

RANDOMIZED COMPARATIVE STUDY ON THE EFFICACY OF MULTI-MODAL INTERVENTION INCLUDING WILLIAM'S FLEXION EXERCISES, TENS AND MULLIGAN MOBILIZATION IN LUMBAR DEGENERATIVE DISORDERS

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A. INTRODUCTION

Lumbar disc degenerative disease is the most common cause of Low back pain throughout the world (1) Intervertebral discs are among the largest avascular tissues in the body, relying on diffusion for nutrition, and consists of inner nucleus pulposus, outer annulus fibrosus and cartilage located superiorly and inferiorly. Intervertebral disc resists compression because of its osmotic properties of the proteoglycans, which help to maintain disc height (2)

Due to excessive loading, deformation of the endplate occurs which leads to decreased intradiscal pressure, loss of intervertebral disc height and increased stress to the surrounding annulus fibrosus and facet joints. Signs of lumbar degenerative disease includes one or all of the following: diminished disc height, narrowing of facet, spondylophytes and endplates degeneration, spinal canal stenosis, narrowing of lateral recess in the spinal canal, disc desiccation, diffuse annular bulging beyond the disc space, defects and sclerosis of the endplates, and bone spur at the vertebral apophyses(3). Lumbar degenerative disc disease can occur at any spinal level, but it most commonly affects the L3-L4 and L5-S1 segment of lumbar spine (4).

The lumbosacral complex is biomechanically vulnerable to segmental instability, contributing to the high prevalence of low back pain among affected individuals (5).

Lumbosacral junction is an important unit of the spine. The lumbar spine comprising 5 distinct vertebrae (L1-L5) articulate with the sacrum, a rigid, fused structure consisting of 5 sacral segments (S1-S5)

The lumbar vertebrae structurally larger and more robust compared to other vertebral segments, a morphological adaptation that facilitates the transmission and support of axial body weight.

Larger vertebral bodies increase surface area for weight distributions and thicker intervertebral discs absorb shock and facilitate weight transfer, stronger pedicles and laminae provide structural support (6)

The lumbar spinal canal demonstrates a relatively narrower and more triangular configuration in cross-section compared to the wider, more oval morphology observed in the cervical and thoracic regions.

The lumbosacral complex in the lumbar spine is susceptible to both mechanical injury such as herniation, or degeneration and non-mechanical injury like degenerative conditions, inflammation due to anatomical, biomechanical and physiological factors including spinal anatomy, ligament support etc. (7)

The intervertebral (I.V.) disc is essential for preserving spinal alignment and contributing to overall spinal stability. Lumbar I.V. disc degeneration may lead to diminished spinal flexibility, neural impingement and disc height reduction (8). The I.V. disc facilitates load distribution and functions to promote spinal flexibility and stability simultaneously (9). According to global research data, degenerative disc disease affects approximately 266 million people worldwide, accounting for approximately 3.66% of the global population; Regional analysis observed Europe had the highest estimated incidence at (5.7%) representing the widest variation in prevalence while Africa (2.4%), respectively.

Epidemiological data suggest a significantly higher prevalence, low- and middle-income countries have 4 times more the number of cases than high-income countries. Globally spondylolisthesis has been identified in approximately 39 million individuals, representing around (0.53%) of the population, degenerative disc disease affects an estimated 403 million (5.5%) individuals worldwide, while lumbar spinal stenosis affects roughly 103 million cases annually (10).

The most frequently affected intervertebral disc level was the L4-L5 segment, accounting for (41.2%) of cases. Disc protrusion emerged as the predominant morphological pattern, observed in 69.4% of patients. The L5-S1 segment exhibited the highest incidence of disc height loss, observed in 32.2% of cases.

The most prevalent distribution pattern was multilevel disc degeneration, observed in 38.2% of population studied, followed by contiguous two- level degeneration, which occurred in (34.7%) with disc bulges frequently reported at L3-

L4 (27.2%) and L4-L5 (26.9%) levels. A significant proportion of intervertebral discs (56.1%) showed modic changes at the site of intervertebral disc prolapse levels. Spondylolisthesis was predominantly observed at the L5-S1 level (56.1%) & was mostly associated with lumbar canal stenosis (38.4%). Posterior osteophytes formation was frequently identified at the L3-L4 (31.1%) and L5-S1 level (31.1%) levels. The D12-L1 levels represented least commonly involved (4.1%) in cases of lumbar degenerative disc disease (11).

The shape of the vertebral foramen becomes more triangular at the lumbosacral junction, specifically at L5, due to pedicle shortening, narrowing of the spinal canal and increases susceptibility to compression. (12)

Lumbar degenerative disorder is a significant cause of disability in the world. Lumbar disc degenerative disease may present as lumbar disc herniation, spondylolisthesis, lumbar spinal stenosis, facet joint arthropathy, disc desiccation condition, can lead to symptoms such as low back pain, leg pain & weakness, radiating pain, numbness and tingling, which may result in decreased mobility and impacting daily life [13].

Intervertebral discs are specialized fibrocartilaginous structures that function as shock absorbers between adjacent vertebrae & facilitate articulation between vertebrae and act as pivot point, enabling spinal flexibility through movement such as flexion, extension and axial rotation. These discs contribute approximately 25% of the total length of the vertebral column.

The intervertebral disc comprises two distinct components: A tough fibrous outer layer known as annulus fibrosus surrounding a gel-like inner called nucleus pulposus. The nucleus pulposus contains approximately 80-85% water in normal cases. Disc desiccation is degenerative disc disease that dries up the water contents. This degenerative process may contribute to annular fissuring, loss of disc integrity, could lead to facet joint arthropathy.

If it progresses, it may protrude or herniate potentially compressing adjacent spinal nerve roots. Disc desiccation is commonly diagnosed because of natural aging process with a high prevalence in older population. However there are many other etiological factors may contribute to disc desiccation such as trauma, sudden weight loss, idiopathic causes (14).

Intervertebral disc desiccation occurs when glycosaminoglycan loss causes the nucleus pulposus to be replaced with fibrocartilage resulting in loss of disc height and function due to reduction in nucleus pulposus volume. (15)

Each vertebrae have an intervertebral disc at the front and facet joints at back form overlapping joints with adjacent vertebrae. Low back pain can be caused by various factors including ligamentous instability affecting spinal discs, compression or pinching of the spinal cord roots. (16)

Degeneration of intervertebral disc causes early-stage looseness and hypermobility of facet joints (17, 18). This can contribute to hypertrophy of facet joints especially the superior articular process leading to narrowing of spinal canal and compression of neural structures.

The most common form of degenerative stenosis generally becomes symptomatic during or after the seventh decade of life (19).

Degenerative narrowing of the lumbar spinal canal characterized by a progressive constriction of spinal canal due to wear and tear changes in the vertebral joints, intervertebral discs and hypertrophy of ligament flavum (20).

As the perineural and perivascular spaces become narrower, prominent clinical manifestations may develop, including neurogenic intermittent claudication, radiating pain in the lower extremities, low back pain and disturbance in the bladder and bowel function. Clinical symptoms may include diminished sensory perception and muscle fatigue in the lower extremities as well as increasing pain in the gluteal region or legs which may worsen when walking or prolonged standing or sustained upright posture (21).

B. MATERIAL & METHODS

A systematic review of Quantitative evidence was conducted using PubMed & google scholar to identify relevant studies. This study was conducted on diagnosed patients with lumbar degenerative conditions. The diagnostic criteria for the lumbar degenerative conditions were established on the basis of abnormal findings in MRI. While clinical evaluation was performed using the Straight Leg Raise test and The Femoral Nerve Stretch test.

1. Study Design

This study is a single blinded randomized control trial.

Inclusion criteria

Patients with chronic low back pain for more than 3 months
age group 25-60 years

Exclusion criteria

Acute disc bulge
Any traumatic injury of spine
Infective conditions
congenital spinal deformities
Malignancy, any history of spinal surgery

Study Duration

The duration of the study is 6 months

Sample size

140 subjects

Purpose

Several treatment method has been used management of lumbar degenerative conditions Williams spinal flexion exercises along with transcutaneous electrical nerve stimulation (TENS) and Mulligan mobilization with movements (MWM) are two Integrated conservative therapeutic techniques used for treating patients with lumbar degenerative conditions.

2. Data Collection

Subjects who voluntarily gave their consent for the participation in the study were enrolled from patients visiting Department of Physiotherapy - Era's Lucknow Medical College, Sarfarazganj, Lucknow and Department of Physiotherapy - Dr Jawahar Lal Rohatgi Hospital, Sarvodaya Nagar, Kanpur.

3. METHODS

Patients were divided into two treatment groups:

Group A - Williams spinal flexion exercises along with Tens.

Group B - Mulligan mobilization with movements techniques. Pain was measured using the Visual Analog Scale (VAS) and mobility with the Modified Oswestry Disability Index (MODI), Mulligan Mobilization belt, Tape measurement for ROM assessment Modified Schober test used in measuring lumbar flexion, extension and side bending to the right and the left data were analyzed using SPSS version 25, with $p < 0.05$ considered significant.

Group A - William's spinal flexion exercise protocol comprises a structured regimen of therapeutic movements designed to improve lumbar spine flexion, avoiding lumbar extension, engaging the abdominal and gluteal muscles. These exercises are typically performed in supine position on the floor or other flat surface for the conservative management of chronic or recurrent lower back pain.

Williams's flexion exercise, emphasizes muscle strengthening as well as stretching of other muscles.

This therapeutic exercise regimen can reduce pain and enhance lumbopelvic stability. It promotes passive flexibility through stretching of the hip flexors and lumbar paraspinal muscles and sacrospinalis group.

When patients perform the Williams spinal flexion exercises regularly, they reduce pain and increase the range of motion. The eight variations of Williams's exercises include: (a) Posterior pelvic tilt (b) Unilateral knee to chest (c) Partial sit-ups (d) Hamstring stretch (e) Hip flexor stretch (f) Lumbar flexion with rotation (g) Seated trunk flexion (h) Piriformis muscle stretching.

(a) Posterior Pelvic Tilt:

Performed in supine position with hips and knees flexed and then tightening & squeezing the muscles of the buttock & abdomen. Hold for 10 seconds & then slowly returning the pelvis to the starting position. 10 – 15 repetitions are advised. Patient is instructed to avoid lifting their hips off the floor.

(b) Unilateral Knee-to-chest:

Performed in supine position and pulling unilateral knee toward shoulder. Hold for 5 to 10 seconds. The contra-lateral limb remains in a extended position. Patient is instructed to extend the knee & return back to the relaxed supine position. Repeat with other knee

(c) Partial sit-up:

Executed in a supine position with knees flexed. Perform the pelvic tilt using abdominal muscles to raise upper back off the floor and then curl the head and shoulder off the floor, curling up towards your knees. Hold for 7 seconds, return slowly to the starting position. 10 repetitions advised.

(d) Hamstring muscle stretch:

Performed in supine position, involving passive or active assisted hip flexion with the knee extended. Hold for 10 seconds & 10 repetitions. Repeat the stretch on the other side.

(e) Hip flexors stretch:

Performed in supine position. Flex one knee keeping their foot flexed, gently stretch knee towards the chest. Hold the stretch for 10 seconds and slowly return to the starting position. Repeat the stretch on other side and perform 10 repetitions.

(f) Lumbar flexion with rotation:

Performed in supine position. Participants were instructed to flex the trunk while maintaining both the knees bent and then gently rotate the knees towards symptomatic side. 10 repetitions advised.

(g) Seated lumbar flexion:

Performed in sitting with spine in an upright position. Lean forward slowly keeping knees straight until patient feel tension in the back. Hold the flexed position for 10 seconds and then slowly return to the starting position.

(h) Piriformis muscle stretching:

Performed in a supine position. Both knees are flexed and cross one leg over the other and then stretch the muscle in opposite shoulder direction. Hold for 10 seconds. Exercise to be performed only by one leg especially on the side where the pain is radiating.



Group B- The Mulligan concept of mobilization with movement (MWM) represents a specialized manual therapy paradigm designed to facilitate, restore optimal arthro-kinematic glide and osteokinematic movements across spinal and peripheral articulations. (22)

Mulligan's concept facilitates the correction of biomechanical and postural dysfunction by performing mobilization with movement, it involves the application of sustained transverse glide to the spinous process of the targeted vertebrae, either actively or passively execution of the restricted peripheral joint movements aiming to restore segmental mobility. (23) All of the Mulligan techniques must elicit pain free response during movement. (24)

When MWM is specifically applied to the spinal facet joints, the technique is referred to as sustained natural apophyseal glides (SNAGs) [25].

SNAGs is a manual therapy technique that enhance joint mobility through the application of passive accessory glide to the lumbar facet joints with concurrent active movement BY the patient performs simultaneously (26)

Exercise-

Participant were seated on a height adjustable plinth with both feet placed on a footrest, positioned in slight plantarflexion. The mulligan belt was secured around the patient anterior superior iliac spine (ASIS) and anchored to the hip joint of the therapist to facilitate the mobilization technique.

SNAGs were applied using the therapist's hypothenar eminence on the transverse process of the affected spinal segment. The mobilization force was directed parallel to the facet joint plane (cranial direction). Concurrently patients were instructed to lean forward while therapist applied mobilization force simultaneously. After attaining the end range of forward flexion, patients were instructed to return to the starting position. During this return movement, a sustained cranially directed mobilization force was maintained. These techniques are usually performed in weight bearing position like standing, sitting (27)(28). Patients must not feel pain during technique application.

C. Statistical Analysis

Reliability test

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Matrix Input		

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	Cases Used	Statistics are based on all cases with valid data for all variables in the procedure.
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Resources	Processor Time	00:00:00.00
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Scale: ALL VARIABLES

Case Processing Summary				
Group			N	%
A	Cases	Valid	68	100.0
		Excluded ^a	0	.0
		Total	68	100.0
B	Cases	Valid	68	100.0
		Excluded ^a	0	.0
		Total	68	100.0

a. Listwise deletion based on all variables in the procedure.

Reliability Statistics		
Group	Cronbach's Alpha	N of Items
A	.511	4
B	.755	4

1) Frequencies

Notes		
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	Cases Used	Statistics are based on all cases with valid data.
Syntax		FREQUENCIES VARIABLES=GROUP /ORDER=ANALYSIS.
Resources	Processor Time	00:00:00.00
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Statistics		
Group		
N	Valid	136
	Missing	0

Group					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	68	50.0	50.0	50.0
	B	68	50.0	50.0	100.0
	Total	136	100.0	100.0	

2) Paired T- test for VAS-Pre and VAS-Post

T-Test

Notes		
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	Cases Used	Statistics for each analysis in a list are based on the cases with no missing or out-of-range data for any variable in that list
Syntax		T-TEST PAIRS=VASPRE WITH VASPOST (PAIRED) /ES DISPLAY(TRUE) STANDARDIZER(SD) /CRITERIA=CI(.9500) /MISSING=LISTWISE.
Resources	Processor Time	00:00:00.00
	Elapsed Time	00:00:00.00

Paired Samples Statistics						
Group			Mean	N	Std. Deviation	Std. Error Mean
A	Pair 1	Visual Analog Scale - Pre	5.88	68	.985	.119
		Visual Analog Scale - Post	3.24	68	1.081	.131
B	Pair 1	Visual Analog Scale - Pre	5.01	68	.801	.097
		Visual Analog Scale - Post	3.72	68	.808	.098

Paired Samples Correlations						
Group			N	Correlation	Significance	
					One-Sided p	Two-Sided p
A	Pair 1	Visual Analog Scale - Pre & Visual Analog Scale - Post	68	.026	.415	.831
B	Pair 1	Visual Analog Scale - Pre & Visual Analog Scale - Post	68	.422	<.001	<.001

Paired Samples Test											
Group			Paired Differences						Significance		
			Mean	Std. Deviation	Std. Error	95% Confidence Interval of the Difference		t			
						Lower	Upper				
A	Pair 1	Visual Analog Scale - Pre - Visual Analog Scale - Post	2.647	1.443	.175	2.298	2.996	15.126	67	<.001	<.001
B	Pair 1	Visual Analog Scale - Pre - Visual Analog Scale - Post	1.294	.865	.105	1.085	1.503	12.339	67	<.001	<.001

Paired Samples Effect Sizes						
Group			Standardizer ^a	Point Estimate	95% Confidence Interval	
					Lower	Upper

A	Pair 1	Visual Analog Scale - Pre	Cohen's d	1.443	1.834	1.441	2.222
		- Visual Analog Scale - Post	Hedges' correction	1.460	1.814	1.425	2.197
B	Pair 1	Visual Analog Scale - Pre	Cohen's d	.865	1.496	1.147	1.841
		- Visual Analog Scale - Post	Hedges' correction	.875	1.479	1.134	1.820

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

3) Paired t-test for Disability pre and post

T-Test

Notes			
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Comments			
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	Split File		Group
	N of Rows in Working Data File		136
Missing Value Handling	Definition of Missing		User defined missing values are treated as missing.
	Cases Used		Statistics for each analysis in a list are based on the cases with no missing or out-of-range data for any variable in that list
Syntax		T-TEST PAIRS=DISPRE WITH DISPOST (PAIRED) /ES DISPLAY(TRUE) STANDARDIZER(SD) /CRITERIA=CI(.9500) /MISSING=LISTWISE.	
Resources	Processor Time		00:00:00.00
	Elapsed Time		00:00:00.00

Paired Samples Statistics						
Group	Pair 1		Mean	N	Std. Deviation	Std. Error Mean
A	Pair 1	Disability - Pre	20.46	68	3.857	.468
		Disability - Post	13.34	68	3.317	.402
B	Pair 1	Disability - Pre	22.07	68	3.293	.399
		Disability - Post	19.28	68	3.424	.415

Paired Samples Correlations						
Group	Pair 1		N	Correlation	Significance	
					One-Sided p	Two-Sided p
A	Pair 1	Disability - Pre & Disability - Post	68	.603	<.001	<.001
B	Pair 1	Disability - Pre & Disability - Post	68	.805	<.001	<.001

Paired Samples Test											
Group	Pair 1		Paired Differences					t	df	Significance	
			Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				One-Sided p	Two-Sided p
						Lower	Upper				
A	Pair 1	Disability - Pre - Disability - Post	-7.118	3.235	.392	6.335	7.901	18.145	67	<.001	<.001
B	Pair 1	Disability - Pre - Disability - Post	-2.794	2.099	.255	2.286	3.302	10.978	67	<.001	<.001

Paired Samples Effect Sizes							
Group				Standardizer ^a	Point Estimate	95% Confidence Interval	
						Lower	Upper
A	Pair 1	Disability - Pre - Disability - Post	Cohen's d	3.235	2.200	1.757	2.639
			Hedges' correction	3.272	2.176	1.737	2.609
B	Pair 1	Disability - Pre - Disability - Post	Cohen's d	2.099	1.331	1.001	1.656
			Hedges' correction	2.123	1.316	.990	1.637

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

4) Independent sample t test for age T-Test

Notes		
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Comments		
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Missing Value Handling	Definition of Missing	User defined missing values are treated as missing.
	Cases Used	Statistics for each analysis are based on the cases with no missing or out-of-range data for any variable in the analysis.
Syntax	T-TEST GROUPS=GROUP('A' 'B') /MISSING=ANALYSIS /VARIABLES=AGE /ES DISPLAY(TRUE) /HOMOGENEITY DISPLAY(FALSE) /CRITERIA=CI(.95).	
Resources	Processor Time	00:00:00.00
	Elapsed Time	00:00:00.00

Group Statistics					
	Group	N	Mean	Std. Deviation	Std. Error Mean
Age of the patient	A	68	41.15	12.528	1.519
	B	68	43.76	12.606	1.529

Independent Samples Test									
		t-test for Equality of Means							
		t	df	Significance		Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
				One-Sided p	Two-Sided p			Lower	Upper
Age of the patient	Equal variances assumed	1.215	134	.113	.227	-2.618	2.155	-6.880	1.645
	Equal variances not assumed	1.215	133.995	.113	.227	-2.618	2.155	-6.880	1.645

Independent Samples Effect Sizes						
			Standardizer ^a	Point Estimate	95% Confidence Interval	
					Lower	Upper
Age of the patient	Cohen's d		12.567	-.208	-.545	.129
	Hedges' correction		12.638	-.207	-.542	.128
	Glass's delta		12.606	-.208	-.545	.131

a. The denominator used in estimating the effect sizes.
 Cohen's d uses the pooled standard deviation.
 Hedges' correction uses the pooled standard deviation, plus a correction factor.
 Glass's delta uses the sample standard deviation of the control (i.e., the second) group.

5) Chi square test for Gender
Chi-Square Test for Independence

Notes		
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	Cases Used	Statistics for each table are based on all the cases with valid data in the specified range(s) for all variables in each table.
Syntax	CHISQUARE INDEPENDENCE /TABLES=GENDER BY GROUP /STATISTICS=CHISQ /FORMAT=AVALUE NOTABLES /CELLS=COUNT.	
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	Cells Available	524245

Case Processing Summary						
	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Gender * Group	136	100.0%	0	0.0%	136	100.0%

Chi-Square Tests					
	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.030 ^a	1	.863		
Continuity Correction ^b	.000	1	1.000		
Likelihood Ratio	.030	1	.863		
Fisher's Exact Test				1.000	.500
Linear-by-Linear Association	.030	1	.863		
N of Valid Cases	136				
a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 29.50.					
b. Computed only for a 2x2 table					

Results and Interpretation

1. Baseline Characteristics

The two groups (A and B, n=68 each) were comparable in their demographic variables.

- **Age:** No significant difference was observed between the groups ($t \approx -1.22$, $p = .23$). The 95% CI of -6.88 to 1.65 crossed zero, confirming similarity in mean age.
- **Gender:** Distribution of male and female participants did not differ significantly ($\chi^2(1) = 0.03$, $p = .863$; Fisher's exact test $p = 1.00$).

Interpretation: Both groups were demographically well matched at baseline, ruling out confounding effects from age or gender.

2. Pain Scores (VAS)

Group A

Pain scores reduced significantly from **5.88 ± 0.99 (pre)** to **3.24 ± 1.08 (post)**. The mean change was $\Delta = 2.65$, highly significant ($t(67) = 15.13, p < .001$). The effect size was very large (Cohen's $d_z \approx 1.83$), corresponding to a **45% reduction** in pain.

Group B

Pain decreased from **5.01 ± 0.80 (pre)** to **3.72 ± 0.81 (post)**. The mean change was $\Delta = 1.29$, also significant ($t(67) = 12.34, p < .001$). The effect size was large ($d_z \approx 1.50$), with a **25.8% reduction**.

Between-group comparison

The improvement in pain was **greater in Group A** compared to Group B. The difference in mean change was **1.35 units** (95% CI [0.95, 1.76], $p < .001$).

Interpretation: Both groups showed significant pain relief, but **Group A experienced nearly double the reduction** compared to Group B.

3. Disability Scores

Group A

Disability scores reduced from **20.46 ± 3.86 (pre)** to **13.34 ± 3.32 (post)**. The mean change was $\Delta = 7.12$, highly significant ($t(67) = 18.15, p < .001$). The effect size was extremely large ($d_z \approx 2.20$), representing a **34.8% improvement**.

Group B

Scores decreased from **22.07 ± 3.29 (pre)** to **19.28 ± 3.42 (post)**. The mean change was $\Delta = 2.79$, also significant ($t(67) = 10.98, p < .001$). The effect size was large ($d_z \approx 1.33$), with a **12.7% improvement**.

Between-group comparison

The mean improvement in Group A was significantly larger than Group B ($\Delta = 4.32$, 95% CI [3.40, 5.25], $p < .001$).

Interpretation: Both groups benefited, but **Group A showed a markedly greater functional improvement** than Group B.

4. Correlation Analysis

- **Pain (VAS):** Weak correlation in Group A ($r \approx 0.03$), moderate in Group B ($r \approx 0.42$).
- **Disability:** Moderate correlation in Group A ($r \approx 0.60$), strong correlation in Group B ($r \approx 0.81$).

Interpretation: In Group B, individual rank-ordering of disability was more stable across time, while Group A showed greater variability—likely due to stronger intervention effects.

5. Reliability Analysis

Cronbach's alpha across the four combined variables (VAS-pre/post, Disability-pre/post) was:

- **Group A:** $\alpha = 0.511$ (poor reliability)
- **Group B:** $\alpha = 0.755$ (acceptable reliability)

Interpretation: The low alpha for Group A suggests limited internal consistency when combining these different measures. Since VAS and disability are distinct constructs measured across two timepoints, Cronbach's alpha is not an ideal reliability metric. Scale-specific or test-retest measures would be more appropriate.

Overall Interpretation

- Both groups showed **statistically significant improvements** in pain and disability.
- **Group A demonstrated larger reductions** in both pain and disability compared to Group B, with very large effect sizes.
- Baseline comparability of age and gender strengthens the validity of these findings.
- Group B's stability in disability correlations suggests consistent improvement patterns, whereas Group A showed greater intervention-driven changes.
- Reliability analysis indicates caution in interpreting alpha values, as constructs and timepoints differ.

D. RESULTS

There were 140 diagnosed patients with lumbar degenerative disease. patients were between the ages of 25 to 60 years & most were the 4th decade of life. all patients included in the study reported a prior history of lower back pain & predominantly, patients presented with either unilateral or bilateral radiating pain and pain aggravation was typically of low back pain occurs mostly during walking, prolonged standing, or lifting tasks.

This study assessed five degenerative spinal pathologies; lumbar intervertebral disc herniation, lumbar canal stenosis, facet arthropathy, disc desiccation, spondylolisthesis condition. lumbar PIVD was identified as the predominant pathology. The following pathological combinations were also observed; (1) lumbar disc herniation combination with lumbar canal stenosis. (2) combination of lumbar Pivd and with facet joint arthropathy (3) herniated disc with spondylolisthesis (4) lumbar canal stenosis with disc desiccation. Randomly divided into two groups

Group A- Received Williams spinal flexion exercises along with TENS

Group B- Received Mulligan movement with mobilization

Baseline and post- intervention outcomes were measured using visual analogue scale (VAS) and Oswestry disability index (ODI) for functional disability and Modified Schober's test for lumbar flexion range.

DISCUSSION

Lumbar intervertebral disc degeneration represents the predominant etiology of lower back pain worldwide and it was observed disc herniation identified as a principal manifestation of degenerative process. These findings support the widely held view that male are more susceptible to intervertebral disc degeneration compared to females, potentially caused by increased mechanical stress and repetitive microtrauma (29).

the Mulligan techniques can decrease pain and improve disability in patients with lumbar degenerative conditions. Sustained natural apophyseal glides (SNAGs) may facilitate immediate pain relief and enhancement in spinal ROM by correcting positional fault in facet joints.

The findings of this randomized control trial demonstrate that combine therapy Williams spinal flexion exercises along with TENS was more effective in reducing pain, enhancing lumbar mobility and reducing functional in patient with lumbar degenerative condition as compared to Mulligan movement with mobilization.

CONCLUSIONS

The lumbar discs most frequently affected by degenerative changes, leading to herniation and spinal stenosis ,are L4,L5 & L5, S1. Awareness is needed of this prevalent condition is essential so that appropriate preventive measures can be taken to reduce disease progression like giving proper time for physical exercise and adopting a healthy lifestyle.

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REFERENCES

1. Weiler C, Lopez-Ramos M, Mayer HM, et al. Histological analysis of surgical lumbar intervertebral disc tissue provides evidence for an association between disc degeneration and increased body mass index. *BMC Res Notes*. 2011; 4:497. doi: 10.1186/1756-0500-4-497. [DOI] [PMC free article] [PubMed] [Google Scholar]
2. Choi YS. Pathophysiology of degenerative disc disease. *Asian Spine J*. 2009; 3:39–44. doi: 10.4184/asj.2009.3.1.39. [DOI] [PMC free article] [PubMed] [Google Scholar]
3. Modic MT, Ross JS. Lumbar degenerative disk disease. *Radiology*. 2007; 245:43–61. doi: 10.1148/radiol.2451051706. [DOI] [PubMed] [Google Scholar]
4. David G, Ciurea AV, Iencean SM, Mohan A. Angiogenesis in the degeneration of the lumbar intervertebral disc. *J Med Life*. 2010; 3:154–161. [PMC free article] [PubMed] [Google Scholar]
5. Bartelink, D. L. (1957). The role of abdominal pressure in relieving the pressure on the lumbar intervertebral discs. *Journal of bone Joint Surgery*, 39B, 718.
6. Coppes MH, Marani E, Thomeer RT, et al. Innervation of “painful” lumbar discs. *Spine* 1997; 22:2342 – 9[discussion: 2349 – 50].
7. Yaszemski MJ, White A, Panjabi M. Biomechanics of the spine. In: Fardon DF, Garfin SR, editors. *Ortho x0002_ pedic knowledge update, spine*. 2nd edition. Rose x0002_ mont (IL) 7 AAOS; 2002. p. 15 – 23.
8. Belfi LM, Ortiz AO, Katz DS. Computed tomography evaluation of spondylolysis and spondylolisthesis in asymptomatic patients. *Spine (Phila Pa 1976)*. 2006; 31: E907–E910. - PubMed
9. Hoy D, March L, Brooks P, et al. The global burden of low backpain: estimates from the Global Burden of Disease 2010 study. *Ann Rheum Dis*. 2014; 73:968-974. <https://journals.sagepub.com/doi/abs/10.1177/2192568218770769>
10. https://journals.lww.com/neur/fulltext/2020/68060/magnetic_resonance_evaluation_of_lumbar_disc.22.aspx
11. Lipson SJ, Muir H. Experimental intervertebral disc degeneration: morphologic and proteoglycan changes over time. *Arthritis Rheum*. 1981; 24:12–21. doi: 10.1002/art.1780240103. [DOI] [PubMed] [Google Scholar]
12. Haun DW, Kettner NW. Spondylolysis and spondylolisthesis: a narrative review of etiology, diagnosis, and conservative management. *J Chiropr Med* 2005; 4(4):206-17
13. Richard S. Snell, *Clinical Anatomy by Regions*, Lippincott Williams and Wilkins, 8th edition, 2007. PubMed 2007 Mar; 106(3):591-614. <https://spineconnection.org/back-pain-conditions/facet-arthropathy/>
14. Holm S. Pathophysiology of disc degeneration. *Acta Orthop Scand Suppl*. 1993; 251:13–15. doi: 10.3109/17453679309160105. [DOI] [PubMed] [Google Scholar]
15. Rauschnig W. Patho anatomy of lumbar disc degeneration and stenosis. *Acta Orthop Scand Suppl*. 1993; 251:3–12. doi: 10.3109/17453679309160104. [DOI] [PubMed] [Google Scholar]
16. Paine KW. Clinical features of lumbar spinal stenosis. *Clin Orthop Relat Res*. 1976;(115):77–82. [PubMed] [Google Scholar]
17. Katz JN, Harris MB. Clinical practice: lumbar spinal stenosis. *N Engl J Med*. 2008; 358:818–25. doi: 10.1056/NEJMcp0708097. [DOI] [PubMed] [Google Scholar]
18. Rauschnig W. Normal and pathologic anatomy of the lumbar root canals. *Spine (Phila Pa 1976)* 1987; 12:1008–19. doi: 10.1097/00007632-198712000-00012. [DOI] [PubMed] [Google Scholar]
19. Baker RT, Nasypany A, Seegmiller JG, et al. The mul ligan concept: mobilizations with movement. *Int J Athl Ther Trai* 2013; 18: 30–34.

20. Mulligan BR. Manual Therapy: “NAGS,” “SNAGS,” “MWMS” etc. Wellington, New Zealand: Plane View Services LTD, 2010.
21. Exelby L. The Mulligan concept: its application in the management of spinal conditions. *Manual Ther* 2002; 7: 64–70.
22. Mulligan, B. “NAGS”, “SNAGS”, “MWMS”, 5th ed.; OPTP: Wellingt, Newzealand, 2004.
23. Moutzouri, M.; Billis, E.; Strimpakos, N.; Kottika, P.; Oldham, J.A. The effects of the Mulligan Sustained Natural Apophyseal Glide (SNAG) mobilisation in the lumbar flexion range of asymptomatic subjects as measured by the Zebris CMS20 3-D motion analysis system. *BMC Musculoskeletal. Disord.* 2008, 9, 131. [CrossRef]
24. Heggannavar A., Kale A. 2015, Immediate Effect of Modified Lumbar Snags In Non-Specific Chronic Low Back Patients: A Pilot Study. *Int J Physiotherapy Res.* 3(3):1018-23. Available from: http://www.physiopeedia.com/Chronic_Low_Back_Pain.
25. 28. Moutzouri M., Billis E., Strimpakos N., Kottika P., Oldham J.A. 2008. The effects of the Mulligan Sustained Natural Apophyseal Glide (SNAG) mobilisation in the lumbar flexion range of asymptomatic subjects as measured by the Zebris CMS20 3-D motion analysis system. *BMC Musculoskeletal disorders.* 9(1):1.
26. Wang YX, Griffith JF. Effect of menopause on lumbar disk degeneration: potential etiology. *Radiology.* 2010; 257:318–320. doi: 10.1148/radiol.10100775. [DOI] [PubMed] [Google Scholar]