AMJ Australasian Medical Journal [AMJ 2017;10(10):838-847]

Thoracic mobilisation and periscapular soft tissue manipulations in the management of chronic Prolapsed Intervertebral Disc (PIVD) - An innovative manual therapy approach

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RESEARCH

Please cite this paper as: Kiran R, Mohanty PP, Pattnaik M. Thoracic mobilisation and periscapular soft tissue manipulations in the management of chronic pivd - An innovative manual therapy approach. AMJ 2017;10(10):838–847.
https://doi.org/10.21767/AMJ.2017.2851

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ABSTRACT

Background
The most common cause of CLBP is discogenic lower back pain. Researches haveshown that connective tissue remodelling occurs in CLBP and thoracic spinal mobility and thoracolumbar mobility have higher correlations with LBP.

Aims
To see the effect of upper back fascia and periscapular muscles stretching and thoracic mobilisation to reduce symptoms in subjects with CLBP due to lumbar disk herniation.

Methods
A total of 40 subjects with CLBP due to Prolapsed Intervertebral Disc (PIVD) were recruited and randomly distributed in two groups. Group 1 was given stretching of upper back fascia and periscapular muscles and thoracic mobilization along with conventional exercises (Cyriax listing correction -I followed by repeated McKenzie back extension exercise and Core muscle strengthening). Group 2 received only conventional exercises. Outcome Measures: visual analog scale, Oswestry Disability Index and Modified Schober's Test. Measurements were taken before and after three weeks of treatment, for five days/week.

Results
Overall results of the study, both Group 1 and Group 2 showed improvement in pain, function and lumbar Range of Motion (ROM) after three weeks of intervention. However, Group 1 improved significantly to a greater extent in pain, ROM and function than the Group 2.

Conclusion
Stretching of periscapular muscles and fascia of the upper back and mobilisation of upper thoracic spine is found to be effective for the management of chronic low back pain due to PIVD.

Key Words
Chronic low back pain, manual therapy, myofascial pain syndrome, periscapular muscle stretching, prolapsed intervertebral disc, thoracolumbar fascia, thoracic mobilisation

What this study adds:

1. What is known about this subject?
LBP with or without radiation to lower extremity due to PIVD is often associated with myofascial pain syndrome of periscapular muscles and cervicothoracic extension dysfunction.

2. What new information is offered in this study?
Periscapular muscles and upper back fascia stretching and upper thoracic spinal mobilisation are found to be effective for the management of CLBP due to PIVD.

3. What are the implications for research, policy, or practice?
Manual therapy techniques applied to a distant connected

part of lumbar spine that reduces the symptoms would be safe and beneficial to patient with PIVD.

Background

Low back pain is extremely common human phenomena. Acute lower back pain refers to lower back pain that lasts less than four weeks. Subacute lower back pain refers to lower back pain that lasts from 4–12 weeks. Chronic lower back pain (CLBP) is lower back pain lasting 12 weeks or longer. Chronic low back pain is one of the common causes responsible for disability. The most common cause of CLBP is discogenic lower back pain.[1] In discogenic lower back pain, a tear occurs from the nucleus pulposus extending out to the outer third or two thirds of the annulus fibrosus. This tear allows the proteins with inflammatory properties to extravagate out to the nerve fibres, which can irritate those fibres and cause pain. DePalma et al. confirmed the disc as the most common etiology of chronic low back pain in adults.[2] Langevin et al. hypothesize that connective tissue remodelling occurs in CLBP as a result of emotional, behavioural and motor dysfunction and increased connective tissue stiffness due to fibrosis is an important link in the pathogenic mechanism leading to chronicity of pain, fear and further movement impairment.[3]

Researchers have shown that abnormal movement patterns can have important influences on the connective tissues that surround and infiltrate muscles.[4] Both increased stress due to overuse, repetitive movement and/or hypermobility, and decreased stress due to immobilization or hypomobility can cause changes in connective tissue.[5] Tissue microinjury, inflammation and fibrosis not only can change the biomechanics of soft tissue (e.g., increased stiffness) but also can profoundly alter the sensory input arising from the affected tissues.[6] Connective tissue is richly innervated with mechano-sensory and nociceptive neurons. Modulation of nociceptor activity has been shown to occur in response to changes in the innervated tissue.[6]

Tissue levels of inflammatory mediators (prostaglandins, bradykinin), have been shown to influence sensory input to the nervous system. Conversely, nociceptor activation has been shown to modify the innervated tissue. Release of Substance P from sensory C-fibers in the skin can enhance the production of histamine and cytokines from mast cells, monocytes and endothelial cells. Increased TGFb-1 production, stimulated by tissue injury and histamine release, is a powerful driver of fibroblast collagen synthesis and tissue fibrosis.[7] Thus, activation of nociceptors by itself can contribute to the development or worsening of fibrosis and inflammation, causing even more tissue stiffness and movement impairment.

In patients with CLBP, connective tissue fibrosis can occur in the thoracolumbar fascia due to one or several of the following factors: (1) decreased activity, (2) changes in muscle activation patterns causing muscle co-contraction, muscle spasm or tissue microtrauma and (3) neurally-mediated inflammation the intrinsic muscles of the back consist of a complex, serially arranged group that extends from the pelvis to skull base. The first layer of the paraspinal muscles consists of the trapezius, latissimus dorsi, rhomboids, and levator scapulae. In muscle, plasticity of perimuscular and intramuscular connective tissue plays an important role in how muscle responds to mechanical stress.[8] It has been shown, that during the early phase of immobilization, loss of muscle length is primarily due to shortening of muscle-associated connective tissue, which is only later followed by actual shortening of muscle fibers.[4]

The perimascular connective tissue of paraspinal muscles is attached to the thoracolumbar fascia i.e., the posterior layer of thoracolumbar fascia, which covers the paraspinal muscles and offers attachment for extrinsic back (shoulder girdle and serratus posterior) muscles and also anterolateral abdominal wall. Via these attachments, the posterior layer is capable of transferring loads between the trunk and extremities. The posterior layer of the thoracolumbar fascia extends from the sacral region through the thoracic region as far as splenius muscle and ligamentum nuchae of the cervical spine. It consists of two laminae: superficial and deep lamina. Priscilla et al. concluded that superior extent of the superficial lamina is upto rhomboids and splenius muscle and inferiorly it has extensive attachments to gluteus medius and maximus, and to the sacrum and ilium.[9] The deep lamina has been reported to blend with thin fascia over splenius cervicis. It is continuous inferiorly with the sacrotuberous ligament and via it with biceps femoris.

Facial continuity between the low back and shoulder girdle and neck muscles respectively, suggests that there is a potential anatomic conduit for the chronic low back pain. Additional attachments to muscles of the upper limb, cervical spine, and head support the view that the posterior layer is capable of transferring loads between this regions.[10] Langevin et al. found that, during a standardized passive flexion test in chronic low back pain patients, shear strain was reduced by ~20 per cent in thoracolumbar fascia, compared to the No-LBP subjects.[3] They concluded the reduced shear strain may result from impaired neuromuscular control and recruitment patterns of these muscles during trunk movements which has been shown to

be associated with chronic LBP. Alternatively, the altered muscle recruitment patterns could lead to altered forces being transferred to the connective tissues, which could cause remodelling as can occur in other types of connective tissues such as ligaments and joint capsules. Over time, the altered movement patterns could worsen connective tissue adhesions resulting in increased movement restriction, especially in the presence of pain and inflammation refs. Mellin et al. investigated joints and spinal mobility associated with low back pain.[11] They concluded that spine, hip and shoulder mobility was significantly smaller in chronic low back pain patients when compared with those without back pain. They also concluded that thoracolumbar mobility had higher correlations with LBP than mobility of the lumbar spine. Thoracic spinal mobility alone also correlated with LBP.[12]

The results suggest that ligamentous or capsular stiffness of the joints may be associated with low back pain. It can be concluded that there may be fascial involvement (tissue stiffness due to adhesion and fibrosis), altered sensory input and reduced shear strain in thoracolumbar fascia and hypomobility in thoracic spine in chronic low back patient due to PIVD. Evidences suggest that fascial stretching may alleviate these secondary changes in the fascia and spinal mobilization may increase the spinal mobility. Considering these facts and incident of chronic low back pain due to herniated disc, purpose of the study is to see the effect of upper back fascia and periscapular muscles stretching to reduce symptoms in subjects with chronic low back pain due to lumbar disk herniation.

Method

Type of study: Experimental

Research design: The Pre-Test, Post-Test Control Group Study Design which is experimental in nature.

Total sample consists of 40 subjects (29 males and 11 females), (age range 22–53 years with mean age was 34) with prolapsed intervertebral disc characterized by a chief complaint of low back pain with dermatomal pain distribution radiating down leg, obliterated lumbar lordosis with trunk deviation away from the painful side as found on inspection, lumbar flexion, extension and side flexion to the painful side reproduce original symptom; and leg pain more than back pain, centralization phenomenon with repeated back extension, a positive straight leg raising (SLR) at less than 60[degrees] reproducing the leg pain; A MRI scan demonstrating a herniated nucleus pulposus in lumbar spine.

Inclusion criteria

Subjects having low back pain for more than three months, age between 20–55 years in both the sex fulfilling the McKenzie posterior derangement syndrome i.e., centralisation phenomenon with repeated extension. Compression of periscapular muscles (levator scapulae/ rhomboids) by manual pressure reproduced the original low back pain with or without radiation.

Exclusion criteria

Age above 60 years, Low back ache due to tumour, spondylolisthesis, spondylolysis, osteoporosis, previous back surgery, known Rheumatic, neurological, or mental diseases, recent trauma or fracture and any other conditions contraindication to manual therapy.

Procedure

A total of 40 subjects both males and females (29 males and 11 females), were evaluated with the mean age of (33.55±5.66) years and average duration of LBP (6.03±2.16) months, recruited from the Department of Physiotherapy, SVNIRTAR based on the fulfilment of the inclusion and exclusion criteria.

The procedure was explained to them. They were given verbal instructions about the study and informed consent was taken prior to their participation in the study. The subjects were randomly divided into two groups.

Before initiating the treatment, subjects were assessed for baseline values of all the dependent variables. Pain by **Visual analogue scale (VAS):** The visual analogue scale is a numeric rating scale which can be used to quantify pain. The simplest VAS is a straight line of 100mm length. The ends are defined as the extreme limits of pain oriented from left to right. It is shown to be valid and sensitive and has a reasonable degree of reproducibility.[13]

Lumbar spine range of motion by Modified Schober's Test: it is a reliable clinical measurement method of lumbar flexion range of motion. The modified Schober method of determining lumbar spinal motion has been shown to be the most reliable than fingertip-to-floor method and two-inclinometer method for a routine, noninvasive, clinical evaluation of lumbar spinal motion.[14]

Functions by Oswestry Disability Questionnaire: it is an effective method for measuring disability in patients with LBA, high degree of severity and different causes. It includes 10 six- point scales. Sum of 10 ODI scores is expressed as a percentage of maximum scores and if patient fails to

complete a section per cent scores is adjusted. 1st section rates the intensity of pain and remaining 9 cover the disabling effects of pain on write out ADLs.

Group 1 (Experimental): 20 subjects (14 males and 6 females, with the mean age 34±5.49 and average duration of LBP (6.15±2.01) months, was given stretching of upper back fascia and periscapular muscles by slow and sustained manual pressure along the direction of the muscle and fascia, which was maintained at the end range for a few second and Maitland's rhythmic graded thoracic mobilization with discomfort along with conventional exercises (Cyriax listing correction I followed by repeated McKenzie back extension exercise and Core muscle strengthening) for five days in a week for three weeks.[15,16]

Group 2 (Conventional): 20 subjects (15 males and 5 females, with the mean age 33±5.94 and average duration of LBP (5.9±2.35) months,) received Cyrix listing correction- I followed by repeated McKenzie back extension exercise and Core muscle strengthening five days in a week for three weeks.[15,16]

Then at the end of three weeks recording of changes in pain on VAS, ROM measure and ODI score were measured.

Data analysis

Statistical analysis was performed using SPSS version 23.0
The dependent variables were analysed using repeated measures ANOVA.

There was one between factor (group) with two levels (groups: stretching and conventional therapy alone) and one within factor (time) with two levels (pre-test and post-test).

All pair wise post-hoc comparisons were analysed using a 0.05 level of significance.

Results

OSWESTRY Disability index (ODI)

As depicted in Figure 1 there was a significant reduction in ODI score in both groups from pre-treatment measurement over a period of 3 weeks, with reduction being significantly more in experimental group than conventional exercise group.

There was a main effect for time F (1,28,0.05)=1057.434, p=0.000. There was also a main effect for group F (1,28,0.05)=19.602, p=0.000. The main effect was qualified into time X group interaction F (1,28,0.05)=54.902, p=0.000. Tukey's Post HOC analysis revealed that both the experimental and control group improved but experimental group improved significantly better than conventional group, from pre-to post test.

Flexion ROM

Figure 2 illustrates that there was improvement in lumbar flexion ROM in both the groups following treatment for three weeks. The experimental group showed greater improvement in the post- treatment measurements as compared to the conventional group.

There was main effect for time F (1,28,0.05)=178.770, p=0.000 There was also a main effect for group F (1,28,0.05)=5.113, p=0.030 The main effects were qualified into time X group interaction F (1,28,0.05)=9.058, p=0.001. Tukey's Post Hoc analysis shows that there was a significant improvement in score for both the groups. However, the experimental group showed significantly greater improvement than conventional group at the end of three weeks.

Extension ROM

Figure 3 illustrates that there was improvement in Extension ROM in both the groups following treatment for three weeks. The Experimental group showed greater improvement in the post- treatment measurements as compared to the Conventional group.

There was main effect for time F (1,28,0.05)=289.949, p=0.000 There was also a main effect for group F (1,28,0.05)=4.415, p=0.042 The main effects were qualified into time X group interaction F (1,28,0.05)=6.734, p=0.013. Tukey's Post Hoc analysis shows that there was a significant improvement in score for both the groups. However, the experimental group showed significantly greater improvement than conventional group at the end of three weeks.

Side flexion ROM to affected side

Figure 4 illustrates that there was improvement in Side flexion ROM to affected side in both the groups following treatment for three weeks. The experimental group showed greater improvement in the post- treatment measurements as compared to the exercise therapy group.

There was main effect for time F (1,28,0.05)=235.204, p=0.000 There was also a main effect for group F (1,28,0.05)=5.272, p=0.027 The main effects were qualified into time X group interaction F (1,28,0.05)=15.448, p=0.000.

Tukey's Post Hoc analysis shows that there was a significant improvement in score for both the groups. However, the experimental group showed significantly greater improvement than conventional group at the end of three weeks.

Side flexion ROM to unaffected side

Figure 5 illustrates that there was improvement in side flexion ROM to unaffected side in both the groups following treatment for three weeks. The experimental group showed greater improvement in the post- treatment measurements as compared to the conventional group.

There was main effect for time F (1,28,0.05)=111.234, p=0.000 There was also a main effect for group F (1,28,0.05)=4.322, p=0.044 The main effects were qualified into time X group interaction F (1,28,0.05)=10.490, p=0.002. Tukey's Post Hoc analysis shows that there was a significant improvement in score for both the groups. However, the experimental group showed significantly greater improvement than conventional group at the end of three weeks.

Pain in VAS

Figure 6 illustrates that there was weekly improvement in VAS for pain in both the groups following treatment in three weeks. The Experimental group showed greater improvement in the post- treatment measurements as compared to the Conventional group.

There was main effect for time F (1,28,0.05)=905.209, p=0.000 There was also a main effect for group F (1,28,0.05)=12.507, p=0.001 The main effects were qualified into time X group interaction F (1,28,0.05)=23.862, p=0.000. Tukey's Post Hoc analysis shows that there was a significant improvement in score for both the groups. However, the experimental group showed significantly greater improvement than conventional group at the end of three weeks.

Discussion

Overall results of the study, both experimental group and conventional group showed improvement in pain measured by VAS, function measured by ODI and ROM measured by modified Schober's test after three weeks of intervention. However, experimental group improved to a greater extent in pain, spinal ROM and function than the control group.

Pain (VAS)

In this study both the groups showed significant reduction in pain over time, but after completion of study, experimental group showed more reduction in pain every week than conventional group.

The performance of repeated movements in the opposite direction would result in a reduction of the derangement and reduction or centralisation of pain. Most McKenzie method exercises are intended to directly and promptly diminish and eliminate patients' symptoms by providing beneficial and corrective mechanical directional end-range loads to the underlying pain generator.[17] Al-Obaidi et al. reported that the McKenzie intervention was effective in the treatment of individuals with discogenic CLBP who demonstrate complete or partial pain centralization.[18] Al-Obaidi et al. following McKenzie intervention for a cohort on CLBP was able to demonstrate significant improvements in all physical performances that remained stable 2-months following intervention.[18] Petersen et al. found that the effectiveness of the McKenzie method was stable in reducing CLBP disability after 2-months follow up.[19]

Core stabilization exercises strengthen the local stabilizers and deep muscles of the back, enhance coordination, enhance trunk stabilization and reduce the pressure on spine and subsequently reduce the low back pain.[20] In the present study, there was significant reduction in pain and improvement in function (ODI) was probably because of effects of stabilising exercises on local stabilizers and deep muscles of the back which leads to increased spinal stability, restrain aberrant micro-motion and reduced associated pain. Studies have shown that compared to general exercise, core stability exercise is more effective in decreasing pain and may improve physical function in patients with chronic LBP.[21]

The experimental group showed more improvement in pain every week than control group. In addition to the McKenzie repeated extension exercises and core stabilisation exercises experimental group received periscapular muscle stretching, upper back fascia stretching and thoracic mobilisation. Effects of stretching of the periscapular muscle, stretching upper back fascia and thoracic mobilisation, may be responsible for the additional improvement in VAS (pain).

Nociceptors that are located in the fascia recognises the pain stimulus which becomes sensitized when chronically stimulated. As pain is considered as an autonomic nervous system phenomenon, this facilitation of the receptors located in the fascia triggers a sympathetic response which was termed as sympathetic tone Shea M, thereby reducing the threshold of pain sensitivity in the subjects resulting in

severe pain.[22] Facilitation of the proprioceptive receptors (Ruffini and Pacini corpuscles) that are located in the fascia during the application of the stretch inhibited the sympathetic facilitation. This inhibition of sympathetic tone further reduced the perception of pain.[22]

The mechanical stimulus of a manual therapy technique triggers a cascade of neurophysiological effects. Studies suggest that these neurophysiological mechanisms result in hypo-algesic responses in patients with musculoskeletal pain.[23,24] Patients with chronic pain conditions often demonstrate increased rates and magnitudes of temporal sensory summation (TSS) compared with pain-free individuals.[25] TSS is a specific short-lasting aspect of central sensitization of the nervous system. TSS is characterized by a progressive increase in output from dorsal horn neurons in response to repeated unchanging low-frequency nociceptor stimuli (Like pain in Chronic LBP due to PIVD).[26]

Repeated exposure to increased nociceptor activity resulting from windup can cause facilitated transmission in dorsal horn neurons and long-lasting changes in synaptic properties.[27] This in turn could drive long-lasting changes in dorsal horn and central nervous system excitability resulting in reduced thresholds to future episodes of nociception. This may be one of the reason for chronicity of pain in cLBP patients.

Bishop et al. speculate that an intervention (SMT at upper thoracic spine) that reduces TSS may inhibit or reduce the potential for central sensitization in maintaining musculoskeletal pain. Additionally, interventions that inhibit or reduce TSS may prevent long-term facilitation from occurring, preventing the progression to central sensitization and persistent pain states.[27]

As the study of Bishop et al. showed there were reductions in TSS that occurred in both the upper and lower extremity.[27] Converging evidence from experimental studies of nociception in animal models suggests that the observed phenomenon (changes in TSS distal to the spinal level of application) could be mediated by propriospinal neurons projecting from the lower cervical cord to the lumbar spine. For example, cervical propriospinal neurons mediate inhibition of neurons in the dorsal horn of cats,[28] and primates.[29] Also, and more specifically, Sandkuhler et al. concluded that propriospinal neurons from the cervical and thoracic cord modulate thermally evoked noxious responses of lumbar dorsal horn neurons.[30] Furthermore, activation of capsacin-sensitive vanilloid receptors in cervical muscles of cats increases the neuronal activity of the cervical and lumbar dorsal horns.[31,32]

Considering above mentioned facts we hypothesise that cervicothoracic SMT given in this study may have an effect on producing hypoalgesia in the areas supplied by the lumbar spine for example pain due to lumbar PIVD.[33]

Range of motion (ROM)

In the present study, there was significant improvement in all the Range of Motions of lumbar spine in both the groups at the end of the study, but experimental group showed more improvement in lumbar ROM than conventional group.

A high proportion of patients who fit the derangement classification demonstrate a limitation of extension range, which improves when treatment procedures that cause a reduction, abolition, or centralization of symptoms, are applied. The performance of repeated movements in the opposite direction would result in a reduction of the derangement and reduction or centralisation of pain and in this way, facilitates spinal extension and gain in extension range. McKenzie passive lumbar extension exercises accentuates momentarily the lordosis and by stretching the shortened periarticular tissues and restore the soft tissue to their original length, thus, correct the dysfunction and increase the ROM.

During the performance of a specific stabilization exercise, patients learn how to recruit the deep muscles of the spine and gradually reduce undesirable excessive activity of other muscles.[34] Another benefit of the CORE exercise program is the restoration of coordination and control of the trunk muscles to improve control of the lumbar spine and pelvis.[35] It is assumed that the CORE exercise program can restore the function of weakened muscles in CLBP patients and augment the ability to support and control the spine and pelvis, thereby alleviating mechanical irritation and pain, ultimately reducing spasm in the low back region. As the protective spasm reduced and muscle strength increased, restriction of trunk movement got reduced and in turn the active spinal range increased.

Myers[36] suggests that stretching can also be applied to 'stuck layer' problems by fixing one layer and appling stretching movement of the adjacent layer, shear stress is created that allows the restoration of increased relative movement between the adjacent planes of fascia Schwind.[37] Inefficient functioning of thoracolumbar fascia in CLBP due to PIVD can be due to fibrotic changes of muscle with loss of elastic properties. Improving length of the fibrotic muscles

will improve the mobility of the lumbar spine and may help in pain relief. Stretching of the levator scapulae in prone lying with the arms crossed across the chest relieves the tightness of the stiff structures that are attached to the Thoraco lumbar Fascia (TLF), thereby increasing the flexibility of the spine.[38]

The fascia demonstrates lengthening in response to an applied uniaxial stretch.[39] Perhaps mechanical stress due to stretching performed in this study might be enough to induce a gel-like state in the fascia leading to increased soft-tissue compliance and subsequently greater lumbar spine ROM.

Increase hydration of thoracolumbar fascia due to upper back fascia stretching may help to increase the extensibility of thoracolumbar fascia and in turn contribute to additional increase in spinal range of motion in experimental group.[40] Since each muscle slip attaches to fascial expansions that then attach to periosteum–ligaments–joint capsules, which ultimately attach to bone, a stretch designed to target a supposedly 'isolated' muscle can be directed laterally, obliquely, or longitudinally to other nearby structures.[41] Thus, stretching of Periscapular muscle also stretch the attached fascia to it and in turn contribute to the increase in Spinal range of motion.

The additional effect of thoracic mobilisation i.e. stretching of tightened structures and increase mobility between the motion segments may be the reason for the greater Improvement in experimental group. Similar results have been seen in other studies with spinal mobilisation in increasing spinal range of motion.[38,42,43]

Function (ODI)

At the end of study both the groups showed reduction in Oswestry Disability Index over time. The improvements in both the groups were significantly different from each other. The difference in improvement of ODI was better in experimental group.

Normal pain free ROM is essential for normal function.[44] This hold true for any joint in the body and accordingly for the lumbar spine. The component of ODI viz. pain intensity, personal care, lifting, walking, sitting, standing, sleeping, social life, travelling and employment are directly related to patient's pain. The reduction in ODI scores seen in both the groups may be due to reduction of pain and improvement in ROM.

Higher effects on pain tend to be paired with higher use of physical function sentence unclear. More reduction in ODI score and more Increase in ROM in experimental group may explain the better improvement in function in experimental group. Similar results were found in the systematic review of Cross et al.[45]

Clinical implication

As in PIVD in lumbar segment, the lumbar spine is the site of pathology. Manual therapy application directly over the lumbar spine will not be safe as it may further aggravate the pathology. Manual therapy techniques applied to a distant part which is connected to the lumbar spine that reduces the symptoms would be safe and beneficial to the patient. So, stretching of periscapular muscles and fascia of the upper back and mobilisation of upper thoracic spine can be a treatment of choice in addition to conventional physiotherapy in patients with chronic low back pain with or without radiation to lower extremity due to PIVD to reduce pain and improve spinal ROM and function.

Limitations

Sample size was small, short duration of study and no follow up to see long term effects.

Conclusion

Chronic low back pain with or without radiation to lower extremity due to PIVD can be associated with myofascial pain syndrome of periscapular muscles and cervicothoracic extension dysfunction. Stretching of periscapular muscles and fascia of the upper back and mobilisation of upper thoracic spine was found to be effective for the management of chronic low back pain due to PIVD.

References

1. Schwarzer AC, Aprill CN, Derby R, et al. The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. Spine. 1995;20:1878–83.
2. DePalma MJ, Ketchum JM, Saullo T. What is the source of chronic low back pain and does age play a role? Pain Med. 2011;12:224–233.
3. Langevin HM, Fox JR, Koptiuch C, et al. Reduced thoracolumbar fascia shear strain in human chronic low back pain. BMC Musculoskelet Disord. 2011;12:203.
4. Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. Med Hypotheses. 2007;68(1):74–80.
5. Cummings GS, Tillman LJ. Remodeling of dense connective tissue in normal adult tissues. In: Currier DP, Nelson RM, editors. Dynamics of human biologic tissues

Contemporary perspectives in rehabilitation, vol. 8. Philadelphia: F.A. Davis; 1992;45–73.
6. Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. Med Hypotheses. 2007;68(1):74–80.
7. Leask A, Abraham DJ. TGF-beta signaling and the fibrotic response. FASEB J. 2004;18(7):816–27. doi: https://doi.org/10.1096/fj.03-1273rev
8. Langevin HM. Potential role of fascia in chronic musculoskeletal pain. Integrative Pain Medicine. 2008;11:123–32.
9. Barker PJ, Briggs CA. Attachments of the posterior layer of lumbar fascia. Spine (Phila Pa 1976). 1999;24(17):1757–1764.
10. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, et al. The posterior layer of the thoracolumbar fascia: Its function in load transfer from spine to legs. Spine (Phila Pa 1976). 1995;20:753–8. doi: https://doi.org/10.1097/00007632-199504000-00001
11. Melling G, Harkapaa K, Hurri H, et al. A controlled study on the outcome of inpatient and outpatient treatment of low back pain. Part IV. Long term effects on physical measurement. Scand J Rehab Med. 1999;22:189–194.
12. Mellin G. Correlations of spinal mobility with degree of chronic low back pain after correction for age and anthropometric factors. Spine (Phila Pa 1976). 1987. doi: 10.1097/00007632-198706000-00008
13. Murrin KR, Rosen M. Pain measurement. In: Smith G and Govindo BG (editors) Acute pain: London: Butterworth. 1985.
14. GiU K, Krag MH, Johnson GB, et al. Repeatability of four clinical methods for assesment of lumbar spineal motion. Spine. 1988;13(1):50–53.
15. Cyriax JH, Cyriax PJ. Illustrated manual of orthopaeic medicine. 2nd edition. Butterwort Heinemann. 1993.
16. McKenzie RA, May S. The lumbar spine: Mechanical diagnosis and therapy (2nd Ed). Waikanae, New Zealand, Spinal Publications. 2003.
17. Wall PD. The dorsal horn. In: Wall PD, Melzack R, editors. Textbook of Pain. 2nd ed. Edinburgh, UK: Churchill Livingstone; 2006.
18. Al-Obaidi SM, Al-Sayegh NA, Ben Nakhi H, et al. Evaluation of the McKenzie intervention for chronic low back pain by using selected physical and bio-behavioral outcome measures. PMR. 2011;3(7):637–646. doi: https://doi.org/10.1016/j.pmrj.2011.04.025
19. Petersen T, Kryger P, Ekdahl C, et al. The effect of McKenzie therapy as compared with that of intensive strengthening training for the treatment of patients with subacute or chronic low back pain: A randomized controlled trial. Spine (Phila Pa 1976). 2002;27:1702–1709. doi: https://doi.org/10.1097/00007632-200208150-00004
20. Bergmark A. Stability of the lumbar spine: A study in mechanical engineering. Acta OrthopScand. 1989;230(Suppl):20–4.
21. Liddle SD. Exercise and chronic low back pain: What works? Pain. 2004;107(1-2):176–90. doi: 10.1016/j.pain.2003.10.017
22. Myofascial Release Textbook, Michael Shea, Ph.D Somatic Psychology, Self- Published, 1995;45–56.
23. Bialosky JE, Bishop MD, Robinson ME, et al. The influence of expectation on spinal manipulation induced hypoalgesia: an experimental study in normal subjects. BMC Musculoskelet Disord. 2008;9:19.
24. Bialosky JE, Bishop MD, Robinson ME, et al. Spinal manipulative therapy has an immediate effect on thermal pain sensitivity in people with low back pain: a randomized controlled trial. Phys Ther. 2009;89:1292–303.
25. Price DD, Staud R, Robinson ME, et al. Enhanced temporal summation of second pain and its central modulation in fibromyalgia patients. Pain. 2002;99:49–59.
26. Woolf CJ, Salter MW. Neuronal plasticity: increasing the gain in pain. Science. 2000;288:1765–9.
27. Bishop MD, Beneciuk JM, George SZ. Immediate reduction in temporal sensory summation after thoracic spinal manipulation. Spine J. 2011;11(5):440–446.
28. Kalezic I, Pilyavskii AI, Maisky VA, et al. Distinctive pattern of c-fos expression in the feline cervico-lumbar spinal cord after stimulation of vanilloid receptors in dorsal neck muscles. Neurosci Lett. 2004;364:94–7.
29. Hobbs SF, Oh UT, Chandler MJ, et al. Evidence that C1 and C2 propriospinal neurons mediate the inhibitory effects of viscerosomatic spinal afferent input on primate spinothalamic tract neurons. J Neurophysiol. 1992;67:852–60.
30. Sandkuhler J, Stelzer B, Fu QG. Characteristics of propriospinal modulation of nociceptive lumbar spinal dorsal horn neurons in the cat. Neuroscience. 1993;54:957–67.
31. Price DD, Bennett GJ, Rafii A. Psychophysical observations on patients with neuropathic pain relieved by a sympathetic block. Pain. 1989;36:273–88.
32. Pilyavskii AI, Maznychenko AV, Maisky VA, et al. Capsaicin-induced effects on cfos expression and NADPH-diaphorase activity in the feline spinal cord. Eur J Pharmacol. 2005;521:70–8. doi: https://doi.org/10.1016/j.ejphar.2005.08.006
33. Haavik-Taylor H, Murphy B. Cervical spine manipulation

alters sensorimotor integration: a somatosensory evoked potential study. Clin Neurophysiol. 2007;118:391–402.
34. Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine. 2001;26:E243–E248.
35. Hodges PW. Core stability exercise in chronic low back pain. OrthopClin North Am. 2003;34:245–254.
36. Myers T. Anatomy trains. Churchill Livingstone, Edinburgh. 2009.
37. Schwind P. Fascia and membrane technique. Churchill Livingstone, Edinburgh. 2006.
38. Mohanty PP, Pattanaik M. Manual Therapy for the Management of Lumbar PIVD- an Innovative Approach. Indian Journal of Physiotherapy and Occupational Therapy. 2015;9(4):98.
39. Iatrides J, Wu J, Yandow J, et al. Subcutaneous tissuemechanical behavior is linear and viscoelastic under uniaxial tension. Connect Tissue Res. 2003;44(8):208–217.
40. Puxkandl R, Zizak I, Paris O, et al. Viscoelastic properties of collagen— synchrotron radiation investigations and structural model. Philos Trans R Soc Lond B Biol Sci. 2002;B357:191–197.
41. Franklyn-Miller A, Falvey E, Clark R. The strain patterns of the deep fascia of the lower limb. Fascial Research II: Basic Science and Implications for Conventional and Complementary Health Care. Elsevier, Munich. 2009.
42. Youn-Bum Sung. Effects of Thoracic Mobilization and Manipulation on Function and Mental State in Chronic Lower Back Pain J Phys Ther Sci. 2014;26(11):1711–1714.
43. Crisco JJ, Panjabi MM. The intersegmental and multisegmental muscles of the lumbar spine. A biomechanical model comparing lateral stabilizing potential. Spine. 1991;16(7):793–9.
44. Drezner J, Herring S. Managing low-back pain: steps to optimize function and hasten return to activity. Phys Sportsmed. 2001;29(8):37.
45. Cross KM, Kuenze C, Grindstaff T, et al. Thoracic spine thrust manipulation improves pain, range of motion, and self-reported function in patients with mechanical neck pain: A systematic review. J Orthop Sports Phys Ther. 2011;41(9):633–642.

ACKNOWLEDGEMENTS

All the participants of the study for their consent and cooperation

PEER REVIEW

Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

FUNDING

None

ETHICS COMMITTEE APPROVAL

Institute Ethical Committee; Letter No: DPT 9A 15 dated 25.01.2013

Figure 1: Pre-and post ODI scores of Experimental and Conventional Group

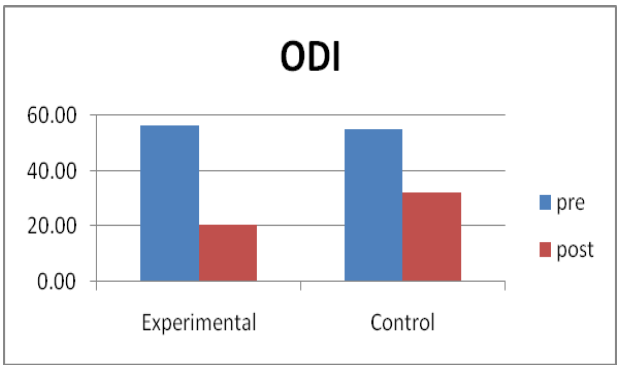

Figure 2: Pre-and post-lumbar flexion ROM of Experimental and Conventional Group

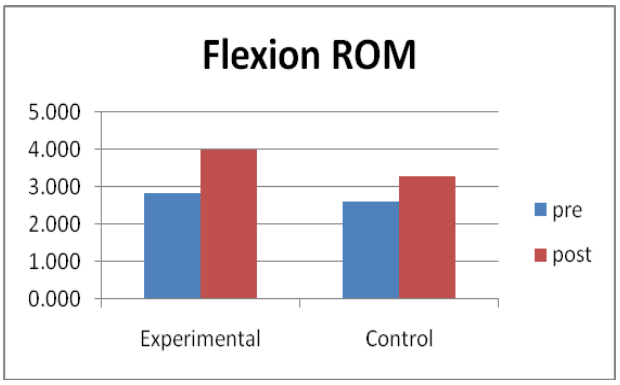

Figure 3: Pre-and post-lumbar extension of Experimental and Control Group

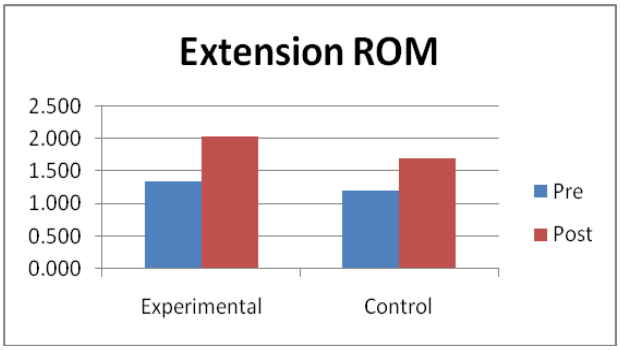

Figure 4: Pre-and post-lumbar Side Flexion ROM affected side of Experimental and Conventional Group

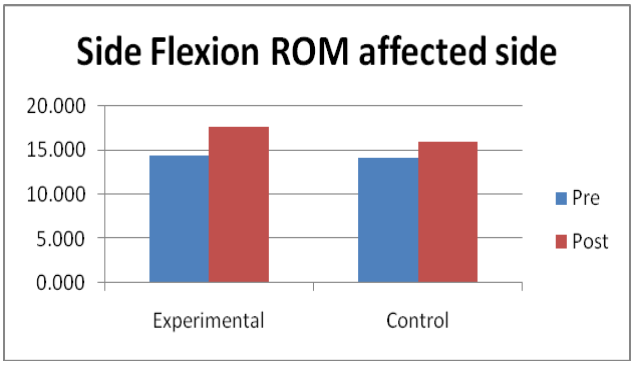

Figure 5: Pre-and post-lumbar Side Flexion ROM unaffected side of experimental and conventional group

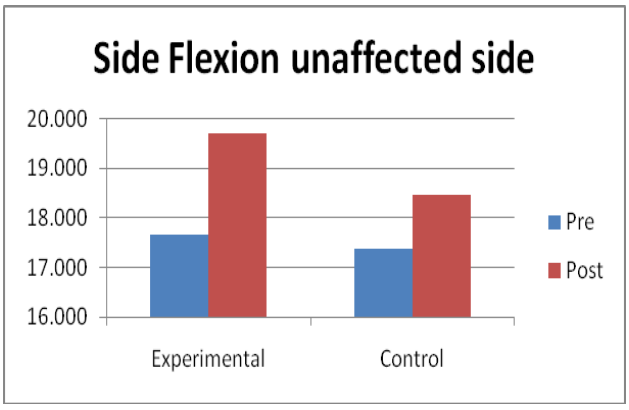

Figure 6: Pre-and post-scores of VAS of experimental and control group