Long-Term Lumbar Multifidus Muscle Atrophy Changes Documented With Magnetic Resonance Imaging: A Case Series

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ABSTRACT

A retrospective case series of three patients with chronic low back pain who received baseline MRI scans revealing multifidus muscle atrophy with fatty replacement is provided. Each patient received spinal manipulative therapy, and two were compliant with low back exercises targeting the multifidus. A follow-up scan performed >1 year later was compared to the baseline scan revealing a decrease in atrophy with fatty replacement in the two patients who performed multifidus-focused low back exercises (15% and 39% on the left and 7% and 32% on the right respectively), and an increase in the patient who underwent spinal manipulation alone (41% and 53%). Interestingly, the decrease in atrophy in the two patients that performed the exercises correlated to functional improvements. Though limited, these results highlight the utility of MRI in quantifying positive and negative long-term changes in multifidus atrophy, which may be an indicator of recovery in chronic low back pain patients.

CASE SERIES

INTRODUCTION

Low back pain (LBP) is a highly prevalent and often recurrent problem (1) creating a major economic burden in the United States. A recent paradigm shift in the view of LBP has gone from viewing it as a self-limited acute condition to a recurrent syndrome (2). From a biomechanical perspective, the lumbar multifidus muscles (LMM) are important stabilizers of the lumbar spine, and dysfunction in the LMM has been found to play a role in chronic and recurrent low back pain (3). The LMM become neurologically inhibited following a low back injury and prolonged inhibition can lead to atrophy with fatty replacement, in which healthy muscle is replaced with fat (3).

This "fatty atrophy" is most readily identified on transverse views of magnetic resonance imaging (MRI) scans of the lumbar spine, and the presence of atrophy is strongly associated with LBP (4). We recently published a brief review of this pathology (3), and have included an anatomical drawing for reference (Fig. 1).

Danneels et al. (5) described clinical success with exercise therapy that was directed at improving the stabilizing function as well as decreasing the amount of fatty atrophy of the LMM as reported by computed tomography (CT) (6). While many previous studies utilizing MRI focus on changes in the total cross sectional area (CSA) of the LMM of study participants

(7-10), a recent study found no significant change in total CSA between LBP patients and control groups and notes that changes in fatty infiltration that decreases quality (less lean muscle tissue and increased fatty deposition) rather than quantity (total CSA) of the multifidus muscle may be more prognostic of recurrent LBP (11). In the present case series we describe three cases of chronic low back pain with MRI-documented and quantified multifidus atrophy. The aim of these reports is to demonstrate the utility of MRI in quantifying long-term changes in LMM fatty atrophy in chronic-LBP patients. As a pilot investigation regarding these issues, we describe MRI changes in LMM fatty atrophy in three chronic-LBP patients undergoing SMT; two with exercise and one without.

CASE DESCRIPTIONS

Three LBP patients presenting to a chiropractic clinic were selected for retrospective record and MRI review case reports based on the availability of baseline MRI scans showing LMM atrophy and LBP complaints. All three patients had chronic LBP complaints for more than six months, and baseline MRI (non-contrast T1-weighted) scans showing LMM atrophy prior to beginning treatment (Fig. 2-4 top panels). Each patient received spinal manipulative therapy (SMT) of the lumbar spine directed at the zygapophyseal (facet) joints. In addition, two of the patients performed regular lumbar stabilization exercises including LMM activation through isometric contractions. The third patient's pain levels made performing the lumbar stabilization exercises not feasible. All patients subsequently received follow-up MRI (non-contrast T1-weighted) scans after at least one year (Fig. 2-4 bottom panels).

The comparison of baseline and follow-up MRI scans was accomplished by identifying the 3 most anatomically comparable transverse images between the two scans, which were imaged at different facilities in all 3 cases at the lumbosacral junction. A retrospective qualitative interpretation of soft copy films was performed by a radiologist who was blinded regarding the timing of the scan and the therapeutic outcome. Further, a quantitative evaluation method described by Danneels et al. (6) was used to estimate the proportion of the LMM that had undergone fatty atrophy (average. of right and left of the three comparable images). In brief, this methodology is as follows: the LMM were isolated and a histogram of the pixel count by grayscale was used to quantify the proportion of fat versus muscle (as a percent of total LMM CSA) where fat appears at a higher intensity. Figures 2-4 demonstrate the three comparative transverse images for each patient with arrows highlighting areas that showed significant change; either positive or negative and the estimation of LMM atrophy with fatty replacement is provided.

Patient 1:
Patient 1 was a 28 year-old male marine engine mechanic who sustained a chronic LBP-producing injury while on-the-job and was completely disabled for 3 years by the time of initial presentation where he presented with LBP, muscle spasms and right leg numbness. The baseline lumbar MRI scan

(non-contrast T1-weighted sequences on a 1.5T scanner with TR: 627.0 and TE: 15.0) revealed L3-S1 disc narrowing, a 4 mm posterior disc protrusion at the L3-L4 level, a 1 to 2 mm posterior disc protrusion at L4-L5, and a left-sided disc herniation at L5-S1 that contacted the left S1 nerve root. Furthermore, the baseline MRI scan revealed asymmetric multifidus muscle atrophy at L5-S1: averages approx. 32.4% on the left and 23.4% on the right (Fig. 2). The patient was treated with SMT approximately 2 times per week for 3 years following initial presentation, and then treated 2 times per month for a subsequent nine months. The patient was educated in lumbar stabilization exercises emphasizing multifidus activation six months prior to SMT by a physical therapist and reportedly continued with a home LMM-strengthening program through the duration of SMT treatment. A follow-up MRI scan (non-contrast T1-weighted sequences on a 1.5T scanner with TR: 450.0 and TE: 10.0) performed 44 months after the baseline scan demonstrated a decrease in LMM atrophy: average approx. 27.5% on the left (15% decrease) and 21.8% on the right (7% decrease) (Fig. 2). At that time, the patient had markedly reduced pain and was able to return to full time work.

Patient 2:
Patient 2 was a 39 year-old male machinist who sustained a chronic LBP-producing injury while on-the-job two years prior to presentation and presented with LBP, pelvic pains and pains into his feet. The patient was able to continue working, but was unable to participate in recreational activities following injury. The baseline lumbar MRI scan (non-contrast T1-weighted sequences on a 1.5T scanner with TR: 650.0 and TE: 14.0) performed two years post-injury after initial presentation revealed slight narrowing of the intervertebral foramen bilaterally at L5-S1 and a posterior disc bulge of about 2-3 mm at the L4-L5 level creating minimal indentation of the anterior thecal sac. Additionally, the baseline MRI revealed asymmetrical multifidus muscle atrophy at L4-S1: average approx. 20.3% on the left and 13.0% on the right (Fig. 3). The patient was treated with SMT at a frequency of approximately 1 time per week for 6 years, and at the same time was educated in lumbar stabilization exercises emphasizing multifidus activation that he performed regularly at home. During the first two years of treatment the patient was able to successfully return to all pre-injury activities, with only episodic LBP thereafter. A follow-up MRI scan (non-contrast T1-weighted sequences on a 1.5T scanner with TR: 500.0 and TE: 13.1) performed 72 months after the baseline evaluation revealed a decrease in LMM atrophy: average approx. 12.3% on the left (39% decrease) and 8.8% on the right (32% decrease) (Fig. 3).

Patient 3:
Patient 3 was a 25 year-old male tile setter who sustained a chronic LBP-producing injury while on-the-job, which left him completely disabled from all work, continued to worsen, and later this patient developed right posterior leg pain. The baseline lumbar MRI scan (non-contrast T1-weighted sequences on a 1.0T scanner with TR: 621.0 and TE: 12.0) performed 12 months later revealed a right paracentral disc protrusion at the L4-L5 vertebral level, and a subtle left paracentral disc protrusion with mild disc degeneration

revealed at L5-S1. The baseline MRI also revealed symmetrical multifidus muscle atrophy at the level of L4-S1: average approx. 12.2% on the left and 11.6% on the right (Fig. 4). The patient was treated with SMT 2 times per week for 2 months, and was prescribed 3 sessions of supervised rehabilitation exercises for multifidus strengthening, which he was unable to complete due to LBP. The patient's symptoms worsened substantially over the 2 months of therapy and he was ultimately referred for a spine surgery evaluation. A follow-up MRI scan (non-contrast T1-weighted sequences on a 1.5T scanner with TR: 550.0 and TE: 13.1) performed twelve months after the baseline scan demonstrated an increase in LMM atrophy: average approx. 17.2% on the left (41% increase) and 17.8% on the right (54% increase) (Fig. 4). Ultimately the patient underwent a right-sided L4-L5 hemilaminotomy and diskectomy.

DISCUSSION

It is well recognized that the LMM are key components in the stabilization and control of the lumbar spine (12). The LMM are unique in that they can produce a large amount of force over a small operating range in comparison with longer muscles of the spine, and makes the LMM ideal for stability rather than movement (13). The LMM can be subdivided into superficial and deep fibers with different functions, and EMG studies have shown differential activation between them. The activity of the superficial fibers has been shown to control flexion and maintain spinal orientation while the activity of the deep fibers serves to control intersegmental motion (12, 14). Therefore, the deep fibers of the LMM are morphologically and biomechanically designed for stabilization. Interestingly, decreased activity in the LMM fibers has been reported after alterations to the facet joint capsule (15). Furthermore, rapid atrophy of the LMM was reported after damage to the medial branch of the lumbar nerve root or induced disc injury (16). Taken together, these findings suggest a facetogenic/discogenic reflex inhibition or dorsal ramus syndrome model for LMM atrophy (15-17).

Numerous studies have described morphological changes of the LMM using the soft-tissue imaging capabilities of MRI as well as computed tomography (CT) and ultrasound [reviewed in (3)]. While CT has long been used to evaluate changes in the LMM (6, 17), it requires moderate to high radiation and provides less tissue contrast than MRI, though the bony structures that partially define the location of the LMM such as the vertebral lamina and facet joints are readily visualized. Ultrasound has been shown to be as effective in measuring the CSA of the LMM as MRI (18), but there is low tissue contrast and limited field of view making ultrasound a low cost and accessible measuring tool for LMM CSA and bilateral asymmetry rather than fatty atrophy.

Prior investigations have established that atrophied fibers of LMM are replaced by fat following low back injuries, primarily but not confined to the deep LMM fibers, and that MRI changes in the LMM are strongly associated with LBP; 81% of 412 adults with LBP were reported to have some MRI defined LMM atrophy (4). The same study reported a higher

risk in women {odds ratio (OR) 3.7} and a higher incidence in adults compared to adolescents (81% compared to 14% respectively) with LBP, but noted that moderators such as body mass index (BMI) and duration of painful symptoms may account for the differences. Fatty replacement primarily occurs in the deep fibers, and LMM atrophy tends to be localized medial and deep along the LMM myofascial sheath at the level and side of greatest symptoms (19). Large-scale studies have previously demonstrated the utility of MRI in documenting the size and composition of the LMM (LMM fatty atrophy is visualized as high intensity areas on T1-weighted sequences) (4, 20). However, previous studies tend to focus on the CSA of the muscle rather than the percent of atrophy with fatty replacement (7, 9, 10), and many studies have enrolled healthy individuals or athletes as subjects rather than chronic LBP patients (7, 10, 21). While total CSA of the LMM may be one prognosticator of LBP, we believe that long-term change in the percent of atrophy of the LMM is another useful indicator as it is not yet fully understood if fatty infiltrations of LMM can be reversed.

In the current case reports, we highlight the use of MRI in monitoring long-term LMM fatty atrophy changes in patients with chronic LBP. In the process, we observed an interesting positive correlation between SMT combination with LMM stabilization exercise and reduced LMM atrophy, which also correlated to symptomatic and functional recovery in two patients. Conversely, a negative correlation of the same parameters was observed in a patient who only underwent SMT. While no substantive inferences can be drawn from such a small number of cases, it is not unreasonable to hypothesize that the correlations were causal as previous reports have shown increased LMM activity following SMT (22, 23). There is evidence that specific exercise protocols including multifidus activation have been shown to reverse atrophy of LMM and that these exercises can reduce LBP (24). Specifically, motor learning principles have been used to effectively increase isometric contraction capability of LMM (25), and patients who facilitate activation of the LMM through isometric contractions show a more rapid and complete recovery of their LBP (26).

While further research is necessary to validate our observations, it is reasonable to suggest that assessment of changes in LMM fatty atrophy should be considered in pre- and post-treatment MRI evaluations of the lumbar spine, as this metric was predictive of recovery in all three cases described. However, the most obvious pitfall of drawing any extrapolable conclusions from these case reports is that random variation is a plausible explanation for the results we described. In particular, the clinical course described for the case study of patient 3, who experienced worsening symptoms eventually requiring lumbar surgery, may have had nothing to do with the lack of LMM exercises, but rather was accounted for by greater lumbar disk pathology.

CONCLUSION

The LMM are essential stabilizers of the lumbar spine and atrophy of these muscles is closely associated with LBP, though this does not rule out other causes of LBP. In our case reports, we found that MRI served as an effective tool for

evaluating and documenting changes in LMM fatty atrophy in chronic-LBP patients. While this study is limited, there was an observed decrease in LMM atrophy in patients who received SMT accompanied with low back exercises, and future studies would help demonstrate whether our results can be consistently reproduced.

TEACHING POINT

Magnetic resonance imaging (MRI) was shown to be a valuable tool in documenting quantifiable long-term positive and negative changes in multifidus muscle atrophy with fatty replacement in chronic low back pain (LBP) patients, and may be an indicator of recovery.

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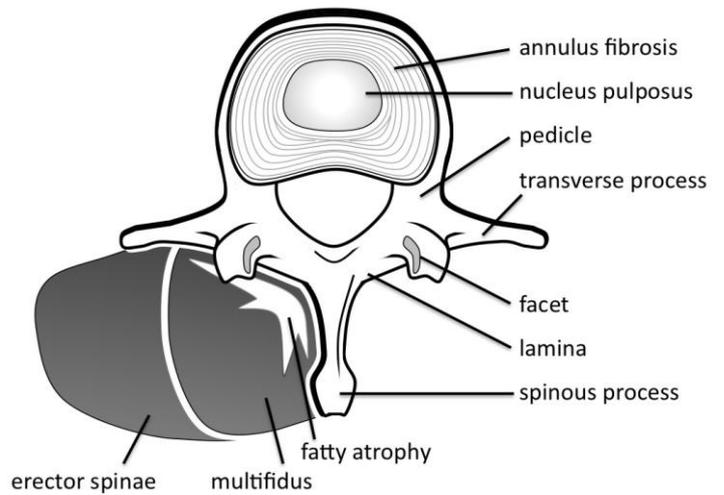
Figure 1 (top): Lumbar multifidus muscle (LMM) fatty atrophy is most commonly seen medially and deep in transverse MR images of the lumbar spine.
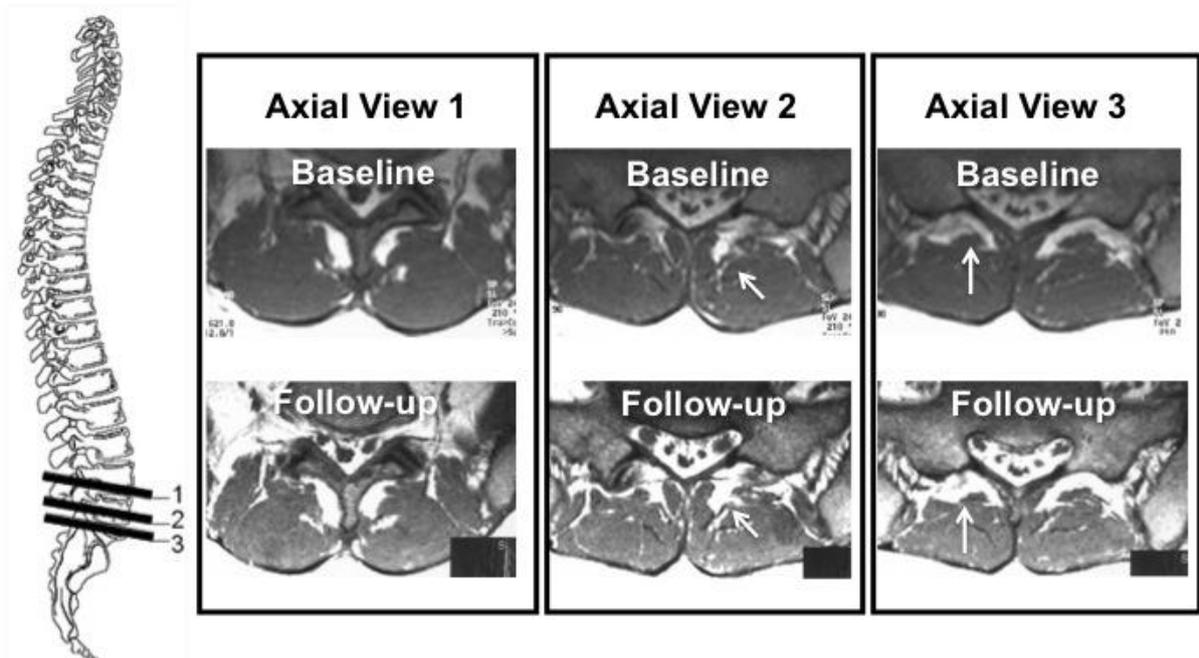
FIGURES
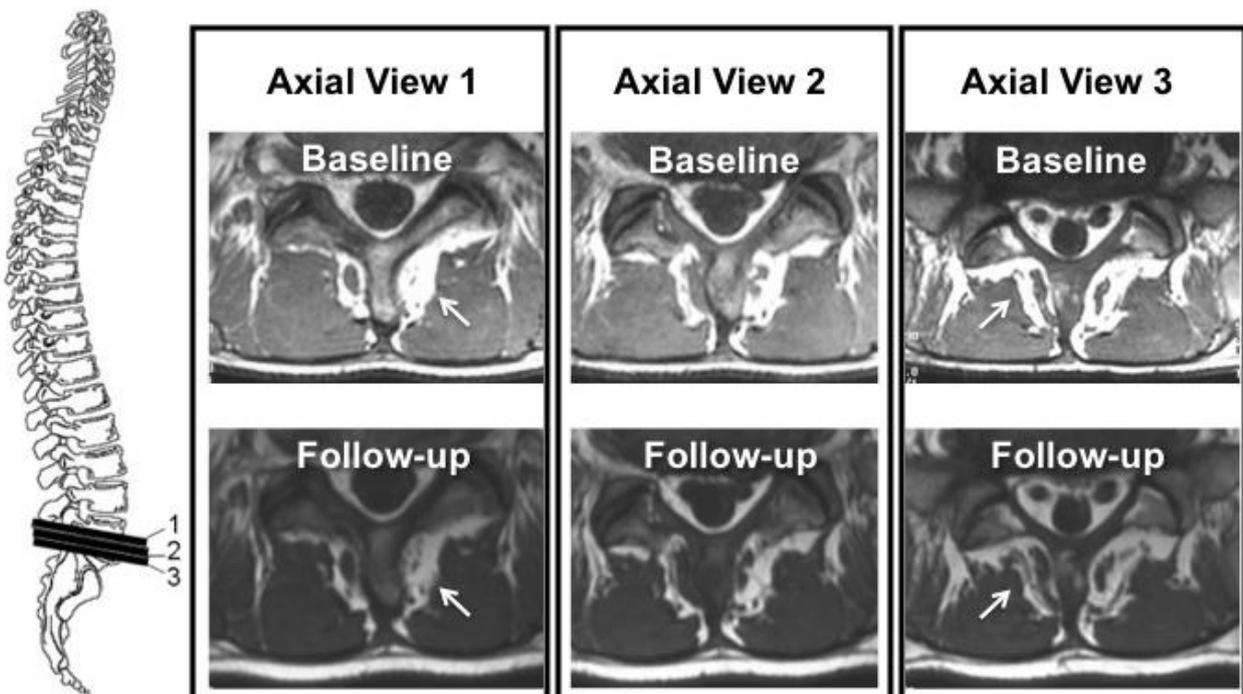
Figure 2: Patient 1: 30 year old male diagnosed with a lumbar sprain/strain, and disc herniations at levels L3-L4, L5-S1, and muscle spasming. FINDINGS: Baseline MRI transverse sequences revealed posterior disc protrusion at the L5-S1 level w/ a focal herniation on the left contacting the S1 nerve root; midline disc protrusion at L3-L4 level; disc degeneration L3-S1 in addition to LMM atrophy (asymmetric multifidus muscle atrophy at L5-S1 avg. approx. 32.4% on the left and 23.4% on the right). The follow-up MRI transverse sequences were performed 44 months post baseline at a different facility revealing decreased LMM atrophy [asymmetric multifidus muscle atrophy at L5-S1 avg. approx. 27.5% on the left (15% decrease) and 21.8% on the right (7% decrease)] compared to baseline MRI. White arrows indicate areas of highest lumbar multifidus fatty atrophy decrease between baseline and follow up. TECHNIQUE: Baseline MRI: 1.5T scanner, transverse non-contrast T1-weighted sequences, TR: 627.0 and TE: 15.0. Follow-up MRI: 1.5T scanner, transverse non-contrast T1-weighted sequences, TR: 450.0 and TE: 10.0.
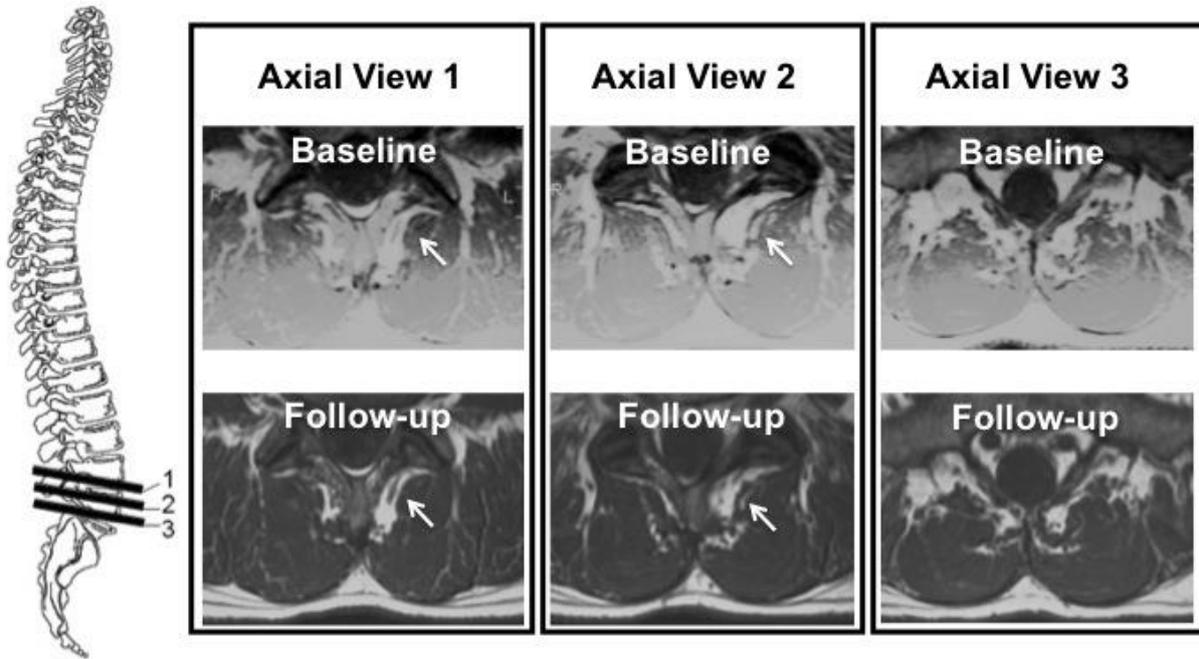
Figure 3: Patient 2: 41 year old male diagnosed with a lumbar sprain/strain, and right foot paresthesias. FINDINGS: Baseline MRI transverse sequences revealed mild posterior disc bulge at L4-L5 level; mild bilateral intervertebral stenosis at the L5-S1 level; intervertebral disc degeneration L4-S1 in addition to LMM atrophy (asymmetric multifidus muscle atrophy at L4-S1 avg. approx. 20.3% on the left and 13.0% on the right). The follow-up MRI transverse sequences were performed 72 months post baseline at a different facility revealing decreased LMM atrophy [asymmetric multifidus muscle atrophy at L4-S1 avg. approx. 12.3% on the left (39% decrease) and 8.8% on the right (32% decrease)] compared to baseline MRI. White arrows indicate areas of highest lumbar multifidus fatty atrophy decrease between baseline and follow up. TECHNIQUE: Baseline MRI: 1.5T scanner, transverse non-contrast T1-weighted sequences, TR: 650.0 and TE: 14.0. Follow-up MRI: 1.5T scanner, transverse non-contrast T1-weighted sequences, TR: 500.0 and TE: 13.1.

Figure 4. Patient 3: 29 year old male diagnosed with a lumbar sprain/strain, herniated discs: L4-L5, L5-S1, sciatic neuralgia, and muscle spasming. FINDINGS: Baseline MRI transverse sequences revealed L4-L5: right paracentral disc protrusion; L5-S1: mild degenerative disc disease, and subtle left paracentral protrusion in addition to LMM atrophy (asymmetric multifidus muscle atrophy at L4-S1 avg. approx. 12.2% on the left and 11.6% on the right). The follow-up MRI transverse sequences were performed 12 months post baseline at a different facility revealing increased LMM atrophy [asymmetric multifidus muscle atrophy at L4-S1 avg. approx. 17.2% on the left (41% increase) and 17.8% on the right (54% increase)] compared to baseline MRI. White arrows indicate areas of highest lumbar multifidus fatty atrophy increase between baseline and follow up. TECHNIQUE: Initial MRI: 1.0T scanner, transverse non-contrast T1-weighted sequences, TR: 621.0 and TE: 12.0. Follow-up MRI: 1.5T scanner, transverse non-contrast T1-weighted sequences, TR: 550.0 and TE: 13.1.

Etiology	Facetogenic/discogenic reflex inhibition; dorsal ramus syndrome
Incidence	High in LBP patients (>80%); general population unknown
Gender ratio	Higher in women with LBP (OR 3.7); BMI may be a confounder
Age predilection	Higher in adults with LBP compared to adolescents
Risk factors	Low back injury including facet or disc injury; incidence and severity increase with length of LBP symptoms
Treatment	LMM focused exercises including isometric contractions
Prognosis	Favorable with treatment; recurrent episodes of LBP without treatment
Findings on imaging	High intensity areas are identified medial and deep along the LMM myofascial sheath on T1-weighted transverse sequences

Table 1: Clinical and epidemiologic characteristics of lumbar multifidus muscle (LMM) atrophy.
LBP = low back pain, OR = odds ratio, BMI = body mass index.

MRI documented Change in Atrophy	left: -15%; right: -7%	left: +39%; right: +32%	left: +41%; right: +54%
Follow-up % LMM Atrophy	L5-S1; left: ~27.5%; right: ~21.8%	L4-S1; left: ~12.3%; right: ~8.8%	L4-S1; left: ~17.2%; right: ~17.8%
Baseline % LMM Atrophy	L5-S1; left: ~32.4%; right: ~23.4%	L4-S1: left ~20.3%; right ~13.0%	L4-S1; left: ~12.2%; right: ~11.6%
MRI Findings	L3-S1 disc narrowing; 4 mm posterior disc protrusion at L3-L4; 1 to 2 mm posterior disc protrusion L4-L5; left-sided disc herniation at L5-S1 that contacted the left S1 nerve root; bilateral LMM atrophy	Narrowing of the intervertebral foramen bilaterally at L5-S1; posterior disc bulge of ~2-3 mm at the L4-L5 level with minimal indentation of the anterior thecal sac; bilateral LMM atrophy	Right paracentral disc protrusion at L4-L5; subtle left paracentral disc protrusion with mild disc degeneration revealed at L5-S1; bilateral LMM atrophy
Follow-up MRI Technique	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 450.0; TE: 10.0	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 500.0; TE: 13.1	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 550.0; TE: 13.1
Baseline MRI Technique	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 627.0; TE: 15.0	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 650.0; TE: 14.0	1.5T scanner; T1-weighted; non-contrast; transverse; TR: 621.0; TE: 12.0
Sex; Age	M; 28	M; 39	M; 25
Patient	1	2	3

Table 2: Pre- and post-treatment imaging findings for three cases. LMM = lumbar multifidus muscle.

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	MRI	Computed tomography	Ultrasound
Lumbar multifidus muscle atrophy	High intensity areas are identified medial and deep along the LMM myofascial sheath on T1-weighted transverse sequences and are used to measure fatty atrophy compared to total CSA	Low density areas are identified medial and deep along the LMM myofascial sheath on transverse plane images and are used to measure fatty atrophy compared to total CSA	Echogenic vertebral lamina and facet joints are used as landmarks to measure total CSA and compare asymmetry between L and R
Advantages	High resolution of soft tissue; high tissue contrast	High resolution of bony structures	Low cost; high accessibility and absence of ionizing radiation
Disadvantages	Less detail of bony structures compared to CT	Moderate to high radiation; less tissue contrast than MRI	Limited field of view; low tissue contrast

Table 3: Comparison of the advantages and disadvantages of MRI, Computed tomography, and ultrasound for assessing lumbar multifidus muscle (LMM) atrophy. CSA = cross sectional area.

ABBREVIATIONS

BMI = Body Mass Index
CSA = Cross-Sectional Area
CT = Computed Tomography
EMG = Electromyography
LBP = Low Back Pain
LMM = Lumbar Multifidus Muscles
MRI = Magnetic Resonance Imaging
OR = Odds Ratio
SMT = Spinal Manipulative Therapy

KEYWORDS

Lumbar multifidus muscle; lumbar multifidus muscle atrophy; multifidi; chronic low back pain; MRI

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