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REVIEW ARTICLE

Epidemiology, Pathophysiology and Symptomatic Treatment of Sciatica: A Review

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ABSTRACT:

Sciatica refers to pain, weakness, numbness or tingling in the leg. Sciatica is a relatively common condition with a lifetime incidence varying from 13% to 40%. The common corresponding annual incidence of an episode of sciatica ranges from 1% to 6 %**.** This review assesses current knowledge of the epidemiology, pathogenesis and treatment of sciatica.

Key words: sciatica, self care, treatment, surgery

INTRODUCTION:

Sciatica refers to pain, weakness, numbness, or tingling in the leg. Sciatica is a symptom of another medical problem, not a medical age [1]. The prevalence of sciatic symptoms reported in the literature varies considerably ranging from 1.6% in the general population to 43% in a selected working population [2]. Although the prognosis is good in most patients [3], a substantial proportion (up to 30%) continues to have pain for 1 year or longer [4, 5]. In approximately 90% of the cases, sciatica is caused by a herniated disc involving nerve root compression. However, lumbar canal stenosis or foraminal stenosis and (less often) tumors or cysts are other possible causes [6].

The most important symptom of sciatica is lumbosacral radicular leg pain that follows a dermatomal pattern radiating below the knee and into the foot and toes [6, 7]. The pain worsens with coughing; patients may report sensory symptoms, limited forward flexion of the lumbar spine, gait deformity and unilateral spasm of the paraspinal muscles. However, most patients present with a less clear clinical picture. In acute sciatica, diagnostic imaging may only be indicated if there are indications of underlying pathology (e.g. infections, malignancies) other than disc herniation. In patients with persistent and severe symptoms who fail to improve following 6–8 weeks of non-surgical treatment, imaging might be useful to identify the presence or absence of a herniated disc with nerve root compression [6].

Management of sciatica varies considerably. Patients are commonly treated in primary care but a small proportion is referred to secondary care and may eventually undergo surgery if complaints remain present for at least 6 weeks. Conservative treatment for sciatica is primarily aimed at pain reduction, either by analgesics or by reducing pressure on the nerve root. There seems to be consensus that surgery is indicated in carefully selected patients for sciatica in presence of a herniated lumbar disc [8], or severe sciatica with serious or progressive neurologic deficits and imaging demonstrating lumbar disc herniation at the nerve root level correlating with the patient's examination findings [3, 9] The primary rationale of surgery for sciatica is that surgery will relieve nerve root irritation or compression due to herniated disc material. The most common type of surgery is open microdiscectomy, surgical removal of part of the disc, performed with or without the use of an operating microscope or other magnifying tools. Other minimally invasive surgical techniques, such as endoscopic surgery have recently been developed [10].

Types of Sciatica: The different types of Sciatica are summarized below:

Acute Sciatica (Short-term):

Acute sciatica may be foregoing between four to eight weeks. It does not typically require professional treatment, symptoms can be significantly reduced with the use of accessible over-the-counter (OTC) painkillers combined with exercise [11].

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Chronic Sciatica (long-term):

Chronic sciatica persists for longer period of time. It may require physical therapy which may include exercise, applied heat, and other techniques. In rare cases surgery may be required [11].

Common Causes of Sciatica: The major causes of sciatica are:

Lumbar Bulging or Herniated Disc:

A bulging disc is also known as a contained disc disorder. This means the gel-like center (nucleus pulpous) remains enclosed within the tire-like outer wall (annulus fibrosus) of the disc. A herniated disc occurs when the nucleus breaks through the annulus. It is called a non-contained disc disorder. Whether a disc bulges or herniated, disc material can press against an adjacent nerve root and compress delicate nerve tissue and cause sciatica. The consequences of a herniated disc are worse. Not only does the herniated nucleus cause direct compression of the nerve root against the interior of the bony spinal canal, but the disc material itself also contains an acidic, chemical irritant (hyaluronic acid) that causes nerve inflammation. In both cases, nerve compression and irritation cause inflammation and pain, often leading to extremity numbness, tingling and muscle weakness.

Lumbar Spinal Stenosis Spinal:

Steno sis is a nerve compression disorder most often affecting mature people. Leg pain similar to sciatica may occur as a result of lumbar spinal stenosis. The pain is usually positional, often brought on by activities such as standing or walking and relieved by sitting down. Spinal nerve roots branch outward from the spinal cord through passageways called neural foramina comprised of bone and ligaments. Between each set of vertebral bodies, located on the left and right sides, is a foramen. Nerve roots pass through these openings and extend outward beyond the spinal column to innervate other parts of the body. When these passageways become narrow or clogged causing nerve compression, the term foraminal steno sis is used.

Spondylolisthesis:

Spondylolisthesis is a disorder that most often affects the lumbar spine. It is characterized by one vertebra slipping forward over an adjacent vertebra. When a vertebra slips and is displaced, spinal nerve root compression occurs and often causes sciatic leg pain. It is categorized as developmental (found at birth, develops during childhood) or acquired from spinal degeneration, trauma or physical stress i.e. weightlifting.

Trauma:

Sciatica can result from direct nerve compression caused by external forces to the lumbar or sacral spinal nerve roots. Examples are motor vehicle accidents, falling down, football and other sports. The impact may injure the nerves or occasionally fragments of broken bone may compress the nerves.

Piriformis Syndrome:

Piriformis syndrome is named for the piriformis muscle and the pain caused when the muscle irritates the sciatic nerve. The piriformis muscle is located in the lower part of the spine, connects to the thighbone and assists in hip rotation. The sciatic nerve runs beneath the piriformis muscle. Piriformis syndrome develops when muscle spasms develop in the piriformis muscle thereby compressing the sciatic nerve.

Spinal Tumors:

Spinal tumors are abnormal growths that are either benign or cancerous (malignant). Fortunately, spinal tumors are rare. However, when a spinal tumor develops in the lumbar region, there is a risk for sciatica to develop as a result of nerve compression.

Obesity & Sciatica:

Most people know that obesity contributes to the development of coronary heart disease, diabetes, high blood pressure and colon cancer. The obesity is a causative factor to back pain. Being overweight or obese can significantly contribute to symptoms associated with osteoporosis, osteoarthritis, rheumatoid arthritis, degenerative disc disease [12].

Symptoms and Signs of Sciatica:

Sciatica pain can vary widely, but some common symptoms are given as follows:

- Some people have sharp pain in one part of the leg or hip and numbness in other parts.
- The affected leg may feel weak and thin than other leg.
- It may feel like a mild tingling, dull ache or a burning sensation.
- The sensations may also be felt on the back of the calf or on the sole of the foot.
- Pain that is worse when you lie down or awakens you at night.
- You have been losing weight unintentionally.
- This episode of back pain has lasted longer than 4 weeks.

- Redness and swelling on the back or spine.
- Sciatica is usually felt in only one leg at a time. Sometimes, a sensation like an electric shock can be felt along the nerve. The pain can range from a mild ache to incapacitating pain. Sciatica pain is often felt when you sneeze, cough, go to the toilet, or when you're sitting, and may be accompanied by lower back pain [13].

EPIDEMIOLOGY:

A number of environmental and inherent factors thought to influence the development of sciatica have been studied, including gender, body habit us, parity, age, genetic factors, occupation, and environmental factors A cross-sectional study of 2946 women and 2727 men showed neither gender nor body mass had an influence on the development of sciatica, although body mass may have been associated with low back pain [14]. Body height may be a risk factor for sciatica, although this appears to be significant only in males in the 50–64 yr age group. Parity of up to six also has been identified as having no association with sciatica [14,15]. The incidence of sciatica is related to age. Rarely seen before the age of 20, incidence peaks in the fifth decade and declines thereafter. This age distribution was also observed in those presenting for lumbar disc herniation surgery [16]. The odds ratio (OR) of an episode of sciatica increased by 1.4 for every additional 10 yr of Age, up to the age of 64[14]. Interestingly the site of disc herniation appears to change with age. Although the majority of disc herniations occur at the L4/5 or L5/S1 level, with Advancing age, there appears to be a relatively increased incidence of herniation at the L3/4 or even L2/3 level [17]. Genetic link with sciatica was first reported in a juvenile population [18]. This has also been observed in the adult population, where both retro- and prospective observational studies identified a higher incidence of sciatica or prolapsed disc among first-degree relatives than controls in a population of patients presenting for surgery on herniated lumbar discs [19, 20]. A study of pairs of adult twins identified the lifetime incidence of sciatica in monozygotic and dizygotic twins as 17.7% and 12%, respectively. The estimated heritability was 20.8% for those reporting sciatica and 10.6% for those admitted to hospital with sciatica [21]. Recreational activities, such as walking and jogging, may influence incidence of sciatica. Regular walking was shown to almost double the incidence of sciatica in a group of workers who were pain free at baseline. This study also showed that jogging had a dual effect on the incidence of sciatica. Although joggers who were pain free at baseline had a decreased incidence of sciatica, those with a previous history of sciatica were more likely to experience more episodes [22]. Physical activity associated with occupation has also been shown to influence incidence of sciatica. Carpenters and machine operators were shown to be more likely to develop sciatica than sedentary office workers [23, 24]. Retired or part-time farmers were less likely to develop sciatica than full-time ones [25]. Risk factors identified for sciatica associated with occupation included awkward working position, working in a flexed or twisted trunk position [22] ,or with the hand above the shoulder. Driving is also positively associated with sciatica or lumbar disc herniation[26,27]. It is possible that driving causes exposure to vibration at around 4–5 Hz which may coincide with resonant frequency of the spine in the seated position and so leading to a direct mechanical effect on the lumbar disc. Smoking has been linked with sciatica and several hypotheses, such as tobacco disturbing the metabolic balance of intervertebral discs, coughing causing marked elevations of intra-disc pressures, or a possible fibrinolytic effect of tobacco, have been proposed. An analysis of eight studies of smoking and sciatica revealed a positive correlation in only four of eight studies in men and one of five studies in women. Although there was a weak association between smoking and sciatica, these studies were cross-sectional and it was impossible to say that smoking preceded the sciatica [28].

PATHOPHYSIOLOGY:

The intervertebral disc was implicated in the path physiology of sciatica [29] and with the assumption that the protruding disc exerted pressure on sciatic nerve roots; the Treatment was surgical removal of the disc. Any subsequent improvement in symptoms was attributed to relief of pressure on the nerve roots. Kelly, however, suggested that pressure on a nerve results in loss of function and is rarely associated with pain [30].

There are several lines of evidence to support this. Disc pathology and steno sis with apparent neural compromise have been shown to be a relatively common finding in asymptomatic patients [31,32,33]. Symptomatic patients with disc herniation may experience marked improvement in symptoms without any alteration of the original pathology [34], whereas the removal of herniated disc material or other causes of nerve root compression

does not always relieve pain. A positive correlation was noted between contact pressure and preoperative neurological impairment, suggesting that pressure led to loss of function rather than pain [35], whereas chymopapain, a substance used for chemonucleolysis of Herniated lumbar discs, may cause a rapid relief of leg pain that precedes any change in the size of the disc herniation or degree of nerve root impingement [36]. These observations suggest that processes other than pressure on nerve roots are involved in the development of sciatic neuralgia. The evidence suggests that a complex interplay of inflammatory, immunological, and pressure related processes may be involved.

Inflammation:

When Lindahl and Rexed [37] found histological evidence of inflammation in posterior nerve roots examined during Laminotomy, they postulated that inflammation rather than pressure was the source of nerve root pain. Support for this theory was provided when injection of autologous nucleus pulpous into canine epidural space provoked an intense inflammatory reaction involving the Dura and nerve roots, with signs of epidural fibrosis present from as early as 2 weeks [38]. High levels of phospholipase A2 (PLA2), an important enzyme in the inflammatory process, were demonstrated in herniated nuclear material of patients with reticular pain [39], whereas PLA2 isolated from human disc

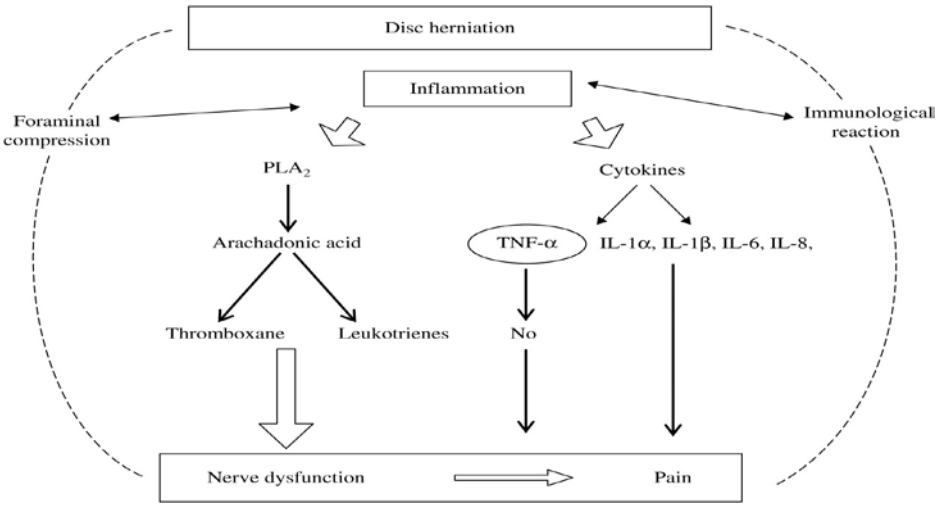

Fig 1: An overview of the pathogenesis of discogenic sciatica.

material was demonstrated to provoke an intense inflammatory reaction[40]. PLA2 activity was noted to be higher in cases of sequestrated rather than bulging discs at the time of surgery, with a strong correlation between disc and plasma PLA2 levels [41]. Injection of PLA2 into rat-Epidural space caused motor weakness and altered sensation of the hind limbs, and sustained ectopic discharge of lumbar dorsal roots was provoked. Histological l examination of nerve roots after 3 days revealed evidence of demyelination [42]. Chymopapain, used for chemonucleolysis of herniated intervertebral discs, has anti-inflammatory properties; reducing PLA2 activity around inflamed sciatic nerves [43]. This may explain why pain relief often precedes shrinkage of the herniated disc. Finally, PLA2 acting on cell membrane, releases arachidonic acid, a precursor of the inflammatory mediator leukotrienes, and thromboxanes. Elevated levels of leukotrieneB4 and thromboxane B2 have been demonstrated in human lumbar discs removed for relief of ridiculer pain [44]. Further evidence for the inflammatory properties of nucleus pulpous was demonstrated by s.c injection of autologous disc material in pigs. Titanium chambers containing autologous nucleus pulpous material attracted significantly more leucocytes than those containing fat or empty 'sham' chambers [45]. The injection of nucleus pulpous suspension also induced increased micro vascular thrombosis and macromolecular leakage in hamster cheek pouch. Autologous nucleus pulpous applied to rat L5 nerve roots reduced blood flow to the dorsal root ganglion by up to 20%. This was a statistically significant reduction compared with controls. Endometrial fluid pressure of the L5 nerve roots was also significantly raised compared with controls [46].

Cytokines have also been implicated in the genesis of this inflammatory response. Analysis of homogenates of 77 discs removed from patients with nerve root pain Revealed the presence of the cytokines interleukin-1a (IL-1a), IL-1b, IL-6, and tumor necrosis factor-a(TNF-a) [47]. High levels of IL-6, IL-8, and prostaglandin E2 (PGE2) were

found in discs removed from patients having surgery for sciatica and low back pain[48]. Raised levels of IL-8 in preoperative samples of cerebrospinal fluid (CSF) and serum in patients undergoing discectomy, correlated with a more pronounced degree of disc herniation noted at surgery [49]. Cytokines, particularly TNF, induce synthesis of nitric oxide (NO), a potent mediator of inflammation. Raised NO synthase activity was detected in rat nerve roots exposed to autologous nucleus pulpous, whereas amino guanidine, an NO synthesase inhibitor, reduced the edemas and adverse effects on nerve conduction in pig nerve roots after exposure to nucleus pulpous[50]. TNF-a appears to be the cytokine most strongly associated with the inflammatory properties of nucleus pulpous. This has been demonstrated to be present in pig nucleus pulpous, although the adverse effects of nucleus pulpous on nerve conduction were completely blocked by doxycycline, a compound that inhibits the effects of TNF-a [51]. The effects on porcine saccrococcygeal cauda equina were inhibited by the selective TNF-an inhibitors etanercept and infliximab. These drugs reduced effects on nerve conduction velocity, intracapillary thrombus formation, and intraneural edema formation compared with enoxaparin and control [52]. Monoclonal anti-TNF-a antibodies were shown to inhibit the enhanced activity that was seen in wide dynamic range neurons of the superficial dorsal horn when Autologous nucleus pulposus was applied to the L5 nerve root [53]. Finally, infusion of the monoclonal anti-TNF-a antibody infliximab in 10 patients with herniated disc-induced sciatica led to significant reductions in pain levels at 1 h, 2 weeks, and 3 months, compared with historical controls [54]. These studies all suggest that TNF-a plays an early and prominent role in the path physiological events that lead to nerve dysfunction and pain when Nucleus pulposus is approximated to lumbar nerve roots.

Immunological:

There is some evidence to suggest that the immune system also may play a part in the reaction between the nerve root and the exposed nucleus pulposus. Glycosphingolipids (GSLs) are particularly abundant in cell types of the central and peripheral nervous system [55,56]. Titers of antibodies to these cell components are normally very low but Become elevated in auto-immune conditions of the nervous system such as Guillan–Barre´ syndrome [57]. Antibodies to GSLs were measured in patients with acute and chronic sciatica and those who had lumbar discectomy for disc herniation. Raised antibody levels to GSLs were an overview of the pathogenesis of discogenic sciatica. Stafford et al. Downloaded from bja.oxfordjournals.org by guest on October 5, 2010 detected in 71% of patients with acute sciatica, 61% at 4 yr follow-up, and 54% of those undergoing discectomy[58]. Markers of glial cell and nerve damage [neurofilament (NFL), glial fibrillary acidic protein, S-100 protein, and neuron-specific enolase] were measured in the CSF of patients presenting for lumbar disc surgery and compared with controls. CSF levels of NFL protein and S-100 were significantly elevated in patients appearing for disc surgery compared with controls. Patients with symptoms of sciatica for, 3 months duration had higher NFL protein levels than those with symptoms for longer. Patients with persistent neurological findings at 3 months post-surgery had higher preoperative NFL levels than those who did not develop_sequelae.[59] these studies suggest that an immune reaction to nervous tissue may be involved in the pathogenesis of both acute and chronic sciatica.

Mechanical compression:

The evidence above strongly suggests that an inflammatory and immune response is involved in the pathogenesis of nerve root irritation and sciatic type pain. There is also some evidence to suggest that nerve root compression may also be involved. Claude equine compression with a nonirritant silicone tube in rats led to significantly higher rates of sural nerve ectopic firing than control animals. Administration of a nitroprusside infusion, a source of NO, led to increased ectopic firing only in those animals with cauda equine compression [60]. An observational study, with magnetic resonance imaging (MRI) in consecutive patients with leg pain, noted that 9.6% had no disc disease, 3.3% bulging, 11.4% protrusion, 68.5% extrusion, and 7.1% disc sequestration, respectively. A statistically significant positive correlation between the severity of disc disease and leg pain, and Roland-Morris and Prolo disability scales were observed, that is, those with larger herniations had more leg (but not back) pain and disability[61]. Another observational study noted the prevalence of swelling of dorsal root ganglia and impingement within the intervertebral foramina at the appropriate level and side in patients with a unilateral monoradiculopathy. Again, the degree of swelling and Impingement correlated well with severity of

leg pain [62]. As already noted, elevated CSF levels of NFL and S-100 were observed in patients with verified disc herniations. These proteins are nervous system specific and their presence indicates damage to central nervous system structures [59]. When either an aneroid constrictor or an autologous nucleus pulposus material was applied to porcine S1 nerve root, it was noted only compression of the S1 nerve root significantly raised levels of NFL and total protein Concentrations in the CSF. This was not seen with nucleus pulposus alone [63]. Another animal model, exposing rats to experimental disc herniation, medial displacement of the fourth dorsal root ganglion, both or sham procedure revealed that exposure to nucleus pulposus without nerve root compression or chronic nerve root displacement alone did not significantly alter mechanical or thermal stimulatory thresholds. However, in animals exposed to both nucleus pulposus and nerve root displacement, there was a significant Reduction in threshold for thermal stimuli that lasted for the 14 day experimental period [64]. Histological examination of nerve roots revealed edemas in both nucleus pulposus Exposed and displaced nerve roots, being slightly more severe in the displaced group. In animals exposed to both, histology of nerve roots indicated significant cellular injury at day 21 with edema, fibrotic reactions, evidence of axonal demyelization, and Schwann cell hypertrophy. From the above evidence, it could be proposed that ridiculer pain in sciatic nerve roots arises from a complex interaction of inflammatory, immune, and pressure-related elements. This can most easily be appreciated in terms of intervertebral disc-mediated pain where the majority of research has been conducted, although it is probably equally applicable to all other forms of sciatic neuralgia. The high incidence of asymptomatic individuals with disc abnormalities associated with neural compromise shows that pressure alone does not cause pain in sciatic nerve roots. Although disc bulging, to a varying degree is common, nucleus pulposus sequestration or extrusion is rarely seen in asymptomatic individuals, the potent inflammatory properties of nucleus pulposus have been outlined earlier and involve the major inflammatory mediators. This causes an inflammatory reaction in sciatic nerve roots which has been shown, in animal models, to lead to sustained ectopic discharge, demyelization [65], decreased blood flows to the dorsal root ganglion, increased endoneurial pressure, and decreased conduction velocity [49]. Response, but the above evidence suggests that an abnormal response may occur, with antibodies being formed to normal neural elements. Crucially, this may also be related to the development of chronic sciatica. This inflammatory process seems to be exacerbated by the effects of nerve root pressure. Lumbo-sacral nerve roots, possibly due to the vulnerability of its venous drainage system, seem to be particularly susceptible to the effects of pressure. This may explain why even minor compression may lead to nerve root edema, intraneural inflammation, and hypersensitivity [66]. This theory is supported by Haddocks, who wrote that 'Surgeons . State that the nerve root that is causing the problem is easily identifiable by its edematous inflammatory character [67]. although passive congestion does not necessarily cause inflammation, this underlines the potential for lumbar nerve roots to become congested and swollen which presumably exacerbates any underlying inflammation. This combination of susceptibility to inflammation and pressure effects with subsequent edema may be what makes the lumbo-sacral nerve roots so particularly vulnerable to neuropathies.

DIAGNOSIS OF SCIATICA:

Sciatica is mainly diagnosed by history taking and physical examination. By definition patients mention radiating pain in the leg. They may be asked to report the distribution of the pain and whether it radiates below the knee and drawings may be used to evaluate the distribution.

Sciatica is characterized by radiating pain that follows a dermatomal pattern. Patients may also report sensory symptoms. Physical examination largely depends on neurological testing. The most applied investigation is the straight leg raising test or Lasègue's sign. Patients with sciatica may also have low back pain but this is usually less severe than the leg pain. The diagnostic value of history and physical examination has not been well studied [68]. **70** No history items or physical examination tests have both high sensitivity and high specificity. The pooled sensitivity of the straight leg raising test is estimated to be 91%, with a corresponding pooled specificity of 26%.The only test with a high specificity is the crossed straight leg raising test, with a pooled specificity of 88% but sensitivity of only 29% [69]. Overall, if a patient reports the typical radiating pain in one leg combined with a positive result on one or more neurological tests indicating nerve

root tension or neurological deficit the diagnosis of sciatica seems justified.

Value of imaging:

Diagnostic imaging is only useful if the results influence further management. In acute sciatica the diagnosis is based on history taking and physical examination and treatment is conservative (non-surgical). Imaging may be indicated at this stage only if there are indications or "red flags" that the sciatica may be caused by underlying disease (infections, malignancies) rather than disc herniation. Diagnostic imaging may also be indicated in patients with severe symptoms who fail to respond to conservative care for 6-8 weeks. In these cases surgery might be considered and imaging used to identify if a herniated disc with nerve root compression is present and its location and extent. It is important as part of the decision to operate that the clinical findings and symptoms Correspond well with the scan findings. This is especially relevant because disc herniations identified by computed tomography or magnetic resonance imaging are highly prevalent (20%-36%) in people without symptoms who do not have sciatica.6 w3 In many people with clinical symptoms of sciatica no lumbar disc herniations are present on scans. At present no one type of imaging method shows a clear advantage over others. Although some authors favor magnetic resonance imaging above other imaging techniques because computed tomography has a higher radiation dose or because soft tissues are better visualized, evidence shows that both are equally accurate at diagnosing lumbar disc herniation. Radiography for the diagnosis of lumbar disc herniation is not recommended because discs cannot be visualized by x-rays [70].

TREATNENT OF SCIATICA:

Self-care at home:

Self care measures can help relieve the symptoms of sciatica and also prevent recurrence.

- Cold and hot packs. Use alternate cold and hot packs to reduce swelling and relieve discomfort.
- Practice good posture. Stand up straight with your ears aligned with your shoulders, your shoulders aligned with your hips and your buttocks tucked in. Your knees should be bent slightly.
- Regular exercise: improves flexibility and helps prevent age-related degenerative changes in your back.
- Lift objects safely. Always lift from a squatting position, using your hips and legs to do the heavy work. Never bend over and lift with a straight back.
- Avoid sitting or standing for extended periods. If you sit at work, take regular breaks to stand and walk around. If you must be on your feet, prop one foot on a small block or footrest, and then switch feet throughout the day.
- Use proper sleeping posture. Take pressure off your back by sleeping on your side or on your back with a pillow under your knees.
- Avoid wearing high heels. Shoes with heels that are more than 1½ inches high shift your weight forward, throwing the body out of alignment.
- Do abdominal crunches. These exercises strengthen the abdominal muscles that help to support your lower back. Lie with your back on the floor, hands behind your head and knees bent. Press your lower back to the floor, lift your shoulders up about 10 inches off the floor, and then lower them. Repeat 10 to 20 times daily.
- Lay in the face down position and clasp your hands behind the lower back, then raise the head and chest slightly against gravity while looking at the floor.
- In the above position with the head and chest lowered to the floor, lightly raise an arm and opposite leg slowly, with the knee locked, 2-3 inches from the floor.
- Stretch. Sit in a chair and bend down toward the floor. Stop when you feel just slight discomfort, hold for 30 seconds, then release. Repeat six to eight times.
- Lie on the back and gently pull the knees to the chest until a comfortable stretch is felt.
- Walk/swim. Walking and swimming can help to strengthen your lower back.[71]

Medications:

Pain medications vary considerably. Specific types and causes of pain may respond better to one kind of pain medication than to another kind. These all may suppress the sciatica pain temporally, not permanently. Also, each person is slightly different in the way they respond to a pain medication. Chronic pain suffers who are on medication may have breakthrough pain. These are uncontrolled severe flares of pain that "break

through" the medication. Medications used to treat pain include:

- *Analgesics*, such as acetaminophen (Tylenol) and tramadol (Ultram), can relieve pain but don't have the anti-inflammatory effects of NSAID'S.
- *Nonsteroidal anti-inflammatory drugs* (NSAID'S) - aspirin, ibuprofen (Motrin, Nuprin, and Advil), naproxen (Naprosyn), and celecoxib (Celebrex) are examples of nonsteroidal anti-inflammatory drugs used to reduce inflammation and relieve pain. Long-term use of analgesics and NSAID'S may cause stomach ulcers as well as kidney and liver problems.
- *Muscle relaxants* such as diazepam (Valium), clonazepam (Klonopin), cyclobenzaprine (Flexeril), and baclofen (Liorisol) can be used to treat pain associated with muscle spasms and spasticity.
- *Anticonvulsants* such as phenytoin (Dilantin) and carbamazepine (Tegretol), gabapentin (Neurontin) can be used to relieve nerve pain as in trigeminal neuralgia.
- *Steroids* can be used to reduce the swelling and inflammation of the nerves. They are taken orally (as a Medrol dose pack) in a tapering dosage over a 5-day period. They have the advantage of providing pain relief within a 24-hour period. Steroid injections into the area of your pain may be prescribed if your pain is severe.

Epidural steroid injections:

This procedure, usually performed under fluoroscopy, involves an injection of steroids and an analgesic numbing agent into the epidural space of the spine to reduce the swelling and inflammation of the nerves. About 50% of patients will notice relief after an epidural injection, although the results tend to be temporary. This procedure is usually done in a series of three, at 2-week intervals, to obtain the best results in the shortest time. If the injections are helpful, the series can be done up to three times a year.

Facet injections:

Facet injections are used for patients with low back pain stemming from inflammation or irritation of the facet joint. They may be performed using a fluoroscope (X-ray), which directs a needle through the skin and muscles to the path of the sensory nerves located in the facet joints. At that point, a mixture of numbing medicine and cortisone is injected into the facet joint [72].

Narcotics (opioids)

Narcotics are very powerful pain relievers that actually deaden a person's perception of pain. They are used for a short period (2 to 4 weeks) after an acute injury or surgery. Common narcotics include codeine (Tylenol 3), meperidine (Demerol), propoxyphene (Darvocet), hydrocodone (Vicodin), and oxycodone (Percocet, Oxycontin). Sumatriptan (Imitrex) and naratriptan (Amerge) are used to relieve migraine headache. Narcotic medications cause impaired mental function, drowsiness, nausea, constipation, and sometimes addiction[72] .

Surgery in case of sciatica:

Surgical intervention for sciatica focuses on removal of disc herniation and eventually part of the disc or on foraminal stenosis, with the purpose of eliminating the suspected cause of the sciatica. Treatment is aimed at easing the leg pain and corresponding symptoms and not at reducing the back pain. Consensus is that a cauda equina syndrome is an absolute indication for immediate surgery. Elective surgery is the choice for unilateral sciatica. Until recently only one relatively old randomised trial was available that compared surgical intervention with conservative treatment for patients with sciatica [73]. This study showed that surgical intervention had better results after one year, whereas after four and 10 years of follow-up no significant differences were found [73].

A Cochrane review summarised the available randomised clinical trials evaluating disc surgery and chemonucleolysis [74]. In chemonucleolysis the enzyme chymopapain is injected in the discus with the purpose of shrinking the nucleus pulposus. The review reported better results with disc surgery than with chemonucleolysis in patients with severe sciatica of relatively long duration varying from more than four weeks to more than four months. Chemonucleolysis was more effective than placebo. Indirectly therefore the review suggested that disc surgery is more effective than placebo. On the basis of data from three trials the authors concluded that evidence is considerable that surgical discectomy provides effective clinical relief for carefully selected patients with sciatica as a result of lumbar disc prolapse that fails to resolve with conservative care. A recent review came to the same conclusion

[75]. The Cochrane review further concluded that the long term effects of surgical intervention are unclear and that evidence on the optimal timing of surgery is also lacking [74].

REFERENCE

1. Clarke JA, van Tulder MW, Blomberg SE, et al. Traction for low-back pain with or without sciatica. Cochrane Database Syst Rev. 2007 ;(2):CD003010.
2. Konstantinou K, Dunn KM (2008) Sciatica: review of epidemiological studies and prevalence estimates. Spine (Phila Pa 1976) 33:2464–2472.
3. Legrand E, Bouvard B, Audran M, Fournier D, Valat JP. Sciatica from disk herniation: medical treatment or surgery? Joint Bone Spine. 2007; 74:530–535. doi: 10.1016/j.jbspin.2007.07.004.
4. Vroomen PC, Krom MC, Slofstra PD, Knottnerus JA. Conservative treatment of sciatica: a systematic review. J Spinal Disord. 2000; 13:463–469. doi: 10.1097/00002517-200012000-00001.
5. Weber H, Holme I, Amlie E (1993) the natural course of acute sciatica with nerve root symptoms in a double-blind placebo-controlled trial evaluating the effect of piroxicam. Spine (Phila Pa 1976) 18:1433–1438.
6. Valat JP, Genevay S, Marty M, Rozenberg S, Koes B. Sciatica. Best Pract Res Clin Rheumatol. 2010; 24:241–252. doi: 10.1016/j.berh.2009.11.005.
7. Tulder M, Peul W, Koes B. Sciatica: what the rheumatologist needs to know. Nat Rev Rheumatol. 2010; 6:139–145. doi: 10.1038/nrrheum.2010.3.
8. Gibson JNA, Waddell G (2007) surgical interventions for lumbar disc prolapse. Cochrane Database of Systematic Reviews CD001350. doi:10.1002/14651858.CD001350.
9. Gregory DS, Seto CK, Wortley GC, Shugart CM. Acute lumbar disk pain: navigating evaluation and treatment choices. Am Fam Physician. 2008;78:835–842.
10. Caroline Gillot.Text book of bone deformities, Garhwal prakashan: maharastra ,1998;96-99.
11. Foley K, Smith MM (1997) Microendoscopic discectomy. Tech Neurosurg 3:301–307.
12. Jean-Jacques Abitbol, MD (Orthopedic Surgeon) leading Causes of Sciatica 2010; 72:556-8.
13. Heler jalan, UBM Medica Australia, Article on Sciatica 2000-2008, 17.
14. Heliovaara M, Makela M, Knekt P, Impiva ara O, Aromaa A Determinants of sciatica and low back pain. Spine 1991; 16:608-14.
15. Heliovaara M Risk factors for low back pain and sciatica. Ann Med 1989; 21:257-64.
16. Spangfort EV The lumbar disc her nation: a computer aided analysis of 2504 operations.Acta Orthop Scand Suppl 1972;142:1-95.
17. Frymoyer JW Back pain and sciatica. N Engl J Med 1988; 318:291-300.
18. Varlotta GP, Brown MD, Kelsey JL, Gold en alfamilial predisposition for herniation of a lumbar disc in patients who are less than twenty one years old. J Bone jointsurgam1991; 73:124-8.
19. Matsui H, Kanamori M, Ishihara H, Yudoh K, Naruse Y, Tsuji H Familial predisposition for lumbar degenerative disc disease. Spine1998; 23:1029-34.
20. Heikkila JK, Koskenvuo M, Heliovaara M, et al Genetic and environmental factors in sciatica. Evidence from a nationwide panel of 9365 adult twin pairs. Ann Med 1989; 21:393-8.
21. Simmons ED, Guntupalli M, Kowalski JM , Braun F, Seidel T. Familial predisposition for degenerative disc disease. Spine1996; 21:1527-9.
22. Miranda H, Viikari-Juntura E, Martikainen R, Takala EP, Riihimaki H. I ndividual factors, occupational loading and physical exercise as predictors of sciatica pain. Spine2002; 27:1002-9.
23. Riihimaki H, Tola S, Videman T, Hannine n K. Low back pain and occupation Spine 1989; 14:204-9 .
24. Riihimaki H, Viikari-Juntura E, Moneta G, Kuha J, Videman T, Tola S Incidence of sciatic pain among men in machine operating, dynamic

physical work and sedentary work. Spine1994; 19:138-42.

25. Manninen P, Riihimaki H, Heliovaara M I ncidence and risk factors of low-back pain in middle aged farmers. Occup Med 1995; 45:141-6.
26. Heliovaara M Body height, obesity and risk of herniated lumbar intervertebral disc. Spine1987; 12:469-72.
27. Kelsey JL, Githens PB, O'Connor T, et al Acute prolapsed lumbar intervertebral disc: an epidemiologic study with special reference to driving automobiles and cigarette smoking Spine 1984;9:608-13.
28. Goldberg MS, Scott SC, Mayo NEA review of the association between cigarette smoking and the development of non specific back pain and related outcomes. Spine2000; 25:995-1014.
29. Mixter WJ, Barr jsrupture of the intervertebral disc with involvement of the spinal canal.N Engl J Med 1934; 211:210-5.
30. Kelly mpain due to pressure on nerves. Spinal tumours and the intervertebral disc. Neurology 1956; 6:32-6.
31. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg[Am] 1990;72:403-8.
32. Boos N, Semmer N, Elfering E, et al Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging. Spine 2000; 25:1484-92.
33. Jensen MC, brantzawadzki MN, Obuchow ski N, Modil MT, Malkasian D, Ross JS Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med1994;331:69-73.
34. Garfin SR, Rydevik BL, Brown RA. Comp ressive neuropathy of spinal nerve roots. Spine 1991; 16:162-6.
35. Takahashi K, Shima I, Porter RW. Nerve root pressure in lumbar disc herniation. Spine1999; 24:2003-6.
36. Kato F, Mimatsu K, Kawakami N, Iwata H , Miura T Serial changes observed by magnetic resonance imaging in the intervertebral disc after chemonucleolysis. A consideration of the mechanism of chemonucleolysis. Spine 1992;17:934-9.
37. Lindahl O, Rexed B. Histological changes in spinal nerve roots of operated cases of sciatica. Acta Orthop Scand 1951; 20:215-25.
38. McCarron RF, Wimpee MW, Hudkins PG, Laros gsthe inflammatory effects of nucleus pulposus: a possible element in the pathogenesis of low back pain. Spine 1987;12:760-4.
39. Saal JS, Franson RC, Dobrow R, White A H, Goldthwaite N High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. Spine 1990; 15:674-8.
40. Franson RC, Saal JS, Saal JA Human disc phospholipase A2 is inflammatory. Spine 1992; 17:5129-32.
41. Piperno M, Helliole Graverand MP, Reboul P, et al. Phospholipase A2 activity in herniated lumbar discs. Spine 1997; 22:2061-5.
42. Chen C, Cavanaugh JM, Ozaktay C, Kalla kuri S, King AI. Effects of phospholipase A2 on lumbar nerve root structure and function. Spine 1997; 22:1057-64.
43. Sawin PD, Traynelis VC, Rich G, et al Chymopapain-induced reduction of pro inflammatory phospholipase A2 activity and amelioration of neuropathic behavioural changes in an in vivo model of acute sciatica. J Neurosurg 1997;86:998-1006.
44. Nygaard OP, Mellgren SI, Osterud bthe inflammatory properties of contained and noncontained lumbar disc herniation. Spine 1997; 22:2484-8.
45. Olmarker K, Blomquist J, Stromberg J, Ha nnmark U, Thomsen P, Rydevik B. Inflam matogenic properties of nucleus pulposus. Spine 1995; 20:665-9.
46. Yabuki S, Kikuchi S, Olmarker K, Myers RR Acute effects of nucleus pulposus on blood flow and endoneurial fluid pressure in rat dorsal root ganglia. Spine 1998; 23:2517-23.
47. Takahashi H, Suguro T, Okazima Y, Mo tegi M, Okada Y, Kakiuchi T. Inflammat ory cytokines in the herniated disc of the lumbar spine. Spine 1996; 21:218-24.

48. Burke JG, Watson RWG, mccormack D, Dowling FE, Walsh MG, Fitzpatrick JM. Intervertebral discs which cause low back pain secrete high levels of proinflammatory mediators. J Bone Joint Surg Br 2002; 84-B: 196201.
49. Brisby H, Olmarker K, Larsson K, Nutu M, Rydevic B Proinflammatory cytokines in cerebrospinal fluid and serum in patients with disc herniation and sciatica. Eur Spine J2002; 11:62-6.
50. Brisby H, Byrod G, Olmarker K, Miller V M, Aoki Y, Rydevik B. Nitric oxide as a mediator of nucleus pulposus-induced effects on spinal nerve roots. J Orthop Res 2000; 18:815-20.
51. Olmarker K, Larsson K Tumor necrosis factor α and nucleus-pulposus-induced nerve root injury. Spine 1998; 23:2538-44.
52. Olmarker K, Rydevik B Selective inhibition of tumor necrosis factor-α prevents nucleus pulposus-induced thrombus formation, intraneural oedema, andreduction of nerve conduction velocity. Spine 2001; 26:863-9.
53. Onda A, Yabuki S, Kikuchi seffects of neutralizing antibodies to tumor necrosis factor-alpha on nucleus pulposus-induced abnormal nociresponses in rat dorsal horn neurons.Spine 2003;28:967-72.
54. Karppinen J, Korhonen T, Malmivaara A, et al Tumor necrosis factor-α monoclonal antibody, infliximab, used to manage severe sciatica. Spine 2003; 28:750-4.
55. Sullivan WJ, Willick SE, Chira-Adisai W, et al. Incidence of intravascular uptake in lumbar spinal injection procedures. Spine 2000; 25:481-6.
56. Svennerholm L, Bostrom K, Fredman P, Mansson JE, Rosengren B, Rynmark BM Human brain gangliosides: developmental changes from early fetal stage to advanced age. Biochim biophysacta 1989; 1005:109-17.
57. Baumann N, Harpin ML, Marie Y, et al. Antiglycolipid antibodies in motor neuropathies. Ann NY Acad Sci 1998; 845:322-9.
58. Brisby H, Balague F, Schafer D, et al Glycosphingolipid antibodies in serum in patients with sciatica. Spine 2002; 27:380-6.
59. Brisby H, Olmarker K, Rosengren L, Cede rlund CG, Rydevik bmarkers of nerve tissue injury in the cerebrospinal fluid in patients with lumbar disc herniation and sciatica. Spine1999; 24:742.
60. Onozawa T, Atsuta Y, Sato M, Ikawa M, Tsunekawa H, Feng X Nitric oxide induced ectopic firing in a lumbar nerve root with cauda equina compression. Clin Orthop2003; 408:167-73.
61. Porchet F, Wietlisbach V, Burnand B, Dae ppen K, Villemure JG, Vader JP. Relations hip between severity of lumbar disc disease and disability scores in sciatica patients Neurosurgery 2002; 50:1253-9.
62. Aota Y, Onari K, An HS, Yoshikawa kdor sal root ganglia morphologic features in patients with herniation of nucleus pulposus. Spine 2001; 26:2125-32.
63. Skouen JS, Brisby H, Otani K, Olmarker K, Rosengren K, Rydevik B Protein markers in cerebrospinal fluid in experimental nerve root injury. Spine 1999; 24:2195-200.
64. Myers Olmarker kpathogenesis of sciatic pain: role of herniated nucleus pulposus and deformation of spinal nerve root and dorsal rooganglion. Pain 1998; 78:99-105.
65. Cicala RS, Turner R, Moran BS, et al. Methylprednisolone acetate does not cause inflammatory changes in the epidural space. Anesthesiology 1990; 72:556-8.
66. Rydevik BL, Pedowitz RA, Hargens AR, S wenson MR, Myers RR, Garfin sreffects of acute, graded compression on spinal nerve root function and structure. An experimental study of the pig cauda equina. Spine 1991; 16:487-93.
67. Haddox JD. Lumbar and cervical epidural steroid therapy. Anesthesiol Clin North America 1992; 10:179-203.
68. Vroomen PCAJ, Krom MCTFM de, Knottnerus JA. Diagnostic value of history and physical examination in patients suspected of sciatica due to disc herniation: a systematic review. J Neural 1999; 246:899-906.
69. Deville WLJM, Windt DAWM, van der Dzaferagic A, Bezemer PD, Bouter LM. The test of Lasegue: systematic review of the accuracy in diagnosing herniated discs. Spine 2000; 25:11407.

70. Jarvik JG, Deyo RA. Diagnostic evaluation of low back pain with emphasis on imaging. Ann internmed 2002137:586-97.

71. Neepa Sevak "Homeopathic Treatment of Sciatica" 1996-602-347-7950.
72. *Dr. Theresa Greenwald, RN, Mayfield Spine Institute "Pain Management" updated: 6.2004.*
73. Weber H. Lumbar disc herniation. A controlled prospective study with ten years of observation. Spine 1983; 8:131-40.
74. Gibson JN, Waddell G. Surgical interventions for lumbar disc prolapse. Cochrane Database Syst Rev 2007. Jan 24 ;(1):CD001350.
75. Van Tulder MW, Koes B, Seitsalo S, Malmivaara A. Outcome of invasive treatment modalities on back pain and sciatica: an evidence-based review. Eur Spine J 2006; 15:S82-92.