"MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"

By

Dr. SUJATA

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 MEDICINE
IN
RADIODIAGNOSIS

Under the Guidance of

Dr. ANIL KUMAR SAKALECHA, MD,
PROFESSOR OF RADIODIAGNOSIS

DEPARTMENT OF RADIODIAGNOSIS,
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

MAY 2017

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

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. ANIL KUMAR SAKALECHA**, Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M.D. DEGREE IN RADIODIAGNOSIS**", the examination to be held in May 2017 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

Dr. SUJATA
Postgraduate in Radiodiagnosis
Sri Devaraj Urs Medical College
Tamaka, Kolar

Date:
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 **"MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"** is a bonafide research work done by **Dr. SUJATA,** under my direct guidance and supervision at Sri Devaraj Urs Medical College, Kolar**,** in partial fulfilment of the requirement for the degree of "**M.D. IN RADIODIAGNOSIS**".

Dr. ANIL KUMAR SAKALECHA, MD
Professor
Department Of Radiodiagnosis
Sri Devaraj Urs Medical College
Tamaka, Kolar

Date:

Place: Kolar

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

CERTIFICATE BY THE HEAD OF DEPARTMENT

This is to certify that the dissertation entitled "**MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"** is a bonafide research work done by **Dr. SUJATA,** under direct guidance and supervision of **Dr. ANIL KUMAR SAKALECHA**, Professor, Department of Radiodiagnosis at Sri Devaraj Urs Medical College, Kolar**,** in partial fulfilment of the requirement for the degree of "**M.D. IN RADIODIAGNOSIS"**.

Dr. PURNIMA HEGDE, MD
Professor & HOD
Department of Radiodiagnosis
Sri Devaraj Urs Medical College
Tamaka, Kolar

Date:
Place: Kolar

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

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT AND PRINCIPAL

This is to certify that the dissertation entitled **"MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"** is a bonafide research work done by **Dr. SUJATA** under the direct guidance and supervision of **Dr. ANIL KUMAR SAKALECHA,** Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M.D. DEGREE IN RADIODIAGNOSIS**"**.**

Dr. PURNIMA HEGDE,
Professor & HOD
Department Of Radiodiagnosis,
Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:
Place: Kolar

Dr. M. L. HARENDRA KUMAR
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, and Kolar has unanimously approved

Dr. SUJATA

Post-Graduate student in the subject of

RADIODIAGNOSIS at Sri Devaraj Urs Medical College, Kolar

to take up the Dissertation work entitled

"MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON-TRAUMATIC ETIOLOGY"

to be submitted to the

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

Member Secretary

Sri Devaraj Urs Medical College,

Kolar–563 101.

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

COPY RIGHT

I hereby declare that 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. SUJATA

Date:

Place: Kolar

ACKNOWLEDGEMENT

I owe debt and gratitude to my parents ***Shri GURUNATH KOLLUR and Smt. LAXMI KOLLUR****, along with my brother* ***SACHIN*** *and my husband* ***Dr. KAILASH MIRCHE*** *for their moral support and constant encouragement during the study.*

With humble gratitude and great respect, I would like to thank my teacher, mentor and guide, ***Dr. ANIL KUMAR SAKALECHA,*** *Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, for his able guidance, constant encouragement, immense help and valuable advices which went a long way in moulding and enabling me to complete this work successfully.*

I have great pleasure in expressing my deep sense of gratitude to ***Dr. PURNIMA HEGDE,*** *Professor and Head, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar. Without her initiative and constant encouragement this study would not have been possible. Her vast experience, knowledge, able supervision and valuable advices have served as a constant source of inspiration during the entire course of my study.*

I would like to express my sincere thanks to ***Dr. PATTABHIRAMAN V.,*** *Professor, Department of Radiodiagnosis, Sri Devaraj Urs Medical College for his valuable support, guidance and encouragement throughout the study.*

I would like to thank ***Dr. RACHEGOWDA N., Dr. A. NABAKUMAR SINGH, Dr. ASHWATHNARAYANA, Dr. NAVEEN G NAIK, Dr. JAGADISH, Dr. VINAY, Dr. KUKU MARIAM SURESH*** *and* ***Dr. ANIL KUMAR T. R.*** *and all my teachers of Department of Radio diagnosis, Sri Devaraj Urs Medical College, Kolar, for their constant guidance and encouragement during the study period.*

I am extremely grateful to the patients who volunteered to this study, without them this study would just be a dream.

I am thankful to my fellow ***postgraduates, especially Dr. Shivaprasad Gangadhar Savagave and Dr. Varun S.*** *for having rendered all their co-operation and help to me during my study.*

My sincere thanks to ***Mrs. Veena*** *along with rest of the computer operators. I am also thankful to* ***Mr. Ravi, Mr. Chandrasekhar, Mr. Aleem, and Mr. Mateen*** *with other* ***technicians*** *of Department of Radiodiagnosis, R.L Jalappa Hospital & Research Centre, Tamaka, Kolar for their help.*

Dr. SUJATA

LIST OF ABBREVIATIONS

ABCs	*Aneurysmal bone cysts*
AF	*Annulus fibrosus*
ALL	*Anterior longitudinal ligament*
AP	*Anteroposterior*
AV	*Arteriovenous malformation*
CNS	*Central nervous system*
CSF	*Cerebrospinal fluid*
CT	*Computerized tomography*
DALYs	*Disability adjusted life years*
EACs	*Extradural arachnoid cysts*
ESR	*Erythrocyte sedimentation rate*
FA	*Facetal arthropathy*
FS	*Fat saturation*
FDA	*Food and drug administration*
GCT	*Giant cell tumour*
ISL	*Interspinous ligament*
I.V	*Intravenous*

LIST OF ABBREVIATIONS

IVD	*Intervertebral discs*
IVF	*Intervertebral foramen*
LBP	*Low back pain*
LF	*Ligamentum flavum*
LFH	*Ligamentum flavum hypertrophy*
LSTV	*Lumbosacral transitional vertebrae*
MRI	*Magnetic resonance imaging*
NMR	*Nuclear magnetic resonance*
NP	*Nucleus pulposus*
OA	*Osteoarthritis*
PLC	*Posterior ligamentous complex*
PLL	*Posterior longitudinal ligament.*
SC	*Spinal cord*
SEM	*Spinal cord epidural metastasis.*
SI	*Signal intensity*
SPECT	*Single-photon emission computed tomography*
SSL	*Supraspinous ligament*
STIR	*Short tau inversion recovery*

LIST OF ABBREVIATIONS

T1 W	*T1 Weighted*
T2 W	*T2 Weighted*
TR	*Repetition time*
TE	*Echo time*
TB	*Tuberculosis*
YLD	*Years lived with disability*

ABSTRACT

Background: Low back pain (LBP) is most commonly encountered complaint in clinical practice and is a common cause for hospital visits and a majority of patients (about 60 to 80%) experience back pain at some point of time or other. MRI helps in complete evaluation of low back pain.

Aims and objectives: The study was conducted to evaluate the changes seen on MRI in patients with LBP due to various non-traumatic causes, to distinguish various causes of low back pain with level of spinal involvement and to evaluate the concordance between clinical diagnosis MR imaging.

Materials and methods: This descriptive observational study was carried out over a period of 18 months from January 2015 to June 2016 in 106 patients with low back pain who underwent MRI of the lower spine. Patients who met the inclusion/exclusion criteria were included in the study.

Results: More than 40% of patients (n = 43; 40.57%) were in the age group of 41 to 60 years with a slight male preponderance in the study (58%). Degenerative changes were the commonest findings in more than 50% of patients (n = 87; 82.08%) followed by infective (n = 20; 18.87%) and neoplastic (n = 11; 10.38 %) etiologies. Four patients each had inflammatory and congenital etiologies (3.77%). Arachnoid cyst was seen in two patients (1.89%). Degenerative changes were considered as cause for low back pain in 65 patients. Among degenerative changes, disc changes were the commonest pathology seen in > 70% of patients (n = 76; 71.7%) followed by endplate changes (n = 63; 59.4%), vertebral changes (n = 58; 54.7%) and joint and ligament

changes (n = 44; 41.5%). Among degenerative disc changes disc bulges were most common (n = 132 discs; 43.7%), followed by disc protrusion (68 discs; 22.52%), annular fissure/tears (40 discs; 13.25%), disc extrusion (37 discs; 12.25%) and disc sequestration (25 discs; 8.28%). Spondylolisthesis was seen in 23 patients and were graded from grades I to grade IV of which Grade I spondylolisthesis was most common (n = 14, 60.9%). Type II Modic end plate changes were commonest (n = 37; 72%). Infective causes of low back pain were tubercular spondylitis and pyogenic spondylitis. Tubercular spondylitis was commonest infective condition seen in 17 patients (85%) followed by pyogenic spondylitis (n = 3; 15%). Pott's spine was seen mostly in patients aged 40 years or older (11 of 16; 68.75%) and T12-L1 was the most commonly involved spinal level (n = 7, 43.75%). There were 10 malignant and one benign tumour. Most of malignant conditions were metastasis seen in eight patients. Among congenital conditions there were two cases of myelomeningocele, one case each of distometamyelia and arteriovenous malformation (AVM) of spinal cord.

Conclusion: Degenerative changes were the commonest cause for low back pain followed by infective and neoplastic etiologies. MRI provides most precise visualization of all spinal elements and paraspinal soft tissues. Additionally, the ability of MRI to detect disc and vertebral signal changes has made it an investigation of choice for evaluation of low back pain.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Low back pain (LBP) is a commonly encountered complaint in clinical practice with a significant economic burden to the society. Globally, LBP has been shown to cause more years lived with disability (YLD) that any other condition and is considered as one of the leading causes of disability adjusted life years (DALYs) among the general population[1].

LBP is a common cause for hospital visits and a majority of patients (about 60 to 80%) experience back pain at some point of time or other[2,3]. In fact, nearly three-fourth of patients older than 30 years, experience back pain and of these about two-third of patients experience LBP[4]. In India, a high incidence of LBP has been found in individuals who are involved in jobs that require handling heavy loads, constant sitting/standing position or working at improper body position and prolonged working hours. In fact, the causative factors for LBP are very wide and ranges from body habitus, habits, work atmosphere, age and gender[5]. However, the commonest cause for LBP is attributed to mechanical disorders, which contribute about 9 out of 10 cases[4].

Currently MRI provides most precise visualisation of all spinal elements and paraspinal soft tissues[4]. The ability of MRI to detect disc and subchondral bone marrow signal changes makes it an investigation of choice for evaluation of LBP.

MRI is the modality of choice for evaluation of spinal cord tumours as it can provide diagnosis or differential diagnoses in majority of cases[6,7].

On magnetic resonance imaging (MRI) studies degenerative changes with intervertebral disc space pathologies are a common cause for low back pain[8]. Other causes include involvement of spinal elements and paraspinal structures, degenerative changes in facet joints, joint effusion, synovitis, sacroiliitis, spondylolysis, degenerative and inflammatory processes of the spinal ligaments and pathologies of paraspinal muscles[9].

As LBP can result from various etiological factors, appropriate diagnosis helps in optimizing management options and ultimately improve patient outcomes, this study has been planned to evaluate the various causes of LBP which will aid in appropriate management.

AIMS AND OBJECTIVES

The aims and objectives of this study were:

- To evaluate the changes seen on MRI in patients with low back pain due to various non−traumatic causes.
- To distinguish various causes of low back pain with level of spinal involvement.
- To evaluate the concordance between clinical diagnosis and MR imaging.

REVIEW OF LITERATURE

ANATOMY OF SPINE AND SPINAL CORD

The spinal cord represents a caudal extension of the medulla oblongata; it terminates in the conus medullaris, typically located at T12 or L1 level in adults. The cord is slightly flattened along its anterior and posterior surfaces and is enlarged in two regions. The cord widens first for the brachial plexus from C3 to T2, then for the lumbosacral plexus from T9 to T12. The filum terminale is a slender fibrous strand extending from conus to the coccyx, and the cauda equina is the spinal nerve roots extending caudal from the conus within the lumbar subarachnoid space[10].

The three-layered meningeal covering of the central nervous system (CNS) is contiguous with the spinal cord, and all lie within the bony spinal canal. The innermost layer, the piamater, is adherent to the surface of the cord. The middle arachnoid membrane remains closely adherent to the outer layer, the duramater. The space between the arachnoid and piamater is called the subarachnoid space, which contains cerebrospinal fluid (CSF). This space is contiguous with the intracranial subarachnoid space. A potential space exists between the duramater and arachnoid membrane, called the subdural space. The dentate ligaments are formed by the extension of piamater laterally from the surface of the cord to attach to the duramater, in a saw-tooth fashion, between the exiting nerve roots. The dura extends from the base of the skull to the S2 level, forming the dural (thecal) sac, which is surrounded externally by epidural fat and loose connective tissue to fill the remainder of the volume of the bony spinal canal. The anterior and posterior venous plexuses are also located in the epidural fat[10].

The gray matter of the spinal cord is located internally, in contradistinction to the gray matter of the brain, and is surrounded by white matter tracts; the proportion of gray matter increases in the cervical and lumbar regions. Both dorsal (sensory) and ventral (motor) roots arise along the entire length of the cord and unite to form a total of 31 paired spinal nerves. There are eight cervical, 12 thoracic, five lumbar, and five sacral nerve roots as well as one coccygeal nerve root[10].

Arterial supply to the cord originates from arterial vessels that enter the spinal canal through the neural foramina at all levels of the spinal cord, which are termed radicular branches. Radicular branches arise from the vertebral arteries, the costocervical trunk, intercostal and lumbar arteries. The distal branches of the radicular arteries that continue on to supply the cord are termed radiculomedullary arteries, which in the adult are variable in number and position. The radiculomedullary arteries terminate in branches that run cephalocaudal along the surface of the cord, anastomosing with those above and below to form the anterior as well as the paired posterior spinal arteries on the cord surface. The anterior spinal artery provides the major blood supply to the spinal cord, supplying the anterior 70% to 80%, while the posterior spinal arteries supply the posterior 20% to 30%. There are a large number of radiculomedullary arteries supplying the posterior spinal arterial system. Radiculomedullary supply to the anterior spinal artery is more variable and its branches. The artery of Adamkiewicz, which arises from a low thoracic or upper lumbar artery, supplies the thoracolumbar region. This important artery usually arises from a left intercostal branch in the region of T9 to T12[10].

Anatomy of Spine

The vertebral column forms the central axis of the skeleton and consists of 33 vertebrae. There are seven cervical, twelve thoracic and five lumbar vertebrae (the true, "moveable" vertebrae), and caudally there are five sacral and four coccygeal segments, all of which are fused as the sacrum and coccyx, respectively (Figure 1)[11].

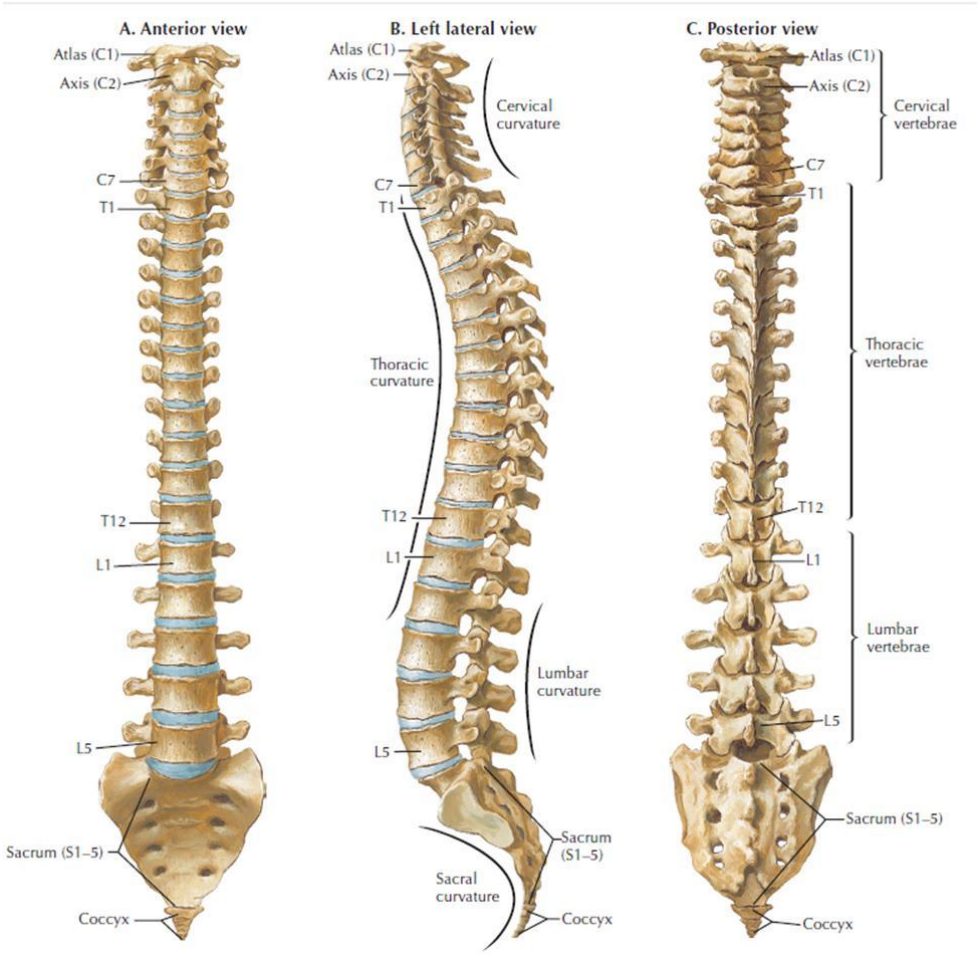


Figure 1. Illustration showing vertebral column with seven cervical vertebrae, 12 thoracic vertebrae and five lumbar vertebrae and 5 sacral vertebrae. Note the cervical, thoracic, lumbar and sacral curvature.

The Vertebral Canal

The vertebral canal transmits the spinal cord and in the lumbar region, the cauda equina. It is formed by the posterior margins of the vertebral bodies and discs anteriorly, and the pedicles and laminae (the neural arch) posteriorly[11].

The Intervertebral Canal (Neural Foramen)

The spinal nerves arise from the spinal cord and leave the spinal canal through the intervertebral canals, each of which is situated between adjacent pedicles (Figure 2). The nerves are accompanied by blood vessels and are supported by extradural fat within each canal[11].

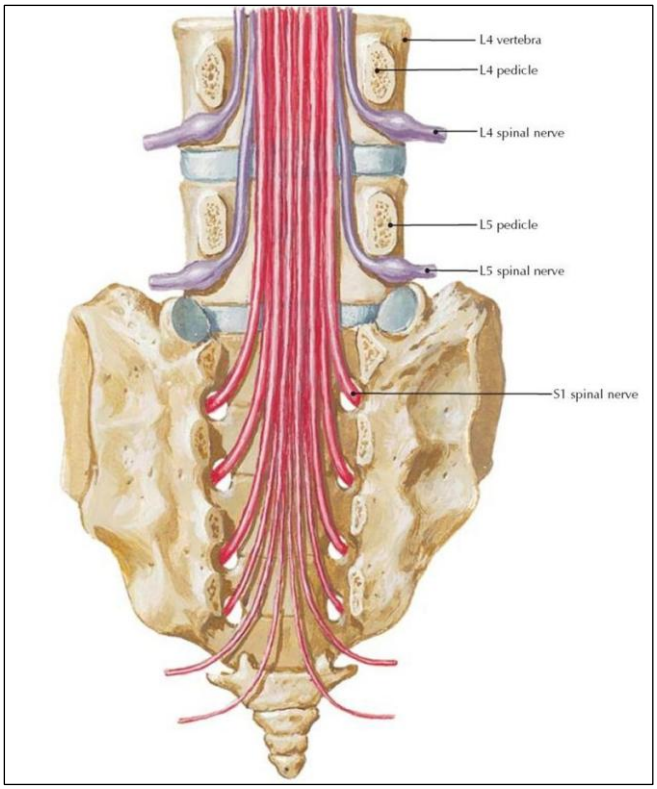


Figure 2. Relationship between the lower spinal nerves and their respective neuroforamina (intervertebral foramina) in lumbosacral spine.

The Ligaments of Vertebral Column

A number of ligaments strengthen the vertebral column. The anterior longitudinal ligament runs superoinferiorly between the anterior surfaces of the vertebral bodies from the occiput to the sacrum. The posterior longitudinal ligament is applied to the posterior surfaces and narrows as it passes downward. The ligamentum flavum joins adjacent laminae and the interspinous ligaments run between the spinous processes (Figure 3)[11].

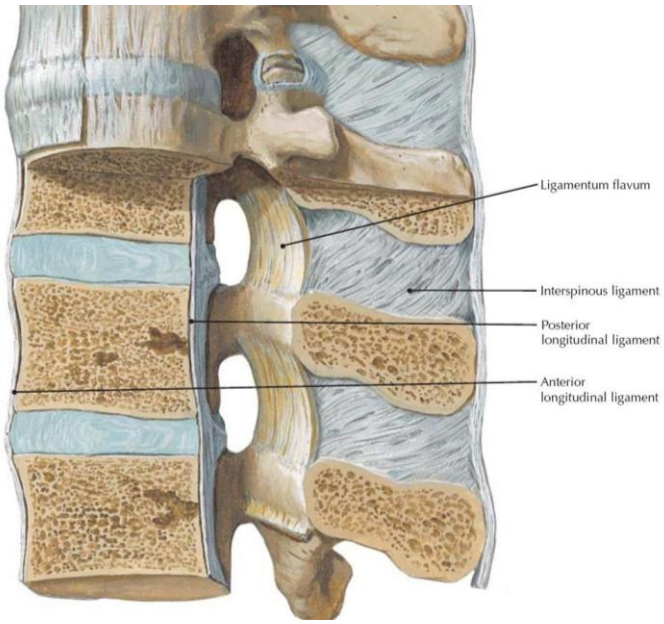


Figure 3. Longitudinal section showing ligaments of spine (lumbar region)

In the axial plane the ligamentum flavum appears V shaped and is thickest in the lumbar region. The vertebral column can be considered as a three-column structure. The anterior column is formed by the anterior longitudinal ligament, the anterior annulus fibrosus, and the anterior part of the vertebral body. The middle column comprises the posterior longitudinal ligament, the posterior annulus fibrosus, and the posterior part of the vertebral body. The posterior column consists of the neural arch and posterior ligamentous complex[11].

Intervertebral Discs

The intervertebral discs (IVD) lie between the superior and inferior cartilaginous endplates of the adjacent vertebrae, being formed of an outer annulus fibrosus (AF) and an inner nucleus pulposus (NP) (Figure 4). The IVD functions to provide motion between individual vertebrae and also allows the effective transfer of load[11].

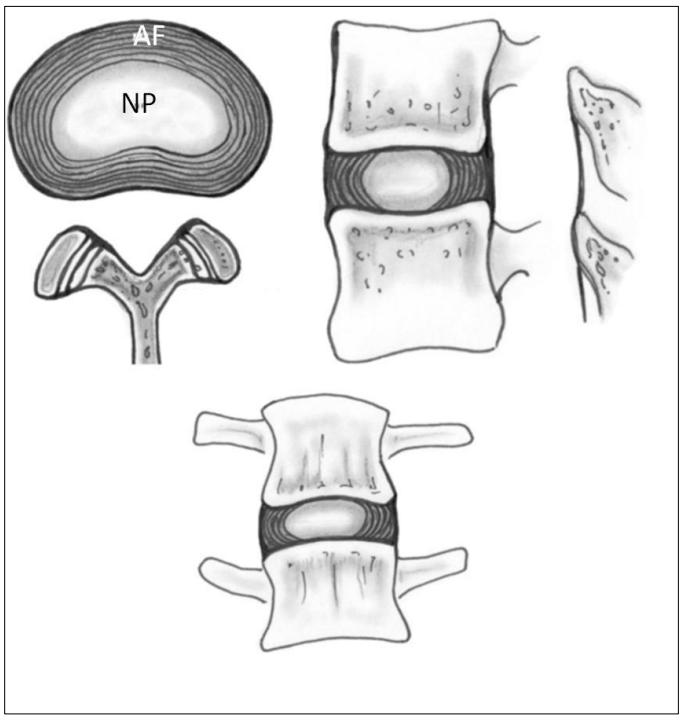


Figure 4. Normal lumbar disc. (Top Left) Axial, (Top Right) sagittal, and (Bottom) coronal images demonstrate that the normal disc, composed of central NP and peripheral AF, is wholly within the boundaries of the disc space, as defined, craniad and caudad by the vertebral body end plates and peripherally by the planes of the outer edges of the vertebral apophyses, exclusive of osteophytes (NP, nucleus pulposus; AF, annulus fibrosus).

The Lumbar Vertebral Column

There are five lumbar vertebrae, the third (L3) being the largest. Lumbar vertebrae have square-shaped anterior vertebral bodies covered by fenestrated cartilage attached to the adjacent disks. Projecting posteriorly are bilateral pedicles composed of thick cortical bone connecting to lamina forming the spinal canal. The articular facets face each other in the sagittal plane, and the transverse distance between the pedicles increases (the interpedicular distance) from L1 to L5. L5 is somewhat atypical with a wedge-shaped body, articulating inferiorly with the sacrum (Figure 5). Not infrequently, it may be fused, wholly or partly, with the body of the sacrum ("sacralization of L5"). Extending from the pedicles is a bony plate called the pars articularis from which extend the superior and inferior articular facets. The posterior superior articular facet of an inferiorly located vertebra connects to the posterior inferior facet of the superior vertebra above creating a diarthrodial synovial lined joint, surrounded by a fibrous capsule posterolaterally with absence of the joint capsule anteriorly, where the ligamentum flavum and synovial membrane are present[11].

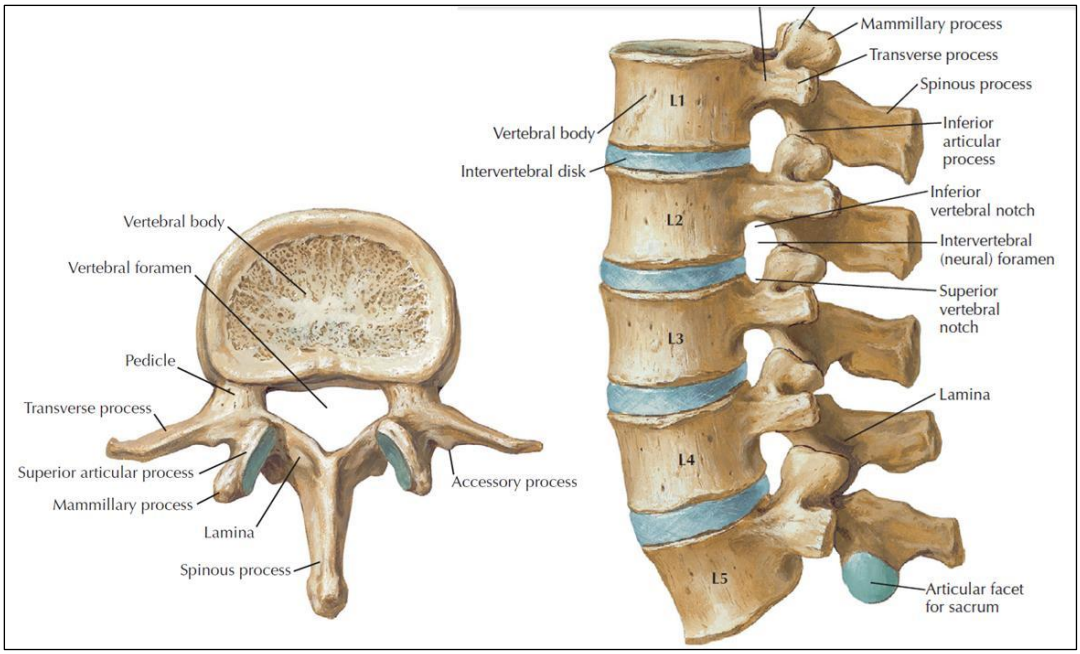


Figure 5. Axial and longitudinal view of lumbar vertebrae showing normal lumbar curvature and vertebral bodies.

MRI ANATOMY OF SPINE

MRI is the primary imaging method for the vertebral column. MRI is ideally suited to the demonstration of soft tissue anatomy of the spinal cord, including the vertebral medullary cavity, the intervertebral discs, the spinal ligaments and the paravertebral musculature[12].

A typical MRI series will consist of T1W and T2W sagittal and axial images. Further coronal images and intravenous gadolinium contrast administration may be undertaken depending on the clinical picture. The tissue discrimination of MRI is superior to CT. MRI is the only method to show an intrinsic abnormality of the spinal cord substance. On T1W images the CSF is dark and, in general, this sequence shows the anatomy. On T2W images the CSF appears white and thus there is a myelographic effect. T2W sequences, in general, demonstrate pathology[11].

The Vertebral Body

The vertebral body contains marrow, MRI signal intensity (SI) of which is dependent upon the proportion of red (haemo-poietic) and yellow (fatty) marrow, this varying with the age. In adults, the high proportion of yellow marrow results in the marrow appearing hyperintense to the IVD, particularly in the lumbar region. Islands of red marrow or marrow fibrosis appear as areas of reduced T1W and T2W SI. Focal areas of fatty marrow may be seen, particularly around the basivertebral veins. In the elderly, the marrow SI may be very heterogeneous[12].

Intervertebral Discs

The annulus fibrosus (AF) is formed by 15-25 laminae of fibrous connective tissue, which due to its fibrous nature appears hypointense on all MR pulse sequences, particularly T2W images. The AF is attached circumferentially to the periphery of the vertebral body via Sharpey's fibres. The anterior annulus is thicker than the posterior annulus, resulting in the NP lying relatively posteriorly within the IVD. The NP consists of a gel-like substance with approximately 90% of its content being water, rendering it hyperintense on T2W images and of intermediate SI on T1W images. The intranuclear cleft appears as a horizontal band of low SI on sagittal T2W images, which is a normal finding after the age of 30 years[12].

The Spinal Canal

The spinal canal is divided into the central canal, the lateral recess, and the intervertebral foramen (IVF). The boundaries of the central canal are vertebral body, IVD and PLL anteriorly; posterior epidural fat pad, LF and base of the spinous process posteriorly. Central canal contains the thecal (dural) sac and CSF, the intradural nerve roots and, in the cervical and thoracic region, the spinal cord[12].

The boundaries of the lateral recess are vertebral body and IVD anteriorly; thecal sac medially; pedicle, facet joint and IVF laterally; and LF and lamina posteriorly. The lateral recess contains epidural fat, the traversing nerve root, and epidural vessels. The boundaries of the IVF are vertebral body and IVD anteriorly;

LF, pars interarticularis and facet joint posteriorly; pedicles of the adjacent vertebrae superiorly and inferiorly. IVF contains epidural fat, the exiting nerve root, radicular epidural vessels and sinuvertebral nerves[12].

The Paraspinal Musculature

The thoracolumbar muscles are divided into anterior and posterior:

- Anteriorly: Psoas major and occasionally psoas minor
- Posteriorly: Multifidus, erector spinae, quadratus lumborum and intertransversarius[12].

Cross-sectional MR Anatomy of Spinal Cord

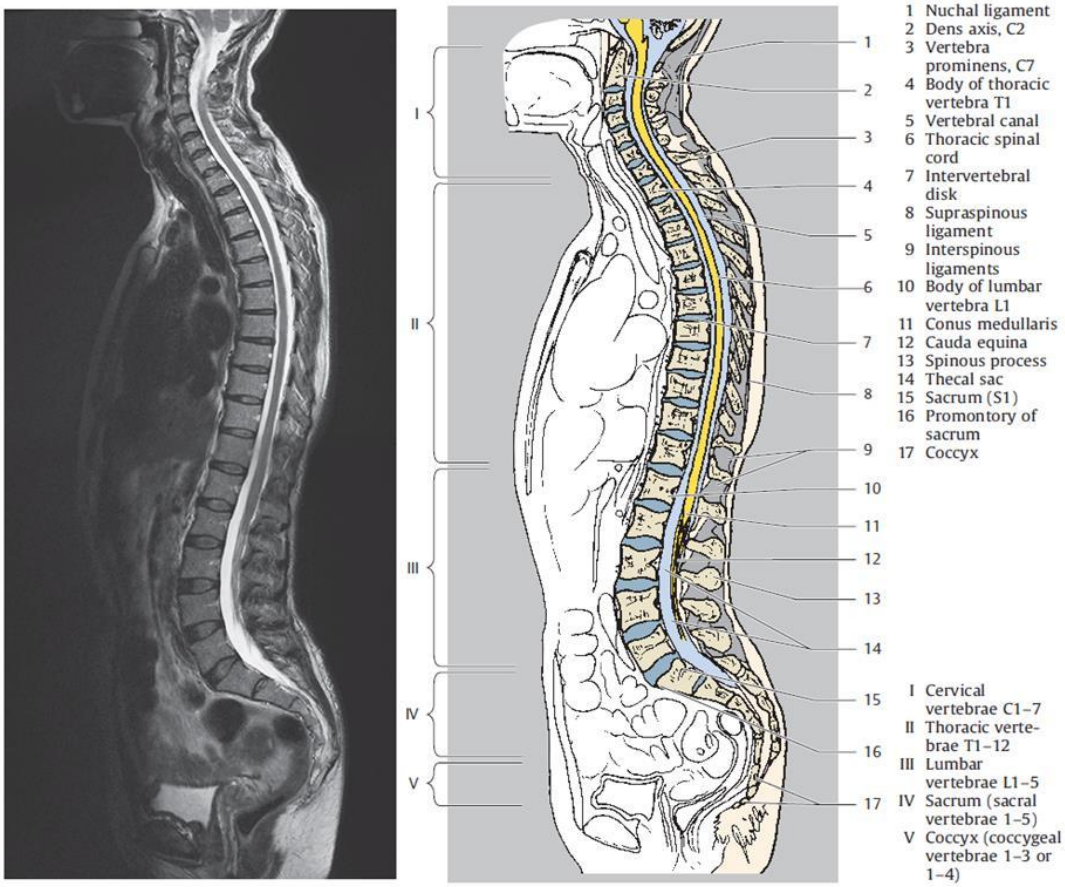
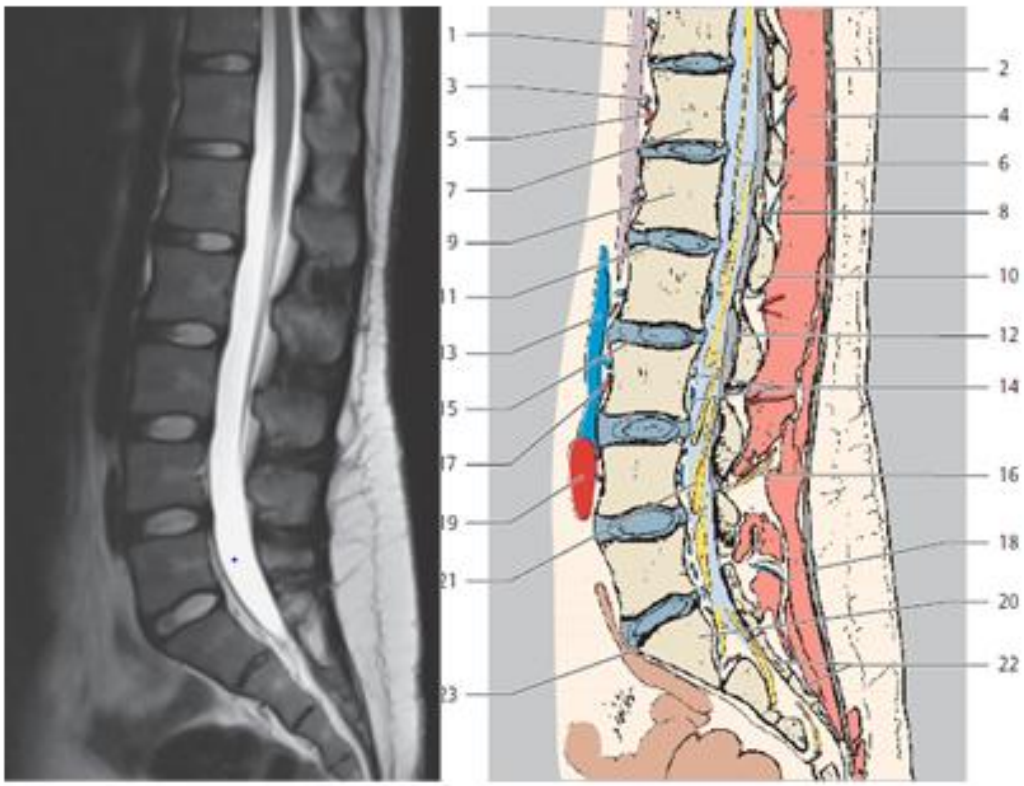
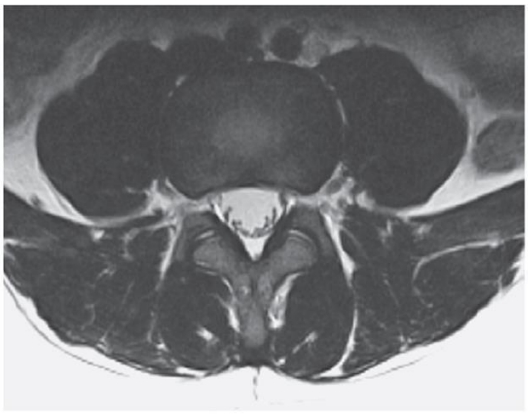


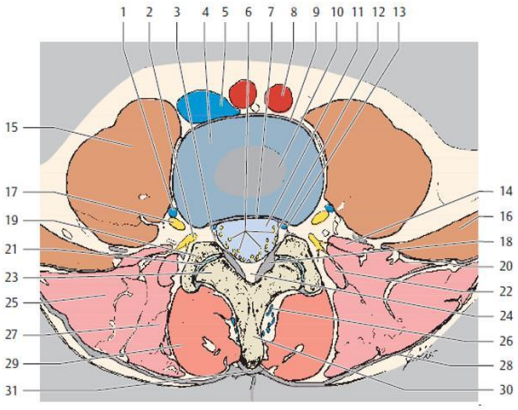
Figure 6. Sagittal T2 WI MRI showing whole spine.[13]



1 Spinal cord
2 Conus medullaris
3 Abdominal aorta
4 Ligamentum flavum
5 Lumbar vertebral body L1
6 Spinous process L1
7 Intervertebral disk L1/L2 (nucleus pulposus)
8 Interspinous ligament
9 Anterior longitudinal ligament
10 Supraspinous ligament
11 Intervertebral disk L2/L3 (annulus fibrosus)
12 Cauda equina
13 Basivertebral vein
14 Epidural fatty tissue
15 Left common iliac vein
16 Posterior longitudinal ligament
17 Sacral canal
18 Thecal sac (lumbar cistern)
19 Promontory of sacrum
20 Dura mater
21 Sacrum (S1)
22 Median sacral crest

Figure 7. T2 WI sagittal image through lumbosacral spine[13].





1 Costal process
2 Nerve filaments
3 Lumbar artery
4 Spinal ganglion in lateral recess L4
5 Anterior internal vertebral venous plexus
6 Inferior vena cava (confluence)
7 Nutrient foramen
8 Lumbar vertebral body L4
9 Anterior longitudinal ligament
10 Left common iliac artery
11 Basivertebral vein
12 Ascending lumbar vein
13 Posterior longitudinal ligament
14 Thecal sac (lumbar cistern)
15 Interarticular portion L4
16 Spinal ganglion L3
17 Psoas major muscle
18 Spinal dura mater
19 Zygapophyseal joint
20 Quadratus lumborum muscle
21 Superior articular process
22 Thoracolumbar fascia (anterior layer)
23 Inferior articular process
24 Ligamentum flavum
25 Posterior vertebral arch (lamina)
26 Epidural fatty tissue (retrospinal/dorsal fatty triangle)
27 Posterior external vertebral venous plexus
28 Erector spinae muscle (lateral tract: iliocostalis lumborum muscle)
29 Erector spinae muscle (medial tract: multifidus muscle)
30 Erector spinae muscle (lateral tract: longissimus muscle)
31 Interspinous ligament
32 Thoracolumbar fascia (posterior layer)
33 Supraspinous ligament

Figure 8. T2 WI axial image through L3-4 disc level

HISTORICAL BACKGROUND

Role of X-rays in Evaluation of Low Back Pain

Plain radiographs were the first imaging modality to be used for evaluation of low back pain. Radiographs have the advantage of being fast, relatively inexpensive, easy availability, reliability, and portability. Acute radiographs are indicated for evaluation of trauma or evaluation of tumour or infection. However, there are two major challenges with radiographs – difficulty in interpretation and very high rate of false-positive findings. A structured approach is needed for evaluation of radiographs so as prevent missing findings. Traditionally, anteroposterior (AP) and lateral views of lumbar spine are taken to evaluate spine for any structural abnormality, tumours or infections (Figure 9).[14].

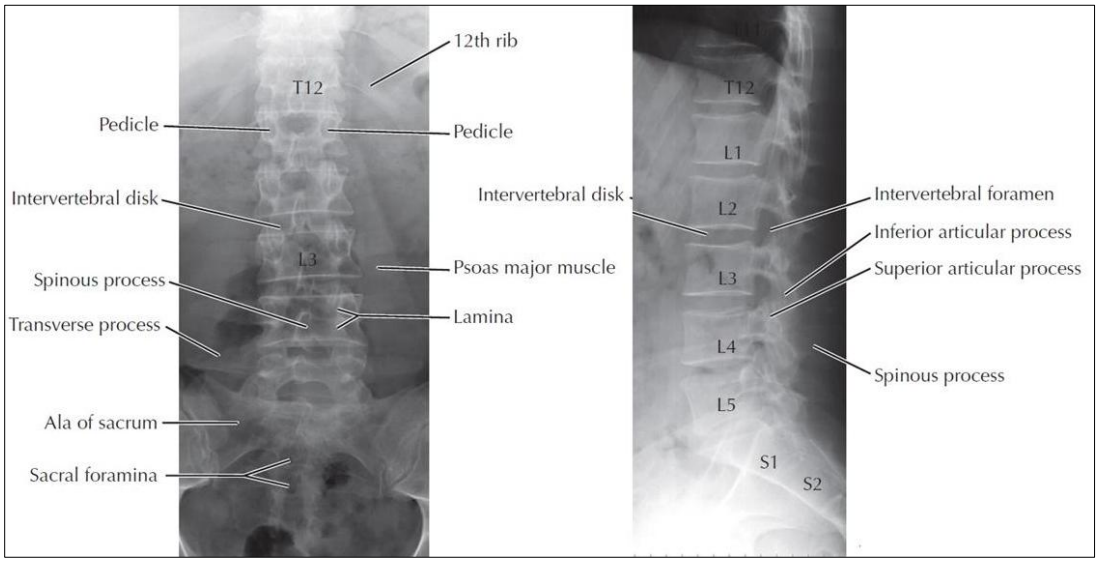


Figure 9. Radiograph of lumbar spine: AP (left) and lateral (right) views showing normal vertebral bodies, lumbar lordosis, pedicle, intervertebral foramina and pedicles

Although degenerative changes may commonly be evident on plain radiographs; one should be careful in making a diagnosis of degenerative disease of spine as they are known to be equally common in asymptomatic individuals and symptomatic persons. Degenerative changes on radiographs are most common in patients > 40 years and are seen in > 70 percent of patients older than 70 years. Intervertebral narrowing and irregular ossification of the vertebral end plates is seen with increasing incidence with advancing age. To summarize, although radiographs have limited role in evaluation of low back pain, they often help to determine degenerative disorders[14].

Role of Myelography in Evaluation of Low Back Pain

Myelography is an invasive procedure in which water-soluble iodine-based contrast agents (iohexol and iopamidol) are introduced into subarachnoid space and is used to evaluate spinal cord and intrathecal nerve roots. Following contrast injection, three views viz. AP, lateral, and oblique views are obtained (Figure 10). Myelography can also be performed for evaluation of disc herniation above or below a segment that may be ambiguous or distorted on MRI secondary to metal placement. It is also indicated in individuals who cannot undergo MRI (such as claustrophobia or pacemaker, or in MRI is otherwise contraindicated)[14].

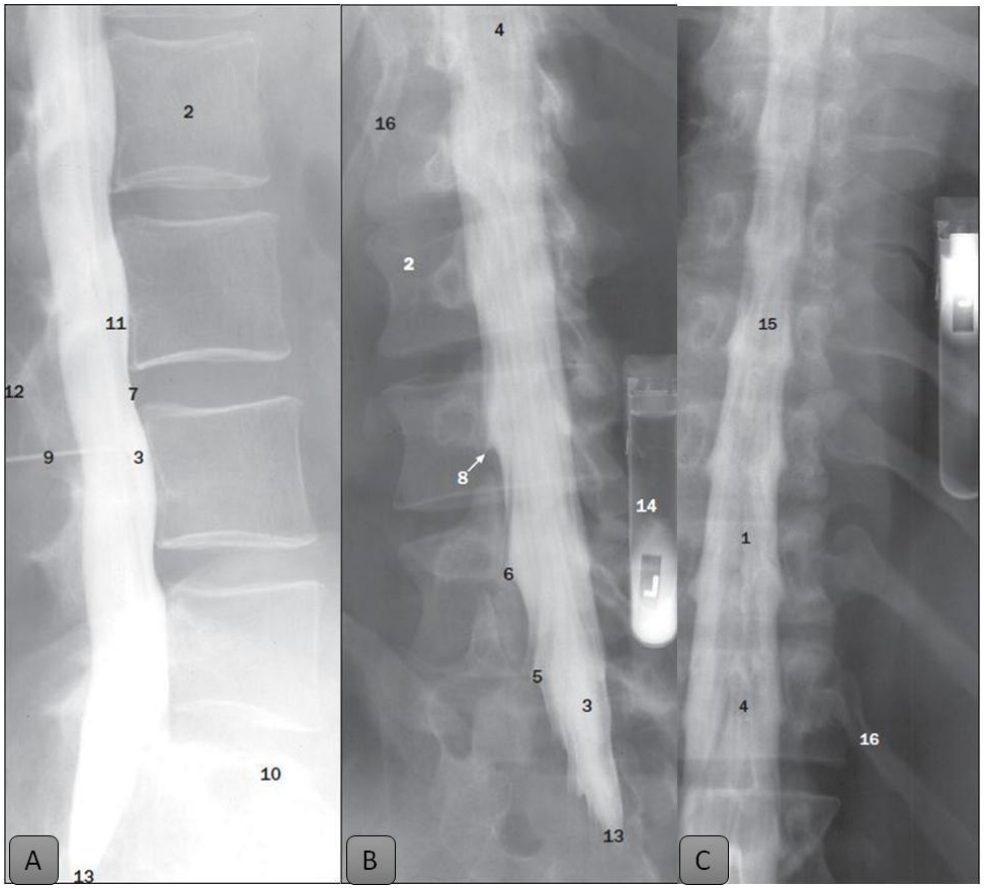


Figure 10. Lumbar myelography (A) lateral, (B) oblique and (C) AP views. Normal study. 1) Anterior median fissure 2) Body of second lumbar vertebra 3) Contrast medium in subarachnoid space 4) Conus medullaris 5) Fifth lumbar spinal nerve 6) Fourth lumbar spinal nerve 7) Intervertebral disc indentations in anterior thecal margin 8) Lateral extension of subarachnoid space around spinal nerve roots 9) Lumbar puncture needle in space between third and fourth lumbar vertebrae 10) Sacral promontory 11) Spinal nerves within subarachnoid space (cauda equina) 12) Spinous process of third lumbar vertebra 13) Terminal theca at first/second sacral vertebra 14) Test tube containing contrast medium to indicate tilt of patient 15) Thoracic cord 16) Twelfth rib.

Additionally, CT scan performed immediately following myelography increased the diagnostic quality and reliability of CT study as there is better visualization of osteophytes, disc herniations, and spinal cord contour (Figure 11)[14].

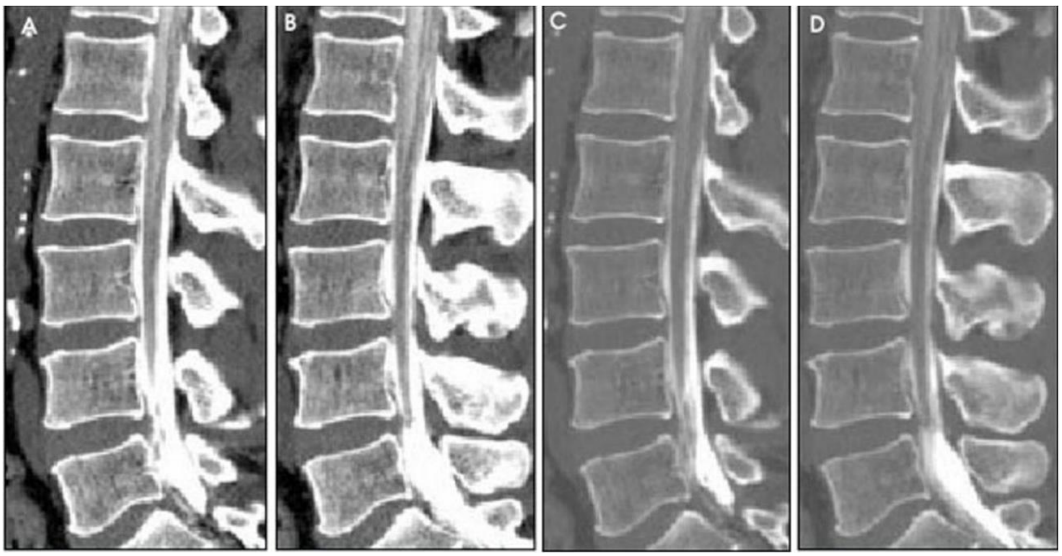


Figure 11. Lumbar CT myelogram (16 x 0.625 mm) performed using a 16-slice MDCT machine (A through D). MDCT = multidetector computed tomography.

An important limitation of myelography is its inability to visualize nerve root entrapment lateral to the termination of the nerve root sheath. Therefore, myelography may not be able to detect far lateral disc herniations, which have a reported incidence of about 1 to 12 percent of all lumbar disc herniations and a predilection for L3-L4 and L4-L5 levels. Possible adverse events with myelography include dural tear, resulting in headaches, nausea, vomiting, pain or tightness in the back or neck, dizziness, diplopia, photophobia, tinnitus, or blurred vision. It is believed that dural tear can lead of loss of cerebrospinal fluid volume, thereby reducing brain's supporting cushion with increasing tension in the brain's anchoring structures[14].

Recent advances in CT and MRI in the form of CT and MR myelography have overcome the limitations of conventional myeolgraphy (invasive procedure and lack of diagnostic accuracy) (Figure 12)[14]

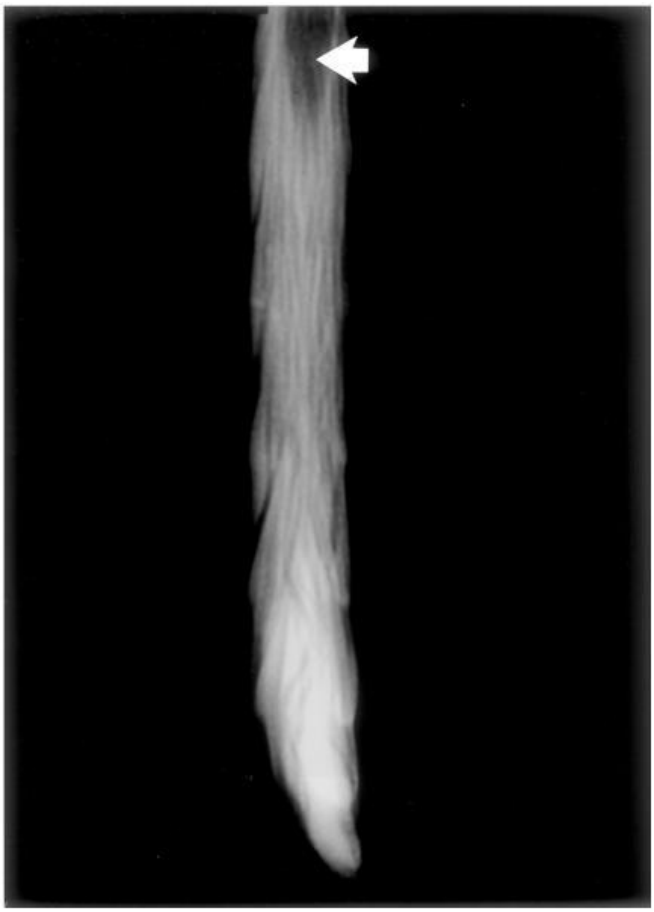


Figure 12. Normal MR myelogram oblique, 45^0 right anterior oblique (RAO) projection in a 24-year-old-male with low back pain. T2 weighted half- Fourier single shot fast spin echo (HASTE) type sequence. The conus medularis (arrow) and nerve roots are clearly identified.

Role of Computed Tomography in Evaluation of Low Back Pain

CT is an excellent tool to assess osseous structures of the spine and their relationship to the neural canal in an axial plane. Therefore, CT scan is now considered as a complementary investigation in addition to radiography,

myelography, and MRI. CT scan is helpful in diagnosing tumours, vertebral fractures, partial or complete dislocations and spondylolisthesis. The data is obtained in the axial images can be reformatted for viewing in any desirable plane (Figure 13). However, CT is not helpful in soft tissue assessment as compared to MRI. Other limitations with CT include possibility of obscuring nondisplaced fractures or simulating false ones. In addition, CT is associated with radiation exposure and results are adversely affected by patient motion. Introduction of spiral CT has addressed few of the limitations by reducing scan time and radiation exposure[14].

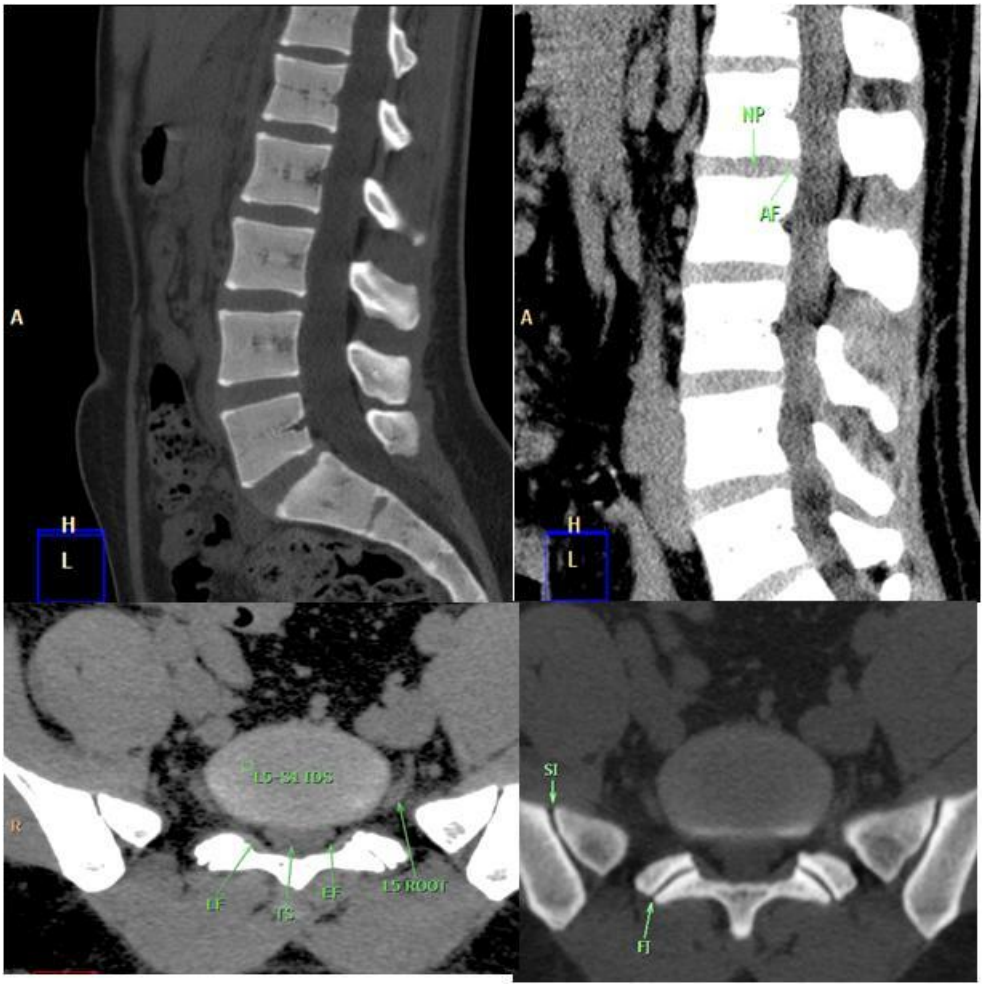


Figure 13. CT lumbar spine sagittal (top row) and axial (bottom row) images showing lumbar spine.

Nuclear Medicine in Evaluation of Low Back Pain

Plain radiographs, CT scans and MRIs have limited role in evaluating biochemical changes in bone and can only reveal morphologic changes in bone. Bone scintigraphy, aids in detection of biochemical changes by scanning and mapping the presence of radioactive compounds (such as technetium (Tc) 99m phosphate or gallium 67 citrate) in vertebrae. These radioactive compounds get incorporated into hydroxyapatite crystals deposited in an osteoid matrix during new bone formation and the radiation is received and mapped onto image. The image produced indicates bone turnover, which is high especially in bone metastases, primary spine tumours, fracture, infarction, infection, and other metabolic bone diseases[14].

Bone metastases appear as multiple foci of increased tracer uptake in the affected regions (Figure 14). In rare instances, where the bone metastases is diffuse, diffusely increased uptake of tracer results in every bone can be reported as a negative study. Aggressive tumours such as myeloma can also yield a negative examination as do not involve an osteoblastic response[14].

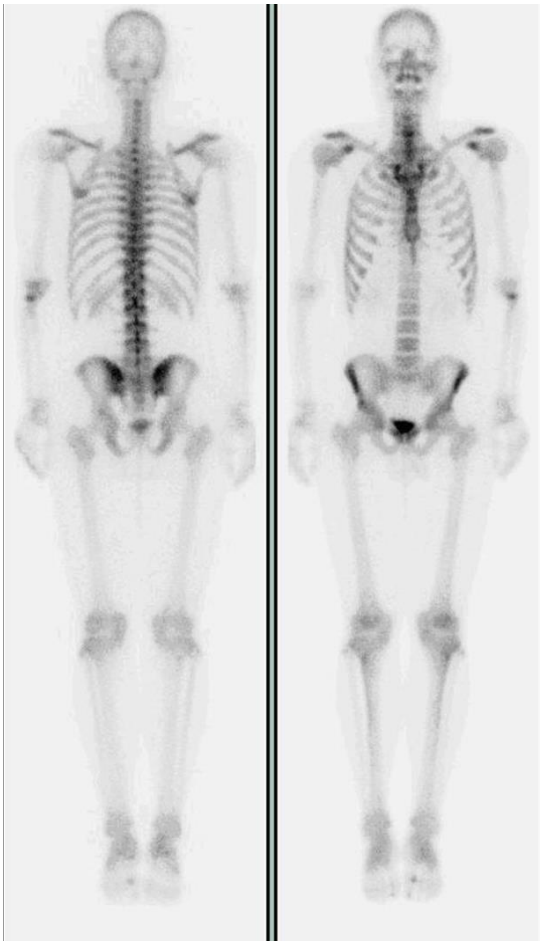


Figure 14. Bone scan in a patient with prostate cancer showing vertebral metastases.

Primary spine tumours are commonly benign. Benign conditions such as osteoid osteoma, osteoblastoma, aneurysmal bone cyst, and osteochondroma can produce an active bone scan. These tumours have predilection to posterior elements of the spine. In these conditions, CT study is recommended to differentiate them and isolate their anatomic position[14].

Recently, introduction of single-photon emission computed tomography (SPECT) helps distinguish benign lesions from malignant lesions. SPECT is superior compared to bone scan due to its ability to provide three-dimensional image, which

allow for more precise determination of lesion location. Malignant lesions have predilection towards pedicles whereas facetal lesions tend to be benign. Vertebral body or spinous process lesions can be either benign or malignant. Gallium 67 is considered as the most effective radioactive tracer for assessing infectious spondylitis. MRI is considered as the next best investigation for assessment of infectious spondylitis with a sensitivity of 96 percent, a specificity of 93 percent, and an accuracy of 94 percent[14].

HISTORY OF MRI

The clinical field of magnetic resonance imaging (MRI) is comparatively new, yet its history spans more than a century and is renowned for several Nobel Prizes and key innovations in science and technology. The study of MRI launched in 1882 with a major breakthrough in Physics: namely, the discovery of the Rotating Magnetic Field by Nikola Tesla. In his honor, the "Tesla" became the international unit of magnetic flux density, which calibrates the strength of the magnetic field used in all MRI systems. The research and development of nuclear magnetic resonance spectroscopy by Felix Bloch of Stanford University and Edward Purcell of Harvard laid the foundation for nuclear magnetic resonance (NMR). Raymond Damadian of the State University of New York discovered a difference in relaxation times between normal and abnormal tissue (e.g., cancer). Paul Lauterbur utilized magnetic field gradients to produce the first nuclear magnetic resonance images. Meanwhile Peter Mansfield was working on improving the calculation used to process images in order to improve

quality. Mansfield was successful and in 1978 presented the first cross-section images of both a finger and the abdomen[15].

Richard Ernst discovered a new reconstruction method for imaging. Ernst found that by altering the magnetic field, one could produce phase and frequency encoding, which continues to be the image reconstruction standard used today. In 2003 the Nobel Prize in Medicine or Physiology was shared by Lauterbur and Mansfield for their work. The final step towards advancing the clinical use of MRI was to build a magnet scanner, which was accomplished in 1977 and approved for clinical use by the FDA (Food and Drug Administration) in 1984. In addition, gadolinium, an MRI contrast agent, was patented and approved by the FDA four years later. Clinical MRI is a rather young field that has yielded extraordinary achievements, most of which occurred in the United States[15].

The advent of CT and, more importantly, MR imaging revolutionized spine imaging. The spinal cord and its surrounding structures could now be noninvasively visualized in great detail. In situations in which myelography is still necessary, advances in contrast agents have made the procedure less painful with fewer side effects. This has resulted in more rapid, more specific diagnosis, therapy, and improved outcomes[16]. MRI also provides excellent images of the thecal sac with soft-tissue contrast[17].

LOW BACK PAIN

Low back pain (LBP) can be defined as activity-limiting ± pain referred into one or both lower limbs. Lower back can be defined as the area on the posterior aspect of the body from the lower margin of the twelfth ribs to the lower gluteal folds[1].

Low backache can be acute or chronic. Acute low back pain can be defined as lumbosacral pain lasting not more than six weeks with or without progressive or disabling symptoms. If symptoms persist for more than six weeks, then it can be referred to as complicated acute LBP. Chronic LBP is defined as low back pain that persists for more than three months[18].

Causes of LBP

Common causes of low back pain are enumerated in Table 1. Lumbar disc diseases are an important cause of low back ache with L4-L5 as the commonest site for degenerative disc disease[19]. According to a meta-analysis by Endean et al, intervertebral disc abnormalities (excluding trauma) is one of the commonest causes of low back ache[20].

Table 1. Common Causes of Low Back Pain[20,21,22,23,24]

Degenerative disease	Degenerative disc diseases	Disc bulge
		Disc protrusion
		Disc extrusion
		Disc sequestration ± migration
	Vertebral body changes	Spondylolysis
		Spondylolisthesis
		Hemangioma
		Schmorl's nodes
		Osteophytes
		End plate changes
	Ligamentum flavum changes	
	Posterior element changes	Facetal arthrosis
Trauma		
Infective/Inflammatory	Tuberculosis spine	
	Pyogenic	
	Echinococcosis	
	Ankylosing spondylitis (including sacroiliitis)	
Neoplasms	Extradural	Lymphoma
		Myeloma
		Metastases
		Hemangioma
		Vertebral body tumours
		Chordoma
		Sarcoma
	Intradural extramedullary	Schwannoma
		Neurofibroma
		Meningioma
		Ganglioneuroma
		Paraganglioma
		Dermoid / Epidermoid
		Arachnoid cyst
		Metastases
		Ependymoma
	Intramedullary	Ependymoma
		Astrocytoma
		Hemangioblastoma
		Glioblastoma
		Metastases
		Hydrosyringomyelia
Congenital	Sacralization/lumbarization	
	Scoliosis/kyphosis	
	Perineural cyst	
Soft tissue related disorders		
Lumbar canal stenosis and associated nerve compression		
Osteoporosis/osteomalacia/osteopenia (work/stress induced)		

Degenerative disc changes

The terms disk degeneration, degenerative disk disease, and spondylosis describe anatomical changes to the vertebral bodies and intervertebral disc spaces in symptomatic individuals. Spinal osteoarthritis (OA) can be defined radiologically by joint space narrowing, osteophytosis, subchondral sclerosis, and cyst formation. Osteophytes can be classified into two primary clinical categories[25].

The first category, *spondylosis deformans* is defined as bony outgrowths arising primarily along the anterior and lateral perimeters of the vertebral end-plate apophyses. It is believed that these changes are more prone to develop at sites of stress to the annular ligament are most common in thoracolumbar level (T9–10 and L3 level). Osteophytes generally have minimal effect on intervertebral disk height can be asymptomatic in most of the individuals[25].

The second category, *intervertebral osteochondrosis* describes formation of pathological end-plate osteophytes, which are associated with disk space narrowing, vacuum phenomenon, and vertebral body reactive changes. If these changes protrude within the spinal canal or intervertebral foramina, they may cause nerve compression with resultant radiculopathy or spinal stenosis. Additionally, these bony changes may limit joint mobility and involve other organs or tissues. The term ‘‘osteoarthritis’’ suggests pathology limited to bone. Nevertheless, in the context of vertebrae, osteoarthritis can affect disks and nerve roots and therefore is not only limited to bone[25].

Disk degeneration involves structural disruption and cell-mediated changes in the intervertebral disc. Traditionally, disc degeneration has been associated to mechanical loading. Age-related changes in vertebral bone and cartilaginous endplate can adversely interfere with normal disc nutrition and result in reduced intradiscal pressure with subsequent bulging and loss of disc height resulting in increased stress to annulus. Alterations in age-related blood supply to the disc have been shown to result in increased stress on annulus and facet joints. Additionally, biochemical factors have been shown to adversely affect discal metabolism and increase the susceptibility of disc for mechanical disruption. Regardless of the initiating mechanism, all these mechanisms are considered interactive and additive, finally resulting in altered functional ability of the disc to resist applied forces[26].

Mechanical, traumatic, nutritional, and genetic factors all play a role in the cascade of disc degeneration. With degeneration and aging, type II collagen increases in the annulus along with greater disc desiccation from the nucleus pulposus. This results in a loss of the hydrostatic properties of the disc, with an overall reduction of hydration in both areas to about 70%. Additionally, proteoglycans form an important biochemical constituent of discs. With degeneration, the relative composition of proteoglycans is altered resulting in reduced tensile strength of the disc and also believed to be responsible for disc desiccation. The disc becomes progressively more fibrous and disorganized, finally resulting in amorphous fibrocartilage with indistinct nucleus and annulus[26].

It has been proposed that annular disruption is the critical factor in degeneration and, when a radial tear develops in the annulus, there is shrinkage with disorganization of the fibrous cartilage of the nucleus pulposus and replacement of the disc by dense fibrous tissue with cystic spaces. Currently most authors believe that in a degenerative disc, annular tears occur secondary to repetitive stress leading to disc herniation. Annular tears initially appear in the outer layers of the annulus pulposus and progress to involve whole annular width and subsequently result in disc herniation. Herniation of nucleus pulposus may involve and compress nerve root resulting in radicular pain[26]. Three types of fissures are generally described, radial, transverse and concentric (Figure 15). It has been shown that all the three types of tears are present in almost all degene`rated discs. Disc desiccation changes on MRI are suggestive of at least one or more small annular fissures. The term annular fissure is preferred over the term annular tear, as tear would imply a traumatic etiology, and therefore is confusing[27].

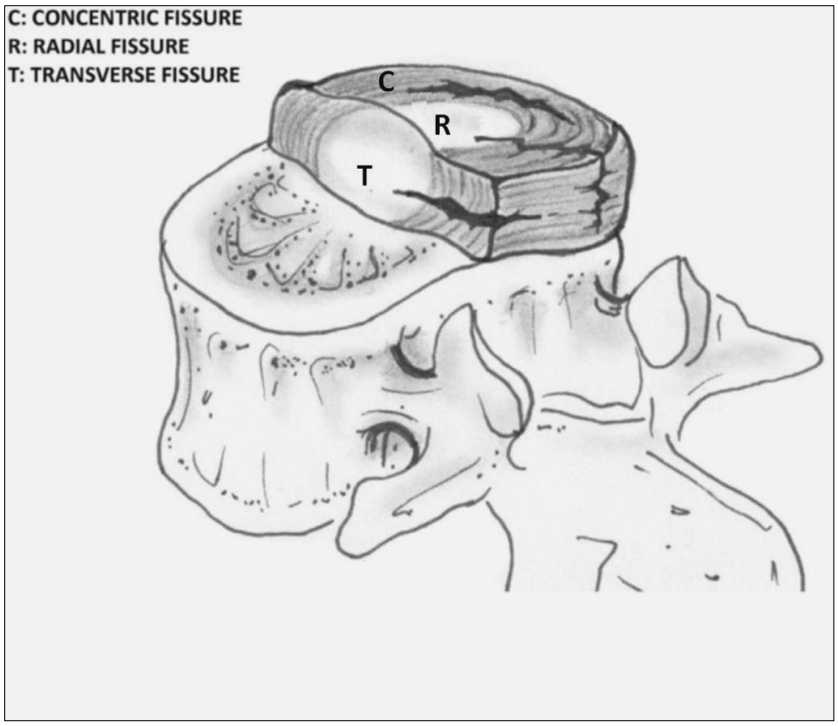


Figure 15. Fissures of the annulus fibrosus. Fissures of the annulus fibrosus occur as radial (R), transverse (T), and/or concentric (C) separations of fibers of the annulus. The transverse fissure depicted is a fully developed, horizontally oriented radial fissure; the term ‘‘transverse fissure’’ is often applied to a less extensive separation limited to the peripheral annulus and its bony attachments

Degenerative changes in the disc can be classified into annular fissure, degeneration and herniation. Degeneration may include desiccation, fibrosis, narrowing of disc space, diffuse bulging of annulus beyond disc space, osteophytes of vertebral apophyses, defects, and sclerosis of end plates. Disc tissue extending beyond the edges of the ring apophyses, throughout the circumference of the disc, is referred to as ‘‘bulging’’. The terms ‘‘bulge’’ or ‘‘bulging’’ refer to a generalized extension of disc tissue beyond the edges of the apophyses. Such bulging involves greater than 25% of the circumference of the disc and typically extends a relatively short distance,

usually less than 3 mm, beyond the edges of the apophyses. Disc bulge can be symmetric, when there is symmetric extension of annular tissue extends beyond vertebral apophysis or asymmetric if the annular tissue extends beyond the vertebral apophysis asymmetrically by > 25% of circumference of disc (Figure 16)[27].

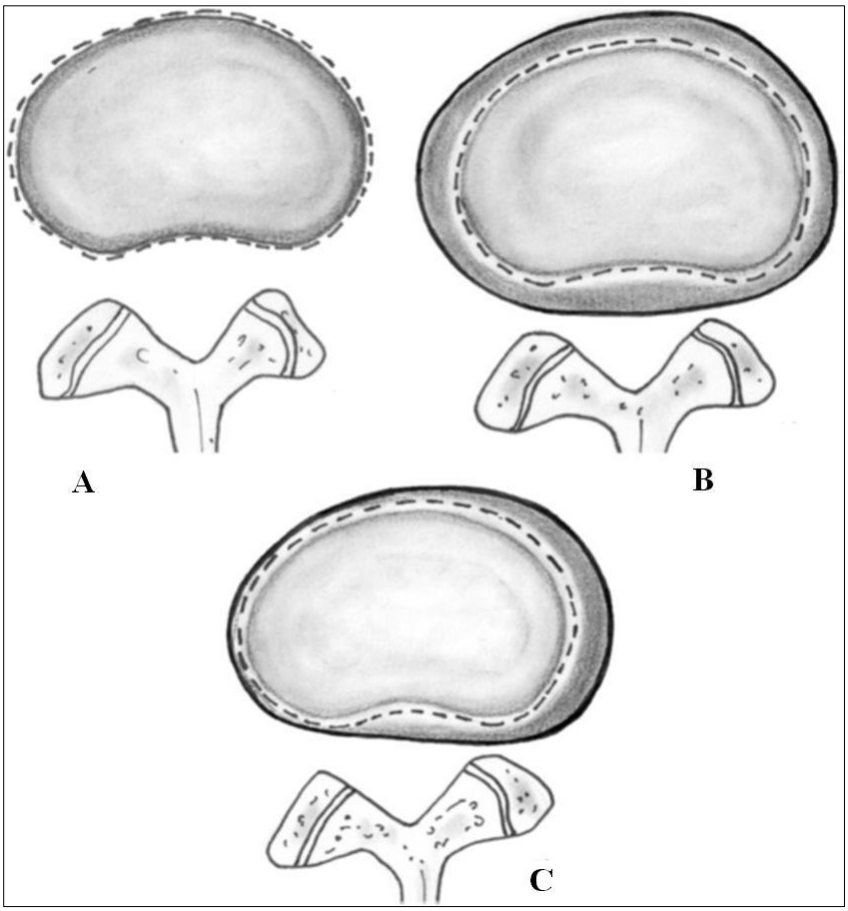


Figure 16. Bulging disc. (A) Normal disc (for comparison); no disc material extends beyond the periphery of the disc space, depicted here by the broken line. (B) Symmetric bulging disc; annular tissue extends, usually by less than 3 mm, beyond the edges of the vertebral apophyses symmetrically throughout the circumference of the disc. (C) Asymmetric bulging disc; annular tissue extends beyond the edges of the vertebral apophysis, asymmetrically greater than 25% of the circumference of the disc.

Herniation is broadly defined as localized or focal displacement of disc material beyond the limits of intervertebral disc space (>25% of peripheral zone on axial plane). Herniated discs may be classified as protrusion or extrusion, based on the shape of the displaced material. Protrusion is present if the greatest distance between the edges of the disc material presenting outside the disc space is less than the distance between the edges of the base of that disc material extending outside the disc space (Figure 17). The base is defined as the width of disc material at the outer margin of the disc space of origin, where disc material displaced beyond the disc space is continuous with the disc material within the disc space[27].

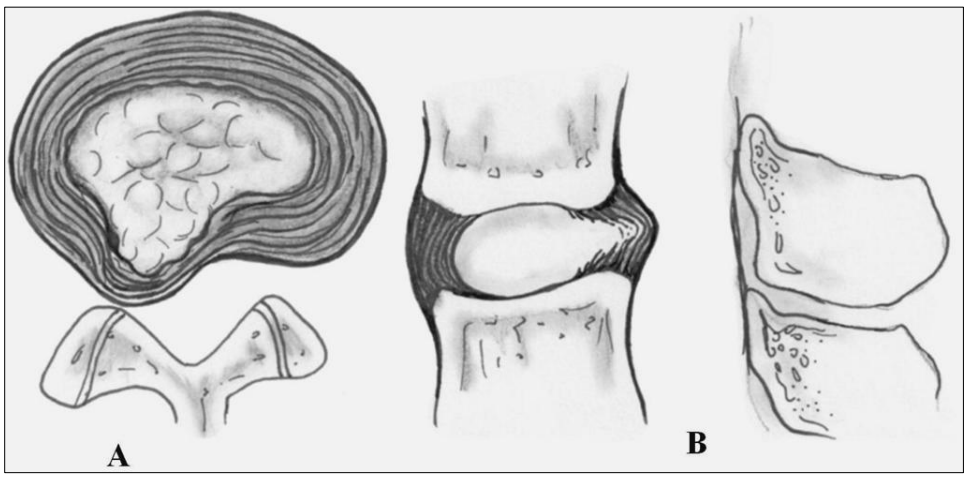


Figure 17. Herniated disc: protrusion. (A) Axial and (B) sagittal images demonstrate displaced disc material extending beyond less than 25% of the disc space, with the greatest measure, in any plane, of the displaced disc material being less than the measure of the base of displaced disc material at the disc space of origin, measured in the same plane.

Extrusion is present when, in at least one plane, any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base of the disc material beyond the disc space or when no continuity exists between the disc material beyond the disc space and that within the disc space (Figure 18). The latter form of extrusion is best further specified or subclassified as sequestration if the displaced disc material has lost continuity completely with the parent disc (Figure 19). The term migration may be used to signify displacement of disc material away from the site of extrusion. Disc herniations in the craniocaudad (vertical) direction through a gap in the vertebral body end plate are referred to as intravertebral herniations (Schmorl nodes) (Figure 20) [27].

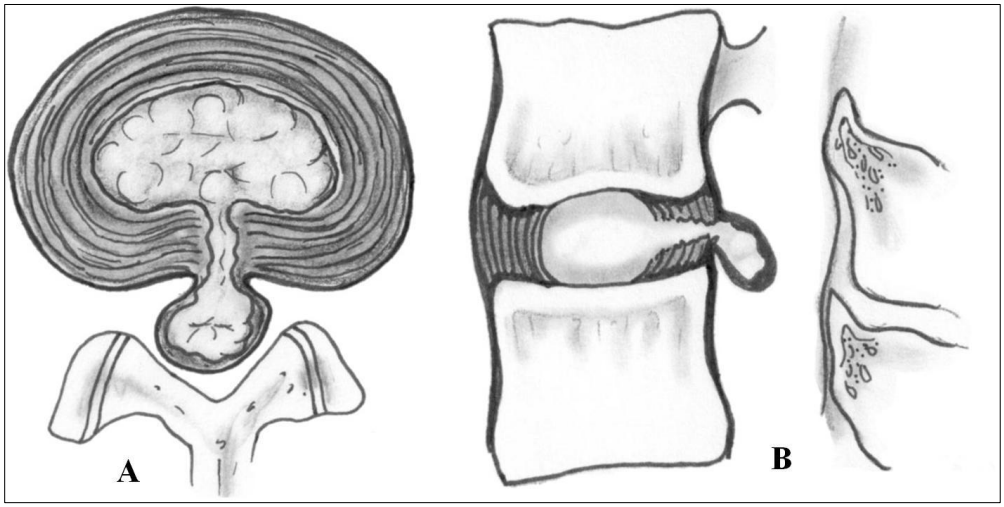


Figure 18. Herniated disc: extrusion. (A) Axial and (B) sagittal images demonstrate that the greatest measure of the displaced disc material is greater than the base of the displaced disc material at the disc space of origin, when measured in the same plane.

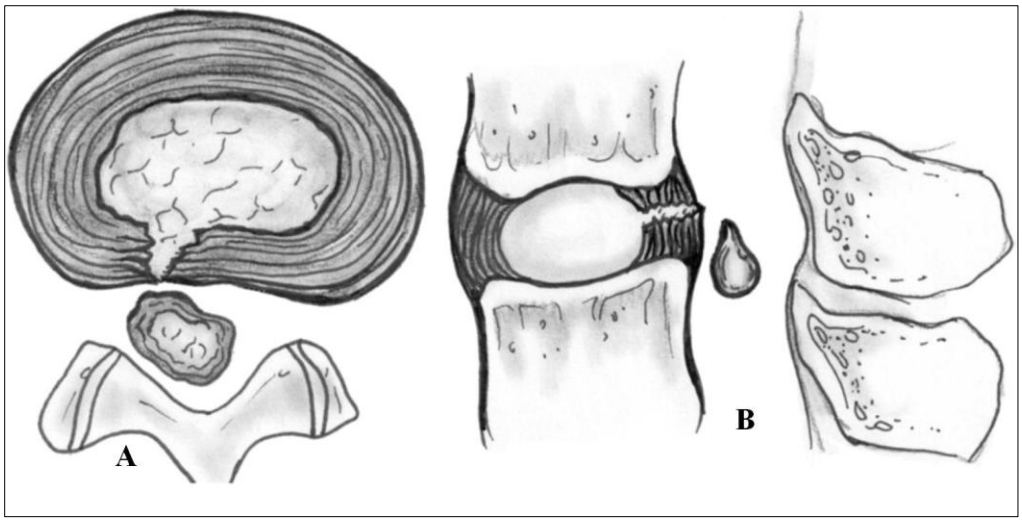


Figure 19. Herniated disc: sequestration. (A) Axial and (B) sagittal images show that a sequestrated disc is an extruded disc in which the displaced disc material has lost all connection with the disc of origin.

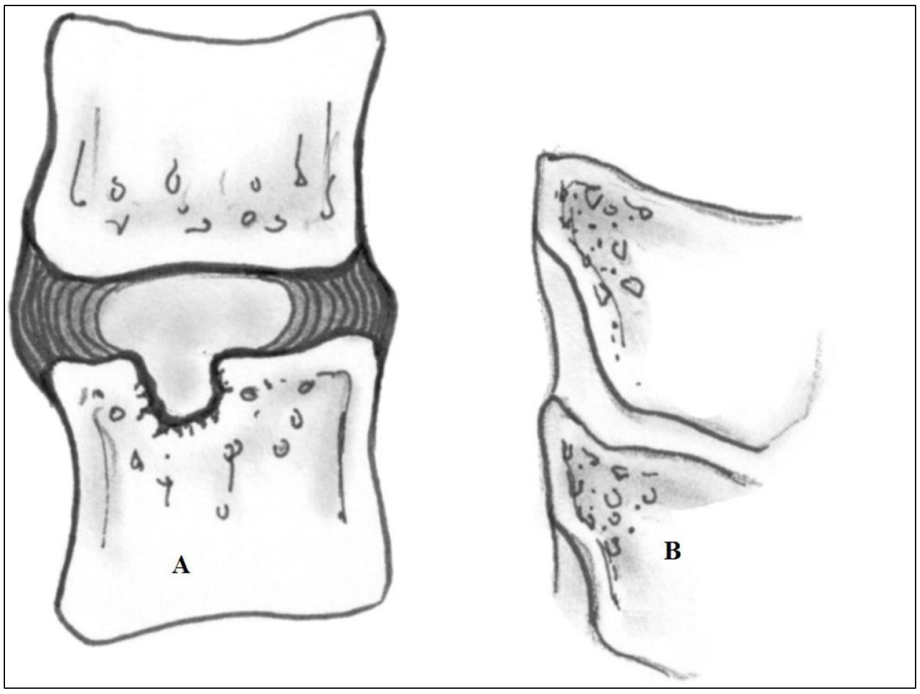


Figure 20. Intravertebral herniation (Schmorl node). Disc material is displaced beyond the disc space through the vertebral end plate into the vertebral body, as shown here in sagittal projection.

Spondylolysis refers to fatigue fracture of pars interarticularis and spondylolisthesis refer to a forward slippage of vertebra in comparison to the one below it. In adolescent athletes spondylolytic spondylolisthesis is most common at L5, especially among those who in engage in repetitive extension type activities. On the other hand in older people, nonspondylolytic spondylolisthesis is common at L4 due to a degenerative elongation of the pars interarticularis. Sometimes, these individuals may complain of radicular signs of nerve entrapment, or signs of central stenosis due to the anterolisthesis. Spondylolisthesis should therefore be considered as more serious condition requiring radiological evaluation to monitor deterioration of spondylolisthesis[28].

Degenerative marrow changes

There are three types of endplate changes. Type I changes are seen as low signal on T1-weighted images (T1 WI) and high signal on T2-weighted images (T2 WI) and likely represent endplate edema. Type II changes are seen high signal on T1 WI and T2 WI but appear dark on fat-suppressed sequences, likely represent fat. Type III changes are seen as low signal on both T1- and T2- WI and represent endplate sclerosis (Figure 21). These endplate changes are commonly referred to as "Modic" changes[26].

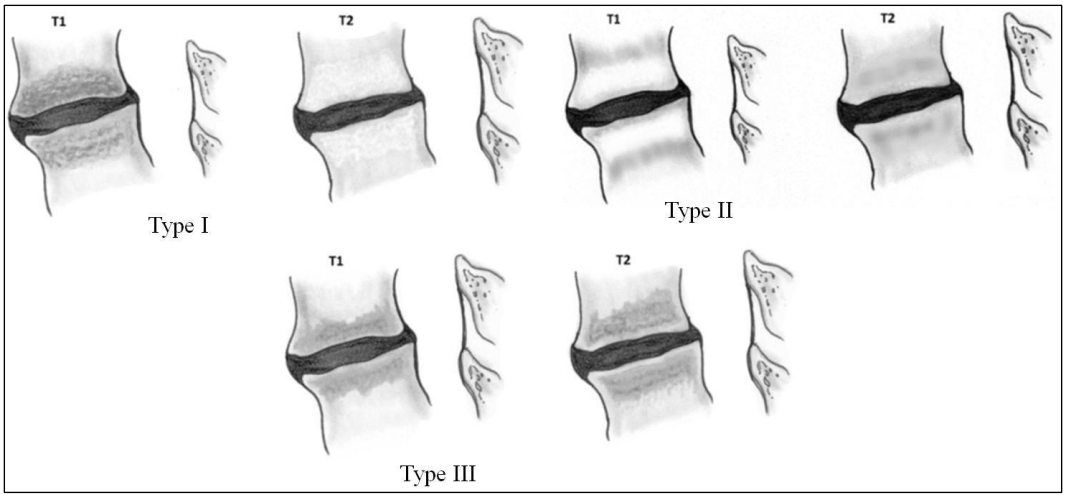


Figure 21. Reactive vertebral body marrow changes. These bone marrow signal changes adjacent to a degenerated disc on magnetic resonance imaging. T1- and T2-weighted sequences are frequently classified as (Top Left) Modic I, (Top Right) Modic II, or (Bottom) Modic III.

Central Canal Stenosis

Spinal stenosis has numerous causes such as congenital spine abnormalities and disk herniation. Disc herniation usually consists of triad of disk bulge, facet hypertrophy and ligamentum flavum hypertrophy[29]. Lumbar spinal canal stenosis is commonly encountered in older patients due to pre-existing spinal degeneration. Patients with lumbar spinal canal stenosis frequently give a history of recurrent low back pain. Multi-level stenosis may be common in patients with diffuse leg pain. Patients can also present with radiculopathy (due to nerve root compression) or neurogenic claudication. Pain may increase with extension of lumbar spine or during walking and gets relived by forward flexion or sitting. Although patients have radiculopathy symptoms, they complain of poorly localized, cramping pain with paraesthesias and

heaviness in the legs. Diagnosis may be difficult as neurologic signs may be present only with walking, in such cases a short walk prior to examination may be helpful. Clinically, signs of radiculopathy include positive straight leg raise test, decreased reflexes and sensory or motor loss[28].

Degenerative facet and ligamentous changes

Alterations of the articular cartilage of lumbar facet joints predispose them for arthropathy. Disc degeneration and loss of disc space height, increase stresses to facet joints with craniocaudal subluxation thereby leading to arthrosis and osteophytosis. The superior articular facet is more severely involved. Facet arthrosis can result in narrowing of the spinal canal, lateral recesses, and foramina and therefore can be considered as an important cause for lumbar stenosis. However, facet arthrosis has also been shown to occur independent of facetal stress and thereby become symptomatic. Furthermore, synovial villi may become entrapped within the joint resulting in joint effusions. Facetal arthropathy may cause low back pain either by nerve root compression from facetal degeneration or by direct irritation of pain fibers from the innervated synovial linings and joint capsule. Osteophytosis and herniation of synovium through the facet joint capsule may lead to formation of synovial cysts; however, etiology is still not completely elucidated. Synovial cysts have also been known to result in low back pain with increasing incidence in patients with facetal degeneration and synovial cysts compared to patients with facetal degeneration alone[28].

Alterations in vertebral articulations can result in ligamentous laxity. Ligamental changes include loss of elastic tissue, calcification /ossification, and bony proliferation at sites of ligamentous attachment to bone. Presence of excessive lordosis or extensive disk space loss results in close approximation and contact of spinous processes and ligamentous degeneration[28].

Infections

Spinal infections can be described based on etiology as pyogenic, granulomatous or parasitic. Most bacteria cause pyogenic response, whereas *Mycobacteria,* fungi, *Brucella spp*, and syphilis induce granulomatous reactions. Anatomical classifications include vertebral osteomyelitis, discitis, and epidural abscess. One more classification is based on the mode of spread – hematogenous, direct inoculation or spread from a contiguous source[30].

Pyogenic spondylitis and tuberculous spondylitis are common causes of spinal infection. It is difficult to differentiate tuberculous spondylitis and pyogenic spondylitis clinically and radiologically. Recently magnetic resonance imaging has been reported to be beneficial for early diagnosis and differential diagnosis of the spondylitis[30].

Pyogenic spondylitis is relatively rare and represents 0.15% to 3.9% of all osteomyelitis cases. Vertebral osteomyelitis is more common in the lumbar region, followed by thoracic and cervical spine (< 10%). Current data has shown that thoracic

spinal vertebrae have been found as the most common areas of tuberculous infection, followed by lumbar and thoracolumbar spine (in descending order by rate). In contrast, lumbar spine is the most commonly affected in pyogenic spondylitis, followed by thoracic and cervical spine (<10%)[30].

Data from various studies have shown that in tubercular spondylitis vertebral body damage is more severe compared to discal changes where as in pyogenic spondylitis disc is affected more commonly compared with vertebral body. This stark difference has been attributed due to lack of proteolytic enzymes in tubercular infections compared with pyogenic infections. The MRI findings in pyogenic spondylitis and tuberculous spondylitis are enumerated below (Table 1)[30].

Contrast enhanced MRI imaging is a good diagnostic tool and has a definite role to play in differentiation of tubercular and pyogenic spondylodiscitis[31].

Spinal tuberculosis is a destructive form of tuberculosis. Common clinical manifestations include constitutional symptoms, back pain, spinal tenderness, paraplegia, and spinal deformities A study reported that skeletal involvement in tuberculosis occurs mainly by hematologic dissemination. Onset is insidious with symptoms ranging from months to three years[32].

Table 1. MRI Findings in Pyogenic Spondylitis and Tuberculous Spondylitis

Variable	Pyogenic spondylitis	Tuberculous spondylitis
Para- or intraspinal abscess	Absence	Presence
Abscess wall	Thick and irregular	Thin and smooth
Postcontrast paraspinal abnormal signal margin	Ill-defined	Well defined
Abscess with postcontrast rim enhancement	Disc abscess	Vertebral intraosseous abscess
Vertebral body rim enhancement pattern	Homogeneous	Heterogeneous and focal
Involvement of vertebral bodies	Involvement ≤2 vertebral bodies	Multiple body involvement
Commonly involved region	Lumbar spine involvement	Thoracic spine involvement
Degree of disc preservation	Moderate to complete disc destruction	Normal to mild disc destruction
Bony destruction more than half	Infrequent and mild to moderate	Frequent and more severe

In TB spondylitis the cortical definition of affected vertebrae is invariably lost in contradistinction to pyogenic spondylitis. The sizes of the paraspinal masses have been noted to be generally larger in TB than in pyogenic infection. Spinal TB represents 25-60% of cases of skeletal TB. Thoracolumbar junction is affected most commonly and is relatively infrequent in the cervical and sacral segments of spine. Neurologic abnormalities may be encountered as a result of spinal cord compression from abscess, granulation tissue and bone fragments, and ischemia of the cord /intramedullary granulomas[33].

Careful correlation of MR imaging findings with clinical findings and lab data is important in outcome prediction in patients with spinal epidural abscess[34].

Neoplasms

Low back pain caused as a result of neoplasia accounts for <1% of cases of back pain; however, these patients may have morbidity if undiagnosed and untreated. Primary spinal cord tumours represent approximately 15% of all neoplasms of the CNS. Although most common initial presenting symptom can be non-specific back pain in individuals in spinal neoplastic tumours, they pose a diagnostic challenge for practitioners as symptoms can often non-specific[35].

Neoplasms are common in patients with age >50 years. Other clinical features include presence of previous malignancy, recent unexplained weight loss, constant pain that is worse at night, duration of pain greater than one month, failure of conservative treatment, elevated erythrocyte sedimentation rate (ESR) and anaemia. Presence of these clinical features should alert the practitioner towards possibility of primary or secondary bone tumour[28].

Primary bone tumours that are seen in lumbar spine include multiple myeloma and osteoid osteomas. Osteoid osteoma is a benign tumour characterized by constant, boring pain that is worse at night and dramatically relieved by aspirin. Scoliosis may also be seen due to chronic muscle spasm with the tumour at the concavity of the scoliosis due to chronic pain/irritation. Osteoid osteoma is seen commonly in 2nd and 3rd decade[28].

In adults multiple myeloma is the commonest malignant tumour affecting the bones. It is most common during 5^{th} to 7^{th} decade; however it is rare in patients <40 years of age. Individuals may present with mild, aching low back pain initially relieved by rest and aggravated by weight bearing. Subsequently, pain may worsen at night and not relieved by rest. Other, associated symptoms may include fever, pallor, diffuse bone tenderness and purpura[28].

Secondary bone tumours include metastasis from various cancers such as breast, prostate, lung and kidney. Although, metastases account for < 1% of back pain, they are 25 times more likely to affect the spine than primary bone tumours. Patients may present with insidious and constant low back pain unrelieved by rest, especially in patient aged > 50 years old. Clinical features suggestive of space occupying lesions may be present if there is extension of tumour into the spinal canal. Screening tests for metastases include ESR, serum protein electrophoresis, serum calcium, alkaline phosphatase, prostate specific antigen and x-rays[28].

Spinal cord tumours are classified according to their anatomical location and are separated into three broad categories: intramedullary, intradural extramedullary, and extradural. Intramedullary tumours are predominantly gliomas (astrocytomas and ependymomas), extramedullary tumours are most commonly peripheral nerve sheath tumours or meningiomas, and extradural lesions are usually metastatic. The most common primary spinal cord tumour in adults is ependymoma.

Spinal cord epidural metastasis (SEM), a common complication of systemic cancer has been seen with an increasing frequency. Prostate, breast and lung cancer are the most common offenders. Metastasis commonly arises in the posterior aspect of vertebral body with later invasion of epidural space. The most common complaint is pain, and two thirds of patients with SEM have motor signs at initial diagnosis. Currently magnetic resonance imaging is the most sensitive diagnostic tool[36].

MRI of the spine for the early diagnosis of Spinal cord compression may be considered useful in patients with extensive skeletal metastasis and back pain[37].

Gadolinium-enhanced MRI is highly important for surgical planning of spinal cord and cauda equina tumours[38].

Intradural tumors account for 10 to 20% of primary central nervous system (CNS) neoplasms in adults of which, about two thirds are extramedullary and, with rare exception, well circumscribed and histologically benign. The vast majority of intraspinal nerve sheath tumors are schwannomas (neurinoma/neurilemmoma) and neurofibroma. Most of these tumours are intradural and extramedullary. These tumors are seen most often in adults between the ages of 20 and 50 years. One sixth of the tumors have a purely extradural location, one sixth have both an intra and extradural component (dumbbell), while remainder are purely intradural[39].

Schwannomas are predominantly located laterally and obliquely to the spinal cord and may have broad based towards the dura. Schwannomas may have an epidural extension through intervertebral foramen with a ‘dumbbell’ shape.[40].

Spinal meningiomas are slow growing tumours, which are initially asymptomatic and with cord compression become symptomatic. MRI is the diagnostic tool of choice for diagnosis of meningiomas[41].

MRI facilitates in differential diagnosis of spinal neurinomas and meningiomas based on the specific MRI patterns[42]. The study concluded that MRI is the best imaging technique for diagnosis of spinal cord meningiomas[43].

Aneurysmal bone cysts (ABCs) although typically occur in the posterior elements they can often expand secondarily into the pedicles and vertebral body. In these patients, neural canal encroachment is common. Additionally, it may cause cord compression and pathological fracture. These patients most common present with pain and swelling[44].

Spinal cysts can cause extradural mass effect. For example, it has been observed that synovial cysts (juxta articular) may be a rare cause of extradural mass effect and is nearly always associated with facet degeneration. MR imaging helps to define the origin of the cyst (example, periarticular). The cyst will be localized adjacent to or dorsal to ligamentum flavum. Cyst may have a thin, well-defined rim of signal void due to its fibrous nature, peripheral calcification and occasional hemosiderin deposition. The signal characteristics of cyst fluid contents may parallel those of CSF but demonstrate no fluid motion. Slight hyperintensity on T1- weighted images has been described as the most common appearance (due to increased protein content). Congenital and acquired arachnoid cysts are uncommon but important

neoplastic causes of extradural mass effect. Extradural arachnoid cysts (EACs) are CSF-filled out pouchings of arachnoid layer that protrude through a dural defect; 2/3rd occur in the mid to low thoracic spine, 20% in the lumbosacral region and 9% at the thoracolumbar junction. Cervical EACs are uncommon. Secondary bony changes include widened interpedicular distance, scalloping of the vertebral bodies or pedicle thinning and erosion[45].

Congenital lesions

Tarlov/perineural cyst

A Tarlov cyst is a nerve root cyst originating between the layers of the perineurium and endoneurium near the dorsal root ganglion. The arachnoid membrane of the nerve root in the spinal dura mater is continuous with the cyst wall. The cyst wall is formed by the nerve fibers or reticulum. These were first reported by Tarlov in 1938 when he reported five such cases as an incidental finding during the autopsy. Tarlov's cyst are seen in about 4.6% in the general adult population with back pain. Although majority of them are asymptomatic (~70%), some of them have additive effects on other pathological conditions (~17%) and about 13% can be symptomatic. Tarlov cysts commonly present with low back pain, radiculopathy or cauda equina syndrome depending on their location, size and relationship to the surrounding nerve roots. The cysts often extend circumferentially around the involved nerves and can cause bony erosion along with compression and impingement on the surrounding structures. MRI is considered as imaging of choice for diagnosis of perineural cysts[46].

Scoliosis and kyphosis

Back pain has been shown to be associated with scoliosis and hyperkyphosis[23]. Congenital scoliosis is the commonest spinal disorder followed by congenital kyphosis and lordosis. Congenital scoliosis is believed to be either failure of formation or failure of segmentation of vertebrae or both, resulting in mixed deformity. Complete failure of formation results in hemivertebra, while wedged vertebra is seen with incomplete failure of formation. These malformations if present laterally cause scoliosis, lordoscoliosis if present posterolaterally, kyphoscoliosis if malformations are present anterolaterally and kyphosis if present ventrally and lastly lordosis if present dorsally. In the lumbar spine, kyphoscoliosis is common due to dorsal part malformation. Studies have shown low back pain with congenital scoliosis[47].

Lumbosacral Transitional Vertebrae

Lumbosacral transitional vertebrae (LSTV) is a congenital anomaly involving segmentation of the lumbosacral spine. LSTV includes either the involvement of L5 in sacrum or S1 into the lumbar vertebrae[48].

Sacralization of lumbar vertebra refers to addition of sacral elements into L5 vertebra. The incorporation of L5 vertebra with sacrum can be unilateral or bilateral and can be partial or complete. Complete sacralization refers to complete bony union between abnormal transverse process and sacrum. Incomplete sacralization shows a well-defined joint line between transverse process of L5 vertebra and sacrum. LSTV is one of the causes of low back ache. Lumbosacral transitional vertebrae (LSTV) occur as a result of congenital anomaly in the segmentation of the lumbosacral spine.

Sacralization means addition of sacral elements by the incorporation of Fifth lumbar vertebra. It is believed that LSTV can produce low back pain due to arthritic changes occurring at the false articulation sites. LSTV are common with the prevalence ranging from 1-20% among general population. There is conflicting evidence regarding role of LSTV in low back pain[48].

Lumbosacral transitional vertebrae are usually identified on lateral and Ferguson radiographs. Furthermore, Castellvi et al have described radiographic classification system of LSTVs based on morphologic characteristics into 4 types (Table 2; Figure 22)[49].

Table 2. Castellvi Radiologic Classification of LSTVs Based on Morphology[49].

Type		**Features**
Type 1	A	Unilateral dysplastic transverse process measuring at least 19 mm in width (craniocaudal dimension)
	B	Bilateral dysplastic transverse process measuring at least 19 mm in width (craniocaudal dimension)
Type II	A	Unilateral incomplete lumbarization/sacralization with enlarged transverse process with diarthrodial joint between itself and sacrum
	B	Bilateral incomplete lumbarization/sacralization with enlarged transverse process with diarthrodial joint between itself and sacrum
Type III	A	Unilateral lumbarization/sacralization with complete osseous fusion of the transverse process(es) to the sacrum
	B	Bilateral lumbarization/sacralization with complete osseous fusion of the transverse process(es) to the sacrum
Type IV		unilateral type II transition with a type III on the contralateral side

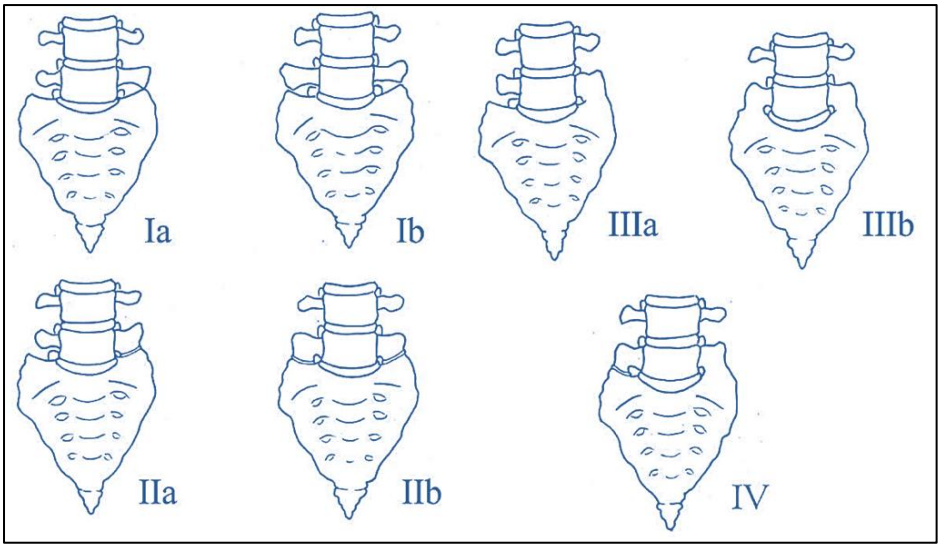


Figure 22. Illustration demonstrating the Castellvi classification of LSTVs.

Various studies evaluated causes, incidence and clinical features of LSTV. Although individuals with LSTV are usually asymptomatic, they may present with symptoms spinal or radicular pain, disc degeneration, L4-L5 disc prolapse, lumbar scoliosis and lumbar extradural defects. Significant lumbosacral intervertebral disc narrowing has also been observed in these individuals. In patients with LSTV, a higher incidence of disc herniation has been observed including in younger individuals. These patients are also prone for spondylolytic spondylolisthesis[49].

CLINICAL STUDIES

A retrospective analysis of LBP using MRI was performed in a tertiary care centre in North India in 232 patients aged 20 to 40 years. The authors concluded that MRI is helpful in depicting objective evidence of lumbar degenerative disc disease in symptomatic young adults in patients with clinical suspicion of disease[50].

The Back Pain Cohort of Southern Denmark (BaPa Cohort) study was conducted with the aim of analysing the clinical relevance of MRI in diagnosis of early spondyloarthritis. The study involved 48 patients who underwent MRI of spine including sacroiliac joints. The authors concluded that there is an excellent agreement for key MRI changes in the spine and sacroiliac joints and that MRI changes associated with non-specific LBP can be used for evaluation of the same and in patients with suspected spondyloarthritis[9].

Low back pain is a common clinical condition, which can result from pathologies involving lumbar spinal structures and sacroiliac joint. Many times, these clinical symptoms can overlap and may not be distinguishable clinically. Therefore MR imaging is necessary to differentiate the same. A study was conducted in 750 patients with low back pain who underwent MRI (age group 7 to 89 years). Study showed that sacroiliitis was an important cause of low back pain. The authors suggested that use of fat suppressed coronal sequence during evaluation of lumbar spine is simple and convenient screening strategy, which will help in evaluation of low back pain[51].

A study was conducted between patients aged 2 to 95 years who underwent MRI of spine in patients with low back pain or trauma or other spinal lesions. The study included 261 patients (187 males and 74 females with mean age of 46.3 ± 15.7 years. Most commonly performed studies were MRI of lumbosacral spine (46.45) followed by cervical spine, thoracolumbar spine. The most common indication for low back pain was low back pain in > 80% of patients. Spondylosis and moderate disc prolapses were seen in 31.5% of patients. Other causes were trauma, Pott's spine, spinal neoplasm, congenital kyphosis, etc. Although MRI was helpful in assessing causes for low back pain, the severity of findings may not necessarily correlate with severity of symptoms[52].

A cross-sectional study was conducted to evaluate etiologies of low back pain in patients with lumbar disk herniation to clarify whether disk herniation is main cause of low back pain and to evaluate if other diseases are responsible for low back pain. The study included 1250 patients with proven lumbar disk herniation by MRI study. Of these, 500 patients had chronic low back pain (40%). The authors concluded that a thorough evaluation of low back pain is essential for evaluation of causes for low back pain[53].

A study was performed by Bechara et al in 39 patients with chronic low back pain to identify relationships between objectively measured and subjectively scored parameters and reported pain. MRI findings of lumbar disc parameters were graded based on T2 signal intensity; nucleus shape, Modic changes, and osteophyte formation to form cumulative. The results showed that the most and least degenerated disks in each patient had the highest negative and positive correlation coefficient and

regression weight contribution respectively. The authors concluded that objective parameters, MRI index can be used as potential biomarkers for identifying and MRI is a potential investigative tool for identifying causative factors for low back pain[54].

A systematic review and meta-analysis was performed by Brinjikji et al to compare the prevalence of MR imaging features of lumbar spine degeneration in individuals aged <50 years presenting with and without low back pain. The meta-analysis included 14 studies involving 3097 individuals (1193, 38.6%, asymptomatic; 1904, 61.4%, symptomatic). Imaging findings with a higher prevalence in symptomatic individuals included disc bulges, spondylolysis, disc extrusion, Modic 1 changes, disc protrusion, and disc degeneration. The authors concluded that MR imaging evidence of disc bulge, degeneration, extrusion, protrusion, Modic changes and spondylolysis were seen significantly more in patients with low back pain compared to asymptomatic individuals[55].

A study was conducted in 200 patients to classify and quantify causes of low back pain based on MRI features. The study results showed that degenerative changes were the commonest cause for low back pain. Among disc herniations, disc bulge was common (79%) followed by disc protrusion (15%), disc extrusion (6%) and disc sequestration (<1%). Although there was no sex difference in disc protrusion there was a male preponderance in disc extrusion. The authors concluded that MRI is useful in classifying spinal lesions. Degenerative disc disease is the single most common cause in majority of patients[18].

A study was conducted in 109 patients with low back pain secondary to lumbar disc degeneration to characterize, extent, and changes associated with degenerative lumbar disk disease by MRI. The study included patients aged between 17 to 80 years. MRI findings were observed at multiple levels and included lumbar lordosis, Schmorl's nodes, decreased disc height, disk annular tear, disc herniation, disc bulge, disc protrusion and disc extrusion. Other imaging findings observed were narrowing of spinal canal, lateral recess and neural foramen with compression of nerve roots, ligamentum flavum thickening and facetal arthropathy. The study results showed that lumbar disc degeneration was the most common cause of low back pain. Although, plain radiograph may be helpful in evaluation of low back pain, MRI should be considered as the standard imaging modality due to excellent soft-tissue contrast and precise location of intervertebral discs changes[56].

A study was conducted by Weishaupt et.al to evaluate the predictive value of MRI in diagnosis of abnormalities of the lumbar intervertebral discs, particularly with adjacent endplate changes, to predict symptomatic disc derangement. The authors concluded that moderate and severe end plate abnormalities are useful in predicting painful disk derangement in patients with symptomatic low back pain[57].

A study was conducted in 108 patients in whom MRI intervertebral disc degeneration with lumbar degenerative disease and disc degeneration was evaluated by Pfirrmann and modified Pfirrmann grading system and correlated with Modic changes. The results showed that Modic changes correlated strongly with disc degeneration. The authors concluded that Modic changes correlate with Pfirrmann and modified Pfirrmann grades of disc degeneration in lumbar degenerative disease[58].

A study showed by that myeloma, breast cancer, prostate cancer, lung cancer and lymphoma are often seen involving the spine or soft tissue in the epidural space. The average age of patient with metastatic epidural disease can range from 53 to 58 years[59].

Metastatic lesions are often destructive and lytic but can be sclerotic, especially in prostate cancer. MRI is extremely sensitive in detection of metastasis in the vertebral bodies or extradural space. The multiplicity of lesions is strong evidence for a metastatic origin. However, in the case of single lesion, differentiation of a metastatic lesion from a primary tumor or from a lesion of another etiology can be difficult[60].

Meningiomas are the second most common intradural spinal tumor, next only to neurofibromas and account for approximately quarter of all primary spinal tumors. Women, usually between their fourth and fifth decades, account for approximately 80% of patients with spinal meningiomas. A review of six major case studies of spinal meningiomas that included 571 cases, showed a frequency of 17% for cervical, 80% for thoracic and 3% for lumbar tumours. Rare extradural extension or purely extradural lesions have been reported to occur at a frequency of 3.5% to 15%[61].

MATERIALS AND METHODS

Source of data:

This descriptive observational study was carried out over a period of 18 months from January 2015 to June 2016 in 106 patients with low back pain who underwent MRI of the lower spine at Department of Radio-Diagnosis, R. L. Jalappa Hospital & Research Centre attached to Sri Devaraj Urs Medical College. Patients who met the inclusion/exclusion criteria were included in the study.

Inclusion Criteria:

- Patients with low-back ache of non-traumatic etiology who underwent MRI of lower spine and had positive findings on MRI.

Exclusion Criteria:

- Patients with previous history of spinal surgery.

Method of collection of data:

The study was conducted in patients who underwent MRI for evaluation of low back pain and agreed to participate in the study. An informed consent was taken from the patient before including them in the study.

The following sequences of the lower spine were performed:

1. T2 weighted imaging (T2 WI) sagittal spine,
2. T1 weighted imaging (T1 WI) sagittal spine,
3. T1 WI axial images of relevant segments of spine,
4. T2 WI axial images of relevant segments of spine,
5. Coronal short τ wave inversion recovery (STIR) sequence of region of interest,
6. T1 fat saturation (FS) sagittal spine
7. T1 FS axial images of relevant segments of spine

Axial - T1 and T2WI & sagittal - T1 and T2WI sequences were performed with following parameters (Table 3):

Table 3. Summary of MR Imaging Parameters

Sequence	Orthogonal plane	Repetition Time (TR)	Echo time TE	Field of View	Matrix (mm)
T1 WI	Sagittal	550 – 700	10 – 21	280	1.1×1.1 ×4.0
T1 WI	Axial	550 – 700	10 – 21	260	0.9×0.9 ×4.0
T2 WI	Sagittal	3500 – 5000	80 – 120	280	0.9×0.9 ×4.0
T2 WI	Axial	3500 – 5000	80 – 120	260	0.8×0.8 ×4.0
STIR	Coronal	3000 – 3500	70 – 90	280	1.1×1.1 ×4.0
T1 FS Plain & contrast enhanced	Axial	500 – 600	10 – 20	280	0.9 × 0.9 ×4.0
	Coronal	500 – 600	10 – 20	300	1.2 × 1.2 ×4.0
	Sagittal	500 – 600	10 – 20	280	1.1×1.1 ×4.0

- Slice Thickness of 4mm
- Spacing - 1mm.`
- Flip angle - 150°.

Contrast MR study (I.V gadolinium injection) was performed wherever required. Patient's renal function was assessed in the form of blood urea and serum creatinine results and only patients with a normal renal function underwent contrast study (whenever indicated).

Baseline demographic data was recorded, which included the patient's age, gender, radiculopathy symptoms and clinical diagnosis. The MRI findings were analyzed with regard to location and extent of abnormality, which included degenerative changes, discal abnormality, vertebral end plate changes, ligamental and facetal changes, presence of lesion(s) and its characteristics (such as infective or neoplastic lesions), spinal canal stenosis, congenital abnormalities/conditions and incidental findings, if any. In patients who underwent surgery, MRI findings were correlated with surgical and pathology findings. A total of 50 patients underwent surgery and surgical findings were correlated. Descriptive statistics was used for data analysis. Microsoft Excel® was used for data analysis.

There are no definitive cut off value for diagnosis of lumbar canal stenosis[62,63]. Based on the experience at our institution and data available from other studies[64], we have defined lumbar canal stenosis as lumbar canal anteroposterior (AP) diameter of less than 10 mm on T2 W axial image. Additionally, the presence of other diseases such as disk protrusion, hypertrophic facet joint degeneration, absent fluid around the cauda equine, and ligamentum flavum hypertrophy in a symptomatic patient was also considered as having lumbar canal stenosis, irrespective of canal AP diameter at disc level[62].

Figure 23. Siemens Magnetom Avanto® 1.5 T MRI scanner used in the study.

RESULTS

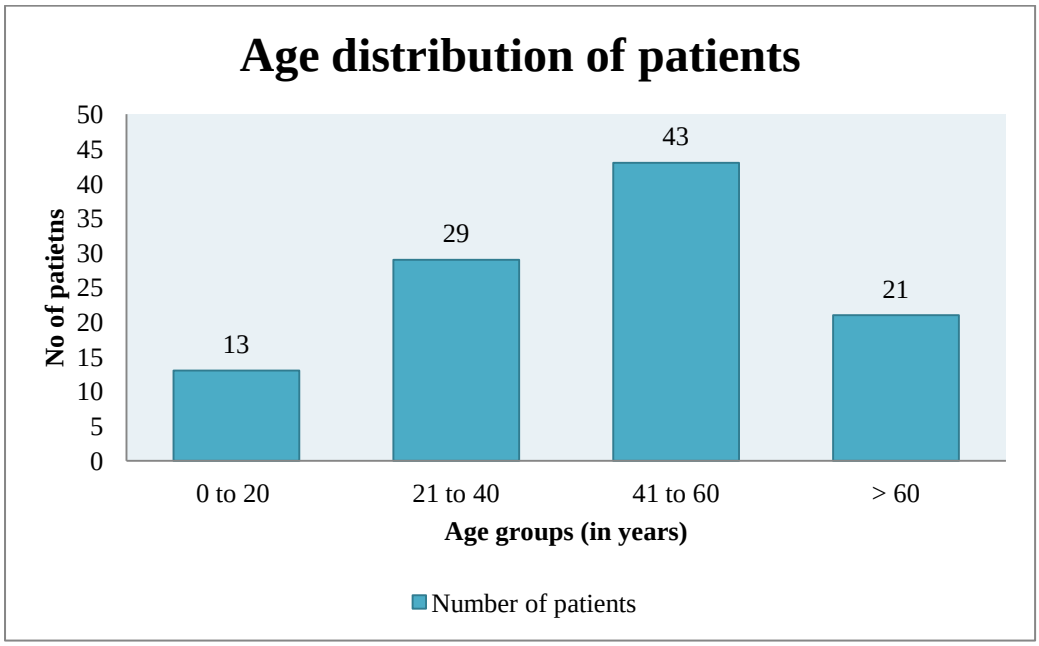


Figure 24. Distribution of patients based on age group.

The study included a total of 106 patients (Table 4). More than 40% of patients (n = 43; 40.57%) were in the age group of 41 to 60 years (Figure 24). There were 29 patients in the age group of 21 to 40 years (27.36%), followed by age group of > 60 years (n = 21; 19.8%) and least patients were in the age group of <20 years (n = 13; 12.26%).

Table 4. Distribution of Patients Based on Age Group.

Age (in years)	Number of patients	%
0 to 20	13	12.26
21 to 40	29	27.36
41 to 60	43	40.57
> 60	21	19.81
Total	106	100

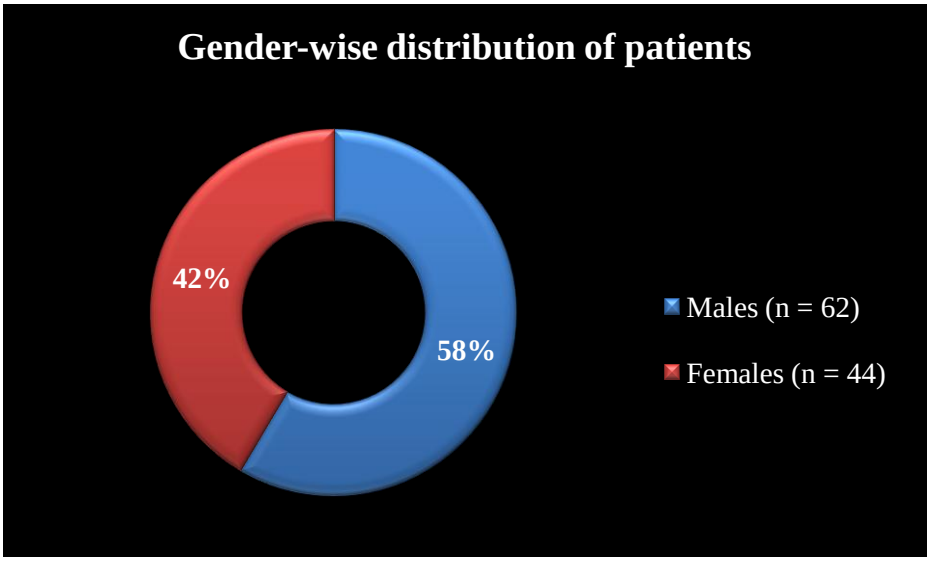


Figure 25. Gender-wise distribution of patients

There was slight male preponderance in the study with 58% of patients being males (n = 62) (Figure 25 and Table 5).

Table 5. Gender-wise distribution of Patients

Gender Distribution	No of patients	%
Males	62	58.49
Females	44	41.51
Total	106	100%

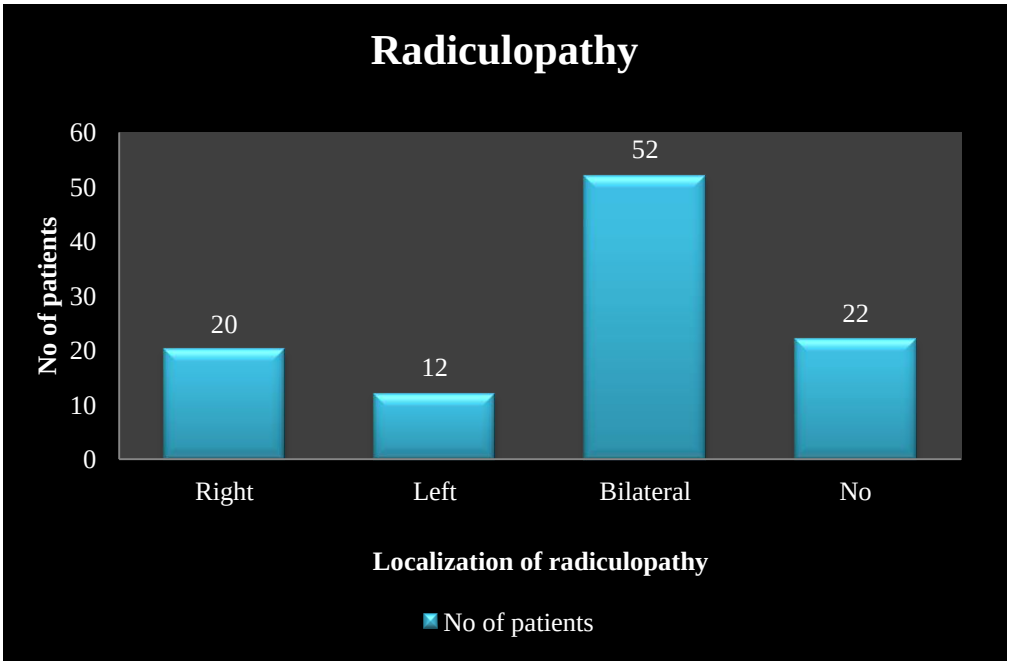


Figure 26. Localization of radiculopathy symptoms.

Nearly half of the patients (n = 52, 49.02 %) had bilateral radiculopathy followed by radiculopathy on right side (n = 20, 18.87%) and lastly left sided radiculopathy in (n = 12, 11.32%) (Figure 26, Table 6).

Table 6. Localisation of Radiculopathy Symptoms.

Radiculopathy	No of patients	%
Right	20	18.87
Left	12	11.32
Bilateral	52	49.06
No symptoms	22	20.75

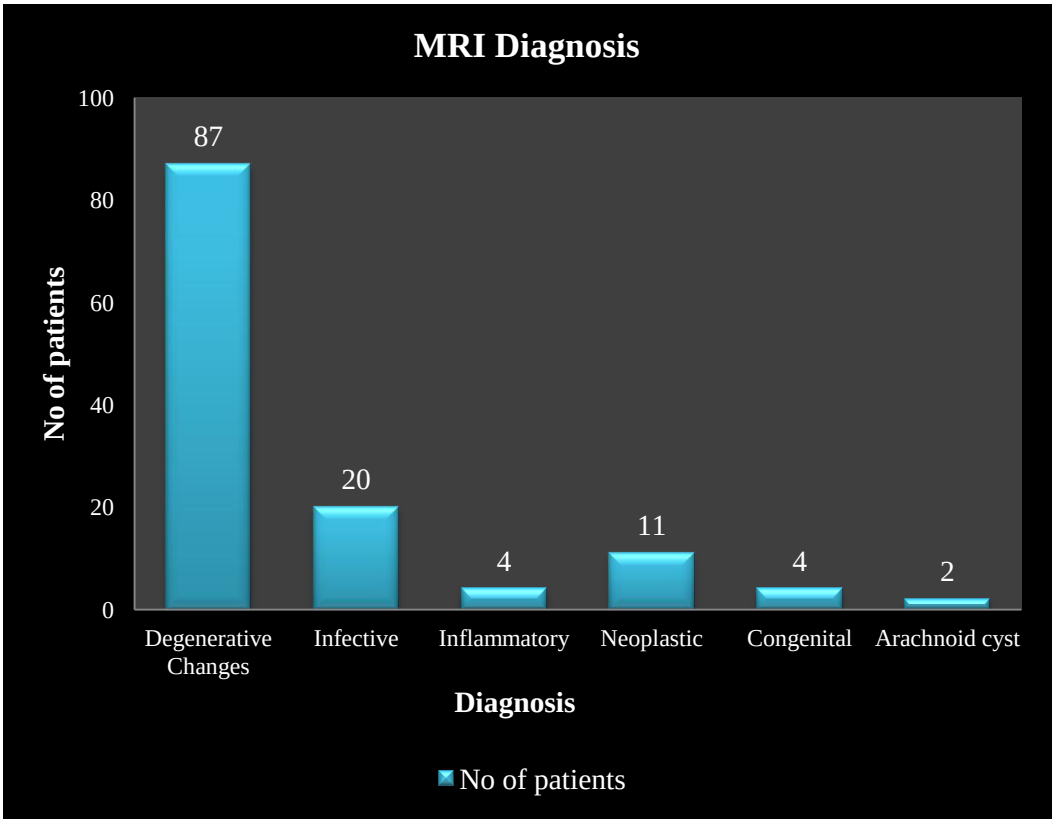


Figure 27. MRI diagnosis of various causes of low back pain

On MRI, degenerative changes were the commonest findings in more than 50% of patients (n = 87; 82.08%) followed by infective (n = 20; 18.87%) and neoplastic (n = 11; 10.38 %) etiologies (Figure 27). Four patients each had inflammatory and congenital etiologies (3.77%). Arachnoid cyst was seen in two patients (1.89%) (Table 7). A total of 65 patients had degenerative changes only and was therefore considered to be cause for low back pain. Remaining 22 patients had additional diagnosis of tuberculosis of spine (n = 7), metastases (n = 6), sacroiliitis (n = 4), arachnoid cyst (n = 2), arteriovenous malformation, multiple myeloma and sacral chordoma (n = 1 each). In these patients degenerative changes were not considered as the primary cause for low back pain.

Other findings reported in our study were LSTV-L (n = 5), LSTV-S (n = 13), Tarlov cyst (perineural cyst) (n = 16), kyphosis/scoliosis (n = 8) (n = 6), all of which were associated with aforementioned MRI diagnoses and were not considered as the primary pathology responsible for low back pain. Incidental findings of uterine fibroids (n = 6) and ovarian cysts (n = 12) were seen.

Table 7. MRI Diagnosis of Various Causes of Low Back Pain

MRI Diagnosis	No of patients	%
Degenerative Changes	87	82.08
Infective	20	18.87
Inflammatory	4	3.77
Neoplastic	11	10.38
Congenital	4	3.77
Arachnoid cyst	2	1.89

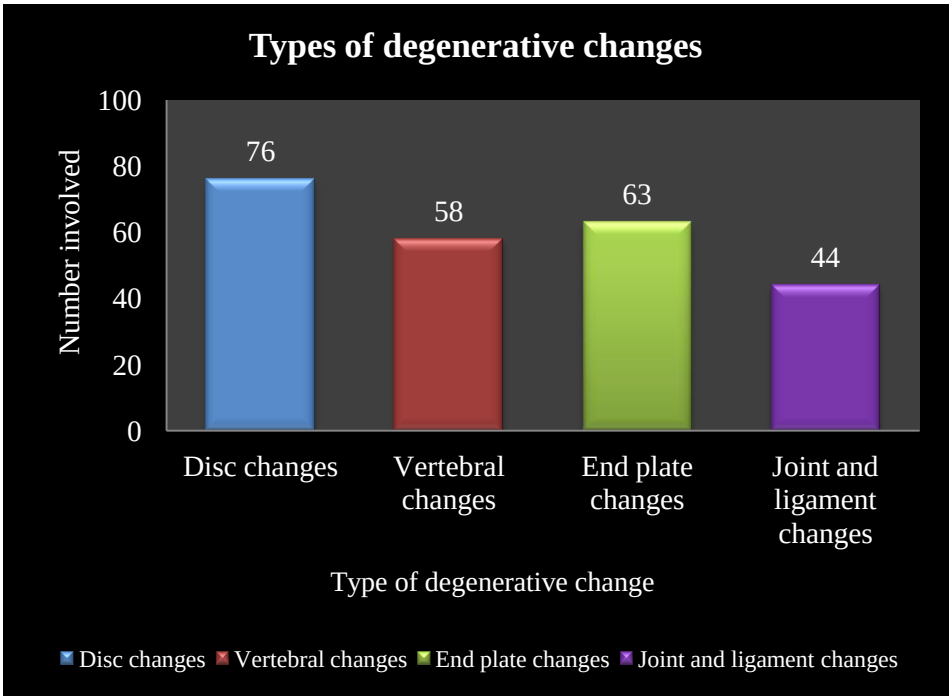


Figure 28. Types of degenerative changes

Among degenerative changes, disc changes were the commonest cause for low back pain seen in > 70% of patients (n = 76; 71.7% of study population) (Figure 28) followed by endplate changes (n = 63; 59.4%), vertebral changes (n = 58; 54.7%) and joint and ligament changes (n = 44; 41.5%) (Table 8).

Table 8. Types of Degenerative Changes

Degenerative Changes	No involved	%
Disc changes	76	71.7
End plate changes	63	59.4
Vertebral changes	58	54.7
Joint and ligament changes	44	41.5

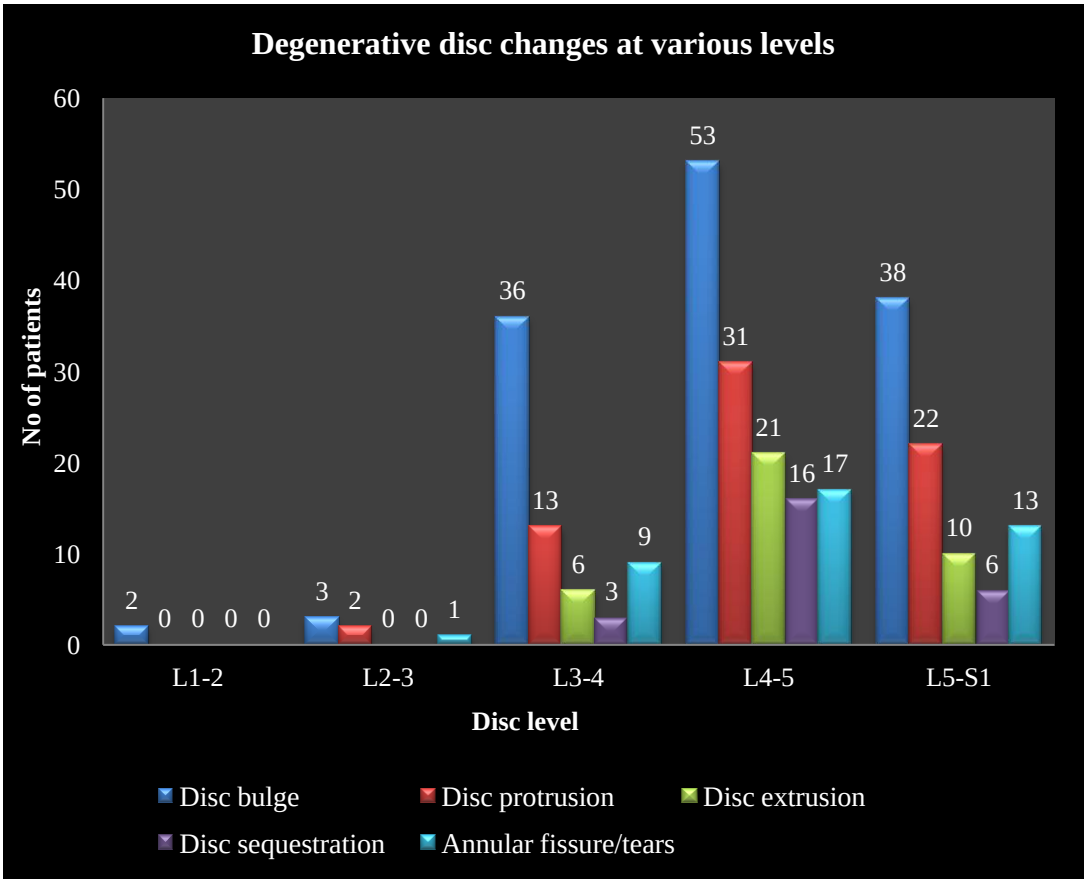


Figure 29. Degenerative disc changes at various levels

Degenerative disc changes were seen at multiple levels. There were a total of 302 degenerative disc conditions in 87 patients. Among them disc bulges were most common, seen in 132 discs (43.7%), followed by disc protrusion (68 discs; 22.52%), annular fissure/tears (40 discs; 13.25%), disc extrusion (37 discs; 12.25%) and disc sequestration (25 discs; 8.28%) (Figure 29). The most commonly affected discs were L4-5 followed by L5-S1 and L3-4. L1-2 and L2-3 were least commonly affected discs (Table 9). L4-5 was the commonest location for disc bulge (40.15%), disc protrusion (45.59%), disc extrusion (56.76%), disc sequestration (64%) and annular fissure/tears (42.5%) followed by L5-S1 and L3-4 levels.

Table 9. Degenerative Disc Changes at Various Levels

Degenerative disc change	**L1-2**	**L2-3**	**L3-4**	**L4-5**	**L5-S1**	**Total**
Disc bulge	2 (1.5%)	3 (2.27%)	36 (27.27)	53 (40.15%)	38 (28.78)	**132 (43.7%)**
Disc protrusion	0 (0%)	2 (2.94%)	13 (19.12%)	31 (45.59%)	22 (32.35%)	**68 (22.52%)**
Disc extrusion	0 (0%)	0 (0%)	6 (16.22%)	21 (56.76%)	10 (27.03%)	**37 (12.25%)**
Disc sequestration	0 (0%)	0 (0%)	3 (12%)	16 (64%)	6 (24%)	**25 (8.28%)**
Annular fissure/tears	0 (0%)	1 (2.5%)	9 (22.5%)	17 (42.5%)	13 (32.5%)	**40 (13.25%)**
Total	**2**	**6**	**67**	**138**	**89**	**302**

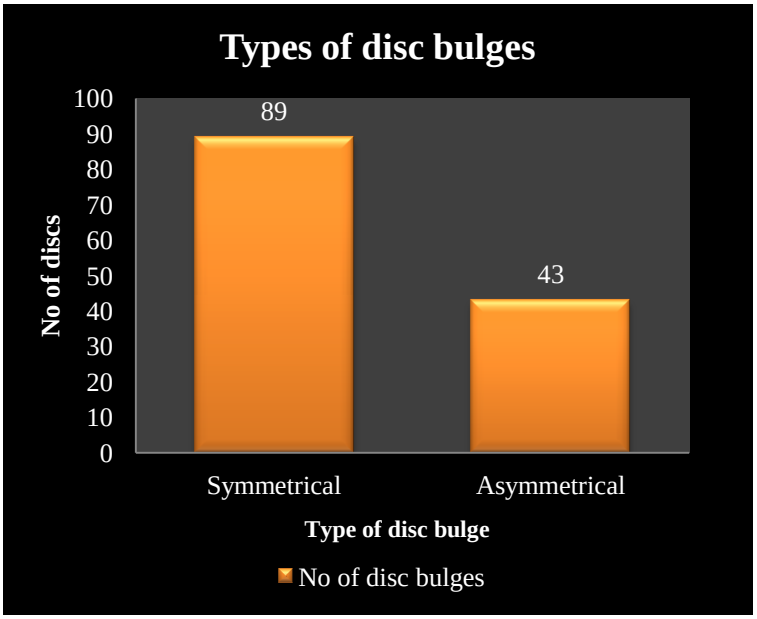


Figure 30. Types of disc bulges

Disc bulges were classified into symmetrical and asymmetrical disc bulges (Figure 30). Symmetrical disc bulges constituted about 2/3^{rds} of total disc bulges (n = 89, 67.42%) followed by asymmetrical disc bulges (n = 43, 32.58%) (Table 10).

Table 10. Types of Disc Bulges

Type of disc bulge	No of disc bulges	%
Symmetrical	89	67.42
Asymmetrical	43	32.58
Total	**132**	**100**

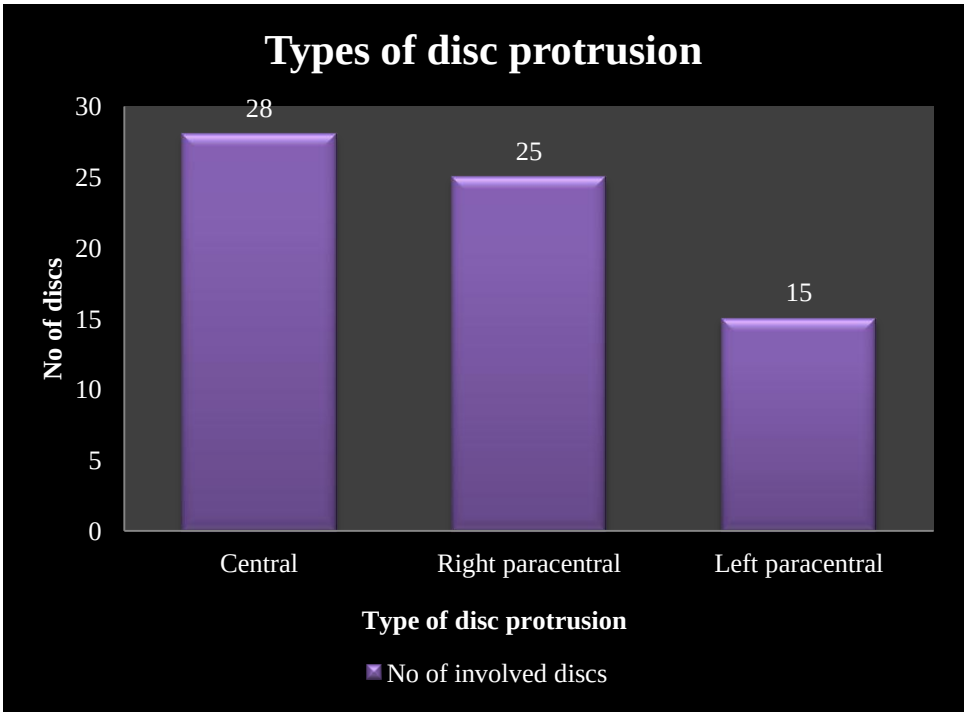


Figure 31. Types of disc protrusion

Disc protrusions were divided into central, right and left paracentral (Figure 31). Central disc protrusions were seen in >40% of involved discs (n = 28, 41.18%) followed by right paracentral disc protrusions (n = 25, 36.76%) and lastly left paracentral disc protrusions (n = 15, 22.06%) (Table 11).

Table 11. Types of Disc Protrusion

Types of disc protrusion	No of involved discs	%
Central	28	41.18
Right paracentral	25	36.76
Left paracentral	15	22.06

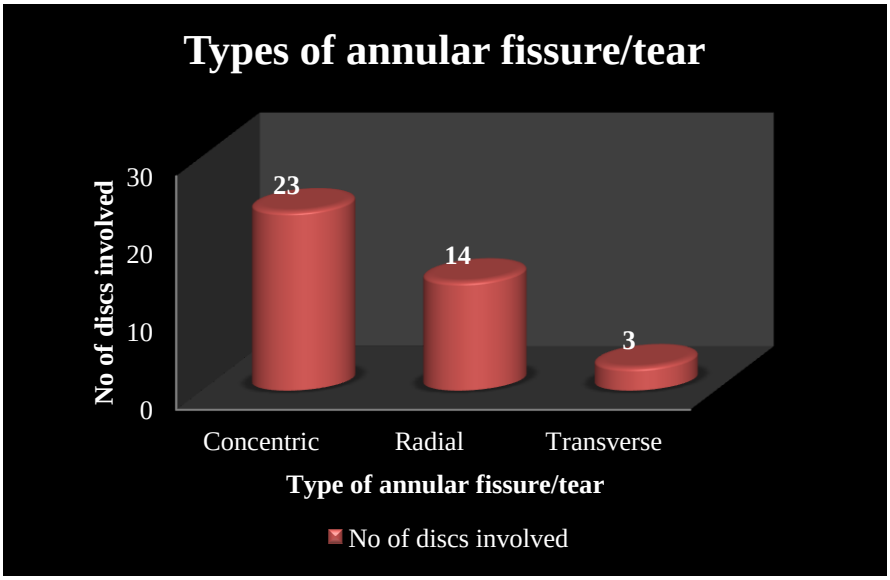


Figure 32. Types of annular fissure/tear

Annular fissure/tears were classified as concentric, radial or transverse (Figure 32). Concentric annular fissure/tears were the commonest type seen in more than 50% of involved discs (n = 23, 57.5%) followed by radial annular fissure/tear (n = 14, 35%) and transverse annular tear (n = 3; 7.5%) (Table 12).

Table 12. Types of Annular Fissure/Tear

Type of annular fissure/tear	No of discs involved	%
Concentric	23	57.5
Radial	14	35
Transverse	3	7.5
Total	**40**	**100**

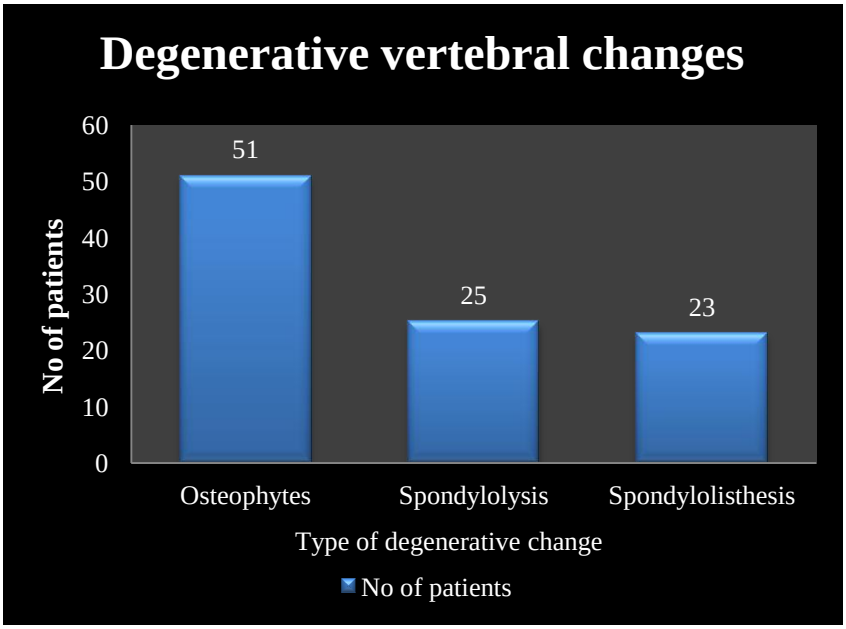


Figure 33. Degenerative vertebral changes.

Degenerative vertebral changes were seen in form of spondylolysis (n = 25 patients, 23.58% of degenerative vertebral changes), spondylolisthesis (n = 23; 21.7%) and osteophytes (n = 51; 48.12) (Table 13).

Table 13. Degenerative Vertebral Changes

Degenerative vertebral changes	**No of patients**	**%**
Osteophytes	51	48.12
Spondylolysis	25	23.58
Spondylolisthesis	23	21.70

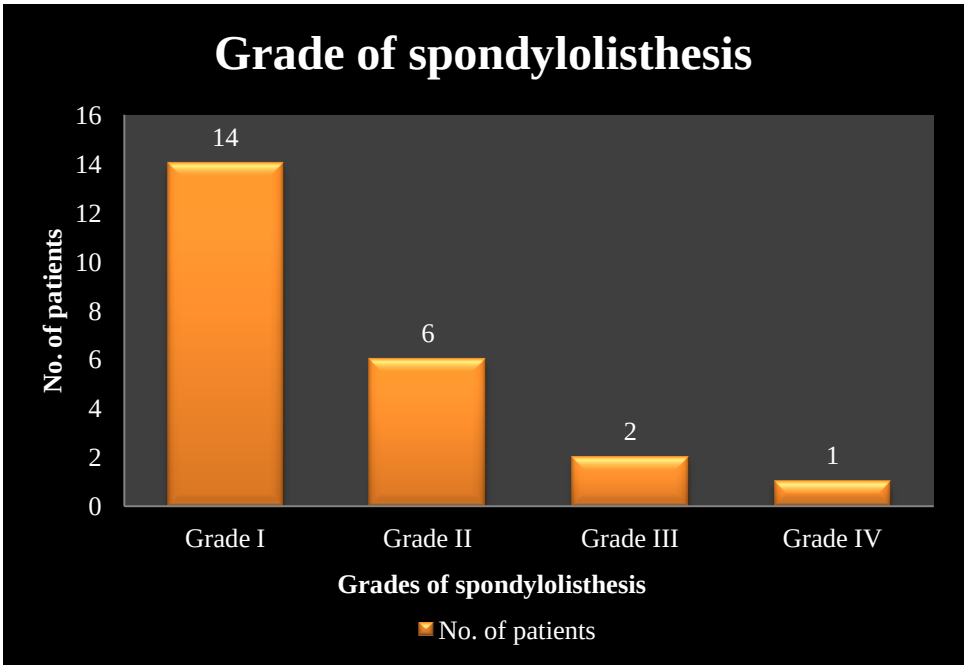


Figure 34. Grades of spondylolisthesis

Spondylolisthesis were seen in total of 23 patients and were graded from grades I to grade IV (Table 14). Grade I spondylolisthesis were seen in more than 60% of patients (n = 14, 60.9%) followed by grade II spondylolisthesis (n = 6, 26.1%), grade III spondylolisthesis (n = 2; 8.7%) and grade IV spondylolisthesis in one patient (4.3%) (Figure 34).

Table 14. Grades of Spondylolisthesis

Grades of spondylolisthesis	No of patients	%
Grade I	14	60.9
Grade II	6	26.1
Grade III	2	8.7
Grade IV	1	4.3
Total	**23**	**100**

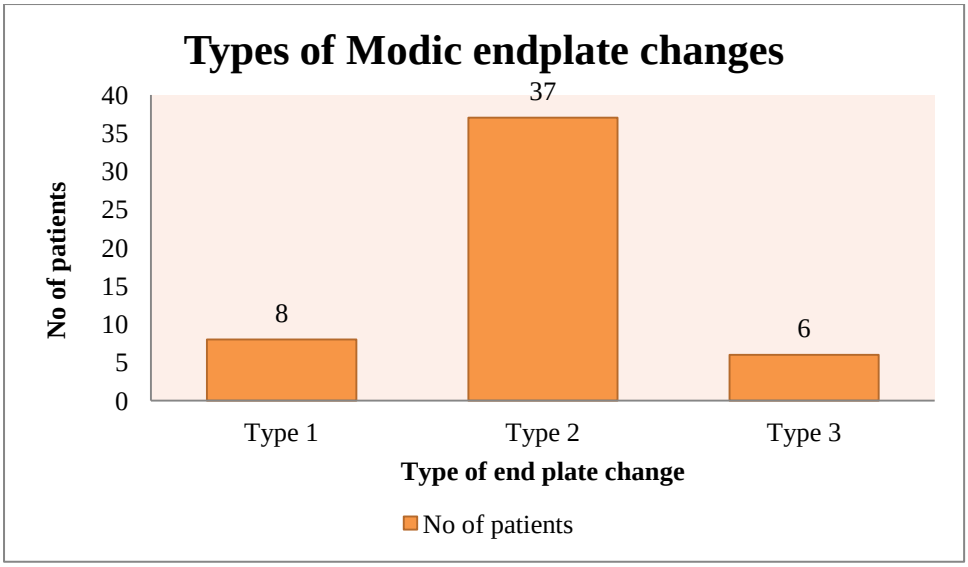


Figure 35. Types of Modic end plate changes.

Vertebral end plate changes were seen in the form of Schmorl's nodes seen in 48 patients and Modic endplate changes seen in 51 patients. Type II Modic end plate changes were commonest and seen in 37 patients (72.55%) followed by type I (n = 8, 15.69%) and type III Modic end plate changes (n = 6; 11.76%) (Table 15) (Figure 35).

Table 15. Types of Modic End Plate Changes

Type of end plate changes	No of patients	%
Type 1	8	15.69
Type 2	37	72.55
Type 3	6	11.76
Total	**51**	**100**

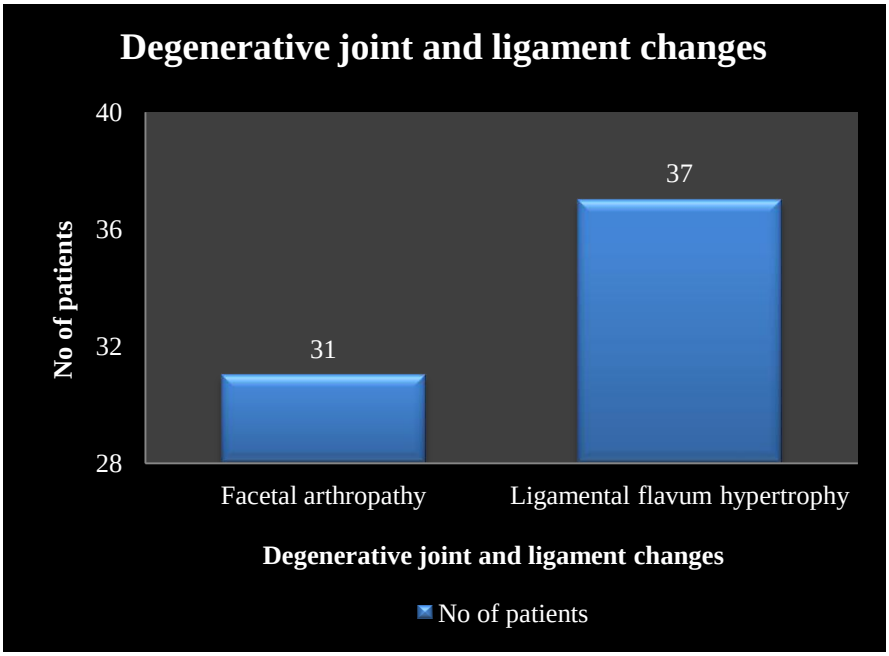


Figure 36. Degenerative joint and ligament changes

Degenerative joint and ligament changes were seen in the form of ligamentum flavum hypertrophy (LFH) and facetal arthropathy (FA) (Figure 36). LFH was seen in 37 patients (34.9% of study population) and facetal arthropathy were seen in 31 patients (29.25%) (Table 16).

Table 16. Degenerative Joint and Ligament Changes

Degenerative Joint and Ligament Changes	**No involved**	**%**
Facetal arthropathy	31	29.25
Ligamentum flavum hypertrophy	37	34.9
Total	**68**	

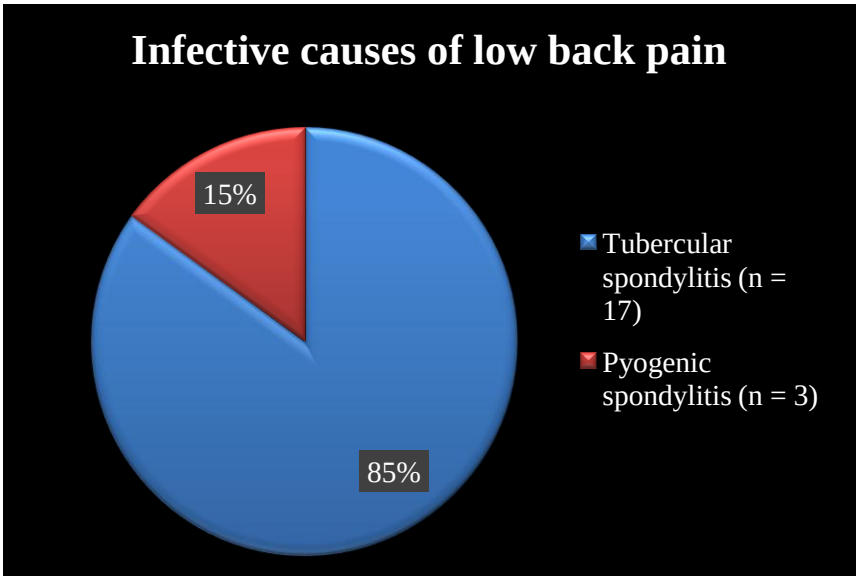


Figure 37. Infective causes of low back pain

Infective causes of low back pain were seen in 20 patients and included tubercular spondylitis and pyogenic spondylitis (Figure 37). Tubercular spondylitis was the commonest infective condition seen in 17 patients (85%) followed by pyogenic spondylitis (n = 3; 15%) (Table 17). In all these cases, final diagnosis was confirmed by demonstrating acid fast bacilli on Ziehl Neelson staining for tubercular spondylitis and culture/sensitivity for pyogenic spondylitis. Among pyogenic spondylitis, *S. aureus* was reported in two cases and *E. coli* in one case. All the cases with tubercular spondylitis were treated with antitubercular treatment and pyogenic spondylitis were treated with appropriate antibiotics.

Table 17. Infective Causes of Low Back Pain

Infective lesions	No of patients	%
Tubercular spondylitis	17	85
Pyogenic spondylitis	3	15
Total	**20**	**100**

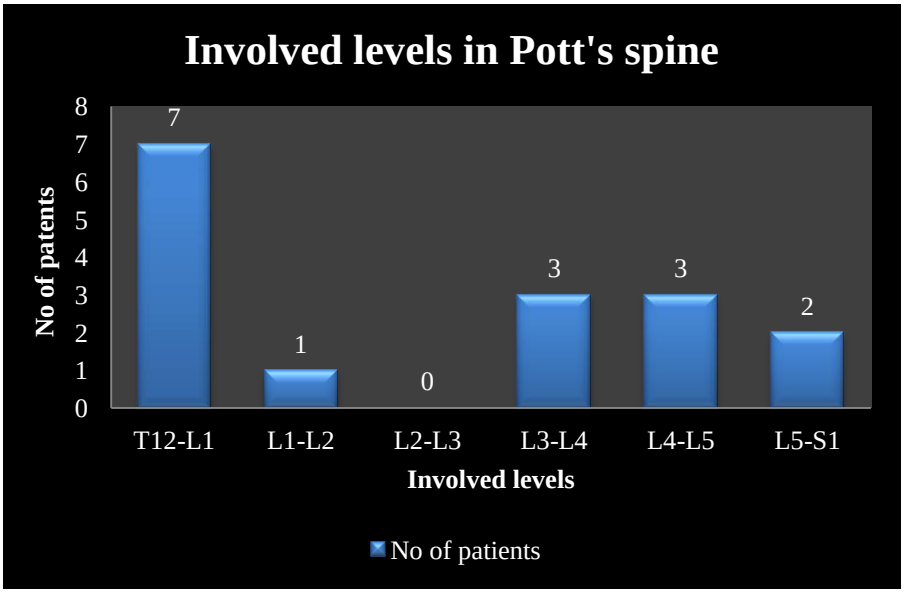


Figure 38. Involved spinal levels in Pott's spine

T12-L1 was the commonest spinal level involved in Pott's spine (n = 7, 43.75%) followed by L4-L5 and L3-L4 (n = 3 each, 18.75%) (Figure 38). L5-S1 was the least commonly involved and was seen in only two patients (12.50%) (Table 18).

Table 18. Involved Spinal Levels in Pott's Spine

Involved spinal levels in tubercular spondylitis	No. involved	%
T12-L1	7	43.75
L1-L2	1	6.25
L2-L3	0	0.00
L3-L4	3	18.75
L4-L5	3	18.75
L5-S1	2	12.50
Total	**16**	**100**

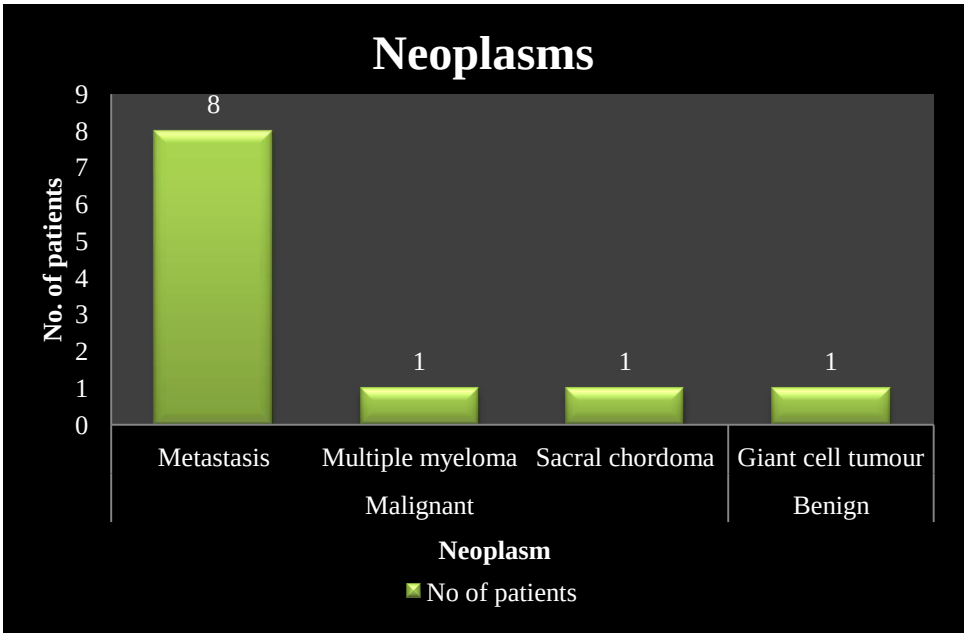


Figure 39. Neoplasms as a cause of low back pain

There were 11 neoplasms noted in our study, 10 were malignant and one was benign. Among the malignant conditions, most of them were metastasis, seen in eight patients. There was one each case each of multiple myeloma and sacral chordoma (Figure 39). There was a case of benign giant cell tumour (GCT).

The primary malignancy in metastasis were carcinoma prostate (n = 4), carcinoma lung (n = 3) and carcinoma esophagus (n = 1). Multiple myeloma was confirmed by electrophoresis. Sacral chordoma and GCT was confirmed by histopathology.

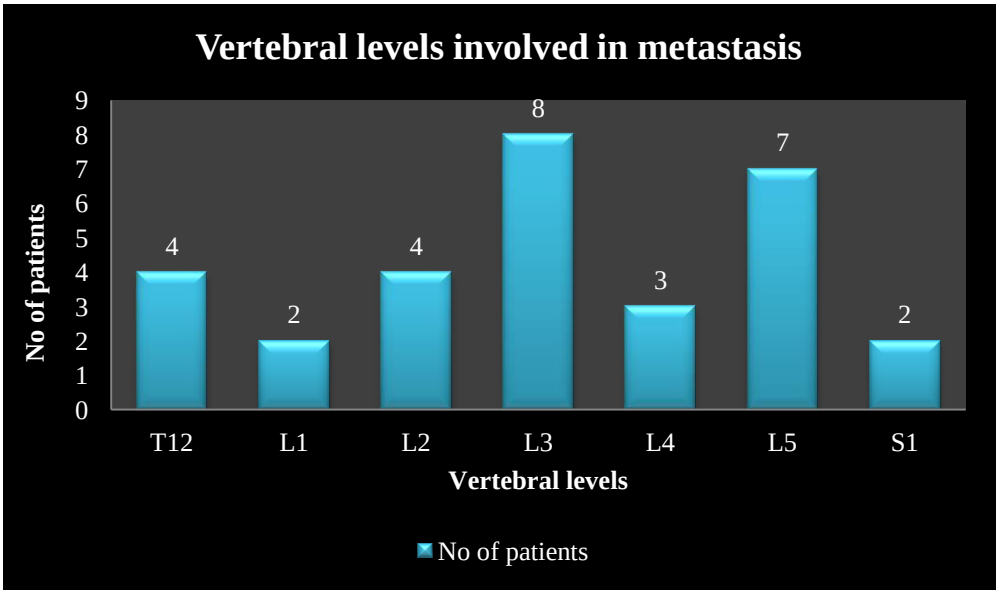


Figure 40. Vertebral levels involved in metastasis

L3 (n = 8 patients) was the most common location for metastasis, which was seen in all the patients with metastasis, followed by L5 (n = 7) (Figure 40). In all the patients the lesions involved multiple (≥2 in number) vertebral levels. L2 and S1 were the least commonly affected vertebral levels (n = 2 patients each) (Table 19).

Table 19. Vertebral levels involved in metastasis

Vertebral level	No of patients	%
T12	4	50
L1	2	25
L2	4	50
L3	8	100
L4	3	37.5
L5	7	87.5
S1	2	25

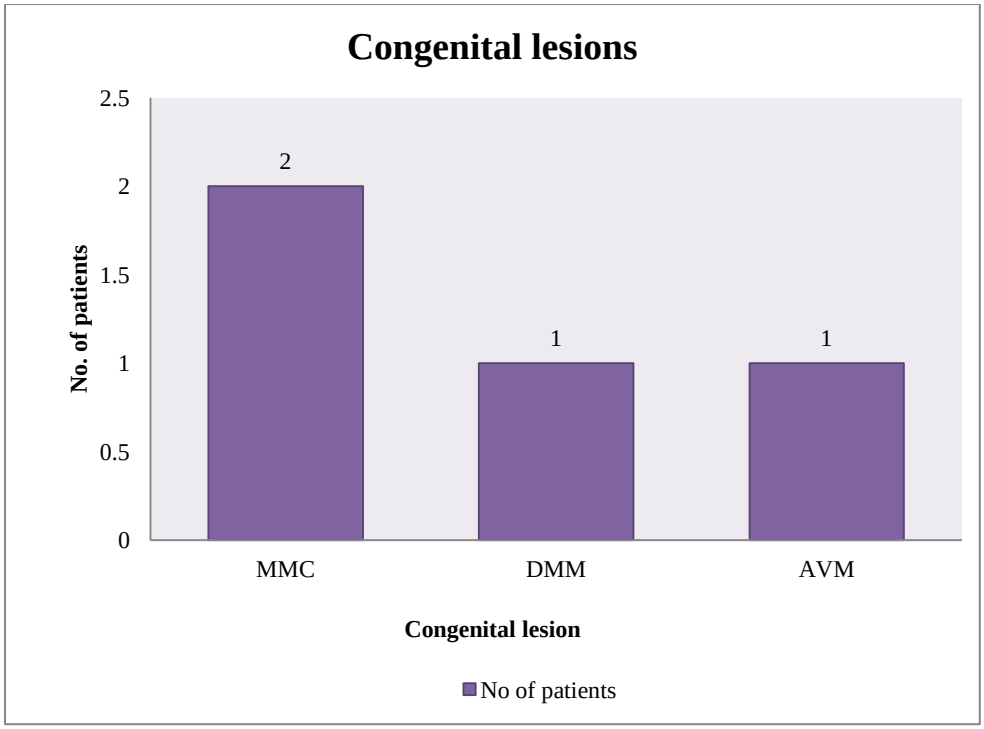


Figure 41. Congenital lesions (MMC – myelomeningocele; DMM – diastematomyelia, AVM – arteriovenous malformation)

The congenital conditions seen in our study are depicted in Figure 41. There were two cases of myelomeningocele, one case each of diastematomyelia and arteriovenous malformation (AVM) of spinal cord. All these cases underwent surgery and final diagnosis was confirmed. Other findings seen in our study were LSTV-L (n = 8), LSTV-S (n = 13), Tarlov cyst (perineural cyst) (n = 16) and kyphosis/scoliosis (n = 8), all of which were seen in patients who had primary MRI diagnoses and were not considered as the primary pathology responsible for low back pain.

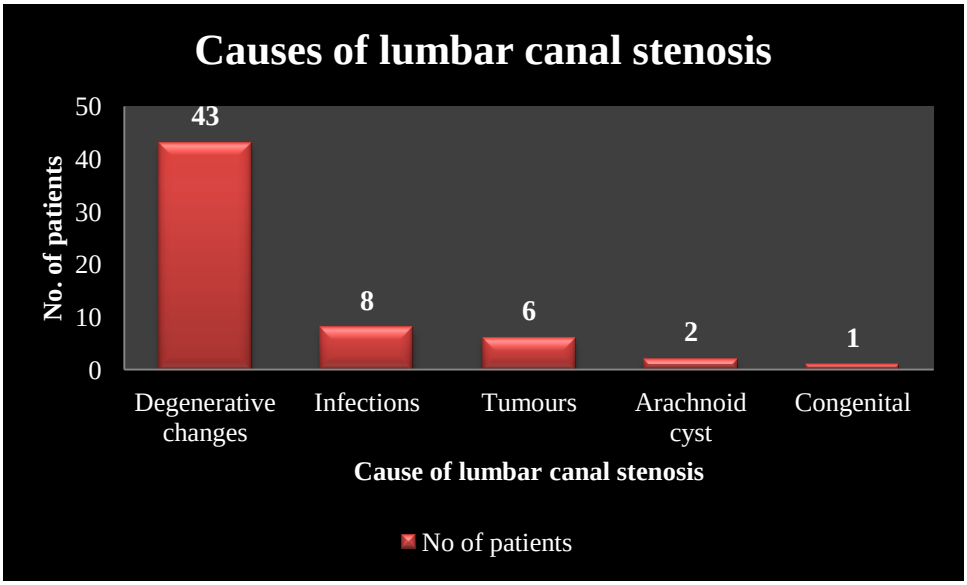


Figure 42. Causes of lumbar spinal canal stenosis

Lumbar spinal canal stenosis was seen in 60 patients in the study (Table 20). Degenerative changes were the commonest cause of lumbar canal stenosis (n = 43, 71.7%) followed by infections (n = 8, 13.3%), tumours (n = 6, 10%), arachnoid cyst (n = 2; 3.3%) and congenital lumbar spinal canal stenosis (n = 1; 1.7%) (Figure 42).

Table 20. Causes of Lumbar Canal Stenosis

Causes of lumbar canal stenosis	No. of patients	%
Degenerative changes	43	71.7
Infections	8	13.3
Tumours	6	10.0
Arachnoid cyst	2	3.3
Congenital	1	1.7
Total	**60**	**100**

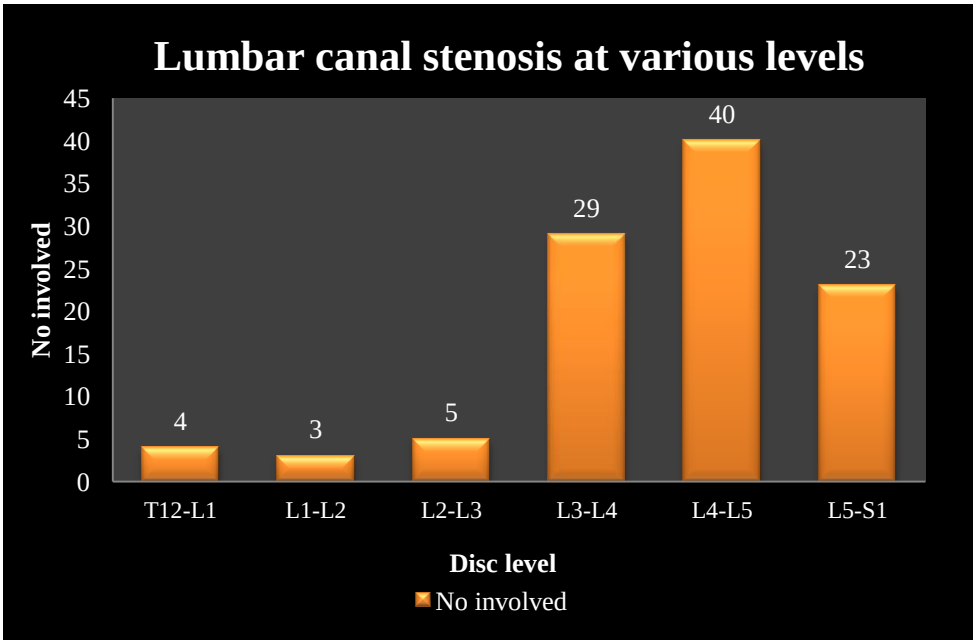


Figure 43. Lumbar canal stenosis at various levels

Figure 43 shows the involved discs of lumbar canal stenosis. There were 104 disc levels causing lumbar canal stenosis in our study. L4-5 was the commonest level involved (n = 40; 38.5%) followed by L3-4 (n = 29; 27.9%) and L5-S1 (n = 23; 22.1%). Remaining levels constituted for only 12 discs (Table 21).

Table 21. Lumbar Canal Stenosis at Various Levels

Level	No involved	%
T12-L1	4	3.8
L1-L2	3	2.9
L2-L3	5	4.8
L3-L4	29	27.9
L4-L5	40	38.5
L5-S1	23	22.1
Total	**104**	**100**

A comparison between MRI diagnosis and final diagnosis is presented in Table 22. There were total of 87 patients who were diagnosed with degenerative changes. A total of 65 patients had degenerative changes only and was therefore considered to be cause for low back pain. No further investigations were performed in these individuals and the final diagnosis in these patients was given as degenerative changes. Twenty two patients had diagnosis of tuberculosis of spine (n = 7), metastases (n = 6), sacroiliitis (n = 4), arachnoid cyst (n = 2), arteriovenous malformation, multiple myeloma and sacral chordoma (n = 1 each). In these patients degenerative changes were not considered as the primary cause for low back pain.

On MRI, there were 20 patients who were diagnosed with having infective etiology. Of these 17 patients were diagnosed with tuberculosis of spine (Pott's spine) and three patients were diagnosed as pyogenic spondylitis. In all these cases, final diagnosis was confirmed by demonstrating acid fast bacilli on Ziehl Neelson staining for tubercular spondylitis and culture/sensitivity for pyogenic spondylitis. Among pyogenic spondylitis, *S. aureus* was reported in two cases and *E. coli* in one case.

There were four patients who were primarily diagnosed with inflammatory sacroiliitis and were treated for the same. No further investigations were performed in these individuals and the final diagnosis was given as inflammatory sacroiliitis (Table 22).

There were 11 patients whose symptoms were attributed to neoplastic conditions on MRI. Among the malignant conditions, most of them were metastasis, seen in eight patients. There was one case where a differential diagnoses of giant cell

tumour (GCT), Brown's tumour and chondroblastoma was provided. Final diagnosis was given as GCT. There was one case of sacral chordoma and one case of multiple myeloma. In the patient with multiple myeloma, an additional differential diagnosis of metastasis was also provided. Final diagnosis of multiple myeloma was confirmed by electrophoresis (Table 22).

There were four cases who had congenital conditions that were determined to be cause for low back pain – arteriovenous malformation (AVM), diastematomyelia, myelomeningocele (thoracolumbar level) and sacrococcygeal myelomeningocele. All these conditions were correctly diagnosed on MRI (Table 22). Other findings seen in our study were LSTV-L, LSTV-S, Tarlov cyst/perineural cyst, and kyphosis/scoliosis. Incidental findings of ovarian cysts (n = 12) and uterine fibroids (n = 6) were also seen in our study.

Table 22. Comparison of MRI Diagnosis and Final Diagnosis.

MRI Diagnosis	No. of Patients	Final Diagnosis	No. of Patients
Degenerative changes*	87	Degenerative changes*	87
Infective - Pott's spine	17	Infective - Pott's spine	17
Infective - Pyogenic	3	Infective - pyogenic	3
Inflammatory - sacroiliitis	4	Inflammatory - sacroiliitis	4
Neoplastic† – GCT/Brown tumour/Chondroblastoma	1	Neoplastic - GCT	1
Neoplastic‡ - sacral chordoma/GCT	1	Neoplastic - sacral chordoma	1
Neoplastic - multiple myeloma/metastasisθ	1	Neoplastic - multiple myeloma	1
Neoplastic - metastasis	8	Neoplastic - metastasis	8
Congenital - AVM	1	Congenital - AVM	1
Congenital - diastematomyelia	1	Congenital - diastematomyelia	1
Congenital - myelomeningocele	1	Congenital - myelomeningocele	1
Congenital - SC myelomeningocele	1	Congenital - SC myelomeningocele	1
Arachnoid cyst	2	Arachnoid cyst	2

*Of 87 patients with degenerative changes, 65 had primary diagnosis of degenerative changes. Other conditions like tuberculosis of spine (n = 7), metastases (n = 6), sacroiliitis (n = 4), arachnoid cyst (n = 2), arteriovenous malformation, multiple myeloma and sacral chordoma (n = 1 each) were believed to cause low back pain in 22 patients.

† In this case three differential diagnoses were provided – GCT, chondroblastoma and Brown tumour

θIn this case two differential diagnoses were provided on MRI – multiple myeloma and metastasis. Final diagnosis was confirmed by electrophoresis.

‡ In this case two differential diagnoses were provided – sacral chordoma and GCT
GCT – Giant Cell Tumour, AVM – Arteriovenous Malformation, SC – Sacrococcygeal

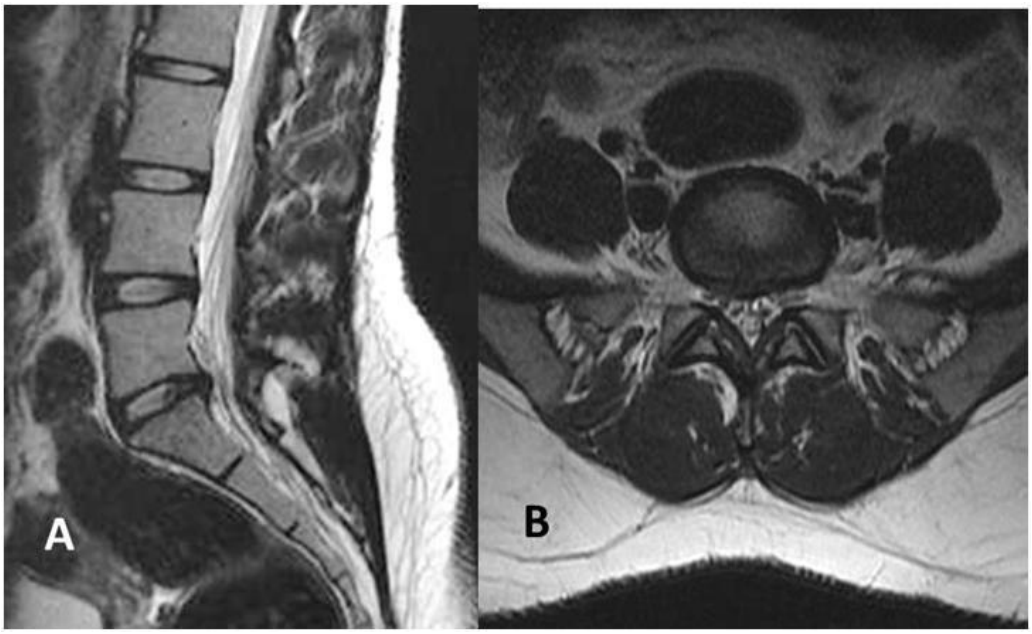


Figure 44. (A) Sagittal and (B) T2 weighted MRI of LS spine showing diffuse disc bulge at L5-S1 level. No significant spinal canal or neural foraminal stenosis.

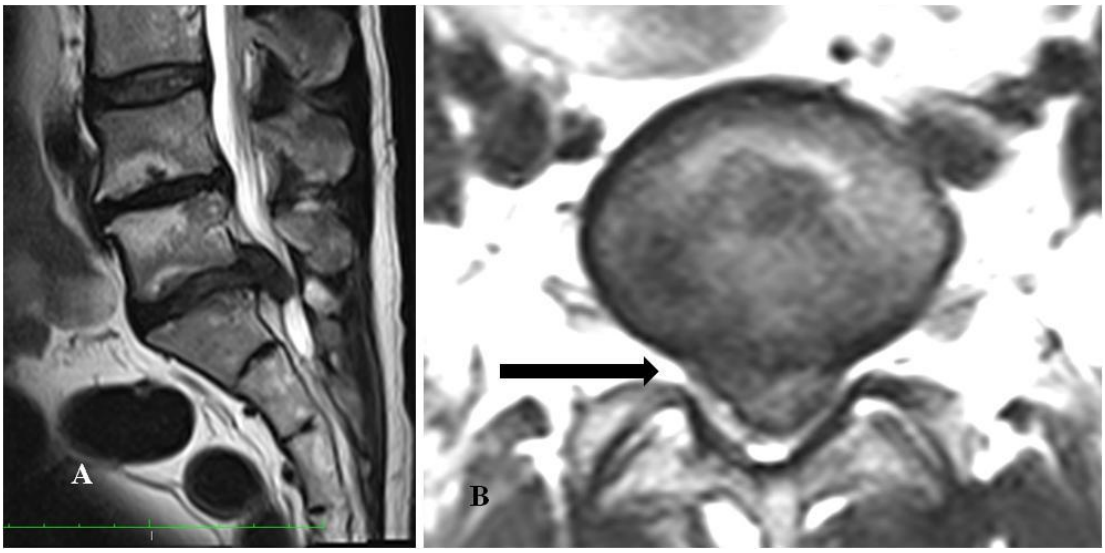


Figure 45. (A) Sagittal and (B) axial T2 weighted MRI of LS spine showing central disc protrusion with caudal extension causing spinal canal stenosis (black arrow).

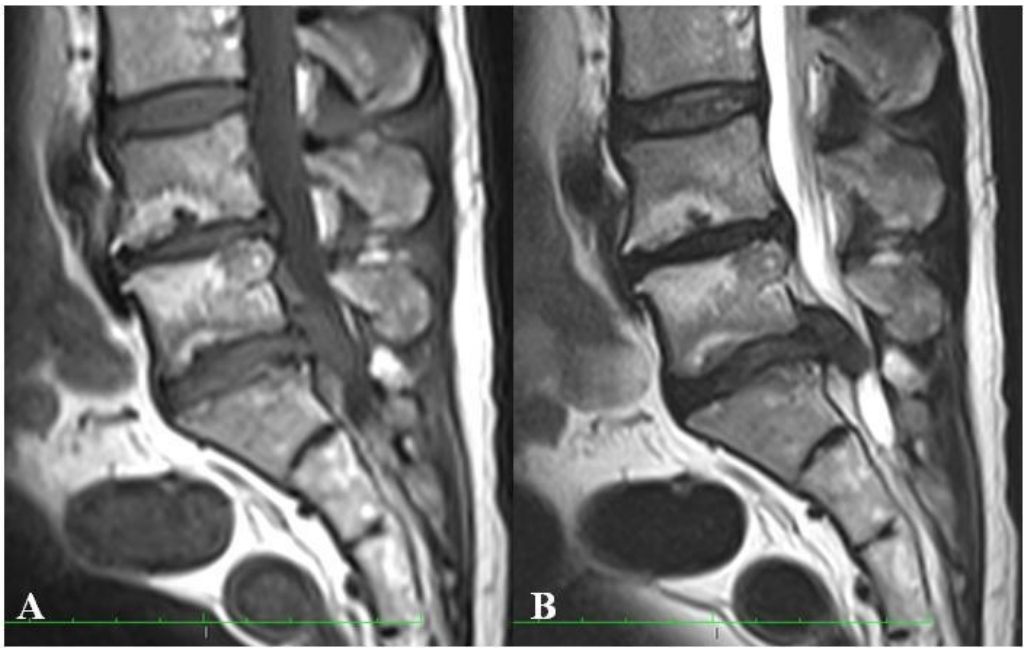


Figure 46. Sagittal (A) T1WI and (B) T2 weighted MRI of LS spine showing hyperintense signal in endplates of L4 and L5 on both sequences, suggestive of Modic type II changes.

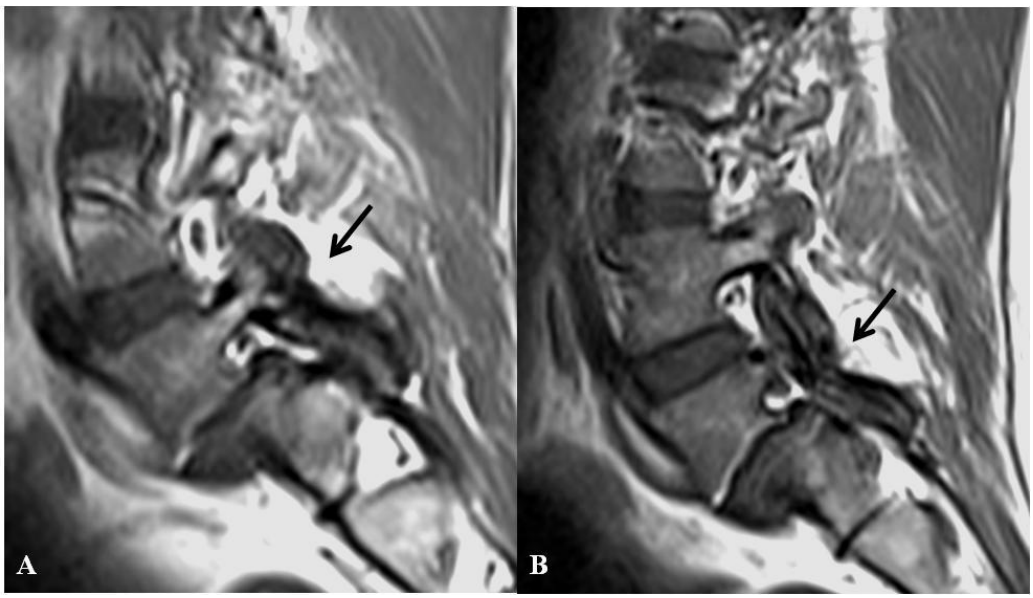


Figure 47. Sagittal (A) Right and (B) left T1weighted MRI of LS spine showing defect in bilateral pars interarticularis (black arrows), suggestive of bilateral spondyolysis.

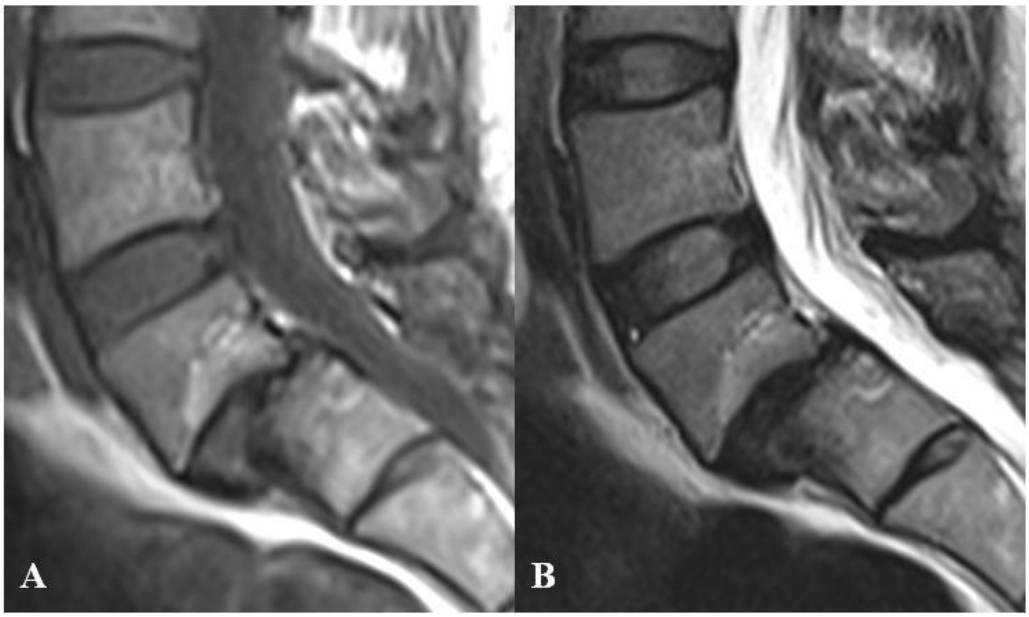


Figure 48 Sagittal (A) T1 and (B) T2 weighted MRI of LS spine showing anterior displacement of L4 vertebral body over L5, more than 25% of AP diameter, suggestive of grade II spondylolisthesis

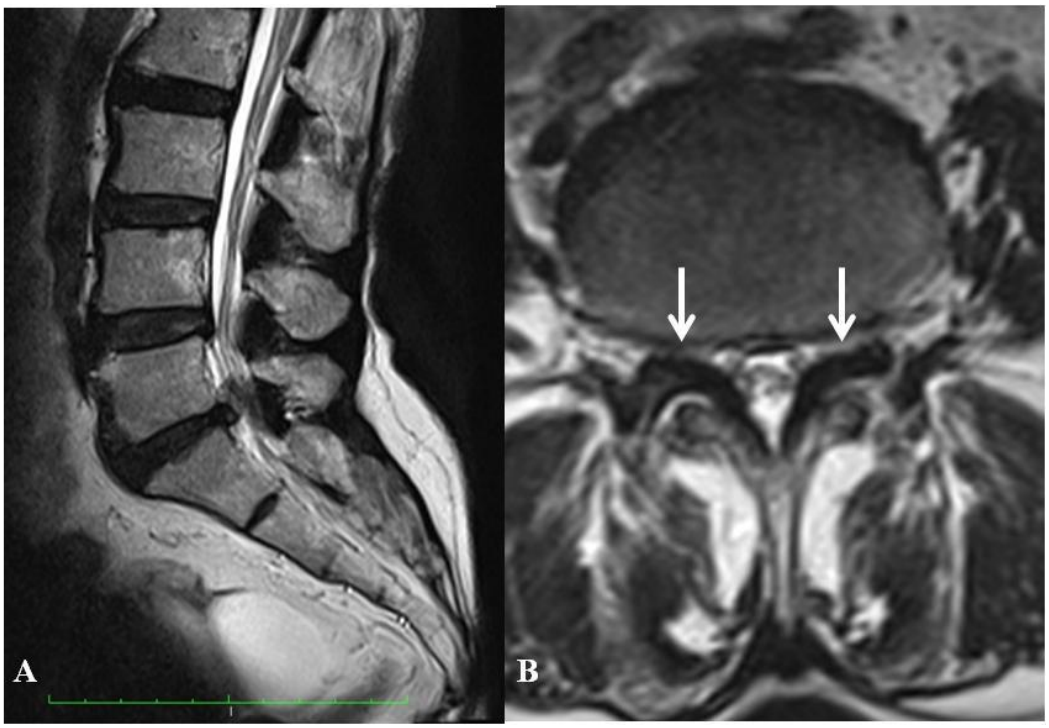


Figure 49 (A) Sagittal T2 and (B) axial T2 weighted MRI of LS spine showing disc bulge with ligamentum flavum thickening (white arrows), causing narrowing of spinal canal.

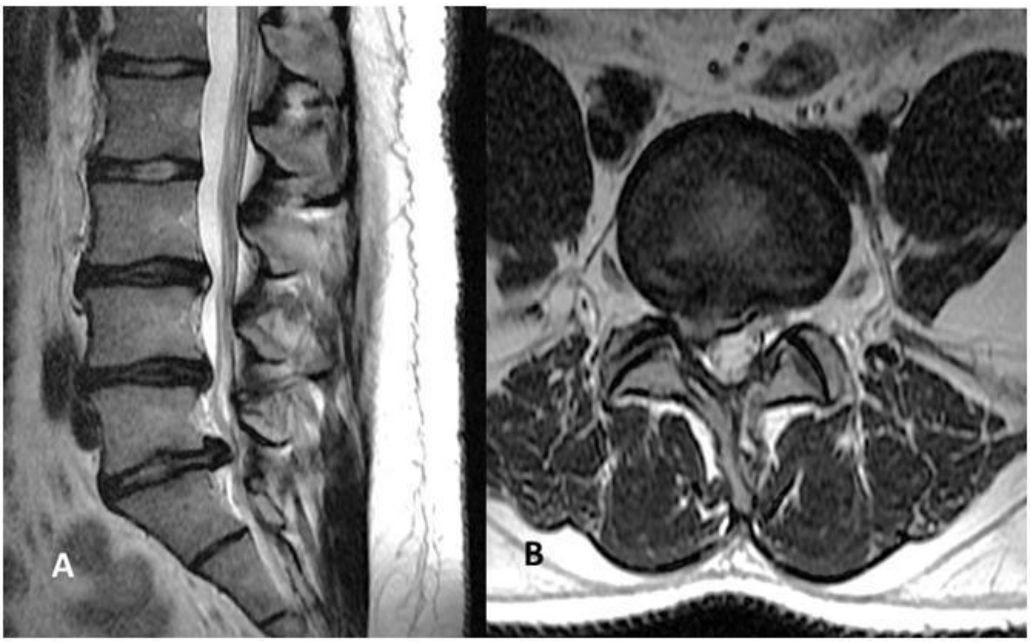


Figure 50 (A).Sagittal and (B) axial T2 weighted MRI of LS spine showing multilevel disc degenerative changes. At L5-S1 there is central and right paracentral disc protrusion causing effacement of the right lateral recess, narrowing of right neural foramen and compressing the right exiting nerve root. At L4-L5 level there is disc bulge. At L3-L4 level there is posterior annular fissure.

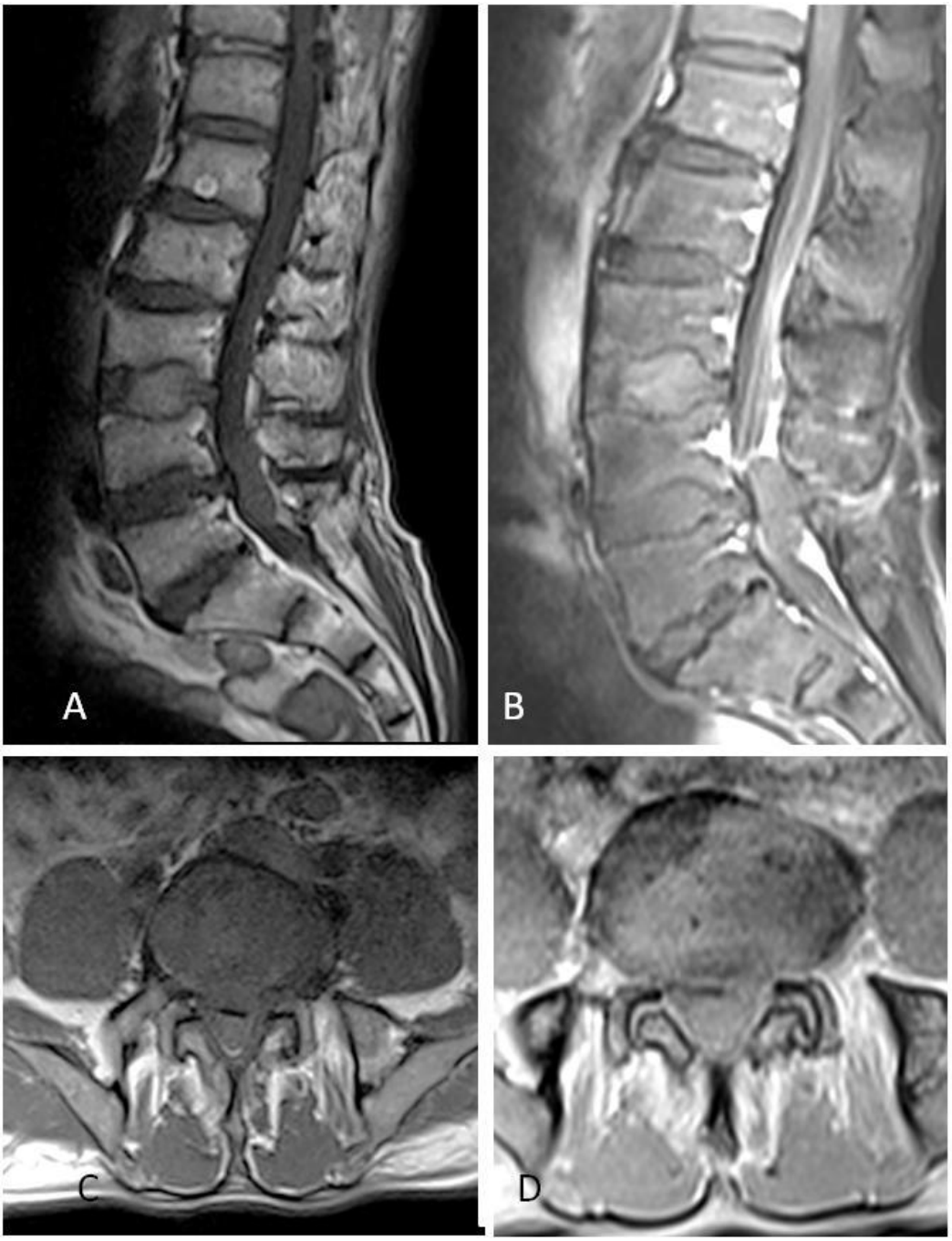


Figure 51. A 65-year-old male patient with complains of low back pain. (A) Sagittal and (C) axial T2 WI showing degenerative changes at L4-L5 and hypointense lesion in the spinal canal at the same level, causing spinal canal stenosis. (B) sagittal and (D) axial T1 weighted fat saturated contrast enhanced MRI showing non-enhancing isointense lesion at L4-L5 level, suggestive of sequestrated and posteriorly migrated intervertebral disc material.

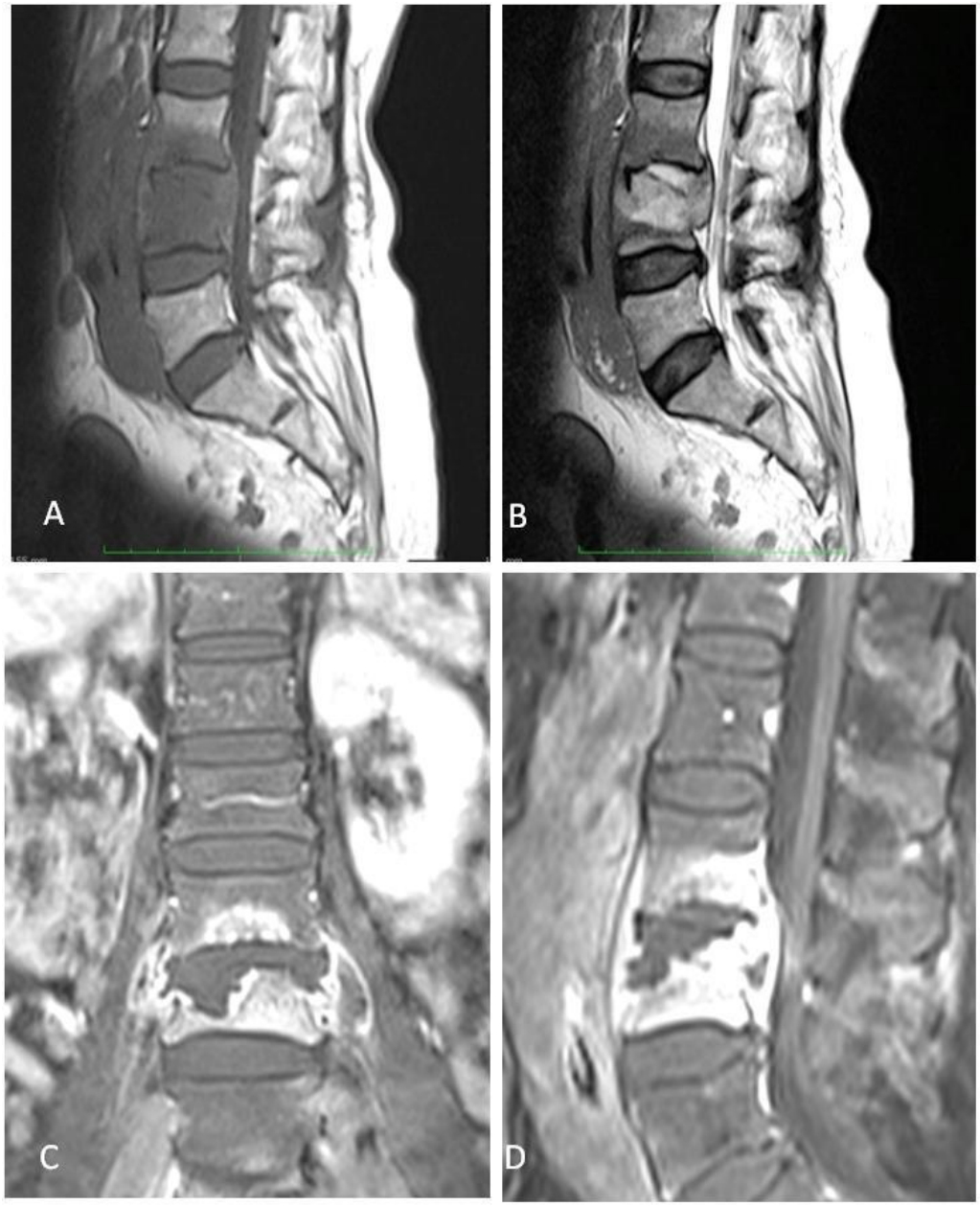


Figure 52 A case of tubercular spondylodiscitis. Sagittal (A) T1 and (B) T2 weighted MRI showing altered marrow signal involving L4 and L3 vertebral bodies causing spinal canal stenosis. (C) Coronal and (D) sagittal T1-weighted fat saturated MRI showing heterogeneously enhancing L4 and L5 vertebral bodies with loculated collections in pre and paravertebral regions at L3-L4 level, extending to epidural space with central non-enhancing necrotic areas– suggestive of Pott's spine.

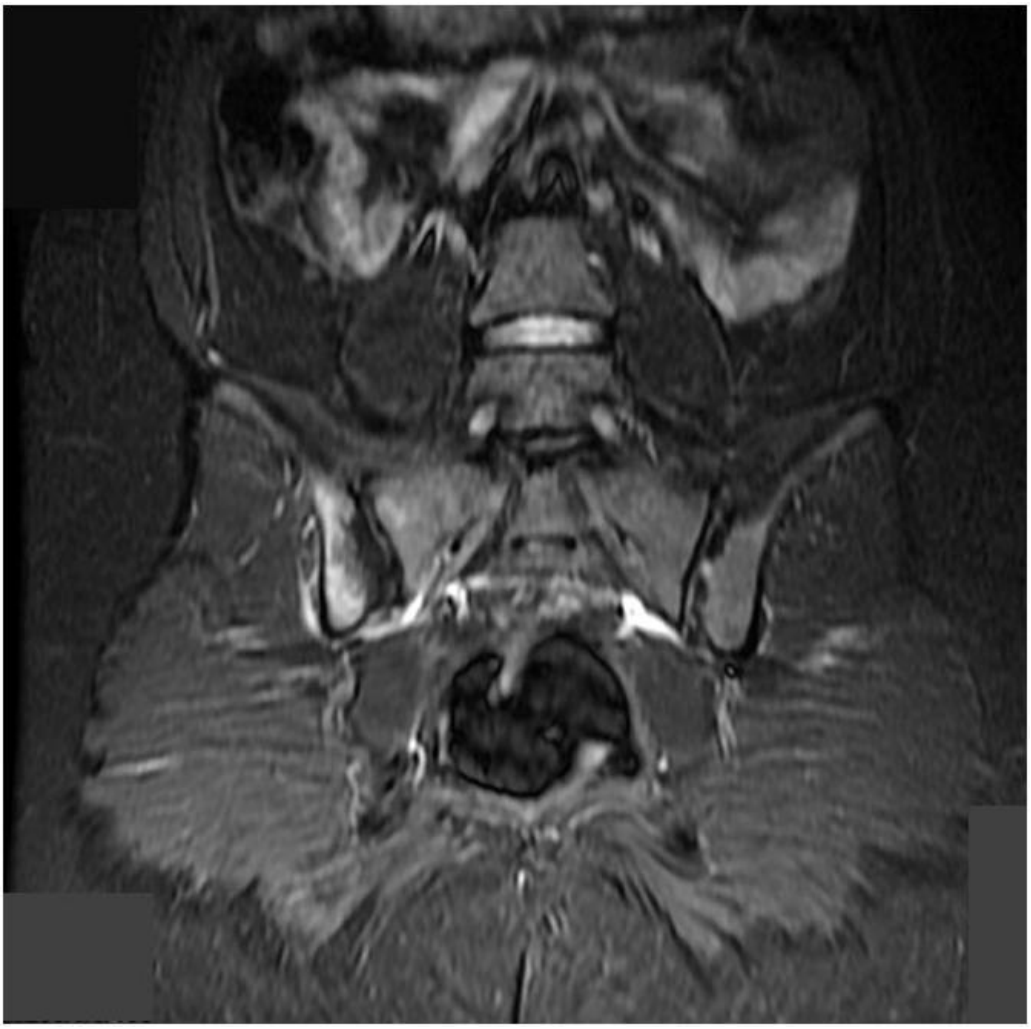


Figure 53 Coronal STIR image showing sacroiliitis on right side, predominantly involving the iliac region.

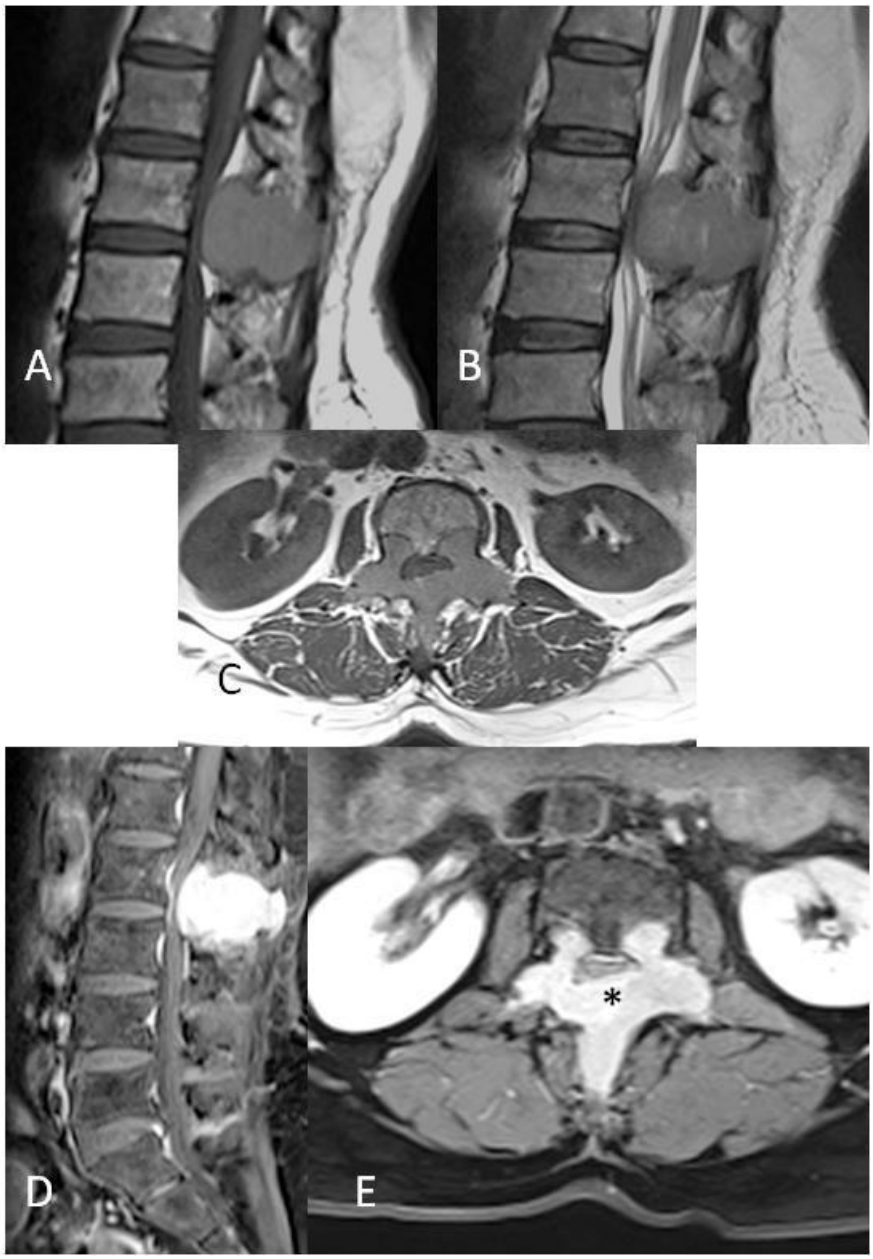


Figure 54 A case of giant cell tumour. Sagittal (A) T1, (B) T2 and (C) axial T1 weighted MRI of LS spine showing a well-defined, symmetric, expansile lesion at L2 vertebral level involving the spinous process, bilateral lamina, pedicles, transverse processes & superior and inferior articular processes and adjacent posterior part of vertebral body. It is mildly hyperintense to muscle on both T1 & T2W images. (D) Sagittal and (E) axial post-contrast T1 weighted fat-saturated MRI shows avid homogenous enhancement of the tumor (*).

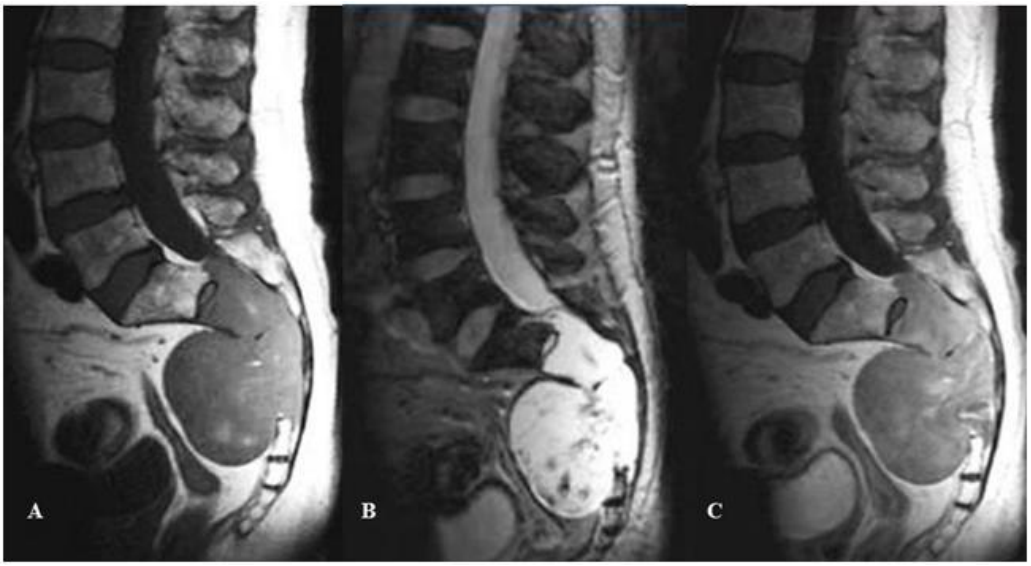


Figure 55. A 19-year-old female patient with sacral chordoma. Sagittal (A) T1 and (B) T2 weighted MRI of LS spine shows a large soft tissue mass lesion involving the sacral vertebrae (S2 to S4) with presacral component. It is hyperintense on both sequences (C) Post-contrast T1 weighted images show heterogeneous and moderate enhancement of the mass lesion.

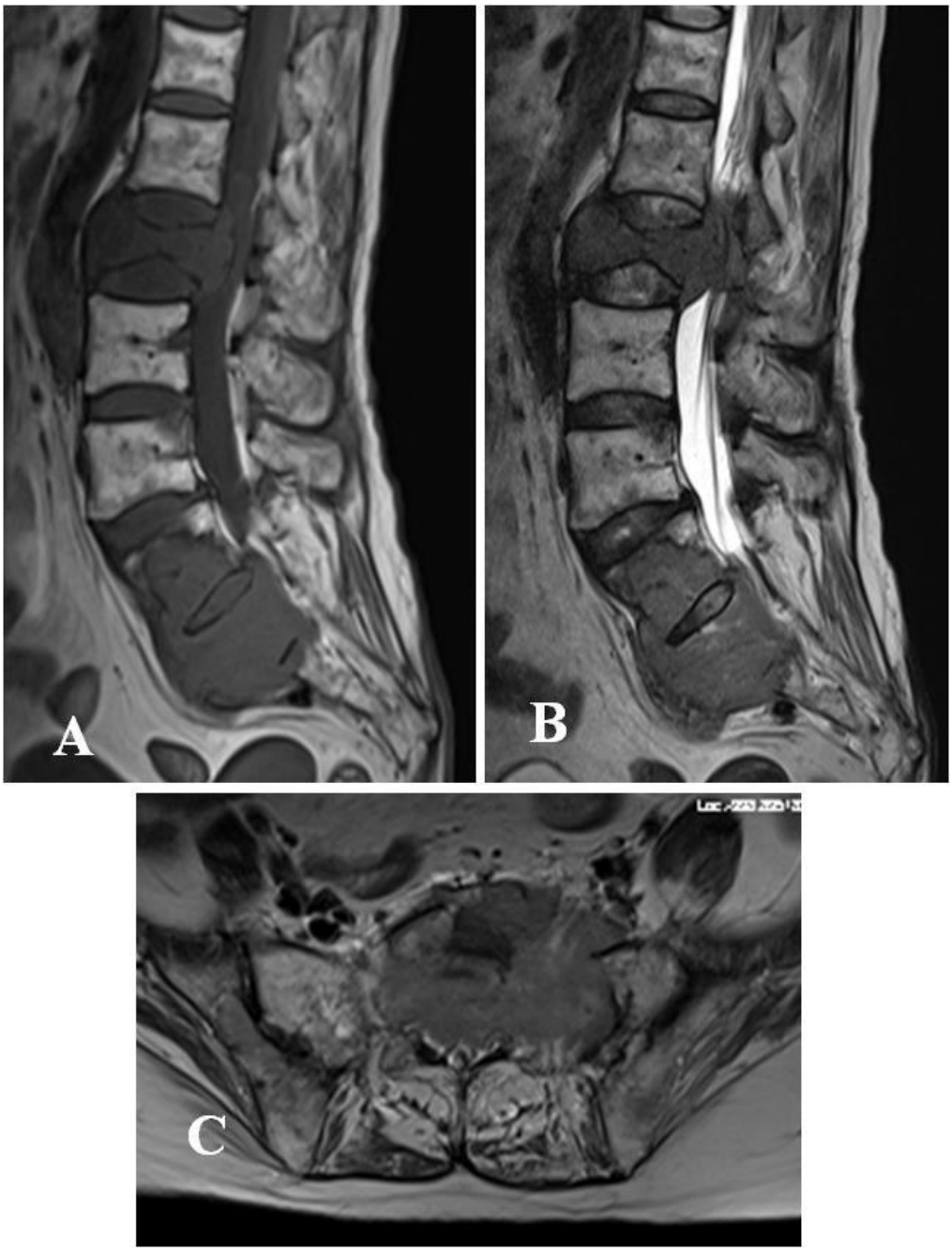


Figure 56. A 68-year-old male patient with low back pain due to multiple myeloma. Sagittal (A) T1 and (B) T2 weighted MRI of LS spine shows diffuse loss of normal signal in multiple vertebral bodies, with one vertebra showing reduction in the height, causing epidural hematoma compressing the cauda equina. The intervertebral discs appear normal. (C) Axial T2 weighted MRI at L5-S1 level shows epidural hematoma causing near total spinal canal stenosis.

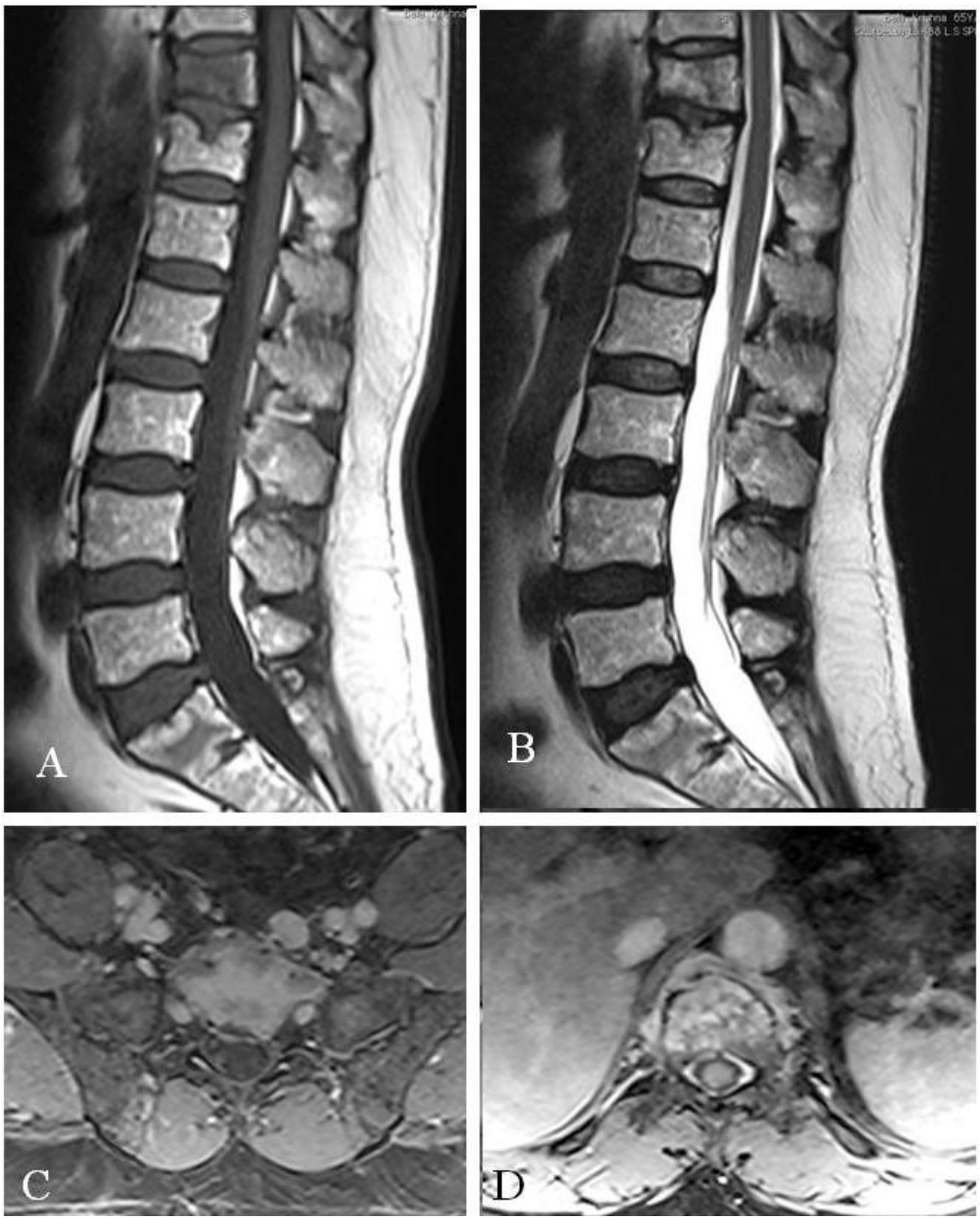


Figure 57. A case of spinal metastasis from prostate carcinoma. Sagittal (A) T1 and (B) T2 weighted MRI of LS spine shows T1 hypointense & T2 heterogeneously hyperintense lesion in the D11 and S1 vertebral bodies. Axial (C) and (D) post contrast T1 weighted fat saturated MR sequence shows diffuse contrast enhancement. There is associated pre & para-vertebral enhancing soft tissue component.

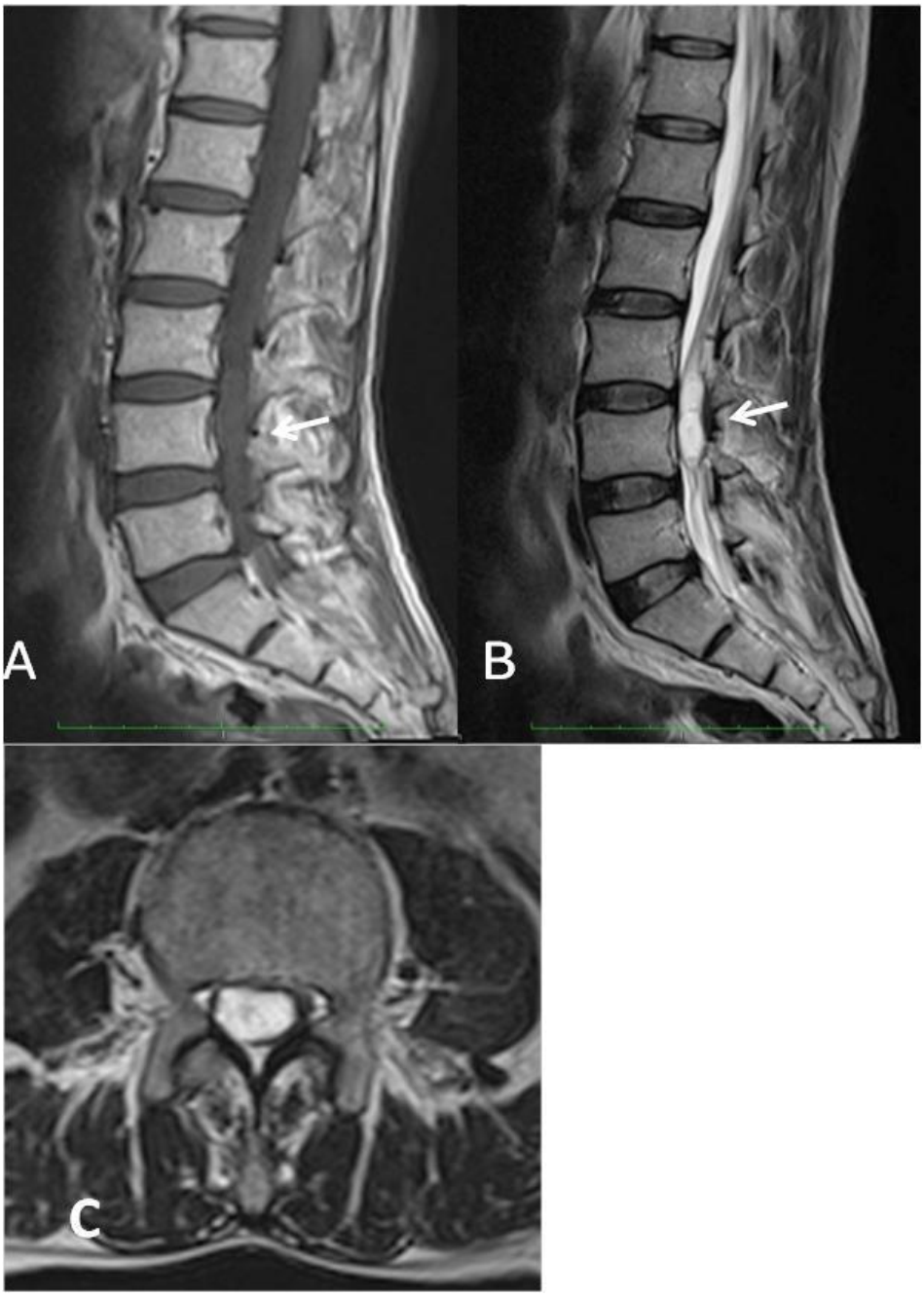


Figure 58. Sagittal (A) T1 and (B) T2 weighted MRI of LS spine shows longitudinally oriented well-defined CSF signal intensity cystic lesion at L3 and L4 vertebral levels (white arrows). (C) Axial T2 weighted MR shows intradural extramedullary location of the lesion obliterating the thecal sac and causing mass effect in the form of displacement/ compression on cauda equina roots.

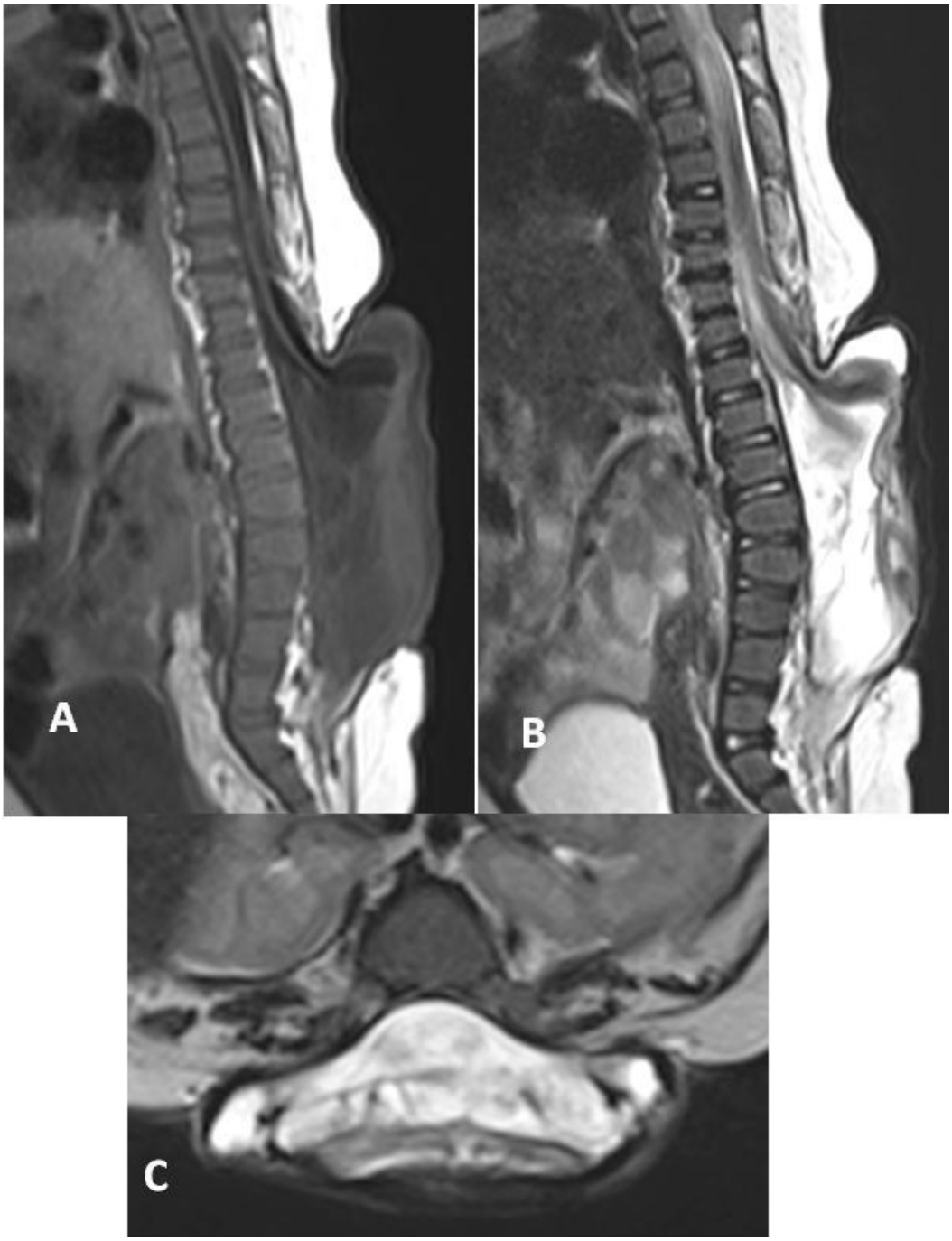


Figure 59 A case of thoraco-lumbosacral spinal dysraphism. Sagittal (A) T1, (B) T2 and (C) axial T2 weighted MRI showing a large myelomeningocele and tethered cord in a 6 year old patient. There is absence of posterior elements spinal elements from D11 to L5 vertebral levels.

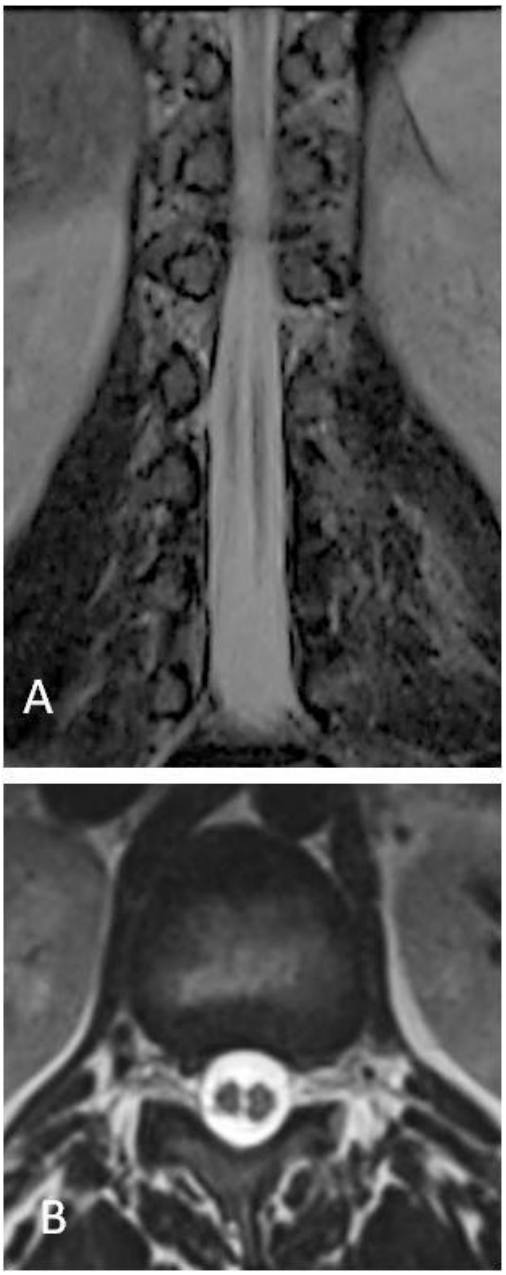


Figure 60. A case of type-II diastematomyelia.(A) Coronal STIR and (B) axial T2 weighted MRI of dorsal spine showing midline longitudinal cleft of distal dorsal spinal cord at T12 vertebral level.

DISCUSSION

The study included a total of 106 patients. More than 40% of patients (n = 43; 40.57%) were in the age group of 41 to 60 years. There were 29 patients in the age group of 21 to 40 years (27.36%), followed by age group of > 60 years (n = 21; 19.8%) and least patients were in the age group of <20 years (n = 13; 12.26%).

A cross-sectional observational study by Freburger et al in 723 patients with low-back pain in patients aged 21 years or more also reported similar findings. In their study among general population, 49.4% of patients with low back pain were in age group 45-64 years. Another 18.3% of patients were in of 35 to 44 years age group. Patients with age group 21 to 34 years constituted to <11% of patients with low back pain. They also observed that low back pain was more prevalent in the age group of 55 to 64 years (28.9%) followed by the age groups of 45 to 54 years (13.5%) and 65 years or more (12.3%)[65].

Although our study was not powered to determine the prevalence of low-back, nonetheless our results also indicate that low back pain is probably more common in the age group of 41 to 60 years.

In our study we observed a slight male preponderance with 58% of patients being males (n = 62).

Our results are in contrast to the gender distribution observed by other studies. Globally, Hoy et al performed a systematic review of global prevalence of low back pain that included 165 studies with 966 age or gender-specific prevalence estimates covering 54 countries. They observed higher prevalence of low back pain in females (females> males)[1]. A similar observation was made by Freburger et al who observed that more females (62.2%) complained of low-back pain compared to males[65]. A study conducted by Ahmad and Rather in North India in 210 patients with low back pain also had a female preponderance[66]. The difference in gender distribution in our study is probably due to the fact that we have included patients who underwent MRI for low back pain. Not all patients with low back pain undergo MRI, and this may have resulted in more males in our study.

Nearly half of the patients (n = 52, 49.02 %) in our study had bilateral radiculopathy, radiculopathy on right side (n=20, 18.87%) and lastly left sided radiculopathy in (n = 12, 11.32%). Radiculopathy was seen in 55 of 87 patients with degenerative changes (63.2%). Similarly radiculopathy was seen in more than 2/3rd of patients with Pott's spine (11 of 16 patients; 68.75%) and 7 of 8 patients with metastasis (87.5%). All the patients with radiculopathy had nerve root impingement/compression on MRI.

Degenerative changes were seen in more than 80% of patients (n = 87), followed by infective (n = 20; 16.26%) and neoplastic (n = 11; 8.94%) etiologies. Degenerative changes were considered primary cause for low back pain in 65 patients. Four patients had inflammatory and congenital causes for low back pain.

Arachnoid cyst was seen in two patients. There were 22 patients with concomitant diagnosis, which were considered as primary cause for low back.

Our study results are similar to study conducted in south India by Gopalakrishnan et al. In their study of 200 patients they observed that majority of the patients with low back pain had degenerative changes (approximately 80%)[18]. There was however lower incidence of infective and neoplastic lesions in their study and constituted only about 3.5% and 2% respectively.

Degenerative Changes

Degenerative changes were observed in 87 patients in our study. Among degenerative changes, degenerative disc changes were the most common abnormality seen in > 70% of patients (n = 76; 71.7%) followed by endplate changes (n = 63; 59.4%), vertebral changes (n = 58; 54.7%) joint and ligament changes (n = 44; 41.5%).

Degenerative Disc Changes

Degenerative disc changes were seen at multiple levels. A total of 186 disc levels with 302 degenerative disc conditions were seen in 87 patients. L4-L5 was the most commonly involved spinal level in our study (n = 71, 66.98%) followed by L5-S1 (n = 50, 47.17%) and L3-L4 (n = 44, 41.51%). Other levels T12-L1 (n = 10;

9.43%), L1-L2 (n = 5; 4.72%) and L2-L3 (n = 7; 6.60%), constituted remaining 11% of spinal level involvement.

Among disc changes, disc bulges (Figure 44) were most commonly seen in 132 discs (43.7%), followed by disc protrusion (68 discs; 22.52%), annular fissure/tears (40 discs; 13.25%), disc extrusion (37 discs; 12.25%) and disc sequestration (25 discs; 8.28%). The most commonly affected discs were L4-5 followed by L5-S1 and L3-4. L1-2 and L2-3 were least commonly affected discs (Table 9). L4-5 was the commonest location for disc bulge (40.15%), disc protrusion (45.59%), disc extrusion (56.76%), disc sequestration (64%) (Figure 51) and annular fissure/tears (42.5%) followed by L5-S1 and L3-4 levels.

A study by Verma et al in North India in 232 patients has shown findings comparable to that observed in our study. Although the age group in their study was 20 to 40 years, the results assume significance considering the similar findings. In their study multiple level disc involvement was seen in about 60% of patients. L4-5 was the commonly involved disc in 79.3% of patients followed by L5-S1 (68.9%) and L3-4 levels (32.8%). Additionally, L1-2 and L2-3 were least commonly involved. Disc bulge, protrusion, extrusion, and sequestration were most common at L4-5 level followed by L5-S1 and L3-4 level, which was observed in our study as well[67].

Gopalakrishnan et al also reported similar disc degenerative changes in their study. They observed that disc bulges constituted majority of disc degenerative

changes (n = 79%) followed by disc protrusion (14.4%), disc extrusion (5.8%) and lastly disc sequestration (seen in only three discs, two at L4-5 and one at L5-S1). L4-5 was the commonest location for disc bulge (39.2%), disc protrusion (42.1%), disc extrusion (45.45%) and disc sequestration (66.67%)[18]. Overall, most commonly involved disc was L4-5 (40.14%) followed by L5-S1 (26.7%) and L3-4 (21.4%). L1-2 and L2-3 levels constituted for remaining 10% of disc involvement[18].

In our study, central disc protrusions (Figure 45) were seen in >40% of involved discs (n = 28, 41.18%) followed by right paracentral disc protrusion (n = 25, 36.76%) (Figure 50) and lastly left paracentral disc protrusion (n = 15, 22.06%). Taken together paracentral disc herniations were the commonest type of disc herniation seen in our study (58.8% of disc protrusions).

Our results are similar to study by Gopalakrishnan et al. They observed that paracentral disc herniation was commonest type of disc herniation seen in about 65% of patients followed by central disc herniation (30%) and foraminal disc herniation (5%)[18]. A similar observation was also found in a study by Knop-Jergas et al who evaluated anatomic position of lumbar disc herniation in 80 patients. They observed that paracentral disc herniation was the commonest type (34.24%) followed by central disc herniation (26.02%) and intraforaminal (20.54%) locations[68].

In our study, annular fissure/tears were classified as concentric, radial or transverse. Concentric annular fissure/tears were the commonest type seen in more than 50% of involved discs (n = 23, 57.5%) followed by radial annular fissure/tear

(n = 14, 35%) and transverse annular tear (n = 3; 7.5%). Annular fissure/tears were most common at L4-5 level (42.5%) followed by L5-S1 levels (32.5%).

Literature suggests that L4-5 is the commonest level involved in annular tears followed by L5-S1[56,67], findings which are similar in our study. A study by Verma et al in 232 patients with low back pain of age group 20 to 40 years has shown that annular tear was most frequent in L4-L5 intervertebral disc (36.2 % cases), followed by L5-S1 (32.8%) and L3-L4 (8.6%)[67]. Suthar et al also reported similar findings for annular tears with L4-5 being the commonest level involved in annular fissure/tear (9.8%) followed by L5-S1 and L3-4 (prevalence of both was nearly same at 18-19%)[56].

MRI is considered to be highly sensitive for diagnosis of degenerative changes of spine in patients with low back pain. However, specificity of MRI is low, as degenerative changes of the spine are also seen in many asymptomatic individuals. However, current evidence suggests that disc bulges and protrusions have poor correlation to symptoms. Disc extrusions are almost always associated with symptoms and therefore may be considered as predictors of response to treatment[29].

Degenerative Vertebral Changes

In our study, vertebral changes were seen in the form of spondylolysis (n = 25; 23.58%) (Figure 47), spondylolisthesis (n =23; 21.70%) and osteophytes (n = 51;

48.11%). Most of the cases of spondylolisthesis were common in patients > 40 years or older (19 of 23 patients; 82.6%). Spondylolisthesis were graded from Grade I to grade IV. Grade I spondylolisthesis were seen in more than 60% of patients (n = 14, 60.9%) followed by grade II (n = 6, 26.1%) (Figure 48), grade III (n = 2; 8.7%) and grade IV was seen in one patient (4.3%). Most commonly affected levels in spondylolisthesis was L4/L5 (n = 16; 69.6%) followed by L5/S1 (n = 5; 21.7%) and lastly two cases were seen at L3/L4 (8.7%).

Our findings although are similar to study conducted by Denard et al who evaluated spondylolisthesis in 300 elderly symptomatic men to determine association of prevalent spondylolisthesis with back pain, neurogenic symptoms and functional limitations. They observed that spondylolisthesis was seen in 31% of patients and the prevalence of back pain was similar in patients with/without spondylolisthesis. There was significantly increased incidence of radiculopathy, paresthesias, and weakness in lower extremities in patients with spondylolisthesis. In their study, almost all of the patients (99%) had grade I spondylolisthesis. The commonest location of spondylolisthesis in their study was L4/5 (44.5%) followed by L5/S1 (23.9%) and L3/4 (2.17%). The authors concluded that although spondylolisthesis alone may not be associated with low back pain, it is commonly associated with radiculopathy, paresthesias, and weakness in lower extremities[69].

A cross-sectional prevalence study was conducted by Kalichman et al to assess prevalence rates of spondyolysis and spondylolisthesis in community-based population. They concluded that spondylolisthesis was more common in patients

aged >40 years and showed increasing prevalence with advancing age[70]. Spondylolisthesis is usually seen in individuals aged over 40 years and has shown an increasing prevalence with increasing age[70,71]. Lumbar spondyolysis was observed in 21 of 188 subjects (11.5%) and was commonest at L5 level. There was no difference in prevalence of spondylolysis among males and females. In the same study, spondylolisthesis was seen in 39 patients (20.7%) of population. The commonest location of spondylolisthesis was L4-5 and L5-S1[70].

Similar results have also been reported by the researchers of the Copenhagen Osteoarthritis Study, who evaluated prevalence and risk factors for degenerative lumbar spondylolisthesis. They observed that spondylolisthesis was significantly more common at L4-L5 and L5-S1 levels[71].

The relatively higher incidence of spondylolysis in our study could be attributed due to different population size and in part due to ethnic variability.

In our study osteophytes were seen in 51 patients. Out of this 37 patients were 40 years or older (72.55%). Our study findings are similar to findings reported by O'Neill et al who conducted a population based survey in 1180 individuals to determine the frequency and distribution of osteophytes. They found that vertebral osteophytes were seen in about 84% of men and 74% of women and showed increasing frequency with advancing age.[72]. It is however important to note that although there is a correlation between prevalence of osteophyte formation in patients

with low back pain, it may also be seen in asymptomatic individuals. Therefore, presence of osteophytes may not necessarily cause low back pain.

Degenerative Vertebral End Plate Changes

In our study vertebral end plate changes were seen in the form of Schmorl's nodes in 48 patients and Modic endplate changes seen in 51 patients. Type II Modic end plate changes (Figure 46) were commonest and seen in 37 patients (72.55%) followed by type I (n = 8, 15.69%) and type III Modic end plate changes (n = 6; 11.76%).

Out study results are comparable to findings reported by Fayad et al. In their study of 74 patients, half of the patients had Modic type I changes (n = 37) followed by Modic type I-2 changes (defined as mixture of Modic type I and type II changes but predominantly edema changes) in 25 patients (33.78%) and Modic type II changes in 12 patients (16.2%) of patients. There were no patients with type III change[8]. Yu et al in their study of end plate changes found type II changes to be commonest followed by type I and lastly type III, which was very rare and was seen in only one patient[58].

A large meta-analysis involving 58 studies had evaluated prevalence and association of Modic changes with non-specific low back pain. The authors found that Modic changes are common in patients with low back pain (mean average 43%) compared with asymptomatic patients (6%), highlighting the role of Modic changes in

low back pain. Additionally, the authors observed that type II changes were more common followed by type I changes, mixed types and least common was type III. Modic changes were also more common in lumbar spine. The authors opined that Modic changes are as a response to injury or may reflect genetic changes associated with response to injury and can contribute to low back pain[73].

In our study Schmorl's nodes were seen in 48 patients. Schmorl's nodes are herniation of nucleus pulposus through cartilaginous and bony end plate into body of adjacent vertebra.

Although Schmorl's nodes are incidental findings on MRI, a study by Hamanishi et al has shown that Schmorl's nodes were observed to be present significantly more in patients with low back pain (19%) compared with those without low back pain (9%). However, an exact relationship has not been confirmed[74].

Degenerative facet and ligament changes

In our study degenerative joint and ligament changes were seen in the form of ligamentum flavum hypertrophy (LFH) (Figure 49) and facetal arthropathy (FA). LFH was seen in 37 patients (34.9%) and FA was seen in 31 patients (29.25%). LFH and FA were almost always associated with degenerative changes (41 of 43 patients; 95.34%).

A study by Suthar et al has shown that facetal hypertrophy/arthropathy and ligamentum flavum hypertrophy is commonly encountered in patients with low back pain and having degenerative changes. Additionally they also observed that ligamentum flavum hypertrophy and facetal arthropathy were common at L4-5 disc level followed by L5-S1 and L3-4 disc levels, which are the common sites for degenerative disc, vertebral and end plate changes[56]. Our results are similar to the study by Munns et al who compared relation between low back pain and ligamentum flavum thickening in asymptomatic (n = 36) and symptomatic patients (n = 27). The authors reported significant correlation between ligamentum flavum thickening with advancing age, lower lumber level and chronic low back pain[75].

Lumbar canal stenosis

Lumbar spinal canal stenosis (defined as <10 mm of AP diameter) was seen in 60 patients in the present study. Degenerative changes were the commonest cause of lumbar canal stenosis (n = 43, 71.7%) followed by infections (n = 8, 13.3%), tumours (n = 6, 10%), arachnoid cyst (n = 2; 3.3%) and congenital lumbar spinal canal stenosis (n = 1; 1.7%). There were 104 discs involved in our study. L4-5 was the commonest level involved (n = 40; 38.5%) followed by L3-4 (n = 29; 27.9%) and L5-S1 (n = 23; 22.1%). Remaining levels constituted for only 12 discs.

Gopalakrishnan et al in their study using cut-off value of <8 mm at intervertebral disc level for lumbar spinal stenosis found prevalence of lumbar spinal canal stenosis to be 19%[18]. One possible reason for the lower prevalence in their study

could be due to use of stringent criteria for lumbar canal stenosis (<8 mm diameter). Similarly Kalichman et al evaluated the prevalence of lumbar spinal canal stenosis in general population and evaluated the association with low back pain. They observed that the relative spinal canal stenosis (defined as spinal canal diameter < 12 mm) was 22.5% and absolute spinal canal stenosis (defined as spinal canal diameter <10 mm) was 7.3%. Furthermore, there was a statistically significant association between absolute spinal canal stenosis with low back pain with three-fold higher risk of low back pain in these patients[76]. Our results are in agreement with their observation as more than 50% of patients in our study had lumbar spinal canal stenosis.

Infections of Spine

Infective causes of low back pain were seen in 20 patients in our study and included tubercular spondylitis and pyogenic spondylitis. Tubercular spondylitis was the commonest infective condition seen in 17 patients (85%) followed by pyogenic spondylitis (n = 3; 15%). In all these cases, final diagnosis was confirmed by demonstrating acid fast bacilli on Ziehl Neelson staining for tubercular spondylitis and culture/sensitivity for pyogenic spondylitis.

Pott's spine (Figure 52) was seen mostly in patients aged 40 years or older (11 of 16; 68.75%) (range 20 to 75 years) without any gender predilection.

T12-L1 was the most commonly involved spinal level in Pott's spine (n = 7, 43.75%) followed by L4-L5 and L3-L4 (n = 3, 18.75%), while L5-S1 constituted the remaining two cases. On MRI, eight patients had kyphotic deformity, 11 patients had vertebral body collapse/anterior wedge compression, nine patients had posterior spinal

element involvement (pedicle involvement), 10 patients showed partial collapse/destruction of disc, 12 patients had spondylodiscitis and 13 patients had epidural cold abscesses. On contrast study, heterogeneous enhancement of the lesions was seen in all the 16 cases.

Our results are similar to review by Narlawar et al in 33 patients who reported that the mean age group of patients with spinal tuberculosis ranged from 13 to 53 years, however, there was no significant gender predilection. Although the authors have evaluated tuberculosis of whole spine, they observed lower thoracic vertebral level and lumbar vertebrae together accounted for about 87% of all cases of tuberculosis, suggestive of predilection of Pott's spine to lower spine. All the patients presented with complaints of back pain[77]. A review of Pott's spine by Ansari et al also revealed lower thoracic and lumbar vertebrae as the commonest sites involved in Pott's spine[78]. Patients with Pott's spine usually complain of back pain and swelling in some cases. Characteristic MRI findings described in tuberculosis of spine are destruction of two adjacent vertebral bodies and opposing end plates, destruction of intervening disc, vertebral body edema, presence of pre-, paravertebral and epidural abscesses and heterogeneous enhancement of vertebral body[78,79], one or more of which were seen in all our patients. Involvement of posterior elements in spinal tuberculosis is not uncommon and pedicles are commonly involved in posterior element involvement[78].

There were three cases of pyogenic spondylitis in our study (n = 3, 15%) L3-L4 is the most commonly involved spinal level (n = 2) followed by L4-L5. Among

pyogenic spondylitis, *S. aureus* was reported in two cases and *E. coli* in one case. All the cases with tubercular spondylitis were treated with antitubercular treatment and pyogenic spondylitis were treated with appropriate antibiotics.

Sacroiliitis

In our study there were four cases of sacroiliitis (Figure 53). Three cases were bilateral and one case was unilateral (left side). MRI showed a high positive predictive value in the diagnosis of sacroiliitis in our study. Changes of sacroiliitis are seen earliest on MRI. Bone marrow edema along the sacroiliac joints was the most common feature and was seen in three patients. Subchondral edema and erosions were seen in two patients and sclerosis was seen in one patient.

Our study findings are in agreement with the observations made by Shankar et al in their evaluation of sacroiliitis in 29 patients showing abnormal MRI findings. MRI abnormality was seen in 21 bilateral joints and eight unilateral joints. There were no joint changes in control subjects, suggesting that MRI is indeed very sensitive and specific for evaluating sacroiliitis (sensitivity of 87.9% and a specificity of 100%). The MRI changes observed were bone marrow edema (89%), synovial enhancement (55%), subchondral edema (41%), erosions (51%) and sclerosis (28%)[80].

Neoplasms

There were 11 neoplasms noted in our study, 10 were malignant and one tumour was benign. Among the malignant conditions, most of them were metastasis, seen in eight patients. (age ranging from 41 to 65 years). The primary lesions were carcinoma prostate in four patients, carcinoma lung in three patients and carcinoma esophagus in one patient. All the lesions were extradural and located in lower thoracic and lumbar spine. There was one each case of multiple myeloma and sacral chordoma. There was a case of benign giant cell tumour.

Most of the patients with spinal metastases (Figure 57) present with non-specific back pain. A history of primary malignancy can go a long way in suspecting metastasis to spine. Lumbar spine is considered to account for about 20% of spinal metastasis[81]. Spinal metastases are commonest tumour involving the spine and are seen in up to 40% of cancer patients. The commonest tumours causing spinal metastases are breast (21%), lung (14%), prostate (8%), renal and gastrointestinal (5% each) and thyroid (3%)[82]. Carcinoma lung is considered as the leading cause for metastatic cord compression in men followed by carcinoma prostate. Spinal metastasis is common in carcinoma prostate and is seen in up to 20% of patients[83]. Carcinoma prostate metastases are reported in 1 to 12% of cases of spinal metastasis[84].

A study by Chen et al in 37 patients who evaluated vertebral metastasis showed that vertebral metastasis is common among the elderly (mean age > 60 years),

which is consistent with our study[85]. A study by Aydinli et al in 168 patients with lung cancer and vertebral metastasis observed that lumbar spine was the second most common location for vertebral metastasis and was seen in 32% of the cases (105 out of 328 patients). It is believed most of lung cancers spread through arterial dissemination and therefore known to occur predominantly in vertebral body due to its high vascularity[86].

Chordomas (Figure 55) are most commonly seen in the sacrococcygeal region (> 50 to 60%)[87]. A retrospective review of 33 patients with sacrococcygeal chordoma the mean duration of symptoms before diagnosis was about 2.3 years (range of 0.5 to 8 years). Back pain was the most common presenting complaint reported in about 85% of cases. Presence of concurrent paresthesias, bladder/bowel disturbance should direct the clinician to suspect sacrococcygeal chordoma[88].

Giant cell tumours (GCT) (Figure 54) involving the spine are rare and comprise <3% of all GCT.[89]. GCT is rarely seen in the lumbar spine. These neoplasms are more common in age group of 20 to 45 years without any sex predilection[90]. In our case the patient was a 50 year-old female who presented with low back pain.

Multiple myeloma (Figure 56) is a commonly encountered primary malignancy of bone and constitutes for approximately 10% of all hematologic malignancies. It is common in males during the age of 40 to 80 years.[91].

Congenital lesions

There were two cases of myelomeningocele, one case each of diastematomyelia (Figure 60) and arteriovenous malformation (AVM) of spinal cord in our study. There were two cases of arachnoid cysts seen in patients aged 49 and 55 years (Figure 58), both of which caused mass effect in the form of compression of cauda equina fibres. All these cases underwent surgery and final diagnosis was confirmed. Other findings seen in our study were LSTV-L (n = 8), LSTV-S (n = 13), Tarlov cyst (perineural cyst) (n = 16) and kyphosis/scoliosis (n = 8), all of which were seen in patients who had primary MRI diagnoses and were not considered as the primary pathology responsible for low back pain.

Myelomeningocele (Figure 59) and diastematomyelia belong to group of congenital disorders referred to as spinal dysraphism. Myelomeningocele comprises of more than 98% of all open spinal dysraphism. Myelomeningocele and myeloceles are associated with diastematomyelia in about 8 to 45% of cases. Diastematomyelia is the commonest form of failed midline integration of notochord[92].

Arachnoid cysts are commonly seen in lower thoracic spine (about 2/3rd) followed by lumbosacral (13%) and thoracolumbar spine (12%). These cysts have been reported to be rare cause of spinal cord compression following enlargement of cyst. Thoracolumbar and lumbar arachnoid cysts are usually seen in the fourth decade of life[93], which was consistent with findings from our study.

CONCLUSION

In this study of 106 patients, degenerative changes were the commonest cause for low back pain followed by infective and neoplastic etiologies. L4-L5 disc was the most commonly involved spinal level in our study followed by L5-S1 and L3-L4. MRI provides most precise visualization of all spinal elements and paraspinal soft tissues. Additionally, the ability of MRI to detect disc and vertebral signal changes has made it an investigation of choice for evaluation of low back pain.

Pott's spine was the commonest infection followed by pyogenic spondylitis. MRI helped in narrowing down differential diagnosis and helped in arriving to more accurate diagnosis.

MRI is the modality of choice for evaluation of spinal cord neoplasms as it can provide diagnosis or differential diagnosis in majority of cases as clinical findings are often insufficient to arrive at working diagnosis.

Our study underscores the importance of MRI in evaluation of low back pain. The ability of MRI to detect morphological abnormalities, extent of lesion and nerve root compression all help in complete evaluation of low back pain.

SUMMARY

The aim of the study was to evaluate the changes seen on MRI in patients with low backache due to various non−traumatic causes, to distinguish various causes of low back pain with level of spinal involvement and to evaluate the concordance between clinical diagnosis and MR imaging.

This descriptive observational study was carried out over a period of 18 months from January 2015 to June 2016 in 106 patients with low back pain who underwent MRI of the lower spine at Department of Radio-Diagnosis, R. L. Jalappa Hospital & Research Centre. MRI of spine was performed with 1.5 Tesla MRI scanner (Siemens® Magnetom Avanto®).

More than 40% of patients (n = 43; 40.57%) were in the age group of 41 to 60 years. There were 29 patients in the age group of 21 to 40 years (27.36%), followed by age group of > 60 years (n = 21; 19.8%). There was slight male preponderance in the study with 58% of patients being males (n = 62)

On MRI, degenerative changes were the commonest findings in more than 50% of patients (n = 87; 82.08%) followed by infective (n = 20; 18.87%) and neoplastic (n = 11; 10.38 %) etiologies. Four patients each had inflammatory and congenital etiologies (3.77%). Arachnoid cyst was seen in two patients (1.89%) A total of 65 patients had degenerative changes only and was therefore considered to be cause for low back pain. Remaining 22 patients had additional diagnosis of tuberculosis of spine (n = 7), metastases (n = 6), sacroiliitis (n = 4), arachnoid cyst (n = 2), arteriovenous malformation, multiple myeloma and sacral chordoma

(n = 1 each). In these patients degenerative changes were not considered as the primary cause for low back pain.

Among degenerative changes, degenerative disc diseases were the commonest cause for low back pain in > 70% of patients (n = 76; 71.7%) followed by endplate changes (n = 63; 59.4%), vertebral changes (n = 58; 54.7%) and joint and ligament changes (n = 44; 41.5%). Degenerative disc diseases were seen at multiple levels. A total of 302 degenerative disc conditions were seen in 87 patients. Among them disc bulges were most common, seen in 132 discs (43.7%), followed by disc protrusion (68 discs; 22.52%), annular fissure/tears (40 discs; 13.25%), disc extrusion (37 discs; 12.25%) and disc sequestration (25 discs; 8.28%). L4-5 was the commonest location for disc bulge (40.15%), disc protrusion (45.59%), disc extrusion (56.76%), disc sequestration (64%) and annular fissure/tears (42.5%) followed by L5-S1 and L3-4 levels. L1-2 and L2-3 were least commonly affected discs.

Symmetrical disc bulges constituted about 2/3^{rds} of total disc bulges (n = 89, 67.42%) followed by asymmetrical disc bulges (n = 43, 32.58%). Central disc protrusions were seen in >40% of involved discs (n = 28, 41.79%) followed by right paracentral disc protrusions (n = 24, 35.82%) and left paracentral disc protrusions (n = 15, 22.39%). Concentric annular fissure/tears were the commonest type seen in more than 50% of involved discs (n = 23, 57.5%) followed by radial annular fissure/tear (n = 14, 35%) and transverse annular tear (n = 3; 7.5%).

Degenerative vertebral changes were seen in form of spondylolysis, spondylolisthesis and osteophytes. Spondylolisthesis was seen in 23 patients and were

graded from grades I to grade IV. Grade I spondylolisthesis were seen in more than 60% of patients (n = 14, 60.9%) followed by grade II spondylolisthesis (n = 6, 26.1%), grade III spondylolisthesis (n = 2; 8.7%) and grade IV spondylolisthesis in one patient (4.3%). Modic endplate changes were seen in 50 patients. Type II Modic end plate changes were commonest (n = 37; 72%) followed by type I (n = 8, 16%) and type III Modic end plate changes (n = 6; 12%). LFH was seen in 37 patients (34.9%) and facetal arthropathy was seen in 31 patients (29.25%).

Infective causes of low back pain were seen in 20 patients and included tubercular spondylitis and pyogenic spondylitis. Tubercular spondylitis was the commonest infective condition seen in 17 patients (85%) followed by pyogenic spondylitis (n = 3; 15%). Pott's spine was seen mostly in patients aged 40 years or older (11 of 16; 68.75%) (range 20 to 75 years) without any gender predilection. T12-L1 was the most commonly involved spinal level in Pott's spine (n = 7, 43.75%) followed by L4-L5 and L3-L4 (n = 3, 18.75%), while L5-S1 constituted the remaining two cases. Eight patients had kyphotic deformity, 11 patients had vertebral body collapse/anterior wedge compression, nine patients had posterior spinal element involvement (pedicle involvement), 10 patients showed partial collapse/destruction of disc, 12 patients had spondylodiscitis and 13 patients had epidural cold abscesses. On contrast study, heterogeneous enhancement of the lesions was seen in all the 17 cases. Among pyogenic spondylitis, *S. aureus* was reported in two cases and *E. coli* in one case.

In our study there were four cases of sacroiliitis (bilateral in three patients and unilateral in one patient on left side). Bone marrow edema along the sacroiliac joints

was the most common feature and was seen in three patients. Subchondral edema and erosions were seen in two patients and sclerosis was seen in one patient. MRI changes are considered as the earliest signs for sacroiliitis.

There were 11 tumours noted in our study, 10 were malignant and one was benign. Among malignancies, most of them were metastasis (n = 8). There was one each case each of multiple myeloma and sacral chordoma. There was a case of benign giant cell tumour (GCT). All the lesions were extradural and located in lower thoracic and lumbar spine.

Among congenital conditions seen in our study there were two cases of myelomeningocele, one case each of diastematomyelia and arteriovenous malformation (AVM) of spinal cord. There were two cases of arachnoid cysts, both of which caused mass effect in the form of compression on cauda equina fibres.

Our study underscores the importance of MRI in evaluation of low back pain. The ability of MRI to detect morphological abnormalities, extent of lesion and nerve root compression all help in complete evaluation of low back pain.

BIBLIOGRAPHY

1 Hoy D, Bain C, Williams G, March L, Brooks P, Blyth F, et al. A systematic review of the global prevalence of low back pain. Arthritis Rheum 2012;64:2028-37.

2 Biering-Sørensen F. A prospective study of low back pain in a general population. I. Occurrence, recurrence and aetiology. Scand J Rehabil Med 1983;15:71-9.

3 Arya RK. Low back pain - signs, symptoms, and management. J Indian Acad Clin Med 2014;15:30-41.

4 Klekot D, Zimny A, Czapiga B, Sąsiadek M. Isolated septic facet joint arthritis as a rare cause of acute and chronic low back pain – a case report and literature review. Pol J Radiol 2012;77:72–6.

5 Bindra S, Sinha AGK, Benjamin AI. Epidemiology of low back pain in Indian population: A review. Intl J Basic Appl Med Sci 2015:166-79.

6 Watts J, Box GA, Galvin A, Van Tonder F, Trost N, Sutherland T. Magnetic resonance imaging of intramedullary spinal cord lesions: A pictorial review. J Med Imaging Radiat Oncol 2014;58:569-81.

7 Do-Dai DD, Brooks MK, Goldkamp A, Erbay S, Bhadelia RA. Magnetic resonance imaging of intramedullary spinal cord lesions: a pictorial review. Curr Probl Diagn Radiol 2010;39:160-85.

8 Fayad F, Lefevre-Colau MM, Rannou F, Quintero N, Nys A, Macé Y. Relation of inflammatory modic changes to intradiscal steroid injection outcome in chronic LBP. Eur Spine J 2007;16:925-31.

9 Arnbak B, Jensen TS, Manniche C, Zejden A, Egund N, Jurik AG. Spondyloarthritis-related and degenerative MRI changes in the axial skeleton-an

inter- and intra-observer agreement study. BMC Musculoskelet Disord 2013;14:274.

10 Blackham K, Sunshine JL. Spinal cord. In: Haaga JR, Dogra VS, Forsting M, Gilkeson RC, Ha HK, Sundaram M, editors. CT and MRI of the whole body. Chapter 17. 5th edition. Mosby Elsevier. 2003:Pg 733-54.

11 Kirsch C. The vertebral column and spinal cord. In: Butler P, Mitchell AWM, Ellis H, editors. Applied radiological anatomy for medical students. Chapter 11. 1st edition. Cambridge University Press. 2008:Pg 105-12.

12 Central nervous system. In: Butler P, Mitchell A, Healy JC, editors. Applied radiological anatomy. 2nd edition. Cambridge University Press. 2012:Pg: 82-8.

13 Spine. In: Moeller TB, Reif E, editors. Pocket Atlas of Sectional Anatomy. Computed Tomography and Magnetic Resonance Imaging. Volume III: Spine, Extremities, Joints. Thieme. 2007:Pg 263-315.

14 Humphreys SC, Eck JC, Hodges SD. Neuroimaging in low back pain. Am Fam Physician 2002;65:2299-306.

15 McCulloch ML. MRI Historical Background. Methodist Debakey Cardiovasc J 2009;5:13-4.

16 Hoeffner EG, Mukherji SK, Srinivasan A, Quint DJ. Neuroradiology Back to the Future: Spine Imaging. AJNR Am J Neuroradiol 2012;33:999–1006.

17 Nakamura T, Yabe Y, Horiuchi Y, Takayama S. Magnetic resonance myelography in brachial plexus injury. J Bone Joint Surg Br 1997;79:764-9.

18 Gopalakrishnan N, Nadhamuni K, Karthikeyan T. Categorization of pathology causing low back pain using magnetic resonance imaging (MRI). J Clin Diagnos Res 2015;9:TC17-20.

19. Biluts HT, Munie M, Abebe. Review of lumbar disc diseases at Tikur Anbessa Hospital. Ethiop Med J 2012; 50: 57-65.

20 Endean A, Palmer KT, Coggon D. Potential of magnetic resonance imaging findings to refine case definition for mechanical LBP in epidemiological studies: a systematic review. Spine 1976;36: 160-9.

21 Thielen TV, van den Hauwe L, Van Goethem JW, Parizel PM. Imaging techniques and anatomy. In: Adam A, Dixon AK, Gilard JH, Schaefer-Prokop CM editors. Grainger & Allison's Diagnostic Radiology. 5th edition. Chapter 54. Churchill Livingstone Elsvier.2007 Pg: 1279-1294.e.1.

22 Diseases of the Spinal cord. Karper DL, Anthony SF, Longo DL, Braunwald E, Hauser SL, Jameson L. editors. In Harrison's principles of internal Medicine. Vol II, 16th Edition. MC Graw Hill companies, USA; 2004: 2440-1

23 Wheeler SG, Wipf JE, Staiger TO, Deyo RA, Atlas SJ, Park L. Evaluation of low back pain in adults. [Internet]. cited 2016 aug 2 [Updated 2016 Apr 27]. Available at: http://www.uptodate.com/contents/evaluation-of-low-back-pain-in-adults?topicKey=PC%2F7782&elapsedTimeMs=4&view=print&displayedView=full#

24. Joshi M, Yadav S. Lumbosacral agenesis. Ind J Radiol Imag 2005; 15(2): 251-4.

25 Middleton K. Fish DE. Lumbar spondylosis: clinical presentation and treatment approaches. Curr Rev Musculoskelet Med 2009;2:94–104.

26 Modic MT, Ross JS. Lumbar degenerative disk disease. Radiology 2007;245:43-61.

27 Fardon DF, Williams AL, Dohring EJ, Murtagh FR, Gabriel Rothman SL, Sze GK. Lumbar disc nomenclature: version 2.0: Recommendations of the combined task

forces of the North American Spine Society, the American Society of Spine Radiology and the American Society of Neuroradiology. Spine J 2014;14:2525-45.

28 Jenkins H. Classification of low back pain. Australas Chiropr Osteopathy 2002;10:91–7.

29 Roudsari B, Jarvik JG. Lumbar spine MRI for low back pain: Indications and yield. AJR Am J Roentgenol 2010;195:550–9.

30 Lee KY. Comparison of pyogenic spondylitis and tuberculous spondylitis. Asian Spine J 2014;8:216-23.

31 Galhotra RD, Jain T, Sandhu P, Galhotra V. Utility of magnetic resonance imaging in the differential diagnosis of tubercular and pyogenic spondylodiscitis. J Nat Sci Biol Med 2015;6:388-93.

32 LaBerge JM, Brant-Zawadzki M. Evaluation of Pott's disease with computed tomography. Neuroradiology 1984;26:429-34.

33 de Roos A, van Persijn van Meerten EL, Bloem JL, Bluemm RG. MRI of tuberculous spondylitis. AJR Am J Roentgenol 1986;147:79-82.

34 Numaguchi Y, Rigamonti D, Rothman MI, Sato S, Mihara F, Sadato N. Spinal epidural abscess: evaluation with gadolinium-enhanced MR imaging. Radiographics 1993;13:545-59.

35 Ngo TP, Dufton J, Stern PJ, Islam O. Myxopapillary ependymoma as a cause of back pain in a young male - A case report. J Can Chiropr Assoc 2013;57:150-5.

36 Mut M, Schiff D, Shaffrey ME. Metastasis to nervous system: spinal epidural and intramedullary metastases. J Neurooncol 2005;75:43-56.

37 Venkitaraman R, Sohaib SA, Barbachano Y, Parker CC, Khoo V, Huddart RA, et al. Detection of occult spinal cord compression with magnetic resonance imaging of the spine. Clin Oncol (R Coll Radiol) 2007;19:528-31.

38 Asazuma T, Toyama Y, Watanabe M, Suzuki N, Fujimura Y, Hirabayashi K. Clinical features associated with recurrence of tumours of the spinal cord and cauda equina. Spinal Cord 2003;41:85-9.

39 Mc PC, Post KD, Stein BM. Intradural extramedullary tumors in adults. Neurosurg Clin N Am 1990;1:591-608.

40 Rothwell CI, Jaspan T, Worthington BS, Holland IM. Gadolinium-enhanced magnetic resonance imaging of spinal tumours. Br J Radiol 1989;62:1067-74.

41 Sandalcioglu IE, Hunold A, Müller O, Bassiouni H, Stolke D, Asgari S. Spinal meningiomas: critical review of 131 surgically treated patients. Eur Spine J 2008;17:1035-41.

42 Matsumoto S, Hasuo K, Uchino A, Mizushima A, Furukawa T, Matsuura Y, et al. MRI of intradural-extramedullary spinal neurinomas and meningiomas. Clin Imaging 1993;17:46-52.

43 Gezen F, Kahraman S, Canakci Z, Bedük A. Review of 36 cases of spinal cord meningioma. Spine (Phila Pa 1976) 2000;25:727-31.

44 Munk PL, Helms CA, Holt RG, Johnston J, Steinbach L, Neumann C. MR imaging of aneurysmal bone cysts. AJR Am J Roentgenol 1989;153:99-101.

45 Kochan JP1, Quencer RM. Imaging of cystic and cavitary lesions of the spinal cord and canal. The value of MR and intraoperative sonography. Radiol Clin North Am 1991;29:867-911.

46 Sharma M, Velho V, Mally R, Khan SW. Symptomatic lumbosacral perineural cysts: A report of three cases and review of literature. Asian J Neurosurg 2015;10:222–5.

47 Kaspiris A, Grivas TB, Weiss HR, Turnbull D. Surgical and conservative treatment of patients with congenital scoliosis: α search for long-term results. Scoliosis 2011;6:12.

48 Wazir S. Sacralisation of lumbar vertebrae: A case report. Int J Anat Res 2014;2:386-89.

49 Konin GP, Walz DM. Lumbosacral transitional vertebrae: classification, imaging findings, and clinical relevance. AJNR Am J Neuroradiol 2010;31:1778-86.

50 Verma S, Gupta P, Munshi A, Goyal P, Verma S, Sardana V. A retrospective analysis of Magnetic Resonance Imaging findings in 20-40 year old patients with low back pain. Experience at a semi urban tertiary healthcare centre in Northern India. Internet J Spine Surg 2010;6:1-7.

51 Sreedhar CM, Sree Ram MN, Alam A, Indrajit IK, Shanmuganandan K. Sacroiliitis in routine MRI for low back ache. Indian J Radiol Imaging 2006;16:643-9.

52 Suwaid MA, Ismail A, Idris MM. Spectrum of spinal abnormalities on magnetic resonance imaging of patients with clinical suspicion of spinal lesions in Kano, Nigeria. J West Afr Coll Surg 2014;4:27–38.

53 Samini F, Gharedaghi M, Khajavi M, Samini M. The etiologies of low back pain in patients with lumbar disk herniation. Iran Red Crescent Med J 2014;16:e15670.

54 Bechara BP, Agarwal V, Boardman J, Perera S, Weiner DK, Vo N, et al. Correlation of pain with objective quantification of magnetic resonance images in older adults with chronic low back pain. Spine (Phila Pa 1976) 2014;39:469-75.

55 Brinjikji W, Luetmer PH, Comstock B, Bresnahan BW, Chen LE, Deyo RA, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. AJNR Am J Neuroradiol 2015;36:811-6.

56 Suthar P, Patel R, Mehta C, Patel N. MRI evaluation of lumbar disc degenerative disease. J Clin Diagnos Res 2015;9:TC04-9.

57 Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. Radiology 1998;209:661-6.

58 Yu LP, Qian WW, Yin GY, Ren YX, Hu ZY. MRI assessment of lumbar intervertebral disc degeneration with lumbar degenerative disease using the Pfirrmann grading systems. PLoS One 2012;7:e48074.

59 Han JS, Benson JE, Yoon YS. Magnetic resonance imaging in the spinal column and craniovertebral junction. Radiol Clin North Am 1984;22:805-27.

60 Smoker WR, Godersky JC, Knutzon RK, Keyes WD, Norman D, Bergman W. The role of MR imaging in evaluating metastatic spinal disease. AJR Am J Roentgenol 1987;149:1241-8.

61 Souweidane MM, Benjamin V. Spinal cord meningiomas. Neurosurg Clin N Am 1994;5:283-91.

62 Mamisch N, Brumann M, Hodler J, Held U, Brunner F, Steurer J; Lumbar Spinal Stenosis Outcome Study Working Group Zurich. Radiologic criteria for the diagnosis of spinal stenosis: results of a Delphi survey. Radiology 2012;264:174-9.

63 Steurer J, Roner S, Gnannt R, Hodler J; LumbSten Research Collaboration. Quantitative radiologic criteria for the diagnosis of lumbar spinal stenosis: a systematic literature review. BMC Musculoskelet Disord 2011;12:175.

64 Jönsson B, Annertz M, Sjöberg C, Strömqvist B. A prospective and consecutive study of surgically treated lumbar spinal stenosis. Part I: Clinical features related to radiographic findings. Spine (Phila Pa 1976) 1997;22:2932-7.

65 Freburger JK, Holmes GM, Agans RP, Jackman AM, Darter JD, Wallace AS, et al. The rising prevalence of chronic low back pain. Arch Intern Med 2009 9;169:251-8.

66 Ahmad SJ, Rather AH. Clinical pattern of chronic low backache. A prospective study of 210 cases at a multidisciplinary hospital. JK Practitioner 2014;19:21-27.

67 Verma S, Gupta P, Munshi A, Goyal P, Verma S, Sardana V. Retrospective analysis of magnetic resonance imaging findings in 20 -40 year old patients with low back pain. Experience at a semi urban tertiary healthcare centre in northern India. Internet J Spine Surg 2010;6:1-7.

68 Knop-Jergas BM, Zucherman JF, Hsu KY, DeLong B. Anatomic position of a herniated nucleus pulposus predicts the outcome of lumbar discectomy. J Spinal Disord 1996;9:246-50.

69 Denard PJ, Holton KF, Miller J, Fink HA, Kado DM, Marshall LM, et al. Back pain, neurogenic symptoms, and physical function in relation to spondylolisthesis among elderly men. Spine J 2010;10:865-73.

70 Kalichman L1, Kim DH, Li L, Guermazi A, Berkin V, Hunter DJ. Spondylolysis and spondylolisthesis: prevalence and association with low back pain in the adult community-based population. Spine (Phila Pa 1976) 2009;34:199-205.

71 Jacobsen S, Sonne-Holm S, Rovsing H, Monrad H, Gebuhr P. Degenerative lumbar spondylolisthesis: an epidemiological perspective: the Copenhagen Osteoarthritis Study. Spine (Phila Pa 1976) 2007;32:120-5.

72 O'Neill TW, McCloskey EV, Kanis JA, Bhalla AK, Reeve J, Reid DM, et al. The distribution, determinants, and clinical correlates of vertebral osteophytosis: a population based survey. J Rheumatol 1999;26:842-8.

73 Jensen TS, Karppinen J, Sorensen JS, Niinimäki J, Leboeuf-Yde C. Vertebral endplate signal changes (Modic change): a systematic literature review of prevalence and association with non-specific low back pain. Eur Spine J 2008;17:1407-22.

74 Kyere KA, Than KD, Wang AC, Rahman SU, Valdivia–Valdivia JM, La Marca F, et al. Schmorl's nodes. Eur Spine J 2012; 21: 2115–21.

75 Munns JJ, Lee JY, Espinoza Orías AA, Takatori R, Andersson GB, An HS, et al. Ligamentum flavum hypertrophy in asymptomatic and chronic low back pain subjects. PLoS One 2015;10:e0128321.

76 Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, et al. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J 2009;9:545-50.

77 Narlawar RS, Shah JR, Pimple MK, Patkar DP, Patankar T, Castillo M. Isolated tuberculosis of posterior elements of spine: magnetic resonance imaging findings in 33 patients. Spine (Phila Pa 1976) 2002;27:275-81.

78 Ansari S, Amanullah MF, Ahmad K, Rauniyar RK. Pott's Spine: Diagnostic imaging modalities and technology advancements. N Am J Med Sci 2013;5:404-11.

79 Garg RK, Somvanshi DS. Spinal tuberculosis: A review. J Spinal Cord Med 2011;34: 440–54.

80 Shankar S, Abhisheka K, Anil Kumar AVS. Evaluation of magnetic resonance imaging and radionuclide bone scan in early spondyloarthropathy. Indian J Rheumatol 2009;4:142-8.

81 Quraishi NA, Esler C. Metastatic spinal cord compression. BMJ 2011;342:d2402.

82 Georgy BA. Metastatic spinal lesions: state-of-the-art treatment options and future trends. AJNR Am J Neuroradiol 2008;29:1605-11.

83 Berruti A, Dogliotti L, Bitossi R, Fasolis G, Gorzegno G, Bellina M, et al. Incidence of skeletal complications in patients with bone metastatic prostate cancer and hormone refractory disease: predictive role of bone resorption and formation markers evaluated at baseline. J Urol 2000;164:1248-53.

84 Tazi H, Manunta A, Rodriguez A, Patard JJ, Lobel B, Guillé F. Spinal cord compression in metastatic prostate cancer. Eur Urol 2003;44:527-32.

85 Chen YJ, Chang GC, Chen HT, Yang TY, Kuo BI, Hsu HC, et al. Surgical results of metastatic spinal cord compression secondary to non-small cell lung cancer. Spine (Phila Pa 1976) 2007;32:E413-8.

86 Aydinli U, Ozturk C, Bayram S, Sarihan S, Evrensel T, Yilmaz HS. Evaluation of lung cancer metastases to the spine. Acta Orthop Belg 2006;72:592-7.

87 Farsad K, Kattapuram SV, Sacknoff R, Ono J, Nielsen GP. Sacral chordoma. Radiographics 2009;29:1525-30.

88 Jeys L, Gibbins R, Evans G, Grimer R. Sacral chordoma: a diagnosis not to be sat on? Int Orthop 2008;32:269-72.

89 Kwon JW, Chung HW, Cho EY, Hong SH, Choi SH, Yoon YC, et al. MRI findings of gaint cell tumours of the spine. AJR Am J Roentgenol 2007;189:246–50.

90 Martin C, McCarthy EF. Gaint cell tumor of the sacrum and spine: Series of 23 cases and a review of the literature. Iowa Orthop J 2010;30:69-75.

91 Hanrahan CJ, Christensen CR, Crim JR. Current concepts in the evaluation of multiple myeloma with MR imaging and FDG PET/CT. Radiographics 2010;30:127-42.

92 Rossi A, Cama A, Piatelli G, Ravegnani M, Biancheri R, Tortori-Donati P. Spinal dysraphism: MR imaging rationale. J Neuroradiol 2004;31:3-24.

93 Liu JK, Cole CD, Kan P, Schmidt MH. Spinal extradural arachnoid cysts: clinical, radiological, and surgical features. Neurosurg Focus 2007;22:E6.

ANNEXURE-I

PROFORMA

Demographic details:

Name:

Age:

Sex: ☐Male ☐Female

Clinical Data:

1. Detailed history and local examination.

2. Straight leg raise test (SLRT)

3. Sensory and motor evaluation the lower limbs

Radiculopathy: Right ☐ Left ☐ Bilateral ☐

MRI findings:

Level of involvement	Disc changes				
	Bulge	Protrusion	Extrusion	Sequestration	Tear/fissure
T12-L1					
L1-2					
L2-3					
L3-4					
L4-5					
L5-S1					

Level of involvement	Vertebral changes		
	osteophytes	Listhesis	Spondylolysis
T12			
L1			
L2			
L3			
L4			
L5			
S1			

Level of involvement	Endplate changes	
	Schmorl's nodes	Modic changes with type
T12		
L1		
L2		
L3		
L4		
L5		
S1		

Level of involvement	Ligament and joint changes	
	Facet arthropathy	Ligamentum flavum hypertrophy
T12-L1		
L1-2		
L2-3		
L3-4		
L4-5		
L5-S1		

Level	Spinal canal diameter (mm)
T12-L1	
L1-2	
L2-3	
L3-4	
L4-5	
L5-S1	

Level of involvement	Nerve root compression		
	Right	Left	Bilateral
T12-L1			
L1-2			
L2-3			
L3-4			
L4-5			
L5-S1			

Infective/inflammation: ☐ Yes ☐ No

Mass lesion: ☐ Yes ☐ No

Congenital anomalies: ☐ Yes ☐ No

Others:

MRI Diagnosis:

Final diagnosis:

ANNEXURE-II

INFORMED CONSENT FORM

Study title: MAGNETIC RESONANCE IMAGING IN EVALUATION OF LOW BACK PAIN OF NON TRAUMATIC ETIOLOGY.

Chief researcher/ PG guide's name: Dr. ANIL KUMAR SAKALECHA

Principal investigator: DR. SUJATA

Name of the subject:

Age :

Gender :

a. I have been informed in my own language that this study is contraindicated in patients with metallic implants, cardiac pacemaker, ear cochlear implants, brain/aneurysmal clip, metal in eyes, metal fragments of shrapnel, magnetic dental implants, implanted electrical device, artificial heart valves, neurostimulator, stents/shunts, tissue expander, insulin pump and IUD. I thoroughly understand its complication and possible side effects.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
e. I confirm that Principal investigator / Chief researcher (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that i may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant’s name and signature/thumb impression (in case of illiterate)

Signature of the witness (in case of illiterate person): Date:

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Principal investigator/ Guide signature Date:

Magnetic Resonance Imaging in Evaluation of Low Back Pain Of Non-Traumatic Etiology

Patient Information Sheet

Principal Investigator: Dr. Sujata/ Dr. Anil Kumar Sakalecha

I, Dr. Sujata, am a post-graduate student in Department of Radio-Diagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "Magnetic resonance imaging in evaluation of low back pain of non-traumatic etiology" for my dissertation under the guidance of Dr. Anil Kumar Sakalecha, Professor, Department of Radio-Diagnosis. In this study, we will assess the role of magnetic resonance imaging study in the evaluation of causes for low back pain. You would have undergone MRI study of the low back spine before entering the study. There will be no additional expenses incurred by you for participation in this study. MRI is considered as a safe and very effective diagnostic test and there is no harm whatsoever for you during the study.

The study will help us understand the causes for low back pain and help us in planning appropriate treatment and improve care of patient.

All of your personal data will be kept confidential and will be used only for research purpose by this institution. You are free to participate in the study. You can also withdraw from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator

Date

ANNEXURE-III

KEY TO MASTER CHART

A	Asymmetrical
AC	Arachnoid cyst
AF/T	Annular fissure/Tear
B	Bilateral
C	Central
Co	Concentric
CON	Congenital
DC	Degenerative changes
DMM	Diastematomyelia
CON	Congenital
EPC	End plate changes
EXT	Extrusion
F	Female
FA	Facetal arthropathy
Fib	Fibroid
INFL/INFEC	Inflammatory/infective
Ky/Sc – A	kyphosis/scoliosis acquired
Ky/Sc – C	kyphosis/scoliosis congenital
L	Left
LEV INV	Level involved
LSTV – L	Lumbosacral transitional vertebra – Lumarization
LSTV – S	Lumbosacral transitional vertebra - Sacralization
LP	Left paracentral

M	Male
Met	Metastasis
Modic	Modic changes
MMC	Myelomeningocele
MRI	Magnetic resonance imaging
N	No
NRC	Nerve root compression
NEO	Neoplastic / Neoplasm
OP	Osteophytes
PNC	Perineural cyst
Pyo	Pyogenic spondylitis
R	Right
Rd	Radial
RP	Right paracentral
SEQ	Sequestration
S	Symmetrical
SCD	Spinal canal diameter
SC-MMC	Sacrococcygeal myelomeningocele
SPL	Spondylolysis
SPLIS	Spondylolisthesis
T	Transverse
TB	Tuberculosis of spine/Pott’s spine
Y	Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
1	182375	65	F	N	L3-4	S	N	N	N	N	N	N	N	N	II	Y	Y	11	N	N	N	PNC	DC	DC
					L4-5	S	C	N	N	N								12						
					L5-S1	S	RP	N	N	N								11						
2	47809	33	F	B	T12-L1	N	N	N	N	N	N	N	N	N	N	N	N	12	Y	N	N	N	TB	TB
3	146249	40	F	L	L4-5	N	C	N	N	N	N	N	N	N	N	N	N	7	N	N	N	OC	DC	DC
					L5-S1	S	LP	N	N	N								11.7						
4	179975	50	F	B	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	8	Y	N	N	Ky/Sc - A	TB	TB
5	176988	28	M	B	L4-5	S	N	N	N	N	N	N	N	N	N	N	N	10.5	N	N	N	Ky/Sc - C N	DC	DC
6	166526	18	M	N	L4-5	S	N	N	N	N	N	N	N	Y	N	N	N	12	N	N	N	N	DC	DC
					L5-S1	S	N	N	N	N								12.8						
7	171824	65	M	R	L4-5	S	N	N	N	N	N	N	N	N	N	N	N	12.3	N	N	N		DC	DC
					L5-S1	A	N	N	N	N								10.8				N		
8	169562	49	M	R	L3-4	S	N	N	N	N	Y	II	Y	Y	II	N	N	13	N	N	N	N	DC	DC
					L4-5	A	N	N	N	Co								11.5						
					L5-S1	N	RP	N	N	N								8.3						
9	168317	44	M	B	L3-4	S	N	N	N	N	N	N	N	N	II	N	N	12	N	N	N	N	DC	DC
					L4-5	S	N	N	N	N								10.5						
					L5-S1	A	N	N	N	N								10.2						
10	167637	38	F	L	L3-4	S	N	N	N	N	Y	N	Y	N	I	N	N	7.7	N	N	N	N	DC	DC
					L4-5	S	N	Y	N	Rd								4						

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
11	167059	70	M	B	L1-2	S	N	N	N	N	N	III	Y	Y	II	Y	Y	10.1	N	N	N	N	DC	DC
					L2-3	S	N	N	N	N								10.2						
					L3-4	S	N	N	N	N								8.2						
					L4-5	S	N	N	N	N								9.9						
					L5-S1	S	N	N	N	N								9.6						
12	167456	61	F	B	L3-4	A	N	N	N	N	N	N	N	N	II	N	Y	13.4	N	N	N	N	DC	DC
					L4-5	S	N											9.9						
					L5-S1	S	N											9						
13	166822	50	F	N	L4-5	A	N	N	N	N	N	N	Y	N	N	N	Y	13.7	N	N	N	N	DC	DC
					L5-S1	A	N	N	N	N								12.5						
14	166597	51	M	N	L4-5	N	N	N	N	N	N	N	N	N	N	N	Y	7.7	Y	N	N	N	TB	TB
15	166427	18	F	R	L4-5	A	N	N	N	N	N	N	N	Y	N	N	N	12	N	N	N	N	DC	DC
					L5-S1	A												13.2						
16	165235	36	M	L	L4-5	A	N	N	N	N	N	N	N	N	N	N	N	8.7	N	N	N	N	DC	DC
17	164683	70	F	B	L4-5	S	N	N	N	N	N	N	N	Y	III	N	Y	10.2	N	N	N	OC	DC	DC
					L5-S1	S	N	N	N	N								10.8						
18	164404	28	M	B	L4-5	S	N	N	N	N	N	N	N	N	N	N	N	12.3	N	N	N	N	DC	DC
					L5-S1	S	N	N	N	N								11.4						
19	139096	35	M	R	L3-4	S	C	N	N	Co	N	N	N	Y	I	Y	Y	3.7	N	N	N	N	DC	DC
					L4-5	S	C	N	N	N								4.2						
					L5-S1	A	RP	N	N	N								7						

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
20	66193	43	M	N	L2-3	S	N	N	N	N	N	N	Y	N	N	Y	Y	10	N	N	N	N	DC	DC
					L3-4	S	N	N	N	N								9.6						
					L4-5	A	N	N	N	N								14.3						
21	175023	40	M	B	L3-4	S	C	N	N	N	N	N	Y	N	N	N	N	6.5	N	N	N	N	DC	DC
					L4-5	A	LP	Y	N	N								5.4						
					L5-S1	N	N	N	N	Rd								9						
22	174990	38	M	N	L3-4	A	N	N	N	N	N	N	Y	N	I	N	N	12	N	N	N	LSTV-S N	DC	DC
					L4-5	A	N	N	N	N								13.2						
23	174443	35	M	B	L3-4	S	N	N	N	N	N	N	N	Y	II	Y	Y	7.4	N	N	N	N	DC	DC
					L4-5	S	C	N	N	N								5.5						
24	173469	45	M	R	L3-4	A	N	N	N	N	N	N	Y	N	N	Y	N	10	N	N	N	N	DC	DC
					L4-5	S	N	N	N	N								12						
					L5-S1	A	N	Y	N	N								11						
25	327737	25	M	R	T12-L1	N	N	N	N	N	N	N	Y	N	II	N	N	6.5	Y	N	N	LSTV-L N	TB	TB
26	120996	37	F	N	T12-L1	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	N	TB	TB
27	118610	65	F	B	L3-4	S	N	N	N	N	Y	IV	N	Y	III	Y	Y	5.5	N	N	N	Ky/Sc - C	DC	DC
					L4-5	A	N	N	N	N								6.4						
28	120998	60	F	B	L1-2	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	N	TB	TB
					L3-4	S	N	N	N	N								13.2						
29	118026	25	M	R	L3-4	S	N	N	N	N	N	N	Y	Y	I	N	Y	7.4	N	N	N	LSTV-S	DC	DC
					L4-5	S	N	N	N	N								5.2						

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
30	115889	53	M	B	L3-4	S	N	N	N	N	N	N	N	N	N	N	N	3.9	N	N	N	N	DC	DC
					L4-5	A	N	Y	Y	N								4						
					L5-S1	A	N	N	N	N								7						
31	117152	20	F	N	L5-S1	N	N	Y	Y	Co	N	N	N	N	N	N	N	11	N	N	N	N	DC	DC
32	1020606	40	F	B	L4-5	N	N	Y	Y	T	N	N	N	N	N	N	N	12	N	N	N	N	DC	DC
33	139552	60	F	R	L4-5	A	RP	N	N	N	Y	I	Y	Y	III	N	N	5.8	N	N	N	N	DC	DC
34	139401	65	M	R	L3-4	N	N	N	N	N	N	N	N	N	N	Y	N	7.2	N	Y	N	N	Met	Met
					L4-5													4.6						
35	137754	36	F	R	L3-4	S	RP	N	N	N	Y	II	Y	Y	II	Y	N	8	N	N	N	FIB	DC	DC
36	130716	60	M	N	L3-4	A	N	N	N	N	N	N	N	Y	II	N	Y	5.8	N	N	N	PNC	DC	DC
					L4-5	N	C	N	N	N								8.4						
37	133557	26	F	R	L4-5	A	N	N	N	N	Y	N	N	N	II	N	N	4.4	N	N	N	N	DC	DC
					L5-S1	A	N	N	N	N								5						
38	134261	20	M	N	L3-4	A	N	N	N	N	Y	N	N	Y	I	N	N	4.8	N	N	N	LSTV-S	DC	DC
					L4-5	N	C	N	N	N								9						
39	134720	45	M	R	L3-4	A	N	N	N	N	N	N	Y	N	II	N	N	8	N	N	N	LSTV-L	DC	DC
					L4-5	A	N	N	N	Co								9.9						
					L5-S1	S	RP	N	N	N								10.2						
40	130223	40	M	B	L2-3	N	C	N	N	N	N	N	Y	Y	II	N	Y	3	N	N	N	N	DC	DC
					L3-4	A	C	N	N	Co								4.2						
					L4-5	S	N	Y	N	N								5.4						
41	1149358	35	M	B	L3-4	S	N	N	N	N	N	N	Y	Y	I	Y	Y	8	N	N	N	LSTV-S	DC	DC
					L4-5	S	N	Y	N	N								7.4						

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
42	129350	20	M	R	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	N	Pyo	Pyo
																		13.5						
43	139871	41	M	N	T12-L1	N	N	N	N	N	N	N	N	N	N	N	N	9	Y	N	N	Ky/Sc - A	TB	TB
44	141772	46	F	B	L4-5	S	N	N	N	N	Y	I	N	Y	II	Y	Y	11	N	N	N	N	DC	DC
45	141961	44	F	B	L5-S1	S	N	N	N	N	Y	I	Y	N	II	Y	Y	9	N	Y	N	N	Neo	SC
46	142035	30	M	R	L4-5	N	N	N	N	N	N	N	N	N	N	N	N	2.4	Y	N	N	N	Pyo	Pyo
47	147022	56	M	B	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	N	Pyo	Pyo
48	147605	65	M	B	L2-3	A	N	N	N	N	Y	I	Y	Y	N	Y	Y	8.7	N	N	N	Ky/Sc - C	DC	DC
					L3-4	S	N	N	N	N								9.4						
					L4-5	S	N	N	N	N								10.4						
49	150755	80	F	B	L4-5	N	C	N	N	N	Y	I	N	Y	III	Y	Y	1.5	N	N	N	FIB	DC	DC
					L5-S1	S	N	N	N	Rd								4.6						
50	153237	20	M	N	L5-S1	N	N	N	N	N	N	N	N	N	N	N	N	12	Y	N	N	Ky/Sc - C	TB	TB
51	146263	32	F	N	L5-S1	S	N	N	N	N	N	N	N	N	N	N	N	11.8	Y	N	N	OC	SI	SI
52	154088	40	F	R	T12-L1	N	N	N	N	N	N	N	Y	N	N	N	Y	11.6	Y	N	N	N	TB	TB
53	160030	58	M	R	T12-L1	N	N	N	N	N	N	N	N	N	N	N	N	12	Y	N	N	N	TB	TB
54	22867	55	M	B	L3-4	N	N	N	N	N	N	N	Y	Y	N	N	N	5.8	N	AC	N	PNC	AC	AC
55	150673	38	F	L	L4-5	A	N	Y	Y	Rd	Y	II	Y	Y	I	Y	Y	8.7	N	N	N	PNC	DC	DC
					L5-S1	S	RP	N	N	N								9.9						
56	240386	64	F	R	L1-2	S	N	N	N	N	N	N	Y	Y	II	N	N	5.6	N	AC	N	OC	AC	AC

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
57	182374	34	F	L	L4-5	S	N	N	N	N	Y	I	Y	N	II	Y	Y	4.3	N	N	N	FIB	DC	DC
					L5-S1	S	LP	Y	Y	N								6.3						
58	203382	60	F	B	L4-5	N	N	N	N	N	N	N	N	N	N	N	N	8	Y	N	N	OC	TB	TB
59	202368	70	M	B	L5-S1	N	N	N	N	N	Y	II	N	Y	N	N	N	11.6	Y	N	N	LSTV-S	TB	TB
60	195611	20	M	N	L1-2	N	N	N	N	N	N	N	N	N	N	N	N	6	N	N	Y	Ky/Sc - A	DMM	DMM
61	195058	65	M	B	L1-2	N	N	N	N	N	N	N	N	N	N	N	N	4.4	N	Y	N	LSTV-S	Met	Met
					L2-3	N	N	N	N	N								5.2						
62	221109	25	M	L	L3-4	S	N	N	N	Co	N	N	Y	N	N	N	N	5.6	N	N	N	LSTV-S	DC	DC
					L4-5	A	LP	Y	Y	N													DC	DC
63	224968	42	M	N	L4-5	A	RP	N	N	Rd	N	N	Y	Y	I	N	N	3.2	N	N	N	N		
					L5-S1	N	LP	Y	Y	N														
64	228312	41	F	B	L4-5	S	RP	N	N	Co	Y	I	Y	Y	II	N	Y	8.7	N	N	N	N	DC	DC
65	240386	49	F	N	L5-S1	N	N	N	N	Co	N	N	Y	Y	II	N	N	5.6	N	N	N	OC	DC	DC
66	244642	75	M	L	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	11.2	N	Y	N	PNC	Met	Met
					L4-5																			
67	252102	52	M	B	L3-4	S	N	N	N	N	N	N	Y	Y	II	Y	Y	7.6	N	N	N	Ky/Sc - C	DC	DC
					L4-5	N	C	Y	Y	Rd								8.4						
					L5-S1	N	LP	Y	Y	N								9.6						
68	253911	56	F	B	L4-5	S	N	N	N	N	N	N	Y	N	II	N	N	5.2	N	N	N	Ky/Sc - A	DC	DC
					L5-S1	A	C	N	N	Rd														
69	251778	20	F	L	L5-S1	N	RP	Y	Y	N	Y	I	N	Y	II	N	N	4.8	N	N	Y	OC	AVM	AVM
70	246224	50	M	B	L4-5	S	LP	Y	Y	N	Y	II	Y	Y	II	Y	Y	7.4	N	N	N	Ky/Sc - C	DC	DC
					L5-S1	N	C	Y	N	Co								6.3						

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
71	247217	36	M	L	T12-L1	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	Ky/Sc - A	TB	TB
72	236209	65	M	B	L5-S1	N	N	N	N	Co	N	N	Y	Y	N	N	N	12	N	Met	N	LSTV-S	Met	Met
73	257196	30	M	B	L4-5	S	RP	Y	Y	N	N	N	N	N	N	N	N	11.4	N	N	N	LSTV-L	DC	DC
					L5-S1	S	LP	N	N	Rd								13						
74	269192	17	M	B	L5-S1	S	N	N	N	N	N	N	N	N	N	N	N	12	Y	N	N	PNC	SI	SI
75	255855	46	M	B	L3-4	A	C	Y	N	Co	N	N	Y	Y	II	Y	Y	7.4	N	N	N	LSTV-S	DC	DC
					L4-5	N	LP	Y	Y	N														
76	263991	68	M	R	L3-4	A	C	N	N	Co	N	N	Y	Y	III	Y	Y	5.4	N	N	N	PNC	DC	DC
					L4-5	S	RP	Y	Y	N								6.3						
					L5-S1	S	C	N	N	N								7						
77	265867	30	F	B	L5-S1	S	N	N	N	N	N	N	N	N	N	N	N	4.3	Y	N	N	OC	SI	SI
78	266754	45	M	L	L3-4	S	N	N	N	N	Y	I	Y	Y	II	Y	Y	11	N	N	N	OC	DC	DC
					L4-5	N	C	N	N	N								13.2						
79	268498	47	F	L	L4-5	S	LP	Y	Y	Co	Y	I	Y	Y	II	N	N	12	N	N	N	N	DC	DC
80	243834	60	F	B	L3-4	S	RP	N	N	N	N	N	Y	N	II	N	N	12	N	N	N	PNC	DC	DC
					L4-5	S	N	N	N	Co								13						
81	272484	68	M	R	T12-L1	N	N	N	N	N	N	N	Y	N	N	N	N	4.6	N	Y	N	LSTV-S	Neo	MM
82	272829	80	M	B	L4-5	S	RP	Y	Y	Rd	N	N	Y	Y	III	Y	Y	8.4	N	N	N	LSTV-S	DC	DC
					L5-S1	S	LP	N	N	T								7.4						
83	273244	35	F	N	L5-S1	S	N	N	N	N	N	N	N	N	II	N	N	11.5	Y	N	N	OC	Infl	SI
84	276029	55	F	N	L4-5	N	N	N	N	N	N	N	N	N	N	N	N	12.8	Y	N	N	Ky/Sc - A	TB	TB

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
85	277276	56	M	B	L3-4	S	RP	N	N	N	Y	I	Y	Y	II	N	N	12.8	N	N	N	PNC	DC	DC
					L4-5	S	LP	Y	N	Co								13						
					L5-S1	S	N	N	N	N								11.6						
86	278054	50	F	N	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	7.7	N	Y	N	FIB	Neo	GCT
87	279907	54	F	R	L3-4	A	C	Y	N	Co	N	N	Y	Y	II	Y	Y	11.9	N	N	N	OC	DC	DC
					L4-5	S	RP	Y	Y	N								12.3						
					L5-S1	S	LP	N	N	N								13.2						
88	283298	20	M	B	L4-5	S	RP	Y	Y	Co	N	N	Y	Y	II	Y	N	11.5	N	N	N	PNC	DC	DC
					L5-S1	A	C	N	N	N								12						
89	283363	47	M	B	L4-5	S	RP	Y	Y	N	N	N	Y	Y	II	N	Y	7.6	N	N	N	N	DC	DC
					L5-S1	A	C	Y	N	Co								6.4						
90	285267	50	F	N	L4-5	A	N	N	N	Rd	N	N	N	N	N	N	N	12.3	N	Y	N	LSTV-L	Met	Met
91	285583	60	M	B	L3-4	A	C	Y	Y	Co	N	N	Y	Y	II	Y	Y	9.2	N	N	N	PNC	DC	DC
					L4-5	S	RP	N	N	N								8.5						
					L5-S1	S	C	N	N	Rd								7.4						
92	285994	45	M	B	L4-5	S	LP	Y	Y	Rd	Y	II	Y	N	II	Y	N	11.6	N	N	N	PNC	DC	DC
					L5-S1	S	RP	N	N	N								12.4						
93	286194	85	M	B	L3-4	S	N	Y	Y	N	N	N	Y	Y	II	N	Y	7.8		N	N	Ky/Sc - A	DC	DC
					L4-5	S	RP	N	N	Co								5.4						
					L5-S1	S	N	N	N	Rd								4.3						
94	286410	80	M	B	L5-S1	N	N	N	N	N	Y	I	Y	Y	N	N	N	12.8	N	Y	N	PNC	Met	Met

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes

Masterchart – Magnetic Resonance Imaging in Evaluation in of Low Back Pain of Non-Traumatic Etiology

Sl No.	ID	Age	Sex	RP	Level Involved	Disc Changes					Vertebral Body Changes			EPC		FA	LFH	SCD (in mm)	INFL/ INFEC	NEO	CON	Others	MRI Diagnosis	Final Diagnosis
						Bulge	PR	EXT	SEQ	AF/T	SPL	SPLIS	OP	SN	Modic									
95	287483	52	F	B	L3-4	A	C	Y	Y	Rd	Y	III	Y	Y	II	Y	N	8.2	N	N		FIB	DC	DC
					L4-5	S	RP	N	N	N								7.4						
					L5-S1	S	LP	Y	N	N								6.2						
96	293073	37	M	B	L4-5	A	C	N	N	Co	N	N	N	Y	N	Y	Y	11	N	N	N	PNC	DC	DC
					L5-S1	S	RP	Y	Y	N								13						
97	315297	55	M	B	T12-L1	N	N	N	N	N	Y	I	N	N	N	N	N	12.3	N	Y	N	Ky/Sc - C	Met	Met
98	301254	50	F	B	L4-5	N	RP	Y	Y	N	N	N	Y	Y	N	N	Y	5.4	N	N	N	FIB	DC	DC
					L5-S1	S	N	N	N	Co								6.5						
99	311645	65	M	B	L2-3	N	N	N	N	N	N	N	N	N	II	Y	Y	2	N	Met	N	PNC	Met	Met
100	299272	50	F	L	L4-5	N	N	N	N	N	N	N	Y	Y	N	N	N	4	TB	N	N	Ky/Sc - A	TB	TB
101	298365	20	F	B	T12-L1	N	N	N	N	N	N	N	Y	Y	N	N	N	3	TB	N	N	OC	TB	TB
102	294559	59	M	B	L3-4	S	RP	Y	N	T	Y	I	Y	Y	N	Y	Y	7.9	N	N	N	Ky/Sc - C	DC	DC
					L4-5	N	C	N	N	N								8.4						
103	264189	60	M	B	L2-3	N	C	N	N	Co	N	N	Y	Y	II	Y	Y	8	N	N	N	PNC	DC	DC
					L3-4	A	C	N	N	N								7.4						
					L4-5	A	N	N	N	N								9.6						
104	198516	2	F	N	L4-5	N	N	N	N	N	N	N	N	N	N	N	N	11.6	N	N	Y	LSTV-L	TL-MMC	TL-MMC
105	285551	6	M	N	L4-5	N	N	N	N	N	N	N	N	N	N	N	N	12.8	N	N	Y	LSTV-S	SC - MMC	SC - MMC
106	30541	75	F	B	L3-4	N	N	N	N	N	N	N	N	N	N	N	N	11	Y	N	N	Ky/Sc - A	TB	TB

A = Asymmetrical; AC = Arachnoid cyst; AF/T = Annular fissure/Tear; AVM = Arteriovenous malformation; B = Bilateral; C = Central; Co = Concentric; CON = Congenital; DC = Degenerative changes; DMM = Diastematomyelia; EPC = End plate changes; EXT = Extrusion; F = Female; FA = Facetal arthropathy; Fib = Fibroid;;; INFL/INFEC = Inflammatory/infective; Ky/Sc – A = kyphosis/scoliosis – Acquired; Ky/Sc – C = kyphosis/scoliosis- Congenital; L = Left; LEV INV = Level involved; LFH = Ligamentum flavum hypertrophy; LSTV – L = Lumbosacral transitional vertebra – Lumarization; LSTV – S = Lumbosacral transitional vertebra – Sacralization; LP = Left paracentral; M = Male; Met = Metastasis; Modic = Modic changes; MM = Multiple myeloma; MMC = Myelomeningocele; MRI = Magnetic resonance imaging; N = No; NRC = Nerve root compression; NEO = Neoplasm; OC = Ovarian cyst; OP = Osteophytes; PNC = Perineural cyst; Pyo = Pyogenic spondylitis; R = Right; Rd = Radial; RP = Right paracentral; S = Symmetrical; SC = Sacral chordoma; SC-MMC = Sacrococcygeal myelomeningocele; SEQ = Sequestration; SPL = Spondylolysis; SPLIS = Spondylolisthesis; SN = Schmorls nodes; T = Transverse; TB = Tuberculosis of spine/Pott's spine; TL-MMC = Thoracolumbar myelomeningocele; Y = Yes