Original Article

Radiology

Disk Degeneration in Adolescence and Young Adults: A Cross-Sectional Magnetic Resonance Imaging Study

1. Abdul Sattar 2.Asif Shahzad 3.Sadia Anjum, 4.Liaqat Ali Jalal

1. Assoc. Prof. of Radiology 2, 3. PG Residents, Dept. of Radiology 4. Senior Registrar of Neurosurgery, Nishtar Medical College & Hospital, Multan

ABSTRACT

Objective: To investigate the frequency and patterns of Magnetic Resonance Imaging (MRI) for disk degeneration in adolescence and young adults, in lumbosacral spine.

Study Design: Cross-sectional study

Place and Duration of Study: This study was carried out at Department of Diagnostic Imaging in Nishtar Hospital, Multan during January 2012 to December 2012.

Materials and Methods: One hundred patients presented with low back pain and were advised MRI, participated in the study. Demographics were assessed by using a questionnaire and their lumbar disk T2 maps were quantified via sagittal imaging protocols at 1.5T.MRI was reported by two radiologists separately. Data was analyzed by using SPSS (v. 16) as well as manually. *P* values less than 0.05 were considered significant.

Results: The frequency of disk degeneration was found to be 44%. Mean age of the patients was 19.47 ± 2.19 years. Mean duration of pain was 6.93 ± 1.49 months. Most patients (53%) have multilevel involvement with 31% having disease at L4-L5 level. Other significant MRI findings included disc bulge (central 46%, paracentral 29%), thecal sac compression 75% and exiting nerve root compression in 54% along with ligamentus hypertrophy 19%. The statistically significant association ($p < 0.009$) between desicatory changes & nerve root compression and between Pfirrmann grades & radiculopathy ($p < 0.01$) were noted.

Conclusion: Frequency of disco-vertebral degenerative disease is significant in juveniles presenting with low back pain. So, any patient especially with chronic low back pain should undergo MRI spine for early diagnosis of degenerative disease. Moreover multilevel involvement and signs of radiculopathy are also significant.

Key Words: Disc Degeneration, Magnetic Resonance imaging, Juveniles

INTRODUCTION

The term degeneration includes a wide variety of clinical, radiological and pathological manifestations. Typically Intervertebral disk degeneration has been attributed to age progression and excessive physical loading which contribute to structural compromise and biochemical degradation of the disk[1].

The real or apparent desiccation, narrowing of the disk space, fibrosis, diffuse bulging of the annulus beyond the disk space, extensive fissuring (ie, numerous annular tears), defects and sclerosis of the endplates and osteophyte at the vertebral apophyses are taken as the features of degeneration[2].

The role of imaging is to provide accurate morphologic information and influence therapeutic decision making. Radiography and conventional CT are the common imaging methods for evaluation of disk morphology and reactive bony changes[3]. MRI is useful in evaluation of Intervertebral disk degeneration. MR imaging can reflect both the macromolecular concentrations and the structural integrity in the Intervertebral disk and has been considered the best noninvasive method[4]. At MRI these changes are manifested as loss of signal intensity from disc seen on T2W images, disc herniation, annulus tear, endplate changes, osteophyte formation etc.

The decreased signal intensity reflects decreased water and proteoglycan concentrations caused by degeneration[5]. Transverse relaxation time (T2) mapping has the potential to quantitatively evaluate deterioration of the molecular composition and structural integrity of Intervertebral disks[6]. T2 is sensitive to water content and the arrangement of the collagen network structure.A high T2 for the nucleus pulposus has been shown in healthy Intervertebral disks; T2 decreases with the decrease of water content associated with disk degeneration[7]. As degeneration progresses, uniformity of T2 in both the nucleus pulposus and the annulus fibrosus will decrease and, finally, the distinction of signal intensity between the nucleus pulposus and the annulus fibrosus will be lost[8]. Numerous studies have revealed that degenerating Intervertebral disks have shorter T2 values than normal disks and that T2 correlate with the Pfirrmann grades[10].

The aims of this study were to investigate the frequency and patterns of Magnetic Resonance Imaging (MRI) for disk degeneration in adolescence and young adults, in lumbosacral spine.

MATERIALS AND METHODS

This cross-sectional study was carried out in department of radiology Nishtar Medical College &

Hospital, Multan in one year from January 2012 to December 2012 over one hundred patients. Socio-demographics such as age, gender and pain duration were collected. Those patients who have age range between 13 to 22 years and having low back pain of ≥ 5 months were included in the study, while the patients having any spinal deformity like scoliosis or kyphosis, any spine related surgery or history of major trauma related to spine were excluded from the study.

T2 weighed sagittal and axial Magnetic Resonance Imaging (MRI) of lumbo-sacral spine was obtained using departmental protocol on 1.5T. The images were then evaluated by consultant radiologist. The desicatory change, annulus tear, disk bulge, schmorl's nodes, marrow changes, thecal sac compression, lateral recess narrowing, ligamentus hypertrophy, facet joint arthrosis and nerve root compression were noted.

The loss of signal intensity from nucleus pulposus was given the score ranging from 0 to 3 based on Schneiderman et al criteria. A score of 0 was associated with no signal changes for the disk, a score of, 1 was assigned when a slight decrease in signal intensity of the nucleus pulposus was present, a score of 2 was associated with the presence of hypo intensity of the nucleus pulposus and normal disc height, and a score of 3 was reserved for a hypo intense nucleus pulposus with disk space narrowing.

Other MRI patterns noted were, fissuring of disk (annuls tear), morphological defects at endplates (schmorl's nodes), high signal intensity adjacent to the vertebral end plate (marrow changes), loss / thinning of fat signals from recess (recess narrowing), ligamentus signal changes (hypertrophy) and slipping of disk beyond spinal line (disk bulge).

The data was analyzed using SPSS version 16 software.The descriptive and frequency statistics of the data set were ascertained. For categorical cross-tabulation data analyses, Pearson's chi-square and Fisher's exact tests were performed when appropriate. *P* values less than 0.05 were considered significant, and the corresponding 95% confidence intervals (95% CIs) of the *P* values were assessed.

RESULTS

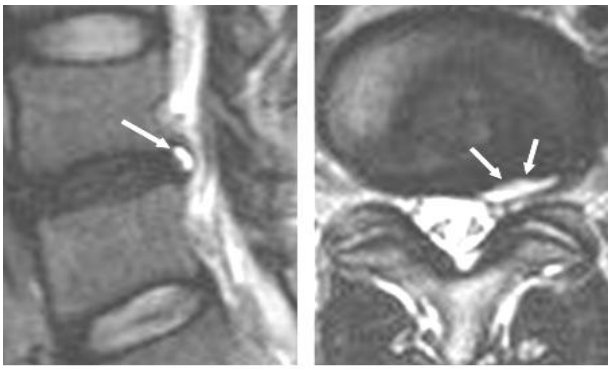
The mean age of the patients was 19.47 ± 2.19 years while mean pain duration was 6.93 ± 1.49 months. Overall 40% patients having evidence of degenerative disease. The common patterns of MRI noted are shown in table I. Among the patients having degenerative disease twenty four percents have grade II disease on Pfirrmann grading system and thirteen percents having grade III disease.

In our study most (52%) patients have multilevel involvement followed by 31% at L4-L5 level and 13% at L5-S1 level. Regarding other levels two patients have involvement at L3-L4 level and only one patient has disease evidence on L2-L3 level. However, we could not found any patient having involvement at L1-L2 level. Other significant findings included signal changes in ligamentus flavum in 19% patients and lateral recess narrowing in 70%.

We found statistically significant association ($p < 0.009$) between desicatory changes & nerve root compression and between Pfirrmann grades & radiculopathy ($p < 0.01$). However we found no significant association between age or pain duration with desicatory changes and annuls tears ($p > 0.10$ & 0.47) and ($p > 0.89$ & 0.38) respectively.

Table No.I: MRI pattern Observed

MRI patterns	Percentages
Desicatory changes	40%
Annulus tear	34%
Disk bulge	
Central	46%
Paracentral	29%
Ligamentus hypertrophy	19%
Thecal sac compression	75%
Nerve root compression	54%



a. b.

Figure No.1: Disc Dessication & Annulus tear in 19 yr old patient

DISCUSSION

Little is known regarding juvenile disk degeneration. It is commonly perceived as wear and tear phenomenon and hence related to age progression. The lumbar spine is subjected to a substantial amount of axial compressive forces, primarily at the level of L4–L5 and L5–S1[21]. Various investigators have reported the high prevalence of disc pathology associated at these levels in comparison with other regions of the lumbar spine[11,12]. In our study we found involvement of L4-L5 level in 31% patients followed by 13% at L5-S1 level.

The Intervertebral disc assumes the role of a "shock absorber" and is subjected to various forces. Therefore, alterations in its biochemical and structural integrity compromise its biomechanical capability and as such alter load accommodation[13,14].

In our study we found 40% patients having evidence of degenerative disease which is comparable to many

international studies having prevalence range from 35 to 42% (15,16). We also found a high percentage 54% of patients having nerve root compression which is quiet alarming in this age group.

With regard to Schmorl's nodes, some authors have suggested that improper or excessive anterior column loading may contribute to such lesions; however, recent evidence suggests that a genetic disposition may exist for the manifestation of Schmorl's nodes. We found schmorl's nodes in 33% patients[17,18].

MR imaging provides a unique means to evaluate the morphologic status of Intervertebral disks and their relationship to neural structures in patients with low back pain. With the use of MR imaging accurate detection of annular tears is possible. The loss of the signal intensity of the central aspect of Intervertebral disks on T2-weighted images is frequently seen in the setting of degenerative disk disease-desicatory change[19,20].

The ability to detect early degeneration of Intervertebral disks may contribute to better understanding of the progression of degeneration seen with age and other risk factors for degenerative disk disease. The ability to detect degeneration in the annulus fibrosus may contribute to investigation of the pathogenesis of lower back pain; primary sensory nerve endings have been found only in the outer annulus fibrosus, and the degeneration is likely to be responsible for disk-related pain. Use of axial T2 mapping may also contribute to evaluation of the effect of conservative and operative treatments for Intervertebral disks, especially for new treatments such as nucleus pulposus replacement, gene therapy, and stem cell transplantation therapy [21,22,23].

Our study has several limitations also. First, neither biochemical nor histological assessment of Intervertebral disks was performed in this study, and therefore the relationship between degenerative grade evaluated by us radiologically and the actual degenerative status of Intervertebral disks is still unclear. Second, signal intensity of Intervertebral disks as evaluated with T2-weighted imaging shows a diurnal variation due to change in the disk water content[24,25]; this is especially true in the nucleus pulposus. To minimize the influence of diurnal variation on T2 in Intervertebral disks, we performed MRI at a fixed time in the afternoon. However further study is needed to evaluate the influence of diurnal variation of T2. Third, BMI of the patients were not collected so, we cannot made an attempt to correlate degenerative grades with obesity levels.

CONCLUSION

Frequency of disco-vertebral degenerative disease is significant in juveniles presenting with low back pain. Moreover multilevel involvement and signs of radiculopathy are also significant. Any patient with chronic low back pain should undergo MRI spine for early diagnosis of degenerative disease regardless of the age.

REFERENCES

1. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG. Classification of age-related changes in lumbar Intervertebral discs: 2002 Volvo Award in basic science. Spine 2002;27:2631–44.
2. Milette PC. Reporting lumbar disk abnormalities: at last, consensus! AJNR Am J Neuroradiol 2001;22:
428–429.
3. Zuo J, Saadat E, Romero A, et al. Assessment of intervertebral disc degeneration with magnetic resonance single-voxel spectroscopy. Magn Reson Med 2009;62:1140–46.
4. Waris E, Eskelin M, Hermunen H, et al. Disc degeneration in low back pain: a 17-year follow-up study using magnetic resonance imaging. **S**pine (Phila Pa **1**976) *2*007;32:681–84.
5. Antoniou J, Pike GB, Steffen T, et al. Quantitative magnetic resonance imaging in the assessment of degenerative disc disease. Magn Reson Med 1998; 40:900–907.
6. Perry J, Haughton V, Anderson PA, Wu Y, Fine J, Mistretta C. The value of T2 relaxation times to characterize lumbar intervertebral disks: preliminary results. Am J Neuroradiol 2006; 27:337-342.
7. Kim DJ, Suh JS, Jeong EK, Shin KH, Yang WI. Correlation of laminated MR appearance of articular cartilage with histology, ascertained by artificial landmarks on the cartilage. J Magn Reson Imaging 1999; 10:57–64.
8. Trattnig S, Stelzeneder D, Goed S, et al. Lumbar intervertebral disc abnormalities: comparison of quantitative T2 mapping with conventional MR at 3.0 T. Eur Radiol 2010;20:2715–22
9. Marinelli NL, Haughton VM, Anderson PA. T2 relaxation times correlated with stage of lumbar intervertebral disk degeneration and patient age. AJNR Am J Neuroradiol 2010;31:1278–82.
10. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine 2001; 26:1873–1878.
11. White AA, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. New York: Lippincott Williams & Wilkins;1990.
12. Hangai M, Kaneoka K, Kuno S, Hinotsu S, Sakane M, Mamizuka N, et al. Factors associated with lumbar intervertebral disc degeneration in the elderly. Spine J 2008;8:732–40.
13. Battie MC, Videman T, Parent E. Lumbar disc degeneration: epidemiology and genetic influences. Spine 2004;29:2679–90.

14. Kuisma M, Karppinen J, Haapea M, Niinimaki J, Ojala R, Heliovaara M, et al. Are the determinants of vertebral endplate changes and severe disc degeneration in the lumbar spine the same? A magnetic resonance imaging study in middle-aged male workers. BMC Musculoskelet Disord 2008;9:51.
15. Spangfort EV. The lumbar disc herniation: a computer-aided analysis of 2,504 operations. Acta Orthop Scand Suppl 1972;142:1–95.
16. Watanabe A, Benneker LM, Boesch C, Watanabe T, Obata T, Anderson SE. Classification of intervertebral disk degeneration with axial T2 mapping. AJR Am J Roentgenol 2007;189:936-42.
17. Adams MA, Freeman BJ, Morrison HP, Nelson IW, Dolan P. Mechanical initiation of intervertebral disc degeneration. Spine 2000; 25:1625–36.
18. Tanaka N, An HS, Lim TH, Fujiwara A, Jeon CH, Haughton VM. The relationship between disc degeneration and flexibility of the lumbar spine. Spine J 2001;1:47–56.
19. Samartzis D, Karppinen J, Mok F, Fong DY, Luk KD, Cheung KM. A population-based study of juvenile disc degeneration and its association with overweight and obesity, low back pain, and diminished functional status. J Bone Joint Surg Am 2011;93(7):662-70.
20. Tertti MO, Salminen JJ, Paajanen HE, Terho PH, Kormano MJ. Low-back pain and disk degeneration in children: a case-control MR imaging study. Radiology
21. Williams FM, Manek NJ, Sambrook PN, Spector TD, MacGregor AJ. Schmorl's nodes: common, highly heritable, and related to lumbar disc disease. Arthritis Rheum 2007;57:855–60.
22. Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. Spine J 2008;8:8–20.
23. Ekman M, Jonhagen S, Hunsche E, Jonsson L. Burden of illness of chronic low back pain in Sweden: a cross-sectional, retrospective study in primary care setting. Spine 2005;30:1777–85.
24. Wenig CM, Schmidt CO, Kohlmann T, Schweikert B. Costs of back pain in Germany. Eur J Pain 2009;13:280–6.
25. Battie MC, Videman T, Parent E. Lumbar disc degeneration: epidemiology and genetic influences. Spine 2004;29:2679–9.

Address for Corresponding Author:
Dr Abdul Sattar
Associate Professor Radiology
Nishtar Medical College & Hospital, Multan
drasanjum@gmail.com
Cell No 0333-4354512