Treatment of Multifidus Muscle Injury in Posterior Lumbar Spine Surgery: A Comprehensive Review

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Abstract Posterior lumbar surgery (PLS) is widely performed to treat lumbar spine disorders; however, it frequently results in iatrogenic injury to the multifidus muscle due to surgical detachment, prolonged retraction, and nerve manipulation, subsequently contributing to postoperative pain, impaired function, and delayed recovery. Accumulating evidence indicates that inflammation plays a central role in the pathophysiological cascade initiated by multifidus injury and is a major contributor to postoperative pain. Accumulating evidence indicates that inflammation plays a central role in the pathophysiological cascade initiated by multifidus injury and is a major contributor to postoperative pain. This review summarizes current findings regarding the mechanisms of multifidus injury and surgery-induced inflammation in PLS, highlights the impact of inflammation on skeletal muscle regeneration, and discusses integrative therapeutic strategies involving both Western medicine and traditional Chinese medicine. The objective is to provide a mechanistic foundation and therapeutic perspective to optimize perioperative protection of the multifidus muscle and improve clinical outcomes after PLS.

Keywords: Posterior lumbar surgery; Multifidus muscle; Rehabilitation therapy; Muscle satellite cells

Posterior lumbar surgery (PLS) is one of the most frequently employed approaches for treating degenerative lumbar spine disorders. It is primarily indicated for conditions involving lumbar instability, neural compression, and mechanical deformity. For patients with common lumbar diseases, such as lumbar disc herniation, lumbar spinal stenosis, and lumbar spondylolisthesis, in whom non-surgical treatments are ineffective, posterior lumbar surgery is a highly selective option and is considered the first-line treatment[1-2].

Despite its therapeutic benefits, the procedure often involves excessive muscle detachment or retraction, which may cause multifidus ischemia, edema, necrosis, and subsequent atrophy. These pathological changes constitute a major source of recurrent low back pain and postoperative dysfunction. In some cases, these postoperative symptoms can even exceed the preoperative level. This phenomenon is referred to in the literature so-called ‘fusion disease’ [3] or ‘failed back surgery syndrome’ (FBSS)[4]. Multifidus muscle injury is a key factor contributing to poor postoperative recovery and the occurrence of various adverse symptoms in patients following posterior lumbar surgery. However, few studies have systematically summarized the inflammatory mechanisms and integrative treatments specifically targeting multifidus injury following PLS. Therefore, in-depth research on multifidus muscle injury and its subsequent effects holds significant clinical importance. This article will provide a review of the mechanisms of the inflammatory response following multifidus muscle injury in PLS, as well as the treatment approaches, including both traditional Chinese and Western medicine.

1. Anatomy and Function of the Multifidus Muscle

The multifidus muscle lies deep within the paraspinal region and possesses the largest attachment area among the lumbar extensors. It is composed of multiple bundles, originating from the lamina and spinous processes of the lumbar vertebrae and inserting into the dorsal surface of the sacrum. It is well developed in the lumbar region and is considered the primary posterior stabilizing muscle of the spine [5]. The muscle extends from the sacrum to the cervical vertebrae, spanning from the sacrum to the second cervical vertebra, and is more developed in the lumbar and cervical regions. It is the only muscle that spans from the lumbosacral region to the back and is a small, deep intervertebral muscle of the spine. It is located deep to the semispinalis muscle and is similar in shape to the semispinalis but shorter. Compared with other paraspinal muscles, the multifidus exhibits a unique short-fiber, large-cross-sectional architectural design optimized for segmental stabilization rather than large-amplitude movement. It has a relatively large physiological cross-sectional area but short fiber length, an architectural design that allows the multifidus to generate significant force over a relatively short distance. [6-7]

From an anatomical perspective, the multifidus muscle consists of superficial and deep layers. The superficial layer originates from the spinous processes of the lumbar vertebrae and inserts into the iliac bone or sacrum. The deep layer originates from the lamina of each lumbar vertebra. Except for the deep muscle fibers originating from the lamina of L5, all other fibers extend caudally, crossing two vertebral levels, and insert into the mammillary processes and the joint capsule of the facet joints. The deep fibers originating from the L5 lamina ultimately insert above the sacral foramen between S1 and S2. The multifidus muscle is composed of five groups of muscle fiber bundles. These fibers extend from the lumbar vertebrae caudolaterally to the coccyx, iliac bone, and sacrum. These five groups partially overlap. Compared with fibers originating from lower lumbar vertebrae, those from upper lumbar levels are more superficially located and positioned more laterally. The multifidus muscle has close anatomical relationships with surrounding structures. It lies bilaterally adjacent to the spinous processes and is enclosed within a myofascial sheath formed by the superficial and middle layers of the thoracolumbar fascia. It is also in proximity to structures such as the lamina and longissimus muscle. In terms of origin and insertion, the lumbar multifidus originates from the mammillary processes and inserts into the inferior margins of the spinous processes two to three levels above.

The function of a muscle is determined by the orientation and morphology of its fibers. The multifidus muscle plays a crucial role in maintaining lumbar spine stability. The superficial multifidus originates from the spinous processes and extends inferolaterally to insert into the mammillary processes, posterior sacrum, or iliac crest, generating an inferolateral force that primarily controls spinal flexion and directional movement. In contrast, the deep multifidus primarily regulates intersegmental motion. Morphologically, the multifidus is short with a large cross-sectional area of the muscle belly and a high density of muscle fibers. These structural characteristics enable it to generate substantial force to support spinal stability. Functionally, the multifidus provides compressive force between vertebral bodies. The orientation of its fibers allows it to apply compressive loads on the lumbar spine. Muscle fibers that insert into the iliac and sacral bones contribute to lumbar extension and help resist excessive spinal rotation and translation. Bilateral contraction of the multifidus prevents vertebral rotational dislocation, thus maintaining the lumbar lordotic curvature [8]. Compared with other paraspinal muscles such as the iliocostalis lumborum and longissimus, the multifidus muscle is more closely connected to the lumbar vertebrae and is located nearer to the spinal axis. When the spine is moved by larger surrounding muscles, the multifidus helps ensure that individual vertebrae do not bend or rotate excessively. In particular, the deep layer of the multifidus plays a critical role in controlling intersegmental spinal rotation, while the superficial layer spans multiple vertebral levels and functions to guide spinal motion in an appropriate direction. This reduces segmental displacement in the lumbar spine, maintains the spine's physiological alignment, and contributes to lumbar stability.

2. Multifidus Muscle Injury and Inflammatory Response Caused by PLS

Multifidus muscle injury commonly occurs in patients undergoing lumbar spine surgery [9]. Radiologically, such injury is typically characterized by muscle atrophy and fatty infiltration, often accompanied by an inflammatory response [10]. Due to its anatomical proximity to the spine, the lumbosacral multifidus is the most severely affected during posterior lumbar surgery (PLS). The primary factors contributing to multifidus injury in PLS include muscle detachment, retraction, and nerve injury.

2.1 Injury Caused by Muscle Detachment

During PLS, subperiosteal detachment of paraspinal muscles disrupts the tendinous attachments and vascular supply of the multifidus, resulting in direct structural and ischemic injury. The procedure typically involves a midline posterior incision, followed by bilateral dissection of the paraspinal muscles to expose the lamina, facet joints, and other anatomical structures necessary for decompression, fusion, and fixation. During this process, the tendinous attachments between the multifidus muscle and the spinous processes or lamina are severed, directly disrupting its original vascular supply and structural integrity. Studies have shown that scar tissue is prone to form at the site of the multifidus postoperatively, with fatty degeneration observed within the muscle, resulting in a reduction of its contractile function [11]. Macintosh et al. [12] reported that creatine kinase levels are significantly higher after posterior lumbar surgery compared to anterior approaches, indirectly reflecting greater multifidus muscle damage. The injury caused by muscle detachment substantially impairs the physiological function of the multifidus and is considered a major contributor to postoperative pain and functional impairment.

2.2 Injury Caused by Retraction

During surgery, the use of self-retaining retractors or laminar retractors to retract the detached multifidus muscle can also cause significant injury. Sustained retraction markedly compromises intramuscular perfusion, and the extent of ischemic damage is strongly correlated with retraction duration. Kawaguchi et al. [13], in a simulated posterior lumbar surgery on pigs, found that retraction of the paraspinal muscles using retractors caused a sharp reduction in local muscle blood flow, with evidence of multifidus necrosis as early as 3 hours postoperatively.

A prospective clinical study by Gejo et al. [14]demonstrated a direct relationship between muscle retraction and muscle injury, particularly in the multifidus. When retraction time exceeded 80 minutes, delayed muscle strength recovery and a higher incidence of postoperative low back pain were observed. In a randomized controlled clinical trial, Kotil et al. [15] reported that relaxing the retraction every 15 minutes for 3 minutes during lumbar discectomy significantly reduced muscle injury. These findings underscore the severity of retraction-induced injury: the longer the retraction time, the greater the damage to the multifidus muscle, which in turn negatively impacts postoperative recovery.

2.3 Nerve Injury

The multifidus muscle is innervated by the medial branch of the dorsal ramus of the lumbar spinal nerve. This anatomical feature makes it particularly susceptible to injury during surgery. During muscle dissection, the medial branch travels along the deep surface of the muscle and is highly vulnerable to damage during surgical manipulation. Once the medial branch nerve is damaged, the muscle undergoes denervation changes postoperatively. In a study by Hodges et al. [16], transection of the medial branch of the dorsal ramus in animal models led to rapid atrophy of the multifidus muscle within a short period. Moreover, the multifidus is innervated by a single nerve branch without communicating branches between adjacent segments, making it more susceptible to denervation-induced atrophy following medial branch injury. This atrophy compromises the normal function of the spine and negatively affects postoperative lumbar mobility and functional outcomes in patients.

3. Inflammatory Response and Skeletal Muscle Regeneration and Repair After Injury

Inflammation orchestrates the early stages of skeletal muscle regeneration; however, excessive or prolonged inflammatory signaling may impair satellite-cell-mediated repair[17-18]. However, excessive inflammation may inhibit the proliferation and differentiation of MSCs and can even exacerbate muscle tissue damage. Although the etiology of skeletal muscle injury may vary, the regenerative process generally follows a consistent pattern, consisting of three overlapping phases: the inflammatory phase, the regeneration and repair phase, and the fibrosis and scar formation phase [19-20].

3.1 Muscle Satellite Cells

In 1961, Mauro was the first to identify the anatomical location and morphological characteristics of MSCs in the tibialis anterior muscle of frogs, laying the foundation for subsequent research on skeletal muscle [21]. MSCs originate from the central portion of the dermomyotome and are undifferentiated mononuclear cells characterized by a large nucleus and relatively scant cytoplasm. Within the cytoplasm, various organelles can be observed, including free ribosomes, microfilaments, intermediate filaments, microtubule vesicles, and occasionally glycogen granules, Golgi apparatus, rough endoplasmic reticulum, and tendon sheaths. Additionally, small mitochondria are typically present around the nucleus, along with centrosomes arranged in pairs adjacent to the nuclear membrane.

After muscle injury, the activation of MSCs is a prerequisite for muscle regeneration. MSCs reside beneath the basal lamina of skeletal muscle fibers and are myogenic precursor cells with the capacity for proliferation and self-renewal. The repair of injured skeletal muscle primarily depends on the proliferation and differentiation of these cells. Under normal physiological conditions, MSCs remain in a quiescent state; however, under certain stress conditions—such as mechanical loading, exercise, or trauma—they become activated. Once activated, MSCs undergo a cascade of events including proliferation, differentiation, and fusion to form myotubes, which subsequently mature into functional muscle fibers. Following activation, the majority of MSCs differentiate and fuse to form new myofibers, thereby repairing the damaged area. A smaller subset of these cells return to a quiescent state to replenish the satellite cell pool, ensuring the capacity for future regeneration [22].

3.2 Effects of Cytokines on Muscle Satellite Cells

Multiple cytokines are involved in the proliferation and differentiation of MSCs. Cytokines released during the inflammatory response influence the expression of members of the myogenic regulatory factors (MRFs) family. Among them, myogenic differentiation factor (MyoD) plays a pivotal role in muscle cell regeneration and is considered the most critical myogenic regulator during myogenesis. MyoD serves both as a marker of MSCs proliferative activity and as a promoter of early myogenic differentiation, making it a key indicator of muscle regeneration. Paired box protein 7 (Pax7) is recognized as the most important marker protein for identifying MSCs. Co-expression of Pax7 and MyoD is typically observed during the proliferation and early differentiation phases of MSCs [23]. Myogenin (MyoG) is expressed during the fusion of MSCs into myotubes and is essential for skeletal muscle development and maturation. It represents a key factor in terminal muscle cell differentiation. MyoD and MyoG can mutually activate each other and bind to E-box elements within promoter or enhancer regions, thereby inducing the expression of a subset of muscle-specific genes. Inhibition of MyoD or MyoG activity can effectively block the myogenic differentiation process [24]. Myosin heavy chain (MyHC) is a key determinant of muscle fiber type (fast or slow) and exhibits temporally regulated expression during the myogenic differentiation process. It is one of the primary markers used in the study of myogenic differentiation [25]. These myogenic regulatory factors are critical for the differentiation, development, and regeneration of skeletal muscle cells. Changes in their expression levels directly affect the progression of skeletal muscle regeneration [26].

3.3 Effects of Inflammatory Cytokines on Muscle Satellite Cells

Following multifidus muscle injury, an inflammatory response is rapidly initiated, with macrophages acting as key inflammatory cells playing an early role. In the initial phase of injury, macrophages migrate to and accumulate in the injured area under the guidance of chemotactic factors [27], predominantly polarizing into M1-type macrophages. M1-polarized macrophages predominate initially and release pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, which stimulate satellite cell activation but may hinder differentiation when excessively sustained [28]. In single-limb perfusion, TNF-α exerts dual effects on blood vessels: on one hand, TNF-α can moderately increase vascular permeability, accelerating the transport and absorption of inflammatory exudates; on the other hand, excessive TNF-α selectively damages endothelial cells involved in angiogenesis, leading to vascular rupture [29].Studies have also shown that TNF-α can inhibit skeletal muscle differentiation under cachectic conditions through the MyoD or caspase pathway, thereby mediating skeletal muscle repair [30]. After interference with IL-6 mRNA, the gene expression of MyoG and α-actin in myogenic cells is also reduced to some extent, suggesting that IL-6 may have potential myogenic activity [31]. Through skeletal muscle cell culture experiments, some researchers have found that activated mononuclear cells can secrete growth factors that stimulate myoblasts to induce the production of IL-6. It is believed that IL-6 secreted by skeletal muscle cells in an autocrine manner can regulate the proliferation of myoblasts and promote the nuclear fusion of myoblasts [32].Double immunofluorescence staining has shown co-localization of IL-6 and Pax7, indicating that muscle satellite cells are capable of producing IL-6 [33].Following tissue injury, the release, transduction, and transmission of inflammatory signals can further activate inflammasomes, stimulating the secretion of IL-1β. A small amount of IL-1β expression can induce an appropriate inflammatory response program, activating specific immune responses and maintaining homeostasis of the injury microenvironment. However, excessive IL-1β expression can expand the scope of the inflammatory response and exacerbate inflammatory injury [34].

With injury resolution, macrophages transition toward an M2 phenotype that secretes IGF-1 and other pro-regenerative factors, promoting tissue remodeling and myofiber maturation. Among them, IGF-1 is a key factor in the resolution of inflammation and macrophage programming during muscle regeneration [35]. It can activate skeletal muscle satellite cells [36], and its overexpression can accelerate the resolution of the inflammatory phase following muscle injury. MSCs are activated and proliferate following skeletal muscle injury, and are capable of differentiating and fusing to generate new myogenic cells. However, TGF-β can inhibit the proliferation and differentiation of MSCs and is involved in the regulation of muscle mass [37]. MRFs promote muscle formation and development through various regulatory mechanisms [38], and TGF-β can modulate the phased proliferation of MSCs by regulating MRFs, thereby reducing transcriptional activity and suppressing skeletal muscle-specific gene expression.

3.4 Relationship Between Extracellular Matrix and Skeletal Muscle Injury Repair

Cells reside within the extracellular matrix (ECM), a complex structure composed of various proteins and polysaccharides. The ECM provides a structural scaffold for cells and tissues, serving as a substrate for cell migration and playing a critical role in cellular signal transduction and responses to pathological stimuli [39].

Inflammatory responses are closely associated with remodeling of the extracellular matrix (ECM) in skeletal muscle. During the injury repair process, the ECM provides structural and biochemical support to muscle cells, with its abundant active molecules serving as a foundation for cellular activities. Transforming growth factor-beta, a multifunctional cytokine, has been demonstrated in studies by Dandan Shi et al. [2,40] to play a role in regulating ECM synthesis and deposition, thereby aiding in the remodeling of skeletal muscle tissue structure to facilitate better functional recovery. Skeletal muscle injury can induce changes in niche signaling, including damage to muscle fibers, which typically affects the basal lamina. This exposes MSCs to altered signals, as the MSC basal lamina can form ECM networks that split from the muscle fiber interstitium [41], impacting the quality of skeletal muscle regeneration and repair.

4. Assessment Methods for Multifidus Muscle Injury after PLS

Currently, there is no dedicated method for independently assessing multifidus muscle injury specifically after PLS. However, common assessment methods for the lumbar multifidus muscle are as follows:

4.1 Ultrasonography

Most current studies focus on morphological changes of the multifidus muscle following treatment of lumbar disc herniation. Zhang Lihong et al. [42] investigated the anteroposterior diameter (A-P), lateral diameter (Lat), and cross-sectional area (CSA) of the multifidus muscle before and after lumbar treatment. Other studies [43] using ultrasonography have found that the CSA, fatty infiltration (FI), and bone mineral density (BMD) of the lumbar multifidus muscle are closely related to the occurrence of adjacent segment disease (ASD). Comparative studies between mature ultrasonography equipment and handheld ultrasound devices have demonstrated that handheld devices also possess reliable measurement capabilities [44]. The widespread application of real-time ultrasound imaging technology has thus become feasible. Moreover, musculoskeletal ultrasound (MSKUS) is a commonly used imaging tool for evaluating the morphology, structure, and function of the multifidus muscle, with advantages including being non-invasive, real-time, dynamic, and high-resolution.

4.2 Magnetic Resonance Imaging (MRI) Assessment

Magnetic resonance imaging (MRI) utilizes magnetic fields and radio waves to generate images of internal body structures. Zhou Min et al. [45] investigated the effect of different postures on the assessment of the multifidus muscle, finding that multifidus degeneration is one of the causes of lumbar disc herniation (LDH), which is significant for studying the etiology and pathogenesis of LDH. In terms of computational methods, image processing software such as ImageJ can be used to quantify CSA of muscles on MRI scans [46]. Clinical studies[47] have shown that multifidus muscle degeneration is most closely associated with lumbar disc herniation, and MRI parameters are commonly used as primary observational indicators. These include measurements of fatty infiltration ratio, multifidus muscle CSA, and vertebral body CSA at the superior endplate levels of L5 and S1 vertebrae to evaluate the degree of postoperative multifidus muscle atrophy [48].

4.3 Other Assessments

In clinical examinations, palpation is commonly used to assess muscle tenderness, tone, atrophy, and to evaluate muscle strength. Electrophysiological assessment is also an important method to evaluate multifidus muscle function, primarily through electromyography (EMG) technology, which provides real-time feedback to guide patients in activating the multifidus muscle during core stabilization training, playing a critical role in rehabilitation [49]. Some studies have reported pathological biopsy analyses of the multifidus muscle to assess muscle fiber types, fatty infiltration, and inflammatory cell infiltration [50], greatly improving the accuracy of evaluation.

5 Treatment of Multifidus Muscle Injury After PLS: Integrative Medicine Approach

5.1 Western Medicine Treatment

5.1.1 Pharmacological Treatment

Pharmacological treatment is one of the commonly used approaches in managing multifidus muscle injury following PLS. Common Western medications include nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, corticosteroids, and others. These drugs work through different mechanisms to alleviate the various complications arising from multifidus muscle injury and to promote muscle repair.

NSAIDs reduce prostaglandin synthesis and are widely used for managing postoperative inflammatory pain. Ibuprofen, in particular, can achieve analgesic effects by inhibiting or decreasing the production of pain-inducing substances at the affected site [51], which greatly facilitates early intervention for patients experiencing low back pain. However, if the patient's symptoms do not improve after a period of medication use, discontinuation is recommended to avoid potential adverse effects caused by prolonged or excessive use.

Muscle relaxants are a class of drugs that promote muscle relaxation by acting on the central nervous system and neuromuscular junctions to suppress muscle spasms and reduce muscle tone. Shen Yingchao [52] found that eperisone can effectively reduce muscle tension in patients with low back pain and restore peripheral blood flow, thereby alleviating back pain. However, some patients may experience drowsiness while taking this type of medication [53], and clinical use should be considered with caution.

Corticosteroids possess potent anti-inflammatory effects and can effectively alleviate inflammation in muscle tissues, thereby reducing low back pain. For example, prednisone has demonstrated good efficacy in reducing inflammation and relieving pain. However, despite their strong anti-inflammatory properties, long-term use of corticosteroids may lead to adverse effects such as increased risk of infection, insomnia, and osteoporosis. Therefore, their use should be tailored and adjusted according to the patient's specific condition.

5.1.2 Physical Therapy

Physical therapy primarily refers to treatment modalities that utilize infrared light, electromagnetic waves, and low-frequency electrical currents generated by modern therapeutic devices to irradiate or electrically stimulate the affected area. Clinically common devices include the Thermal Design Power (TDP) electromagnetic spectrum therapy instrument, which emits electromagnetic waves of specific wavelengths. These waves are absorbed by biological tissues with matching absorption spectra in human cells, leading to biological effects through transmission, transformation, and utilization. These effects include enhancing microcirculation, promoting metabolism, inhibiting inflammatory factors, enhancing tissue repair, increasing bioavailability, improving endothelial function, lowering regional nerve excitation thresholds, and increasing sensitivity to pain stimuli, thus exerting a definite analgesic effect on multifidus-related pain [54].Electrical stimulation therapy devices (medium- and low-frequency) apply electrode pads to the affected area. When current is applied, the electrodes emit weak electrical currents of specific frequencies, which can inhibit nerve impulses, relieve local muscle spasms and tension, reduce fascial adhesions, suppress inflammatory cell activity, and reduce the production of inflammatory factors—ultimately relieving pain [55]. Magnetic therapy can alter the magnetic field of local tissues, exerting a calming effect on local nerves, improving microcirculation, reducing cellular immune responses, and thereby achieving pain relief [56].

5.1.3 Exercise Therapy

Also known as core strength training or functional training, exercise therapy in rehabilitation medicine typically involves strengthening the lumbar and back muscles in the affected area. This enhances muscle strength, regulates muscle tone, improves lumbar structural stability, and reduces local nerve sensitivity [57]. Different training focuses are applied at various stages of lumbar muscle injury recovery.

In the early stages, simple activation exercises can be performed, such as diaphragmatic breathing in the supine position, which activates core muscle groups of the abdomen and lower back, including the multifidus, laying the foundation for further rehabilitation. As recovery progresses, the intensity of training can be gradually increased, incorporating lumbar stability exercises in a standing position, such as single-leg stance or side plank holds, to progressively strengthen the multifidus muscle's power and control, promoting its regeneration and repair.Core strength training can enhance the stability of the lumbar skeletal muscles and restore muscle strength. Targeted functional exercises aimed at the site of injury can help alleviate pain by addressing the underlying muscular deficiencies.

5.2 Traditional Chinese Medicine (TCM) Treatment

5.2.1 Herbal Formula Therapy

In the treatment of multifidus muscle injury following posterior lumbar surgery (PLS), traditional Chinese herbal formulas play a unique role. Feng Ning et al. [58] found that Shen Tong Zhu Yu Tang can improve local blood circulation at the site of injury, promote the dissipation of blood stasis, and relieve pain caused by qi and blood stagnation resulting from multifidus muscle injury. This has a positive effect on improving the postoperative local qi-blood circulation around the multifidus.

Additionally, animal studies have shown that the use of Bu Yang Huan Wu Tang in rats with denervated skeletal muscle fibrosis can improve local blood circulation and nutritional supply, reduce the degree of muscle fibrosis, delay the progression of denervation-induced muscle atrophy, and promote skeletal muscle functional recovery [59].

5.2.2 Acupuncture Therapy

Acupuncture is a representative external treatment method in traditional Chinese medicine. Its efficacy in treating muscle injuries and relieving pain has been widely recognized by clinical practitioners [60]. Acupuncture techniques include filiform needle therapy, electroacupuncture, fire needle therapy, and the gradually evolved acupotomy therapy. Relevant studies have shown that acupuncture has significant clinical effects in treating muscle injuries, and even when used alone, it can effectively alleviate pain—sometimes even achieving acupuncture anesthesia effects [61]. Liu Tong et al. [62]found that the use of "bone-penetrating and muscle-regulating" acupuncture techniques had a good therapeutic effect on multifidus muscle injury after PLS, improving long-term pain outcomes.

In modern times, electroacupuncture has been developed by combining traditional acupuncture with electrical stimulation. Yan et al. [63]found that electroacupuncture at the Zusanli (ST36) acupoint could promote macrophage polarization after skeletal muscle contusion, thereby contributing to the regeneration of contused skeletal muscle. Animal experiments have confirmed that acupuncture can promote the proliferation, differentiation, and repair of satellite cells in the multifidus muscle. In a rat model of lumbar multifidus muscle injury, Xia et al. [64]demonstrated that electroacupuncture facilitates the repair of multifidus muscle cells by regulating mitochondrial function. Zhang Jiayi et al. [65] studied the timing of electroacupuncture intervention in a rat model of multifidus muscle injury and found that electroacupuncture may be most effective when applied 24 hours after injury, as it enhanced the expression of various muscle proteins. Electroacupuncture also modulates pain transmission pathways and improves local microcirculation, creating favorable conditions for the repair of the multifidus muscle.

5.2.3 Moxibustion Therapy

As recorded in *Lingshu · Guan Neng*, "What cannot be treated by acupuncture may be treated by moxibustion." Moxibustion is believed to have functions such as warming the meridians and dispelling cold, expelling wind and dampness, supporting Yang and preventing collapse, unblocking channels and collaterals, and harmonizing the viscera to balance Yin and Yang. When acupuncture is limited in its application, moxibustion can serve as an effective alternative. As an important modality of acupoint therapy, moxibustion exerts its therapeutic effects primarily through thermal stimulation, neural regulation, and immune modulation. It can activate the body's immune system and enhance its resistance to disease.

Moxibustion therapy has been widely recognized by clinicians for its advantages in treating lumbar muscle injuries, including minimal adverse effects and significant clinical efficacy. Studies have shown that Du Meridian moxibustion can improve the strength of core lumbar muscle groups and enhance mobility in patients with lumbar disc herniation caused by kidney Yang deficiency [66]. Lumbar muscle injuries are primarily associated with inflammatory responses, and moxibustion has demonstrated a distinct advantage in modulating inflammatory cytokines. Hu Xiuwu et al. [67] found that long snake moxibustion in patients with cold-dampness type low back pain may exert its therapeutic effect by reducing TNF-α levels. In addition, Zhenni et al. [68] analyzed that moxibustion effectively reduces the levels of pro-inflammatory cytokines, thereby achieving anti-inflammatory effects, providing theoretical support for its clinical application. Moxibustion also exhibits anti-inflammatory, analgesic, immune-enhancing, and circulatory-improving properties. Basic research has confirmed that moxibustion suppresses the expression of inflammatory factors, reduces cellular autophagy, and promotes cell proliferation [69].

5.2.4 Other Traditional Chinese Medicine Therapies

Other TCM treatments for multifidus muscle injury include cupping therapy, acupoint catgut embedding, bloodletting therapy, auricular acupressure, and health-preserving exercises such as Baduanjin, Tai Chi, and Wuqinxi. These modalities are commonly used as adjunctive therapies. For instance, cupping is often combined with Tui Na (Chinese therapeutic massage), acupuncture is paired with catgut embedding or bloodletting, and patients may be instructed to practice health-preserving exercises alongside their primary treatments. While these methods are frequently employed as complementary approaches, their clinical applications and therapeutic efficacy require further exploration and validation.

6. Discussion and Summary

Growing evidence underscores the essential role of the multifidus in maintaining spinal stability, and its vulnerability to surgical injury has become increasingly appreciated. Currently, although numerous achievements have been made regarding multifidus muscle injury caused by PLS, some deficiencies remain. In terms of injury mechanisms, while studies have identified factors leading to multifidus muscle damage and the impact of post-injury inflammatory responses on skeletal muscle regeneration, the more detailed injury mechanisms are not yet fully elucidated, especially regarding the involved signaling pathways, which require further in-depth investigation.

In summary, the multifidus muscle is indispensable for maintaining spinal stability, and direct injury from PLS is the primary cause of multifidus muscle damage. The factors and specific mechanisms underlying multifidus muscle injury warrant further exploration. Alongside improving and innovating minimally invasive posterior lumbar surgical techniques, efforts should be made to reduce multifidus muscle injury from the outset. On one hand, combining cell-tracing techniques to explore the intrinsic mechanisms of multifidus muscle injury and repair may help identify more effective therapeutic targets; on the other hand, actively conducting multicenter, large-sample, and long-term clinical studies to optimize the current integrative treatment strategies of traditional Chinese and Western medicine is necessary. These measures aim to better address the clinical challenge of multifidus muscle injury after posterior lumbar surgery, improve postoperative patient quality of life, and reduce the incidence of complications.

Abbreviations

PLS	Posterior lumbar surgery
MSCs	Muscle satellite cells
MRFs	Myogenic regulatory factors
MyoD	Myogenic differentiation factor
MyoG	Myogenin
Pax7	Paired box protein 7
MyHC	Myosin heavy chain

Declarations

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Consent for publication

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