pairing and grouping in the NP region. The distribution of Actin and Vimentin, which are teh main proteins of the cell skeleton, is disrupted. The shape of the cell is diformed. Gap connections with connexin 43 and 45, which provide intercellular connection, decrease. In addition to all these mechanical causes, nutrient and oxygen diffusion in cells is also diminished from metabolic perspective [1].

Diffusion occurs with posterior and anterior vertebral vessels. Factors such as endplate calcification, narrowing of lamina cribrosa pores and decreased local blood flow increase diffusion. With increased amount of lactate due to anaerobic metabolism, matrix synthesis is degraded. Martrix degradation increases and degraded molecules deposit. Combined with genetic/ systemic factors and smoking; necrosis increases, NP is hyalinized, annulus becomes weaker due to disorganization, proteoglycan distribution alters and water retention decreases [1].

In a degenerated disc, matrix proteins are also altered. Proteoglycans provide viscoelasticity with water retention and increase the tensile- compressive strength of discs. Chondroitin sulfate and keratan sulfate are predominant proteoglycans, which aggregate by binding on hyaluronat molecules. The largest aggregating molecule is aggrecan, which is mainly contained in AF. Versican, decorin, biglycan, fibromodulin and lumican, which are mainly found in fetal disc, are also present. Link proteins stabilize proteoglycans as glycoprotein. Chondroitin sulfate synthesis is disrupted due to degeneration and replaced by keratan sulfate. This leads to reduced water retention in NP and degradation of gel form. Furthermore, reduced water decreases diffusion at molecular level. Collagen and matrix connections provide mechanical resistance and stability to discs. Collagen Type 2 increases resistance to compressive forces. This stability that is preserved by cross connections is distorted due to degeneration and replacement of type 1, 2,2 3i and 5 by type 1, 4 and X. At advanced stages of degeneration, anaerobic respiration impairs cross connections and removes cross connections. Fibronectin is a glycoprotein with increased release in osteoarthritis that includes binding points of collagen-glycoprotein-integrin and membrane proteins. In recent years, it has been found decrease proteoglycan synthesis in NP l in a degenerated disc while increasing proteoglycan synthesis in AF with an inverse effect and its secretion has been found to increase during annulus repair. Fibronectin particles have been observed to stop the production of aggrecan that originates from chondrocytes and increase metalloproteinase, which is responsible for cartilage degradation. A polymorphism in aggrecan gene has been associated with disc degeneration. Differences in the sequence of protein chains are inculpated for degenerated discs. Chondromodulin-l (ChM-I) is thought to play a role in chondroprotective effect by preventing vascularization and fibrous change in early degeneration of discs. This molecule is secreted during the gestational period and ensures the growth of chondrocytes, which are the cartilage-related growth plate. It is thought to be also secreted in matured NP and AF cells. Other tissues are also affected due to the spontaneous elevation of inflammatory mediators such as nitric oxide (NO), interleukin-l B (IL-I interleukin-6 (IL-6).ı tumor necrosis factor alpha (TNF-oc). prostaglandin E2 (PGE2).ı matrix metalloproteinase (MMP) in disc cells. Proteoglycan synthesis is inhibited in articular cartilage. Increased IL1 starts cartilage degradation. MMP increases its elevation while exogenous IL-I p, NO, İL-6 and PG-E2 increases inflammation. Phospholipase activation due to the migration of CD68 cells during neovascularization is the main cause of pain and destruction. This mechanism may explain the inflammatory emchanism of facet joint degeneration associated with disc [1].

Mechanical factors during disc degeneration increase endplate damage, decrease proteoglycan synthesis due to increased NO content caused by increased intradiscal hydrostatic pressure and decrease water retention. Vibration decreases intracellular aggrecan content. As a result, increased MMP-I leads to matrix degradation. Moreover, vibration disrupts controlled flow of ATP in Ca channels. As a consequence, cell feed is disrupted, matrix production decreases, degradation and degeneration develop [1].

The impact of growth factors has been observed in degeneration as external factors. The severity of these effects varies at different stages from apoptosis to matrix organization. In degenerated discs, presence of LacZ and Luciferase signs demonstrate that genetic inheritance may occur in degeneration independent of age and sex. It is argued that the genetic factors of disc generation, maybe its root cause can be eliminated in the future through gene transfer with adenoviruses. Masuda et al. increased cell and matrix proteoglycan synthesis in rats through the mitogenic effect of recombinant human osteogenic protein-l (rHOP-1). Growth factors can be used in treatment as they have similar effects [1].

In short, disc degeneration is caused by several extrinsic, intrinsic and genetic factors. The main framework to determine treatment options is to decide at which stage the condition that results in stenosis in the functional lower segment of the spine develops.

Pathological anatomy of degenerated lumbar spine

Spinal canal stenosis may develop in the central, lateral recess and pedicular regions on coronal plane. Stenosis on the sagittal plane may occur at pedicular, intermediate and disc levels [3,4].

Lateral stenosis may occur at three levels including the entrance of the spinal to the foramen (subarticular), foramen and its outlet (extraforaminal). The first part described is located on encepahlon, medial and lower side of superior articular facet. It only has anterior and posterior osseous walls. Medial and lateral sides are normally open. The mid segment includes the foramen and is

located below the pars interarticularis of the lamina and pedicle. The vertebral body forms the anterior wall. Pars interarticularis forms the posterior wall while the pedicle forms the lateral wall. Medial wall opens up to the spinal canal and is normally open. Its outlet is surrounded by intervertebral foramen. Disc is located in its anterior side, and lower part of the facet joint lies in the lateral side [3-5].

Anatomical classification of degenerative spine required therapeutic classification due to the emerging need to plan treatment. Hansraj divides stenosis into two categories, which are simple or typical and complex. According to this classification, typical stenosis refers to cases who don't have instability or have first grade spondylolisthesis and scoliosis less than 20 degrees. These patients usually benefit from decompression therapies alone. For complicated cases, decompression therapy may have to be combined with fusion and instrumentation [5,6].

Clinical Picture

It usually becomes symptomatic in older age. It is more prevalent among women. L3-4, 14-5 levels are the most commonly affected segments. Cervical involvement is observed in 5% of patients [2]. Patients report back pain, low-back pain as well as pain in hip, thigh, legs and feet. Bilateral involvement is common. Neurological claudication, increased pain while walking and standing, pain relief while lying down and extending legs and increased pain with concussion are typical. Posture is slightly in flexion.

With increased pain, functional capacity gradually decreases and walking distance gets shorter. Daily activities are hampered. Pain may be at a varying scale. Visual analogue pain scoring system-VAPS- is one of the most widely accepted scale [7]. Work-related disability can be scored with work-related disability scoring system (WL-26). Deyo categorized questions related to spinal diseases in six groups under core set and created a useful system. Impact of spinal diseases on functional capacity can be quantified using disability-scoring systems such as Oswestry. Scoring systems such as SF-36 that assess overall health status are helpful before surgery [6,7]. The items in these systems should be easy to understand for patients. Roland disability scoring system was translated into Turkish and its validity was proven statistically. It is a test specific and sensitive to low back pain [8].

The need for analgesics and response to analgesics should be recorded. This may given an idea about the degree of stenosis. Bladder functions should be definetely assessed.

In lumbar lordosis; flattening, paravertebral spasm, increased pain with movement and decreased range of motion especially in extension may be common findings. Range of flexion decreases but is concomitant with pain. Straight leg raising test is usually negative. Decreased motor strength can be detected with provocative tests. Decreased sensation may sometimes be selective in the relevant dermatoma but it is not sensitive as minor sensory changes are expected at these ages [6-9].

Radiologically, deceased disc space, osteophytes and faced hypertrophy are pathognomonic. Defects in pars interarticularis, spondylolysis and narrowed pedicle spaces should be recorded for stenosis. The boundaries of foramens should be examined, hypertrophy of facet joints and relation of osteophytes with the canal should be assessed. Presence of scoliosis, kyphosis, hyperlordosis, sacralization and lumbarization should be investigated via flexion-extension and dynamic scans in standing posture. In spondylolysis, fibrocartilage-like structure is present in the etiology of foraminal stenosis. Listhesis degree should be graded with standard indicators, Psoas shadow and robustness of pedicles should be noted [9,10].

Tomographic evaluation remains important to understand the organization of osteophytes. Lateral recess and foraminal structures limited by bony structures can be clearly evaluated. Magnetic resonance imaging (MRI) decreased tomography and myelographical evaluations substantially. With MRI, relation of disc structures with the canal, intradiscal pathologies and fibrous tissues causing foraminal stenosis can be assessed. MRI is also the most sensitive evaluation method for disc degeneration. However, CT-myelogram is as valuable as MRI for pre- operative planing in cases who have metal implants and are contraindicated for MRI [5,9-11].

In laboratory tests, neurofilaments specific to nerve injuries and proteins like S-1 00 have been demonstrated to increase in the cerebrospinal fluid (CSF) and blood. Total protein, albumin, lgG, IL-8 have been found to increase in CSF while ApoE amount has been found to increase in both CSF and plasma [5].

Discography is the most effective method to reveal disc pathology and take a dynamic decision in treatment planning. Discography also facilitates point targeting in the treatment of complicated radicular symptoms, locating the annular tear, and determining its relation with the clinical picture. Injection of an anaesthetic agent and steroid into disc may provide therapeutic effect and help physicians in differential diagnosis. White and Pancabi demonstrated the effects of pathological loads on disc by measuring disc pressure. Dynamic measurement of disc pressure can be taken as a reference to reveal mechanical problems.

EMG, which is the most commonly used electrodiagnostic tests doubtlessly, has an objective contribution. It shows the exact radicular level of lesions. It is more sensitive to work with evoked sensory potentials. Electrodiagnostic tests do not show decompression or treatment area. However, surgical site should be decided by comparing with other diagnostic tests. Diagnostic discography should be definetely repeated peroperatively to confirm the surgical intervention area. Selective root blocks can be used to differentiate the cause of pain in multi-segmental stenosis [5,9-12].

Diagnostic algorithm provides guidance for treatment planning.

Flow chart should first differentiate low-back pain due to medical diseases and non-medical pain. Pain that does not respond to conservative treatment and bed-rest requires the restart of differential diagnosis steps.

Differential Diagnosis

Disc herniations should be assessed very carefully for differential diagnosis. Disc usually bulges slightly in a stenotic degenerated spine. Symptoms should not be attributed to a disc disease and treatment should be limited to only dsicectomy or medical therapy.

Medical evaluation flow chart should be followed carefully to differentiate medical and non-mechanical low-back pain [9].

If cauda equina syndrome develops acutely, diffuse disc herniation can be suspected. For differential diagnosis, spinal cord tumors, primary and metastatic bone tumors, infections and fractures should be considered [2].

Vascular claudication is the most common clinical condition. Such pain increases with walking and lying down contrary to stenosis and decreases while standing. A careful vascular examination helps diagnosis. EMG is necessary for differential diagnosis in patients with diabetic neuropathy [2].

Treatment

Anti-inflammatory treatment is the first line for degenerative lumbalgia. Muscle relaxants can be combined with physical therapy for reflex muscle spasms. In resistant cases, epidural steroid and anesthetics can facilitate switch to functional therapies [13,14]. Gababentin or amiltriptilin combinations provide significant results in cases of epidural injection [13]. In degenerative lumbalgia, successful outcomes have been reported with the use of calcitonin [10]. Cases, who do not respond to conservative therapies, have neurological dysfunction (bladder, radicular motor deficit etc.), have low functional disability scores can be referred to surgical treatment. Surgery should decrease pain, increase mobility and prevent neurological deficit. The goal of the treatment is to provide adequate decompression and preserve joints and pedicles to restore stability. Today's surgery can be summarized as ensuring decompression by preserving anatomic integrity and avoiding fusion as much as possible. Minimally invasive procedures can meet these needs with increasingly advancing techniques. Treatable stenosis can easily be accessed with bone resections through percutaneous operations, scope guided interventions, and decompression and fusion can be performed through the vital vascular and nervous structures. Open surgical decompression operations have decreased pulmonary complications under spinal anesthesia. However, peroperative events which may increase CSF pressure (coughing etc.) may lead to large tears and tears hard to treat [1 1].

Surgical treatment is planned by assessing: 1. location of stenosis, 2. number of segments involved, 3. stability, 4. degenerative spondylolysthesis, 5. past history of surgical treatment, recurrence and iatrogenic factors and 6. Concomitant scoliosis and kyphosis. Surgerical treatment flow chart is summarized [11].

Non-Fusion Techniques and Disc Surgery

Disc degeneration and decreased disc height are the main reasons of spinal degeneration. Preservation of disc height before facet joint involvement occurs can prevent degeneration. Disc damage and damage- related pain can be relieved by damage repair. Several treatment methods are used for anulus and nucleus [15,16].

Lyman W. Smith performed the first trial-targeting nucleus in disc surgery in 1963 through chimopapain injection. Chimopapain degrades proteoglicans contained in nucleus after injection into nucleus, decreases volume and ensures decompression. However, it creates an effect that damages neural tissues. Mortality cases due to transverse myelitis, paraplegia and anaphylactic shock have been reported [11,15].

Techniques that target nucleus in nucleus containing disc herniations disk can be grouped under nucleoplasty. It has developed as a safe and effective therapy performed under local anaesthesia and becoming popular with laser discectomy and nucleotomy in early 1990s with shorter rehabilitation time [17].

The goal is to evaporate water content of nucleus by using similar equipment used in annuloplasty and energy. In this way, pressure in posterior annulus decreases. One should be more selective in patient selections compared to annuloplasty.

In nucleoplasty, Arthrocare Perc-D Coblation device, which is among the options, applies localized energy to nucleus and evaporates it with bipolar radioenergy. Around six canals are opened in nucleus for decompression and nucleus is converted to plasma form with its water content and removed out through cannula [17-20].

Thermal energy in nucleus decompression is applied with radiwave Radionics probes. In this therapy briefly abbreviated as PIRFT, heat effect of energy is used. Contrary to annuloplasty, in nucleoplasty, the temperature reaches 70-80 degrees. The contribution of this energy to annular denervataion is controversial while its pain relief mechanism has not been clarified yet [17,19].

In laser nucleoplasty, evaporation effect and heat effect of energy are reflected in nucleus proportionally. Nucleus is degraded by laser energy.

Intradiscal electrothermal therapy (IDET) is a treatment procedure for annulus. The goal of this technique is to treat annulus tear, that's why it is referred to as ''Annuloplasty". In 1997, Saai and Saal intervened defects in annular tears with a thermal wire. The basis of this treatment is the stabilization of collagen fibrils on arthroscopic capsulography. Thermal effect can be obtained from

electrothermal, radio wave or laser energy. With thermal effect, symptoms of collagen stabilization and annulus denervation regress. Neural structures are damaged in thermal therapy when it exceeds 42 Celsius [17].

The argument that narrowed disc space is the onset of degenerative spine process resulted in disc replacement materials. Facet instability, foraminal narrowing and subsequent degenerative conditions are secondary to the narrowing of disc space. To restore disc space, artificial nucleus replacements (ANR) have been tried. Polylmethylmetacrylate injection into nucleus and silicon materials have been disappointing. Metal nucleus results published by Fernstrom in 1966 are still controversial. Urbaniak observed that reactive bone formation and resorption continued in silicon-dacron composite implants in chimpanzees. An ideal material described by Edeland in 1981 should have vital functions such as water permeability in addition to nucleus-like tensile reactions. Ray and Gobbin created nucleus-like effect by using hygoscopic thixotroPic gel like hyaluronic acid with polymeric material impregnated high molecular weight polyethilene fiber capsule. Hou et al. Completed cadaver biomechanical study on lumbar intervertebral disc prosthetics (LIDP) shaped like a horseshoe they implanted with anterior paramedian retroperitoneal access. Eastomer reinforced polyurethane nucleus modified by Sulzer Spine-tech, hydrogel nucleus that can be delivered through a 5 mm cannula developed by Rao and Higham in 1991 and finally Ray modification that includes hydrogel in a polyethilene sheath (Prosthetic Disc Nucleus PDN). Are aim at increasing disc height and enabling nucleus to give a physiological response to overloads.

Scheme on anatomical locations of degenerative Lateral stenosis is classified as subarticular, foraminal and extraforaminal stenosis while central stenosis may occur at pedicular disc and interdiscal levels (inspired by Kuslich [27].

Sizing is the most important technical problem in disc prosthetics. Undersized or oversized disc prosthetics will pose a challenge [1,7,9,11].

Decompressive Procedures

They are planned according to the growth zone of stenosis. The goal is to eliminate pressure without impairing spinal stability. Procedures, which may cause instability, should be fixed with fusion procedures.

Central canal stenosis: Stenotic segment is treated through lumbar laminectomy. Decompression should be started from maximum narrowing zone, which should be enlarged in caudal and cepalic directions. Medial facet joint should be preserved to to prevent instability. Decompression is terminated when one makes sure that nerve root is relaxed. If there is any sense of locking in dura, the medial size of superior facet can be included in excision [1,4].

In lateral canal stenosis, nerve root can be treated with unilateral laminotomy. Stenosis at the inlet requires medial facetectomy. Facetectomy should be as long as it can ensure 1 cm medialization of nerve. In case of stenosis in the middle part, dorsal root is compressed. Total facetectomy should include pars region for decompression. For stability, fusion and instrumentation are needed. Osteophytes originating from hyperthrophic facet at the outlet and osteopthitic edges around disc lead to compression. With open techniques, this region is decompressed through Witse paraspinal approach. Transvers process is excised to access the region. The success of minimally invasive techniques such as foraminoscopyand patient satisfaction are higher. Mr Knight et al. perform fusion decompression in their minimally invasive foraminoscopic decompression treatments. (4/10,1 6,1 8720). Knight et al. reported successful results by applying annuloplasty and nucleoplasty in the same session in addition to decompression through the combined use of laser and radiowave energies [12].

Multiple laminotomies can be performed for mild to moderate stenosis by preserving spinous process in the midline. Expansive Lumbar Laminoplasty which was a technique applied by Tsuji et al. for the first time provides decompression by preserving stability [11].

In Distraction Laminoplasty, lumbar canal is decompressed by preserving maximum bone. Distraction devices are used to excise the medial side of facet and inner areas of lamina [11].

Distraction devices such as spinous process x-stop and PEEK decrease the compression in the narrowing area due to ligamentum flavum through indirect decompression. It can be performed under local anaesthesia [11].

Dynamic Intervertebral disc prosthetics is an emerging technique as an alternative non-fusion technique to fusion. Since Edeland, constrained or non-constrained disc prosthetics fixed on bone have been designed such as total joint prosthetics that allow mobility. The validity of these implants is controversial in animal and biomechanical experiments such as Kostuik design while their main goal is preserve spinal mobility by preserving disc height [21].

Fusion Surgeries

They preserve stability through arthrodesis. Posterior instrumentation and pedicle screw fixation are performed in large decompressions leading to instability and multisegmental laminectomies. In the presence of spondylolisthesis and mechanically distorted spine like scoliosis, they are used to provide corrective effect and prevent progression. Distraction and correction of alignment after a large decompression may contribute to decompression. Arthrodesis relieves pain and prevents progression. As fusion can be achieved through rigid fixation, it can be combined by performing it between vertebral bodies in addition to posterior elements. Instrumentation is needed to achieve fusion and preserve stability until fusion. Several

studies comparing pseudoarthrosis rates argue that pedicle screw fixation should be replaced by spondylolisthesis, scoliosis and multisegmental decompression as well as selective fusion.

Interbody Fusion

It can be performed posteriorly or anteriorly. It allows selective fusion. It can also be performed under scope in addition to open procedure [10-12]. It is particularly popular in scopic decompression surgeries requiring fusion and also it is one of the indispensable techniques because it can be combine with posterior procedures in which large decompression is performed. Lumbar interbody fusion (LIF) that has developed rapidly since Cloward [1950) can be performed posteriorly and anteriorly. Posterior LIF is classified according to the access corridor: Paramedian access (PLIF), transforaminal access (TLIF) [23]. Both techniques can be performed percutaneously, minimally invasively or open [4,22,24-33]. Implant is a cancellous-like titanium cylinder like Bagly and Kuslich design (BAK) named cage [4]. Transforaminal access is an ideal treatment option in cases with grade 1-2 spondylolisthesis and without neurological deficit [25]. It provides disc height in the foraminal region and decreases foraminal stenosis.

Anterior LIF can be performed through open surgery with transperitoneal or paramedian retroperitoneal access as well as laparoscopically [26-29]. ALIF under scopy is more advangegous than PLIF as it does not cause dural damage. PLIF allows combination of decompression and fusion while dural and neural damage risk is higher. ALIF restores disc height like posterior procedures and achieves decompression in the foraminal region following discectomy but its inability to eliminate facet hyperthrophy and osteophyte organizations is considered as a disadvantage [20,27-29,32].

Conclusion

87% of back pain does not relieve despite treatment. Every treatment that is initiated without describing the stage of the pathology may increase symptoms. Therefore, physiopathological staging should be done carefully. Decompressive procedures performed on a stable spine, which may lead to instability, may aggravate symptoms. If fusion surgery is not performed properly, patient satisfaction may decrease. Minimally invasive interventions have clear boundaries. In cases requiring large decompression, conservative surgical methods should not be avoided [30,33-36].

After making sure that degenerative spine causes pain and dysfunction, degree of lumbar degeneration and disc pathology should be determined. Degeneration is a progressive process but can be slowed down. A healthy disc space restored conservatively or surgically will delay stenotic spinal disease. Clear benefits and harms of stenosis surgery have been well established. Today, the approach should be to discuss conservative, genetic and surgical, especially minimally invasive techniques that preserve disc space before degeneration starts [37,38].

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