ISSN: 2643-4091

Jani et al. Clin Arch Bone Joint Dis 2024, 7:019
DOI: 10.23937/2643-4091/1710019
Volume 7 | Issue 1
Open Access

Clinical Archives of Bone and Joint Diseases

REVIEW ARTICLE

Mechanical Low Backache: Review of Anatomical Basis of Low Back Pain and Significance of Muscle Strengthening Exercises

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Abstract

Mechanical low back pain (MLBP) is a significant health problem in the community. It affects all strata of the population. Various investigation modalities have been helpful in successfully diagnosing the underlying aetiology of LBP, and patients with such definite aetiology can be offered specific medical or surgical treatment to cure it. However, almost 70% of the cases fall in the Mechanical Low Back Pain criteria. Despite technological advances, treatment of MLBP remains restricted to conservative with supportive measures like analgesics and physiotherapy. However, the response to the treatment still needs to be satisfactory. It is necessary to understand the anatomical basis of MLBP. The lumbosacral spine and surrounding soft tissue as a complex undergo several degenerative changes over a while. Adding to the insult is recurrent microtrauma of that region due to recurrent but otherwise trivial exertion, lousy posture, faulty ergonomic work habits, etc. These factors give rise to a new order of muscle dynamics in this region. They need to be strengthened and assigned to the altered dynamics of the lumbosacral complex, which would improve the symptoms of MLBP in patients and reduce the chances of developing MLBP among the healthy population. This review article explains the variety of changes that occur at micro and macro levels in all the components of this complex by studying their anatomy and their effects on spinal movement and stability with an attempt to explain the anatomical basis of MLBP. It also emphasises paraspinal muscle strengthening in reducing Mechanical lower back pain.

Keywords

Mechanical low back ache, Paraspinal muscles, Thoracolumbar fascia, Disc degenerative changes, Lumbosacral mobility

Introduction

Mechanical Low Back Ache (MLBA) is pain, muscle tension, or stiffness in the lumbosacral region from the lowermost costal margin up to the gluteal fold on either side, with or without radiation to one or both legs. Low back pain is a symptom and not a disease in itself. It is one of the common disabling conditions seen in routine OPD of the general surgical, orthopaedic, neurological or neurosurgical facility. A significant volume of cases go unreported, especially in rural populations. Low Back Ache (LBA) directly impacts the ability to work and perform routine activities and, in turn, adversely affects livelihood. In addition, the psychosocial impact of LBA on individuals is also very significant. It involves the self-confidence and zeal to take on new challenges. Hence, the disability caused by LBA is more than that seen in Out Patient Dept (OPD) practice. Fayote, et al. have systematically reviewed LBA's clinical and economic burden, which is like the tip of an iceberg where many cases go unregistered, especially in the rural population [1].

Citation: Jani BK, Swaminathan S, Aswath KG, Tripathi SP (2024) Mechanical Low Backache: Review of Anatomical Basis of Low Back Pain and Significance of Muscle Strengthening Exercises. Clin Arch Bone Joint Dis 7:019. doi.org/10.23937/2643-4091/1710019

Accepted: August 20, 2024: **Published:** August 22, 2024

Though the causes of LBA are various and can arise from almost every systemic disorder, most of the causes are widely grouped under the following broad headings. Spondylogenic causes constitute nearly 90% of the cases of (LBA) which can further be divided into degenerative (disc, vertebral changes), traumatic (musculoskeletal), infectious (Tuberculosis) and neoplastic (benign, malignant, metastatic) causes. Systemic aetiology includes osteoporosis, osteopenia and Rheumatogenic (Ankylosing spondylosis, spondyloarthropathy, etc) causes. Other comparatively rare causes of LBA can be Vascular (aneurysm or peripheral vascular disease origin), Neurogenic (lesion anywhere along the central nervous system, particularly spine), Viscerogenic (disorders of organs of the abdominal sac, pelvis, retroperitoneal structures, etc.) and psychogenic back pain [2].

Unlike muscles of the extremities, back muscles are constantly in action to maintain posture. The lumbar spine, intervertebral disc (IVD), paraspinal muscles, intervertebral ligaments and thoracic-lumber fascia (TLF) constitutes a finely designed, ergonomically balanced complex where every component works in coordination with the other. All the movements of the lower spine, like flexion, extension, lateral rotation and even minor movements during breathing, also require coordinated movements of muscles [3].

The synchronised contraction/relaxation of muscle groups while performing a particular action at the lumbosacral spine is essential for effective yet pain-free action. Manohar Punjabi, et al. postulated that spinal stability has passive components (vertebral bodies, intervertebral discs, zygapophyseal joints, zygapophyseal joint capsules, and spinal ligaments), the active component comprising the muscles and the neutral system that interacts between these two systems. The neural control acts as a coordinator between the two, receiving input from the passive and the active subsystems and then directing the spinal musculature to stabilise the spine. The neural system should act at the right time and amount to protect the spine from injury and allow the desired movement. In a healthy state, the three systems interact and provide stability; however, following injury or degeneration to the passive system, the active system needs to work harder to compensate for the decreased contribution from the passive system. Hence, any sudden, jerky, unaccustomed or unplanned movement can give rise to uneven force distribution to any of these components, injuring one or more of them that leads to symptoms [4].

Here, understanding the anatomical and biomechanical basis of MLBA and the exact origin of pain in MLBA is essential to suggest possible corrections, treatment and prevention of further symptoms. Commonly, MLBA is managed with clinical evaluation of symptoms and signs and imaging of the lumbosacral spine by X-ray, CT scan and MRI. CT scan is used to delineate bony lesions of the vertebral column. At the same time, MRI can identify the changes/lesions in the spinal cord and surrounding soft tissue, including the intervertebral disc, its degeneration with or without prolapse, causing thecal sac narrowing, impingement of traversing or exiting nerve roots, etc. and in a small number of cases specific conditions like tumour, metastasis, aneurysm and so on. This review excludes lower back arising from systemic and non-spondylotic causes like fracture, tumour, metastasis, prolapsed intervertebral disc with neurological compromise, aneurysm, etc., since such patients require specific treatment for their underlying condition for mitigating the lower back pain [5]. This article focuses mainly on the spondylotic and MLBA arising from disc or musculoskeletal components.

Despite the evaluation advances, the complexity and biomechanics of the spine make it challenging to identify a specific anatomic lesion, with a precise diagnosis made in only 20% of cases [6].

Symptom mitigation in such patients requires conservative management in the form of analgesics, back stretching exercises, physiotherapy or image-guided injection of local anaesthetics in various combinations or sequences. Treatment aims to reduce the symptoms by causing back muscles to gain an additional range of motion, improve flexibility, etc. Most often, it is observed that the chief complaints of such patients are the inability to stand or sit for a long duration, which occurs due to muscle fatigue in maintaining an erect posture like sitting/standing in an anatomically/ergonomically altered (due to disc or related issues) structure. Hence, target-oriented physiotherapy can be more fruitful for these patients [7,8].

Objective

To understand the anatomical basis of MLBA and analyse the significance of muscle-strengthening exercises in its management.

Review of Literature

Mechanical low back ache -definition and statistics

MLBA is pain, muscle tension, or stiffness in the lumbosacral region from the lowermost costal margin up to the gluteal fold on either side, with or without radiation to one or both legs. An episode of low back pain is called acute if it has arisen for the first time in a patient's life or after a pain-free interval of at least six months and lasts no longer than six weeks. It is defined as chronic when it persists for 12 weeks or more. Mechanical LBA arises from the musculoskeletal component of the lumbosacral spine, which includes the lumbar spine (including vertebrae and IVD Disc), paraspinal muscles and Thoracolumbar Fascia [7,9].

Low back pain is a common global problem. The point prevalence of low back pain (LBP) in 2017 was estimated to be about 7.5% of the worldwide population or around 577.0 million people [10]. However, the epidemiological data of MLBA in the Indian population could not be found.

Lumbosacral anatomy and intervertebral joints

The dorsolumbar and sacrococcygeal vertebrae provide a framework for paraspinal muscles and thoracolumbar fascia attachments. Intervertebral discs are immobile yet are movement facilitators that allow the smooth movement of spinal vertebrae over one another in respective directions. Nucleus Polposus, being semi-fluid in nature, will enable it to alter its shape and permit smooth movement of vertebrae in either direction, as in flexion and extension of the vertebral column. Intervertebral joints between vertebral arches are synovial joints which provide minor movement between the superior and inferior articular processes [11]. The articular facets are covered with hyaline cartilage, and the joints are surrounded by capsular ligaments like Supraspinous (joining tips of the adjacent spinous process), Intertransverse (between adjacent transverse processes), Interspinous ligaments (connecting the length of the adjacent spinous process) and ligamentum flavum (connects laminae of adjoining vertebra) [12-14]. Recurrent branches of rami communicantes supply the anterior longitudinal ligament. However, the supply is segmental, and branches arising from adjacent nerve roots supply that segment of the Anterior Longitudinal Ligament [15].

The small meningeal branches of each spinal nerve innervate these joints between the vertebral bodies. The joints between the articular processes are innervated by branches from the posterior rami of the spinal nerves in addition to that of meninges, ligaments, and intervertebral discs. The joints of any particular level receive nerve fibres from two adjacent spinal nerves (Figure 1) [16,17].

Intervertebral disc and its role

Intervertebral discs are cushion-like structures situated between two adjacent vertebral bodies. These are the most significant structures in the human body that do not have their blood supply and absorb the required nutrients by osmosis. The components of the disc are the nucleus pulposus (central gelatinous material), annulus fibrosus (outer fibrous structure) and cartilaginous end-plates (that receive the blood supply) on which the disc rests over the vertebra. The nerve supply is through the sinovertebral nerve (see Table 1). Biochemically, the essential constituents of the disc are collagen fibres, elastin fibres and aggrecan. Age-related degenerative changes in the disc primarily affect the hydration of the nucleus pulposus. The disc loses height and develops fissures in the annulus fibrosus, which causes disc bulges and sometimes protrusion of nuclear material. Even if the protruded disc material or bulge is not compressing the nerve roots, this nociceptive atomic material is the cause of discogenic pain. A variety of nerves supplies these. More so, a single IVD is provided

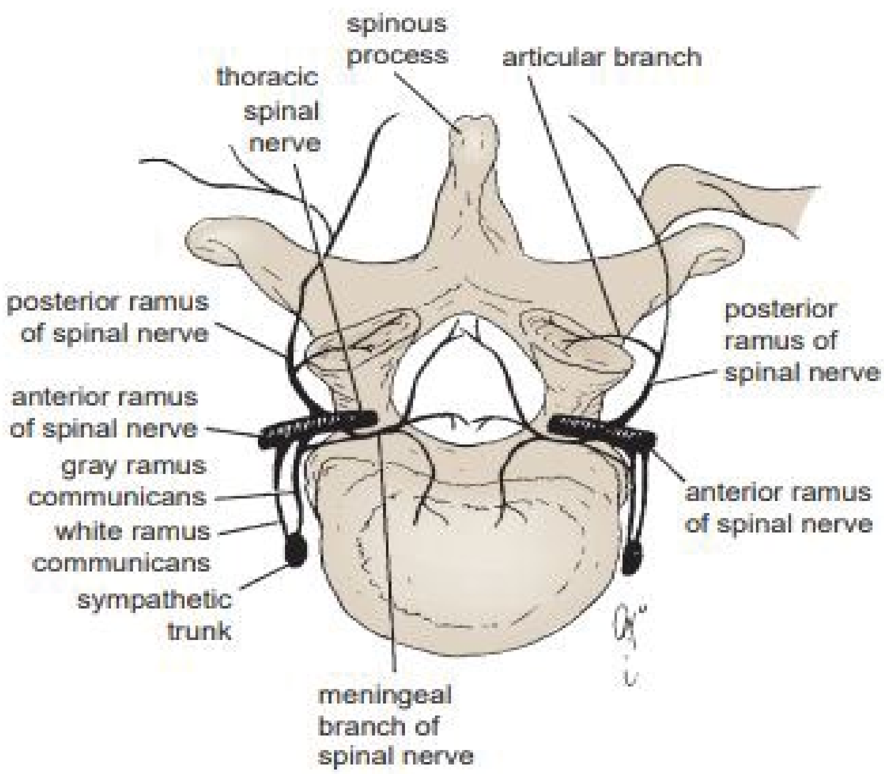

Figure 1: Nerve supply of intervertebral joint and intervertebral disc.

Table 1: Nerve supply of intervertebral disc.

Aspect of Disc	Nerve Supply
Posterior aspects of the discs and the posterior longitudinal ligament	Sinuvertebral nerves
Posterolateral aspects of the discs	Adjacent ventral primary rami and grey rami communicantes
The rest of the parts of the Intervertebral Disc	Rami communicantes cross intervertebral discs and are embedded in the connective tissue of the disc deep to the origin of the psoas.

Table 2: Paraspinal muscles.

Superficial layer	Splenius (splenius capitis, splenius cervicis)
Intermediate layer	Erector spinae (iliocostalis, longissimus, spinalis)
Deep layer	Transversospinales (semispinalis, multifidus, rotatores)
Deepest layer:	Segmental muscles (levatores costarum, interspinales and intertransversarii

by different nerves or their branches to varying aspects of it [15].

Effect of smoking and obesity on IVD

In addition to the micro-level mechanical trauma and dehydration, smoking is also found to be a contributing factor in disc degeneration. Bose N, et al. reported that smoking had not been shown to affect the prevalence of IVD degeneration [18]. On the other hand, some authors have reported both smoking and obesity are detrimental factors for lumbar IVD degeneration. Battie M, et al. evaluated subjects in a narrow age range and used MR imaging to study pairs of identical twins highly discordant for cigarette smoking and reported 18% greater mean IVD degeneration scores in the lumbar spines of smokers than nonsmokers [19]. Like, et al. also studied the association between overweight and lumbar IVD degeneration in 129 working middle-aged men. They showed that BMI ≥ 25 kg/m^2 increased the risk of lumbar IVD degeneration [20].

Effect of aging and hydration on IVD

The degenerative process starts at the NP as a change of the collagen to more fibrotic tissue. These factors effectively dehydrate the NP from a gelatinous semifluid-like structure to comparatively thick fibrous tissue, thereby severely restricting its shock-absorbing capacity. A recent study by Murakami, et al. has shown a significant difference in the water content of NP in adults and younger populations, which was found to be a marked reduction in water content in the adult population compared to the younger population. Similarly, type I and II collagen were significantly higher in more immature tissue, which is comparatively better for shock-absorbing properties. NP is the most affected tissue, and its decay constitutes one of the most significant enablers of furthering disc degeneration, thereby severely restricting the shock-absorbing capacity of the disc [21]. Antoniou, et al. demonstrated that synthesis of type II collagen in the NP tissues dropped with ageing; however, adult NP cells have significantly higher amounts of proteoglycans and collagen when compared to adolescent cells. This suggests the involvement of NP in repairing the extracellular matrix. Hence, the article postulated that after 5th decade, IVD could not be improved, and similarly, ECM alteration in the older age group leads to progressive, linear degeneration of IVD [22].

Even though programmed cell death (apoptosis) is an integral part of the ageing process, uncontrolled or excessive apoptosis is a prominent contributor to IVD degeneration [23].

Paraspinal muscle's anatomy and their significance

In addition to Quadratus lumborum, psoas, and posterior abdominal wall muscles, the paraspinal muscles provide a mobile and dynamic component of lumbosacral spinal stability. These muscles are arranged in different layers (Table 2), covering the entire length of the spine and providing support to maintain posture and stability of the spine by their synchronous micromovement. Moreover, the size of muscle spans between two adjacent vertebrae, intersegmental and the entire length of the spine, at this moment providing various movement options to the spine. This arrangement is beneficial for two adjacent segments of the spine where one is relatively more mobile than the other (viz. cervico-dorsal, dorso-lumbar and lumbosacral) [3,14].

These muscles are arranged in different layers extending from the cervical to the lumbar spine. The posterior rami of spinal nerves innervate these muscles. These muscles act in coordination with other muscles to facilitate a particular action. These muscles also cover the interveretebral synovial joints, essential for muscle bulk and flexibility. They play an important role in promoting the micro-movement at the interveretebral joint. Local Irritation of muscle (due to osteophyte) can cause poorly synchronised muscle contraction, resulting in spasms.

Thoracolumbar fascia

The thoracolumbar fascia (TLF) is a girdling structure

Table 3: Broad categories of LBA.

	Mechanical LBA	LBA with Neurological component
Imaging findings	Osteophytes, disc bulges, annular fissure, Ligamentum flavum hypertrophy, abutting the nerve roots *but not* impinging or compressing them	Disc extrusion and migration, Disc Protrusion, *impingement of exiting nerve roots, compression of traversing nerve roots, Nerve compression, canal stenosis, etc*
Symptoms	Inability to stand or sit for a long duration, Pain aggravated while bending forward, Pain is more at the onset of movement. Pain is Comparatively better when the body is warm-recurrent paraspinal muscle spasms.	Radiculopathy pain is relatively constant. *Tingling, numbness, paresthesia, pain in lower limbs more than back ach*e per se with or without bladder and bowel symptoms. *Weakness in the affected* lower limb is more prominent.
Signs	Paraspinal Muscle Spasm No signs of neurological compression/irritation No autonomic involvement	Absent or sluggish reflexes or Hyperreflexia Sensory motor involvement Autonomic involvement Paraspinal muscle spasm
Compression/Irritation of spinal cord or nerve roots	Absent	Present
Anatomical component giving rise to symptoms.	Osteoligamentous complex + IVD + Paraspinal Muscles	Nerve Root/spinal cord In addition to Osteoligamentous complex + IVD + Paraspinal Muscles

which separates paraspinal muscles from the posterior abdominal wall muscles. It is a conglomeration of several aponeurotic and facial layers which fuse in a thick composite that attaches firmly to the posterior superior iliac spine and sacrotuberous ligament. This thoracolumbar composite (TLC) assists in maintaining the integrity of the lower lumbar spine and the sacroiliac joint [13]. It is spread over the back, joining the posterior thoracic wall to the lumbosacral and sacroiliac joint, thereby providing a second layer of stability in addition to the spinal vertebrae. Being facial and aponeurotic in origin, its flexibility and range of motion are comparatively less than the muscles.

Discussion

LBA has been described as a twentieth-century healthcare disaster. Maniadakis N and Gray A reported that management of LBP in the United Kingdom has been estimated to cost the National Health Service (NHS) 151 million pounds annually. The clinical and economic burden of LBA is quite significant [10].

Origin of LBA- source of pain

Understanding the source of pain in LBA is essential to ensure symptomatic relief to the patient. The degenerative and disc-related anatomical changes are clearly seen on MRI imaging of the lower spine. However, they do not always corroborate with the clinical profile and symptoms and signs elicited from the patient.

LBA can be divided into two main categories. Low Back Ache that arises due to involvement of the spinal cord (meninges) or other neural elements like impingement or compression of transiting or exiting nerve root and, therefore, presents along with radiculopathy on the affected side comprises the first group. The second group of patients present with only LBA without any neurological symptoms or a brief period of radiating pain in the lower limb, not classically described as radiculopathy. This group is classified under the term 'Mechanical Low Back Ache' [24]. The following table (Table 3) broadly explains both entities.

From the above, it is evident that neurological symptoms and signs are the main distinguishing factors in the clinical profile of an individual that direct the mode of management. In general, the involvement of the spinal cord or nerve root to the extent of causing neurological symptoms necessitates neurosurgical intervention. They do not comprise mechanical low back aches; hence, they would not be discussed further.

Pathophysiology of MLBA

Effects of sudden jerks, lifting weight, during an un-synchronous muscle contraction: In standing or sitting positions, the more significant part of body weight falls anterior to the vertebral column (Figure 2) [25]. Moreover, most of the day-to-day activities require flexion of the spine, which is invariably gravity-assisted and requires less effort. However, getting back to the erect position is achieved by contraction of back muscles in an antigravity direction. In addition, the rotation motion of the spine also has some components of the extension of the spine. It is, therefore, not surprising to find that the paraspinal muscles of the back are well-developed in humans [3,14]. Over the life span of an individual, the paraspinal muscles develop muscle memory to maintain specific posture and flexibility of the spine while performing a variety of activities like standing, sitting, bending forward, and lateral and rotational movement by an individual [26,27].

These back muscles overlap and crisscross each other

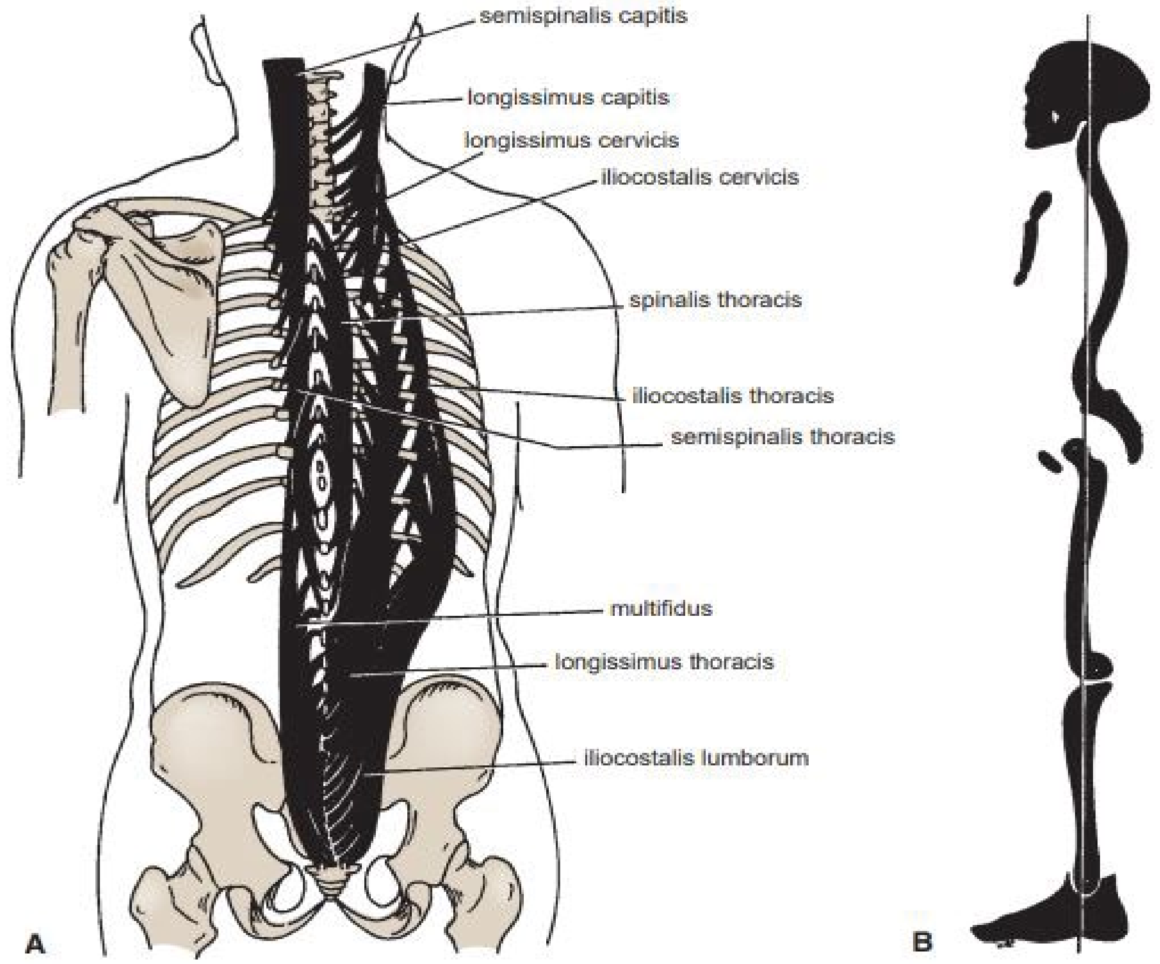

Figure 2: Center of mass tends to fall anterior to the spine. Paraspinal muscles act as a counterforce to keep the spine straight to maintain an erect posture.

at multiple segments of the spine to facilitate various segmental movements of the spine smoothly by a fine, delicately coordinated contraction/relaxation sequence of muscles. The postural tone of these muscles is the primary factor responsible for maintaining the standard curves of the vertebral column. Each muscle in this group may be regarded as a string, which, when pulled or contracted, causes one or several vertebrae to be extended or rotated on the vertebra below. The spines and transverse processes of the vertebrae act as levers that facilitate these muscle actions [17].

While lifting a heavy weight, it is essential to keep the spine as the centre of mass to synchronise the necessary group of back muscles. Due to sudden jerks or lifting weights in an awkward or uncomfortable posture due to compulsions in the local milieu, the load gets distributed unevenly over paraspinal muscles, causing asynchronous muscular contraction of one or more muscle(s). This can trigger sudden spasms of the affected muscle(s) [21].

In case of pre-existing degeneration of the lumbosacral spine or degenerative disc disease, the possibility of such muscle spasms is higher, even with lesser load/weight. This condition worsens with advancing age as well. In such a scenario, these muscles tend to relearn the quality and strength of contraction required to effect a particular movement. However, the threshold for failure and, therefore, onset of muscle spasm is lowered in such circumstances [26]. There is also a possibility of degenerative changes (osteophytes, arthropathy, etc) causing Irritation of the nerve roots supplying these paraspinal muscles. Microtraumata of the deep muscular tissues can arise when the vertebra's motion exceeds its physiological boundaries, which occurs when the demand for spinal muscle control is high [17]. It also presents as recurrent back muscle spasms, inability to stand and sit long, and reduced ability to bend forward and lift the weight. The net result of all the above mentioned stressors may lead to short-term or long-term painful muscle contraction [16].

Role of IVD and its hydration in mechanical LBA

The intervertebral disc comprises a semifluid nucleus pulposus in the centre encircled by a fibrous sheath known as annulus fibrosus. The nucleus is capable of changing its shape according to the compression load on it. It permits one vertebra to rock anteriorly or posteriorly on another, as in flexion and extension of the vertebral column. Annulus fibrosus provides a resilient multilayered support and prevents outward thrust of the nucleus. A sudden increase in the compression load on the vertebral column causes the semifluid nucleus pulposus to become flattened. Sometimes, the outward thrust exceeds the resilience of the annulus fibrosus, resulting in the rupture of its fibres, which in turn allows the nucleus to herniate and protrude into the vertebral canal, where it may press on the spinal nerve roots/trunk or even the spinal cord. With advancing age, the water content of the nucleus pulposus diminishes and is replaced by fibrocartilage. The collagen fibres of the annulus also degenerate, and as a result, the annulus cannot always contain the stress over the nucleus pulposus and gives way. In addition, the stress load on the disc is only sometimes compressive alone but also could be tensile and shear stresses, albeit to a lesser extent [11,15].

Novomu I, et al. revealed that in a healthy adult, the load on the L3/4 level disc in a sitting posture and a standing posture with 20 degrees flexion was 250% of the total body weight, although the portion of the body above the L3/4 level represented only 60% of total body mass on an average. Such large loads have been validated with mathematical models. Therefore, it is evident that the body mass contributes to only 25% of actual stress on the disc, while the remaining 75% of the load is an internal input. The external load is the body's weight above the lumbar disc, and the internal load is the muscle force required to stabilise the spine under different postures [21].

Therefore, it can be safely concluded that muscle-strengthening exercises of paraspinal musculature provide counter-correction to the decreasing disc compliance over age. Hence, the strength and stability of the spine are directly proportional to the strength of the paraspinal muscle. This indicates that spinal strengthening exercises have a significant role in reducing MLBA [21,28,29].

Role of paraspinal muscle strengthening in rehabilitation

Over the life span of an individual, the paraspinal muscles develop muscle memory to maintain specific postures and aid in the spine's flexibility while performing various activities like standing, sitting, bending forward, and lateral and rotational movement. Therefore, whenever disc degeneration or other mechanical factors lead to LBA, retraining of paraspinal muscles as a compensatory mechanism by muscle strengthening exercises would provide the patient with symptom relief. Hence, rehabilitation of the muscle by spinal strengthening exercises is essential [26,27].

Intervertebral joints are richly innervated by rami communicantes from adjacent nerve roots. The degenerative changes in the spinal vertebrae and interveretebral ligaments form a source of chronic pain in MLBA. However, these changes are irreversible, and so is the pain. As the paraspinal muscles provide second-line defence in spinal stability, spinal muscle strengthening can aid in significant clinical recovery.

The paraspinal retinacular sheath (PRS) is the deep lamina of the posterior layer of the thoracolumbar fascia, extending from the spinous to transverse processes. This PRS is in a critical position to act as a 'hydraulic amplifier', assisting the paraspinal muscles in supporting the lumbosacral spine. Though not quantified in the literature, TLF [30]. It plays a significant role in establishing a unified complex along with the paraspinal muscles. However, it is safe to assume that age-related changes, including microtrauma, would affect TLF similarly to muscles, intervertebral ligaments and other soft tissues, though the severity and extent of degeneration may vary. Therefore, the only modifiable elements among this complex are paraspinal muscles, which provide stability and strength to the spine.

Significance of retraining the paraspinal muscles by muscle strengthening exercises

Till now, it is aptly clear that degenerative changes in the spinal vertebrae, IVD, intervertebral ligaments and decreased or dysfunctional paraspinal muscles cause MLBA. With increasing degenerative changes in the disc, the paraspinal muscle mass starts reducing, which was established by the study of G Sudhir, et al. Patients with advanced disc degeneration tend to have wasting of paraspinal muscles, which could be an essential aggravating factor for back aches in these patients. Similarly, those with reduced muscle mass could risk disc degeneration due to the excess load transferred to the disc. Back muscle stretching exercises increase the spine's range of motion in the respective direction; however, maintaining posture for a prolonged time requires adequate spinal stability and strength in paraspinal muscles. Muscle strengthening exercises will improve the muscle mass. This enhanced muscle strength compensates for the degenerative changes and positively affects the patient outcome [28].

In addition, fatty infiltration of the muscles due to obesity or muscle atrophy due to ageing has a significant role in low back pain. The consensus is that obese individuals, regardless of age, have a greater absolute maximum muscle strength compared to non-obese persons, suggesting that increased adiposity acts as a chronic overload stimulus on the antigravity muscles (e.g., quadriceps and calf), thus increasing muscle size and strength. However, when maximum muscular strength is normalised to body mass, obese individuals appear weaker. Reduced mobility, neural adaptations and changes in muscle morphology may cause this relative weakness [19]. Peng X, et al. studied fatty degeneration of paraspinal muscles across the adult lifespan of Chinese females. They presented the standardised reference data showing fatty infiltration in paraspinal muscles increasing with age and BMI, while muscle loss may be associated with ageing. Either change (fatty infiltration or atrophy) may cause or aggravate MLBA and requires strengthening of the paraspinal muscles for symptomatic relief [31]. Hence, correcting BMI, weight reduction, smoking cessation, and paraspinal muscle strengthening exercises are essential adjuncts in managing mechanical low back aches.

Conclusion

MLBA has a voluminous presence in society, and its prevalence in the peak productive age group is causing a significant economic and clinical burden. Various literature reviews suggest mechanical or spondylogenic as the most common cause of LBA. Interplay of multiple components viz IVD, intervertebral ligaments, paraspinal muscles and TLF and their fine coordination at micro and macro levels ensures smooth and pain-free movement. Age-related changes or repeated microtrauma in the vertebral spine and any of the above components lead to MLBA and the inability to maintain posture for a long time. In addition, smoking and obesity show a linear association in increasing the severity of MLBA. In addition to the other conservative and supportive measures, practising postural rehabilitation (by retraining and strengthening paraspinal muscles) would be an essential adjunct in improving the quality of life by reducing pain. Age-related degenerative changes in IVD, vertebrae and paraspinal soft tissue are irreversible, whereas strengthening paraspinal muscles is the only adjustable element that could counter these effects for symptomatic relief in mechanical LBA. Paraspinal muscle strengthening exercises are more likely to improve the quality of life in mechanical low back aches than paraspinal muscle stretching exercises.

Author Contributions

Conceptualization: Dr Bharat Khemji Jani, Dr Aswath KG; Formal Analysis: Dr Bharat Khemji Jani, Dr Subramaniam Swaminathan; Investigation: Dr Bharat Khemji Jani, Dr Subramaniam Swaminathan; Methodology: Dr Bharat Khemji Jani, Aswath KG, Dr Satya Prakash Tripathi; Project Administration: Dr Bharat Khemji Jani, Dr Subramaniam Swaminathan; Writing – Original Draft: Dr Bharat Khemji Jani, Dr Satya Prakash Tripathi; Writing – Review & Editing: Dr Bharat Khemji Jani, Dr Satya Prakash Tripathi

Support

Armed forces medical services.

Conflicts of Interest

Nil to declare.

References

1. Fatoye F, Gebrye T, Mbada CE, Useh U (2023) Clinical and economic burden of low back pain in low- and middle-income countries: A systematic review. BMJ Open 13: e064119.
2. Disorders and Diseases of the Spine Editors: Page 204 table 12.1, 12.2. In: Swiontkowski Marc F, Stovitz Steven D, Manual of orthopaedics. (6th edn), 2001 Lippincott Williams & Wilkins Manual of Orthopaedics Swiontkowski | PDF | Hip | Knee (scribd.com)).
3. Ocran E (2023) Deep back muscles: Anatomy, innervation and functions | Kenhub.
4. Studnicka K, Ampat G (2022) Lumbar Stabilization. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing.
5. Casazza BA (2012) Diagnosis and treatment of acute low back pain. Am Fam Physician 85: 343-350.
6. Will JS, Bury DC, Miller JA (2018) Mechanical low back pain. Am Fam Physician 98: 421-428.
7. Casser H-R, Seddigh S, Rauschmann M (2016) Acute lumbar back pain: Investigation, Differential Diagnosis, and Treatment Dtsch Arztebl Int 113: 223-234.
8. Chou R, Oregon Health & Science University, Portland, Oregon (2011) Low back pain (chronic). Clinical Evidence Handbook, American Family Physician 84: 437.
9. Adams MA, Hutton WC (1982) Mechanical factors in the etiology of low back pain. Orthopedics 5: 1461-1465.
10. Maniadakis N, Gray A (2000) The global burden of low back pain. International Association for the Study of Pain (IASP). The economic burden of back pain in the UK. Pain 84: 95-103.
11. Waxenbaum JA, Reddy V, Futterman B (2023) Anatomy, back, intervertebral discs. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
12. Iwanaga J, Simonds E, Yilmaz E, Schumacher M, Patel M, et al. (2019) Anatomical and biomechanical study of the lumbar interspinous ligament. Asian J Neurosurg 14: 1203-1206.
13. Willard FH, Vleeming A, Schuenke MD, Danneels L, Schleip R (2012) The thoracolumbar fascia: anatomy, function and clinical considerations. J Anat 221: 507-536.
14. Goubert D, Oosterwijck JV, Meeus M, Danneels L (2016) Structural changes of lumbar muscles in non-specific low back pain: A systematic review. Pain Physician 19: E985-E1000.
15. Bogduk N, Tynan W, Wilson AS (1981) The nerve supply to the human lumbar intervertebral discs. J Anat 132: 39-56.
16. Snell RS. The Back, Chapter 12. In: Clinical anatomy by regions: Richard S. Snell. (9th edn), 690.
17. Panjabi MM (2003) Clinical spinal instability and low back pain. J Electromyogr Kinesiol 13: 371-379.
18. Boos N, Semmer N, Elfering A, Schade V, Gal I, et al. (2000) Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: Predictors of low back pain-related medical consultation and work incapacity. Spine 25: 1484-1492.
19. Battié MC, Videman T, Gill K, Moneta GB, Nyman R, et al. (1991) 1991 volvo award in clinical sciences. Smoking and lumbar intervertebral disc degeneration: An MRI study of identical twins. Spine 16: 1015-1021.

20. Liuke M, Solovieva S, Lamminen A, Luoma K, Leino-Arjas P, et al. (2005) Disc degeneration of the lumbar spine in relation to overweight. Int J Obes (Lond) 29: 903-908.
21. Inoue N, Espinoza Orías AA (2011) Biomechanics of intervertebral disk degeneration. Orthop Clin North Am 42: 487-499, vii.
22. Antoniou J, Steffen T, Nelson F, Winterbottom N, Hollander AP, et al. (1996) The human lumbar intervertebral disc: Evidence for changes in the biosynthesis and denaturation of the extracellular matrix with growth, maturation, ageing, and degeneration. J Clin Invest 98: 996-1003.
23. Wang C, Yu X, Yan Y, Yang W, Zhang S, et al. (2017) Tumor necrosis factor-α: A key contributor to intervertebral disc degeneration. Acta Biochim Biophys Sin 49: 1-13.
24. Patrick N, Emanski E, Knaub MA (2014) Acute and chronic low back pain. Med Clin North Am 98: 777-789.
25. Snell RS. Chapter 12, The Back. In: Clinical anatomy by regions/Richard S. Snell. (9th edn), Includes index, 604.
26. Saeterbakken AH, Stien N, Andersen V, Scott S, Cumming KT, et al. (2022) The effects of trunk muscle training on physical fitness and sport-specific performance in young and adult athletes: A systematic review and meta-analysis. Sports Med 52: 1599-1622.
27. Liu Q, Jorgensen E (2011) Muscle memory. J Physiol 589: 775-776.
28. Sudhir G, Jayabalan V, Sellayee S, Gadde S, Kailash K (2021) Is there an interdependence between paraspinal muscle mass and lumbar disc degeneration? A MRI based study at 2520 levels in 504 patients. J Clin Orthop Trauma 22: 101576.
29. Siemionow K, An H, Masuda K, Andersson G, Cs-Szabo G (2011) The effects of age, sex, ethnicity, and spinal level on the rate of intervertebral disc degeneration: A review of 1712 intervertebral discs. Spine (Phila Pa 1976) 36: 1333-1339.
30. Schuenke MD, Vleeming A, Van Hoof T, Willard FH (2012) A description of the lumbar interfascial triangle and its relation with the lateral raphe: Anatomical constituents of load transfer through the lateral margin of the thoracolumbar fascia. J Anat 221: 568-576.
31. Peng X, Li X, Xu Z, Wang L, Cai W, et al. (2020) Age-related fatty infiltration of lumbar paraspinal muscles: A normative reference database study in 516 Chinese females. Quant Imaging Med Surg 10: 1590-1601.