

**STUDY OF EFFICACY OF EPIDURAL METHYL
PREDNISOLONE ACETATE IN LUMBAR
RADICULOPATHY**

By

Dr. VENKATA SATYANARAYANA.K



DISSERTATION SUBMITTED TO
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH ,
KOLAR , KARNATAKA IN PARTIAL FULFILMENT OF THE
REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF SURGERY

IN

ORTHOPAEDICS

Under the guidance of

Dr. N.S.GUDI. M.S.

Professor



**DEPARTMENT OF ORTHOPAEDICS
SRI DEVARAJ URS MEDICAL COLLEGE**

TAMAKA, KOLAR-563101

APRIL-MAY 2014

Sri Devaraj Urs Academy Of Higher Education And Research,
Tamaka , Kolar Karnataka,

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled “**STUDY OF EFFICACY OF EPIDURAL METHYL PREDNISOLONE ACETATE IN LUMBAR RADICULOPATHY**” is a bonafide and genuine research work carried out by me under the guidance of **Dr.N.S.GUDI** M.S. **Professor.** Department Of Orthopaedics Sri Devaraj Urs Medical College, Tamaka, Kolar.

Date:

Dr. Venkata Satyanarayana.k

Place : Kolar .

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled “**STUDY OF EFFICACY OF EPIDURAL METHYL PREDNISOLONE ACETATE IN LUMBAR RADICULOPATHY**” is a bonafide research work done by **Dr.VENKATA SATYANARAYANA.K** in partial fulfillment of the requirement for the degree of **DOCTOR OF SURGERY(M.S.)** in **ORTHOPAEDICS.**

Date :

Place : Kolar

Dr.N.S.GUDI, M.S.

Professor.

Department Of Orthopaedics
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

CERTIFICATE BY THE CO-GUIDE

This is to certify that the dissertation entitled “**STUDY OF EFFICACY OF EPIDURAL METHYL PREDNISOLONE ACETATE IN LUMBAR RADICULOPATHY**” is a bonafide research work done by **Dr.VENKATA SATYANARAYANA.K** in partial fulfillment of the requirement for the degree of **DOCTOR OF SURGERY(M.S.) in ORTHOPAEDICS.**

Date :

Place : Kolar

Dr.RAVI.M.DNB,MNAMS

Professor.

Department Of Anesthesiology & Critical care

**Sri Devaraj Urs Medical College,
Tamaka, Kolar.**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

ENDORSEMENT BY THE HOD, PRINCIPAL / HEAD OF THE
INSTITUTION

This is to certify that the dissertation entitled “**STUDY OF EFFICACY OF EPIDURAL METHYL PREDNISOLONE ACETATE IN LUMBAR RADICULOPATHY**” is a bonafide research work done by **Dr.VENKATA SATYANARAYANA.K.** under the guidance of **Dr.N.S.GUDI_{M.S.} Professor** Department of Orthopaedics Sri Devaraj Urs Medical College, Tamaka, Kolar.

Dr.P.V.Manohar,
Professor and HOD.
Department of Orthopaedics
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

Place:Kolar

Dr. M.B.Sanikop,
Principal,
Sri Devaraj Urs Medical College.
Tamaka, Kolar.

Date:

Place: Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved

Dr.VENKATA SATYANARAYANA.K

Post-Graduate student in the subject of

ORTHOPAEDICS

at

Sri Devaraj Urs Medical College, Kolar

to take up the Dissertation work entitled

**“STUDY OF EFFICACY OF EPIDURAL METHYL
PREDNISOLONE ACETATE IN LUMBAR RADICULOPATHY”**

to be submitted to

**SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND
RESEARCH TAMAKA, KOLAR, KARNATAKA,**

Date:

Place: Kolar

Member Secretary
Sri Devaraj Urs Medical College,
Kolar-563101

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR, KARNATAKA,

COPY RIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Sri Devaraj Urs Academy Of Higher Education And Research, Kolar ,Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic /research purpose.

Dr. VENKATA SATYANARAYANA.K

Post-Graduate
Orthopaedics
SDUMC,Kolar

Date :

Place : Kolar

© SRI DEVARAJ URS UNIVERSITY , KOLAR , KARNATAKA,

ACKNOWLEDGEMENT

I take this opportunity to acknowledge my sincere heartfelt thanks to all those who contributed in many ways to the success of this study and made it an unforgettable experience for me.

I begin by expressing my unwavering gratitude to the **Almighty** for all his blessings and to **my patients** who kindly obliged for being part of my study and making it a success.

Any attempt at any level cannot be satisfactorily completed without the support and guidance of my teacher. At the outset, I am pleased to express my admiration and gratitude to **Dr.N.S.Gudi** for his guidance, timely suggestions, diligent directions and constant supervision which went a long way in moulding this dissertation study and without whose help this study would not have been possible.

I convey my deepest regards and heartfelt thanks to my respected **Professor and Head of the department Dr.P.V.Manohar**, his valuable advice and guidance in completing this dissertation.

I gratefully acknowledge my co-guide **Dr. Ravi.M** for his understanding, constant guidance and unwavering help in clearing my doubts and encouraging me during my study.

I remain thankful to **Dr. B.S.Nazeer, Dr. Arun.H.S** and all my **assistant professors** and **lecturers** for their guidance, motivation and moral support during my entire postgraduate course which enabled me to complete my work.

I express my sincere thanks to my **colleagues** and **dear friends** **Dr.Nithin Krishna, Dr.Gopinath, Dr.Ram Bhupal Varma,Dr.Praneeth Reddy** and **Dr.Girish** for their co-operation and help in carrying out this study.

I thank my seniors **Dr.Kiran, Dr.Jaya Krishna, Dr.Siddarth, Dr.Veera Satyanarayana** for keeping me on my toes by asking me thought provoking questions throughout my study. I thank my **Juniors** for providing the useful tips and clues in completing this vast work.

No words can describe the constant motivation, moral support that my **Parents, Mr.K.Adisheshaiah** and **Mrs.Jaya Pradha** for all their sacrifices, encouragement and support. Also, my gratitude goes to my brother **Dr.K.Sabari Girish** for always being there to help me in every way he can. I am very much thankful to my wife **Dr.K.Anusha** and my daughter. **K.Loukhya** for their love.

I am also thankful to **Dr. Mahesh** for his immense help in doing the tedious statistical work.

Last but not the least I would like to thank all the **OT and paramedical Staff** for their kind help.

Dr.VENKATA SATYANARAYANA.K

ABSTRACT

Background:

Chronic back pain with sciatica represents an important health problem in orthopaedic practice. The number of patients presenting with low back pain with radiculopathy constitute nearly 60% of OPD practice. Patients presenting to us are of rural background involved in heavy physical labour, these symptoms temporarily disables them in earning a livelihood. So an approach has to be worked out to alleviate their symptoms early.

Objective:

To assess the efficacy of epidural steroid injection on pain relief and functional return in lumbar radiculopathy.

Materials and Methods:

Patients admitted in the department of orthopaedics from December 2011 to July 2013 in R L Jalappa Hospital and research centre attached to Sri Devaraj Urs Medical College, Tamaka , Kolar with lumbosacral nerve root pain which has not resolved within a minimum of six weeks was included in the study.

After selecting the cases on the basis of inclusion and exclusion criteria,all patients had 80 mg Methyl Prednisolone Acetate(40mg/ml) along with 4cc of 2% Xylocard in lumbar epidural space. A total of hundred patients were included in this study.Efficacy was analysed using parameters like SLRT,ODI score,VAS score.Followup is for a period of minimum 3 months.

Results:

The mean age group was 42.53 and 51% were in the middle age group between 30 to 50 years. In the study it was observed that the majority i.e. 65% of patients had symptoms <12 months. There was statistically significant improvement in SLRT from 63.5 mean preintervention to 81.29 mean 3 months post steroid injection on right side, 62.35 to 79.22 on left side. ODI improved from 45.65 prior to treatment to 39.17 at 3 months. VAS before treatment was 7.45 mean, 1.46 at 3 months.

Conclusion:

We conclude that epidural steroid injection is an effective non surgical treatment option for patients with low backache and radicular leg pain. Epidural steroid should be considered before surgical intervention. It is only after adequate pain control that rehabilitation can be effective and function restored in many low backache patients.

Key words: Lumbar radiculopathy, Epidural steroid, Oswestry Disability Index, Visual Analogue Scale

LIST OF ABBREVIATIONS

1. Oswestry Disability Index – ODI
2. Straight Leg Raising Test - SLRT
3. Visual Analogue Scale – VAS
4. Intervertebral Disc Prolapse-IVDP

TABLE OF CONTENTS

Sl.No	CONTENT	Page No.
1	INTRODUCTION	1
2	OBJECTIVES OF THE STUDY	3
3	REVIEW OF LITERATURE	4
4	METHODOLOGY	57
5	RESULTS	64
6	DISCUSSION	89
7	CONCLUSION	92
8	BIBLIOGRAPHY	93
9	ANNEXURES	101

LIST OF TABLES

Sl.No	TABLE	Page No.
1	Root Involvement	34
2	Age distribution	65
3	Sex distribution	66
4	Distribution of patients according to Occupation	67
5	Distribution of patients according to side of Sciatica	68
6	Distribution of patients according to Duration	69
7	Distribution of patients according to previous treatment	70
8	Distribution of patients according to Co-morbid conditions	71
9	Distribution of patients according to spasm	72
10	Distribution of patients according to degree of SLRT before treatment	73
11	Distribution of patients according to Neurological Signs	74
12	Distribution of patients according to ODI score before treatment	75
13	Distribution of patients according to VAS score before treatment	76
14	Distribution of patients according to Levels	77
15	Distribution of patients according to Radicle affected	78
16	Distribution of patients according to Complications	79
17	Mean Scores of SLRT Score before and after treatment	80
18	Correlation of SLRT Score before and after treatment	80
19	Paired t test showing mean difference of SLRT score before and after treatment	81
20	Mean scores of ODI	81
21	Correlation of ODI Score before and after treatment	82

22	Paired t test showing mean difference of ODI score before and after treatment	82
23	Mean scores of VAS	83
24	Correlation of VAS Score before and after treatment	83
25	Paired t test showing mean difference of VAS score before and after treatment	84
26	Distribution of patients according to Patient satisfaction	85
27	Comparison of ODI score	86
28	Association of ODI score before treatment with Patient satisfaction	87
29	Association of ODI score after 1 month with Patient satisfaction	87
30	Association of ODI score after 3 month with Patient satisfaction	88

LIST OF FIGURES

Fig.No.	FIGURES	Page No.
1	Spinal column	11
2	Spinal curves pattern	12
3	Lumbosacral canal contents(coronal view)	13
4	Spinal canal contents	14
5	Spinal Nerve roots	14
6	Intervertebral Disc	15
7	Annulus fibrosus(Fibers alignment)	16
8	Nucleus Pulposus	17
9	Nourishment of Disc	18
10	Nerve supply of Disc	19
11	Stages of disc herniation	20
12	Stages of disc herniation(axial view)	21
13	Epidural space	23
14	MRI Intervertebral disc prolapse(axial section)	38
15	MRI Intervertebral disc prolapsed(Sagittal section)	38
16	Fluroscopic Anterio Posterior View Interlaminar approach- Needle Placement	51
17	Fluroscopic Lateral View Interlaminar approach Needle Placement(A),Contrast Injection(B)	52
18	Caudal approach Fluroscopic View-Lateral(A), Anterio Posterior(B)	53
19	Local infiltration	60
20	Epidural needle	60
21	Loss of resistance technique	61
22	Steroid injection	61
23	Lateral posture-Epidural injection	62

LIST OF CHARTS

Sl.No	CHARTS	Page No.
1	Bar diagram showing Age distribution of patients	65
2	Pie diagram showing sex distribution of patients	66
3	Bar diagram showing distribution of patients according to Occupation	67
4	Pie diagram showing distribution of patients according to Side of sciatica	68
5	Bar diagram showing distribution of patients according to Duration of symptoms	69
6	Bar diagram showing distribution of patients according to previous treatment	70
7	Bar diagram showing distribution of patients according to Co-morbid conditions	71
8	Pie diagram showing distribution of patients according to Spasm	72
9	Bar diagram showing degree of SLRT before treatment	73
10	Bar diagram showing distribution of patients according to Neurological signs	74
11	Pie diagram showing distribution of patients according to ODI score	75
12	Bar diagram showing distribution of patients according to VAS Score before treatment	76
13	Bar diagram showing distribution of patients according to Levels of lesion	77
14	Bar diagram showing distribution of patients according to Radical affected	78
15	Distribution of patients according to Complications	79
16	Bar diagram showing distribution of patients according to Patient satisfaction	85
17	Comparison of ODI score	86

Introduction

1.INTRODUCTION

Back pain and related symptoms rank among the second most frequent medical complaints. Disability from low back pain is second only to the common cold as a cause of lost work time and is the most common cause of disability in people under 45 years of age.¹

Chronic back pain represents an important health problem in orthopaedic practice. The number of patients presenting with low back pain constitute nearly 80% of OPD practice. Patients presenting to us are of rural background involved in heavy physical labour. Low back pain temporarily disables them in earning a livelihood. So an approach has to be worked out to alleviate their symptoms early. Multiplicity of causes and difficulties in its treatment render low back ache one of the most frequent problems that a orthopaedic surgeon encounters. Decisions regarding optimal management are not easy. There are many therapeutic interventions available; however, none seems to be clearly superior². At one end of the spectrum is the regime of bed rest and analgesics, forming the main pillar of the treatment while at the other, there is the operative removal of the prolapsed disc. Even in the conservative line of treatment, there is no universally accepted policy and the best form of treatment. This wide and haphazard spectrum of treatment suggests that there is no single satisfactory method of treatment that ensures permanent and long lasting cure .³

Radicular pain defined as pain that radiates from the site of a pinched nerve in the low back to the area of the body aligned with that nerve, such as the back of the leg or into the foot⁴. Radicular pain occurs due to an ectopic discharge physiologically generated in either the dorsal root or the dorsal root ganglion. An important cause of radicular pain is mechanical compression exerted by degenerative changes in the facet joint, the posterior longitudinal ligament or the herniated disc. Another cause is

chemical irritation produced by phospholipase A2 or substance P secreted from the prolapsed intervertebral disc^{5,6}. As a result, they contribute to pain mechanisms by triggering venous congestion and/or neural edema around the nerve root, for this reason, the local delivery of steroids seems to be a rational option.⁷

Steroids inhibit the inflammatory response caused by chemical and mechanical sources of pain. They inhibit the formation of nerve root edema, have an anti-inflammatory effect, increase blood flow to neural elements thus improving ischemic neuritis and block conduction in nociceptive nerve fibers⁸. Steroids also work by reducing the effect of the immune system to react to inflammation associated with nerve damage⁹. The purpose of epidural steroid injections is not to cure anatomic abnormalities but to improve symptoms. In this way, patients can commence rehabilitation, allowing a quicker return to a “normal” lifestyle with maximum function and activity.¹⁰

Objectives

2. OBJECTIVE

To assess the efficacy of epidural steroid injection on pain relief and functional return in low backache with radiculopathy.

*Review of
Literature*

3. REVIEW OF LITERATURE

Historical Review:

Backache & sciatica are symptoms that have been recorded for centuries, but their common pathology & relationship has been recognized only recently. Various related points were known in isolation, but it was left to Mixter & Barr¹¹ in 1934, to correlate them & put them in a comprehensive form.

The first attempt to correlate sciatica with backache was in 1867, by Lasegue who described the posture & gait in sciatica, & also devised the Sciatic nerve stretch test. In 1888, Charcot described the spinal deformity associated with sciatica, & Brissot in 1890 called it Sciatica Scoliosis.

Early in the twentieth century, Goldthwait noticed a sudden development of cauda equina lesion, in a patient under treatment for lumbosacral strain. He attributed the symptoms to subluxation at the lumbosacral joint, & suggested that it was posterior displacement of the discs. The same year, Middleton & Teacher described sudden paraplegia in a man due to disc retropulsion while lifting heavy weight.

In 1916, Sicard put forth his theory which stated that sciatica was due to irritation of the nerve roots, which he termed as neurochorditis. His theory of root irritation was supported by Putti (1927), who felt that the lesion commonly was at the intervertebral foramen & due to arthritis of the facet joints following variations in their planes.

In the late 1920's, Schmorl with Junghans, described the pathological anatomy of the spinal column & intervertebral discs, & that the herniation of the disc can occur in any direction, including posteriorly into the spinal canal, & anteriorly into the prevertebral region. During the same period Dandy confirmed by his studies

that the tumors earlier described by Elsberg were in fact cartilaginous loose fragments extruded from the disc.

In 1934, Mixter & Barr summarised the previous knowledge regarding disc lesions & they suggested surgical removal for relief of the symptoms. They documented their findings in 30 clinical cases with unilateral sciatica. In 1941, Dandy described the “Concealed Disc” or intermittent herniation of the nucleus pulposus. Key, Burns & Young pointed out that, disc herniations could produce low back pain without sciatica. Charnley suggested fluid imbibition; Scott stated the factor of mental stress, & Lindblom suggested compression as a causative factor. With these postulations, factors other than mere trauma came to receive increasing attention, & aided in establishing the present view of multifactorial origin of low back pain & sciatica.

Evolution of Epidural Injections:

In 1855, Wood of Edinburgh popularized the use of a hollow needle, which has been described 10 years earlier by Rynd of Dublin, & the use of the glass syringe, which has been devised by Paraviz of France. Introduction of the syringe & needle into clinical practice proved a big milestone in the management of many ailments & disease disorders, which were treated by injection of opiates, chloroform, bromides, etc., near the nerve trunks.

Leonard Corning¹² used the epidural space in 1885 to produce spinal analgesia with injections of cocaine in the dog & subsequently used the same in man for seminal incontinence & spinal weakness.

The first epidural injection was reported in 1901, Sicard¹³ employed extradural cocaine by the caudal route to treat cases of lumbago & sciatica. He subsequently described the interspinous approach to the epidural space. During this period, there

were various attempts to establish epidural injection. One such attempt was made by Caussade & Queste in 1909, reviewed several cases of sciatica relieved by spinal injection of stovocaine, but their selection criteria & technique cases & description of technique were vague. Page¹⁵ described the technique of Lumbar epidural analgesia. The technique was refined in 1925 when Viner¹⁰ reported injecting 20 mL of 1% procaine in 50 to 100 mL of Ringer's solution, normal saline.

Evans described treating sciatica with epidural injections from a caudal approach in 1930. He administered 1% novocaine, often over 100 mL, to most of his patients. He reported such side effects as, abnormal sensations or paraesthesiae, such as formication. A few patients said that they had found it difficult to control the desire to shout or scream.” Despite these effects, he reported that 22 of the patients were cured, and another 5 were improved.

Dogliotti¹⁶ (1933), an Italian described at greater length the technique of lumbar epidural injections, & described the loss of resistance sign.

Around 1950, Cyriax described the use of epidural injections with procaine as a diagnostic test of differentiate between lesions outside the canal & those inside it, and at the same time noted its therapeutic value. He later laid down compressive indications for selection of patients & advocated epidural anesthesia as the conservative treatment of choice in patients of low lumbar disc lesions with nerve root pressure & neurological signs, the one contra indication being the presence of bladder symptoms.

Lievre et al¹⁹ administered hydrocortisone into the epidural space via an S1 foraminal approach in 46 patients with sciatica in 1953. They described 8 patients with a very good response, 15 patients with a good response and 8 patients with a moderate response. The first report of epidural steroid injections in the United States

was in 1961, when Goebert et al^{10,17} described using 3 daily injections of 30 mL procaine and 125 mg hydrocortisone acetate. Barry & Kendal¹⁸ in 1962 got similar results as Goebert with the use of cortisone.

The largest study of the 20th century came from Swerdlow & Sayle-Creer in 1970, who described a series of 5000 cases of backache. They were treated with saline & lignocaine epidural injections, saline injections only, & injections containing lignocaine with methyl prednisolone, through the lumbar & caudal routes and were followed up for at least 12 months. They conclude from their studies that in time of recovery from severe pain, that hospitalization or long periods of rest should be avoided & physiotherapy should be started early. They stated that the epidural injection may avoid the need for surgery.

There was more research with different combinations by Pamela Daly, Beliveau²⁰ (1971) & Warr et al²¹ (1972). The latter opined though the epidural injection is not the cure-all of any back pain, it is the best method currently available, short of laminectomy, for all cases of syndrome, young or old, mild or severe, acute or chronic, but for the contraindications. They concluded by saying that the ability to achieve in hours what may other-wise take weeks or months, commends this form of treatment.

Dilke, Burry & Grahame²² in 1973, published the results of a study of a doubleblind controlled trial on 100 cases given epidural corticosteroid injections by the lumbar route & 100 control cases. They reported statistically highly significant differences with respect to relief of pain & resumption of normal duties in favor of the group treated by extradural injections & feel that it seems to be a valuable adjunct to the management of lumbar nerve root compression syndromes, associated with degenerative disc disease.

In 1977, Dr. R. K. Sharma has reported on a series of 201 cases of low back pain with sciatica, given 40 ml of saline, 0.5% lignocaine hydrochloride with 80 mg of methylprednisolone via the caudal route he obtained good to very good results in 56.2%, fair in 23.9% & no improvement in 19.9% of cases. In the same year Brevick et al²³ discussed a series of 53 low backaches with sciatica cases which they treated with Caudal Epidural injections using Depomethylprednisolone & Bupivocaine with excellent long & short term results.

In 1997 Carette et al²⁴ reported their results in the New England Journal of Medicine on a 158 cases with disc prolapse & radicular pain, given methyl prednisolone (80 mg) & saline (8 ml). They remarked significant improvement following these lumbar injections & reported a good short term follow up.

In the beginning of the twenty first century, Manchikanti et al²⁵ reported a case series of 62 patients with discogenic pain, who were given caudal epidural injections & remarked a positive short as well as long term follow up. In 2004, Elva G. Delport et al²⁶ reported a series of 140 patients concluding that majority of the patients were satisfied with epidural steroid injections as a form of treatment in assisting them through the more painful periods of their condition, although many required reinjections for periodic flare ups over the 3 year period. In 2005, Arden et al²⁷ reported on a series of 228 cases of low back ache with unilateral sciatica, given 80mg of Triamcinolone Acetate & 10 ml of 0.25 % Bupivocaine via the lumbar route they obtained excellent results in the short term. At the same time, Wilson-McDonald et al²⁸ reported on a series of 93 patients with MRI evidence of a disc prolapse, spinal stenosis, with lumbosacral nerve root pain which had not resolved within a minimum of 6 weeks.

Ackerman et al²⁹ published their findings in discogenic pain using different routes of epidural injections in 2007. Salahadin Abdi et al³⁰ in a study conducted in Miami stated that there is limited evidence in the lumbar spine for long-term relief by interlaminar lumbar injections while both lumbar transforaminal & caudal had moderate long-term relief in managing nerve root pain & chronic low back pain. In 2007 Kenneth Botwin³¹ concluded that Fluoroscopically guided caudal epidural steroid injections may help reduce bilateral radicular pain and improve standing and walking tolerance in patients with Degenerative Lumbar Spinal Stenosis.

Abdi et al³² once again evaluated the effect of lumbar interlaminar epidural injections with or without steroids in managing various types of chronic low back & sciatica. In 2009, Sayegh et al³³ reported a case series of 193 cases with low back pain & sciatica. They evaluated these cases after using Caudal Epidural injections with or without steroid & concluded that steroid containing preparations demonstrated better & faster efficacy.

In 2010 Digambar Prasad Nawani³⁴ reported a case series of 50 patients concluding pain relief was very good in 37 (74%) patients while good response was seen in 9 (18%) patients and no significant pain relief was seen in 4 (8%) patients following epidural injection of 15 ml (10 ml bupivacaine + 2 ml tramadol (50 mg/ml) + 100 mg depomed (40 mg/ml & 20 mg/ml). Jafar Mobaleghi et al³⁵, 2011 in a prospective, single-blind uncontrolled study, 60 patients with radicular pain due to lumbar spinal stenosis or herniated disks who underwent Epidural Steroid Injection and were observed for 6 months, concluded that Epidural methylprednisolone injection has less analgesic effect in patients with Lumbar Spinal Stenosis compared to Herniated Disc, with less permanent effect. In 2012 a study conducted by Laxmaiah Manchikanti et al³⁶, involving 120 patients concluded that lumbar interlaminar

epidural injections, with or without steroids, are effective for managing chronic function-limiting low back pain and lower extremity pain secondary to lumbar spinal stenosis. In appropriately selected patients, significant functional status improvement and pain relief can be achieved with approximately 4 injections a year.

Anatomy:^{37,38,39,40}

The central axis of the human skeleton is formed by the vertebral column. At its proximal end through two modified vertebrae – the Atlas & the Axis it supports the skull. In the thoracic region it articulates with the rib cage which, in turn articulates with the pectoral girdle & upper limbs. Finally through sacral vertebrae, it articulates with the pelvic girdle to which the lower limbs are attached.

Kester has introduced the concept of the functional spinal unit, which consists of:

(a) Two adjacent vertebrae with their articulations

(b) An intervening Disc

(c) Ligaments

-Anterior Longitudinal Ligaments

-Posterior Longitudinal Ligaments

-Ligamentum Flavum

-Interspinous Ligaments

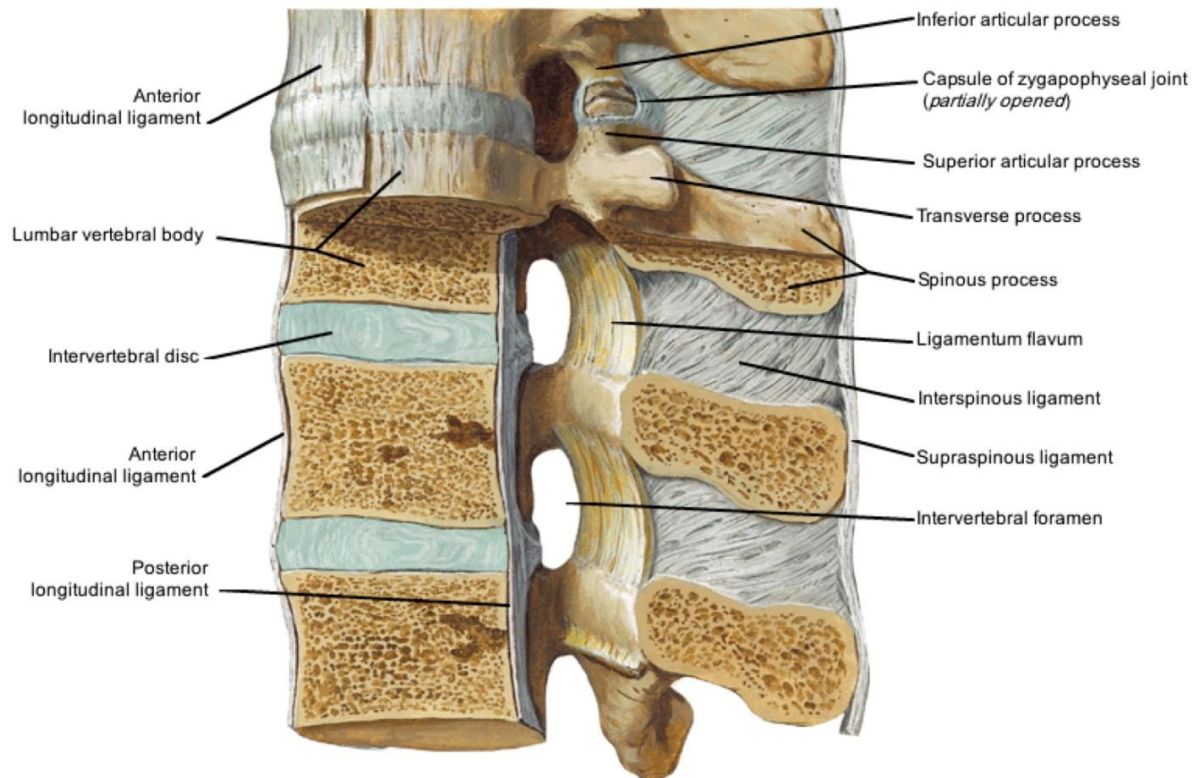


Fig 1: Spinal Column

In the position of normal stance, balance is maintained by intrinsic structural stability & supported by the tone of the various postural muscles.

The weight of the trunk is centered over its base by three curves :

- (i) Cervical Lordosis
- (ii) Thoracic Kyphosis
- (iii) Lumbar Lordosis

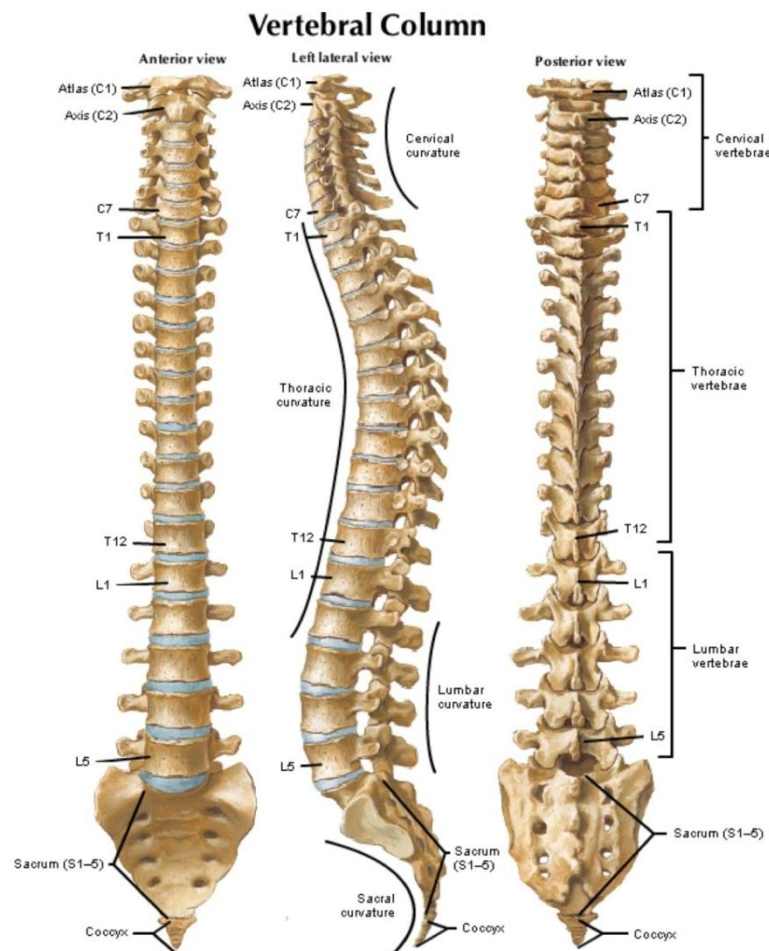


Fig 2: Spinal Curve Pattern

Spinal Canal :

Spinal canal is formed by superimposition of vertebrae, bounded ventrally by the dorsal surfaces of the bodies of the vertebrae, intervertebral discs, & the posterior longitudinal ligament. Dorsally & laterally it is bounded by pedicles, laminae, transverse processes & spinous processes, & the ligamentum flavum, which can be quite thick at times. The posterior longitudinal ligament is less well developed over the lower two vertebrae, which is loosely attached to the bodies but firmly attached to the inter-vertebral disc.

Contents – Lumbo - Sacral Spinal Canal:

- (a) The Dural Sac containing the spinal cord & the nerve roots, which ends at S2– S3, & becomes filum terminale
- (b) Cauda Equina.
- (c) Cerebro-spinal fluid.
- (d) The Epidural Space-This is wider at the dorsal side as a result of the dural sac lying more closely against the cerebral bodies. It contains fat, connective tissue, venous plexuses, & the emerging spinal roots.

The spinal cord ends between L1 & L2. From here, the dural sac contains only the lower nerve roots & the conus medullaris, which together form the cauda equina.

Protection of the delicate nervous system from shocks is provided by:

1. The Bony Spinal Canal
2. Cerebrospinal fluid
3. Epidural cavity

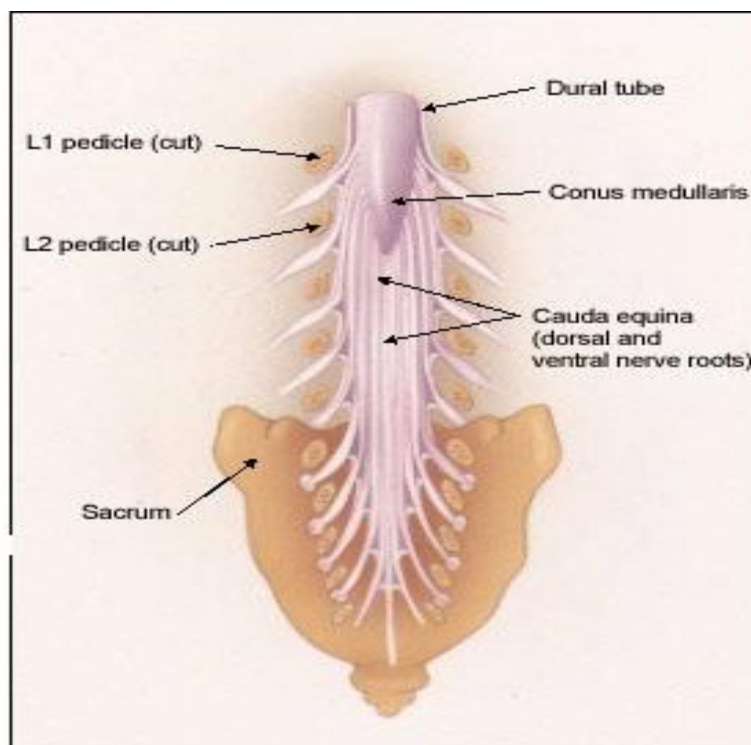


Fig 3: Lumbo Sacral Canal Contents(Coronal view)

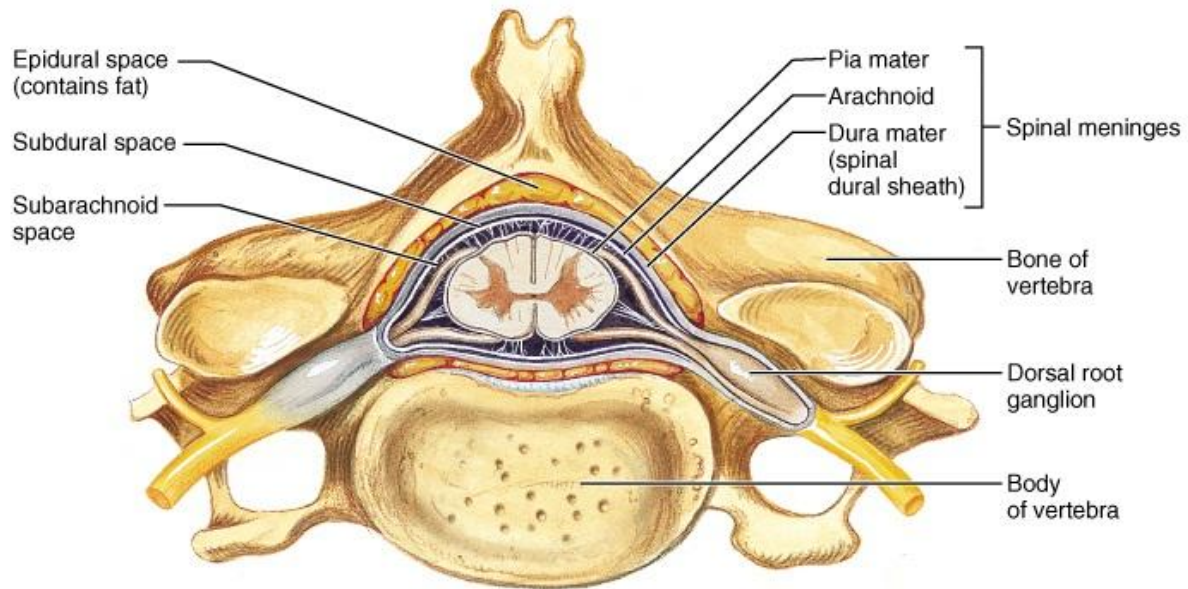


Fig 4:Spinal Canal Contents(Axial view)

Spinal Nerve Roots :

The lumbar nerve roots emerging from the dural sac reach the intervertebral foramina after descending obliquely in the sulci lateralis & along the pedicles. From the point of exit from the dural sac they are surrounded by the dural sheath. They are susceptible to be compressed the sulci lateralis as there is little room for displacement.

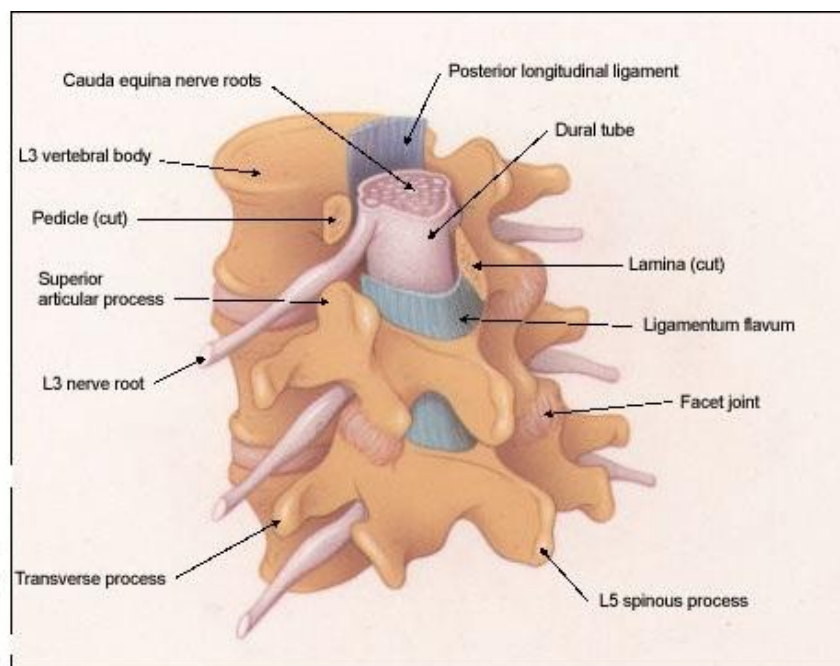


Fig 5:Spinal Nerve Roots

Intervertebral Foramina:

These are formed by two adjacent vertebrae, the dorsal boundary of which is formed by articular processes both superior & inferior, their joint capsules, & ligaments. The ventro-medial boundary of the foramen is bounded by the bodies of adjacent vertebrae & the intervertebral disc. L5 root is more likely to be compressed because of the anatomical peculiarities of the intervertebral canal, through which the root passes.

Due to longer, narrow & oblique canal formed, the nerve root remains in close contact with the disc over a greater distance. In lateral prolapse of the disc, there will be little chance for the nerve to escape from pressure. This is in contrast with the other intervertebral canals, which are roomy & less oblique.

The Intervertebral Discs:

The discs contribute to a third or fourth of the length of the articulated vertebral column, & variations in their thickness anteriorly & posteriorly play a great role in maintaining the primary & secondary curves of the column. Each intervertebral disc consists of **two plates of hyaline cartilage**, united by a ring of fibrous tissue, the **annulus fibrosus**, in the middle of which lies the **nucleus pulposus**.

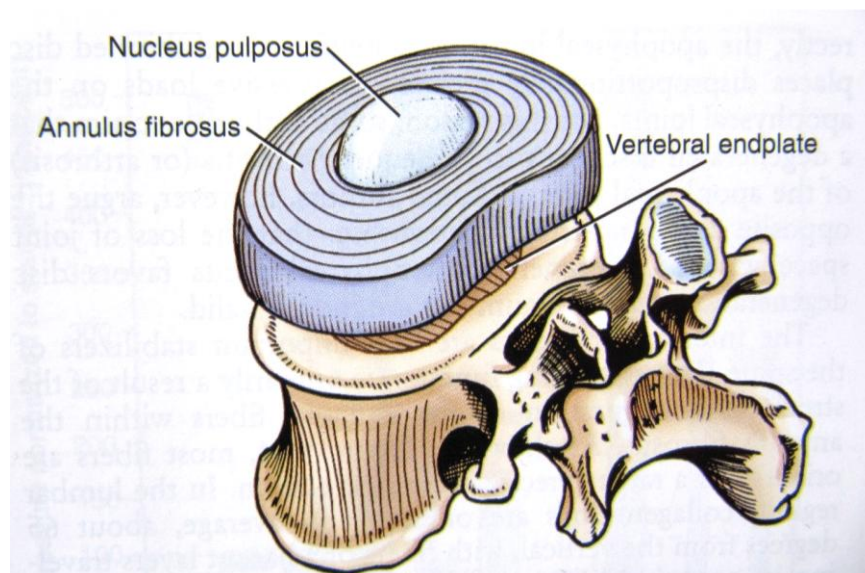


Fig 6: Intervertebral Disc

The plates of **hyaline cartilage** are 1 mm thick & are placed horizontally. They are attached to the bodies of adjacent vertebrae. On completion of skeletal growth, they fuse with the surrounding epiphyseal ring. Until the age of about 30 years they contain vascular channels which later disappear & are replaced by scar tissue.

The **annulus fibrosis** forms a rigid firm bond between the vertebral bodies by being attached to the vertebral margins & to the anterior margins of the cartilage plates. It consists of fibrous tissue arranged in 10-12 concentric laminae. The fibers of the laminae are all placed obliquely at an angle of about 45 degrees to the adjacent vertebral bodies; but the layers contain fibers lying alternately in the 45 degrees slope, at right angles to each other, thus offering it enough strength to withstand strain in any direction. Some fibers are attached to the anterior & posterior longitudinal ligaments & are reinforced by them. Puschel (1930) stated that its water content is 78% at birth, reduces to 70% in the third decade, & remains fairly constant thereafter.

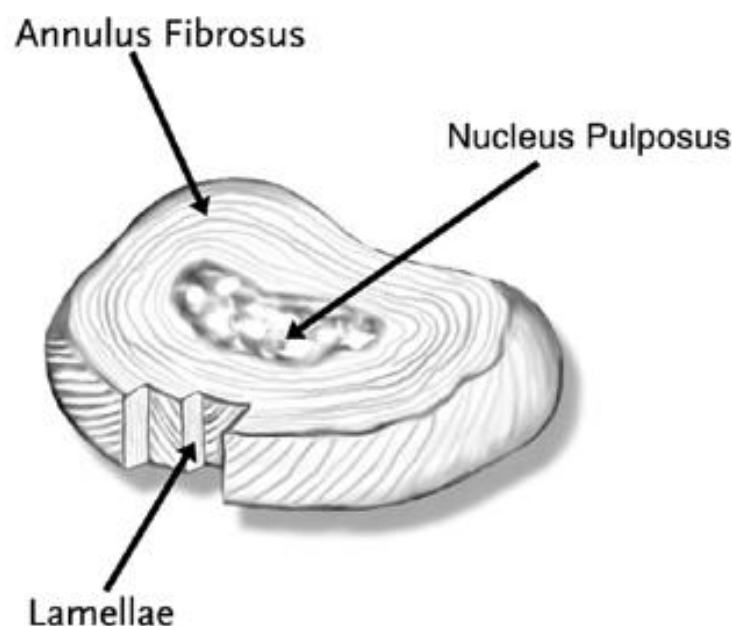


Fig 7:Annulus Fibrosus(fibers alignment)

The **nucleus pulposus** is a greyish white, translucent, & semigelatinous material consisting of a polysaccharide/protein gel, in which are interspersed collagen fibrils, & cartilage cells. The notochordal cells usually disappear by 10th year followed by gradual increase in fibrous tissue. Water content gradually reduces from about 88% at birth to about 67% by age of 70 years. Due to water imbibition during the night, man is 1-2 cms taller in the morning than at night. The intradiscal pressure varies with posture, time of day & age. As the nucleus pulposus is situated not centrally but a little posteriorly the annulus fibrosis is thinner posteriorly.

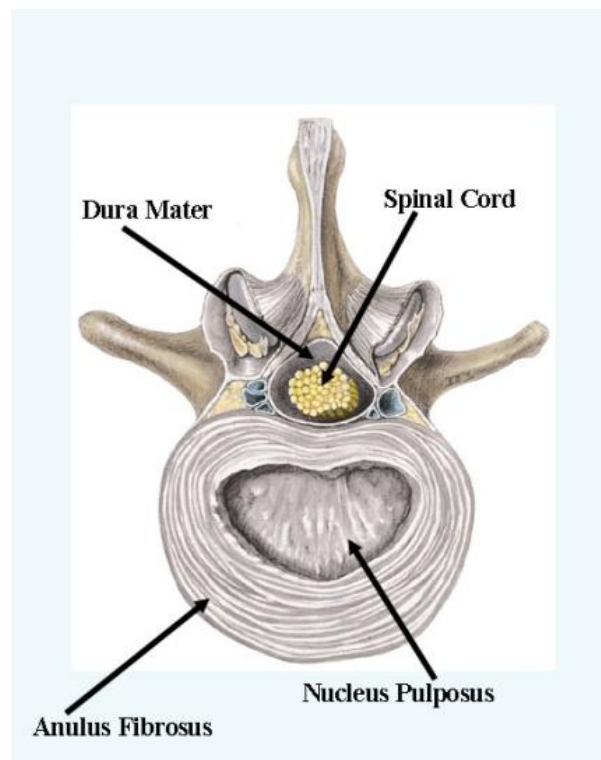


Fig 8:Nucleus Pulposus

The **nourishment** of the disc is through the lymph which diffuses from the marrow cavity of adjacent vertebrae, through perforations in the cartilage plates. Some investigators period suggests their existence through adult life. The pattern of the vascular supply of the annulus fibrosis has interested some workers, such as

Larcher, Prader & Tondury, 1947, who described that annulus is permeated by blood vessels almost exclusively at the posteriolateral corner. Over the period of growth these are replaced by fibrous tissue which leads to decreased resistance at this site. According to Thurel, fibers in the posterior part of the annulus fibrosis are less stronger than in ventro-lateral part, another factor also tending to weaken the annulus in this region.

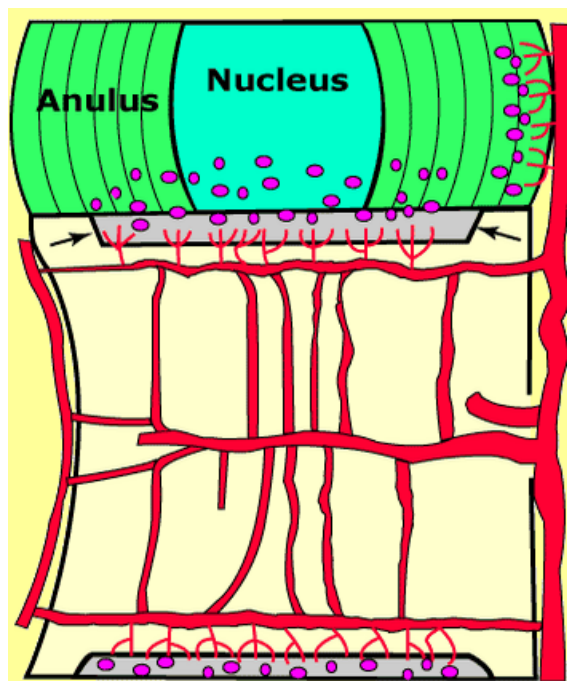


Fig 9:Nourishment of Disc

The **nerve supply** of the disc is from sinu vertebral nerve which is a branch from posterior primary division, reenter the spinal canal and supply even the posterior longitudinal ligament.

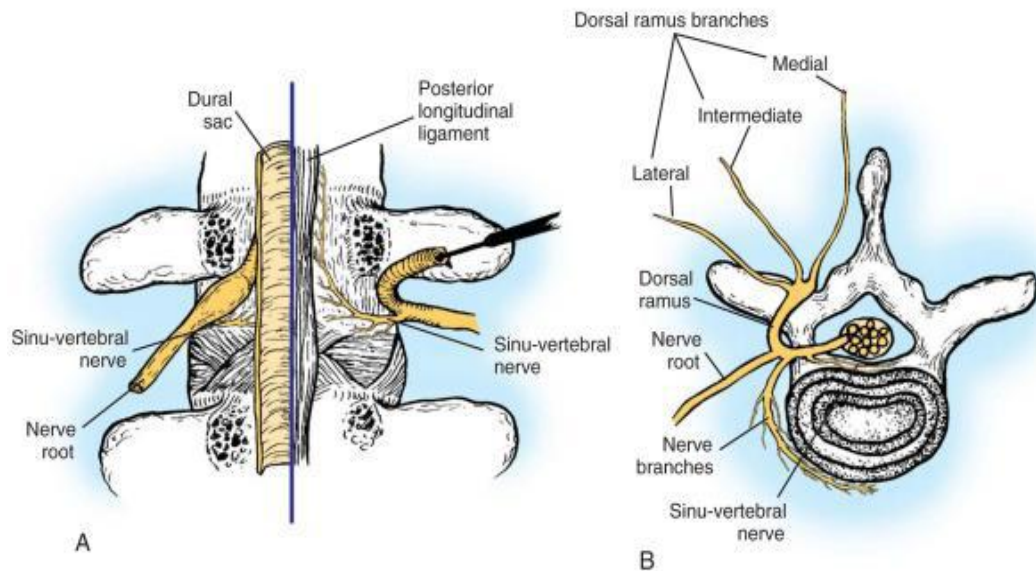


Fig 10: Nerve supply of Disc

The intervertebral disc, as a whole acts as an excellent shock absorber, a vertical compressive force while walking produces centrifugal forces in nucleus, in flexion and extension of spine annulus compression on concave side is prevented in a similar manner.

Types of Disc Lesions

There is no clear-cut distinction between degenerative changes due to age & pathological conditions of the intervertebral disc. The various disc lesions are classified by **Bartschi** as follows:

1. **Atrophia Simplex**, which is the simplest form of primary degeneration of the disc, which may or may not be characterized by persistent low back pain.
2. **Protrusion**, which is characterized by protrusion of the disc through the weakness of the annulus fibrosis & consequently resulting in stretching of the sensitive posterior longitudinal ligament. It is accompanied by severe low back pain & sciatica & other radicular compression symptoms, depending on whether or not the nerve roots are pressed. In this stage, there is also new cartilaginous tissue formation of the margin of

the vertebral body, probably as a result of irritation, when it ossifies & forms osteophytes.

3. **Extrusion**, in this stage, there is complete eruption of the nucleus pulposus through the annulus fibrosis, whereby it comes to lie on the posterior longitudinal ligament. Two types are recognized.

(a) The mobile type (concealed ruptured disc or intermediate prolapse). In this type, the prolapsed portion returns to the same tear intermittently

(b) The fixed prolapse, when the prolapsed nucleus can no longer be reduced.

4. **Sequestration** occurs when the nucleus pulposus leaks out through a tear in the annulus fibrosus and separates from the disc itself. The portion of disc material that has detached may exert pressure on nearby spinal nerves, causing symptoms of pain, tingling, numbness, or weakness throughout the muscles and skin innervated by that nerve. It is also possible that the sequestered disc material will be broken down and resorbed back into the blood stream.

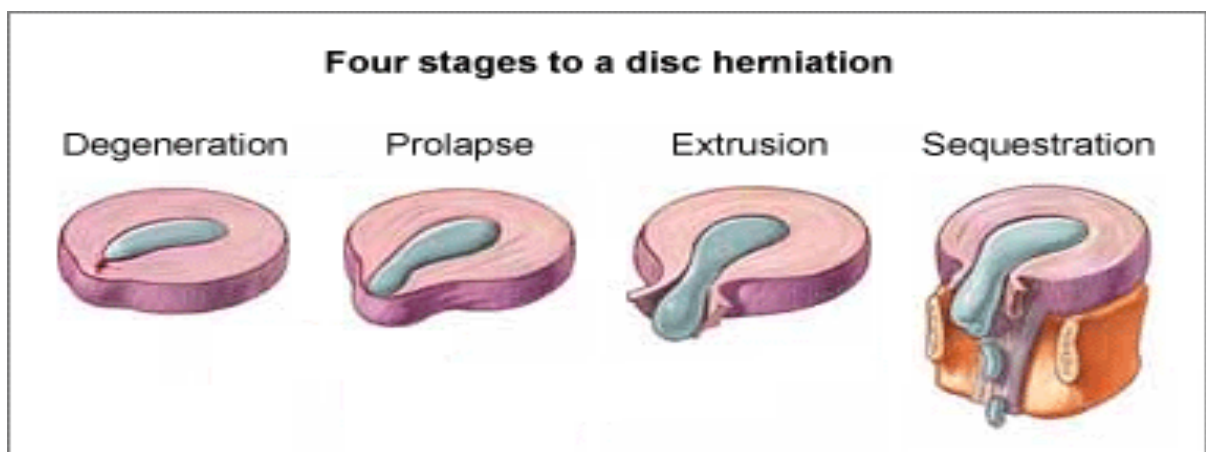


Fig 11: Stages of disc herniation

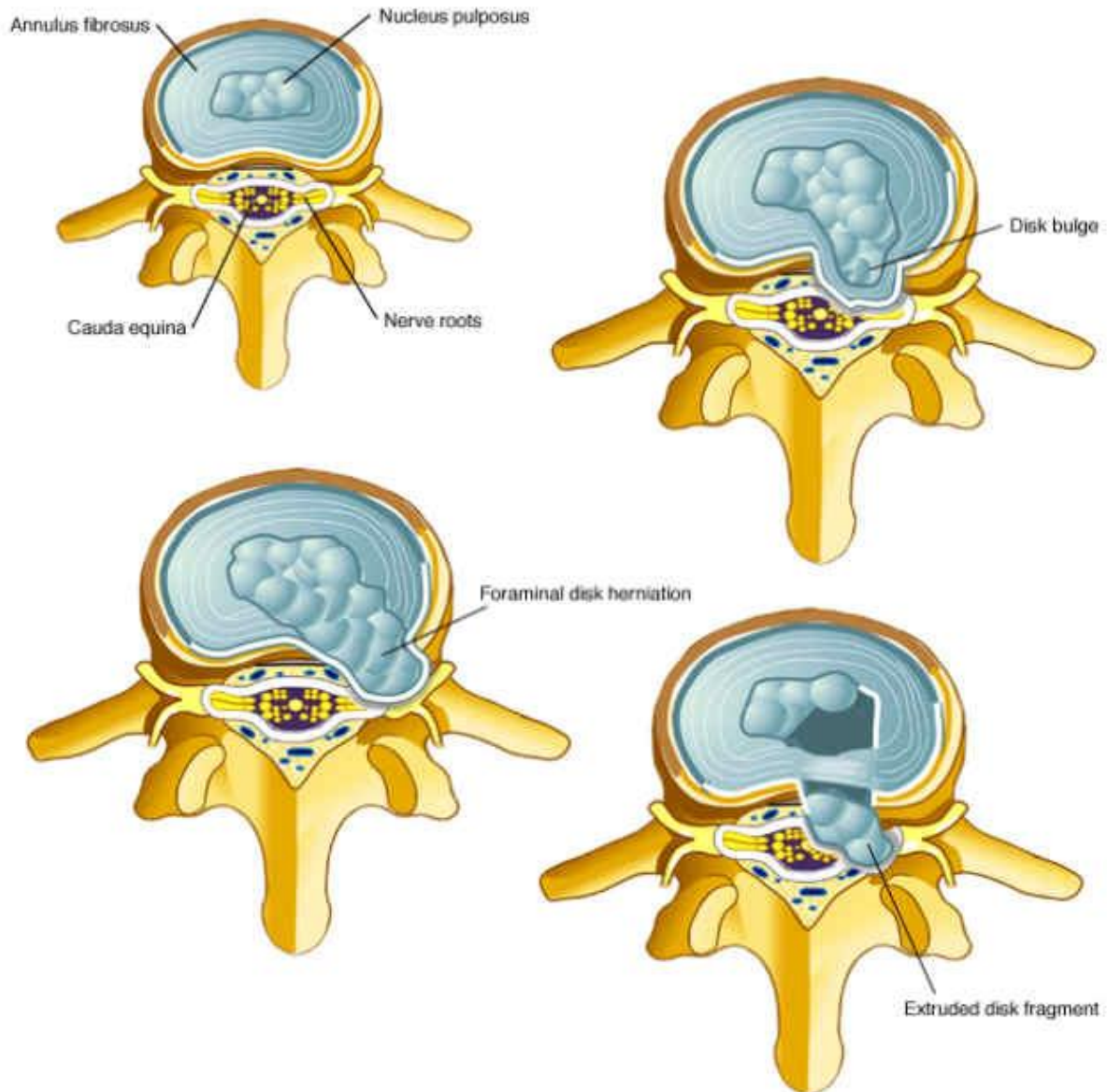


Fig 12: Stages of Disc Herniation(axial view)

Presentation of Disc Protrusion

- (a) The commonest protrusion is fairly well circumscribed bulging of the disc, yellowish or white glistening covered with attenuated annulus fibrosus or with soft elastic summit.
- (b) Less commonly free lying completely extruded disc material in the epidural space, which may or may not be embedded in dense fibrous tissue.
- (c) Intermittent herniation of Falconer, or concealed disc of Dandy.

Nerve Sensitivity of the Structures of the Intervertebral Canal

The pain secondary to disc protrusion may be either due to irritation, be it chemical, mechanical or auto-immune, of the adjacent direct irritation of the inflamed nerve or even a combination of all the factors.

The **pain sensitive tissues** in this area are:

- (a) Anterior longitudinal ligament
- (b) Vertebral bodies
- (c) Posterior longitudinal ligament
- (d) Capsule of the intervertebral joint
- (e) Nerve roots & the muscles
- (f) Inter-spinous ligament – this may or may not contain pain fibers.

The **non-sensitive tissues** are:

- (a) Ligamentum flavum
- (b) Annulus fibrosis & nucleus pulposus of the disc
- (c) Inter-spinous ligament – this may or may not contain pain fibers

The Epidural Space-

The epidural space is a potential, elliptical, or annular space between the spinal dura & the bony vertebral canal.

Formation:

In the cranial cavity, the dura is arranged in two layers, the periosteal & investing layers, which are finally adherent to each other except where they split to enclose the venous sinuses. The outer periosteal layer is the periosteum of the inner surface of the skull which in spine, acts as the periosteum of the spinal canal. The inner investing layer is continued on from the brain in the cranium to the spinal dura. The space is therefore present only in the spinal canal.

Boundaries & Extent:

Its upper boundary is the foramen magnum, inferiorly at the end of the sacral canal. It ends where the hiatus is closed by the sacro-coccygeal membrane. Anteriorly, the space is bounded by the posterior longitudinal ligament, laterally by the pedicles of the vertebral laminae. It communicates laterally with the paravertebral space through the intervertebral foramina. Since a few fibrous bands hold the dura against the posterior longitudinal ligament, there is very little of epidural space there.

Contents:

Contents of the epidural space include the dural sac & spinal nerve roots, & the extradural plexus of veins, spinal arteries, lymphatic's & fatty tissue. The extradural plexus of veins, also called the venous plexus of Batson, are relatively large, thin walled, & contain no valves. They are arranged in 4 vertical channels, two on either side of the posterior longitudinal ligament & two in front of the vertebral arches, & are interconnected by venous rings at each segment level. At each segment, they receive the basivertebral veins from the spinal cord. Serially they also receive communicating branches from the vertebral, cervical, deep cervical, intercostals, lumbar, ilio-lumbar & lateral sacral veins through the intervertebral & sacral foramina. They form connecting limbs with the cerebral veins above & the pelvic below.

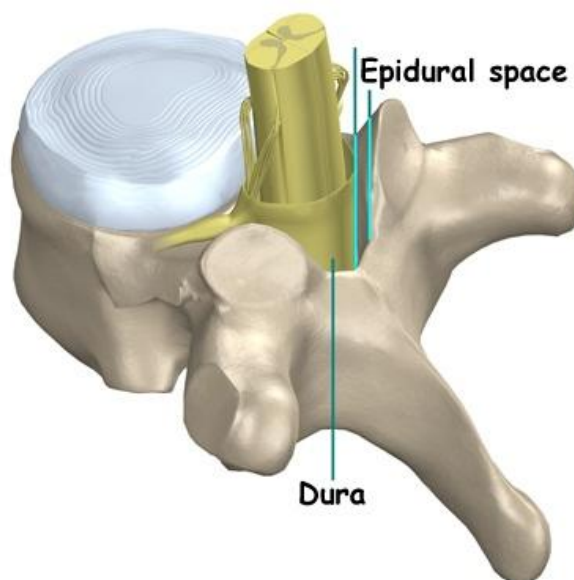


Fig 13:Epidural space

Negative Pressure in the Epidural Space:

Though the epidural space was first used in 1885 by Corning to inject cocaine & produce analgesia in dogs, the sacral approach used in 1901 by Sicard & Cathelin, and the interspinous approach by Sicard & Forrester in 1906, it was Janzen who first described the negative pressure in the epidural space in 1926. Negative pressure is said to exist in extra dural space. This, so called negative pressure is greatest at point of firm attachments. It is greatest in thoracic region (1-3cm H₂O), less in lumbar region (1cm H₂O) and least in sacral region (0.5cm H₂O). This difference in pressures make hanging drop technique at thoracic region and loss of resistance technique at lumbar region the preferable methods of identifying the epidural space.

Transmission theory:

Negative epidural pressure is created from transmission of intrapleural negative pressure through the intervertebral foramina. Clinically negative pressure in epidural space decreases on marked flexion and in patients who are tense and straining. While decreased subarachnoid pressure will increase negative epidural pressure.

Sacral Canal

It is the continuation of the vertebral canal and naturally curved like the bone. Cross sectionally it is triangular at base and becomes flatter caudally. Anteriorly it is bounded by posterior longitudinal ligament and the body. Laterally and posteriorly it is bounded by fused pedicle and lamina.

At the caudal extremity, the lamina of the 5th, & often of the 4th sacral vertebra, fail to meet in the midline, leaving the sacral hiatus at the termination. The hiatus is covered by:

(a) Prolongations of the supra spinous ligament

(b) Superficial posterior coccygeal ligament, which serves as a roof

(c) Over the end of the sacral canal.

Methods of Identifying the Epidural Space¹⁶

Though there are a large number of methods, they are but variations in the methods enumerated below.

1. Dogliott's Method:

An 18 - 19 bore needle with a short bevel & blunt end is fixed on a syringe. As the needle is gradually advanced across the interspinous space, the point of **loss of resistance** to the injection of air is noted. This denotes entry into the space.

2. Hanging drop sign of Gutierrez

A needle similar to the one above is used, no syringe is attached to it, but a drop of liquid is placed in its hub. As the needle is advanced across the interspace, entry in to the epidural space is shown by the **drop of liquid being sucked in by the negative pressure**.

3. Odom's method

To the hub of the needle is attached a three way cannula. The second limb of the cannula is attached to the pre-flattened rubber balloon. Initially, the knob is kept in such a way that the two do not communicate with each other. As the tip of the needle traverses the interspace the knob is turned so that the balloon communicates with the needle. When the epidural space is encountered the **balloon collapses due to the negative pressure**.

4. Spring Loaded Syringe

This special syringe makes use of a spring on its position I such a way that it contracts as soon as the negative pressure of the epidural space is reached by the needle tip.

5. Whoosh Test

Once the needle has been placed in the epidural space, air is injected into the space & a **sound (gush of air)** can be auscultated with a stethoscope just above point of insertion of the needle.

Fluoroscopic Contrast Dispersion Method

Isohexol is used as a dye to document placement of the needle in the epidural space & also to study the dispersion of the steroid & the extent of its reach. The advantage is highly accurate needle placement & the study of dispersion pattern also helps to decide the extent. Disadvantages include the need of a fluoroscopic table & dye related complications .

Etiopathogenesis⁴¹

Sciatica denotes pain in the distribution of the sciatic nerve, but in many instances of sciatic pain due to involvement of the lumbosacral roots the pain involves only a part of the sciatic trunk and is felt only part way down the limb. The term is therefore descriptive but not accurate. In those cases associated with root syndromes also, the pain though felt along branches of the sciatic trunk is in point of fact root pain and the term sciatica is therefore misleading in the sense that it refers to pain along the peripheral trunk though its origin and distribution is in fact radicular.

Various causes include-Herniated lumbar disc, Degenerated discs, Sciatic neuritis, Spinal cord varices, Arthritis of spine, Protruding discs with adhesions, Hypertrophied ligamentum flavum, Lumbosacral strain, Chronic radiculitis, Spondylolisthesis, Defect of L5 pedicle, Inflammation around L5 lamina, Unstable lumbosacral mechanism, Suppurative periostitis of L4 vertebra, Spinal cord

tumour⁵³. The predominance of herniated disc over all other causes of sciatica reaches as high as 90%, or in some instances even higher percentages.

According to the causative factor it is classified as:

1) Viscerogenic Sciatica:

This type of neuralgia is due to visceral causes, e.g. diseases of the bladder, prostate, ovaries, or of structures like joints, ligaments & muscles. The main features of this type of neuralgia are that neurological disturbances do not correspond to known patterns of nerve distribution & there are no objective neurological signs.

2) Sciatic Neuritis:

In this form, it is a manifestation of either a general systemic disease like diabetes or syphilis, or of a generalized toxemia like alcoholism, lead & arsenic poisoning etc. It can be diagnosed by other signs & symptoms of the underlying disease or toxemia.

3) Secondary Sciatica:

This is due to peripheral neuritis & is due to pressure on the nerve, which may be.

- a) Outside the spinal canal such as by pelvic tumors
- b) Non-disc lesions inside the spinal canal such as extra/intra-dural masses
- c) Intervertebral disc lesions

The etiological factors as well as pathophysiology can be conveniently considered under the following headings though the division is but arbitrary & each cannot be separated from the other:

- a) Mechanical causes
- b) Changes in the annulus fibrosis
- c) Changes in the nucleus pulposus
- d) Changes in the adjacent structure

Observations of O'Connell (1951 - 500 Cases)⁴² & Armstrong (1958 -10,000 Cases) also bear out a higher incidence in the lower discs with the L 5 – S 1 accounting for almost 50 % of the cases. According to Roaf (1960)⁴³, disc degeneration is more common in the lumbosacral region due to the facts enumerated in the earlier section, due to greater range of mobility & a higher frequency of congenital anomalies.

Changes in the Annulus Fibrosus:

It has been stated earlier that the annulus is narrower posteriorly. Malmos points out that the posterior longitudinal ligament also is not as well developed in the lower lumbar region as higher up, at the same time being narrower. Larcher, Prader & Thubury (1947) described vessels permeating the posterolateral aspects of the annulus, & then branching out without penetrating the disc. According to them, these vessels disappear with growth, usually in the second decade, & are replaced by scar tissue. Weak points are thus formed..When, due to bad posture, abnormal stresses or other mechanical causes, the line of weight bearing lies in the posterior part of the disc & hence over the annulus instead of over the nucleus as in the normal, the annulus is weakened further. The nucleus being one-fourth of the total size of the disc in the lower lumbar region makes matters worse. It may be repeated minor stresses result in small tears which form weak spots (Lindblom, 1952, 1957) through which herniation of the disc is later facilitated. ⁴⁴

Changes in the Nucleus Pulposus:

Paulson, Sylven, Snellman & Hirsch et al⁴⁵ (1952) from their clinical, anatomical, microscopic studies stated that more than mere mechanical stress is responsible for the nuclear degeneration. The reduction in water content has already been noted. There is a decrease in the mucopolysaccharides & an increase in the proteins, which leads to a decrease in the water-binding capacity of the disc. As the protein content increases, there is a loss of delineation between the annulus & the nucleus. In such degenerated discs, pain perception increases.

Now, considering the changes in the annulus & in the nucleus together, the nucleus gradually escapes, from the confines of the annulus & herniation occurs.

- Anteriorly, No structures near enough to be affected hence no symptoms
- Posteriorly, which is the most important is considered in detail
- A third type of herniation is vertically into the adjacent vertebral bodies, which again produces no symptoms. It is commonly seen in adolescent Kyphosis & the radiological appearance is known as “Schmorl’s Nodes”

Further, the posterior herniation can occur in the midline (which is least common due to the presence of the posterior longitudinal ligament), paramedially or laterally. The more lateral the protrusion, the greater the occurrence of pain radiating down the lower limb. In a series of cases where clinical & radiological findings were correlated with the operative findings, it has shown that:

- Central protrusions mostly produce back ache & rarely radiating pain
- Lateral protrusions produce radiating pain more frequently than back ache
- The intermediate protrusions produce back ache & radiating pain

Clinical Application:

The poor localization & radiation of low back pain may well be related to the fact that each sino vertebral nerve & each posterior primary ramus supplies at least two levels. Entrapment of spinal nerves is an obvious cause of pain, sensory disturbances & muscle weakness. Irritation of these branches may be relevant in the pain of the disc degeneration & herniation especially in spinal stenosis.

On the other hand irritation of the posterior primary rami is a feature of segmental instability. In performing an inter-transverse spinal fusion, the posterior primary rami are nearly always sectioned. This may be one of the beneficial effects of this operation. Irritation of sino vertebral nerve may cause back pain & sciatica. This has special relevance to the variety of conditions that produce lateral recess narrowing at L 4-5 & L 5 -S 1 levels.

Production of Pain⁴⁶

There are various theories as to how pain is produced by the prolapsed intervertebral disc. These fall into the following groups:

Derangement of Mechanics of the Spine.

Inflammatory Changes around Nerve Roots.

Dural Origin.

Pressure on Nerve Roots.

The exact mechanism by which pain is produced is still a matter debate. Similarly, the intermittent nature of the symptoms is explained as due to one or more of the following factors:

- Rest offered by forced inactivity
- Partial or complete reduction of the disc (mobile)
- Healing of tears in the annulus fibrosis

- Desiccation & hence reduction in the size of extruded disc material
- Relief of compression of the nerve roots due to suppression of inflammatory reaction & venous stasis
- Adaptation of nerve roots to pressure & tension with lengthening.

Clinical Symptoms & Signs

The clinical diagnosis of low back pain & sciatica (Stevens J, 1968) covers a large number of different disorders, the commonest being prolapsed intervertebral disc (90 %), then soft tissue disorders, such as lumbo-sacral strain, fibrositis, myositis, osteoarthritis, & miscellaneous, in that order of frequency. Other, although rare, conditions are equally important, e.g. specific infections such as pyogenic osteomyelitis, tuberculous spondylitis & brucellosis, & non-specific conditions, such as ankylosing spondylosis, rheumatoid arthritis, osteomalacia, Paget's disease etc., neoplastic such as myelomatosis, neurofibromatosis, hemangiomas, lipomas, & primary or secondary malignant tumors of the spine, & metabolic disorders such as severe osteoporosis⁴⁷.

Symptoms in degenerative lumbar spinal stenosis are caused by a combination of central, lateral recess, and intervertebral foraminal stenosis that occurs as a part of a degenerative cascade. Neurogenic claudication is the hallmark for symptomatic LSS. Classically described as lower limb or buttock pain brought on by prolonged standing or walking, the pain distribution may be unilateral or bilateral, monoradicular or polyradicular, and may include a component of paresthesias and weakness. Low back pain is common but is not always present. Activities involving lumbar extension such as prolonged overhead reaching or walking downhill are typical exacerbating factors. Lumbar flexion postures, such as bending forward,

pushing a shopping cart, or sitting down typically relieves symptoms. Patients may complain of walking with a stooped-forward posture. Degenerative LSS results from the development of marginal osteophytes of disc ventrally, zygapophyseal joint hypertrophy laterally, and hypertrophy or buckling of the ligamentum flavum dorsally. Other conditions include disc herniation, tumor, congenitally short pedicles, vertebral fracture, and spondylolisthesis.⁴⁸

The onset of backache or lumbago may be sudden, after lifting a weight or after bending forwards to pick up an object, or it may have an insidious onset & progress slowly. Often the backache is mild & intermittent, brought on by exertion & relieved by rest. This persists for a few days or weeks till a sudden strain aggravates the trouble. This pain may be felt over the spine, the sacro-iliac joints or the iliac crest or occasionally even the groin, & is due to the stretching of the annulus & the posterior longitudinal ligament. The backache is accompanied by stiffness of the spine due to reflex-muscle spasm. Bilateral spasm leads to scoliosis, which may be either towards or away from the side of the disc. In some cases alternating scoliosis is observed. The patient assumes the posture, which gives him the least pain. Because of muscle spasm, all movements of the spine are limited especially forward bending. In case of a disc prolapse, generally only one movement is restricted. A generalized restriction should make us investigate for alternative causes of pain. All these symptoms get relieved by bed rest. Gardner's test for malingerers states that if the patient is able to sit with his legs & trunk at right angles, it is probable that he has a straight leg raising test (SLRT) to 60-70 degree.

Neurological Symptoms & Signs:

Neurological symptoms may appear simultaneously with pain & stiffness of the spine or often much later. The earliest evidence of root involvement in a lumbar disc prolapse is radiating pain along the course of the sciatic nerve, commonly called sciatica. The exact distribution of the pain depends upon the particular root involved. Usually it is a shooting, episodic pain, related to movement, & going down the back of the thigh a varying distance either along the posterior or postero-lateral aspect of the calf. The distribution of pain will naturally be different if the disc prolapse is at a higher level. The radiating pain is usually often aggravated by coughing, sneezing, or straining. Besides pain, compression of the root may produce parasthesia, tingling or numbness in the area of the distribution of the particular root. As the compression progresses objective neurological deficits i.e. sensory, motor or reflex appear. Usually the symptoms & signs are restricted to one root, but occasionally other adjacent roots or the whole of the cauda equina may be compressed (Tandon & Sankaran, 1967)⁴⁹. The signs & symptoms of specific root involvement are summarized below in Table. An important feature of the disease is the intermittent exacerbation & remission of varying duration & severity. The sciatic pain in some rare instances may get suddenly relieved leaving behind motor weakness & numbness, if the nerve root loses its conductivity due to extreme compression or ischemia. Occasionally the backache may disappear with the appearance of the sciatic (root) pain & is due to the fact that with the extrusion of the disc, the stretch on the annulus has eased.

Table 1:Common Root Involvement

Root	Cause	Sensory Deficit / Parasthesia	Motor Deficits	Reflexes
L 4	L 3 – L 4 IVD, L 4 Foraminal Stenosis	Postero-lateral Thigh, Anterior Knee, Medial Leg	Quadriceps Hip Adductors	Patellar tendon
L 5	L 4 – L 5 IVD, L 5 Foraminal Stenosis	Anterolateral Leg, Dorsum of the foot, Great Toe	Extensor Hallucis, EDL, EDB, Gluteus Medius	Usually None
S 1	L 5 – S 1 IVD, S 1 Foraminal Stenosis	Lateral Malleolus, Lateral foot, Heel, Web of 4th & 5th Toes	Peroneus Longus & Brevis, Gastro - Soleus, Gluteus Maximus	Achilles Tendon

Straight-Leg Raising Test (SLRT - Lasegue's Test):

Compression or stretching of any one of the nerve root constituting the sciatic nerve, results in limitation in straight leg raising due to the extra stretch that this maneuver causes. This sign is present in the vast majority of cases of root compression due to protruded discs. Usually, the pain begins when the leg is raised to about 40 degrees. Extension of the contralateral leg may also be limited by the onset of pain on the affected side. This happens in 43% of cases in unilateral disc lesions & 88.3% of cases where the disc has protruded en mass. The raising of the contralateral leg tugs at the sensitive nerve root causing pain. In our country, limitation of straight leg raising may not be seen in some patients thought a root compression by a prolapsed disc is proved later at surgery. This is explained by the fact that in India most people, especially women, adopt the stooping posture to perform many of their

daily chores. Thus the nerve is elongated & has a certain amount of elastic flexibility which permits full straight leg raising despite the compression by the disc protrusion .

To differentiate the pain arising from stretching of the sciatic nerve roots from that of lumbosacral or sacro-iliac disease, the straight leg raising test can be modified. The patient is made to do the straight leg raising test, till the pain just appears, the leg is now lowered a little to make the pain disappear. At this angle, a dorsiflexion of the ankle will reproduce the pain, if it is due to nerve root stretching & not from any other causes. The Kernig's test also is useful in such cases to confirm root irritation. With the patient lying supine the hip & knee are flexed; when the hip flexion is about 90 degrees, the knee is slowly extended. This causes the sciatic pain to appear in cases of ruptured discs. The commonest roots to be involved are the L5 & S1 nerve roots.

Investigations

Plain X – Rays:

Straightening of the normal lordosis, scoliosis, narrowing of the affected disc space & osteophyte formation are the usual features. Oblique x-ray films may reveal associated spondylolysis in the lumbar region but are more useful in cervical disc lesions to show osteophytes & foraminal narrowing. Rarely the ruptured & extruded discs may calcify & thus show up on plain films.

Neurological signs aided when necessary by MRI / Myelography gives more accurate information. Plain x-rays may often be normal in a case of disc prolapse. Plain x-rays are particularly useful in excluding other lesions causing backache & sciatica, like spondylolisthesis, arthritis, tuberculosis & secondary tumors.

In India, one must always keep in mind the possibility of tubercular lesion of the spine mimicking the picture of a disc prolapse. In reviewing x-rays of the spine in a case of sciatica, very often the surgeon is so occupied with the height of the intervertebral disc & the pedicle or transverse processes, which may be the seat of a tuberculous focus.⁴⁹

A preliminary estimation of the size of the canal can be made by observing the ratio of the size of the canal to the size of the adjacent vertebral body at each segment. In cases presenting with symptoms of intermittent claudication, such a study helps to determine the cause of the claudication as neural rather than vascular (Jones & Thompson, 1968). It may, however, be mentioned that routine plain x-rays may not reveal this lesion. Axial tomography is required to demonstrate the shape & size of the spinal canal.

Myelography:

This is performed only in doubtful cases, after careful thought. Any study that introduces a foreign material, however, innocuous, into the subarachnoid space should be advised only when absolutely necessary. In case neurological localization of the affected root is definite there is no need for myelography.

Computed Tomography (CT):

This is an excellent non-invasive method of investigating the spine. This can be either Plain or Contrast enhanced. The invention of CT has helped to accurately diagnose any abnormality in the vertebral body & its appendages. Thinner sections or cuts can be taken in the later generation of CT scanners which help to delineate the pathology better. The main disadvantage being the expense & availability but with due course of time this is increasing exponentially.

Magnetic Resonance Imaging (MRI):

MRI is currently the standard for advanced imaging of the spine. MRI is superior to CT in most circumstances, in particular, identification of infections, tumors, & degenerative changes within the discs. More importantly, MRI is superior for imaging the disc & directly images neural structures. Also, MRI typically shows the entire region of study (i.e., cervical, thoracic, or lumbar). Of particular value is the ability to image the nerve root in the foramen, which is difficult even with post myelography CT because the subarachnoid space & the contrast agent do not extend fully through the foramen. Despite this superiority, there are circumstances in which MRI & CT, with or without myelography, can be used in a complementary fashion.

The demonstrated findings must be carefully correlated with the clinical impression. The best way to obtain meaningful clinical information from MRI of the spine is to have a specific question before the study. This question is derived from a patient's history & a careful physical examination & is posed using the parameters of (1) neural compression, (2) instability, & (3) deformity. In each case, the specific location of the abnormality should be suspected before MRI & confirmed with the study. Only abnormalities in one or a combination of these categories are important because surgical techniques can treat only these problems. Failure to interpret an imaging study in this way, especially MRI, which is sensitive to anatomical abnormalities, would inevitably lead to poor clinical choices & outcomes. T1 is used for vertebral body structure, while T2 is used to enhance the spinal cord. It is much more expensive & availability is restricted to a few centers in the country.

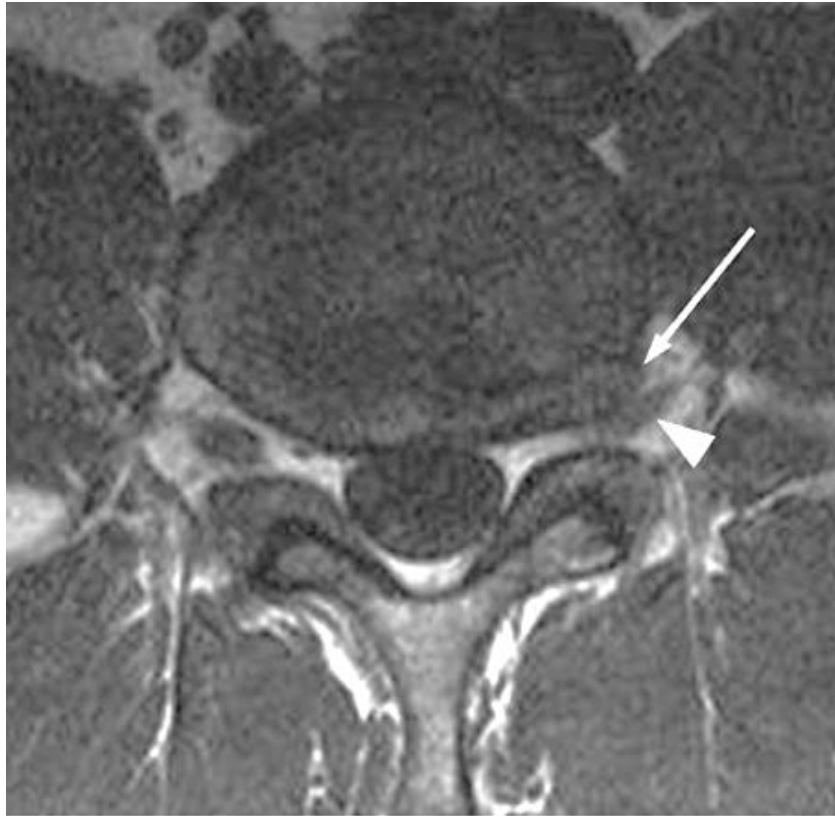


Fig 14: MRI Intervertebral Disc Prolapse(Transverse Section)



Fig 15: MRI Intervertebral Disc Prolapse (Sagittal Section)

Treatment

From the onset it must be emphasised that there is no a single treatment for backache & sciatica offering a complete & permanent cure. The three most effective components are (Cyriax)

- (a) Exercise & heat to lumbar spine
- (b) Manipulation & traction
- (c) Epidural injection

Broadly speaking total management of backache falls into three groups:

1. Prophylactic
2. Operative
3. Conservative.

1)Prophylactic Treatment

There is no prophylaxis in so far as there are no means available to prevent backache from occurring. The main role of prophylaxis comes after the completion of conservative & operative treatment & is aimed at prevention of recurrence of symptoms. Prophylaxis consists of the correction of both the dynamic & static posture & to avoid undue & unnecessary stresses & strains which predispose the herniation of the disc.

2)Surgical Treatment

This involves removal of the extruded disc either through fenestration surgery or wide laminectomy, which may or may not be followed by arthrodesis of the spine. The indication for surgical intervention, the technique & management, are controversial & much has yet to be found out before the fundamental principles become universally accepted.

Operative removal of the prolapsed disc tissue for the relief of persistent sciatica is now accepted as a valuable & safe procedure, attended with very good prospect of cure & minimal morbidity. Even with surgical removal the cure obtained is only about 40-50 percent as stated by Shinnars & Hamby (1949)^{50,51}, Dunkerley (1971), Love (1949), Raaf & Berglund (1949).⁵²

Better results of surgery are obtained in cases who displayed objective neurological evidence than in those without neurological signs (Shinner & Hamby). There is no logic in the wait & watch policy, a more realistic & aggressive attitude is more desirable & beneficial for the patient once conservative treatment has failed to relieve the symptoms. MRI can form a useful pre-operative investigation in locating & defining lesions. A cauda equina lesion is an indication per excellence for urgent exploration.

3)Conservative Treatment⁵³

This, in almost all the cases suffering from disc syndrome, forms the first line of treatment. Various modalities are:

Oral drugs-

Nonnarcotic analgesics

Acetaminophen

Aspirin

Nonsteroidal anti-inflammatory agents

Narcotic analgesics

Muscle relaxants

Antidepressants

Corticosteroids

Physical measures-

Bed rest

Activity modification

Exercise

Manipulation

Local heat

Superficial (hot packs)

Deep (ultrasound or diathermy)

Local cold

Massage, mobilization, and other soft-tissue techniques

Corsets

Traction

Injection drugs-

Anesthetics (epidural and facet joint)

Corticosteroids (epidural and facet joint)

Intradiskal chymopapain

Stimulation-

Acupuncture

Transcutaneous electrical nerve stimulation (TENS)

Implanted neurostimulators

There appears to be general agreement in that all forms of conservative treatment are effective & beneficial to the patient in some form or other. The choice & plan of conservative treatment is very much a matter of the clinician's own likings & dislikings, & is indeed his ideas, interests & beliefs.

Bed rest :

It is by far the most widely form of conservative treatment aimed at both physical & mental rest. Putting the patient in bed in itself reduces the intradiscal pressure considerably & also, by minimizing the movement, reduces the chances of nerve irritation & thereby affords relief of displaced cartilage (Cyriax), eliminates the work & the gravity stresses, & diminishes the muscle spasm. For the treatment to be effective the patient must be at complete rest, not only physically but also mentally. The patient cannot rest if agitated or worried (Raaf).

The obvious disadvantages if prolonged rest in bed is that the patient should ideally be hospitalized which entails the patient moving out of his home surroundings with its accompanied disadvantages.

Other disadvantages are:

- (i) slowness in affording relief,
- (ii) economic factors both involving the hospital & the patient,

Medication

Various medicaments that have been used fall mainly into 3 groups:

1. Simple and narcotic analgesics,
2. Muscle relaxants – these were used in the hope to combat painful spasm & also to produce sedation & tranquillization (Krayenbuld & Zender). Good results have been claimed by some.
3. Anti - inflammatory drugs – use of these drugs is based on the fact that no respective inflammatory processes be that due to mechanical, chemical or of autoimmune nature has been confirmed by histological means (Rexed & Lindahl).

The obvious logical step was to use anti-inflammatory drugs to combat this inflammatory process.

Other drugs that have been tried are:

1. Cortisone – systemic use is hazardous & dangerous for obvious reasons. Locally its use is effective & useful, both in reducing inflammatory process & reducing the subsequent fibrosis & adhesion formation,

2. Non-steroid drugs – such as:

Phenylbutazolidin

Tanderil

Indomethacin

Physiotherapy

Heat, & massage according to many is soothing & may help to reduce the spasms, but Cyriax believes that in disc lesions heat & massage, though futile are quite harmless. Hydrocollator packs-moist heat-is more comfortable than dry heat. Exercises in the acute stage are not only undesirable & illogical, but are positive harmful. In the acute stage, the patient needs rest rather than mobility but in the convalescent period exercises should form the integral part of the prophylactic managements by correcting the posture, by improving the tone & muscle balance, undue strain of movements & gravity are minimized.

Supports:

Cyriax considers it a logical treatment to be advocated after reduction of the disc & this he believes is to maintain the reduction. Supports can be given either as:

(a) Surgical appliances such as corsets or lumbo-sacral supports.

(b) Plaster of Paris jacket.

Advantages of Supports

- (a) They permit earlier mobilization, ambulation & resumption of activities.
- (b) They restrict movements of the deranged segment of the spine - a permanent factor in the production of pain, i.e. it immobilizes spine.

Disadvantages & Limitations of the Supports

- (a) If continued without exercise, the muscles waste in a very short time.
- (b) They do not eliminate the stresses of gravity
- (c) Proper molding & fit of the corset is of the utmost importance to that they can position & maintain the spine in the proper physiological curve.

Short corsets are no good. They in fact increase lordosis.

Back Braces

Back braces often offer no advantage over corset. On the other hand, they are less comfortable.

Manipulation⁵⁴

They are a great deal of controversy & debate as to the indications, value & mechanism by which manipulation affords relief. Some people believe that by manipulation of the spine, one is breaking the peri articular adhesions. Cyriax believe that manipulation reduces the displaced fiber cartilage part of the disc & therefore is only successful in those cases, & not when there is protrusion of the nucleus pulposus.

Manipulation is contraindicated in conditions such as.

- (a) Signs of involvement of both sacral roots.
- (b) Pregnancy
- (c) Spinal claudication.
- (d) Neurosis.

It is likely to be a useless procedure if the disc protrusion is large & soft. The value of manipulation in post laminectomy cases is doubtful.

Epidural steroids

The use of epidural steroid injections should be viewed as only one of the components of a comprehensive functional rehabilitation program. Patient education regarding the pathophysiology of neurogenic claudication is often helpful to the patient's independent pain-control efforts. A comprehensive flexion-biased physical therapy program is an essential component in the overall management of the patient. Pain relief obtained with epidural steroid injections often facilitates the patient's tolerance for and progression through a functionally oriented rehabilitation program. The effectiveness & utility of nerve blocks as diagnostic & therapeutic measures in the management of disc sciatica syndrome is depended upon inherent properties of local anesthetic to interrupt specific sensory & somatic, motor & anatomic pathways & also on the anti-inflammatory & anti-proliferative properties of steroid.

In the recent years, epidural injections are gradually finding their well-deserved place in the management of backache & sciatica. The results of relief from the treatment so far published are quite encouraging.

Mechanism of Action of Epidural Injections

Epidural injections have been used successfully for decades in treatment of low back pain & sciatica. The mechanism of action of the drugs is uncertain. Hence, there have been many theories as to what should be injected & what is being accomplished by the injection as well as the best way to approach the space, these include the following.

Anti-Inflammatory Effect:⁴⁸

Symptoms have been hypothesized to be a result of a combination of ischemic neuritis of the cauda equina or nerve root, impaired epidural venous return resulting from an increase in cerebrospinal fluid pressure below the level of compression, or disruption of nerve root microcirculation when standing. This process can result in the formation of nerve root edema as a result of microvascular injury inside the nerve roots. Edema has been noted to produce pain in nerve roots. The stenotic nerve root canal can result in mechanical compression of the exiting nerve root. The nerve roots themselves as they exit the neural foramen have a poorly developed epineurium, rendering them particularly vulnerable to mechanical and chemical injury.

Compression of the large venous plexus within the intervertebral foramen may occur, leading to congestion, ischemia, intraneural edema, and increased intraneural pressure. Nerve root inflammation can potentially occur from multiple sources in the degenerative spine. Degenerative changes present in the spine may lead to hyperemia, venous congestion, and perhaps leakage of neurotoxin substances. Multiple studies have demonstrated adverse histologic, inflammatory, and electrophysiologic effects of material from the nucleus pulposus on neural tissues.

Degenerative disc disease of the lumbar spine with annular disruption and herniation could lead to leakage of neurotoxic substances. Multiple studies have demonstrated the adverse histologic and electrophysiologic effects of discogenic inflammatory mediators on neural structures. The discovery of elevated levels of phospholipase A2 at the neural interface with herniated disc material by Saal and associates in 1990 helped confirm the role of inflammation in painful lumbar conditions. Other inflammatory mediators such as leukotriene B4 and thromboxane B2 have also been demonstrated in human nuclear material⁵⁵. The role of these

inflammatory mediators in the genesis of discogenic and neurogenic pain syndromes is now well accepted.

Corticosteroids have been noted to have potent anti-inflammatory properties. These effects are a result of inhibition of specific leukocyte functions including inhibition of leukocyte migration, prevention of degranulation of granulocytes, mast cells, and macrophages, and stabilization of lysosomal membrane and other membranes. Corticosteroids have been shown to be able to block nociceptive C-fiber conduction and also to inhibit prostaglandin synthesis. The potential mechanisms of action of pain relief of corticosteroid in the neural axis include the inhibition of nerve root edema with resultant improved microcirculation and reduced ischemia, a potential reduction in sensitivity of the prostaglandin-sensitized dorsal horn neurons by inhibiting inflammatory mediators such as phospholipase A₂, and by direct inhibition of C-fiber neuronal membrane excitation.

Inhibition of the Autoimmune Response to Nucleus Pulposus:

The nucleus pulposus is normally contained tightly within the annulus & after its embryological formation it no longer normally takes any vascular contact with the systemic circulation. This system is analogous to other human situation in which derangement is known to produce an autoimmune response. It has been shown in experimental findings in the rabbit that autogenous nucleus pulposus material can excite an autoantibody response in regional lymph nodes & if this is true, corticosteroids can suppress this antigen antibody reaction & also chronic inflammation proved by this reaction.

The Mechanical Effect:

Evans in 1930 published intra sacral epidural injection in the treatment of sciatic. By injecting high volumes either of an local anesthetic or normal saline (up to 100 ml), he observed displacement of the posterior & lateral aspects of the dural sac & reasoned that the physical displacement of the neural elements caused by the injected fluid may lead to stretching & lysis of the neural adhesions & even to anaesthesia from compressive effects. This breaking down of scar tissue in the epidural space by causing neurolysis can cause relief of radiating pain, but this has never been proved by any study. Also it was found by the injection contrast media into the epidural space that high volumes simply pass out of intervertebral foramen. When there has been previous surgery the contrast is guided away from the scar tissue site & takes the path of least resistance.

Indications:⁵⁶

Indications can be conveniently divided into two main groups:

1. Diagnostic

Epidural injection is useful in determining the cause of:

(a) Uncharacteristic Backache

(b) Referred pain, which will not be affected or altered (If a correctly performed epidural injection fails to relieve the patient's symptoms it is likely that the lesion lies outside the spinal canal.)

(c) Psychoneurosis

(d) Differential spinal block can establish or exclude a diagnosis of malingering. This test is based on the fact that different strengths of a local anaesthetic agent selectively block conduction in nerve fibres of different diameters.

2. Therapeutic indications

- a) Acute 'lumbago'. Epidural injection in this state generally affords instant relief from pain.
- b) Intractable sciatic pain.
- c) Chronic backache
- d symptoms of intervertebral disc prolapse complicating pregnancy
- e) Root pain with or without neurological signs
- f) Nocturnal cramp & coccydinia
- g) A patient with discogenic pain with or without sciatica, not relieved by adequate conservative methods (Acute / Chronic)
- h) As a non-operative treatment in patients with lumbar canal stenosis.
- i) failure after laminectomy or other methods of treatment.

Contraindications:

- Absolute contraindications to performing epidural injections include a) known hypersensitivity to agents.
- b) Local infection and sepsis are also contraindications because of the potential for hematogenous spread through Batson's plexus.
 - c) local malignancy.
 - d) anticoagulant therapy or coagulopathy, because of the risk for epidural hematoma.
 - e) congestive heart failure.
 - f) uncontrolled diabetes mellitus- causes elevation in blood sugars.
 - g) Cauda Equina syndrome, which is a surgical emergency.

Technique:^{57,10}

There are several accepted techniques to access the lumbar epidural space. These techniques include the interlaminar technique, the caudal technique, and the transforaminal technique. If there is an adequate posterior epidural space on the cross-sectional study, preferred level for injection is L4-5. The next most common level for injection is L3-4, and it is important to note that the posterior epidural space at L5-S1 is typically small or nonexistent and much less suitable for injection. Some operators believe that fluid reliably spreads only a couple of vertebral levels from the needle tip, typically in a cephalad direction. However, Harley¹⁰ showed that 6 mL of contrast injected at L 4-5 consistently spread above L1 and down over the sacrum. Nevertheless, most operators, prefer to be at or no more than 2 levels below the worst level of pathology.

The lumbar route has a slightly greater risk of dural puncture which can however be overcome by a careful technique, but the advantages are more ease of identification, a lesser degree of variation in the anatomy of the lumbar region, nearer to the probable site of lesion, thus enhancing its therapeutic value.

Patient positioning is very important, with the patient prone with head flat on table and a bolster or pillow under the abdomen at the level selected in an effort to reverse the normal lumbar lordosis and open the spinous processes. Standard sterile provodone iodine preparation and draping are followed by local anesthesia of the skin and subcutaneous tissues with lidocaine. The approach selected for a lumbar epidural injection, will depend on the operator's preference and experience.

Interlaminar approach

These injections are performed either using a midline or paramedian approach. The patient is positioned prone or sitting posture. Most authors recommend using either an 18- or 20- gauge epidural Tuohy or Crawford needle connected to a loss-of-resistance syringe. The needle is advanced first through the supraspinous ligament, through the interspinous ligament, and eventually to the ligamentum flavum. Once the ligamentum flavum is penetrated, a loss of resistance occurs, indicating placement in the epidural space. Non-ionic contrast is injected under live fluoroscopic visualization to confirm epidural localization and to exclude inadvertent placement in the paraspinal musculature, intraligamentous soft tissues, arterial or venous blood vessel, or thecal sac. Once placement in the epidural space is confirmed, a total of 3 to 5 mL of preservative-free 1% lidocaine is given, followed by 1 to 2 mL of the corticosteroid preparation.



Fig 16:Fluoroscopic Anterio Posterior View Interlaminar approach-Needle Placement

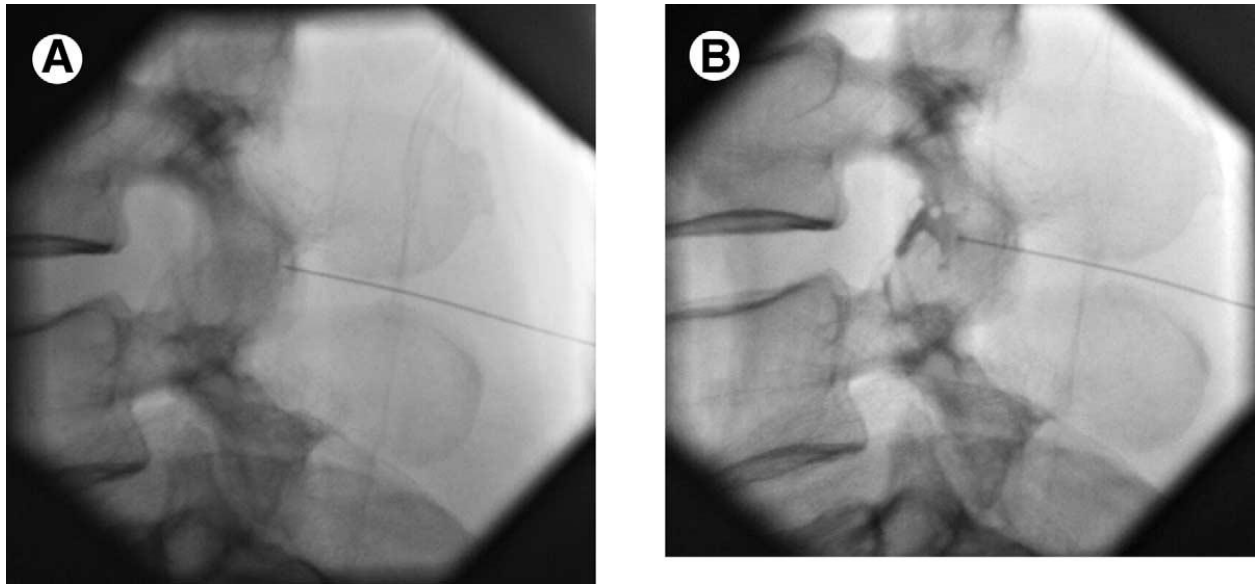


Fig 17: Fluroscopic Lateral View Interlaminar approach-Needle Placement(A),
Contrast Injection(B)

Caudal approach

The procedure using a caudal approach is performed with the patient in a prone position. A wedge-shaped pillow is placed under the hips to tilt the pelvis and bring the sacral hiatus into greater prominence. The sacrococcygeal area is prepared using an iodine-based antiseptic solution. The preferred method for identification of hiatus is by using the sterile-gloved middle finger on the dominant hand to locate the tip of the coccyx through palpation. A mark is then placed under the interventionalist's proximal interphalangeal. Other method of identification of the sacral hiatus is by running a finger down the middle of the sacrum until the paired cornua are identified a few centimeters proximal to the coccyx. The cornua are the lateral eminences of the sacral notch, which is the opening to the sacral hiatus at the S4 level. Local anesthetic is given, and anteroposterior view is obtained to ensure that a midline position is noted. Creating a gentle curve at the end of the needle may help the needle to follow the slope of the sacrum. With a 45° angle of entry and the bevel down, the needle is advanced through the sacrococcygeal ligament and stopped when

the underlying bone is encountered. A lateral fluoroscopic image should be obtained to assess needle depth . The needle is then withdrawn slightly, the bevel is turned up, and the needle hub is dropped nearly horizontally. Ideally, the needle shaft will be in the same plane as the sacral canal. The needle is advanced with a gentle twisting motion to decrease the likelihood of lodging in the sensitive periosteum. Needle advancement is stopped at S3 to avoid the caudal margin of the thecal sac

Most authors recommend using either a 20-gauge 3.5-inch/90-mm Tuohy needle or a 22-gauge 3.5-inch/90-mm spinal needle, placed into the sacral hiatus. Non-ionic contrast is placed into the caudal canal to exclude intravascular, intrathecal, and soft tissue infiltration. Intravascular uptake has been shown to occur in up to 10.9% of caudal injection procedures . Once an epidurogram is obtained, a variable volume of anesthetic and corticosteroid preparation can be injected. The total volume of injectate used is debatable. Studies have shown that 80% of the time 10 mL of injectate volume will reach the L4-5 interspace.



Fig 18: Caudal approach Fluroscopic View-Lateral(A),Anterio Posterior(B)

Transforaminal approach

The transforaminal approach requires fluoroscopic guidance. Andrade and Eckman⁵⁸ have shown that translaminar fluoroscopic epidural injection results in dorsal flow of contrast agent, whereas the foraminal approach shows ventral flow.

The technique described by Derby et al is recommend for performing transforaminal injection. The patient is placed prone on a radiology table. The patient's back is prepared using an iodine-based antiseptic solution. A 22- or 25-gauge spinal needle is inserted under intermittent fluoroscopic guidance to the dorsal/ventral aspect of the neural foramen at the suspected symptomatic radicular levels. An A-P or slightly oblique fluoroscopic view is obtained to assure that the needle is directed to approximately at the 5:30 position on the right and the 6:30 position on the left, using the pedicle as a clock face. A lateral fluoroscopic view is then obtained to confirm that the needle is positioned just beneath the pedicle in the anterior epidural space. Aspiration is performed once in this location. If the aspirate is negative for blood, non-ionic contrast agent is injected to confirm epidural flow of the injectate and to rule out intravascular, intrathecal, or soft tissue infiltration. The overall rate of intravascular injection has been described as 10.8% to 11.2%; with a transforaminal injection at S1, the rate is 19.9% to 21.3%⁵⁹. Once an epiduragram is obtained, selected drug is injected.

Complications^{10,57}

1) Due to technical difficulties -

a) Most common complication is that the injection becomes an intrathecal, with recovery of spinal fluid. In these situations the needle can be withdrawn & the next level above or below selected. An occasional intrathecal injection is accompanied by transitory "Spinal" headache which usually is relieved within 24 hours by the use of

oral analgesics. Dural puncture is a common complication of lumbar translaminar epidural injections, with an incidence of 5% to 17%.

b) Epidural needle may break & should be removed surgically as soon as possible.

2) Exacerbation of radicular pain has been documented in translaminar epidural injections with an incidence of over 4%. This exacerbation may be related to the injection of large volumes of therapeutic agents into an epidural space already compromised by disc herniation, spinal stenosis, or epidural fibrosis. It is postulated that this symptom may be avoided by injecting slowly.

3) Commonly transitory mild weakness in one or both lower extremities is noted secondary to the addition of local anesthetic in the epidural space. This usually subsides after 15-20 minutes after which the patient is able to resume normal ambulatory activities

4) Complications after intra-spinal use of long acting steroid preparation have included tuberculous meningitis, adhesive arachnoiditis, aseptic meningitis, sclerosing spinal patchy meningitis.

5) Other rare complications of epidural injections are meningitis, epidural abscess, cerebrospinal fluid-cutaneous fistula, epidural hematoma, allergy to steroids, retinal hemorrhage, and extradural abscess

6) Lignocaine sensitivity

7) Transient Neurogenic Bladder

8) Paraparesis

9) Corticosteroids have several known side effects. Fluid retention can lead to congestive heart failure. An uncommon but serious complication is the development of Cushing's syndrome as the result of excess glucocorticoid administration. This

development has usually resulted from exceptionally high doses of corticosteroid given over a short period.

Various steroids commonly used are:⁶⁰

- Dexamethasone - Particles are 5-10 times smaller than red blood cells, contain few particles, and show no aggregation.
- Triamcinolone - Particles varies greatly in size, are densely packed, and form extensive aggregations.
- Betamethasone - Particles varied greatly in size, were densely packed, and form extensive aggregations.
- Methylprednisolone - Particles are relatively uniform in size, smaller than red blood cells, and densely packed and do not form very many aggregations.

Methodology

METHODOLOGY

This study was taken up to evaluate the efficacy of lumbar epidural steroid in low backache with lumbosacral radiculopathy cases. The study was conducted for a period of one & half year, from December, 2011 to July, 2013, with the last three months being allocated to follow up under the department of Orthopaedics in R L Jalappa Hospital and research centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

MATERIALS:

Study population: A total of hundred patients satisfying the inclusion criteria from December, 2011 to July, 2013 were taken up for the study.

Inclusion Criteria:

1. Age more than 18 years
2. Patients with low backache with lumbosacral nerve root pain which has not resolved within a minimum of six weeks and is of an intensity to warrant some intervention.

Exclusion criteria:

1. Previous surgeries at the same motion segment.
2. Those with unclear topographical diagnosis.
3. Those with severely disabling neurological deficit.
4. Those with structural deformities like scoliosis, kyphosis.
5. Allergy to Steroids, Bleeding diatheses, Pregnancy.
6. Uncontrolled hypertension, uncontrolled diabetes mellitus.

After selecting the cases on the basis of the above mentioned criteria, they were counselled about the study & were included after a written informed consent to participate in the study.

All the patients underwent a thorough clinical evaluation in way of a history of the illness, including the details of pain, as well as the nature of the conservative treatment they have received in the past, examination including neurological assessment of the lower limb as per a proforma prepared for the study (attached later). They were subjected to questionnaires including the Visual Analogue Scale, Oswestry Disability Index Score(ODI) the scores were evaluated before & after the intervention and at follow up.

The investigations that were done for every patient included an X-ray Lumbo-Sacral Spine – AP and LAT, MRI Lumbosacral Spine, Routine Hemogram, HIV and HBsAg status, Bleeding and Clotting time.

Preparation & Position of the Patient:

The procedure is carefully explained to the patient, who is told to expect increase in intensity of his symptoms during the injection. All the injections were carried out in the operation theatre with dry, sterile materials. Neurological Status & SLRT are re-assessed at this stage. With the patient in sitting or lateral decubitus posture (affected side down in those who could not sit), lumbo sacral region was prepared with spirit & povidine iodine several segments above & below the laminar interspace to be injected. The patient is draped in a sterile fashion. Sitting position was preferred as the fully flexed spine lead to opening of the interspinous spaces which were being used for the injection.

Procedure:

After positioning the patient & preparation of the skin, the target laminar interspace was identified. Commonly used anatomical landmarks were the highest point of the iliac crest, with L 4 spinous process. Using a needle, the skin over the target interspace was injected with 1 to 2 ml of 2% preservative-free Xylocaine without epinephrine & was anaesthetized. The target space was within one level above or below the level of symptomatic disc. Then a 3½-inch, 18-gauge Tuohy epidural needle was inserted & advanced it vertically within the anesthetized soft tissue track until contact with the lamina has been made. "Walk off" the lamina with the Tuohy needle onto the ligamentum flavum was done. Stylet from the Tuohy needle was removed & attached to a 10-ml syringe filled with air. It was advanced into the epidural space using the loss of resistance technique. This was followed by securing the needle & then injecting 4ml 2% Xylocard and 2 ml of 40 mg/ml Methyl Prednisolone Acetate (Depomedrol) one after the other, a total of 6ml. Finally the tuohy needle is withdrawn & the spot was sealed with tincture benzoin. All the procedures were done by a anaesthetist.

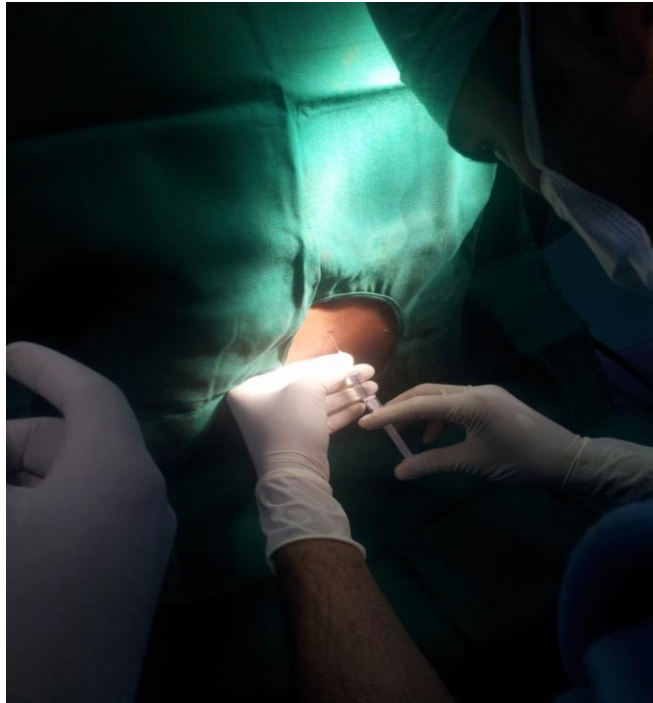


Fig 19:Local infiltration



Fig 20:Epidural needle in situ



Fig 21:Loss of resistance technique

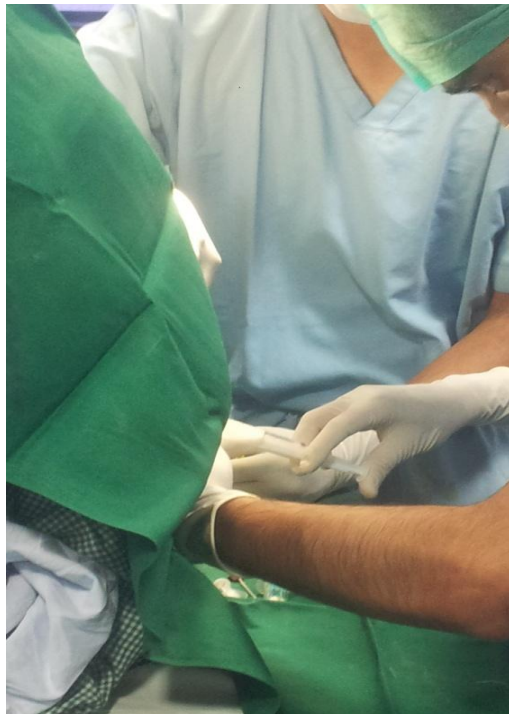


Fig 22:Steroid injection



Fig 23:Lateral posture-Epidural Injection

Post Injection Protocol

At the conclusion of the injection a note is made of the following: relief of pain & its extent measured subjectively as well as by straight leg raising test, & motor & sensory examination. The patient is advised that apart from a feeling of warmth in the legs & perhaps a sensation of walking on cotton wool, there should be no other neurological signs or untoward effect. The patient is further warned that after injection the pain may be worsened for a few days before it begins to settle. The patient is advised to lie flat for at least 45 min after the injection which helps to avoid headache developing on sitting up. The patient was advised to pass urine before leaving the hospital. Back extension exercises were continued after the injection as a routine protocol.

Clinical evaluations were performed immediately after the injection, at 1 & 3 months. The Visual Analogue Score, Oswestry Disability Index score & the Straight Leg Raising Test (SLRT) were used to differentiate patients whose symptoms improved from those who remained symptomatic. Patient satisfaction was documented at 3 months. Excellent->75% reduction of symptoms/disability, Good->50% reduction, Fair-<50% reduction, Poor-same or worse.

Results

RESULTS AND OBSERVATION

Statistical analysis:

Data was entered into excel sheet after coding and analyzed using SPSS 11 version software. Frequencies and proportions was computed for qualitative data, Mean and Standard deviation was computed for quantitative data. Paired t test is the test of significance for Quantitative data to check the difference before and after treatment. Chi-square test is the test of significance for categorical data to check the association.

Results:

Table2: Age distribution of Patients

Age Distribution	Frequency	Percent
18-30 years	22	22.0
30 to 40 years	27	27.0
40 to 50 years	24	24.0
>50 years	27	27.0
Total	100	100.0

In the study it was observed that majority of the patients i.e. 51% were in the middle age group between 30 to 50 years, 27% of them in age group >50years and 22% in <30years age group. The mean age group of the patients in the study was 42.53 ± 11.9 years.

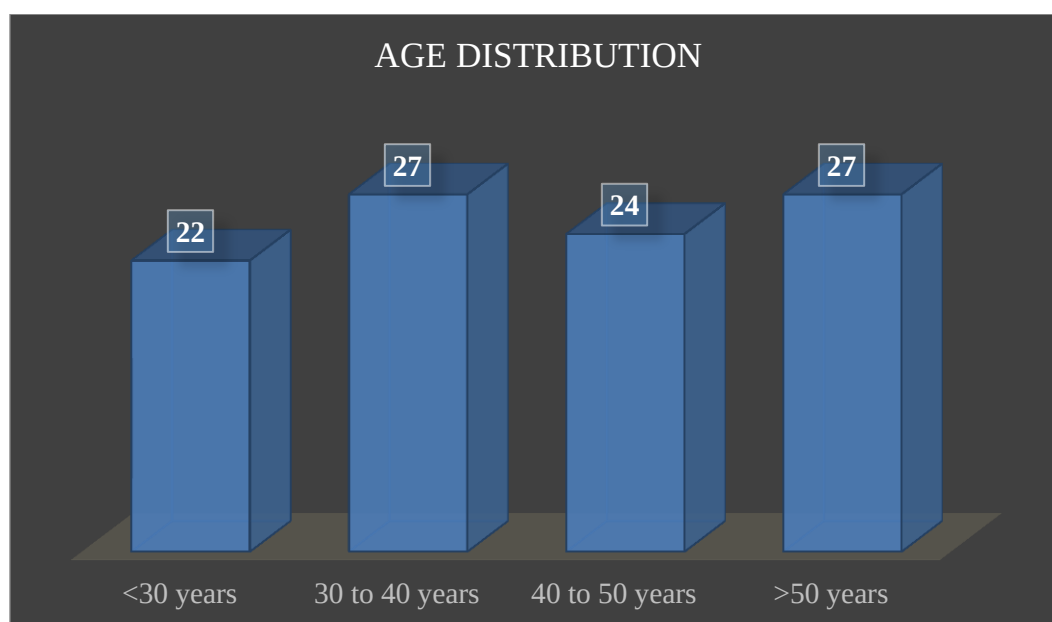


Chart 1: Bar diagram showing Age distribution of patients

Table3: Sex distribution of patients

Sex	Frequency	Percent
Female	48	48.0
Male	52	52.0
Total	100	100.0

In the study it was observed that majority i.e. 52% of patients were males and 48% of them were females.

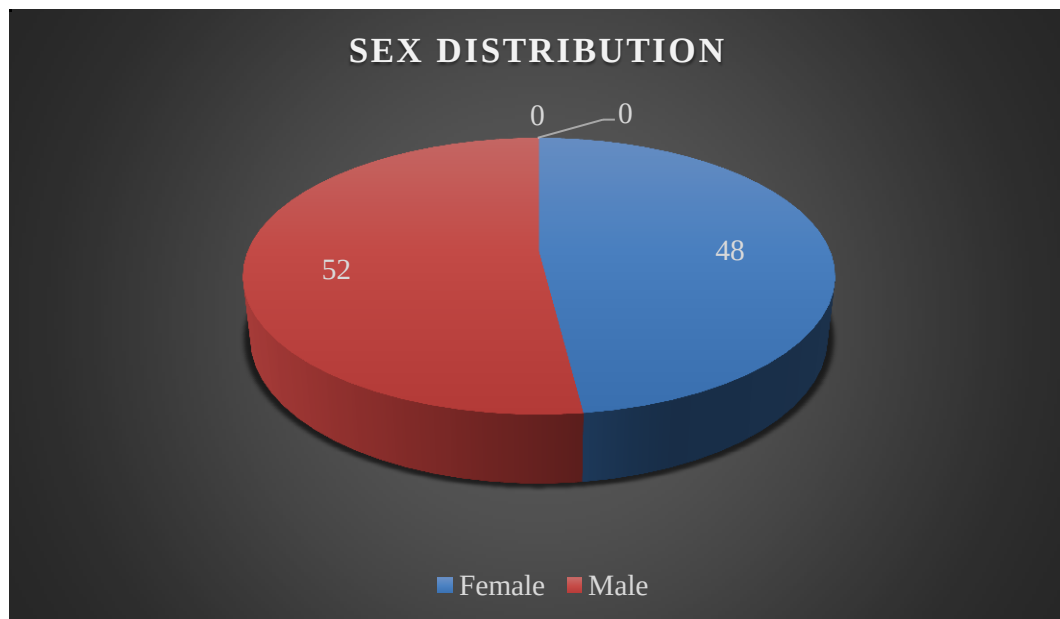


Chart 2: Pie diagram showing sex distribution of patients

Table 4: Distribution of patients according to Occupation

Occupation	Frequency	Percent
Professional/Semi professional	4	4
Skilled	16	16
Semiskilled/Unskilled	29	29
Student/Dependent	12	12
Housewife	39	39

Professional and Semiprofessional – Accountant, Advocate, Bank Employee, Teacher

Skilled – Business, Carpenter, Driver, Ration shop, Mechanic

Semi-Skilled – Agriculture, Laborer, Mason

In the study it was observed that Majority of the patients were housewife i.e. 39%, followed by Semi-skilled / Unskilled occupation i.e. 29%.

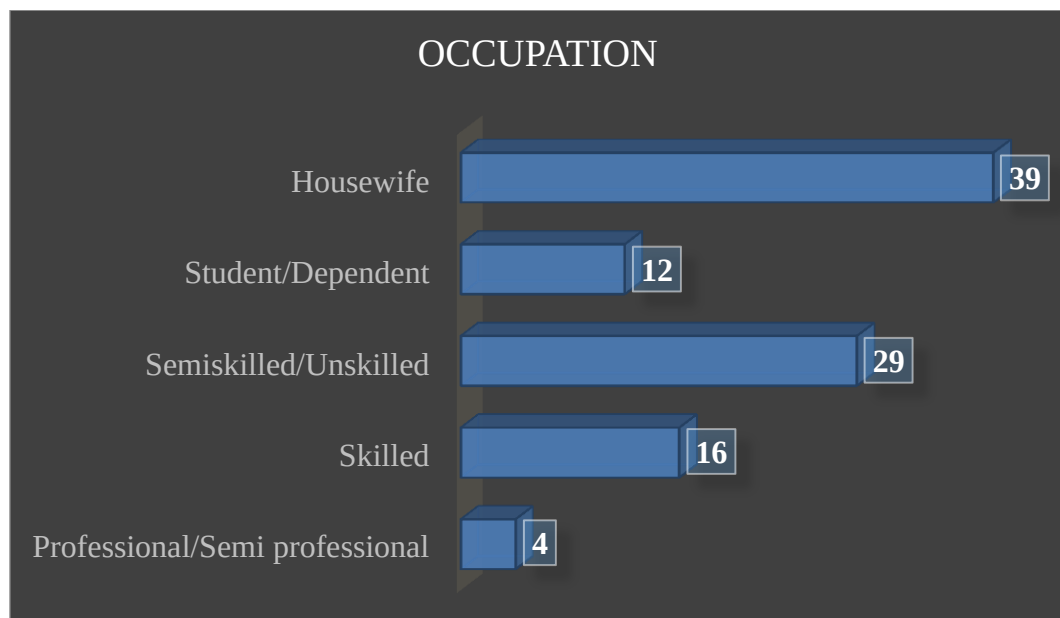


Chart 3: Bar diagram showing distribution of patients according to Occupation

Table 5: Distribution of patients according to side of Sciatica

Sciatica	Frequency	Percent
Bilateral	20	20.0
Left side	36	36.0
Right side	44	44.0
Total	100	100.0

In the study it was observed that majority i.e. 44% of patients had sciatica on right side, 36% on left side and 20% had sciatica on both the side.

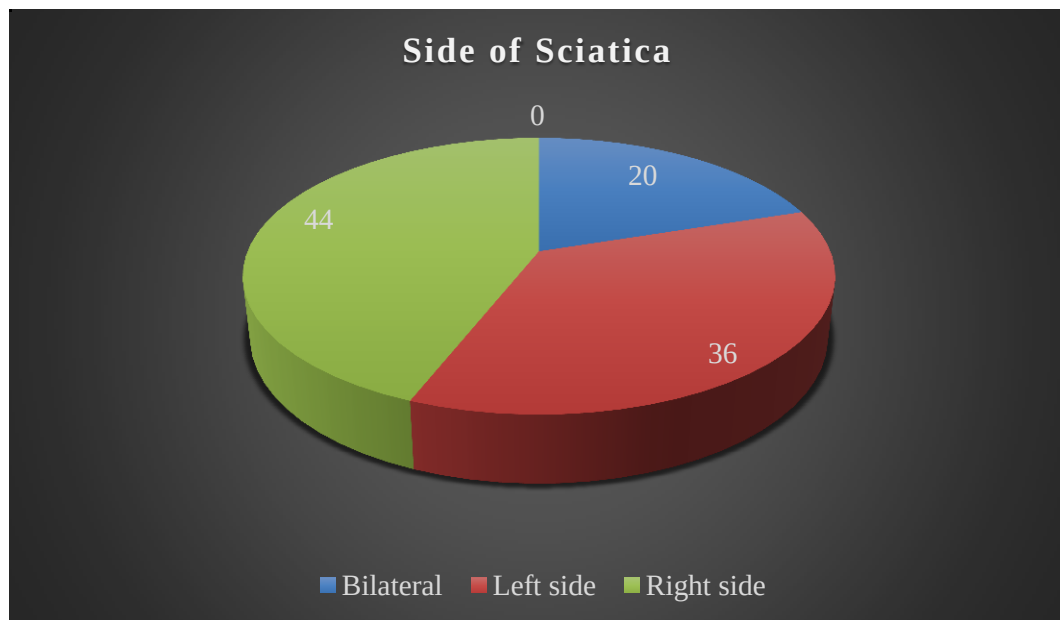


Chart 1: Pie diagram showing distribution of patients according to Side of sciatica

Table 6: Distribution of patients according to Duration

Duration	Frequency	Percent
<12 months	65	65.0
13 to 36 months	22	22.0
36 to 60 months	10	10.0
>60 months	3	3.0
Total	100	100.0

In the study it was observed that the majority i.e. 65% of patients had symptoms <12 months. 22% had symptoms between 13 to 36 months.

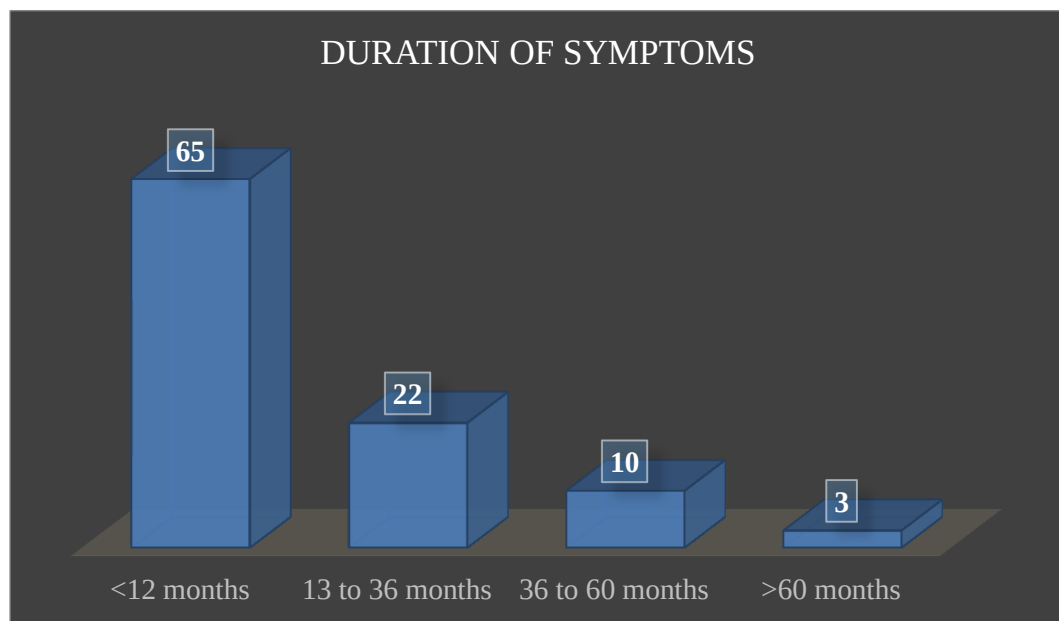


Chart 2: Bar diagram showing distribution of patients according to Duration of symptoms

Table 7: Distribution of patients according to previous treatment.

Previous treatment	Frequency	
	Yes	No
Rest/Analgesics	68	32
Physiotherapy	52	48
Traction	32	68

In the study it was observed that 68% of patients were treated by Rest/Analgesics, 52% were treated by Physiotherapy and 32% were treated by Traction.

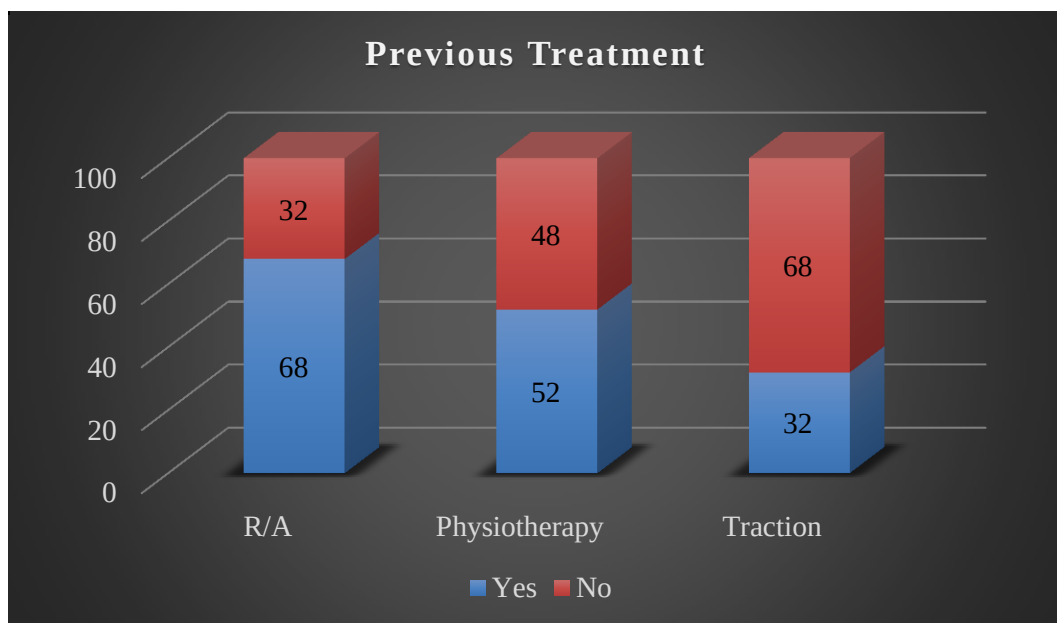


Chart 3: Bar diagram showing distribution of patients according to previous treatment

Table 8: Distribution of patients according to Co-morbid conditions.

Co-morbid conditions	Frequency	
	Yes	No
Diabetes Mellitus	13	87
Hypertension	13	87

In the study it was observed that 13% of patients had both diabetes and Hypertension.

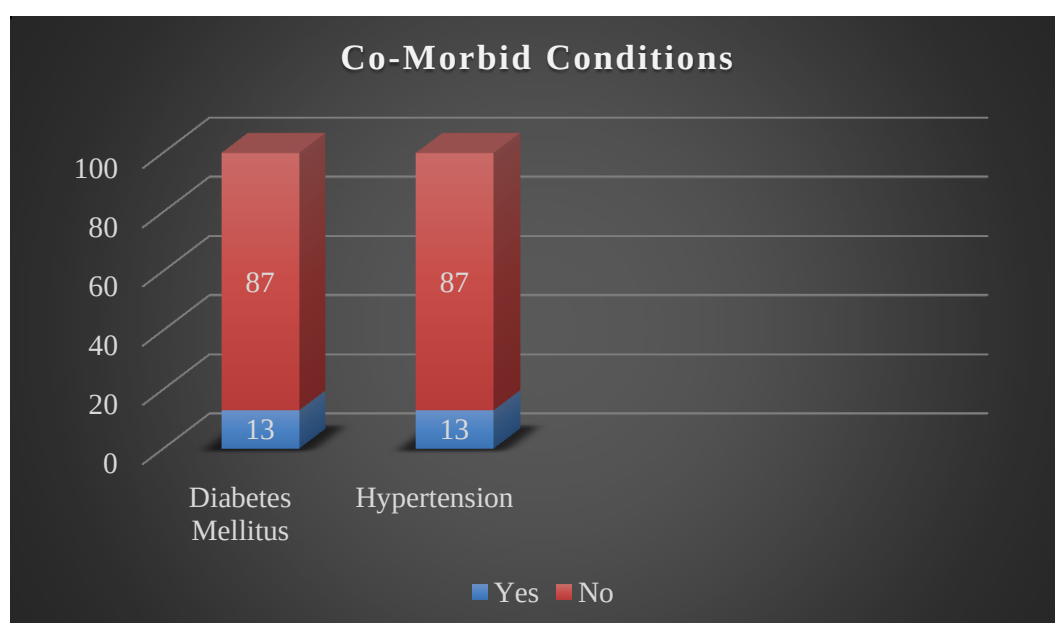


Chart 4: Bar diagram showing distribution of patients according to Co-morbid conditions

Table 9: Distribution of patients according to spasm

Spasm	Frequency	Percent
No	17	17.0
Yes	83	83.0
Total	100	100.0

In the study it was observed that majority i.e. 83% of patients had spasm and 17% did not have spasm.

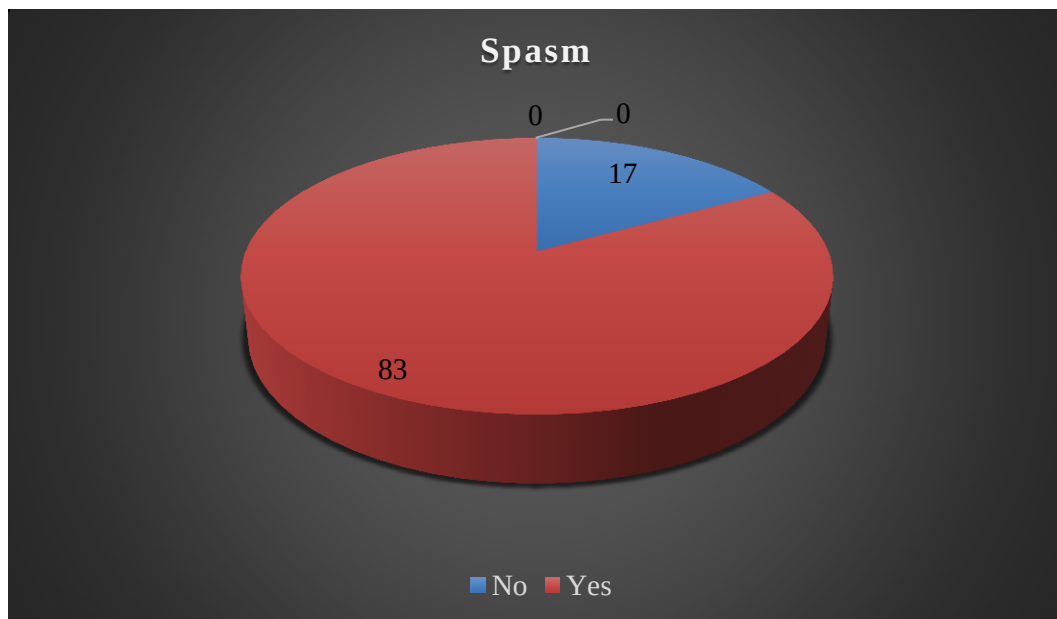


Chart 8: Pie diagram showing distribution of patients according to Spasm

Table 10: Distribution of patients according to degree of SLRT before treatment

SLRT	Frequency	Percent
Right side		
>70	35	35
≤ 70	65	65
Left side		
>70	48	48
≤ 70	52	52

In the study it was observed that 65% of patients had $SLRT \leq 70$ on right side and 52% of patients had $SLRT \leq 70$.

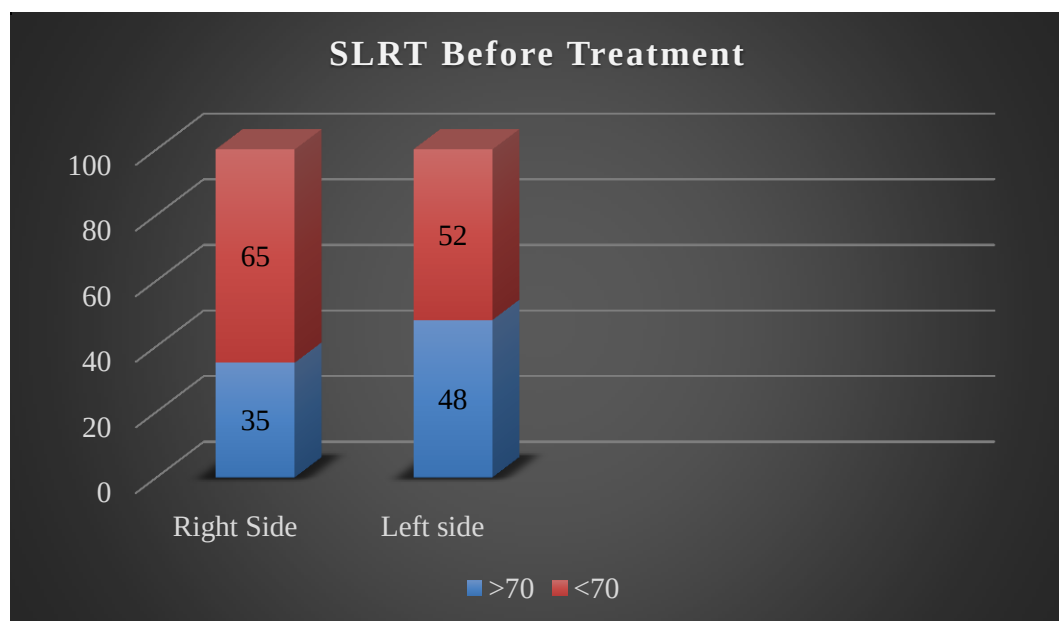


Chart 11: Bar diagram showing degree of SLRT before treatment

Table 11: Distribution of patients according to Neurological Signs

Sensory Signs	Frequency	Percent
No	65	65.0
Yes	35	35.0
Motor Signs		
No	84	84.0
Yes	16	16.0

In the study it was observed that 35% of patients had sensory signs and 16% of patients had Motor signs.

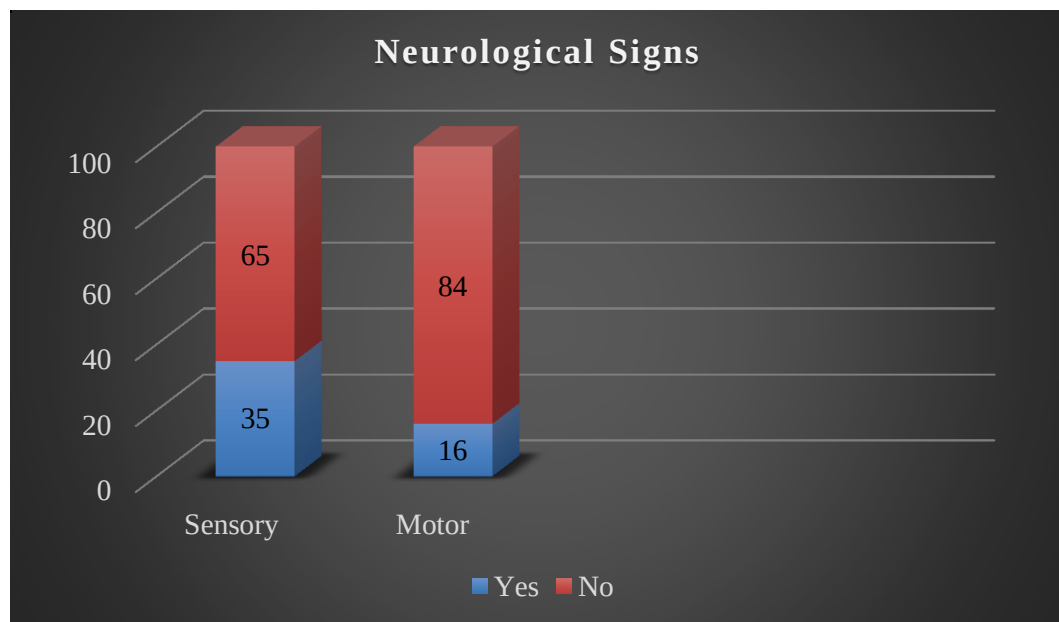


Chart10: Bar diagram showing distribution of patients according to Neurological signs

Table 12: Distribution of patients according to ODI (Oswestry Disability Index) score before treatment

ODI before treatment	Frequency	Percent
20 to 40%	24	24.0
40 to 60%	76	76.0
Total	100	100.0

In the study it was observed that majority i.e. 76% of patients had a score of 40 to 60% and 24% had ODI score of 20 to 40%.

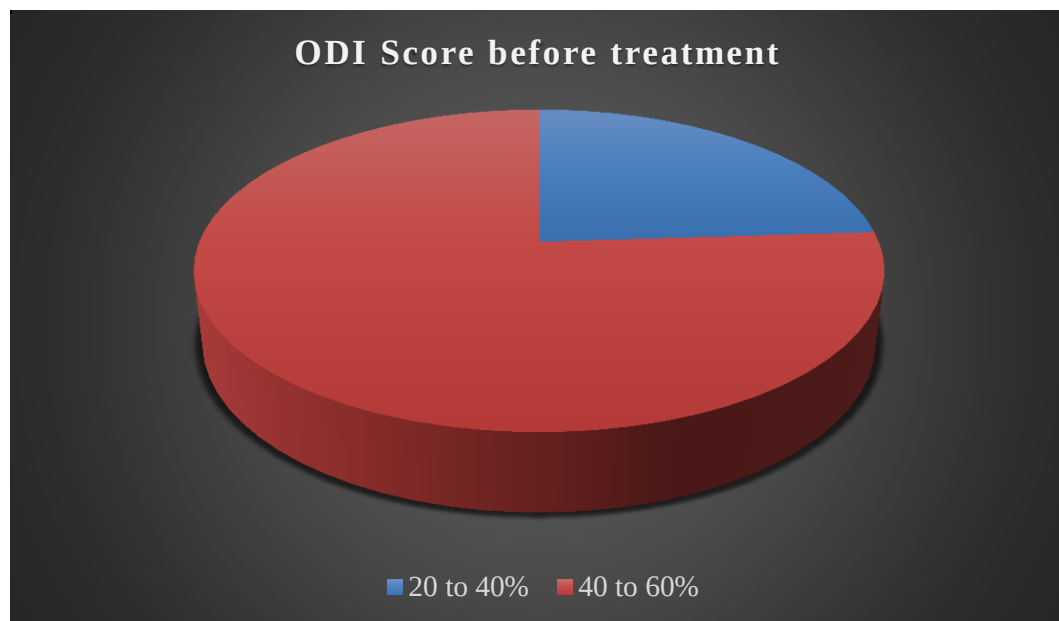


Chart 115: Pie diagram showing distribution of patients according to ODI score

Table 13: Distribution of patients according to VAS score before treatment

VAS Score before treatment	Frequency	Percent
6	13	13.0
7	36	36.0
8	40	40.0
9	11	11.0
Total	100	100.0

In the study it was observed that majority i.e. 40% of the patients had a VAS score of 8, and 36% had VAS score 7.

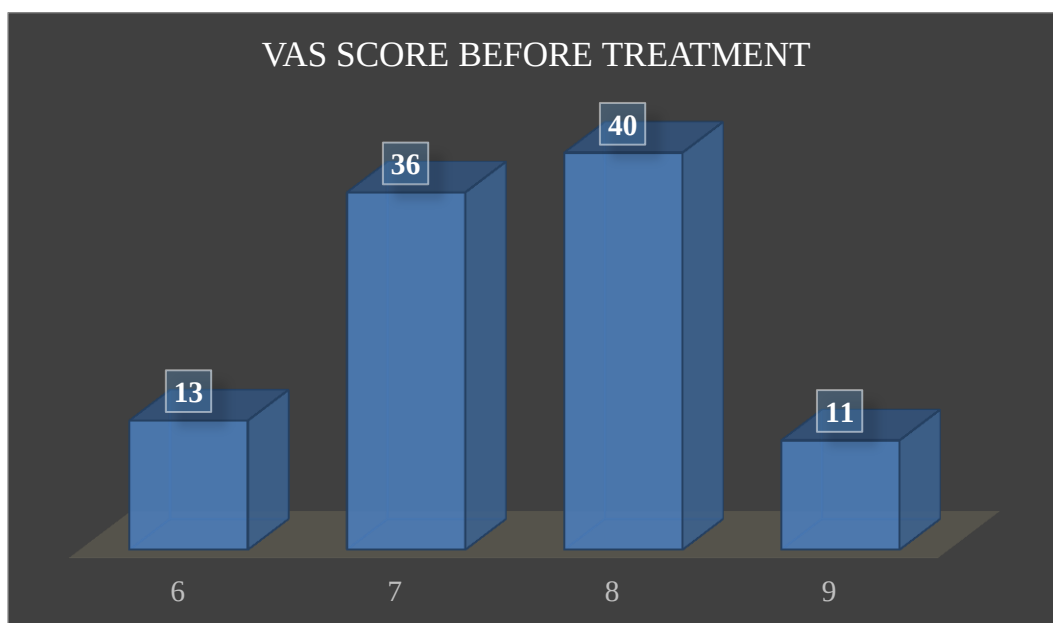


Chart 62: Bar diagram showing distribution of patients according to VAS Score before treatment

Table 14: Distribution of patients according to Levels

Level	Frequency	Percent
0	1	1.0
1	67	67.0
2	26	26.0
3	6	6.0
Total	100	100.0

In the study it was observed that majority i.e. 67% of patients had lesion at single level. 26% of patients had lesions at 2 levels and 6% of patients had lesions at multiple level.

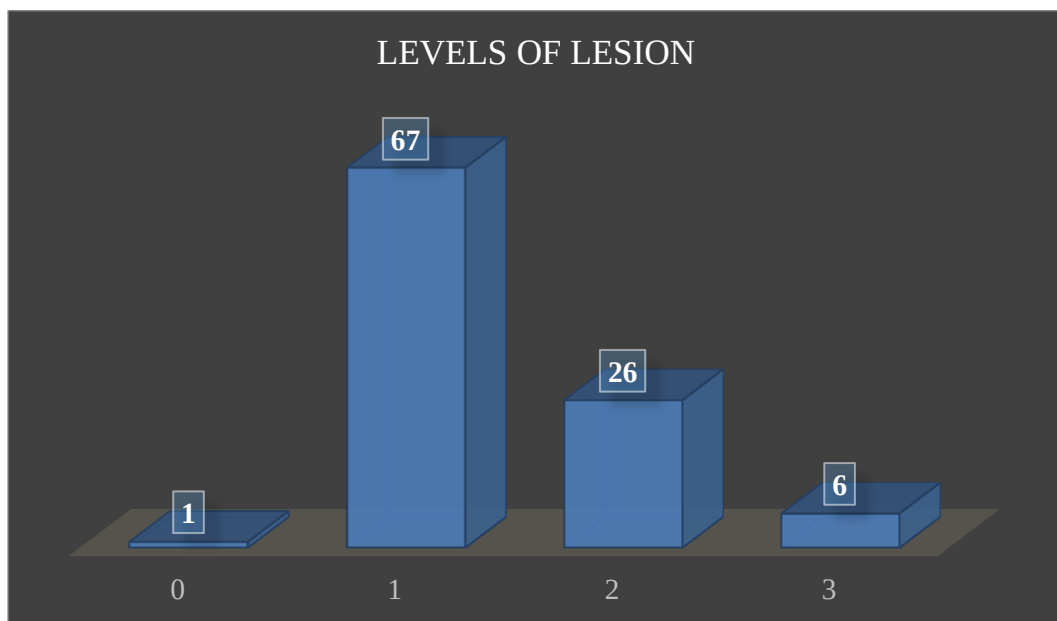


Chart 13: Bar diagram showing distribution of patients according to Levels of lesion

Table 15: Distribution of patients according to Radicle affected

Root Affected	Frequency	Percent
L3	2	2.0
L4	15	15.0
L5	36	36.0
L5- S1	7	7.0
S1	40	40.0
Total	100	100.0

In the study it was observed that in majority i.e. 40% of patients S1 radicle was affected, in 36% of patients L5 radicle was affected.

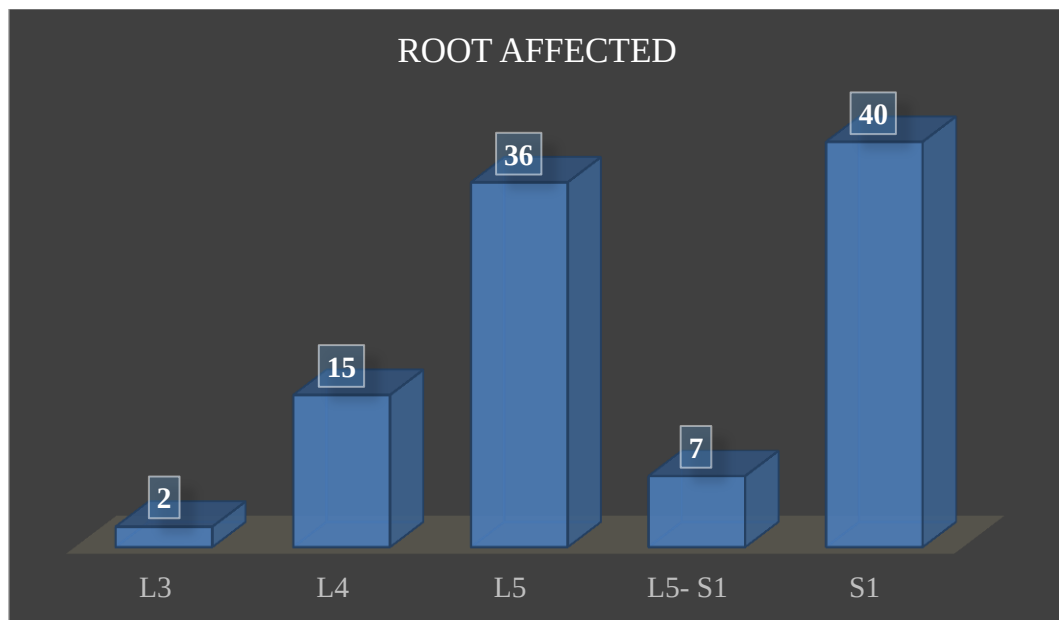


Chart 74: Bar diagram showing distribution of patients according to Radicle affected

Table 16: Distribution of patients according to Complications

	Yes	No
Dural Puncture	15	85
Pain at Injection Site	27	73
Headache	10	90

In the study it was observed that 15% of patients had Dural puncture, 27% of patients had pain at injection site and 10% had headache.

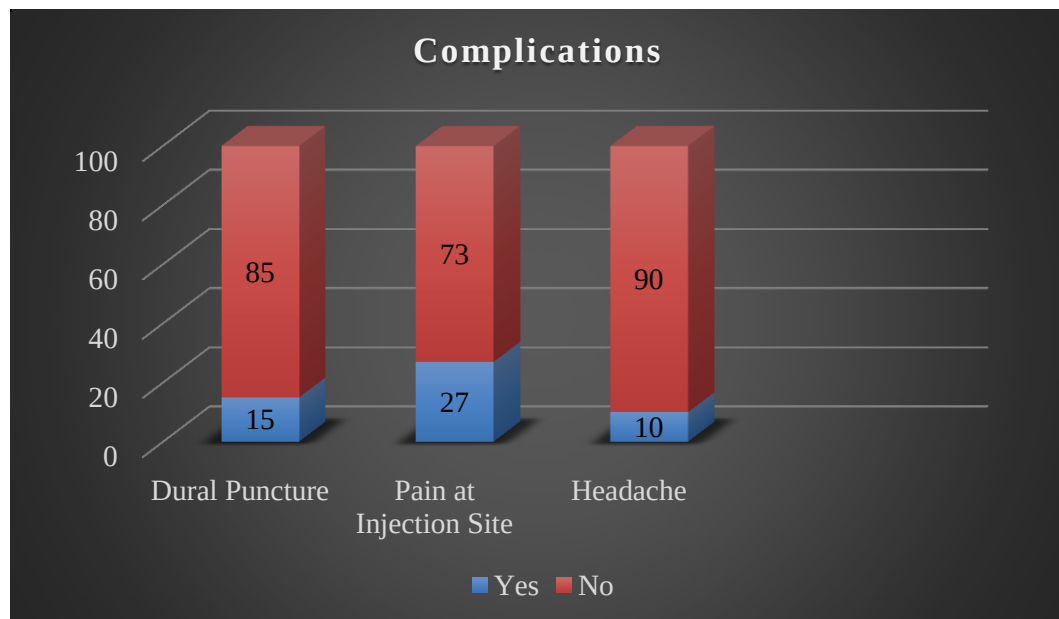


Chart 15: Distribution of patients according to Complications

Table 17: Mean Scores of SLRT before and after treatment among the Patients with SLRT ≤ 70

SLRT	Mean (n=65)	Std. Deviation
Right side (Before treatment)	63.55	7.377
Right side (after 1 month)	77.10	7.97
Right side (after 3 months)	81.29	7.125
	Mean (n=52)	Std. Deviation
Left side (before treatment)	62.35	8.32
Left side (after 1 month)	74.71	11.01
Left side (after 3 months)	79.22	10.92

In the study it was observed that mean scores of SLRT improved on both sides after treatment.

Table 18: Correlation of SLRT Score before and after treatment

SLRT	Correlation (n=65)	p value
Right side (before treatment) & Right side (after 1 month)	0.372	0.003**
Right side (before treatment) & Right side (after 3 months)	0.223	0.081
Right side (after 1 month) & Right side (after 3 months)	0.673	0.000**
	Correlation (n=52)	p value
Left side (before treatment) & Left side (after 1 month)	0.640	0.000**
Left side (before treatment) & Left side (after 3months)	0.537	0.000**
Left side (after 1 month) & Left side (after 3months)	0.829	0.000**

**p value significant at <0.01

In the study it was observed that there is significant positive correlation for SLRT on both sides before and after treatment. i.e. as the SLRT score increases before treatment there is also increase in SLRT score after treatment on both sides and VIZ.

Table 19: Paired t test showing mean difference of SLRT score before and after treatment

SLRT	Mean Difference (n=65)	Std. Deviation	t	p value
Rt (before treatment) – Rt (after 1 month)	-13.548	8.607	-12.39	0.000**
Rt (before treatment) – Rt (after 3 months)	-17.742	9.039	-15.45	0.000**
Rt (after 1 month) – Rt (after 3 months)	-4.194	6.153	-5.366	0.000**
	Mean Difference (n=52)	Std. Deviation	t	p value
Lt (before treatment) – Lt (after 1 month)	-12.35	8.56	-10.3	0.000**
Lt (before treatment) – Lt (after 3 months)	-16.86	9.53	-12.62	0.000**
Lt (after 1 month) – Left (after 3 months)	-4.51	6.42	-5.01	0.000**

**p value significant at <0.01

In the study it was observed that there is significant mean difference in SLRT scores before and after treatment at 1 month and 3 month on both sides. Hence it can be concluded that treatment improves the symptoms at a significant level.

Table 20: Mean scores of ODI (Oswestry Disability Index)

ODI score	Mean	Std. Deviation
ODI (before treatment)	45.65	5.508
ODI (after one month)	19.73	8.929
ODI (after 3 months)	9.1753	10.086

In the study it was observed that mean scores of ODI improved after treatment.

Table 21: Correlation of ODI(Oswestry Disability Index) Score before and after treatment

ODI	Correlation	Sig.
ODI (before treatment) & ODI (after 1 month)	0.416	0.000**
ODI (before treatment) & ODI (after 3 months)	0.251	0.013**
ODI (after 1 month) & ODI (after 3 months)	0.622	0.000**

**p value significant at <0.01

In the study it was observed that there is significant positive correlation for ODI scores before and after treatment. i.e. as the ODI score increases before treatment there is also increase in ODI score after treatment and VIZ.

Table 22: Paired t test showing mean difference of ODI score before and after treatment

	Mean difference	Std. Deviation	t	p value
ODI (before treatment) – ODI (after 1 month)	25.918	8.316	30.694	0.000**
ODI (before treatment) – ODI (after 3 months)	36.47423	10.20528	35.200	0.000**
ODI (after 1 month) – ODI (after 3 months)	10.55670	8.33038	12.481	0.000**

**p value significant at <0.01

In the study it was observed that there is highly significant mean difference in ODI scores before and after treatment at 1 month and 3 month. Hence it can be concluded that treatment improves the symptoms at a significant level.

Table 23: Mean scores of VAS (Visual Analogue Score)

VAS score	Mean	Std. Deviation
VAS (before treatment)	7.45	.842
VAS (after 1 month)	2.95	1.278
VAS (after 3 months)	1.46	1.528

In the study it was observed that mean scores of VAS reduced after treatment.

Table 24: Correlation of VAS(Visual Analogue Score) before and after treatment

VAS Score	Correlation	p value
VAS (before treatment) & VAS (after 1 month)	0.342	0.001**
VAS (before treatment) & VAS (after 3 months)	0.312	0.002**
VAS (after 1 month) & VAS (after 3 months)	0.626	0.000**

**p value significant at <0.01

In the study it was observed that there is highly significant positive correlation for VAS scores before and after treatment. i.e. as the VAS score increases before treatment there is also increase in ODI score after treatment and VIZ.

Table 25: Paired t test showing mean difference of VAS score before and after treatment

	Mean difference	Std. deviation	t value	p
VAS (before treatment) – VAS (after 1 month)	4.505	1.268	35.004	0.000**
VAS (before treatment) – VAS (after 3 months)	5.990	1.496	39.420	0.000**
VAS (after 1 month) & VAS (after 3 months)	1.485	1.234	11.847	0.000**

**p value significant at <0.01

In the study it was observed that there is highly significant mean difference in VAS scores before and after treatment at 1 month and 3 month. Hence it can be concluded that treatment improves the VAS score at a significant level.

Table 26: Distribution of patients according to Patient satisfaction

Patient satisfaction	Frequency	Percent
Excellent	38	38.0
Fair	14	14.0
Good	39	39.0
Poor	9	9.0
Total	100	100.0

In the study it was observed that 39% of patients had Good satisfaction, 38% had excellent satisfaction, 14% had fair satisfaction and only 9% had poor satisfaction after treatment.

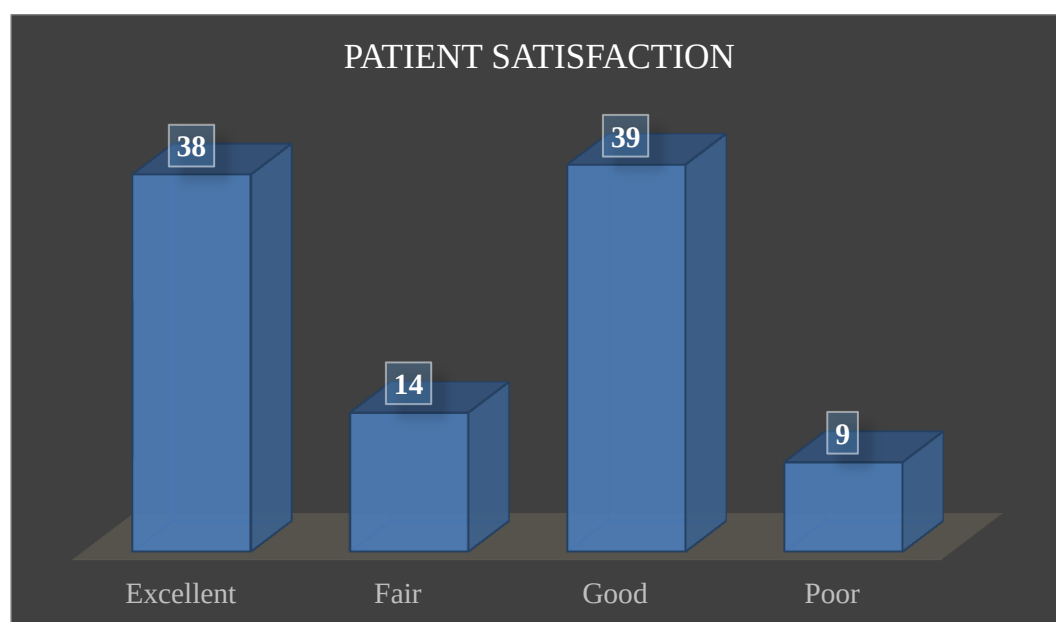


Chart 18: Bar diagram showing distribution of patients according to Patient satisfaction

Table 27: Comparison of ODI(Oswestry Disability Index) score

ODI Score	Before treatment	At 1 month	At 3 months
0-20%	0	63	90
20 to 40%	24	36	9
40 to 60%	76	1	1
Total	100	100	100

In the study it was observed that ODI score improved or reduced after treatment at 1st month and 3rd month.

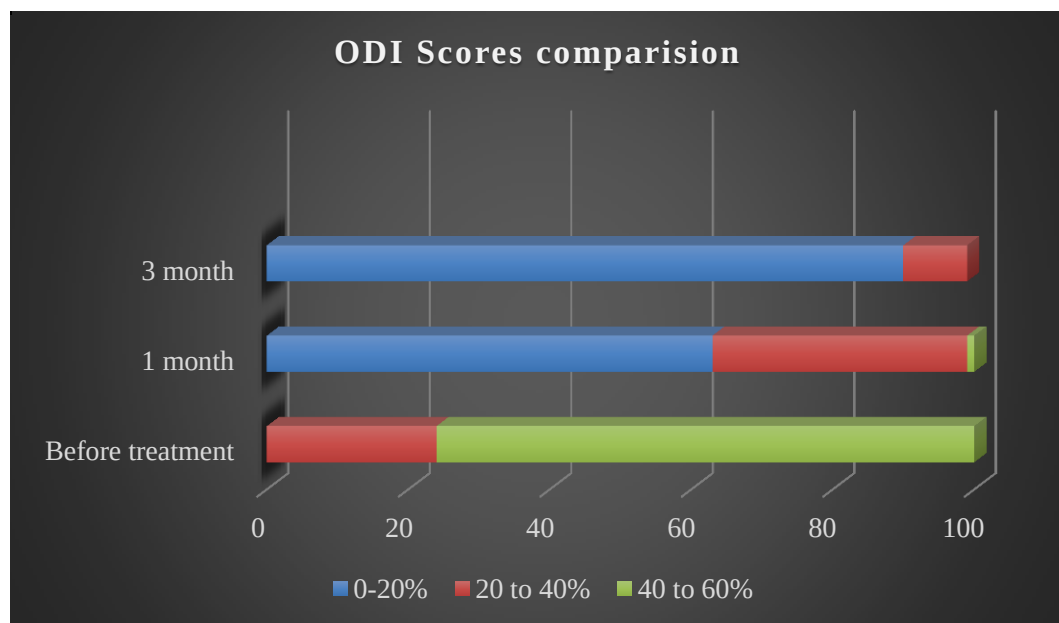


Chart 17: Comparison of ODI score

Table 28: Association of ODI score before treatment with Patient satisfaction

ODI before	Patient satisfaction				Total	
	Excellent	Fair	Good	Poor		
20 to 40%	14	4	6	0	24	$X^2=8.025$ df =3, p =0.045
40 to 60%	24	10	33	9	76	
Total	38	14	39	9	100	

In the study it was observed that there was slightly significant association between ODI score before treatment and Patient satisfaction. p value <0.05

Table 29: Association of ODI(Oswestry Disability Index) score after 1 month with Patient satisfaction

ODI After 1 month	Patient satisfaction				Total	
	Excellent	Fair	Good	Poor		
0 to 20%	32	3	24	4	63	$X^2=28.774$ df =6, p = 0.001**
20 to 40%	6	11	15	4	36	
40 to 60%	0	0	0	1	1	
Total	38	14	39	9	100	

In the study it was observed that there was highly significant association between ODI score after treatment at 1 month and Patient satisfaction. p value <0.001

**Table 30: Association of ODI(Oswestry Disability Index) score after 3 month
with Patient satisfaction**

ODI After 3 month	Patient satisfaction				Total	
	Excellent	Fair	Good	Poor		
0 to 20%	38	10	39	3	90	
20 to 40%	0	4	0	5	9	
40 to 60%	0	0	0	1	1	
Total	38	14	39	9	100	

$\chi^2=49.277$ df
=6,
p = 0.0001**

In the study it was observed that there was very high significant association between ODI score after treatment at 1 month and Patient satisfaction. p value <0.0001. Hence it can be concluded that as the duration of treatment increased there was significant improvement in patient satisfaction.

Discussion

DISCUSSION

Low backache due to lumbar nerve root compromise is a debilitating problem frequently afflicting otherwise healthy individuals. Pain and reduced mobility severely compromise their quality of life. The aim of any therapy should be to return to a normal lifestyle as soon as possible, whether it be by treatment of the underlying cause or merely by symptomatic relief. It is recommended that surgery to be undertaken only when conservative management fails.⁶¹

Etiology of low backache remains controversial. Degeneration, herniation, or by an inflammatory reaction could be responsible for lower backache & sciatica⁵⁹. An epidural injection can decrease inflammation in the epidural space as well as the pain in the affected nerve root.

In this study, a total of hundred patients with lumbosacral radiculopathy were evaluated for a period of minimum 3 months post epidural injection of steroid, in terms of effectiveness. The mean age group was 42.53 and 51% were in the middle age group between 30 to 50 years.

In the study it was observed that the majority i.e. 65% of patients had symptoms <12 months.

There was statistically significant improvement in SLRT from 63.5 mean preintervention to 81.29 mean 3 months post steroid injection on right side, 62.35 to 79.22 on left side. These results were comparable to a study conducted by Karppinen⁶² who studied results in 160 patients with sciatica, showed significant improvement in SLRT following epidural injection of methylprednisolone and anesthetic.

We observed that 67% of patients had symptomatic lesion at single level, 26% of patients had lesions at 2 levels and 6% of patients had lesions at multiple level. This was similar to the observation made by Wilson-MacDonald²⁸ where 80% patients

showed single level ,16% at 2 levels and 4% multiple.We observed in 40% of patients S1 root was affected, in 36% of patients L5 root was affected.

In this study we observed that the mean scores of ODI improved from 45.65 prior to treatment to 9.17 at 3 months.Similar observation was made by N. K. Arden et al²⁷ who showed improvement from 44 to 12 at end of 3 months.

VAS before treatment was 7.45 mean, 1.46 at 3 months.These results were comparable to a study conducted by Pirbudak et al⁶³ who showed significant improvement in 92 patients with sciatica treated with epidural steroid.

In the study it was observed that 39% of patients had Good satisfaction, 38% had excellent satisfaction, 14% had fair satisfaction and only 9% had poor satisfaction after treatment.

There were no major complications like meningitis,allergic reactions or cauda equine syndrome.In the study it was observed that 15% of patients had Dural puncture, 27% of patients had pain at injection site and 10% had headache.these results were comparable to a study done by Michael J.DePalma⁸ who showed injection site pain in 17.1% ,headache in 3.1%.

Karppinen et al⁶² demonstrated that either local steroid injection or saline injection around a nerve root would improve referred pain, but found that when a combination of steroid and local anaesthetic were used there was a rebound phenomenon after three to six months, with increasing deterioration of symptoms the longer the period between the injection and follow-up.Our patients had significant improvement by 1 month and only 3 patients underwent surgery due to poor relief. It has been shown that epidural steroid injection with local anaesthetic is better than injection of local anaesthetic alone and thus the steroid does appear to be important in reducing pain in these patients.⁶³

Many studies show that only 10% to 15% of patients with sciatica presenting to a specialist eventually require operation. The reason for the low take up of surgery may have been that many of the older patients did not wish to undergo surgical treatment. A small number of patients refused to have surgery despite the failure to improve over the course of time because of the perceived risks.

This study helps us to say that epidural steroid injection in early stage of back ache helps in relief of symptoms, early return to normal daily activities and work. Epidural steroid injection helps in patients in whom spontaneous improvement is expected by accelerating the rate of recovery. However in long term followup it may not preclude the need for surgery.

Conclusion

CONCLUSION

This study was done to evaluate the efficacy of epidural steroid in low backache with radiculopathy. The results statistically showed significant improvement in terms of pain relief measured with Visual Analogue Scale score and patient satisfaction, clinical improvement measured by SLRT and functional return measured by Oswestry Disability Index. Complications encountered were injection site pain and headache.

We conclude that epidural steroid injection is an effective non surgical treatment option for patients with low backache and radicular leg pain. Epidural steroid should be considered before surgical intervention. After early adequate pain control, rehabilitation can be effective and function can be restored. Epidural steroid in selected cases fulfills this criteria.

Bibliography

BIBLIOGRAPHY

1. Thomas von Rothenburg, Robert Drescher, Odo Koester, Gebhard Schmid. Magnetic resonance imaging of the lumbar spine after epidural and nerve root injection therapy: evaluation of soft tissue changes. *Clinical Imaging* 2006;30:331–334.
2. Bart W. Koes , Rob J.P.M. Scholten , Jan M.A. Mens, Lex M. Bouter. Efficacy of epidural steroid injections for low-back pain and sciatica: a systematic review of randomized clinical trials. *Pain* 1995; 63:279-288.
3. Botwin KP et al. Fluoroscopically guided lumbar transforaminal epidural steroid injections in degenerative lumbar stenosis. *Am J Phys Med Rehabil* 2002;81:898-905.
4. Wilson MacDonald J, Burt G, Griffith D, Glynn C. Epidural steroid injection for nerve root compression. *J Bone Joint Surg* 2005;87-B:352-5.
5. Ahsan K, Mahmud. A clinical trial of epidural steroid injection in the treatment of low back pain. *Dinajpur Med Col J* 2011;4(1).
6. Iversen T et al., Effect of caudal epidural steroid or saline injection in chronic lumbar radiculopathy: multicentre, blinded, randomized controlled trial. *BMJ* 2011;343.
7. Hee-Seung Nam, Yong Bum Park. Effects of Transforaminal Injection for Degenerative Lumbar Scoliosis Combined with Spinal Stenosis. *Ann Rehabil Med* 2011; 35: 514-523.

8. Michael J. DePalma, Curtis W. Slipman, Evidence-informed management of chronic low back pain with epidural steroid injections. *The Spine Journal* 2008;45-55.
9. Nawani DP, Agarwal S, Asthana V, Single shot epidural injection for cervical and lumbosacral radiculopathies. *Korean J Pain* 2010;23:254-257.
10. Willard G. Hession, Jeffrey D. Stanczak, Kirkland W. Davis, James J. Choi. *Seminars in Roentgenology* 2004;39:7-23.
11. Viner, Norman. Intractable Sciatica – A Sacral Epidural Injection. *The Canadian Medical Association Journal* 1925;15:630.
12. Cyriax, J. (1957) *Textbook of Orthopaedic Medicine*, Third Edn, p. 644. Cassell, London.
13. Sicard A. Les injections medicamenteuses extra-durales par voie sacro coccygienne. *Compt Rend Soc De Biol* 1901; 53: 396–8.
14. McManus, Frank, Sheehan, James. Cauda equina compression following epidural injection for disc prolapse. *Irish Journal of Medical Science*. 1975;144:447-448.
15. Pagés F. Anesthesia metamerica. 1921;3:3–30.
16. Dogliotti AM. A new method of block: segmental peridural spinal anesthesia. *Am J Surg* 1933; 20: 107–18.
17. Goebert, H. W. Jallo, S. J, Gardner, W. J. & Wasmuth, C. E. Painful radiculopathy treated with epidural injections of Procaine & Hydrocortisone Acetate. *Anaesthesia & Analgesia* 1961;40:13.

18. Coomes, E.N.A comparison between epidural anaesthesia & bed rest in sciatica. British Medical Journal 1961;1:20.
19. Becker, A., Held, H., Redaelli, M., Strauch, K., & Chenot, J. F.. Low back pain in primary care: costs of care & prediction of future health care utilization. Spine 2010;35:1714-1720.
20. Beliveau, P. A comparison between epidural anaesthesia with & without corticosteroid in the treatment of sciatica. Rheumat. P h y s. M e d 1971; 11:40-43.
21. Warr, A. C, Wilkinson, J. A. Bum, J. M. B. ,Lanydon, L. Chronic lumbo-sciatic syndrome treated by epidural injection & manipulation. Practitioner 1972;209:53-59.
22. Dilke, T.F.W., Burry, H.C., Grahame, R.Extradural corticosteroid injection in management of lumbar nerve root compression. British Medical Journal 1973;2:635.
23. R. K. Sharma. Indications, technique & results of caudal epidural injection for lumbar disc retropulsion. Postgraduate Medical Journal 1977; 53:1-6.
24. Carette S, Leclaire R, Marcoux S, Morin F, Blaise GA, St-Pierre A, Truchon R, Parent F, Levesque J, Bergeron V, Montminy P, Blanchette C. Epidural corticosteroid injections for sciatica due to herniated nucleus pulposus. N Engl J Med 1997; 336:1634-1640.
25. Manchikanti L, Abdi S, Lucas LF. Evidence synthesis & development of guidelines in interventional pain management. Pain Physician 2005; 8:73- 86.

26. Elva G, Delport, Anthony R, Cucuzzella, Julie K, Marley, Christine M, Pruitt, J, Rush Fisher. Treatment of lumbar spinal stenosis with epidural steroid injections: A Retrospective outcome study. Arch Phys Med Rehabil 2004; 85: 479-84.
27. Arden NK, Price C, Reading I, Stubbing J, Hazelgrove J, Dunne C, Michel M, Rogers P, Cooper C, WEST Study Group. A multicentre randomized controlled trial of epidural corticosteroid injections for sciatica: the WEST study. Rheumatology (Oxford) 2005; 44: 1399-1406.
28. Wilson-MacDonald J, Burt G, Griffin D, Glynn C. Epidural steroid injection for nerve root compression: a randomized, controlled trial. J Bone Joint Surg Br 2005; 87-B: 352-355.
29. Ackerman, W. E., & Ahmad, M. The efficacy of Lumbar epidural steroid injections in patients with lumbar disc herniations. Anesthesia Analog 2007; 104: 1217-1222.
30. Abdi, S., Datta, S., Trescot, A. M., Schultz, D. M., Adlaka, R., Atluri, S. L., et al. Epidural steroids in the management of chronic spinal pain. Pain Physician 2007; 10: 185-212.
31. Kenneth Botwin, Lee Ann Brown, Mark Fishman, Sanjiv Rao. Fluoroscopically Guided Caudal Epidural Steroid Injections in Degenerative Lumbar Spinal Stenosis. Pain Physician 2007; 10: 547-558.
32. Parr, A. T., Diwan, S., Abdi, S. Lumbar interlaminar injections in managing chronic low back & lower extremity pain: a systematic review. Pain Physician 2009; 12: 163-188.

33. Sayegh, E. F., Kenanidis, I. E., Papavasiliou, A. K., Potoupnis, E. M., Kirkos, M.J., & Kapenetenos, A. G. Efficacy of Steroid & Nonsteroid Caudal Epidural For Lowback Pain and Sciatica. *Spine* 2009;34:1441-1447.
34. Digambar Prasad Nawani, Sanjay Agrawal, Veena Asthana. Single Shot Epidural Injection for Cervical and Lumbosacral Radiculopathies: A Preliminary Study. *Korean J Pain* 2010; 23: 254-257.
35. Jafar Mobaleghi, Faramarz Allahdini, Karim Nasser, Behzad Ahsan, Shoaleh Shami, Mansour Faizi, Fardin Gharibi. Comparing the effects of epidural methylprednisolone acetate injected in patients with pain due to lumbar spinal stenosis or herniated disks: a prospective study. *International Journal of General Medicine* 2011;4 875–878.
36. Laxmaiah Manchikanti, Kimberly A. Cash, Carla D. McManus, Kim S. Damron, Vidyasagar Pampati, Frank J.E. Falco. Lumbar Interlaminar Epidural Injections in Central Spinal Stenosis: Preliminary Results of a Randomized, Double-Blind, Active Control Trial. *Pain Physician* 2012; 15:51-63.
37. Stitz MY, Sommer HM. (1999) Accuracy of blind versus fluoroscopically guided caudal epidural injection. *Spine* ;24:1371–6.
38. Canale, S. T., & Beaty, J. H. (2008). *Cambell's Operative Orthopaedics* (11th Edition ed., Vol. 2). Philadelphia: MOSBY Elsevier.
39. Duthie B. R. & George Bentley; *Mercer's Orthopedic Surgery* 10th Edition (2003).

40. Grey's Anatomy. The complete 20th U.S. edition of Gray's Anatomy of the Human Body, published in 1918.
41. Bernard J. Alpers. Sciatica problem. *Canad. M. A. J.* Aug. 1952, vol. 67.
42. John A. O'Connell. Protrusion of the lumbar intervertebral disc. A Clinical Review Based on Five Hundred Cases Treated by Excision of the Protrusion. *J. Bone Jt Surg* 1951; 33B:8.
43. Roaf, R. A study of the mechanics of spinal injuries. *Journal of Bone & Joint Surgery* 1960;42:810-823.
44. Lindblom. Experimental Ruptures of Intervertebral Discs in Rats. *J of Bone Joint Surg* 1952;34A:123-8.
45. Hirsch C, Paulson S, Sylven B, Snellman O. Biophysical & physiological investigations on cartilage & other mesenchymal tissues. Characteristics of human nuclei pulposi during aging. *Acta Orthop Scand.* 1953; 22(3):175–183.
46. Edgar, Nundy, S. Innervation of the spinal dura mater. *Journal of Neurology, Neurosurgery & Psychiatry* 1966;29:530-534.
47. Jack Stevens. Low Back Pain. *Medical Clinics of North America* 1968;52.
48. Walter S. Bartynski, Stephen Z. Grahovac, William E. Rothfus. Incorrect Needle Position during Lumbar Epidural Steroid Administration: Inaccuracy of Loss of Air Pressure Resistance and Requirement of Fluoroscopy and Epidurography during Needle Insertion. *Am J Neuroradiol* 2005;26:502–505.

49. Tandon PN, Sankaran B. Cauda equina syndrome due to lumbar disc prolapse. Indian J Orthop 1967; 1:112-9.
50. Shinnars, Hamby. The results of surgical removal of protruded lumbar intervertebral discs, J. Neurosurg 1944; 1:117.
51. O'Connell, J. E. A. Protrusions of the lumbar intervertebral discs. A clinical review based on five hundred cases treated by excision of the protrusion. J. Bone Jt Surg 1951.88:8-30.
52. Raaf, J., & Berglund, G.: Results of operations for lumbar protruded intervertebral disc, J. Neurosurg 1949;6:160.
53. Steven J. Atlas, Richard A. Deyo. Evaluating and Managing Acute Low Back Pain in the Primary Care Setting. J Gen Intern Med 2001;16:120-131.
54. McMorland, G., Suter, E., Casha, S., du Plessis, S. J., Hurlbert, R. J. Manipulation or microdiscectomy for sciatica? A prospective randomized clinical study. J Manipulative Physiol Ther 2010;33:576-584.
55. Ramamurthi B. (1974) Spinal Arachnoiditis. Indian Journal of Medical Sciences.15:776-781.
56. R. K. Sharma. Indications, technique and results of caudal epidural injection for lumbar disc retropulsion. Postgraduate Medical Journal 1977;53:1-6.
57. Kenneth P. Botwin, Robert D. Gruber. Lumbar epidural steroid injections in the patient with lumbar spinal stenosis. Phys Med Rehabil Clin N Am 2003;14:121–141.

58. Carron H. Relieving pain with nerve blocks. *Geriatrics* 1978; 33:49 –57.
59. Boswell MV, Hansen HC, Trescot AM, et al. Epidural steroids in the management of chronic spinal pain & radiculopathy. *Pain Physician* 2003; 6: 319–34.
60. Kim D, Brown J. Efficacy and safety of lumbar epidural dexamethasone versus methylprednisolone in the treatment of lumbar radiculopathy: a comparison of soluble versus particulate steroids. *Clin J Pain*. Jul-Aug 2011;27(6):518-22.
61. Kane WJ: The incidence rate of laminectomy. Presented at the International Society for the study of the lumbar spine meeting. New Orleans, May, 1980.
62. Karppinen J, Malmivaara A, Kurunlahti M, Kyllonen E, Pienimäki T, Nieminen P, Ohinmaa A, Tervonen O, Vanharanta H. Periradicular infiltration for sciatica. *Spine* 2001; 26:1059-1067.
63. Rogers P, Nash T, Schiller D, Norman J. Epidural steroids for sciatica. *Pain Clinic* 1992;5:67-72.

Annexures

ANNEXURE

Oswestry Disability Index(ODI)

Section 1 – Pain Intensity

- ☐ I have no pain at the moment.
- ☐ The pain is very mild at the moment.
- ☐ The pain is moderate at the moment.
- ☐ The pain is fairly severe at the moment.
- ☐ The pain is very severe at the moment.
- ☐ The pain is the worst imaginable at the moment.

Section 2 – Personal Care (washing, dressing, etc.)

- ☐ I can look after myself normally but it is very painful.
- ☐ I can look after myself normally but it is very painful.
- ☐ It is painful to look after myself and I am slow and careful.
- ☐ I need some help but manage most of my personal care.
- ☐ I need help every day in most aspects of my personal care.
- ☐ I need help every day in most aspects of self-care.
- ☐ I do not get dressed, wash with difficulty, and stay in bed.

Section 3 - Lifting

- ☐ I can lift heavy weights without extra pain.
- ☐ I can lift heavy weights but it gives extra pain.
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned (i.e. on a table).
- ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.

- ☐ I can lift only very light weights.
- ☐ I cannot lift or carry anything at all.

Section 4 – Walking

- ☐ Pain does not prevent me walking any distance.
- ☐ Pain prevents me walking more than 1 mile.
- ☐ Pain prevents me walking more than $\frac{1}{4}$ of a mile.
- ☐ Pain prevents me walking more than 100 yards.
- ☐ I can only walk using a stick or crutches.
- ☐ I am in bed most of the time and have to crawl to the toilet.

Section 5 – Sitting

- ☐ I can sit in any chair as long as I like.
- ☐ I can sit in my favorite chair as long as I like.
- ☐ Pain prevents me from sitting for more than 1 hour.
- ☐ Pain prevents me from sitting for more than $\frac{1}{2}$ hour.
- ☐ Pain prevents me from sitting for more than 10 minutes.
- ☐ Pain prevents me from sitting at all.

Section 6 – Standing

- ☐ I can stand as long as I want without extra pain.
- ☐ I can stand as long as I want but it gives me extra pain.
- ☐ Pain prevents me from standing more than 1 hour.
- ☐ Pain prevents me from standing for more than $\frac{1}{2}$ an hour.
- ☐ Pain prevents me from standing for more than 10 minutes.
- ☐ Pain prevents me from standing at all.

Section 7 – Sleeping

- ☐ My sleep is never disturbed by pain.
- ☐ My sleep is occasionally disturbed by pain.
- ☐ Because of pain, I have less than 6 hours sleep.
- ☐ Because of pain, I have less than 4 hours sleep.
- ☐ Because of pain, I have less than 2 hours sleep.
- ☐ Pain prevents me from sleeping at all.

Section 8 – Sex life (if applicable)

- ☐ My sex life is normal and causes no extra pain.
- ☐ My sex life is normal but causes some extra pain.
- ☐ My sex life is nearly normal but is very painful.
- ☐ My sex life is severely restricted by pain.
- ☐ My sex life is nearly absent because of pain.
- ☐ Pain prevents any sex life at all.

Section 9 – Social Life

- ☐ My social life is normal and cause me no extra pain.
- ☐ My social life is normal but increases the degree of pain.
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests, i.e. sports.
- ☐ Pain has restricted my social life and I do not go out as often.
- ☐ Pain has restricted social life to my home.
- ☐ I have no social life because of pain.

Section 10 – Traveling

- ☐ I can travel anywhere without pain.
- ☐ I can travel anywhere but it gives extra pain.

- ☐ Pain is bad but I manage journeys of over two hours.
- ☐ Pain restricts me to short necessary journeys under 30 minutes.
- ☐ Pain prevents me from traveling except to receive treatment.

Section 11 - Previous Treatment

Over the past three months have you received treatment, tablets or medicines of any kind for your back or leg pain? Please check the appropriate box.

- ☐ No
- ☐ Yes (if yes, please state the type of treatment you have

SCORING TECHNIQUE FOR THE OSWESTRY LOW BACK DISABILITY QUESTIONNAIRE

1. Each of the 10 sections is scored separately (0 to 5 points each) and then added up (max. total = 50).

Example:

Section 1. Pain Intensity Point Value

- A. ____ I have no pain at the moment 0
- B. ____ The pain is very mild at the moment 1
- C. ____ The pain is moderate at the moment 2
- D. ____ The pain is fairly severe at the moment 3
- E. ____ The pain is very severe at the moment 4
- F. ____ The pain is the worst imaginable 5

2. If all 10 sections are completed, simply double the patient's score.

3. If a section is omitted, divide the patient's total score by the number of sections completed times 5.

Formula: Patient's Score

$X 100 = \text{_____ \% DISABILITY}$

No. of sections completed x 5

Example:

If 9 of 10 sections are completed, divide the patient's score by $9 \times 5 = 45$.

Patient's Score 22

Number of sections completed: $9 (9 \times 5 = 45)$

$22/45 \times 100 = 48\%$ disability

Interpretation of disability scores :

0-20% Minimal disability :Can cope with most ADLs. Usually no treatment is needed, apart from advice on lifting, sitting, posture, physical fitness, and diet. In this group, some patients have particular difficulty with sitting and this may be important if their occupation is sedentary (typist, driver, etc.)

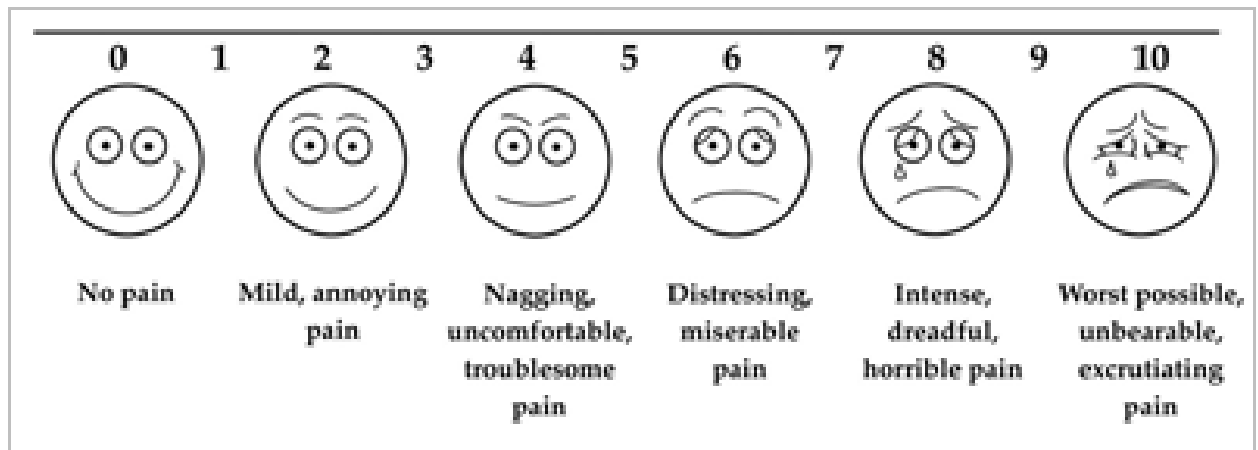
20-40% Moderate disability: This group experiences more pain and problems with sitting, lifting, and standing. Travel and social life are more difficult and they may well be off work. Personal care, sexual activity, and sleeping are not grossly affected, and the back condition can usually be managed by conservative means.

40-60% Severe disability: Pain remains the main problem in this group of patients, but travel, personal care, social life, sexual activity, and sleep are also affected. These patients require detailed investigation.

60-80% Crippled: Back pain impinges on all aspects of these patients' lives both at home and at work. Positive intervention is required.

80-100% :These patients are either bed-bound or exaggerating their symptoms. This can be evaluated by careful observation of the patient during the medical examination.

Visual Analogue Scale



INFORMED CONSENT

My doctor has advised me that due to my medical condition, the chances for my improvement or recovery will be significantly helped by receiving epidural steroid. The doctor has explained the benefits that are expected from the procedure, as well, the risk. I understand that this procedure will help to reduce my symptoms. I also understand the complications (i.e. allergic reaction, head ache, injection site pain, rarely meningitis, cauda equina syndrome) and chances of persistence of symptoms. I would give my consent to undergo the procedure and participate in the study and follow up regularly.

I do not hold the treating doctor, hospital staff for any untoward consequences.

Witness:

Patient's Name:

Time:

Patient's signature:

Date:

**EFFICACY OF EPIDURAL METHYL PREDNISOLONE IN LUMBAR
RADICULOPATHY –PROFORMA**

NAME:

AGE:

SEX:

HOSPITAL NUM:

Date Of Admission:

Date Of Procedure:

Date Of Discharge:

ADDRESS:

OCCUPATION:

HISTORY OF PRESENTING ILLNESS:

Pain in low back region-

Onset:

Duration:

Event related to onset-

Trivial fall

Inappropriate lifting of weight

Direct trauma

Uneventful

Nature:

Intensity:

Radiation: Unilateral or Bilateral

Aggravating factors:

Relieving factors:

Numbness in the lower limbs:(Y/N)

If yes site of numbness:

Weakness in the lower limbs:(Y/N)

If yes specify:

H/O previous similar episodes and duration:

Bowel and bladder disturbances:

Limitation of daily activity:(Y/N)

If yes specify:

TREATMENT HISTORY:

Bed rest:(y/n) if yes, duration:

Physiotherapy:(y/n) if yes, specify:

Massage:(y/n)

Taction:(y/n)

Epidural steroid:(y/n) if yes,drug given and time:

Surgery:(y/n) if yes,specify:

OTHER MEDICAL ILLNESS:

MENSTRUAL History:

HABITS: smoking(y/n): alcohol intake(y/n):

Physical examination:

Built and weight:

Vital signs- Pulse(beats/mt): BP(mm of Hg):

SYSTEMIC EXAMINATION:

CVS:

RS:

ABDOMEN:

MUSCULO SKELETAL EXAMINATION OF SPINE:

Gait:

Attitude:

Inspection:

Palpation:

Tenderness-

Spasm-

Deformity:

Movements:

Flexion-

Extension-

Lateral flexion-

Rotation-

Others:

Special tests:

SLRT:

Rt

Lt

Active-

Passive-

Cross SLRT:

Lasegue test:

Femoral nerve stretch test:

Bow string test:

Neurological examination:

HMF:

Cranial nerves:

Sensations:

Pain-

Temperature-

Fine touch-

Crude touch-

Motor:

Rt

Lt

Bulk- Thigh

Calf

Tone-

Power-

Hip

Flexion

Extension

Abduction

Adduction

Knee

Flexion

Extension

Ankle

DF

PF

EHL

EDL

Inversion

Eversion

Reflexes:

Superficial-

Plantar

Cremastic

Deep tendon-

Knee jerk

Ankle jerk

Pre procedure Oswestry Disability Index (ODI)score:

1. Pain Intensity
2. Personal Care (washing, dressing, etc.)
3. Lifting
4. Walking
5. Sitting
6. Standing
7. Sleeping
8. Sex life (if applicable)
9. Social Life
- 10.Traveling
- 11.Previous Treatment

Total score:

% DISABILITY:

VAS:

Investigations:

Routine Blood Tests:

Hb(gm%):

BT:

HIV/HbsAg:

CT:

Plain X-ray LS Spine—AP &Lateral:

MRI findings:

Proceure findings:

Procedure complications

Post procedural complications:

Post procedural status:

Pain/Radiculopathy :

Degree of improvement(VAS)-

Radiation/numbnesss-

Ability to walk-

Sensory status:

Motor functions:

Follow up:

ODI score at 1 month-

% DISABILITY

VAS:

SLRT:

ODI score at 3 months-

% DISABILITY

VAS:

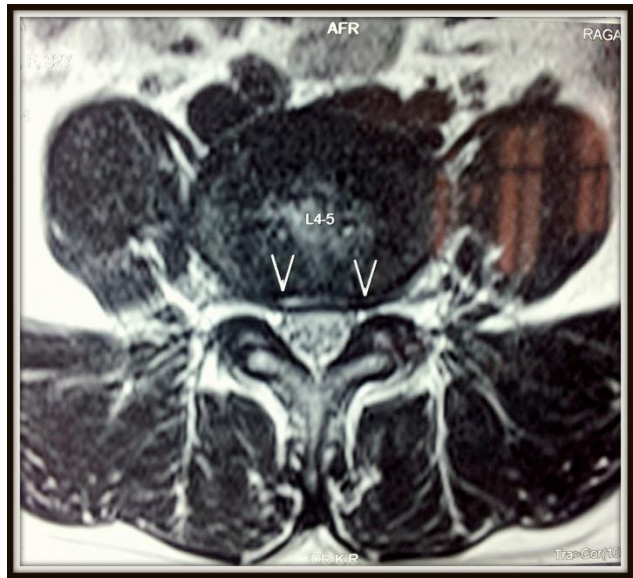
SLRT:

Formula: Patient's Score X 100 = _____ %

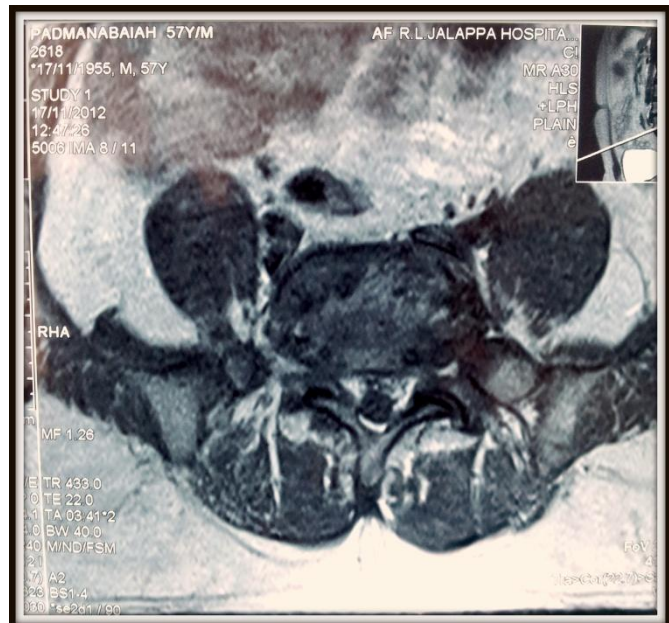
DISABILITY

No. of sections completed x 5

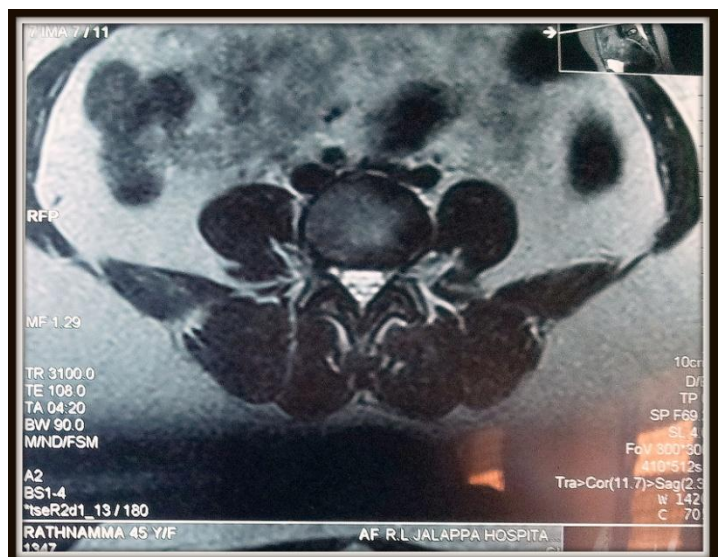
Case 1: A 32 yr female with Lt sided radiculopathy; MRI showing IVDP L4-5
compressing L>R roots (pt name: Manjula, Sl.no:25)



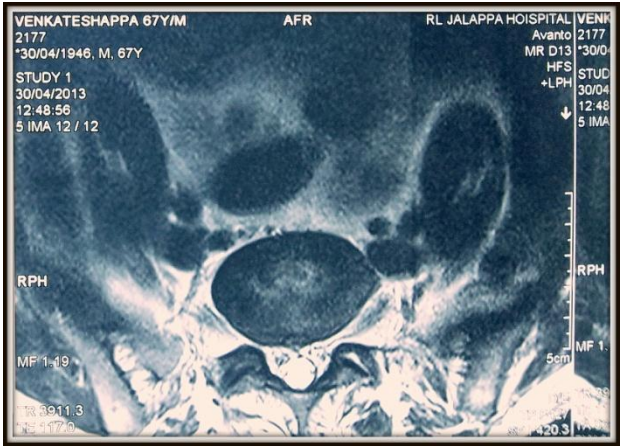
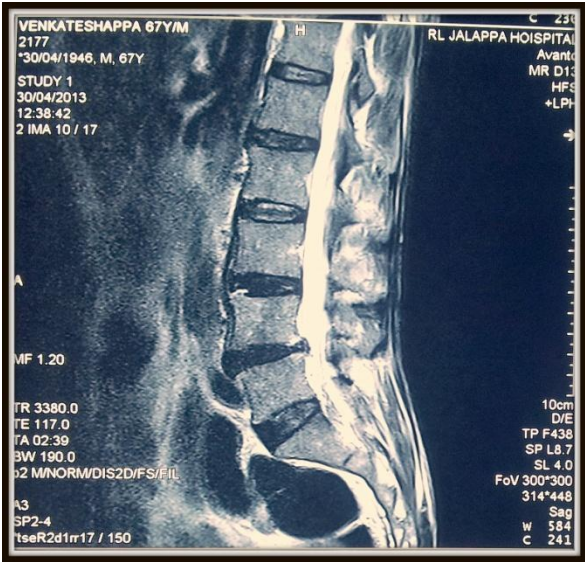
Case 2: MRI showing IVDP L4-5, L5-S1 with B/L root compression, patient had left sided symptoms. (pt name: Padmanabaihan, Sl.no: 29)



Case 3:IVDP L4-5,L5-S1,patient had left side radiculopathy(pt name:Rathnamma,SI no:57)



Case 4: IVDP L4-5 Central and paracental(Venkateshappa,Sl no:64)



X-Ray showing narrowed L4-5 space(Aziz ur rehman,Sl.no:1)



X-Ray showing narrowing of L4-5,L5-S1(suresh,Sl.no:12)



KEY TO MASTER CHART

Hosp.No:Hospital Number	Surg:Surgery in followup
Dur:Duration of symptoms	R:Right
R/A : Rest / Analgesics	L:Left
Tr : Traction	Pt.Satisf:Patient Satisfaction
Ph : Physiotherapy	ODI:Oswestry Disability Index in %
DM:Diabetes Mellitus	VAS:Visual Analogue Scale in centi meters
HTN:Hypertension	Inj.St.P:Injection Site Pain
y:Yes/Present	Root.in:Root invoved
n:No/Absent	D.P:Dural Puncture

Sl.No	Name	Age	Sex	Occupation	Hosp.No	Symptoms		Previous treatment			Comorbid		Clinical signs				ODI	VAS	MRI findings	Root.in	Complications			Surg	Followup(1mon)				Follow Up(3mon)					
						Sciatica	Dur	R/A	Phys	Tr	DM	HTN	Spasm	SLRT		Neurological Signs						D.P	Inj.St.P	Headache		ODI	VAS	SLRT		ODI	VAS	SLRT		Pt.Satisf
														R	L	Sensory	Motor											R	L			R	L	
1	Aziz ur rehman	40	m	bussiness	755149	L	2 yrs	y	y	y	n	n	y	80	45	y	y	52	9	IVDP L4-L5	L5	n	y	n	n	22	5	80	70	22	5	80	80	Fair
2	Ramdas	30	m	labourer	755172	B/L	1 yr	y	y	y	n	n	y	70	70	n	n	48	7	Bulge L4-L5,L5-S1	L5,S1	n	y	n	n	22	3	90	90	10	2	90	90	Good
3	Ramesh	37	m	ration shop	764585	B/L	6 mon	y	n	n	n	n	y	70	70	n	n	44	7	IVDP L5-S1	S1	n	n	n	n	20	2	90	90	0	0	90	90	Excellent
4	Thimarayappa	40	m	agriculture	770580	B/L	3 yrs	y	n	n	n	n	n	60	60	y	y	50	8	IVDP L4-5	L5	n	n	n	n	20	4	80	80	10	2	80	80	Good
5	Chinnakka	54	f	dependent	772066	R	5 yrs	y	y	y	n	n	n	60	80	n	n	50	8	IVDP L5-S1	S1	y	y	y	n	30	4	70	90	10	2	80	90	Good
6	Suchitra	28	f	house wife	774050	R	2yrs	y	y	y	n	n	n	60	80	n	y	42	7	IVDP L2-L3	L3	n	n	n	n	20	4	80	90	0	0	90	90	Excellent
7	Geetha	22	f	house wife	774564	B/L	1 yr	y	y	y	n	n	y	70	70	n	n	46	8	Multiple level	L5,S1	n	n	n	n	20	3	90	90	0	0	90	90	Excellent
8	Lakshmamma	55	f	house wife	776492	R	4 yrs	y	n	n	n	n	y	70	80	n	y	46	8	IVDP L5-S1	L5	n	y	n	n	20	3	80	80	10	2	80	80	Good
9	Raja shekar	30	m	agriculture	777144	L	3 yrs	n	n	n	n	n	y	80	60	n	y	40	7	Multiple level	L5,S1	n	n	n	n	0	1	80	80	0	1	80	80	Excellent
10	Muniyamma	55	f	house wife	782527	L	5 yrs	y	n	n	n	n	n	80	60	n	n	46	7	IVDPL5-S1	S1	n	n	n	n	10	2	80	70	0	1	80	80	Excellent
11	Manoj kumar	38	m	agriculture	784159	R	3 yrs	n	n	n	n	n	y	60	90	n	n	40	7	IVDP L4-L5	L5	n	n	n	n	0	2	90	90	0	1	90	90	Excellent
12	Suresh	32	m	agriculture	790177	B/L	2 yrs	y	y	y	n	n	n	70	70	n	n	36	6	Bulge L4-L5,L5-S1	S1	y	y	n	n	10	2	80	80	0	1	80	80	Excellent
13	Shilpa	28	f	house wife	793920	R	8 mon	n	n	n	n	n	n	70	90	n	y	40	7	IVDP L4-L5	L5	n	n	n	n	10	4	70	90	20	4	70	90	Fair
14	Nazia begum	42	f	house wife	793993	R	6 mon	y	y	n	y	y	y	50	90	n	n	50	7	IVDP L4-L5	L5	n	n	n	n	30	3	70	80	0	1	70	80	Excellent
15	Lakshmidevamma	55	f	dependent	795357	B/L	2 mon	y	y	n	n	y	y	60	90	n	n	42	8	Multiple level	S1	n	n	n	n	10	2	90	90	0	1	90	90	Excellent
16	Munikrishna	37	m	agriculture	796576	L	6 mon	n	n	n	n	n	y	90	60	n	n	42	7	IVDP L3-L4	L4	n	n	n	n	10	3	80	80	0	1	90	90	Excellent
17	Narasamma	60	f	dependent	796602	R	6 yrs	y	y	y	y	y	y	60	60	y	y	54	8	IVDP L4-L5	L5	n	y	n	n	10	3	70	70	10	3	80	80	Good
18	Anand	58	m	bussiness	798929	B/L	3 mon	y	n	n	y	y	y	65	65	n	n	50	7	IVDP L3-L4	L4	n	n	n	n	30	5	70	70	20	3	70	70	Good
19	Nagaraj M	40	m	agriculture	799523	L	6 mon	n	n	n	n	n	n	90	70	n	n	38	6	IVDP L3-L4	L4	n	n	n	n	10	2	80	80	0	1	90	90	Excellent
20	Supriya	33	f	house wife	807475	L	8 mon	n	n	n	n	n	y	90	80	n	n	48	8	Bulge L3-L4	L4	n	n	n	n	10	2	80	80	0	2	80	80	Good
21	Basappa	45	m	labourer	809184	B/L	5 yrs	y	y	y	n	n	y	60	60	y	y	54	9	IVDP L5-S1	S1	n	n	n	n	20	4	70	70	10	3	80	80	Fair
22	Chandrashekar	36	m	mason	815549	L	8 mon	n	n	n	n	n	y	70	70	n	n	54	9	IVDP L2-L3	L3	n	n	n	n	30	4	70	70	20	2	70	70	Good
23	Rama krishana	57	m	agriculture	817716	R	6 mon	y	y	y	y	y	n	70	90	n	n	42	6	IVDP L4-L5	L5	n	n	n	n	10	4	90	90	10	2	90	90	Good
24	Muninarayana	38	m	house wife	825112	R	1 yr	y	y	n	n	n	y	60	70	n	n	48	8	IVDP L3-L4	L4	n	y	n	n	20	2	70	70	0	1	70	70	Excellent
25	Manjula	32	f	house wife	828459	L	10 yr	y	y	y	n	n	y	90	60	n	n	44	7	Bulge L4-L5,L5-S1	L5	y	y	y	n	20	2	90	90	0	0	90	90	Excellent
26	Nancharappa	60	m	agriculture	832001	B/L	6 mon	n	n	n	n	y	y	70	70	y	y	52	7	IVDP L4-L5	L5	n	n	n	n	30	4	80	80	10	2	80	80	Good
27	Khaisai fathima	50	f	house wife	836714	L	1 yr	y	n	n	n	n	y	80	70	y	y	54	8	IVDP L4-L5	L5	n	y	n	n	30	4	80	80	10	2	80	80	Good
28	Gouse mohamadin s	59	m	bussiness	845771	L	3 mon	y	y	n	y	y	y	80	50	y	y	40	7	IVDP L4-L5	L5	n	n	n	n	30	3	90	60	20	4	90	70	Fair
29	Padmanabhaiah	57	m	agriculture	852079	L	3 mon	y	n	n	n	n	y	80	60	n	n	52	7	IVDP L4-L5,L5-S1	S1	n	n	n	n	20	2	80	70	10	1	80	80	Good
30	Sarasamma	35	f	house wife	852579	R	3 yrs	y	y	y	n	n	y	90	90	n	n	50	8	IVDP L3-L4	L4	n	n	n	n	20	4	90	90	10	2	90	90	Good
31	saroja	40	f	house wife	853692	L	3 yrs	n	n	n	n	n	n	80	70	n	n	44	7	IVDP L4-L5,L5-S1	S1	y	y	n	n	20	2	80	70	10	1	80	80	Good
32	Mohammed kafaar	59	m	bussiness	856261	L	6 mon	y	y	y	n	n	y	90	70	n	n	42	6	IVDP L4-L5	L5	n	n	n	n	10	2	90	90	0	0	90	90	Excellent
33	Chikka rangaiiah	49	m	agriculture	860043	L	2 yrs	y	y	n	y	n	y	90	60	n	n	48	8	IVDP L3-L4,L4-L5	L5	n	y	n	n	10	3	90	80	0	0	90	90	Excellent
34	Gayatramma	25	f	house wife	860291	L	2 yrs	y	y	y	n	n	y	80	80	y	n	40	6	NO bulge	S1	n	n	n	n	10	2	80	80	10	2	80	80	Good
35	Sudarshan	18	m	driver	866163	L	1 yr	n	n	n	n	n	y	90	50	n	n	46	8	IVDP L5-S1	S1	y	n	n	n	10	2	90	80	10	2	90	80	Good
36	Bayappa	52	m	agriculture	868217	R	2 mon	n	n	n	n	n	y	30	80	y	n	54	8	Bulge L5-S1	S1	n	y	n	y	40	6	50	80	40	7	50	80	Poor
37	Ramesh	28	m	bussiness	868633	B/L	2 yrs	y	y	y	n	n	n	70	70	n	n	52	7	IVDP L4-L5	L5	n	n	n	n	30	2	90	90	10	2	90	90	Good
38	Chalapathi	32	m	bussiness	875386	R	6 mon	n	n	n	n	n	n	60	80	y	y	48	8	IVDP L4-L5	L5	n	n	n	n	10	2	80	90	0	0	90	90	Excellent
39	Puttaswamy	53	m	agriculture	875409	L	8 mon	n	n	n	n	n	n	80	60	y	n	56	7	IVDP L3-L4,L4-L5	L4	y	n	n	n	20	3	80	70	10	1	80	80	Good
40	Noushad	28	m	carpenter	877069	L	1 yr	n	n	n	n	n	n	80	70	n	n	50	8	IVDP L4-L5,L5-S1	S1	n	y	n	n	10	4	80	80	0	0	80	80	Excellent
41	Lakshmamma	60	f	dependent	874488	L	2 yrs	y	y	y	n	n	n	80	40	n	n	48	8	Bulge L4-L5	L5	y	y	y	n	48	8	80	40	48	8	80	40	Poor
42	Shantamma	45	f	house wife	878680	L	3 mon	n	n	n	n	n	y	90	70	y	y	52	7	IVDP L4-L5,L5-S1	S1	n	n	n	n	20	3	90	70	10	1	90	90	Good
43	Ramachandrappa	45	m	agriculture	878516	B/L	5 yrs	y	y	n	n	n	y	70	70	n	n	48	6	IVDP L4-L5,L5-S1	S1	y	n	y	n	20	3	70	70	20	3	70	70	Fair
44	Lakshmidevamma	71	f	dependent	882109	R	8 mon	y	n	n	n	n	n	60	80	y	n	46	8	IVDP L5-S1	S1	n	n	n	n	26	4	70	80	10	2	80	80	Good
45	Manasa	27	f	house wife	882419	L	4 mon	y	n	n	n	n	y	90	55	n	n	38	7	Bulge L5-S1	S1	n	n	n	n	22	3	90	70	0	0	90	90	Excellent
46	Lingamma	56	f	house wife	882423	R	9 mon	y	y	y	n	y	y	60	90	y	n	42	7	IVDP L4-L5,L5-S1	L5,S1	n	n	n	n	20	4	80	90	10	1	80	90	Good
47	Revathi	28	f	student	884493	R	1 yr	y	y	y	n	n	y	50	90	n	n	40	8	Bulge L4-L5	L5	n	n	n	n	24	2	70	90	0	0	90	90	Excellent
48	Muni akkayamma	50	f	house wife	884613	R	6mon	y	n	n	n	n	y	60	80	y	n	42	9	IVDP L4-L5,L5-S1	S1	y	n	y	n	28	3	60	80	10	2	70	80	Good
49	Rajesh	32	m	bussiness	890311	B/L	6 mon	n	n	n	n	n	y	60	60	n	n	50	8	IVDP L4-L5	L5	y	y	y	n	20	4	70	70	10	1	80	80	Good
50	Basavaraj	37	m	agriculture	890584	L	3 mon	n	n	n	n	n	y	90	60	n	n	42	7	IVDP L5-S1	S1	n	n	n	n	10	2	90	90	0	0	90	90	Excellent
51	Girish	26	m	student	890590	R	6 mon	y	y	n	n	n	Y	55	90	n	n	44	8	Bulge L5-S1	S1	n	n	n	n	16	2	80	90	10	1	90	90	Good
52	Vishwanath	35																																

55	Partha saradhi	30	m	agriculture	890698	R	3 mon	y	n	n	n	n	y	70	90	n	n	34	6 Bulge L5-S1	S1	n	n	n	n	10	2	80	90	0	0	90	90	Excellent
56	Basappa	57	m	agriculture	890591	R	10 mon	y	n	n	y	n	y	60	80	y	n	40	8 IVDP L4-L5,L5-S1	L5,S1	n	n	n	n	22	4	60	80	10	2	70	80	Fair
57	Rathnamma	45	f	house wife	893547	L	4 yrs	y	y	y	y	y	y	80	60	y	n	48	6 IVDP L4-L5,L5-S1	S1	n	n	n	n	20	2	80	70	0	0	80	80	Excellent
58	Papanna	54	m	agriculture	894487	L	2 yrs	y	y	y	n	n	y	80	50	n	n	42	7 Multiple level	S1	n	n	n	n	18	3	80	70	30	5	80	60	Poor
59	Shiva bhagyam	46	m	house wife	891629	L	3 mon	n	n	n	y	y	y	80	60	n	n	50	7 IVDP L4-L5	L5	n	n	n	n	22	2	80	70	0	0	80	80	Excellent
60	Girish	38	m	bussiness	894581	R	10 mon	y	y	n	n	n	y	60	90	n	n	52	9 IVDPL5-S1	S1	n	n	n	n	24	3	70	90	10	2	90	90	Good
61	Kantharaj	39	m	agriculture	895421	L	1 yr	y	y	y	n	n	n	90	45	y	n	46	8 IVDP L5-S1	S1	n	y	n	n	28	3	90	60	10	1	90	80	Good
62	Kamala	30	f	house wife	905058	R	6 mon	y	n	n	n	n	y	60	80	y	n	50	7 IVDP L3-L4,L4-L5	L5	n	n	n	n	20	2	80	80	0	0	80	80	Excellent
63	Arjun	32	m	bussiness	905142	L	1 yr	y	n	n	n	n	y	90	60	y	y	50	8 IVDP L4-L5	L5	n	n	n	n	30	3	90	60	30	3	90	70	Poor
64	Venkateshappa	67	m	agriculture	906254	L	2 mon	n	n	n	y	y	y	90	80	n	n	50	8 IVDP L4-L5	L5	n	y	n	n	20	5	90	90	0	0	90	90	Excellent
65	Kousalya	27	f	house wife	900336	R	6 mon	y	y	n	n	n	y	60	80	n	n	38	8 Bulge L3-L4	L4	y	n	y	n	20	3	80	80	0	0	80	80	Excellent
66	Navya	22	f	student	900284	R	2 mon	y	n	n	n	n	y	50	80	n	n	40	7 Bulge L5-S1	S1	n	y	n	n	20	2	80	80	10	1	80	80	Good
67	Gowramma	45	f	house wife	908992	L	2 yrs	y	y	y	y	n	y	80	45	y	n	42	9 IVDP L5-S1	S1	n	n	n	n	30	5	80	50	30	5	80	50	Poor
68	Ramalakshmi	50	f	house wife	906554	R	8 mon	y	y	n	n	n	y	60	80	y	n	46	8 IVDP L4-L5,L5-S1	L5,S1	n	n	n	n	28	4	70	80	20	2	80	80	Fair
69	Meenakshamma	38	f	house wife	907941	R	3 mon	n	n	n	n	n	y	70	80	n	n	40	7 IVDP L3-L4,L4-L5	L4	n	n	n	n	20	3	80	80	10	1	80	80	Good
70	Hanumakka	50	f	house wife	915381	B/L	6 mon	y	y	n	y	n	y	70	70	n	n	50	8 IVDP L3-L4,L4-L5	L4	y	y	y	n	10	2	80	80	0	0	90	90	Excellent
71	Neelamma	32	f	house wife	919185	B/L	1 yr	y	y	y	n	n	y	60	60	y	n	38	7 Multiple level	L5,S1	y	n	n	n	20	3	70	70	10	1	80	80	Good
72	Venkatamma.B.M	60	f	dependent	915320	L	3 mon	n	n	n	n	n	y	80	70	n	n	52	8 IVDP L4-L5,L5-S1	S1	n	n	n	n	20	3	80	80	10	1	80	80	Good
73	Shaha taj	45	f	house wife	919193	L	2 yrs	y	y	y	n	n	Y	80	50	y	n	48	9 IVDP L5-S1	S1	y	y	y	n	40	7	80	50	40	7	80	50	Poor
74	Sumangala	42	f	house wife	913313	R	8 mon	y	y	n	n	n	y	70	90	n	n	54	8 IVDP L4-L5,L5-S1	S1	n	n	n	n	26	4	80	90	20	2	80	90	Fair
75	Mohammed iliaz	45	m	mechanic	913198	R	1 yr	n	n	n	n	n	y	70	80	n	n	48	6 IVDP L3-L4	L4	n	n	n	n	30	4	80	80	10	1	80	80	Good
76	Chinnapapammma	50	f	house wife	919123	B/L	2 yrs	y	y	n	n	n	y	70	70	y	n	56	8 IVDP L4-L5,L5-S1	S1	n	y	n	n	30	4	70	70	10	1	80	80	Good
77	Dayananda	31	m	advocate	921076	R	6 mon	n	n	n	n	n	y	70	90	n	n	46	6 IVDP L4-L5,L5-S1	S1	n	n	n	n	30	4	70	90	10	1	90	90	Good
78	Munivenkatamma	55	f	house wife	920190	R	10 yrs	y	y	n	n	n	y	70	90	n	n	52	8 IVDP L4-L5	L5	n	n	n	n	36	5	90	90	0	0	90	90	Excellent
79	Markondappa	37	m	agriculture	924296	L	6 mon	n	n	n	n	n	y	90	80	n	n	48	7 IVDP L4-L5	L5	n	n	n	n	10	2	90	90	0	0	90	90	Excellent
80	Byamma	47	f	house wife	924279	R	6 mon	n	n	n	n	n	y	70	90	n	n	50	8 IVDP L4-L5,L5-S1	S1	n	n	n	n	20	2	70	90	0	0	90	90	Excellent
81	Raniyamma	60	f	dependent	926961	B/L	5 yrs	y	y	n	n	n	Y	60	60	n	n	50	8 IVDP L4-L5	L5	n	y	n	n	30	3	70	70	20	3	70	70	Fair
82	Narayana swamy	58	m	dependent	931685	R	10 yr	y	y	n	n	n	Y	70	90	y	n	50	8 IVDP L4-L5	L5	n	y	n	n	28	4	90	90	20	2	90	90	Fair
83	Muni Venkatamma	55	f	house wife	931618	B/L	4 mon	n	n	n	n	n	y	70	70	n	n	50	7 IVDP L3-L4	L4	n	n	n	n	26	2	80	80	22	2	80	80	Fair
84	Narayanamma	52	f	house wife	932806	R	6 mon	y	n	n	n	n	y	60	80	y	n	52	8 Bulge L4-L5	L5	n	y	n	n	10	2	80	80	0	0	80	80	Excellent
85	Mamthaj.S	28	f	teacher	935850	B/L	5 yrs	y	y	n	n	n	y	70	80	n	n	46	7 IVDP L5-S1	S1	n	n	n	n	26	2	70	70	26	2	70	70	Fair
86	Ramesh.N	25	m	accountant	931953	R	3 yrs	y	y	n	n	n	y	70	90	n	n	34	6 IVDP L4-L5	L5	n	n	n	n	20	1	80	90	0	0	90	90	Excellent
87	Pillamma	48	f	house wife	933920	R	1 yr	y	y	y	n	n	y	70	90	n	n	52	8 IVDPL3-L4	L4	n	n	n	n	22	4	80	90	22	4	80	90	Fair
88	Kalpana	29	f	house wife	933958	B/L	4 mon	n	n	n	n	n	y	65	65	n	n	44	7 IVDPL3-L4,L4-L5	L5	n	n	n	n	20	2	80	80	10	1	80	80	Good
89	Mahalakshmi	45	f	bank emplo	934967	L	2 yrs	y	y	y	n	y	y	70	80	n	n	40	6 IVDP L4-L5,L5-S1	S1	n	n	n	n	10	1	80	80	0	0	80	80	Excellent
90	Nagaraj	45	m	bussiness	940475	R	4 yrs	y	y	y	n	n	y	60	80	y	n	42	8 IVDPL4-L5	L5	n	n	n	n	22	2	80	80	0	0	80	80	Excellent
91	Rathnamma	35	f	house wife	942485	R	6 mon	n	n	n	n	n	y	70	80	n	n	50	8 IVDP L4-L5	L5	n	n	y	n	40	4	80	80	40	4	80	80	Poor
92	Reddamma	46	f	house wife	948679	R	3 yrs	y	y	y	n	n	y	35	70	y	n	60	9 Bulge L4-L5	L5	n	n	n	y	54	8	40	70	54	8	40	70	Poor
93	Badhrachala	28	m	labourer	948694	R	3 mon	y	n	n	n	n	y	40	80	n	n	42	8 IVDP L4-L5	L5	n	n	n	n	10	2	80	80	10	2	80	80	Good
94	Ashwath Narayana	65	m	dependent	949753	R	5 yrs	y	y	y	y	n	y	40	80	y	y	38	7 IVDP L5-S1	S1	n	n	n	n	20	3	60	80	10	1	70	80	Good
95	Bhagyamma	50	f	house wife	942591	B/L	2 mon	n	y	y	n	n	y	70	70	n	n	38	7 IVDP L4-L5,L5-S1	S1	n	n	n	n	10	2	80	80	0	0	80	80	Excellent
96	Manjamma	30	f	house wife	944731	R	2 mon	n	n	n	n	n	y	50	80	y	y	58	9 IVDP L4-L5	L5	y	y	n	y	50	9	60	80	50	9	60	80	Poor
97	Srinivasa reddy	30	m	labourer	950943	L	6 mon	y	y	y	n	n	y	90	70	n	n	34	6 IVDP L4-L5	L5	n	n	n	n	10	2	90	80	10	1	90	80	Excellent
98	Balappa	39	m	labourer	952108	R	2 mon	n	n	n	n	n	y	60	80	y	n	44	9 IVDP L4-L5,L5-S1	S1	n	n	n	n	10	1	80	80	10	1	80	80	Good
99	Rama	48	m	mechanic	956398	R	6 mon	y	y	y	n	n	y	70	90	n	n	40	7 Multiple level	S1	n	n	n	n	0	0	90	90	0	0	90	90	Excellent
100	Srinivasan	55	m	agriculture	957051	L	3 yrs	n	y	n	n	n	y	90	60	y	n	40	9 IVDP L3-L4	L4	n	y	n	n	10	1	90	90	10	1	90	90	Good