Evaluation Of Intervertebral Disc Degeneration In Post Menopause Women With Chronic Lower Back Pain Using 3 Tesla MRI

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Abstract

Obiective

This study was conducted to evaluate increase of intervertebral disc degeneration in post menopause women with chronic lower back pain in comparison of premenopausal and post-menopausal women using 3Tesla MRI imaging.

Method

This study included 100 women in age range of 35years and above, who presented main complain of chronic lower back pain. MRI exam imaging was done for diagnostic purpose and data of patient history, demography and radiology report were collected. Intervertebral disc degeneration was graded by 1-5pfirman grading system-spine.

Results

Using IBMSPSS statistics-28 software, relationship and causal correlation of intervertebral disc degeneration and post menopause were evaluated using chi-square and One Way ANOVA inferential statistics. By considering critical p value of 0.005, this study has shown positive causal correlation of intervertebral disc degeneration and

post menopause, with P<0.001 in L1/l2, L2/3 and L3/4, P=0.035 in L4/5 and P=0.001 in L5/S1. this study also showed increase in severity of intervertebral disc degeneration from premenopause, perimenopause and post menopause at percentage of 18.79%, 27.06% and 54.13% respectively.

Conclusion

There is increase in intervertebral disc degeneration in post menopause women in comparison to premenopausal and perimenopausal women associated with decrease of estrogen hormone causing estrogen deficiency induced intervertebral disc degeneration.

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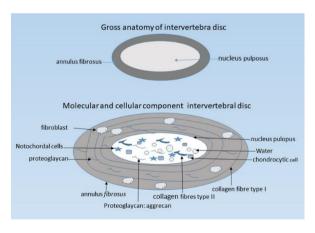
I. Introduction

Intervertebral discdegeneration is the most common cause of chronic lower back pain in adult population [1][2]. There are numerous causes of intervertebral disc degeneration including aging, genetic factors, lifestyle factors, occupational factors, mechanical stress, smoking, obesity, trauma and hormonal changes. In postmenopausal women, early IVD degeneration is asymptotic however the progression is associated with lower back pain due to hormonal changes caused by reduction of estrogen. Capability of 3TMRI to create high resolution of soft tissues is a foundation in evaluation of back painby diagnosis and grading of IVD degeneration. It's essential to understand macro and microanatomy of IVD, hormonal changes in post-menopausal women and their relation to the pathophysiology of back pain.

Anatomy of intervertebral disc

The intervertebral disc comprises two primary components: the outer annulus fibrosus and the inner nucleus pulposus. The annulus fibrosus consists of concentric bands of connective tissue, primarily type I collagen in the outer layers, which provide tensile strength and structural support. These layers resist the torsional and tensile forces exerted by the nucleus pulposus.

The inner annulus fibrosus contains type II collagen and elastic fibers, contributing to compressive strength and maintaining the disc's resilience and hydration [3]. The nucleus pulposus is a gel-like structure composed predominantly of water, proteoglycans such as aggrecan, and type II collagen. Proteoglycans attract and retain water, preserving the disc's hydration and compressive properties. Notochordal and chondrocyte-like cells within the nucleus pulposus secrete and maintain the extracellular matrix. Nutrient transport occurs via diffusion from the highly vascular cartilage endplates to the avascular disc layers. Fibroblast-like cells in the annulus fibrosus produce and maintain collagen fibers, while chondrocytes sustain the cartilage endplate matrix. This intricate cellular and structural organization enables the intervertebral disc to absorb mechanical loads and maintain spinal stability [4][5].



Etiology of chronic lower Back Pain

Chronic lower back pain (LBP) is a common clinical problem mostly in adults with multiple causes. Its etiology can be broadly divided into mechanical, non-mechanical, and referred pain sources. Mechanical causesby far the most commoninclude lumbar muscle strain, ligament sprain, intervertebral disc degeneration or herniation, facet joint arthropathy, andspondylolisthesis. These often result from repetitive stress, poor posture, heavy lifting, or trauma[6]. Non-mechanical causes may involve inflammatory disorders such as ankylosing spondylitis, infections like vertebral osteomyelitis, and neoplastic processes including spinal metastases. Referred pain may originate from visceral structures, such as renal stones or gastrointestinal disease, which share neural pathways with the lumbar region[7].

Chronic discogenic lower back pain

Discogenic lower back pain originates from pathological changes within the intervertebral disc commonly caused by intervertebral disc degeneration, a progressive process involving dehydration and breakdown of the disc's nucleus pulposus and annulus fibrosus. These changes can be due to aging, repetitive mechanical stress, poor posture, vibration exposure, or acute trauma. Micro tears in the annulus fibrosus allow ingrowth of nociceptive nerve fibers and blood vessels into normally avascular disc tissue. Genetic predisposition, and metabolic factors also contribute to disc degeneration. In some cases, herniation, protrusion and internal disc disruption directly irritates nerve endings within the disc, triggering pain[8].

Pathophysiology of discogenic chronic lower back pain

The intervertebral disc is normally a relatively aneural and avascular structure, with only the outer third of the annulus fibrosus containing nerve endings. In disc degeneration, tension overload, and reduced nutrient supply led to loss of proteoglycans and water content in the nucleus pulposus, decreasing its shock-absorbing capacity. This results in increased stress on the annulus fibrosus, causing fissures. These fissures permit the ingrowth of sensory nerve fibers, nociceptors and blood vessels deep into the disc, where they are exposed to inflammatory mediators such as tumor necrosis factor-alpha $(TNF-\alpha)$, interleukins, and prostaglandins. These substances sensitize nerve endings, generating nociceptive pain. Persistent inflammation can lead to peripheral sensitization, and chronic pain may involve central sensitization in the spinal cord and brain. Mechanical stress on the degenerated disc perpetuates the inflammatory response, creating a self-sustaining cycle of pain[9].

IVD degeneration in post menopause women

Intervertebral disc (IVD) degeneration in postmenopausal women is mostly due to hormonal changes, specifically the decrease in oestrogen levels. Oestrogenis important in maintaining disc hydration, collagen integrity, and anti-inflammatory balance within the intervertebral disc. After menopause, reduced oestrogen leads to increased oxidative stress, inflammation, and catabolic activity in disc cells, accelerating matrix degradation and loss of proteoglycans and water content in the nucleus pulposus. This hormonal shift contributes to disc height reduction, annular fissures, and neoinnervation, which are key features of degenerative disc disease and discogenic pain. Consequently, postmenopausal women are at higher risk for chronic low back pain due to these degenerative changes[10].

Evaluation of intervertebral disc degeneration: role of 3T MRI

3-tesla MRI plays a crucial role in evaluating intervertebral disc degeneration in postmenopausal women by providing high-resolution images with superior signal-to-noise ratio and tissue contrast, enabling detailed visualization of subtle structural and biochemical changes in the disc. Its enhanced spatial resolution allows precise assessment of disc hydration through T2-weighted imaging, detection of early nucleus pulposus dehydration, and evaluation of annulus fibrosus integrity. It allows assessment of feature of degeneration such as disc bulge, disc protrusion, disc extrusion, disc sequestration, change in disc signal intensity, disc height loss, annular tear, and schmorl's node. It allows evaluation of modic changes of vertebra body endplates such as bone marrow edema, and inflammation, marrow ischemia, and subchondral bone sclerosis which are highly associated with intervertebral disc degeneration[11].

II. Method And Materials

An observational study was approved and done in radiology department of Maharishi Markandeshwar institute of medical science and research-Mullana supervised by both radiology and community medicine staff.

Subject

100 female patients presented with chronic lower back pain and indicated for lumbosacral MRI imaging were enrolled for study. Age group was select to be classified as premenopausal 35-44, perimenopausal 45-54 years old and post-menopausal above 55 years old age groups.

Materials

3-tesla superconducting magnetic resonance imaging (United magnetic resonance omega) with surface coil Duration of study: from Mayup toOctober 2025

Inclusion criteria

- Premenopausal, perimenopausal and post-menopausal female patients
- IPD and OPD patients
- Patients with chronic lower back pain

Exclusioncriteria

- patients with history of trauma
- patients with past history of spinal surgery
- patients with prior history of infectious or neoplasmic diseases
- patients with other diseases that influence estrogen metabolism
- patients with diseases that influence spinal structure
- patients with active endocrine treatments
- MRI contraindicated patients
- unconsented patients
- medical legal cases

Study variables

Demographic factors: Age group and patient condition.

Data collected

- patient demographic data
- patient history
- MRI radiology report

MRI protocols

Eligible patients who were undergoing for lumbosacral spineMRIimaging Were selected. MRIexam was performed using 3Tesla MRI scanner, and surface coil, and the following scanning parameters were used for clinical image optimization:T1 weighted fast spin echo-axial (TE=110, TR=4400), T2 weighted fast spin echo axial ((TE=10, TR=4400), T2 weighted fast spin echo sagittal (TE=:110, TR= 4400), slice thickness:3mm, Field of view:180x410, interslice gap:10mm, number of signal acquisition:2. MRI images were reported by radiologists, and grading of IVD degeneration was done using 5grades Pfirrman grading system of intervertebral disc degeneration where grade 1 was considered normal with no IVD degeneration, grade 2 was considered mild degeneration, grade 3 was considered moderate degeneration, both grade 4 and 5 were considered severe degeneration.

Data analysis

One-way ANOVA was used for statistical analysis of significance of intervertebral disc degeneration in post-menopausal women in comparison of premenopausal and perimenopausal women, and chi-square test was used to evaluate statistical correlation. A P value < 0.05 was considered significant. IBM SPSS Statistics 28 software was used for statistical data analysis.

III. Results

The selected study sample of 100menopausal women who came for evaluation of chronic lower back painwas classified by age wise into premenopausal women with age range of 35 to 44 years, perimenopause with age range of 45 to 54 years and postmenopausalwomen with age range of \geq 55years. The sample had an average age of 49.71 \pm 11.72years, range of 37.99 to 61.43 years. The average age at menopause was 45 \pm 1.95, range of 41 to 54. The years since menopause was 11.98 \pm 8.73, range of 1 to 30. The sample had overall increase in IVDdegeneration from L1/2 to L5/S1 except L4/5 with had the highest degeneration of 3.34 \pm 1.34 (table 1).

Table 1: Characteristics of study sample

	Mean ± Standard deviation	Range
Age	49.71±11.72	35-78
Age at menopause	45±1.95	41-54
Years since menopause	11.98±8.73	1-30
	Mean grade of IVD degeneration	
L1/2	1.9 ± 1.08	1–5
L2/3	2.26±1.27	1–5
L3/4	2.67±1.36	1–5
L4/5	3.34±1.32	1–5
L5/S1	2.99±1.57	1–5

In total sample of 100, premenopausal women were 37 (37%) with mean age of 37.97 ± 3.1 years, range of 35-43 years, perimenopausal women were 31(31%) with mean age of 49.03 ± 2.73 , range of 45 - 54 years and postmenopausal women were 32 (32%) with mean age of 63.94 ± 6.81 , range of 55 - 78. In premenopausal and perimenopausal women, L4/5 had highest mean degeneration of 2.92 ± 1.47 and 3.45 ± 1.15 respectively, while in

postmenopausal L5/S1 had the highest degeneration of 3.78 ± 1.07 . There was significant positive relationship of statistical comparative analysis of IVD degeneration in premenopausal, perimenopause and postmenopausal with P < 0.001 at L1/2, L2/3, L3/4, P value of 0.035 at L4/5 and P value of 0.001 at L5/S1 (Table 2).

Table 2: Statistical	significance	of IVD	degeneration

	Perimenopause(n=37)	perimenopause (n=31)	post menopause(n=32)	p-values
mean age	37.97 ± 3.10	49.03 ± 2.73	63.94 ± 6.81	
L1/L2	1.35 ± 0.75	1.9 ± 0.94	2.53 ± 1.21	< 0.001
L2/L3	1.54 ± 0.9	2.1 ± 1.12	3.25 ± 1.16	< 0.001
L3/L4	1.84±1.06	2.71 ± 1.29	3.59 ± 1.13	< 0.001
L4/L5	2.92±1.47	3.45 ± 1.15	3.72 ± 1.17	0.035
L5/S1	2.49±1.74	2.77 ± 1.52	3.78 ± 1.07	0.001

In premenopausal group, L1/2 disc were normal (G1) at 27.27% and L4/5 were least normal at 10%. In same group, L5/S1 were highly degenerated at 52.94%, while there was no grade 5 degeneration in L1/2, L2/3, and L3/4. In perimenopause group, L1/2 disc were highly normal (G1) at 28.84% and L4/5 were least normal at 5.77%. In same group, L4/5 were highly degenerated at 42.86%, while there was no grade 5 degeneration in L1/2 and L2/3. In post menopause group, L1/2 discs were highly normal (G1) at 47.62% and L5/S1 were least normal at 4.76 %. In same group, L5/S1 were highly degenerated at 32.35%, while L1/2 had least grade 5 degenerations with of 5.88 %. (Table 3)

Table 3: Percentage of severity of IVD degeneration

Premenopause						
IVDD						
grade	L1/2	L2/3	L3/4	L4/5	L5/S1	TOTAL
1	30 (27.27%)	27(24.54%)	22 (20%)	11(10%)	20 (18.18%)	110 (100%)
2	1(50%)	0(0%)	1(50%)	0(0%)	0(0%)	2(100%)
3	6(12.5%)	10(20.83%)	12(25%)	15(31.25%)	5(10.41%)	48(100%)
4	0(0%)	0(0%)	2(25%)	3(37.5%)	3(37.5%)	8(100%)
5	0(0%)	0(0%)	0(0%)	8(47.056%)	9(52.94%)	17(100%)
Tota1	37	37	37	37	37	185
			Perimenopause			
1	15 (28.84 %)	14(26.92%)	9(17.31%)	3(5.77%)	11(21.15%)	52(100%)
2	4(40%)	3(30%)	1(10%)	1(10%)	1(10%)	10(100%)
3	12(21.05%	11(19.30%)	14(24.56%)	12(21.05%)	8(14.04%)	57(100%)
4	0(0%)	3 (13.64%)	4(18.18)	9(40.91%)	6(27.27%)	22(100%)
5	0(0%)	0(0%)	3(21.43%	6(42.86%)	5(35.71%)	14(100%)
Tota1	31	31	31	31	31	155
Post menopause						
1	10(47.62%)	4(19.05%)	3(14.29%)	3(14.29%)	1(4.76%)	21(100%)
2	2(50%)	1(25%)	0(0%)	0(0%)	1(25%)	4(100%)
3	15(23.81%)	15(23.81%)	11(17.46%)	9(14.29%)	13(20.63)	63(100%)
4	3(7.89%)	7(18.42%)	11(28.95%)	11(28.95%)	6(15.79%)	38(100%)
5	2(5.88%)	5(14.71%)	7(20.59%)	9(26.4%)	11(32.35%)	34(100%)
Total	32	32	32	32	32	160

There were decrease in normality of IVD degeneration from Premenopause N=110/500~(60.10~%) of total normal IVD), perimenopause N=52/500(28.41%) of total normal IVD) and post menopause N=21/500(11.47%) of total normal IVD), with premenopause group having more normal intervertebral discs. There were increase in severity of IVD degeneration at extent of moderate and severe in premenopause perimenopause and post menopause groups; premenopause with moderate degeneration of 48(28.57%) and severe degeneration of 25(18.79%), perimenopause with moderate degeneration of 57(33.92%) and severe degeneration of 36(27.06%), and, post menopause with moderate degeneration of 63(37.5%) and severe degeneration of and 72(54.13%) respectively, collectively suggesting that post menopause have lowest normality of IVD degeneration and highest severity of IVD degeneration (Table 4 and chat 1)

Table 4: percentage of IVD degeneration in menopausal groups

	1 5	0	1 5 1	
	Normal (G1)	Mild(G2)	Moderate(G3)	Severe(G4+5)
Premenopause	110(60.10%)	2(12.5%)	48(28.57%)	25(18.79%)
Perimenopause	52(28.41%)	10(62.5%)	57(33.92%)	36(27.06%)
Post menopause	21(11.47%)	4(25%)	63(37.5%)	72(54.13%)
Total	183(100%)	16(100%)	168(100%)	133(100%)

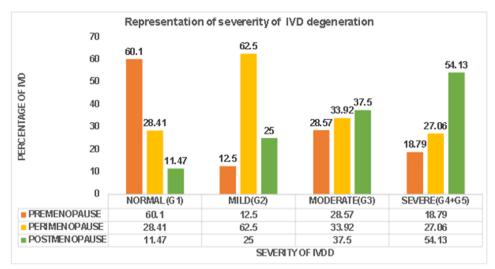


Figure 1: Percentage of IVD degeneration in menopausal groups

IV. Discussion

This study has used 3T MRI for evaluation of chronic lower back pain in premenopausal, perimenopausal and postmenopausal women. The sample had age range of 35 -78 years, years at menopause of 41-49 years, and years since menopause 1-30 years. L4/5 was the most degenerated intervertebral disc with the mean degeneration of 3.34 ± 1.32 . Postmenopausal women had highest severity of intervertebral disc degeneration at percentage of 54.13% comparing to premenopausal and perimenopausal groups with percentage of 18.79% and 27.06% respectively. This study has also show that premenopausal and postmenopausal have more normal and the mild intervertebral disc degeneration in comparison to postmenopausal women at percentage of 60.1%, 28.41%, 54.13% respectively.

Statistical comparative analysis of IVD degeneration in premenopausal, perimenopausal and postmenopausal groups has shown positive regression with P value<0.001 at L1/2, L2/3, L3/4, P value= 0.035 at L4/5 and P value= 0.001 at L5/S1. Theoccurrence of increased intervertebral disc degeneration in premenopausal and postmenopausal is due to reduced estrogen hormone causing estrogen deficiencybone diseases[12][13].

Estrogen helps to maintain the extracellular matrix of intervertebral disc bysupporting proteoglycan and collagen synthesis which maintain the hydration and tension resistance of intervertebral disc, hence reduced secretion acceleratesIVD degeneration due to matrix degeneration[10].

Different clinical studies have reported the causal correlation of reduction of estrogen hormone level in postmenopausal women and the progressive intervertebral disc degeneration [14][15][16]. Using 1-8 Pfirman grading system, C. Lou et al. has used 1.5 T MRI have reported that postmenopausal women had more severe (G6+7+8) IVD degeneration at percentage of 13.4 percent compare to premenopausal and postmenopausal whose 4.5 % and 6.7%. They have also shown that L4/5 had highest mean IVD degeneration of 4.41 \pm 1.34, same as our study [17].

A study conducted by Chao Lou et al. for evaluation of lumber disc degeneration in menopause women using MRI have reported that L4/L5 had highest degeneration of 5.14 ± 1.31 , and postmenopausal women had highest severe degeneration of 2.1% (n=132) compare to premenopausal and postmenopausal whose 0% and 0.7% (n=3) respectively, and this study shows same result as ours as they werehighest severe degeneration in postmenopause and severe degeneration in premenopause and perimenopausal groups[18].

Another review study A. Bisht et al, whose objective is to explore the relationship between menopause and lumbar disc degeneration in postmenopausal women using 1.5 T MRI. This study was conducted by reviewing past published studies on evaluation of disc degeneration in post-menopausal women using 1.5 T MRI and it concluded that decline in estrogen level during menopause contribute to osteoporosis and disc degeneration due to extracellular matrix degradation and loss of disc water content which destroy dis0c integrity[19].

Y. Zhang et al, has shown that, there is positive correlation of increased IVD degeneration in post menopause women and bone mineral density across all age groups P<0.05 which is different to other studies however this couldn't be evaluated in our studies[20].

M. Gambacciani et al, have reported that there is reduction of IVD space in 10-15 years since menopause due to estrogen deficiency causing rapid decline of connective tissue metabolism in IVD[2]

V. Conclusion

There is increase in intervertebral disc degeneration in post menopause women in comparison to premenopausal and perimenopausal women associated with decrease of estrogen hormone causing estrogen deficiency induced intervertebral disc degeneration.

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