The Role of the Lumbar Multifidus in Chronic Low Back Pain: A Review

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Low back pain (LBP), a highly prevalent problem in society, is often a recurrent condition. Recent advances in the understanding of the biomechanics of LBP have highlighted the importance of muscular stabilization of the “neutral zone” range of motion in the low back. The lumbar multifidus muscles (LMM) are important stabilizers of this neutral zone, and dysfunction in these muscles is strongly associated with LBP. The dysfunction is a result of pain inhibition from the spine, and it tends to continue even after the pain has resolved, likely contributing to the high recurrence rate of LBP. Persisting LMM dysfunction is identified by atrophic replacement of multifidus muscle with fat, a condition that is best seen on magnetic resonance imaging. Muscle training directed at teaching patients to activate their LMM is an important feature of any clinical approach to the LBP patient with demonstrated LMM dysfunction or atrophy.

INTRODUCTION

Low back pain (LBP) is a highly prevalent problem in society; approximately 60% to 80% of the population will experience an episode of LBP during their lifetime, and 60% to 86% of these people will have more than one episode of LBP [1,2]. A relatively recent shift in the view of LBP has gone from classifying it as a self-limited acute condition to a recurrent syndrome [3]. The purpose of this review is to discuss the role of the lumbar multifidus muscles (LMM) in recurrent LBP, as well as to discuss literature-based clinical approaches to assessment and treatment of multifidus dysfunction.

Spinal stabilization therapy has been observed to be more effective over time in treating LBP than minimal intervention and exercise therapy alone, and has also been observed to reduce pain, disability, and medication intake, as well as recurrence rates [4,5]. As a consequence, treatment focus has shifted to reactivation and strengthening of the smaller muscles of the spine to improve long-term stabilization of the vertebral column.

Biomechanical research has increased the understanding of mechanisms of low back injury and pain, specifically regarding the ability to stabilize the “neutral zone” of the lumbar spine with tonic muscle control. Panjabi [6] has described the neutral zone as the part of the range of intervertebral motion, measured from the neutral position, in which spinal motion can occur with minimal nonmuscular passive resistance from the spine. Suni and colleagues [2] described a randomized controlled trial of a neuromuscular training program directed at neutral zone stabilization on a population of patients with recent back injury. These authors demonstrated a significant decrease in the intensity of LBP in the treatment group that was not seen in the control group, and concluded that control of the lumbar neutral zone was an important component of LBP and disability prevention.

MULTIFIDUS FUNCTION

It is well established that the LMM are important stabilizers of the lumbar neutral zone; Wilke et al [7] found that the actions of the multifidi account for more than two thirds of the stiffness of the spine when in the neutral zone. In comparison with all lumbar muscles, the LMM are short and stout, with a high cross-sectional area (CSA) and short muscle fibers. Furthermore, these...
The authors found that the level of LMM asymmetry correlated with the neurologic assessment of the LMM using MRI, computed tomography (CT), and ultrasound (US). Kjaer and colleagues [12] evaluated the lumbar magnetic resonance imaging (MRI) results for 412 adult and 442 adolescent subjects in a cross-sectional study of LMM atrophy. These authors categorized the degree of observed atrophy in the LMM as none, slight, and severe, and correlated the findings with complaints of LBP. They found that fatty infiltrations of the LMM were strongly associated with LBP in adults, and that the association was independent of body mass index (BMI).

Kader et al [13] performed a retrospective study of 78 patients with LBP and either with or without leg pain. The authors assessed the correlation between MRI changes in the LMM and leg pain. It was reported that LMM atrophy was present in 80% of the patients with LBP and that there was a significant correlation between LMM atrophy and referred leg pain. The authors theorized that LMM atrophy may be caused by dorsal ramus syndrome, which has been described as LBP with referred leg pain, produced by irritation of anatomic structures supplied by the dorsal ramus nerve, i.e., the facet joints and LMM. The authors further concluded that abnormalities of the LMM may explain referred leg pain in the absence of other MRI abnormalities.

Hides et al [14] described a study of 26 subjects with unilateral LBP, some with referred lower extremity pain. The authors assessed LMM atrophy among the subjects using ultrasound imaging, noting marked asymmetry of the LMM that was localized to a single vertebral level in most cases. The authors noted that the level of LMM asymmetry correlated with the neurologic level of the symptoms in 24 of the 26 subjects in the study.

**IMAGING OF MULTIFIDUS ATROPHY**

There are numerous publications describing the morphologic assessment of the LMM using MRI, computed tomography (CT), and ultrasound (US).

Kader et al [13] used MRI in evaluating LMM atrophy, which they defined as muscular replacement with fat and fibrous tissues. The authors established a ranked grading scale for LMM atrophy consisting of mild, moderate, and severe, corresponding to atrophy in less than 10% of CSA of the LMM, more than 10% and less than 50%, and more than 50%, respectively (Figures 1-3). Two readers assessed the degree of LMM atrophy among the subjects, with good interobserver agreement noted.

Kjaer et al [12] described the blinded assessment of LMM atrophy in 854 MRI scans of adults and adolescents using a grading scale similar to that used by Kader et al [13]. The authors found good intraobserver and interobserver agreement among the readers for the adult group. The authors noted that fatty infiltration after LMM atrophy could be measured in a noninvasive manner using MRI.

Barker et al [15] performed MRI on 50 patients presenting to a back pain clinic with unilateral persisting LBP. The authors measured the CSAs of the left and right psoas and LMM and correlated their findings with the distribution and duration of symptoms among the subjects. A significant positive correlation between the side of LMM atrophy and the distribution of the LBP was found, as well as between the degree of atrophy and the duration of the symptoms.

CT has been evaluated as a relatively lower-cost and more accessible alternative to MRI for assessment of LMM atrophy. Danneels et al [16] described a study of CT assessment of 32 LBP patients and 23 active volunteers. The study design included CT of the CSA of the LMM at 3 levels. The authors found macroscopic atrophic changes in the LMM in 80% of the patients with LBP, and that the atrophy in the LMM at the lowest levels of the lumbar spine correlated significantly with LBP status. The authors concluded that the most important finding from their study was the finding of a significant correlation between LMM atrophy and radicular and nonradicular leg pain.

The utility and reliability of US relative to MRI scanning, have also been evaluated as a means of assessing LMM CSA. Hides et al [17] compared the CSA assessment of the LMM among healthy young adults using MRI and US, following a strict protocol. The authors determined that LMM CSA could be measured as accurately with US as with MRI, although they did not assess the ability of US to discriminate the degree of atrophy in the LMM.

**REFLEX INHIBITION MODEL OF LOCALIZED MULTIFIDUS ATROPHY**

Macintosh et al [11] described the morphology of the LMM by means of the dissection of 12 adult cadaver spines. In contrast to previous determinations that each LMM is innervated by a number of spinal nerves, these authors found that the LMM is divided into 5 distinct myotomes that are each innervated by a single spinal segment. They found that all muscle fibers attaching to the spinous process or lamina of a particular vertebra are segmentally innervated by the same nerve; the medial branch of the dorsal ramus that originates...
inferior to the respective vertebra (Figure 4). Thus, the muscle that moves a given spinal segment is supplied by the nerve for that segment. The authors postulated that from an anatomic perspective, the shared innervation of the zygapophyseal joints means that pain emanating from these joints could result in a reflex inhibition of the LMM at the same level.

Indahl et al [18] noted that the injection of saline into the zygapophyseal joint in a porcine model resulted in decreased activity of the multifidus muscle. They concluded that the effect of the injection was to activate a stretch reflex in the joint capsule, which in turn excited inhibitory interneurons in the spinal cord, which in turn inhibited the motor neurons and decreased the muscle response. The authors inferred that LMM atrophy seen in the human spine is a result of reflex inhibition caused by afferent feedback from the zygapophyseal joint, which in turn impedes the voluntary activation of the LMM. The authors pointed to their results as evidence that multifidus atrophy seen in back pain patients is more likely a result of dysfunction rather than disuse. The authors postulated that inhibitory discharges from the zygapophyseal joints may explain the efficacy of manual medical approaches to back pain such as manipulation and mobilization directed at the zygapophyseal joints.

Hodges et al [19] demonstrated, also in a porcine model, the rapid onset of LMM atrophy within 3 days after an experimentally induced nerve root injury. After transection of the medial branch of the L3 nerve root, the ipsilateral LMM CSA adjacent to the L4, L5, and L6 spinous processes was reduced by 13%, 20%, and 12%, respectively, by 72 hours after injury. The changes were isolated to the side of injury; there were no differences in the CSA or muscular activity levels of the contralateral LMM, as determined by US assessment. The authors also investigated the changes in the LMM after experimentally induced disk injury in the porcine model. A stab wound was introduced to the left anterolateral aspect of the L3-4 intervertebral disk, and this was followed by focal atrophy and a reduction of the CSA of the left LMM by 17%. The authors opined that it was unlikely that the injury resulted from LMM denervation (owing to nerve root injury) as the atrophy was only at the level of the disk, whereas single nerve roots innervate three levels of LMM. The authors noted that the traumatic changes in the LMM noted in their study were undoubtedly attributable to a reduction in neural drive to the muscle, but that the mechanism that accounted for the reduction was unclear.

Hides et al [20] provided further evidence that the pattern of LMM atrophy is local rather than general in a study that compared multifidus size and bilateral symmetry between chronic LBP patients and healthy asymptomatic subjects. The authors reported that at the L4 and L5 levels, asymptomatic subjects were found to have significantly larger multifidus muscles in comparison with chronic LBP patients. They also found that the greatest asymmetry was seen at the L5 vertebral level in patients with unilateral pain presentations. The authors concluded that their findings support a clinical approach to LBP that uses exercise therapy that focuses on localized muscle impairments.

Wallwork et al [21] demonstrated that LMM atrophy was associated with a reduction in the ability to voluntarily contract the muscle. The authors used diagnostic US to measure contractions of the LMM by comparing the thickness of the muscle at rest to when it was contracted. Study subjects with chronic LBP and LMM atrophy demonstrated significantly decreased ability to perform isometric contractions of their LMM. These study results reinforced the findings of prior authors and emphasized the clinical perspective that rehabilitation in LBP patients may need to target localized impairments in motor control.

**Multifidus Atrophy Treatment**

Danneels et al [4] assessed the efficacy of 3 different treatment modalities on multifidus CSA in chronic LBP patients. A group of 59 patients were randomly allocated to one of 3 programs: stabilization training, stabilization training combined with dynamic resistance, and stabilization training combined with dynamic-static resistance. Using CT scanning, LMM CSAs were measured before and after 10 weeks of training. The CSA of the LMM muscle was significantly increased at all vertebral levels only in the dynamic-static resistance training group. The authors concluded that the static holding component between concentric and eccentric contraction phases was critical to induction of muscle regeneration.

A randomized controlled trial performed by Van et al [22] used real-time visual feedback by means of US to improve the isometric contraction capability of the LMM in healthy sub-
The authors applied motor learning principles to train the subjects in voluntary contraction of the multifidus and gave verbal feedback to one group, and verbal and visual feedback to the other. They found that providing the subjects with visual feedback of their performance resulted in better quality muscle contraction of the LMM as well as better memory of how to contract the multifidi after reassessment a week later.

Sung [23] evaluated the question of whether an exercise program designed to increase the ability to contract the LMM improves functional status or reduces LBP. This author described a short-term cohort study that assessed the effect of a 4-week spinal stabilization exercise program in a group of LBP patients, reporting a significant improvement in LMM function, as well as a reduction in LBP disability.

Hides et al [5] described the effects of stabilization training on LMM CSA among elite athletes (cricketers) with LBP, concluding that LMM atrophy can exist in highly active, elite athletes. The authors demonstrated that the stabilization training increased the CSA of the LMM, and that the training was associated with a decrease in symptoms of LBP. Their results showed that specific training that aims at activation of the LMM is associated with both an increase in functional status and a reduction in LBP.

In an earlier study by Hides et al [24], the authors demonstrated that LMM function recovery is not necessarily associated with the resolution of painful symptoms in LBP patients. The authors studied a group of 39 patients with a first episode of acute unilateral LBP and corresponding unilateral segmental inhibition of the LMM. The patients were randomly allocated to 2 groups, only one of which performed specific exercises designed to reactivate the LMM through facilitating an isometric contraction of these muscles. The group that did not perform the exercises had decreased LMM size at a 10-week follow-up examination even though they had resumed normal levels of activity and had a remission of painful symptoms, whereas the group that performed the isometric contractions showed more rapid and complete LMM recovery. The authors suggested that the continued dysfunction of the LMM in such patients may be one reason for the high recurrence rates of LBP after an initial episode.

MacDonald et al [25] assessed muscular control of long and short LMM fibers in 15 recurrent LBP patients and 19 control subjects. The authors used EMG to determine whether LMM control at the L5 level differed between the groups. They found that there were changes in motor control of the LMM among the patients that primarily affected the shorter fibers, and that these changes were greater on the previously painful side of the low back. These findings support the use of a specifically designed stabilization exercise program directed toward the LMM and specifically the deeper shorter fibers that serve to stabilize the spine. The authors concluded that the abnormal pattern of muscle control they observed may leave the spine vulnerable to reinjury, thus predisposing the spine to recurrent episodes of LBP. The authors postulated that the observed motor control problem among the recurrent LBP patients implied that pain and functional performance should not be the only outcome measures after an acute episode of LBP.

**CONCLUSIONS**

The LMM are important stabilizers of the lumbar spine neutral zone, and atrophy of the muscle decreases the ability to
control the neutral zone and is strongly associated with LBP. This atrophy appears to help perpetuate an inhibitory feedback loop that begins with pain in the spine, possibly stemming from the intervertebral disks or zygapophyseal joints, followed by reflex inhibition of the multifidus, and then atrophy and fatty replacement of the muscle. Amelioration of back pain does not necessarily result in resumption of normal LMM function, and decreased LMM function is likely implicated in recurrent LBP. Muscle training directed at teaching patients to activate their LMM is an important feature of any clinical approach to the patient with LBP who demonstrates LMM dysfunction or atrophy.

REFERENCES


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