HOW FAR HAS CLINICAL TREATMENT GONE IN DEGENERATIVE DISC DISEASE?

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Treatment plans for patients suffering from low back pain should be based on basic sciences. The proper assessment of these patients requires knowledge of functional anatomy, biomechanics, and degenerative process to correctly interpret the imaging and electrophysiological studies for understanding the nonorganic factors that may cause the pain.

The basic functional unit of the spine is the motion segment. Each motion segment in the spine (with the exception of very specific anatomical arrangement from the occiput to C2) is called a *threejoint complex*. The three-joint complex consists of three joints: the discovertebral joint in the front and a pair of facet (*zygapophyseal*) joints at the back.

Motion segments surround the neural structures with which they are associated and consist of the superior and inferior vertebral bodies; the interspinous, intertransverse, costovertebral, and longitudinal ligaments, including the intervertebral disc and facet joints; and such interosseous fibrous connections as the ligamentum flavum⁽¹⁾.

The existence of a congenital or acquired pathology in the main structure of any of the 23 or 24 motion segments along the spine primarily affects the other structures in the same motion segment, followed by the motion segments at the other levels of the spine, particularly the neighboring segments⁽²⁾.

The motion of the entire spine is actually the sum of its segmental motions. The motion observed along the entire spine does not completely explain the complex spinal mechanism. Even when there are underlying segmental motion abnormalities, the range of motion observed in the spine as a whole can be normal. The main segmental motions are rotations in the sagittal, axial, and coronal planes, which are observed as flexion, extension, torsion, and lateral flexion. Segmental motion is complex, and simultaneous motions occur along more than one axis; this is called *coupled motion* ⁽³⁾.

The motion observed in the lumbar spine, however, consists of each motion segment up to the thoracolumbar junction, the lumbosacral area, and lumbopelvic rhythm coming from the hip joints. Therefore, the treatment plan, especially exercises, should involve the entire kinetic chain rather than focusing on a single area.

The intervertebral discs, facet joints, ligaments, and muscles contribute in different proportions to segmental stability. It is thought that the muscles provide the most important support to protect the three-joint complex from excessive shear forces⁽⁴⁾. An understanding of the abdominal and lumbar muscles that functionally provide dynamic stability is necessary for spine rehabilitation.

1. Stages of Spine Degeneration

Spine degeneration is a genetically determined process in which mechanically triggered biological factors play a role. In this process, natural aging is considered the only important contributor. Degenerative changes starting in any of the motion segments can initiate degenerative changes in the neighboring segments as well.

The same patient can exhibit different phases of the degenerative process in different motion segments. Spine degeneration consists of three phases: *functional impairment, instability,* and *stabilization*.

Kirkaldy-Willis⁽⁵⁾ defined the degenerative process as occurring separately in two main areas of the three-joint complex: the intervertebral disc and posterior joints. The author stated that each structure in the motion segment affects the another in this process. According to Kirkaldy-Willis's argument, the degenerative process, which proceeds as synovitis, hypomobility, degeneration, capsular laxity, subluxation, and widening in the articular process in facet joints, reveals itself as circumferential laceration, radial laceration, internal disc disruption, disc resorption, and osteophytes in the intervertebral disc. As a result, the interaction among facet joints and the degeneration in the intervertebral disc causes dysfunction, segmental instability, and various types of spinal stenosis.

2. Phases of the Degenerative Process: The Effects of Changes Morphological- and Cellular-Level Changes on Clinical Findings

Degenerative changes at the morphological and cellular levels due to aging are universal; however, the point in this process at which pain begins is unknown.

The first phase of the degenerative process is segmental functional impairment. In this phase, the facet joint functions are the first to be disrupted. Facet joint pain, inflammation, and hypomobility begin as a result of reactive synovitis and articular cartilage degeneration, and the movement of the motion segment is restricted. The short segmental extension and contraction of rotator muscles further limit the joint movements. During this phase, the clinical findings for patients with acute low back pain due to facet joint degeneration include pain that worsens with standing up, walking, extension, and rotation in extension. Generally, the patient is comfortable in flexion. Sometimes, irritation due to distension in the facet joint capsule and pain when stooping due to muscle spasm can also arise. Local sensitivity, muscle spasms, and limited joint range of motion are observed, but no neurological deficit is found. As nonradicular low back pain may be due to functional impairment of the facet joint, the pain can occur in the hip and proximal to the knee, but it does not come down below the knee.

In intervertebral disc, however, degeneration and annular lacerations occur during the first phase. Facet joint dysfunction also provides the basis for the formation of annular lacerations by causing loads to be transferred to the intervertebral disc. Annular lacerations cause disc protrusion and disc herniation in the second phase; however, annular lacerations can also cause acute pain without protrusion or classical herniation because of the many nerve endings in the external annular tendons.

In the patient with discogenic low back pain related to acute annular laceration, the pain intensifies considerably during such activities as flexion, coughing, and straining. Very severe atypical discogenic pain can be observed in cases of central disc herniation because of the many free nerve endings in the posterior longitudinal ligament. With the existence of extruded and sequestered fragments, radicular pain can occur without low back pain. A loss of strength can also occur without low back or leg pain.

In cases of far lateral disc herniation, the nerve root is compressed as it exits the foramen enters the extraforaminal area. This causes lower extremity pain (as opposed to lower back pain), sensory impairment, and a loss of strength in the muscles for which the third, fourth, and fifth lumbar (L3-L4-L5) vertebrae nerve roots are responsible. Because the disc material is close to the dorsal root ganglion, the pain can be very severe. The foraminal stenosis that develops because of the disc material can cause neurogenic claudication pain that decreases during sitting and increases while walking and standing up. Although very serious pain occurs with far lateral disc herniation, normal results can be obtained during a straight leg raising test.

In spine degeneration, segmental functional impairment is followed by the instability phase. This phase is characterized by excessive movement at the segmental level and segmental instability. Capsular laxity and joint hypermobility in the facet joints occurs. In this phase, instability may not be measured by radiography, and t standard flexion-extension results may be normal ⁽⁶⁾.

Diagnosing patients in the instability phase with a clinical examination is more difficult. Patients with no previous discogenic pain or symptoms may consult a doctor because of their instability. Clinical diagnosis is considerably important at this point. It is more important to know the quality of the patient's

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low back range of motion than to know how extensive the range of motion is. The most important determinant of segmental hypermobility in the spine is a good physical examination.

The feeling of getting stuck during spine movements, a painful arc, irregular rhythm during motion, the development of functional scoliosis, sensitivity, and spasm can result from instability. Instability can occur without any symptoms. The flexion-extension measurements can be abnormal in asymptomatic people⁽⁷⁾. In the light of this information, pain that appears in the instability phase seems to result from inflammation-triggered disc and facet joint abnormalities.

Disc-related disorders in the instability phase include internal disc disruption and a narrowing of the intervertebral disc space. As disc degeneration progresses, increased multiple annular lacerations cause internal disc disruption, and the disc becomes a pain generator.

The cause of the pain may be annular nociceptive tendons or biochemical factors. In this phase, the disc cannot resist torsional stresses and the symptoms increase.

A reduction in disc height triggers facet joint laxity, which leads to the narrowing of the intervertebral foramen and lateral recess. Radiculopathy pain may develop due to both compression from the disc material and the biochemical factors revealed by internal disc disruption.

The instability phase is followed by the segmental stabilization phase. In the stabilization phase, increases in fibrous tissue, expansion, and arthrosis of the facet joints appear; in intervertebral disc, degeneration increases, desiccation emerges, vertebral end plates become closer to each other, and osteophytes are formed. These changes in the facet joints and the disc limit the motion segment considerably and lead to spondylosis.

Although the pain is not very apparent, the limitation of movement is. Spinal nerve root compressions can appear due to lateral stenosis, central canal stenosis, and degenerative spondylolisthesis.

Acquired degenerative changes, especially at the floor of a congenitally narrow canal, can reveal themselves as bilateral and multilevel radiculopathy. Central and lateral canal stenosis can be observed at the same and different levels. The most striking clinical finding in patients in the segmental stabilization phase is neurogenic claudication (*pseudoclaudication*). While standing up and walking increase the symptoms, stooping, and other flexion movements typically decrease the pain. During this phase, a straight leg raising test generally yield normal results. Although disc herniation is not often observed during this phase, paresthesia and pain due to spinal stenosis are more common⁽⁸⁾.

3. Spine Degeneration and Pain

Low back pain can arise from different anatomical structures in the spine; the exact source of the pain is generally not known. Immunohistochemical studies have increased our understanding of the sensory innervation of the spine, revealing the innervations of ligaments, myofascial, and neurovascular structures, particularly intervertebral discs and facet joints that form the three-joint complex (9). It is thought that inflammation contributes to pain development, as do rich sensory innervation, mechanical compression, biochemical agents, and central sensitization⁽¹⁰⁾. Radicular symptoms can also occur without having a significant impact on the nerve root, and a good response to anti-inflammatory treatment can occur when the nerve root is involved. The relationship between degenerative disc disease and low back pain is not yet understood completely. Pain may not always parallel the morphological changes in the intervertebral disc⁽¹¹⁾.

Experimental studies show that the intervertebral disc is an important pain generator⁽¹²⁾.

4. Discogenic Pain

The two main reasons for primary discogenic pain are internal disc disruption and degenerative disc disease. Disc degeneration contributes to the pathogenesis of secondary diseases, such as disc herniation, spinal stenosis, and degenerative spondylolisthesis.

The concept of internal disc disruption was characterized by Henry Crock ⁽¹³⁾ as a painful increase in the biological activity of the intervertebral disc and defined as a clinical condition in which radiographs, computational tomography (CT), and myelographs are normal, but abnormal discography findings are obtained.

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Recently, however, it has been defined as a clinical condition in which the height and shape of the disc is preserved on magnetic resonance (MR) imaging, but which is characterized by the darkening of the disc. However, there is a problematic situation here; the patient may not experience pain even when the disc appears black on the MR imaging; thus, MR alone cannot make an accurate diagnosis.

It is thought that pain arising from internal disc disruption occurs due to the mechanical and chemical stimulation of the nociceptors in the external annulus tendons and ligaments surrounding the disc. The radiological and clinical findings for internal disc disruption differ from the findings for degenerative disc disease.

Disc herniation and segmental instability do not exist in internal disc disruption. No radiologic disorder other than darkening in the disc is observed. Nerve root irritation findings, radicular pain, and neurological deficits are not observed in the clinical examination of the internal disc disruption⁽ⁱ¹⁾. While direct images are radiologically normal, early degeneration findings can be observed with MR. Another important diagnosis method involves confirming the tear in the annulus with discography and finding positive responses to pain provocation. CT completed after discography clearly displays morphological distortion.

Patients between the ages of 20 and 50 can suffer from repetitive low back pain attacks. These patients' pain generally centers around the low back area, and they do not have radiculopathy complaints.

However, referred pain spreading to the hips can occur, and in rare cases, the pain can spread distal to the knee. Patient reports may reveal a history of lifting heavy objects with a sudden movement, picking up a very light object or simply coughing just before the annulus laceration. Coughing and straining can increase the pain, as can small movements, rotation, and leaning to the front, sides, or back.

The pain partially decreases with resting. Sitting is not possible because the pain increases. Pulling the knees toward the belly while lying on one side can decrease the pain. Leg pain, if it exists, is a late finding and does not have a dermatomal distribution. No neurological deficit is observed, and findings other than limitations in low back movements seem normal upon physical examination. In the straight leg raising test, low back pain can be observed, but the pain does not spread to the legs. In degenerative disc disease, however, diagnosis is based on radiology, CT, and MR findings. Disc degeneration is usually observed at more than one level, and the natural aging process is the typical cause of this disc degeneration. Discography can be used to understand the level of the disc causing the pain.

Permanent low back pain occurs with lumbar degenerative disc disease. The pain is distributed to the sacroiliac joint area, the hips, and the backs of the legs. The symptoms increase during sitting, and long periods of walking can increase the pain. During the progression period, degenerative disc disease can cause radicular and claudication-type pain by giving rise to herniation and spinal stenosis. Sensitivity in the lumbar area and increasing pain, particularly while straightening up from the flexion position and with lumbar flexion, are observed. The pain may decrease more in the extension position. If there is no accompanying disc herniation and foraminal stenosis, radicular pain spreading to below the knees is not observed.

The natural course of degenerative disc disease is not yet completely understood. In their study, Smith et al. ⁽¹⁴⁾ observed 25 patients with a positive discography for an average of 4.9 years and found that 68% of the patients recovered without any surgical intervention. Despite the limited number of cases studied, this result shows that two out of three patients with positive discography can recover with conservative treatment. Because discography is only administered to patients with very serious complaints, these results can be interpreted as showing that the course of the degenerative disc disease is good in many patients with low back pain who have fewer complaints and who do not undergo discography.

5. Clinical Treatment

Disease history plays an important role in treatment planning for patients with spine degeneration. Treatment plans should not be based solely on radiological findings and the patient's current clinical presentation. The spine undergoes degenerative changes in people as they age, and the MR findings of 30 to 60% of people who do not have any back pain complaints also reveal spinal abnormalities ⁽¹⁵⁻¹⁶⁾.

Many patients respond very well to conservative treatment. The main objectives of conservative

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treatment in the acute phase are patient education, which includes thoroughly teaching the patients how to protect the low back, modifying activity to protect the low back, using non-narcotic analgesics and nonsteroidal anti-inflammatory drugs, returning to normal activities as soon as possible, and educating the patient about exercise.

In the subacute phase, the objectives include ensuring painless movements in the low back, hip, shoulder junction, and the entire kinetic chain affecting the spine through the two upper and lower extremities; improving the strength, resistance, and coordination of the neuromuscular system affecting the spine; and maintaining normal activity to keep low back pain from becoming repetitive and chronic. The most important point is to review surgical options when repetitive low back pain intensifies and progresses towards becoming chronic.

The formation and perception of pain that cannot be evaluated objectively and that has both physiological and psychological aspects varies from person to person. Acute and chronic spinal pain differ from one another, especially in their treatment approach. The behavior of the chronic pain patient may be much more important than the physical problem. Psychological stress initiated by the physical problem leads to disease behavior and the deterioration of social relations⁽¹⁷⁾.

During conservative treatment, the early recognition of and intervention for chronic pain can decrease the rate of disability resulting from chronic disease. Chronic pain reveals itself as functional disorders; thus, combating chronic pain becomes more difficult. Both somatic symptoms and the dimensions of the chronic pain based on many factors should be targeted in the treatment. Both physical and psychosocial functioning must be addressed.

The factors that delay recovery and induce chronic pain can be related to work, psychological factors, or medical problems.

Work-related factors include work dissatisfaction, a heavy physical work load, low education levels, and time spent away from work. *Psychological factors* include depression, anxiety, a self-perception of poor health, a lack of strategies for fighting the disease, somatization, and a history of sexual or physical abuse. *Medical factors* include low back pain at the time of consultation, distribution of the symptoms, severe leg pain, comorbidities, and the frequency and interval of previous low back pain attacks. The most important medical signs that low back pain is becoming chronic are previous low back pain attacks and the existence of severe leg pain⁽¹⁸⁾.

In general, psychosocial factors are believed to be very important determinants in the chronic progression of low back pain⁽¹⁹⁾. Stress, depressive mood and somatization are related to low back pain becoming chronic; however, depressive symptoms in chronic spinal pain primarily occur as a result of the pain⁽²⁰⁾.

Because the number and frequency of previous attacks are important factors in the chronicity of low back pain, it is very important that the doctor following the patient notices the onset of chronicity at the right time, especially in the conservative treatment process. As low back pain becomes chronic, the patient consults to the doctor more frequently, describes how his/her increasing complaints influence his/her life and environment, describes his/ her desperation with more emotional words, and may behave reactively and extremely during physical examinations.

Waddell⁽²¹⁾ states that some test and clinical examinations may indicate that the patient's pain may not have an organic origin; however, the diagnosis and treatment should not be based on these results alone. Segmental instability findings in patients who consult the doctor frequently and express desperation should not be left unnoticed.

If segmental instability can be diagnosed in the light of the patient's history, physical examination, and imaging methods, surgical stabilization systems should primarily be considered. Surgery should be considered particularly for young patients who have frequent low back pain attacks, do not respond to conservative treatment, consult every 3 to 4 weeks, and cannot work for a week or more after an attack.

Internal disc disruptions for which no neurological deficit is observed over the course of degenerative disc disease may eventually lead to disc herniation and spinal stenosis. The precise indication for surgical intervention is increasing neurological deficit. If there is no medical contraindication for surgery, it should be performed regardless of the existence of neurological deficit. Relative indications, however, vary from doctor to doctor and patient to patient. While deciding whether to intervene surgically, patient complaints and clinical findings should

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be supported with radiological imaging. The patient's desire to return to work quickly, impatience with the conservative treatment, lack of expectations of secondary benefits and psychological treatment support the decision to intervene surgically.

Proper conservative treatment planning should be tailored to the patient. The patient's age, activity level, accompanying comorbidities, expectations, and profession are the determinants of the form and duration of conservative treatment.

For patients who consult the doctor due to very severe acute pain, cannot move, and are diagnosed with internal disc disruption and acute annular laceration with physical and radiological findings, the most important determinant of the treatment plan is the patient herself/himself. If the patient, complains of intolerable pain and she/he has to return to work within a short period (24 hours), has to travel, wants pain relief within hours, and has not responded to anti-inflammatory and analgesic treatment within several hours, she/he should be offered intradiscal electrothermal therapy (IDET), a minimally invasive method, as the first option and should be informed of all side effects.

One or two days of bed rest and movement within the limits of the pain should be recommended to patients who consult with the same complaints, respond to the analgesic and anti-inflammatory medication within several hours, and can move. The principles of low back protection should be described in detail. While anti-inflammatory medications and intramuscular steroid injections should be offered to the patients as medical treatment options, the patients should be informed about possible side effects. Severe, acute pain that begins in the floor of the acute annular laceration and responds to medical treatment disappears within 3 to 6 weeks⁽²²⁾.

Patients diagnosed with internal disc disruption and annular laceration should begin exercise programs as soon as their pain is controlled to strengthen and increase the resistance of the entire kinetic chain. The muscles of the entire trunk and extremities can be included with *core stabilization* and *dynamic stabilization* programs. Such programs first provide segmental stabilization by working the local stabilizer muscles of the spine, especially the multifidus and transversus abdominis; later, they work the kinetic chain as a whole. Aerobic capacity, flexibility and increased coordination are the other main objectives of well-planned exercise programs. For patients whose pain cannot be controlled, exercise programs should not be initiated, especially in the early period. The important point is to prescribe exercises that do not trigger pain in the patient. If a patient complains of increasing pain during or after exercise, the program should be reorganized. If a patient involved in a strengthening program consults the doctor with a severe discogenic pain attack, it could be the first indication that the low back pain is becoming chronic. Among patients who begin an exercise program that is reevaluated for modification every three weeks, important indicators of recovery include no recurrence of pain, a straight stance, and no problems during daily activities.

The fastest recovery occurs within the first weeks. Patients who are in the recovery process, apply the principles of low back protection as a life style, and implement the exercise programs should be followed conservatively.

One-third of the patients who visit a doctor for acute low back pain return with moderate pain after one year, and one-fifth of these recurrences arise from increasing activity limitations⁽²³⁾. Other treatments, such as pharmacological treatment, exercise, spinal manipulation, cognitive behavioral therapy, relaxation techniques, massage, and acupuncture should be described and recommended as effective treatments for patients who report moderate low back pain at their first visit (24). However, for patients who complain of increasing pain attacks and disabling chronic pain despite using these treatment methods, interventional treatment and other surgical options should be reviewed. When considering these options, it is most important to determine the specific anatomical structure that may be the source of the pain and to target the invasive treatment to the area. The use of such invasive tests as provocative discography to determine the source of the pain is problematic, and false positive results may be observed.

Other important causes of chronic pain are psychological and environmental factors. Interventional and surgical treatments alone cannot be effective for this aspect of the chronic pain. For this reason, a multidisciplinary approach is very important. While treatments directed to the specific source of the pain are planned, treatments that address the psychological

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and environmental factors affecting the patient's experience of pain should also be offered.

Low Back Pain: An Evidence-Based Clinical Practice Guideline, published by the American Pain Society (APS) in 2009, recommends an interdisciplinary rehabilitation approach, in the form of exercise treatment combined with cognitive behavior treatment, for patients with chronic low back pain and no radicular findings who do not respond to medical treatment and exercise ⁽²⁴⁻²⁶⁾. The disadvantages of this treatment, recommended two to three times per week for a total of 100 sessions, are its high cost, substantial time commitment, and lack of private health insurance coverage ⁽²⁷⁾.

Interdisciplinary treatment options are not recommended if radiculopathy and symptomatic spinal stenosis exist.

The guidelines also recommend that surgical options be reviewed for patients who have no radicular findings, complain of severe low back pain leading to disability, and have degenerative changes in the spine. The patient should be informed about other treatment options, especially interdisciplinary rehabilitation. Patients should be informed that surgery may not reduce the pain or improve functioning⁽²⁶⁾.

The patient should be involved in the decision to pursue surgical intervention⁽²⁸⁾. After the patient

is informed about all possible results and complications, especially when there are two or more treatment options, a common decision should be reached. The patient should actively participate in the treatment decision. Indeed, the patient's preferences and goals should serve as guidelines in reaching a decision⁽²⁹⁾.

While patients with disc herniation and lumbar radiculopathy findings and who want to recover as soon as possible may choose surgery, conservative treatment is recommended for patients with the same symptoms who do not want surgery until it is the last resort.

Many disc herniations can become smaller or disappear with time; however, this process can take months or sometimes years. The surgical success rate is lower for patients who experienced longterm pain before surgery compared with those who experienced short-term pain before surgery. For this reason, two points should be considered when planning treatment for low back pain related to degenerative disc herniation: first, conservative treatment should not be prolonged unnecessarily in a way that could affect the success of surgery; and second, surgery should be avoided in cases of a disc herniation that can disappear completely within several months or can become asymptomatic⁽³⁰⁾.

References

- 1- De Palma AF, Rothman RH: The intervertebral disc. Philedelphia WB Saunders, 1970.
- 2- Wesley W, Parke CM, et al: Applied anatomy of the spine in the Spine Rothman-Simeone. (5th ed), Saunder, Elsevier, 2006.
- White AA, Panjabi MM: Clinical biomechanics of the spine. (2nd ed), Philadelphia, JB Lippincott, 1990.
- 4- Gracovetsky S, Kary M, Levy S et al: Analysis of spinal and mus cular activity during flexion/extension and free lifts. Spine 15:1333-1339, 1990.
- 5- Kirkaldy-Willis WH: Pathology and pathogenesis of low back pain. In Kirkaldy-Willis WH, Burton CV (eds): Managing low back pain. (3rd ed), New York, Churchill-Livingstone, 1992, pp 49-79.
- 6- Kirkaldy-Willis WH, Farfan HF: Instability of the lumbar spine. Clin Orthop 165:110-123, 1982.
- 7- Hayes MA, Howard TC, Gruel CR, et al: Roentgenographic evaluation of lumbar spine flexion extension in asymptomatic individuals. Spine 14:327-331, 1989.
- 8- Weinstein SM, Herring SA, Standaert C: Low back pain. In DeLisa JA, Gans BM (eds): Physical medicine and rehabilitation. Philadelphia, Lippincott Williams and Wilkins 2005, pp 653-679.
- **9-** Bogduk N: The innervation of the lumbar spine. Spine 8(3):286-293, 1983.
- **10-** Garfin SR, Rydevik BL, Brown RA, et al: Compressive neuropathy of spinal nerve roots. A mechanical or biological problem. Spine 16(2):162-166, 1991.
- Crock HV: Internal disc disruption: A challange to disc prolapse fifty years on. Spine 11:650-663, 1986.
- 12- Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N: The prevalence and clinical features of internal disc distruption in patients with chronic low back pain. Spine 20(17):1878-1883, 1995.
- Crock HV: A reappraisal of intervertebral disk lesions. Med J Aust 1, 1(20):983-999, 1970.
- 14- Smith SE, Darden BV, Rhyne AL, Wood KE: Outcome of unoperated discogram positive low back pain. Spine 20:1997-2000;1995.
- 15- Boden SD, McCowin PR, Davis DO, et al: Abnormal magnetic resonans scans of the lumbar spine in asymptomatic subjects: A prospective investigation. J Bone and Joint Surg Am 72:403-408, 1990.
- **16-** Jarvik JJ, Hollingworth W, Heagerty P, et al: The longitudinal ass esment of imaging and disability of the back (LAID Back) study: Baseline Data Spine 26:1158-1166, 2001.

17- Waddle G, Main CJ, Morris EW, et al: Chronic low back pain,

psychological distress and illness behaviour. Spine 9:209-213, 1984.

- 18- Fransen M, Woodward M, Norton R, et al: Risk factors associated with the transition from acute to chronic
- occupational back pain. Spine 27:92-98, 2002.
- **19-** Linton SJ: A review of psychosocial risk factors in back and neck pain. Spine 25:1148-1156, 2000.
- **20-** Pincus T, Burton AK, Vogel S et al: A systematic review of psychosocial factors as predictors of chronicity/disability in prospective cohorts of low back pain. Spine 27:E109-E120, 2002.
- **21-** Waddell G, McCulloch JA, Kummel E, et al: Nonorganic physical signs in low back pain. Spine 5:117-125, 1980.
- 22- Pengel LH, Herbert RD, Maher CG, et al: Acute low back pain: Systematic review of its prognosis. BMJ 327:323-327, 2003.
- 23- Von Korff M, Saunders K: The couse of back pain in primary care. Spine 21:2833-2839, 1996.
- 24- Chou R, Qaseem A, Snow V, et al: Diagnosis and treatment of low back pain: A joint clinical practice guideline from the American Collage of Physicians and the American Pain Society. Ann Intern Med 147:478-491, 2007.
- **25-** Guzman J, Esmail R, Karjalainen K, et al: Multidisciplinary rehabilitation for chronic low back pain: Systematic review. BMJ 322:1511-1516, 2001.
- 26- Chou R, Loeser JD, Owens DK, Rosenquist RW, Atlas SJ, et al: Interventional therapies, surgery and interdisciplinary rehabilitation for low back pain. An evidence-based clinical practice guideline from the American Pain Society. Spine 34:1066-1077, 2009.
- 27- Loisel P, Lemaire J, Poitras S, et al: Cost benefit and cost effectiveness analysis of a disability prevention model for low back pain management: A six year follow up study. Occup Environ Med 59:807-815, 2002.
- 28- Owens DK: Spine update: Patient preferences and the development of practice guidelines. Spine 1073-1979, 1998.
- 29- Whitney SN, McGuire AL, McCullough LB: A trypology of shared decision making, informed consent, and simple consent. Annn Intern Med 140:54-59, 2003.
- **30-** Postacchini F: Results of surgery compared with conservative management for lumbar disc herniations. Spine 21(11):1383-1387, 1996.

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