Correlation of Magnetic Resonance Imaging Findings with Clinical Features of Low Back Pain in a Tertiary Hospital in Delta State Nigeria

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Abstract

Background

Low back pain is a common presenting complain frequently encountered in clinical practice It affects mostly adultpopulation of any age with several associated symptoms. Magnetic Resonance Imaging (MRI) as an imaging modality has excellent soft tissue resolution with the ability to clearly visualize abnormalities in the lumbosacral spine.

Aim of study

The aim of this study was to correlate the MRI findings with clinical features of patients with low back pain in DELSUTH.

MaterialsandMethod

A hospital based cross-sectional prospective study of 150 consenting adults with history of low back pain, recruited from the patients referred for lumbosacral MRI, was carried out using a 1.5Tesla MRI scanner (Toshiba excelart vantage March 2015). Total population sampling was used. Analysis of the collated data was done using Statistical Package for Social Sciences version 25.0 (SPSS Inc. ILUSA).

Results

The age range of the participants was 20 to 105years with a mean age of 59.9 ± 13.2years with 96(64%) males, while 54(36%) were females. The MRI findings showed abnormalities in 141(94%) of participants while 9(6.0%) participants had normal findings. There was significant correlation between protrusion,extrusion and other MRI findings with sciatica, radiculopathy, difficulty in walking and urinaryincontinence. ($p \leq 0.005$)

Conclusion

There was statistically significant positive correlation between MRI findings and age of the participants with LBP. There was also positive correlation between MRI findings of disc protrusion, disc extrusionwith sciatica.

Key words: Magnetic resonance imaging, sciatica, low back pain

Introduction

Lowbackpain (LBP) is one of the most common causes of disability with increased prevalence in the developed nations and a common disease of the musculoskeletal system[1,2]. It arises from pathologies in the lumbosacral region[3,4] such as spondylosis, sprains and strains, intervertebral disc degeneration, herniated orruptured discs, radiculopathy, sciatica, spinal stenosis, spondylolisthesis and trauma. Other underlying conditions can also result in LBP[5]. Predisposing factors for LBP also abound, such as increasing age, sedentary lifestyle, pregnancy, genetic conditions(ankylosing spondilitis), occupation, (jobs that that require heavy lifting, pushing or pulling, twisting or vibrating the spine, or inactive job such as a desk job) poor posture, mental health factors(anxiety, depression and stress causing muscle tension)[1–3,6,7]. Clinical features that may arise alongside LBP range from as inability to walk, run, paresthesia, sciatica, radiculopathies, fecal or urinary incontinence to paralysis. Magnetic Resonance Imaging (MRI) has been found to be most useful in evaluating the lumbosacral spine especially in degenerative spine disease which has been found to be the commonest cause of low back pain[8]. Due to its excellent soft tissue resolution and the non-use of ionizing radiation. MRI is able make help diagnosis of low backpain pathologies and detect abnormalities and in the vertebral bodies, intervertebral disc, spinal cord, spinal canal, spinal foramina and nerve roots in patients with low back pain. However, it has been reported that a significant number (35%) of asymptomatic individuals before 40 years of age may have significant degenerative changes in the lumbosacral spine at one or more vertebral levels on MRI images. Correlation with the clinical evidence is therefore important before any clinical relevance is attached to their presence[4,5]. Hence, this studywas aimed at correlating the MRI findings in the lumbosacral spine with clinical features and symptom complex of the participants with low back pain to evaluate how this relationship is important in arriving at a proper diagnosis to aid clinicians in deciding management decisions in patients with low back pain[9].

MaterialsAndMethod

A hospital based cross-sectional prospective study of 150 consenting adults, recruited from the patients referred for lumbosacral MRI with history of low back pain, from the Neurology, Neurosurgery, Orthopedics and other clinics of the hospital, using a Total population sampling method. A structured questionnaire was then administered to consenting patients to obtain relevant information about the clinical symptoms they experience alongside the low back pain. The clinical course and duration of low back pain un this study was described and categorized as acute (0-4weeks), subacute (5-12 weeks) and chronic (>12weeks). On a scale of 1-10, participants were told to rate their pain. 0-4 was grouped as mild, 5-7 grouped as moderate while 8-10 was grouped as severe. Other relevant clinical information was obtained from the MRI request cards presented in the Radiology department and from the case notes of the patients from Records department of the hospital. Pregnant women and adults with non-MRI compatible devices or implants were excluded from the study. MRI was carried out using a 1.5Tesla MRI scanner (Toshiba excelart vantage March 2015).The patients were positioned supine and comfortably in the MRI scanner with their legs straight in psoas tight position to ensure lumbar lordosis with the median sagittal plane of the patients equidistant to the table edges. A radio frequency surface coil was placed over the patients to cover thelumbar spine. Centering was at L2.

The table was then set in motion until the patient was at the isocentre[11]. The sagittal images were acquired by covering the entire width of the spine including the neural foramina. The axial images were acquired parallel to the discs covering the adjacent margins and including the endplates of theadjacent vertebral bodies. MRImagingwasperformedusingconventionalspinechopulsesequences.
SagittalT_2weightedfastspinecho[repetition time(TR)/echotime(TE), 3000-10000/80-150], SagittalT_1weighted (T1W) sequences (TR/TE, 300- 800/10-40*). Sagittal STIR sequence (TR/TE 4650/108)wasobtained using a matrix of192 ×320, 350mm field of view and 4/0.4mm section thickness/gap. AxialT_2weighted fast spin echo (TR/TE, 3000-10000/80-150) sequences was obtained using a matrix of 320×192, 240-mm field of view and 4/0.4-mm section thickness/gap.[11] AxialT_1weighted fast spin echo (TR/TE 300-900/15-30) sequences was obtained using a matrix of 320×192, 240-mm field of view and 4/0.4-mm section thickness/gap.

Technicalspecifications included slicethickness of3 and 4 mm forsagittal and axial images respectivelywith 1 mm gap as well as 90°flipangle forT1and180°anglefor T2. T1- and T2-weighted, STIR sequences were taken in the, sagittal and coronal planes with slices extending from the superior aspect of L1 to the coccyx. Also, axial images through the L1/L2, L2/L3, L3/L4, L4/L5 and L5/S1 discs were also taken in the sequences. Intravenous gadolinium though not administered routinely to all patients, was however administered to those who better characterization of the abnormality seen in the spine was required. Dose was 10mls stat.

The acquired images were stored in PictureArchivingComputerizedSystem(PACS), viewed and reported according to a standardized protocol.

An MRI worksheet which was used to document the MRI findings of the participants. It had a 42 items in a 2 point Linkert scale to determine either normal or abnormal features in the MRI findings lumbosacral spine findings such as disc degeneration, disc herniation (bulge, protrusion, extrusion or sequestration) and nerve root displacement/compression, spinal stenosis, central canal stenosis, lateral recess, foraminal and extraforaminal narrowing, Modic endplate abnormalities, lumbar lordosis, osteophytes, presence or absence of HIZ (annular fissures, at each ofthe spinal levels were documented[10–13,14](Fig. 1.).These features seen were then correlated with the Clinical features.

Ethical consideration

Ethical approval was obtained from the health research and ethics committee of DELSUTH before the commencement of the study. Written informed consent was obtained from the participants in this study using an informed consent form, following thorough explanation of the study objectives, methods of examination. Possible risks and benefits. To ensure confidentiality, codes were used on the data collection sheets of the patients. Participants were not offered any form of inducements as participation was entirely voluntary.

DataAnalysis

Data entry and analysis were carried out using Statistical Package for Social Sciences for Windows version 25.0 (SPSS Inc. Chicago, IL, USA)[15]. Data comparison (statistical test of significance) was done using Pearson Correlation analysis, chi square test foe categorical variables, student t-test for statistical test of significance.

Categorical data was expressed in frequency and percentage while continuous data was expressed as mean (+/- standard deviation). Analyzed data was presented in the form of tables and graphs. P-values of less than or equal to 0.05 was considered statistically significance at 95% confidence interval.

Result

In this study of 150 participants, 96(64%) were males and 54(36%) were females (Male to female ratio of 1.7:1). The mean age was 59.9 ± 13.2 year with 50-59years as the majority 45 (30.0%) of the study population.

All participants in this study had low back pain. The clinical course and duration of low back pain in this study was describedand categorized as acute (0-4weeks), subacute (5-12 weeks), and chronic (>12weeks). Majority of the participants 120(80%) had chronic LBP, over one-tenth 25(16.7%) had subacute while only 5(3.3%) had acute LBP. (Table 1)

On a scale of 1-10 participants were told to rate their pain. 0-4; was grouped as mild. 5-7 grouped as moderate while 8-10 was grouped as severe. Only 2(1.3%) patients had mild LBP, about one third of patients 35(23.3%) had moderate pain while majority of the participants 113(75%) had severe LBP.(Table 1)

Associated symptoms of low back pain included sciatica 131(77.3%), 65 (43.3%) had bilateralsciatica. There was equal proportion 33(22.0%), of those that had unilateral (right or left) sciatica. Other neurologic features included; difficulty in walking 80(53%), paraesthesia 42(28.0%), limb weakness 77(51.3%), urinary incontinence 14(9.3%) and fecal incontinence 9(6.0%)(Table 1)

The commonest provisional diagnoses were radiculopathy in 41(27.3%) cases, lumbar spondylosis 31(20.7%), spinal stenosis 21(14.0%), Chronic back pain? cause 40(26.7%) and disc prolapse 5(3.3%) transverse myelitis 4(2.7%). The least was Potts spondylitis in 1(0.7%) cases. Overall, radiculopathy was the commonest provisional diagnosis as an entity or in association with other symptoms. (Table 2) of the 150 participants, in 141(94%) of participants showed abnormal MRI findings in their lumbosacral spine, while 9(6.0%) participants had normal findings. (Figure 1)

In correlating the clinical features with MRI findings, Sciatica was seen to be present in 131participants. (Table 3) A large number of patients with disc protrusion 93(92.1%), extrusion10 (7.3%). had sciatica. Only a small proportion of these participants had disc bulge. There was statistically significant relationship between sciatica and disc protrusion (p=0.012) and disc extrusion (p= 0.002). (Table 7)

There was statistically significant relationship between sciatica and vertebral abnormalities/ canal narrowing (p= 0.044). However, there was weak but not significant negative correlationbetween sciatica and disc desiccation (r= -0.002, p=-1000),annular fissure, (r=-0.135, p=-0.114),ligamentum flavum hypertrophy (r=-0.040, p=-0.633) and spinal canal stenosis (r= 0.165, p=-0.121)

In total, there was positive significant correlation between sciatica and vertebral abnormalities and canal narrowing. (r=0.032, p=0.039) (Table 8)

There was statistically significantly relationship between difficulty in walking (p= 0.013); urinary incontinence (p= 0.032) and disc protrusion, with positive significant correlation (r=0.203, p=0.015); (r=0.175, p=0.036) respectively. (Table7)

There is also statistically significant relationship between annular fissure and difficulty in walking (p= 0.020) with a positive significant correlation (r= 0.195, p=0.020). (Table7)

All the patients who had urinary and fecal incontinence had disc protrusion though there was no statistically significantly relationship between fecal incontinence and the MRI findings. (Table 8)

There was statistically significant relationship between the provisional diagnosis of the patient with disc protrusion (p= 0.029), spinal canal stenosis (p= 0.005) and lateral recess narrowing/nerve compression. (p= 0.030). with statistically significant relationship (p= 0.012),(p= 0.0001) and positive correlation for total disc and vertebral abnormalities/canal narrowing and provisional diagnosis.(r=0.015, p=-.662);(r=0.040, p= 0.198) respectively. (Table7), (Table 8).

DISCUSSION

The Majority of the 150 participants in this study had severe and chronic LBP (>12weeks duration). This must have been due to late presentation or patient seeking other forms of remedies before presenting in the hospital. And the later the presentation, the more the likelihood of worsening of symptoms as well as the pathologic condition. The study by Adekanmi*et al*[6]reported similar pattern of LBP duration. In a study by Yong *et al*[7], where he studied the Correlation of clinical presentation, radiography and MRI for Low Back Pain in 57 patients in Malyasia,majority of them presented with chronic low back pain. It was found that in most of the patients the duration of low back pain was 1-2yrs, which is comparable to study done by Ng *et al*[8] where the duration of low back pain was 15 months. Similar results were found in the study by Chan *et al*[16]. The chronicity of LBP at presentation may be attributed to LBP being previously managed with analgesics or other means either by patient or managing physicians. It may also be due to late presentation after patient may have previously sought several remedies.

In this study also, more of the MRI findings were seen in patients with chronic back pain as well as severe back pain. This may be due to the fact that the longer the duration of the back pain and the causative pathology without treatment, the more the possible complications that could occur.

Sciatica was a presentation in majority of patients with affectation of both lower limbs having higher prevalence and in equal proportions of unilateral cases. Radiculopathy and lumbarspondylosis amongst others were major provisional diagnoses made by the referring physiciansin this study. Adekanmi*et al*[6] found similar results in their study where lumbar spondylosiswas the common provisional diagnosis, either alone or in association with other symptoms. The least common finding was pain in the lower limbs/sciatica in 2(1.9%) cases. This was however a retrospective study which may be a reason for the difference in these findings. It was also confirmed from another study that after low back pain and sciatica, the main complaint of patients with disc degeneration was neurogenic claudication, a main feature of lumbar spinal stenosis. It was 100% positive in all cases of lumbar stenosis and combination of stenosis and herniation. It was also concluded that the LBP and sciatica were due to nerve root compression, which was significantly associated with disc degeneration[9].

Many studies also reported disc degeneration to be the commonest MRI finding[10–12,17].

Yong *et al*[7] demonstrated in 56 patients with low back pain, disc degeneration 52 (91.2%) occurred commonly in MRI images of Japanese. Multiplicity in the disc level involvement was common compared to the single disc involvement[7]. This is consistent with that seen in other studies[14,18].

More participants with degenerative disease of the spine had chronic LBP with negative correlation with LBP severity. They also had sciatic pain with numbness, 92.9% with urinary incontinence and 100% of those with fecal incontinence all had degenerative disease of the spine. In a population-based study on 975 participants, Teraguchi*et al*[18]showed that whereas disc degeneration alone was not associated with chronic LBP, the combination of disc degeneration and end plate changes was highly associated with chronic LBP[18].

Disc herniation was one of the commonest MRI findings in 126 (84%) participants with LBP and was seen more at the L4/L5 disc level followed by L3/L4 and was statistically significant. The most common type of herniation seen in this study was disc protrusion followed by extrusion. Both had the commonest occurrence at L4/L5. It also can be deduced that the lower the lumbar level the higher the prevalence of disc herniation. Sequestration or migration was not observed in this study.

All the 5 participants who had acute LBP had disc herniation. Majority of the participants with chronic LBP also had disc herniation. This is expected as disc protrusion can significantly lead to nerve root compression with resultant pain and various neurologic symptoms. There was statistically significant correlation between sciatica and protrusion and extrusion. There was also statistically significant correlation with protrusion and difficulty in walking and urinary incontinence. The degree of disc herniation correlated well with the symptoms seen in different participants. This is expected as disc protrusion can significantly lead to nerve root compression and lead to various neurologic symptoms. In the study of Modic et al[19], he found that 43% of the patient who had lumbar disc herniation were had L4 L5 level disc involved. Similar results were found by Garrdio*et al*[20] in the study, the correlation was made between clinical findings and MRI findings and it was found that 65 (87%) of the patients had significant correlation while 10 (13%) of that patients had no significant correlation. Similar conclusion was made in a number of studies done[21–25]. However, in the study by Kjaer *et al*[26], they reported that disc herniation and nerve root compromise were not associated with LBP. The reason for this may be because their study was done on 40year old males and females only[26]. This index study however, included older patients where increased degree of disc degeneration and herniation were observed.

Annular tear or fissure also referred to as high intensity zone (HIZ) was seen in 27 participants. Majority of participants with chronic LBP and of those with severe LBP had annular fissure. None of the patients with mild LBP had annular fissure.

Some studies have shown that the HIZ was associated with acute LBP. They performed a histological study on excised disks with HIZ, concluded that the HIZ may be a pointer for the inflammatory reaction of a painful disc[27]. However, other studies have shown that HIZ is frequently observed in asymptomatic subjects[28]. As for recurrent LBP, Videman *et al*[29]showed that annular tear on axial MRI scans are associated with recurrent LBP.

Out of the total participants in this study, 58(38.7%) were found to have spondylolisthesis. Patients with previous history of trauma with possible traumatic spondylolisthesis, were excluded. Spondylolisthesis was also graded using the Meyerdings grading system where the antero-posterior diameter of the superior surface of the lower vertebral body was divided into four equal parts and a slip of <25%, 25-50%, 50-75% and >75% is Graded as I, II, III and IV respectively.[13,30] The prevalence of spondylolisthesis was far higher than that seen in the study by Saleem *et al*[10] who had a prevalence of spondylolisthesis of 7 (18.9%) which mostly occurred at the level of L5/S1 3 (8.1%) In the study by Saleem *et al*[10]and Yong *et al*[7], spondylolisthesis was seen in 12.3% of participants. 52(34.7% of total participants) had Grade I while 6(4.0% of total participants) had Grade II spondylolisthesis and no participants with Grade III or IV spondylolisthesis in this study. The presence of degenerative spondylolisthesis at that level was thought to be related to the more sagittal orientation of facet joint that makes them increasingly prone to anterior displacement[31]. Higher grades of spondylolisthesis can lead to a narrowed spinal canal with nerve compression and was more commonly found in the patients of spinal stenosis as compared to disc herniation, reflecting the fact that during stenosis, laxity of capsules and ligaments may result in the development of spondylolisthesis. There was statistically significant association between spondylolisthesis and central canal stenosis (p=0.003) in this study. 51(87.9%) of the 58 participants with spondylolisthesis had spinal canal stenosis while 7(12.1%) did not. 6 participants had Grade II while 45 had Grade I spondylolisthesis meaning that all the 6(100%) participants who had Grade II all had spinal canal stenosis while 45(86.5% of participant with spondylolisthesis) who had Grade I had central canal stenosis. This further prove that the higher the grade of spondylolisthesis, the more the likelihood of spinal canal compromise. Despite this, there was no statistically significant difference in the clinical features between the groups of patients with and without spondylolisthesis. Majority however, had severe LBP.

About half of those with spondylolisthesis had limb weakness and difficulty in walking.

Spinal stenosis has been found to be associated with a plethora of clinical symptoms and has also been defined as the focal, segmental or diffuse narrowing of the central canal or root canals by bony and/or soft tissue elements resulting in encroachment on the neural structures[32]Anatomically, spinal stenosis has been classified as central, when it affects the spinal canal and dural sac, foraminal, when it affects the spinal foramina, or lateral, when it affects the lateral recess. Central and lateral stenosis, are described as distinct entities as in this index study. In this study, 113(75.4%) had central spinal canal stenosis. This was similar to the study done by Premchandran*et al*[33] where out of 43 (73%) symptomatic cases, 34 (79%) cases were found to have narrow diameters of the spinal canal. It has been reported that three or four different syndromes, including neurogenic claudication, nerve root compression, central lower back pain and non-radicular referred lower extremity pain occurs in patients with spinal canal stenosis with classic presentation of neurogenic claudication.[34,35] Substantial reduction in walking tolerance because of neurogenic claudication has been reported to be a common clinical finding. More participants with chronic LBP had stenosis and almost equal proportion had constant and intermittent pain. However, the correlation between symptoms and the dural sac area has been considered to be poor

as observed in studies by Boden *et al*[23] They showed that 20% of asymptomatic subjects over 60 years old had spinal stenosis on MRI. Haig *et al* [36]also showed in their study that imaging could not differentiate symptomatic individuals from asymptomatic individuals as regards spinal stenosis, Spinal canal stenosis was however observed to significantly correlate with the provisional diagnosis of the patients in this study.

Lateral recess narrowing/nerve root compression were observed in 73(48.7%) of participants. there was statistically significant correlation with the provisional diagnosis of the patients. (p=0.030. Ligamentum flavum hypertrophy was present in 71(47.3%) participants .and was worse at L4/L5 level. This was similar to findings in other studies. Facet joint arthropathy was present in 71(47.3%) participants .and was worse at L4/L5 level. This was similar to findings in other studies[7,11,37]. Of the 71 participants who had facet joint arthropathy, 8(11.3%) had it on only right side, 6(8.4%) had on only left side while majority 57(80.3%) had on both sides. More participants with FJA had chronic LBP, had almost same occurrence in patients with both mild and severe LBP. There was statistically significant association between FJA and difficulty in walking. FJA, LFH (p=0.0001), disc herniation (p=0.0001) and spinal canal stenosis (p=003).

Modic vertebral body changes were seen in 69 (46% of total participants). A greater number, had Modic I changes followed by Modic II changes while only one participant had Modic III changes. Both L4 and L5 vertebrae equally had the highest number, followed by L2 S1 and L1 vertebrae. However, it was expected that Modic type 1, probably a reflection of edema, hyper vascularization, and inflammation, would be strongly associated with acute pain. This was similar to studies done by Modic *et al*[38]Kjaer *et al*[26] reported that most degenerative disc abnormalities were moderately associated with low back pain. The strongest associations were noted in Modic changes and anterolisthesis. Kjaer *et al*[26] also suggested that Modic changes constituted the crucial element in the degenerative process and the disc in relation to low back pain and clinical findings where degenerative disc disease with Modic changes was much frequently associated with clinical symptoms. Most authors agreed that, among Modic changes, Type 1 changes were most commonly found in patients with low back pain[26,39,40] contrary to the finding in the study by Ebubedike*et al*[41] where Modic Type 3 was the most common and Type 1 the least. No definite reason was found to account for this difference. Mitral *et al*[42] found a positive trend between the evolution of Type 1 Modic changes into Type 2 changes and the improvement of pain symptom. In addition, they observed that patients in whom Type 1 changes increased were clinically worsened. This may be due to the fact that Type1 Modic changes depicted an ongoing acute inflammatory process.

Incidental findings of vertebral hemangioma in 1 patient (i.e. 0.7% of total patients), scoliosis in 4 patients (i.e. 2.7% of total patients), laminar fracture in 1 patient (i.e. 0.7% of total patients), ependymoma in 2 patients (i.e. 1.4% of total patients) and spinal cord syrinx in 1 patient (i.e. 0.7% of total patients), multiple uterine fibroid, follicular cysts, bilateral renal cortical cysts, pulmonic disease, in 1 patient (i.e. 0.7% of total patients) respectively were found. This further proved that in a small percentage of patients, LBP can be caused by pathologies other than degenerative disease of the spine as seen in other studies[43,44].

CONCLUSION

There was positive correlation between MRI findings of disc protrusion, disc extrusion with sciatica; difficulty in walking and urinary incontinence as well as with provisional diagnosis of patients of which LBP with radiculopathy was the commonest.

Recommendations

The use of MRI in the management of lumbosacral spine abnormalities in patient with low back pain and other spine abnormalities should be encouraged due to the invaluable role of MRI in providing valuable information and the precise localization of the underlying pathology causing the clinical features the patients presents with.Government at all levels should facilitate improvement of management of spinalabnormalities by making MRI scanners more available and accessible in tertiary and possibly secondary healthcare centers. Increased availability will lead to reduced cost and therefore increase the use by both clinicians as well as patients ultimately leading to improved patient management.

TABLE 1: CLINICAL FEATURES OF LOW BACK PAIN IN PARTICIPANTS

Variables	Frequency (n=150)	Percent (%)
Duration of LBP		
0-4weeks (Acute LBP)	5	3.3
5-12weeks (Subacute LBP)	25	16.7
>12weeks (Chronic LBP)	120	80.0
Severity of LBP		
Mild (0-4)	2	1.3
Moderate (5-7)	35	23.3
Severe (8-10)	113	75.4
Sciatica	131	87.3
Right lower limb	33	22.0
Left lower limb	33	22.0
Both	65	43.3
None	19	12.7
Other neurologic symptoms.	80	53.0
Difficulty in walking	42	28.0
Paraesthesia	77	51.3
Limb weakness	14	9.3
Urinary incontinence	9	6.0
Fecal incontinence		

Table 2: PROVISIONAL DIAGNOSIS OF PARTICIPANTS

PROVISIONAL DIAGNOSIS	FREQUENCY/ (%)
LBP? Cause	40(26.7)
Lumbar spondylosis	31(20.7)
Radiculopathy	41(27.3)
Paraesthesia/spinal stenosis	21(14.0)
Urinary fecal incontinence	2(1.3)
Spondylolisthesis	3 (2.0)
Disc prolapse	5 (3.3)
Potts spondylitis	1 (0.7)
Transverse myelitis	4 (2.7)
Others	2 **(**1.3)

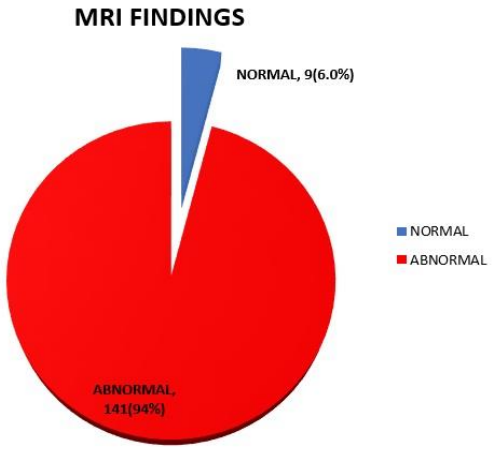

Figure1: Pie chart showing the proportion of normal and abnormal MRI findings in participants with low back pain.

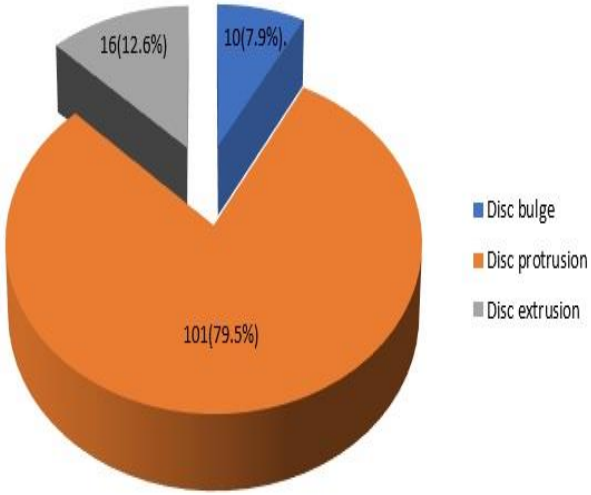

Figure 2: Pie chart showing the types of disc herniation

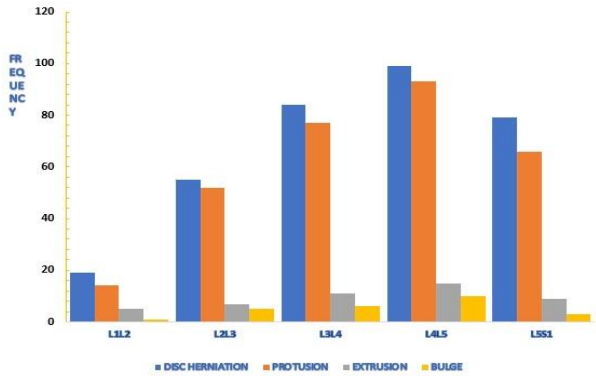

Figure 3: Types of disc herniation at different vertebral levels.

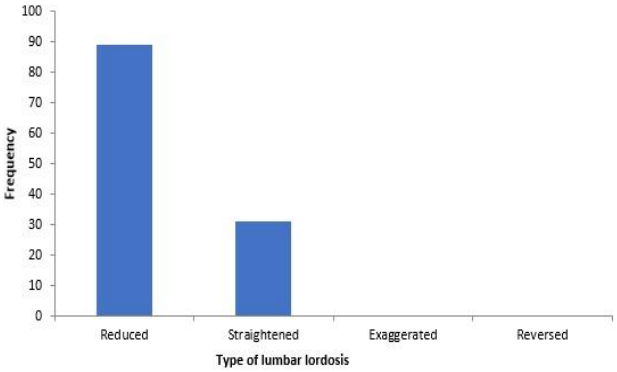

Figure 4: Bar chart showing lumbar lordotic curve in participants

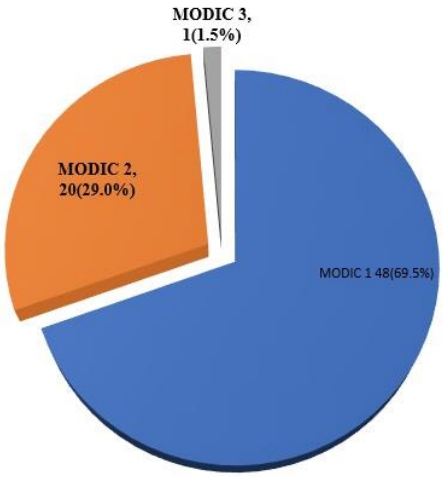

Figure 5: Pie chart showing the types of Modic changes in participants

TABLE3:MRI Findings Of Vertebral AbnormalitiesInParticipants With Low Back Pain.

VERTEBRAL ABNORMALITIES	L1	L2	L3	L4	L5	S1	χ^2	P value
Modic changes	14(9.3)	24(16.0)	30(20.0)	38(25.3)	38(25.3)	22(14.7)	20.003	0.001*
Vertebral osteophytes	22(14.7)	56(37.3)	68(45.3)	79(52.7)	84(56.0)	54(36.0)	69.131	0.0001*
Schmorl's nodes	8(5.3)	21(14.0)	24(16.0)	23(15.3)	18(12.0)	28(18.7)	13.502	0.019*
Collapsed vertebra	3(2.0)	1(0.7)	4(2.7)	5(3.3)	4(2.7)	1(0.7)	4.762	0.458

*= p-value ≤ 0.05 (statistically significant), χ^2 = Chi-square value, *p* value
Values were calculated with total participants. (n=150 (%))
L= intervertebral disc level. with multiple responses as MRI finding were observed in more than one level,.

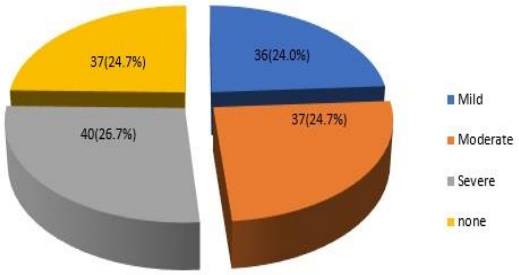

Figure 6: Pie chart showing central canal stenosis in participants

TABLE4: Spinal Canal, Lateral Recess And Foraminal Narrowing In Participants With Low Back Pain.

Canal Narrowing	L1/L2	L2/L3	L3/L4	L4/L5	L5/S1	χ^2	p-value
Spinal canal stenosis	19(12.7)	56(37.3)	86(57.3)	105(70.0)	84(756.0)	119.839	0.0001*
Lateral recess narrowing/nerve root compression	20(13.4)	38(26.7)	49(41.3)	60(44.7)	45(30.0)	43.289	0.0001*
Foraminal narrowing	9(10.7)	26(31.0)	56(66.7)	54(64.3)	39(46.4)	56.129	0.0001*
Ligamentum flavum hypertrophy	7(4.7)	19(12.7)	42(28.0)	46(30.7)	17(23.9)	53.034	0.0001*
Facet joint arthropathy	20(13.3)	38(25.3)	49(32.7)	60(40.0)	45(30.0)	28.973	0.0001

*= p-value ≤ 0.05 (statistically significant), χ^2 = Chi-square value, *p* value
Values were calculated with total participants. (n=150 (%))
L= intervertebral disc level with multiple responses as MRI finding were observed in more than one level,

TABLE 5: Correlation Between Clinical Features, Disc Abnormalities In Participants With Low Back Pain

Clinical features	Disc desiccation (n=126)	Annular fissure	Disc herniation (n=126)	Disc bulge	Disc protrusion	Disc extrusion	Total (Number of responses)
Duration of LBP							
Acute	5 (4.0)	2 (7.4)	5 (3.9)	0 (0.0)	4 (4.0)	1 (6.3)	17 (4.2)
Subacute	20 (15.9)	3 (11.1)	21 (16.5)	1 (10.0)	17 (16.8)	3 (18.8)	65 (16.0)
Chronic	101 (80.2)	22 (81.5)	101 (79.5)	9 (90.0)	80 (79.2)	12 (75.0)	325 (79.9)
χ^2	1.252	2.258	0.937	0.777	0.394	0.560	1.804
p value	0.535	0.535	0.626	0.678	0.821	0.756	0.406
r	0.022	0.025	0.051	-0.072	0.041	0.055	0.018
p value	0.832	0.831	0.656	0.533	0.729	0.594	0.590
Severity of LBP							
Mild	2 (1.6)	0 (0.0)	1 (0.8)	0 (0.0)	1 (1.0)	0 (0.0)	4 (1.0)
Moderate	28 (22.2)	2 (2.0)	31 (24.4)	1 (10.0)	26 (25.7)	4 (25.0)	92 (22.6)
Severe	96 (76.2)	25 (92.6)	95 (74.8)	9 (90.0)	74 (73.3)	12 (75.0)	311 (76.4)
χ^2	0.874	5.320	2.293	1.265	1.218	0.260	0.969
(*p* value)	0.646	0.070	0.318	0.179	0.123	0.878	0.616
r	-0.030	-0.188	-.001	-0.091	0.053	-0.007	-0.028
p value	0.812	0.022	1.000	0.266	0.521	1.000	0.432

TABLE 6: Correlation between Clinical Features, Vertebral Abnormalities in Participants with Low Back Pain

Clinical features	Vertebral osteophyte (n=94)	Ligamentum flavum hypertrophy (n=71)	Facet joint arthropathy (n=71)	Spondylolisthesis (n=58)	Modic Changes	Spinal canal stenosis	Lateral recess narrowing	Total (Number of responses)
LBP								
Acute	4(4.3)	1(1.4)	2(2.8)	3(5.2)	2(2.9)	3(2.7)	4(5.5)	19 (3.5)
Subacute	17(18.1)	10(14.1)	9(12.7)	8(13.8)	11(15.9)	17(15.0)	14(19.2)	86 (15.7)
Chronic	73(77.7)	60 (84.5)	60(84.5)	47(81.0)	56(81.2)	93(82.3)	55(75.3)	444 (80.9)
χ^2	1.118	2.380	1.738	1.441	0.134	1.659	2.889	0.859
p value	0.527	0.304	0.419	0.487	0.935	0.436	0.236	0.651
r	0.085	-0.123	-0.096	-0.013	-0.030	-0.105	0.134	-0.016
p value	0.317	0.134	0.256	1.000	0.718	0.252	0.139	0.619
LBP Severity								
Mild	1(1.1)	1(1.4)	1(1.4)	0(0.0)	1(1.4)	1(0.9)	1(1.4)	6 (1.1)
Moderate	26(27.7)	13(18.3)	12(16.9)	13(22.4)	12(17.4)	21(18.6)	13(17.8)	110 (20.0)
Severe	67(71.3)	57(80.3)	58(81.7)	45(77.6)	56(81.2)	91(80.5)	59(80.8)	433 (78.9)
χ^2	2.707	1.902	0.3119	1.359	2.522	6.762	2.431	1.359
(*p* value)	0.258	0.386	0.210	0.507	0.283	**0.034***	0.297	0.507
r	0.105	-0.099	-0.127	-0.061	-0.113	-0.211	-0.113	-0.08
p value	0.202	0.296	0.163	0.482	0.222	**0.014***	0.223	0.007

* = p-value ≤0.05 (statistically significant),.χ^2 = Chi-square value, *p* value = probability value, *r* = Correlation coefficient

TABLE 7: Correlation between Clinical Features and Disc Abnormalities In Participants

Clinical features	Disc desiccation (n=126)	Annular fissure	Disc herniation (n=126)	Disc bulge	Disc protrusion	Disc extrusion	Total (Number of responses)
Sciatica	110(87.3)	21(77.8)	112(88.2)	9(90.0)	93(92.1)	10(62.5)	355 (87.2)
χ^2	0.001	2.718	0.548	0.69	6.295	9.985	0.008
p value	0.979	0.099	0.459	0.793	**0.012***	**0.002***	1.000
r	-0.002	-0.135	0.060	0.021	0.205	0.258	–0.003
p value	1.000	0.114	0.496	0.795	**0.012***	**0.007***	1.000
Difficulty in walking	71(56.3)	20(74.1)	70(55.1)	3(30.0)	61(60.4)	6(37.5)	231 (56.8)
χ^2	2.878	5.691	1.060	2.344	6.197	1.804	3.499
(*p* value)	0.090	**0.020***	0.303	0.126	**0.013***	0.179	0.061
r	0.139	0.195	0.804	–0.125	0.203	–0.110	0.062
p value	0.118	**0.020***	0.366	0.190	**0.015***	0.197	0.070
Urinary incontinence	13(10.3)	4(14.8)	13(10.2)	0(0.0)	13(12.9)	0(0.0)	43 (10.6)
χ^2	0.901	1.169	0.798	1.103	4.573	1.844	1.332
(*p* value)	0.342	0.280	0.372	0.294	**0.032***	0.175	0.248
r	0.780	0.088	0.073	–0.086	0.175	–0.111	0.038
p value	0.470	0.466	0.476	0.599	**0.036***	0.365	0.252
Fecal incontinence	9(7.1)	2(7.4)	9(7.1)	0(0.0)	8(7.8)	1(6.3)	29 (7.1)
χ^2	1.824	0.116	1.734	0.684	2.023	0.002	1.668
(*p* value)	0.177	0.734	0.188	0.408	0.155	0.964	0.196
r	0.110	0.028	0.108	–0.068	0.116	0.004	0.043
p value	0.356	1.000	0.356	0.640	0.272	1.000	0.207
Provisional diagnosis							
LBP? Cause	32(25.4)	8(29.6)	32(25.4)	1(10.0)	29(28.7)	2(12.5)	104(25.5)
Lumbar spondylosis	28(22.2)	6(22.2)	28(22.2)	1(10.0)	25(24.8)	3(18.8)	92(22.6)
Radiculopathy	33(26.2)	5(18.5)	33(26.2)	6(60.0)	25(24.8)	2(12.5)	104(25.6)
Paraesthesia/spinal stenosis	19(15.1)	4(14.8)	19(15.1)	1(10.0)	12(11.9)	6(37.5)	61(15.0)
Urinary fecal incontinence	2(1.6)	1(3.7)	2(1.6)	0(0.0)	2(2.0)	0(0.0)	7(1.7)
Spondylolisthesis	3(2.4)	1(3.7)	3(2.4)	0(0.0)	3(3.0)	0(0.0)	10(2.5)
Disc prolapsed	5(4.0)	2(7.4)	5(4.0)	0(0.0)	3(3.0)	2(12.5)	17(4.2)
Potts spondylitis	1(0.8)	0(0.0)	0(0.0	0(0.0)	0(0.0	0(0.0)	1(0.2)
Transverse myelitis	2(1.6)	0(0.0)	2(1.6)	1(10.0)	0(0.0	1(6.3)	6(1.5)
Others	1(0.8)	0(0.0)	2(1.6)	0(0.0)	2(0.0)	0(0.0)	5(1.2)
χ^2	9.686	6.140	15.050	9.101	18.552	16.219	12.268
(*p* value)	0.367	0.726	0.105	0.428	**0.029***	0.062	**0.012***
r	0.039	0.014	-0.018	-0.066	0.161	-0.157	0.015
p value	0.658	0.834	0.338	0.467	**0.049***	0.059	0.662

* = p-value ≤0.05 (statistically significant), χ^2 = Chi-square value, *p* value = probability value, *r* = Correlation coefficient

TABLE 8: Correlation between Clinical Features, Vertebral Abnormalities Canal Narrowing

Clinical features	Vertebral osteophyte(n=94)	Ligamentum flavum hypertrophy (n=71)	Facet joint arthropathy(n=71)	Spondylolisthesis (n=58)	Modic Changes	Spinal canal stenosis	Lateral recess narrowing	Total (Number of responses)
Sciatica	85(90.4)	61(85.9)	63(88.7)	53(91.4)	62(89.8)	96(85.0)	65(89.0)	485(88.3)
χ^2	2.176	0.254	0.239	1.399	0.735	2.341	0.375	1.059
(*p* value)	0.140	0.621	0.625	0.237	0.391	0.126	0.540	**0.044***
r	0.120	-0.040	0.040	0.097	0.070	-0.125	0.050	0.032
p value	0.203	0.633	0.806	0.316	0.465	0.161	0.627	**0.039***
Difficulty in walking	59(62.8)	39(54.9)	44(62.0)	36(62.1)	39(56.5)	59(52.2)	44(60.3)	320 (58.3)
χ^2	9.001	0.138	4.042	2.899	0.522	0.231	2.752	11.348
(*p* value)	**0.003***	0.710	**0.044***	0.089	0.470	0.631	0.097	**0.001**
r	0.0245	0.030	0.164	0.139	0.059	-0.039	0.135	0.104
p value	**0.003***	0.745	**0.050***	0.096	0.514	0.706	0.105	**0.001**
Urinary incontinence	11(11.7)	8(11.3)	7(9.9)	5(8.6)	7(10.1)	12(10.6)	9(12.3)	59(10.7)
χ^2	1.670	0.596	0.044	0.057	0.099	0.895	1.508	2.717
(*p* value)	0.196	0.440	0.834	0.812	0.752	0.344	0.219	0.099
r	0.106	0.063	0.017	-0.019	0.026	0.077	0.100	0.051
p value	0.253	0.576	1.000	1.000	0.785	0.519	0.268	0.111
Fecal incontinence	7(7.4)	4(5.6)	4(5.6)	5(8.6)	6(8.7)	5(4.4)	6(8.2)	37(6.7)
χ^2	0.934	0.032	0.032	1.152	1.646	2.015	1.242	1.116
(*p* value)	0.334	0.858	0.858	0.283	0.199	0.156	0.265	0.060
r	0.079	-015	-015	0.088	0.105	-0.116	0.091	0.033
p value	0.485	1.000	1.000	0.309	0.202	0.225	0.268	0.301
Provisional diagnosis								
LBP? Cause	24(25.5)	19(26.8)	18(25.4)	13(22.4)	15(21.7)	29(25.7)	12(16.4)	130 (23.7)
Lumbar spondylosis	1920.2)	20(28.2)	16(22.5)	15(25.9)	14(20.3)	28(24.8)	19(26.0)	131 (23.9)
Radiculopathy	28(29.8)	25(61.0)	20(28.2)	17(29.3)	19(27.6)	30(26.5)	18(24.7)	148 (27.0)
Paraesthesia/spinal stenosis	14(14.9)	16(22.5)	12(16.9)	8(38.1)	14(20.3)	15(71.4)	15(20.5)	86 (15.7)
Urinary fecal incontinence	1(1.1)	8(11.3)	0(0.0)	1(1.7)	1(1.4)	1(0.9)	2(2.7)	7 (1.3)
Spondylolisthesis	1(1.1)	1(1.4)	2(2.8))	2(3.4)	1(1.4)	3(2.7)	1(1.4)	13 (2.4)
Disc prolapsed	4(4.3)	3(4.2)	2(2.8)	2(3.4)	3(4.3)	5(4.4)	4(5.5)	24 (4.4)
Potts spondylitis	0(0.0)	4(5.6)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0 (0.0)
Transverse myelitis	3(3.2)	0(0.0)	1(1.4)	0(0.0)	2(2.9)	0(0.0)	1(1.4)	7 (1.3)
Others	0(0)	0(0.0	0(0.0)	0(0.0	0(0.0))	2(1.8)	1(1.4)	3 (0.5)
χ^2	8.023	17.302	7.012	7.531	7.967	23.432	18.487	37.769
(*p* value)	0.532	**0.044***	0.636	0.582	0.537	**0.005***	**0.030***	**0.000**
r	0.036	0.110	0.090	0.080	-0.037	0.120	-0.099	0.040
p value	0.675	0.180	0.289	0.332	0.648	0.155	0.237	0.198

* = p-value ≤0.05 (statistically significant), χ^2 = Chi-square value, *p* value = probability value, *r* = Correlation coefficient

REFERENCES

1) Norasteh AA. LOW BACK PAIN. Norasteh AA, editor. Rijeka Croatia: Janeza Trdine Croatia; 2012. 12–19 p.
2) Delitto A, George SZ, Van Dillen LR, Whitman JM, Sowa G, Shekelle P, et al. Low back pain: Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association. J Orthop Sports Phys Ther [Internet]. 2012 Apr [cited 2017 Sep 18];42(4). Available from: http://www.ncbi.nlm.nih.gov/pubmed/22466247
3) Church EJ, Odle TG. Diagnosis and treatment of back pain. [Internet]. Vol. 79, Radiologic technology. 2007 [cited 2017 Sep 19]. p. 1–25. Available from: http://adxcorp.net/wp-content/uploads/2012/articles/article_1.pdf
4) Watson N (Nicholas A., Chapman S. Chapman and Nakielny's guide to radiological procedures. Saunders; 2014.
5) Roudsari B, Jarvik JG. Lumbar spine MRI for low back pain: Indications and yield. Am J Roentgenol. 2010;195(3):550–9.
6) Adekanmi A, Bello T, Atalabi O, Jimoh K, Ogunseyinde O. Magnetic resonance imaging of lumbosacral intervertebral discs in nigerians with low back pain. West African J Radiol [Internet]. 2017 [cited 2017 Sep 19];24(1):61. Available from: http://www.wajradiology.org/text.asp?2017/24/1/61/192753
7) Yong PY, Alias NAA, Shuaib IL. Correlation of Clinical Presentation , Radiography , and Magnetic Resonance Imaging for Low Back Pain — a Preliminary Survey. J HK Coll Radiol. 2003;(6):144–51.
8) Ng LCL, Tafazal S, Sell P. The effect of duration of symptoms on standard outcome measures in the surgical treatment of spinal stenosis. Eur Spine J. 2007 Feb;16(2):199–206.
9) Shambrook J, McNee P, Harris EC, Kim M, Sampson M, Palmer KT, et al. Clinical presentation of low back pain and association with risk factors according to findings on magnetic resonance imaging. Pain. 2011 Jul;152(7):1659–65.
10) Saleem S, Aslam HM, Rehmani MA khan, Raees A, Alvi AA, Ashraf J. Lumbar Disc Degenerative Disease: Disc Degeneration Symptoms and Magnetic Resonance Image Findings. Asian Spine J. 2013;7(4):322.
11) Rayoffor O, Nwankwo N, Ugboma E, Rayoffor E. Evaluation of magnetic resonance imaging findings in adult patients with nontraumatic low back pain in South-South Nigeria. West African J Radiol [Internet]. 2016 [cited 2017 Sep 19];23(2):64. Available from: http://www.wajradiology. org/text.asp? 2016/23/2/ 64/ 164 871
12) Uduma FU, Ongolo P, Assam G, Fokam P, Motah M. Evaluation of Pattern of Magnetic Resonance Images of Lumbo-Sacral Spine in Cameroon - A Pioneer Study. Glob J Med Res. 2011;11(2).
13) Suthar P, Patel R, Mehta C, Patel N. MRI evaluation of lumbar disc degenerative disease. J Clin Diagn Res [Internet]. 2015 Apr [cited 2018 Jan 17];9(4):TC04-9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26023617
14) J. T, J. K, J. N, S. T, S. N, M.-R. J, et al. Prevalence of degenerative imaging findings in lumbar magnetic resonance imaging among young adults. Spine (Phila Pa 1976) [Internet]. 2009;34(16):1716–21. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L355118373%0Ahttp://dx.doi.org/10.1097/BRS.0b013e3181ac5fec%0Ahttp://vu.on.worldcat.org/atoztitles/link?sid=EMBASE&issn=03622436&id=doi:10.1097%2FBRS.0b013e3181ac5fec&atitle=Prevalen

15) Strand S. Statistics for research: With a guide to SPSS, 2nd edition. Br J Educ Psychol. 2007;77:497.
16) Chan ST, Fung PK, Ng NY, Ngan TL, Chong MY, Tang CN, et al. Dynamic changes of elasticity, cross-sectional area, and fat infiltration of multifidus at different postures in men with chronic low back pain. Spine J. 2012 May;12(5):381–8.
17) Sakai Y. Low back pain pathogenesis and treatment. InTech. 2012. 34–36 p.
18) Teraguchi M, Yoshimura N, Hashizume H, Muraki S, Yamada H, Oka H, et al. The association of combination of disc degeneration, end plate signal change, and Schmorl node with low back pain in a large population study: The Wakayama Spine Study. Spine J. 2015 Apr 1;15(4):622–8.
19) Udby PM, Bendix T, Ohrt-Nissen S, Lassen MR, Sørensen JS, Brorson S, et al. Modic Changes Are Not Associated with Long-term Pain and Disability: A Cohort Study with 13-year Follow-up. Spine (Phila Pa 1976). 2019 Sep 1;44(17):1186–92.
20) Garrido E, Connaughton PN. Unilateral facetectomy approach for lateral lumbar disc herniation. In: Journal of Neurosurgery. 1991. p. 754–6.
21) Boos N, Dreier D, Hilfiker E, Schade V, Kreis R, Hora J, et al. Tissue characterization of symptomatic and asymptomatic disc herniations by quantitative magnetic resonance imaging. J Orthop Res [Internet]. 1997 Jan [cited 2017 Sep 20];15(1):141–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/9066539
22) Jensen GM. Biomechanics of the lumbar intervertebral disk: a review. Phys Ther [Internet]. 1980 Jun [cited 2017 Sep 15];60(6):765–73. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6445569
23) Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal Lumbar Magnetic-Resonance Spine Scans of the in Asymptomatic. J Bone Jt Surg [Internet]. 1990 [cited 2019 Jun 27];72(3):403–8. Available from: https://link.springer.com/chapter/10.1007/978-1-4471-5451-8_60
24) Kerr MS, Frank JW, Shannon HS, Norman RWK, Wells RP, Neumann WP, et al. Biomechanical and Psychosocial Risk Factors for Low Back Pain at Work. Am J Public Health [Internet]. 2001 [cited 2017 Sep 18];91(7):1069–1075. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1446725/pdf/11441733.pdf
25) Masui T, Yukawa Y, Nakamura S, Kajino G, Matsubara Y, Kato F, et al. Natural history of patients with lumbar disc herniation observed by magnetic resonance imaging for minimum 7 years. J Spinal Disord Tech. 2005 Apr;18(2):121–6.
26) Kjaer P, Leboeuf-Yde C, Korsholm L, Sorensen JS, Bendix T. Magnetic resonance imaging and low back pain in adults: A diagnostic imaging study of 40-year-old men and women. In: Spine. 2005. p. 1173–80.
27) Dongfeng R, Hou S, Wu W, Wang H, Shang W, Tang J, et al. The expression of tumor necrosis factor-α and CD68 in high-intensity zone of lumbar intervertebral disc on magnetic resonance image in the patients with low back pain. Spine (Phila Pa 1976). 2011 Mar 15;36(6).
28) Tonosu J, Oka H, Matsudaira K, Higashikawa A, Okazaki H, Tanaka S. The relationship between findings on magnetic resonance imaging and previous history of low back pain. J Pain Res [Internet]. 2016 Dec 29 [cited 2019 Mar 15];Volume 10:47–52. Available from: https://www.dovepress.com/the-relationship-between-findings-on-magnetic-resonance-imaging-an-peer reviewed-article-JPR

29) Videman T, Battié MC, Gibbons LE, Maravilla K, Manninen H, Kaprio J. Associations Between Back Pain History and Lumbar MRI Findings. Spine (Phila Pa 1976). 2003;28(6):582–8.

30) Kalichman L, Hunter DJ. Diagnosis and conservative management of degenerative lumbar spondylolisthesis. Eur Spine J. 2008 Mar;17(3):327–35.

31) Modic MT, Ross JS. Lumbar Degenerative Disk Disease. Radiology [Internet]. 2007 Oct [cited 2018 Jan 19];245(1):43–61. Available from: http://www.ncbi.nlm.nih. gov / pubmed/17885180.

32) Fortin JD, Wheeler MT. Imaging in Lumbar Spinal Stenosis. Pain Physician. 2004;7(1):133–9.

33) Divya Premchandran Vasudha V Saralay, Mahale Ajith. Predicting Lumbar Central Canal Stenosis. 2014;8:11. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4290304/

34) Szpalski M, Gunzburg R. Lumbar spinal stenosis in the elderly: An overview. Vol. 12, European Spine Journal. 2003.

35) Atlas SJ, Deyo RA. Evaluating and managing acute low back pain in the primary care setting. J Gen Intern Med. 2001 Feb;16(2):120–31.

36) Haig AJ, Sigmundsson FG, Strömqvist B, Jönsson B. Correlation between disability and MRI findings in lumbar spinal stenosis. Acta Orthop [Internet]. 2011 Oct 24 [cited 2017 Oct 25];82(5):637–8. Available from: http://www.tandfonline.com/doi/full/10.3109/17453674.2011.627496

37) Hussaini S, Karimi N, Ezzati K, Hossein Zadeh S, Rahnama L, Arslan S. Reliability of Magnetic Resonance Imaging Findings Interpretation in Patients with Lumbar Disk Herniation. Phys Treat Specif Phys Ther [Internet]. 2015 [cited 2018 Mar 31];5(2). Available from: https://pdfs.semanticscholar.org/45e3/c206a568c29a7c706f71734503cab3b02235.pdf

38) Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. Radiology [Internet]. 1988 Jan [cited 2018 Feb 2];166(1):193–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/3336678

39) Cassar-Pullicino VN. MRI of the ageing and herniating intervertebral disc. Eur J Radiol [Internet]. 1998;27(3):214–28. Available from: http://www.ncbi.nlm.nih.gov/pubmed/9717637

40) Toyone T, Takahashi K, Kitahara H, Yamagata M, Murakami M, Moriya H. VERTEBRAL BONE-MARROW CHANGES IN DEGENERATIVE LUMBAR DISC DISEASE AN MRI STUDY OF 74 PATIENTS WITh LOW BACK PAIN. J BoneJoint SurgBr] [Internet]. 1994 [cited 2018 Mar 10];76(76):757–64. Available from: https://pdfs.semanticscholar.org/32be/e361a592e17303714140413933feac663cc8.pdf

41) Ebubedike U, Umeh E, Ogbole G, Ndubuisi C, Mezue W, Ohaegbulam S. Pattern of lumbosacral magnetic resonance imaging findings in diagnosed cases of disc degenerative disease among Nigerian adults with low back pain. West African J Radiol [Internet]. 2016 [cited 2019 Jun 16];24(1):25. Available from: http://www.wajradiology.org/text.asp?2017/24/1/25/192752

42) Mitra D, Cassar-Pullicino VN, Mccall IW. Longitudinal study of vertebral type-1 end-plate changes on MR of the lumbar spine. Eur Radiol. 2004 Sep;14(9):1574–81.

43) Galukande M, Muwazi S, Mugisa DB. Aetilogy of low back pain Mulago Hospital , Uganda Aetiology of low back pain in Mulago Hospital , Uganda. Afr Health Sci. 2005;5(2):164–7.

44) Bratton R. Assessment and management of acute low back pain. Am Fam Physician [Internet]. 1999 [cited 2019 Jun 27]; 60(8): 2299–308. Available from: https:// europepmc. org/ abstract/med/10593321