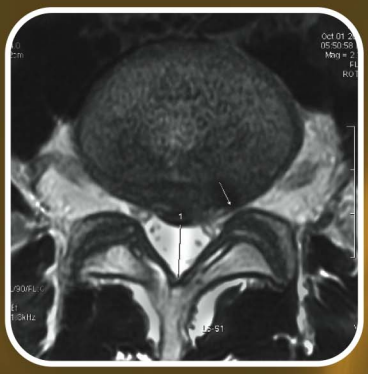
Degenerative Lumbar Spine Disorder & Its Conservative Treatment





Mahesh Pandya

With Interactive DVD-ROM

JAYPEE

CONTENTS

1.	Introduction	1
2.	Relevant Anatomy, Physiology and Biomechanics	28
3.	Chain of Events in Degenerative Lumbar Spine Disorder	46
4.	Clinical Examination	58
5.	Diagnosis	85
6.	Conservative Treatment	123
7.	Recent Advances to Support Triangular Lumbar Spine Distraction	151
	Bibliography	*167*
	Index	*169*

3 CHAPTER

Chain of Events in Degenerative Lumbar Spine Disorder

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It is universally accepted that basic changes responsible for pain in lower back occur in intervertebral disc. Nucleus pulposus gets its way out through tears in annulus. It may be sudden or gradual.

In degenerative lumbar spine disorder, gradually due to accumulated effect of mechanical load over a period of time, there is loss of water content or dehydration of the disc. This is the ultimate event. There are many changes occurring before this final event. Thus, disc degeneration is overall effect of changes in anatomy and physiology of lumbar spine and pelvis.

It is thought by many that all spines degenerate; that is true but at the same time it is also true that all degenerated discs do not give rise to pain. The pain is either due to increased intradiscal pressure and/or compression or inflammation around a nerve root or it is due to stretching of a ligament or a muscle due to change in normal anatomy.

If one looks at literature, there are many views regarding the initial event responsible. It is difficult to say whether the first event is degeneration of the disc due to reasons beyond control or subtle anatomical changes that are responsible for the disc degeneration. Looking at the high incidence rate of the problem, I believe that though there could be familial predisposition or tendency to develop the problem, it can never be entirely genetic, because no disc is immune to degeneration.

As we saw in anatomy and biomechanics, there remains balance of muscles acting on pelvis and lumbar spine like extensors of spine and abdominals, hamstrings, iliopsoas, gluteus maximus and rectus femoris, this balance decides the lumbar lordotic curve, sacral angle and lumbosacral angle (Fig. 3.1).

Pelvic tilt angle increases with weak abdominals. This leads to counternutation (anterior rotation of pelvis on femoral heads) with tightening of hamstrings and iliopsoas (Fig. 3.2).

It is not always possible to pinpoint the beginning of the problem of disc degeneration. Probably, it is inability of disc to take up the load either due to stiffness or some structural variations like sacralization leading to hypomobility (anatomical reasons). This may be due to poor biomechanics like work environment, postural reasons or increased weakness of muscles. This could be due to changes in molecular biology of disc triggered by unknown factor (presently thought to be genetic).

Physiologically, there are some biochemical changes occurring in disc cells. The loss of balance between proteoglycan catabolism and its synthesis is the main event leading to disc degeneration. This happens due to improper nutrition of the disc. Disc being an avascular structure largely depends on diffusion through the end plate for its nutrition. The combined changes in anatomy as well as physiology of intervertebral disc lead to degeneration.

The main work of the disc is to absorb the shock coming on lumbar spine during various activities. The pressure within disc is usually not allowed to increase by strong muscle activities. If there is an excessive mechanical loading of a disc as it happens in sports injury, appropriate proprioception of muscles should be able to prevent disc injury. Similar prevention is possible in degenerative disc disorder also.

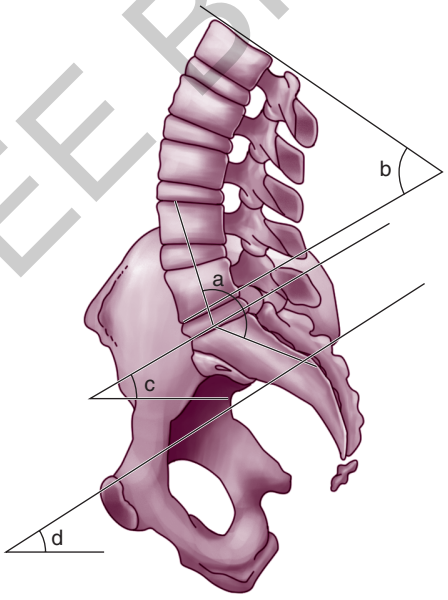

Fig. 3.1 Various angles:
a: Lumbosacral angle
b: Lumbar lordosis angle
c: Sacral inclination
d: Pelvic angle

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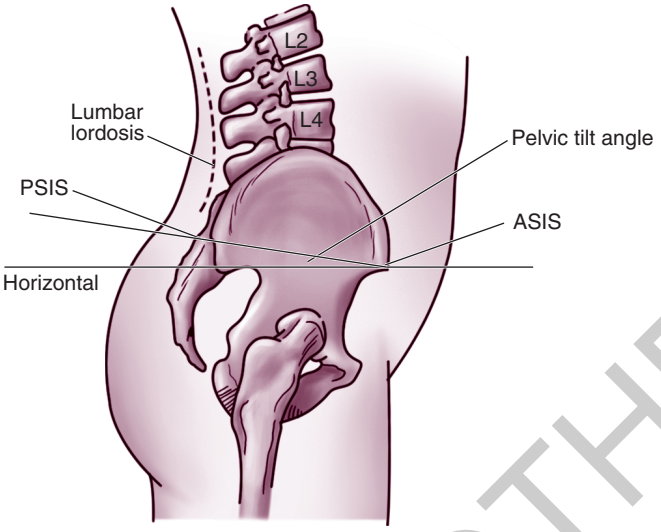

Fig. 3.2 Pelvic tilt angle measurement

The anatomical changes occurring in degenerative disc pathology can be described in two categories. The first one includes changes occurring in muscles working on pelvis, lumbosacral spine and hip joints and the changes in various anatomical angles associated with this muscle imbalance. The second category includes anatomical changes occurring in a mobile segmental unit of lumbar spine.

The important muscles responsible for mobility and stability of lumbar spine are primary flexors like abdominals and secondary flexors like quadratus lumborum and iliopsoas, primary extensors like erector spinae as well as secondary extensors like gluteus maximus, gluteus medius and hamstring muscles and rectus femoris (Fig. 3.3)

Abdominals are probably the most important of all. These muscles are rectus abdominis, external and internal obliques and transverse abdominis. They are powerful stabilizers in front and maintain curvature of lumbar spine. They prevent increased lumbar lordosis. The laxity or weakness of these muscles leads to increased lordosis and increased extension of lumbar spine.

To compensate the increased extension at lumbar spine, the hip flexors contract to limit uninhibited extension of spine. The rectus femoris also helps in controlling the increased extension of spine. But this leads to anterior rotation of inominate bone. To counteract this anterior rotation of inominate bone, hamstrings contract from behind. Associated weakness or stretching of gluteus maximus due to overactive iliopsoas further increases hamstring tightness (Fig. 3.4).

These changes lead to abnormal transmission of load on lumbar spine. This causes lumbar disc degeneration. There is shifting of load posteriorly more on facet joints. This brings in facet joint synovitis.

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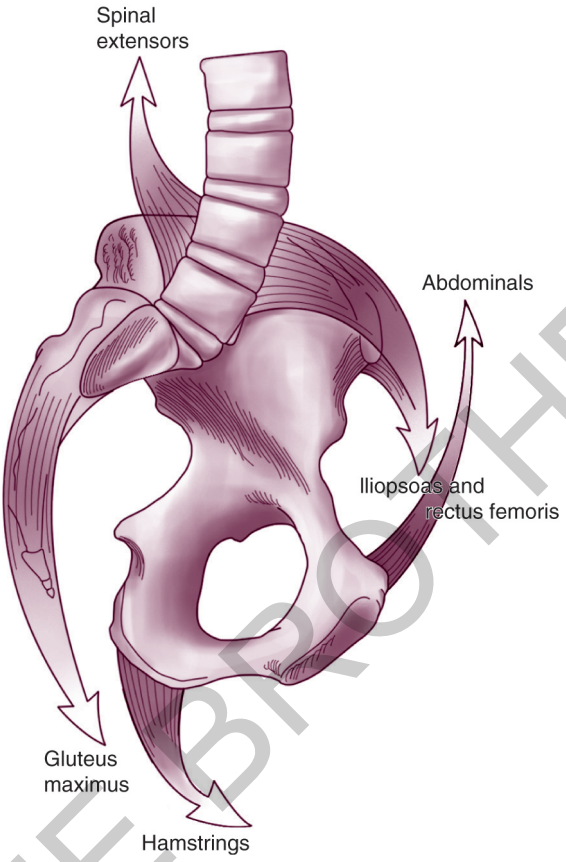

Fig. 3.3 Important muscles acting on lower back

There is permanent adaptive shortening of erector spinae, iliopsoas and hamstring muscles. There is abnormal laxity of abdominals and glutei.

Although the initial event may be just laxity of abdominals, but over a period of time it is difficult to correct permanent adaptive shortening of muscles and subsequent change in important angles in lumbosacral spine simply by strengthening of abdominals.

One has to aim at correcting muscle tightness for successful conservative treatment. There are many more changes occurring at mobile segment of lumbar spine in disc degeneration. If they are not understood and corrected, then also the conservative treatment does not prove to be successful.

The changes occurring at mobile segments of lumbar spine occur in a sequence. There is reduction in incompressibility of disc either due to muscle imbalance or from some kind of insult affecting the nutrition of the disc material. So, there is some loss of disc height. This leads to shifting of load posteriorly on facet joints in early stage leading to increased lumbar

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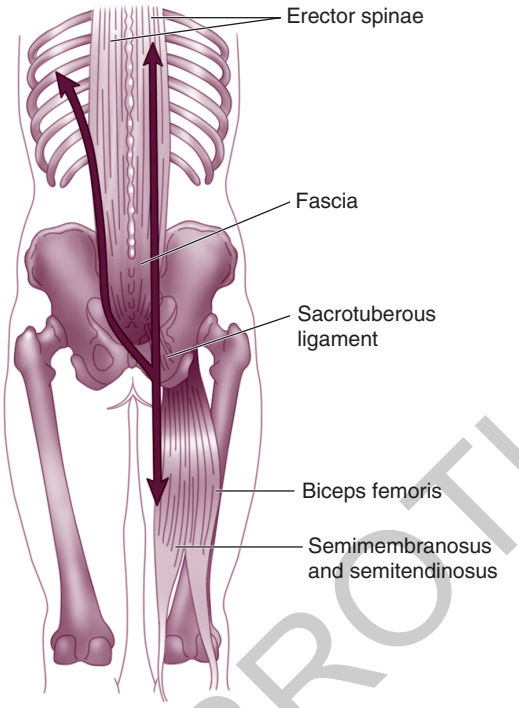

Fig. 3.4 Tightness of extensors of spine brings in hamstrings tightness

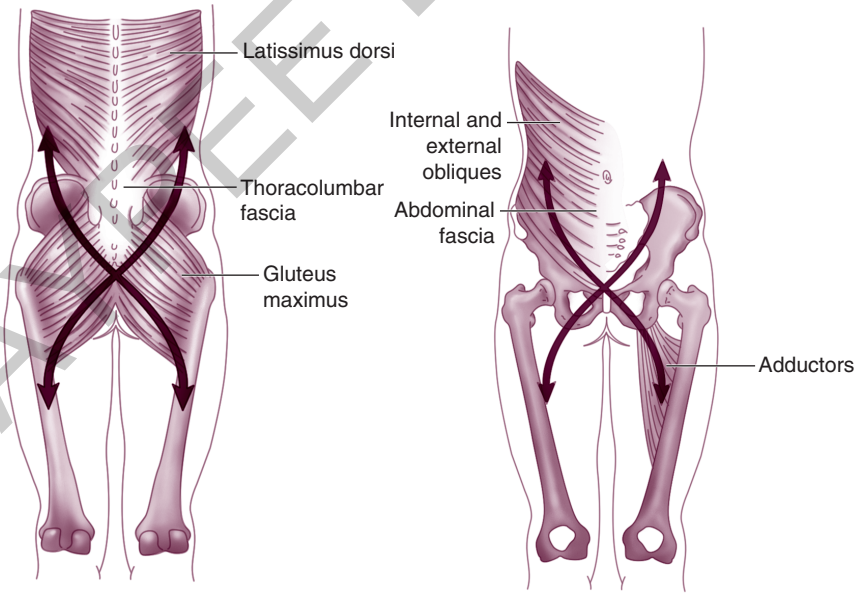

Fig. 3.5 Weakness of gluteus maximus leads to increased activity of secondary extensors of lumbar spine. Action of abdominals on pelvis is balanced by adductors of opposite side

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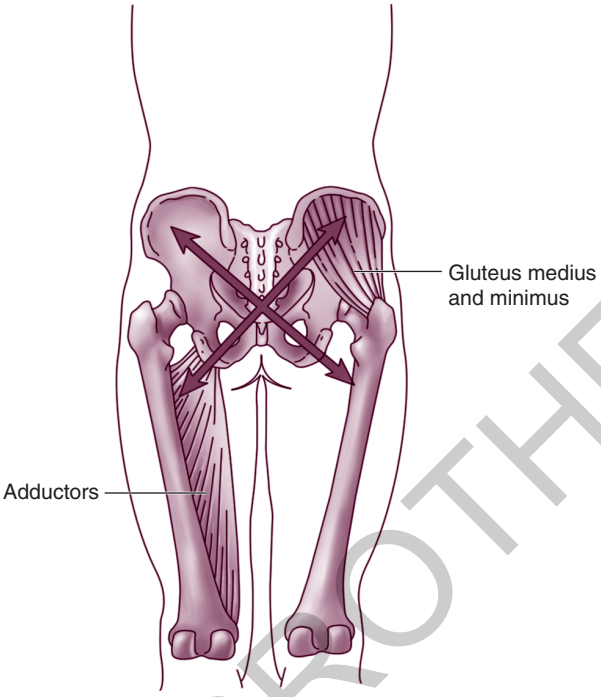

Fig. 3.6 Overactivity of hip adductor is counteracted by opposite side gluteus medius and minimus

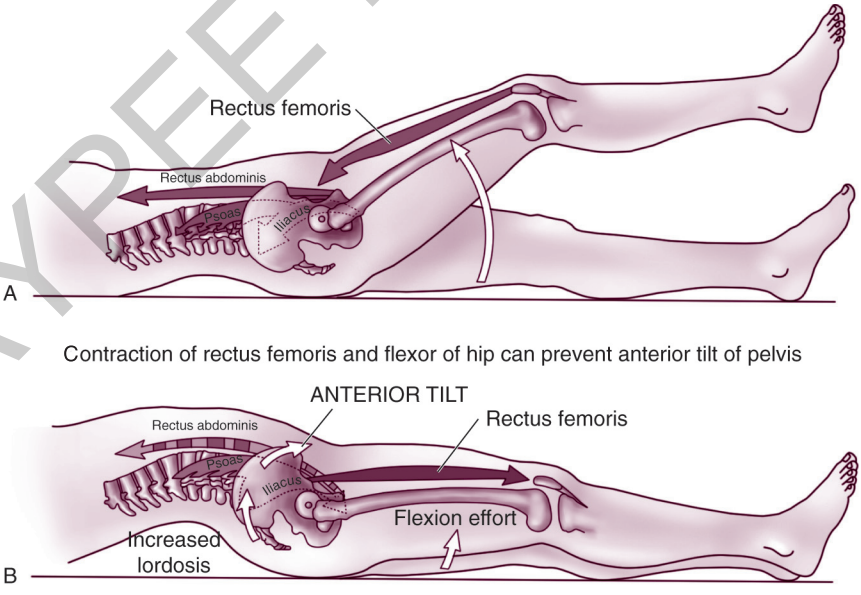

Fig. 3.7 Relaxation of rectus femoris and hip flexor can increase anterior abdominal tilt in presence of weak abdominals

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lordosis. There is synovitis of facet joints. This is known as stage of dysfunction. On further progression, there is buckling of yellow ligament—ligamentum flavum—into the canal. There is true reduction in disc space. This is the stage of instability (Figs 3.5 to 3.7).

At last there is an attempt at creating stability. There is spur formation at the attachments of all ligaments, mainly anteriorly. There is reduction in height of foraminal spaces and spinal canal. There is overall crumpling of the space in lumbar vertebral column. There is formation of hard disc from the material extruded from the disc space. This is a vicious cycle. All the events occurring in this cycle actually increase the next event and that further increases the original triggering event. Once an event is triggered and not controlled, the end result is foraminal stenosis and lumbar canal stenosis. Figures 3.8 to 3.10 explain this chain of events.

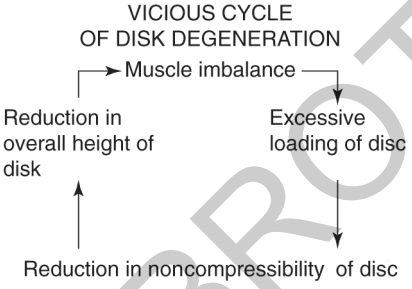

Fig. 3.8 Vicious cycle of disc degeneration [The initial event may start from any level out of four events mentioned in vicious cycle diagram. Then it continues to progress relentlessly. Lumbar spine distraction carried out on continuous basis can break this effectively in large number of patients (See the text for details.)]

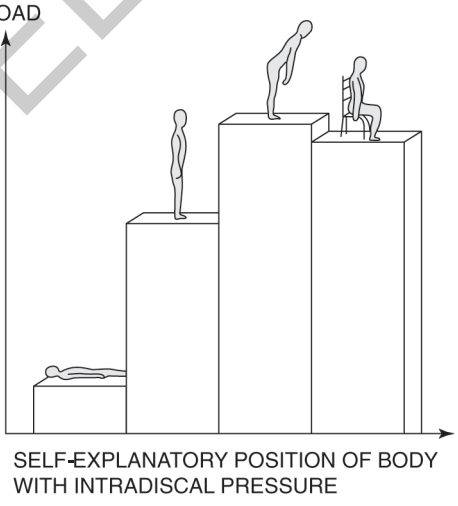

Fig. 3.9 In lying down position, there is minimum load on intervertebral disc. Bending forward has highest load on disc. Standing is more advantageous than sitting for intervertebral disc load. In standing, the load is shared with two lower limbs

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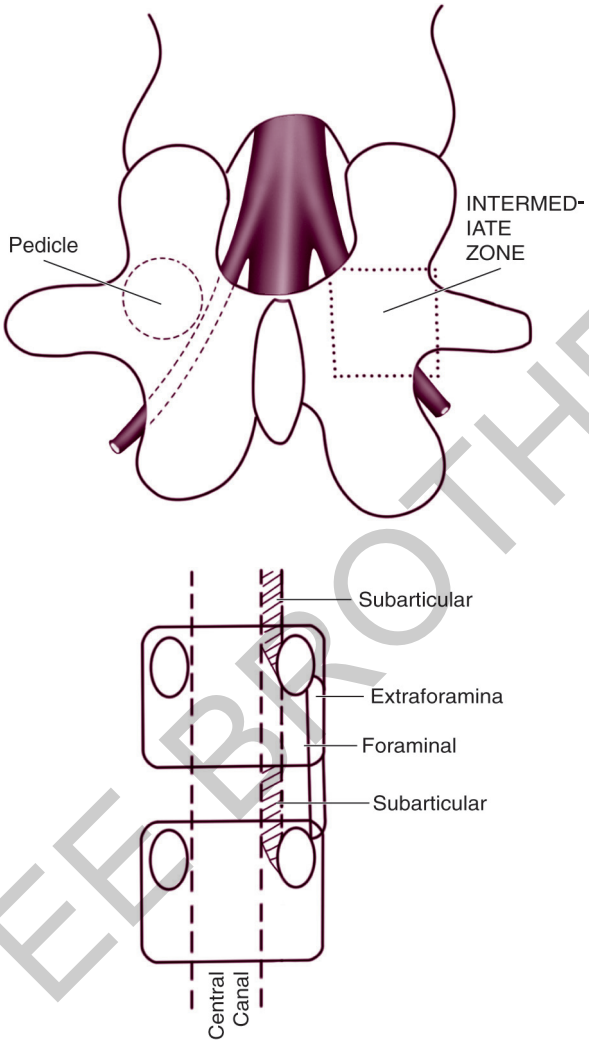

Fig. 3.10 Various types of stenosis

BIOCHEMICAL OR PHYSIOLOGICAL CHAIN OF EVENTS

The young healthy disc is made up of 85-90 percent water. As the disc ages, it loses its macromolecules particularly aggrecan. This happens due to imbalance between proteoglycan catabolism and synthesis. This leads to water loss from the disc.

The capillary beds within subchondral bone in vertebral body become less dense. There is sclerosis of subchondral bone. This leads to reduced permeability of the subchondral plate. Ultimately, delivery of nutrients to

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the disc reduces. Thus there is loss of disc height, increased pressure on surrounding structures, cracking and fissuring of annulus and then disc herniation.

The reduced ability of disc to retain water leads to reduced blood supply. This further leads to reduced oxygen tension, lower pH balance and accumulation of waste products inside the disc material. This ultimately reduces the healing in disc after any insult or injury.

There are enzymes responsible for such imbalance of catabolism and anabolism. These enzymes like aggrecanases and matrix metalloproteinase family of enzymes degrade collagen, proteoglycan and other structural components of intervertebral disc.

There is upregulation of nitric oxide, interleukin 6 and prostaglandin E2 in degenerated disc specimen. This upregulation is responsible for loss of disc and development of back pain.

Progressive cell loss with loss of notochordal cells from nucleus pulposus by the beginning of second decade coincides with the early stage of disc degeneration. These cells are very important in early repair and maintenance of extracellular matrix.

Under pure compressive and eccentric compressive loading, there is uniform distribution across the entire end plate in a normal healthy disc.

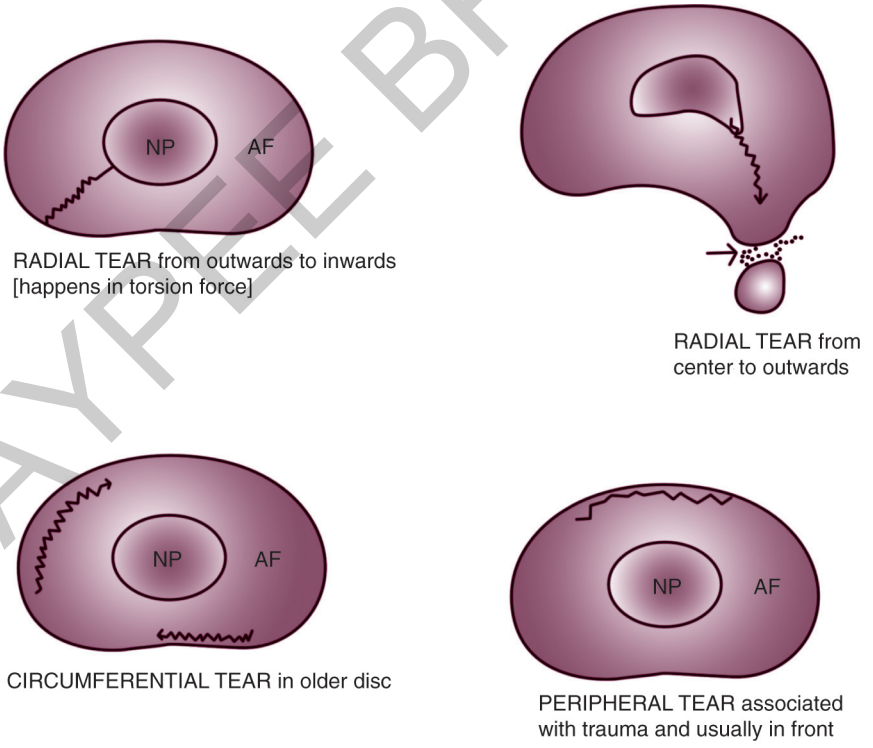

Fig. 3.11 Types of annular tear

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In degenerated disc, this uniform distribution is not found at least for eccentric compressive load.

There are three types of annual tears found in disc. The most common radial tear is located in the posterior annulus fibrosus. The circumferential tears are due to interlaminar shear stresses and are more common in older discs. Peripheral rim tears are located anteriorly and are usually associated with trauma (Fig. 3.11).

The movements most affected due to disc degeneration are axial rotation and flexion because intervertebral disc provides the most resistance to axial compression. The facet joints allow for rotation, lateral bending and extension.

The facet joint complex is very important in stabilizing segmental unit. Due to increased disc degeneration, there is increased stress applied posteriorly. This leads to facet joint arthrosis. This ultimately leads to change in the mechanical forces coming on intervertebral disc.

Spine degeneration is the end result of interplay between subtle alterations in mechanical and biochemical properties of the intervertebral disc and facet joint complex.

The degeneration can start from disc and subsequently may affect facet joints. It may start in facet joints as it happens in spondylosis. Subsequent stress applied on the facet joint complex leads to degeneration of the intervertebral disc.

The disc degeneration happens in all spines. Let us see what factors increase or speed up the events in the disc degeneration. First and foremost is age. Second is family history. Third is nicotine consumption like smoking. Fourth is job pattern requiring heavy repetitive loading of the spine and jerking or prolonged vibration as it happens in prolonged driving.

Factors Increasing the Events in Disc Degeneration

Age

Young disc is capable of converting compressive loads coming on the outer annulus fibrosus into tensile hoop stress while allowing controlled motion of intervertebral segment. The vertebral endplate transmits the compressive load to the nucleus pulposus leading to elevated pressure on the hydrated gel of the nucleus pulposus.

Young and healthy disc transmits all the forces coming on the disc to all surrounding tissues of the disc evenly and in outward directions. The circumferential fibers of the outer annulus fibrosus also dissipate some of the stress.

With increased aging, the changes begin in nucleus pulposus. There is loss of water content and large proteoglycans. In the annulus collagen, fibers deteriorate and fissures develop into the inner region which ultimately extends outwards. There is subsequent overloading of the bony endplates, which become sclerotic and form peripheral osteophytes.

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Family history

This factor came into consideration because of studies mentioning involvement of multiple members of a family having symptomatic disc degeneration. Study of twins suggests a strong link between heredity and disc degeneration. Genes found responsible are some alleles of the vitamin D receptor and matrix metalloproteinase-3. Mutation in the type IX collagen alpha-2 gene is also considered responsible for disc degeneration.

Looking at the high prevalence of the problem, how much this factor contributes to the pathology remains unclear. I believe it is a contributory factor to disc degeneration and not sole responsible factor for disc degeneration.

Nicotine consumption

It is not clear how this works. Nicotine consumption probably leads to disc degeneration by reducing blood supply to the end plate. This could compromise the nutrition of the disc. Further smoking leads to osteoporosis. Moreover, smokers get dry cough that leads to microfractures in end plates. It also reduces the O_2 carrying capacity of hemoglobin. It leads to poor oxygenation of the disc.

Job pattern

At present, this is the most proven cause. As discussed earlier, high and eccentric compressive forces lead to degeneration very fast.

Activities with frequent jerks or vibrations can trigger the disc degeneration. As it happens in prolonged driving, continuous repetitive compressive small amount of forces would lead to tear in annulus.

Diabetes

Like anywhere in body, diabetes mellitus brings in degeneration much faster.

SUMMARY

As discussed earlier, there is difference between symptomatic disc degeneration and degenerative disc disease. What makes a degenerated disc painful is ingrowth of blood vessels and free nerve endings in full thickness annual fissures. In normal disc, the nerve endings are found only upto 1 or 2 mm of periphery of annular surface. In degenerated disc, free nerve endings with receptor for nociceptive substances like substance P and calcitonin gene-related peptide are found in annular disruptions.

In the final stage of lumbar disc degeneration lumbar spinal stenosis, compression leads to neural ischemia. Over and above that, the spinal root's

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nutritional support is half dependant on cerebrospinal fluid diffusion. Mechanical compression leads to decreased nutrition delivery, reduced neural activity and ischemic neuritis. All these changes are responsible for generation of symptoms in lumbar canal stenosis.

The cascade of degeneration of lumbar spine involves interplay of both biomechanical and biologic factors. No single factor can be held responsible for disc degeneration.

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Degenerative Lumbar Spine Disorder & Its Conservative Treatment



Mahesh Pandya is a Consultant Orthopedic Surgeon at Baba Advanced Orthopedic Hospital, Vadodara, Gujarat, India. He did his Masters in Orthopedics in 1989. He is attached to many hospitals as a consultant in city of Vadodara, Gujarat. His area of interest is degenerative disorders in orthopedics. He has done a lot of work in reconstructive surgery of various degenerative disorders. He was awarded a Fellowship in reconstructive surgery of knee and hip joints at Queens Medical Centre, University of Nottingham, UK in 2004 under the auspices of Nirman Foundation, USA.

He has made many innovations in operative treatment of degenerative disorders. He developed a technique of posterior midline approach for decompression and simultaneous posterolateral fusion of lumbar spine. After looking at wound-related problems in open surgery of repair of degenerative rupture of tendo-calcaneus [TC], he did a level 4 study on percutaneous repair of tendo-calcaneus. This study was presented as poster and it bagged the first prize at a state level conference. For relatively young patients of medial compartment osteoarthritis of knee joint, he studied the effectiveness of High Tibial Osteotomy with ring fixator in comparison to conventional osteotomy. The basic idea of using ring fixator was to preserve bone stock and patellar tracking for future total knee arthroplasty.

He has commendable above average percentile achievements in examination in orthopedic surgery conducted by American Academy of Orthopedic Surgery (AAOS). The same examination is taken up by orthopedic consultants in the USA for Practice Ready Assessment (PRA) credit for continuing licensure. He is attached to AAOS for the last three years. He has also successfully participated in an evaluation in specialty of orthopedic surgery by Centre for the Evaluation of Health Professionals Educated Abroad (CEHPEA), Toronto, Canada. He is a life member in Indian Orthopedic Association and Gujarat Orthopedic Association.

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