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Various Mechanisms of Low Back Pain in Elderly Population

Priyanka Tanwar[1], Mamta Naagar[2], Garima Malik[3], Omveer Singh[4], Tarun Singh[5], Md. Shamshir Alam [6], Manish Kumar Maity[7*]

[1]*Department of Pharmacology, Bhagvan Mahavir Institute of Medical Sciences, Sonipat, Haryana, India*
[2,3,4,5,7]*Department of Pharmacy Practice, MM College of Pharmacy, Maharishi Markandeshwar (Deemed to be University), Mullana, Ambala, Haryana, India*
[6]*Department of Pharmacy Practice, College of Pharmacy, National University of Science and Technology, Bosher-Muscat, Sultanate of Oman*

***Abstract*: Chronic low back pain (CLBP) is a pain that lasts at least three months in the lower back area. CLBP is the world's second greatest cause of disability, and it is a serious social and economic issue. CLBP has grown by more than 100% in the previous decade in the adult population and continues to rise substantially in the ageing population, impacting both men and women of all ethnic groups and having a considerable influence on functional capacity and occupational activities. Psychological issues such as stress, sadness, and worry might also have an impact. Given this complication, diagnosing patients with CLBP can be difficult and necessitates sophisticated clinical decision-making. Answering the issue "what is the pain generator?" among the several structures that might be implicated in CLBP is critical in the treatment of these patients, since a misdiagnosis can lead to therapeutic errors. The myth that the etiology of 80 – 90 % of LBP cases is unknown has been passed down through the generations. In the majority of cases, low back pain can be ascribed to a single pain generator, each with its own set of features and therapeutic options. Radicular pain, facet joint pain, sacro-iliac pain, lumbar stenosis pain, and discogenic pain are all discussed here. In this review, our goal is to provide doctors with a basic guide to help them identify pain generators in a safer and faster manner, based on a right diagnosis and subsequent treatment strategy.**

***Keywords*: Low back pain, Chronic low back pain (CLBP), Spine, Intervertebral discs (IVDs).**

1. Introduction

Low back pain (LBP) is the most prevalent musculoskeletal ailment in adults, affecting up to 84 % of the population [1]. Chronic low back pain (CLBP) lasts at least 12 weeks in the lower back region [2]. Many researches propose that chronic pain be defined as pain that lasts longer than the predicted time for recovery, thereby bypassing the near time criteria. This classification is crucial because it emphasizes that CLBP has well-defined underlying pathological reasons and is an illness, not a symptom. CLBP is the biggest cause of disability in the globe, and it is a huge social and economic issue [1]. Given this complication, diagnosing individuals with LBP can be difficult and necessitates sophisticated clinical decision-making. Answering the question, "What is the pain generator?" among the several structures that might be implicated in CLBP is critical in the treatment of these patients, because a diagnosis that is not based on a particular pain generator can lead to therapeutic errors. This article seeks to give a quick clinical guidance that can aid in the identification of pain generators by providing a detailed anatomical description, guiding physicians to the right diagnosis and treatment.

2. Low Back Pain Epidemiology

LBP is a significant social and economic issue. CLBP prevalence among French healthcare professionals is reported to vary from 15-45 % [3], whereas the point prevalence of CLBP in US individuals aged 20 - 69 years old was 13.1 % [4]. CLBP is expected to be present in 5.91 % of the Italian population [5]. In the recent decade, the frequency of acute and CLBP in adults has doubled and continues to rise substantially in the ageing population, affecting both men and women of all ethnic groups [6]. Pain limits occupational activities and is a major source of absenteeism, therefore LBP has a considerable influence on functional ability [7]-[9]. Its economic cost is directly indicated by increased health-care consumption and indirectly by lower productivity [7], [9]. In the next years, these prices are anticipated to climb considerably higher. According to a 2006 study, the entire cost of LBP in the United States exceeds $100 billion per year, with two-thirds of that cost coming from missed income and productivity [10].

3. Pain Generator

Nerve roots, muscle, fascial structures, bones, joints, intervertebral discs (IVDs), and organs in the abdominal cavity are all potential anatomic causes of LBP symptoms. Furthermore, symptoms may arise as a result of abnormal neurological pain processing, resulting in neuropathic LBP [11], [12]. The diagnosis of individuals with LBP can be difficult, and it necessitates complicated clinical decision-making. Nonetheless, identifying the treatment method requires establishing the etiology of the discomfort [13]. Furthermore, a clinician must note that psychological issues such as stress, sadness, and anxiety might alter LBP during the clinical

*Corresponding author: manishkumarmaity@gmail.com

examination [14], [15]. Substance use exposure, thorough health history, job, habits, and psychosocial aspects should all be included in the history [16]. Clinical information is the most important factor in forming a first impression, whereas magnetic resonance imaging (MRI) should only be used if clinical aspects are unclear or if neurological impairments or other medical disorders are present [17]. The American College of Radiology advises against imaging for LBP within the first six weeks unless there are red flags. Weight loss or fever without a recognized reason, immunosuppression, a past cancer diagnosis, intravenous drug use, chronic corticosteroids usage or osteoporosis, being over 70 years old, and focal neurologic deficiency with increasing or debilitating symptoms are among them [18]-[20].

The results of imaging tests are only sporadically linked to symptoms. One cross-sectional investigation of asymptomatic people aged 60 years and above found that 36% had a ruptured disc, 21% had spinal stenosis, and more than 90% had a deteriorated or bulging disc [21].

Although it is impossible to estimate precisely, it is plausible to think that these conditions may cost more than $50 billion per year, if not more, in direct and indirect costs [22]. According to a recent research, lumbar radiography was conducted 66 million times in the United States in 2004, with each scan costing $54 [23]. MRI prices appear to be 10 to 15 times higher [23], [24], while estimates vary significantly based on geographic location, insurance status, and other considerations.

According to the most recent clinical standards, while dealing with LBP patients, the doctor should conduct a thorough diagnostic of the mechanisms that maintain acute and/or chronic pain. These pathways must be addressed specifically in treatment. In this way, we may avoid the usual error of diagnosing "just low back pain," which leads to inadequate treatment of a definition rather than a complicated disease. Because persistent LBP might have numerous pain generators at the same time, a multidisciplinary diagnosis and multimodal therapy are required [25].

4. Anatomy of the Low Back

There are five vertebrae in the lumbar spine (L1–L5). The lumbar spine's complex structure is made up of a series of robust vertebrae connected by joint capsules, ligaments, tendons, and muscles, as well as considerable innervation. Because it must protect the spinal cord and spinal nerve roots, the spine is built to be sturdy. At the same time, it is extremely adaptable, allowing for motion on a variety of planes.

The symphyseal joints between the vertebral bodies, with an IVD in between, offer motion to the vertebral column. The facet joints, which are placed between and behind neighbouring vertebrae, help to stabilise the spine. They can be found at all levels of the spine and contribute around 20% of the torsional (twisting) stability in the neck and lower back. [20] Ligaments help to keep joints stable during rest and activity, preventing hyperextension and hyperflexion injuries. The anterior longitudinal ligament (ALL), posterior longitudinal ligament (PLL), and ligamentum flavum are the three primary ligaments (LF). The canal is flanked on one side by vertebral bodies and discs and on the other side by laminae and LF. The ALL and PLL respectively run the whole length of the spine, anteriorly and posteriorly. The intervertebral foramen allows spinal nerves and arteries to exit to the side. The corresponding foramen beneath each lumbar vertebra is where spinal nerve roots escape. The L1 neural foramina are positioned directly below the L1 vertebra, from whence the L1 nerve root emerges.

The IVDs are found between the vertebrae. They are compressible constructions that use osmotic pressurisation to distribute compressive stresses. The annulus fibrosus (AF), a concentric ring structure of organised lamellar collagen that surrounds the proteoglycan-rich inner nucleus pulposus in the IVD, is a concentric ring structure of organised lamellar collagen that surrounds the proteoglycan-rich inner nucleus pulposus (NP). Except for the periphery, discs are avascular throughout maturity. The human disc has some vascular supply at birth, but these arteries quickly fade, leaving the disc with minimal direct blood supply in a healthy adult [27]. As a result, the cartilaginous endplates close to the vertebral body provide metabolic support for much of the IVD. The recurrent sinuvertebral nerve, a meningeal branch of the spinal nerve, innervates the region around the disc space [28].

Extensors, flexors, lateral flexors, and rotators are the four functional groups of muscles that control the lumbar spine. The lumbar vertebrae are supplied with blood via lumbar arteries that branch out from the aorta. The lumbar arteries branch out into smaller anterior and posterior branches when they enter the intervertebral foramen at each level [29]. The venous drainage system runs parallel to the artery system [30].

The conus medullaris is formed by the termination of the spinal cord within the lumbar spinal canal at the lower border of the L2 vertebra [31]. The link between the dorsal or posterior (somatic sensory) root from the posterolateral side of the spinal cord and the ventral or anterior (somatic motor) root from the anterolateral aspect of the cord gives rise to all lumbar spinal nerve roots [31]. The roots then travel through the spinal canal, forming the cauda equina, before emerging as a single pair of spinal nerves at their respective intervertebral foramina. Motor nerve fibre cell bodies are found in the ventral or anterior horns of the spinal cord, while sensory nerve fibre cell bodies are found in the dorsal root ganglion (DRG) at each level. The lumbar spinal nerves have one or more recurrent meningeal branches, known as sinuvertebral nerves. The sinuvertebral nerve, also known as Luschka's nerve, is a recurrent branch of the spinal nerve formed by the merger of the grey ramus communicans (GRC) with a tiny branch from the proximal end of the anterior main ramus. This mixed nerve re-enters the spinal canal and gives out ascending and descending anastomosing branches for the posterolateral annulus, the posterior vertebral body and periostium, and the ventral meninges [32,33]. The sinuvertebral nerves communicate with radicular branches both above and below the site of entry, as well as on the contralateral side, making it difficult to pinpoint pain caused by their involvement [34]. In addition, the facet joints are innervated on two levels, including somatic and autonomic components. The autonomic afferents transmit referred pain, whereas the former transmit well-defined local

pain.

5. Pathophysiology of Spinal Pain

Nociceptors, which are specialized peripheral sensory neurons that warn us to potentially harmful stimuli at the skin by transducing these stimuli into electrical impulses that are sent to higher brain regions, are responsible for pain [35]. The DRG houses the neuronal body of nociceptors, which are pseudo-unipolar primary somatosensory neurons. The peripheral branch innervates the skin, whereas the central branch synapses on second-order neurons in the dorsal horn of the spinal cord [36]. The second-order neurons project to the mesencephalon and thalamus, which link to the somatosensory and anterior cingulate cortices, respectively, to control sensory-discriminative and affective-cognitive pain aspects. [37] The spinal dorsal horn, which is made up of multiple interneuron populations that create descending inhibitory and facilitatory pathways that can influence the transmission of nociceptive signals, is a prominent location of somatosensory information integration [38]. If the unpleasant stimulus continues, peripheral and central sensitization can develop, turning acute pain into chronic pain. The increase in excitability of neurons in the central nervous system is known as central sensitization, and it occurs when normal inputs start to elicit aberrant responses [37]. It causes tactile allodynia, or pain triggered by gentle skin brushing, as well as the extension of pain hypersensitivity beyond a region of tissue injury. TMD, LBP, osteoarthritis, fibromyalgia, headache, and lateral epicondylalgia are only a few of the chronic pain diseases that cause central sensitization [39]. Despite advances in our understanding of the mechanisms that contribute to central sensitization, [40], [41] remains difficult to treat. LBP chronification is aided by peripheral and central sensitization. In fact, even minor posture alterations can cause long-term inflammation in the joints, ligaments, and muscles that support the low back column's stability, contributing to both peripheral and central sensitization. Furthermore, delta fibres are abundantly innervated in joints, discs, and bone and their continual stimulation might readily lead to central sensitization.

6. Type of Spinal Pain According to Pain Generator

Despite the International Association for the Study of Pain's [41] efforts, there is still some ambiguity in the medical profession over the definitions of back pain, referred pain, radicular pain, and radiculopathy. However, in order to determine the appropriate therapy, a detailed diagnostic assessment is required. Inadequate diagnostic abilities of a non-specialized clinician in this illness, attributable to poor clinical and instrumental analysis, or a therapeutic strategy aimed toward managing the symptom (pain) rather than the pain generating processes [25].

LBP is commonly thought to be nonspecific [42], and the myth that the origin of 80–90% of LBP patients is unclear has persisted for decades [43]-[47].

For example, among fibromyalgia patients, muscle tension and spasm are among the most prevalent causes of LBP. In other circumstances, LBP is caused by a variety of pain generators, each with its own set of symptoms, such as radicular, facet joint, sacroiliac, and discogenic pain, as well as spinal stenosis.

7. Radicular Pain

Radicular pain is the pain caused by ectopic discharges from an inflamed or lesioned dorsal root or its ganglion; the pain usually radiates in a dermatomal pattern from the back and buttocks into the leg [46]. The most prevalent cause is disc herniation, and the most common pathophysiological mechanism is inflammation of the afflicted nerve rather than compression. Radicular pain is a type of pain that radiates down the nerve root without causing neurological damage. Even though it is nociceptive pain, radicular pain differs from typical nociception in that the axons are activated from the perinevrium rather than along their path or in their peripheral terminals [42], [48]. Radicular discomfort is distinct from radiculopathy in a number of ways. Conduction down a spinal nerve or its roots is hampered by radiculopathy. Numbness (dermatomally distributed) is caused by sensory fibre impairment; nevertheless, blocking of motor fibres induces weakness (myotomal). Reduced reflexes can be caused by sensory or motor obstruction [46]. Although radiculopathy and radicular pain are frequently seen together, radiculopathy can occur without pain and radicular pain can occur without radiculopathy [48], [49]. It is vital to note that, contrary to common opinion, there is no way to tell the difference between L4, L5, and S1 radicular pain patterns [50], [51]. An MRI may be the best noninvasive test to establish lumbar disc herniation with radiculopathy based on the patient's history and physical examination results. This is especially significant if an invasive therapy or a clearer definition of the neurological dysfunction is required. The test for determining the existence of lumbar disc herniation is computed tomography (CT) or CT myelography, which is useful for people who cannot have an MRI because it is contraindicated or for those who have an MRI that is inconclusive. Electrodiagnostic investigations can also be used to diagnose nerve root compression, albeit they cannot discriminate between lumbar disc herniation and other types of nerve root compression. Regrettably, we must point out that radiculopathy can exist without radicular discomfort, and vice versa. For these reasons, electrodiagnostic testing is only indicated as a second-line strategy to determine if there is a simultaneous presence of peripheral neuropathy or neuralgia, or to monitor the lesioned nerve's deterioration [52].

8. Facet Joint Syndrome

The lumbar zygapophyseal joints are the lumbar column's posterior articular process. They are made up of the upper vertebra's inferior process and the lower vertebra's superior articular process [53]. The medial branches of the dorsal rami supply them. There are many free and encapsulated nerve terminals in these joints [54], which activate nociceptive afferents and are regulated by sympathetic efferent fibres [55]. Up to 30% of CLBP cases are thought to be due to lumbar

zygapophyseal or "facet" joint pain [56], with nociception originating in the synovial membrane, hyaline cartilage, bone, or fibrous capsule of the facet joint [57].

Facet joint syndrome is sometimes difficult to diagnose and requires a thorough clinical examination as well as a thorough study of radiological examinations. Patients commonly report of LBP that radiates to the thigh or groyne, with or without somatic referral to the legs terminating above the knee. There is no radicular pattern to be found. Back discomfort is often off-center, and the severity of the pain is greater than that of the legs; pain worsens with hyperextension, rotation, lateral bending, and walking uphill. It gets worse when you get out of bed or try to get up after a lengthy period of sitting. Finally, patients frequently complain of back stiffness, which is most noticeable in the morning [58, 59]. Jackson was able to link seven characteristics to facet pain: increased age, past bouts of LBP, normal gait, maximum pain with lumbar extension but failure to worsen pain with the Valsalva technique, and lack of leg pain or muscle spasm [59, 60].

There are no pathognomonic features to look for when diagnosing lumbar facet syndrome using radiography [61]. We can use MRI to detect non-specific symptoms of arthrosis, osteophytes, and flaval ligament hypertrophy. However, CT is the recommended imaging tool for better studying arthrosis disorders, even if radiation exposure should be considered [58]. X-rays, particularly dynamic projections, are one of the most significant tests because they can demonstrate column instability (can be enhanced with flexion and extension of the low back column) as well as an evident overload of these joints [60]. In conclusion, despite the value of neuro-imaging in the diagnosis of facet joint disorders, history and clinical examination remain essential.

9. Sacroiliac Joint Pain

The sacroiliac joints (SIJs) are responsible for providing solid yet flexible upper-body support [62], [63]. SIJs have a role in sacral movement, which has a direct impact on the discs and most likely, the upper lumbar joints. Its innervation is unknown, however it has been suggested that it is innervated by branches from the ventral lumbopelvic rami [64], though this has not been proven. Small branches from the posterior rami, on the other hand, have been found to innervate the SIJ [65], [66]. Patel et al. [66] reported that neurotomy of the L5 dorsal main ramus and lateral branches of the dorsal sacral rami from S1 to S3 successfully alleviated SIJ discomfort in 2012 research [63]. As a result, there is adequate evidence that this method is useful in determining diagnosis and prognosis. In many individuals with CLBP, the SIJ is widely established as a cause of pain [67], [68]. Ligamentous or capsular tension, external compression or shear pressures, hypermobility or hypomobility, changed joint mechanics, and myofascial or kinetic chain dysfunction generating inflammation are all considered to cause pain [69]. Osteoarthritis is an intra-articular cause of SIJ pain; extra-articular causes include enthesis/ligamentous sprain and primary enthesopathy. Ligamentous, tendinous, or fascial attachments, as well as other cumulative soft tissue injuries that occur posterior to the dorsal portion of the SIJ, might cause pain. In a physical examination, it is critical to look at how the joint moves, such as with a stress test that involves pressing down on the iliac crest (pelvis) or upper thigh, which might mimic the patient's symptoms.

SIJ discomfort is frequently misdiagnosed. Every time a patient complains of postural LBP that worsens in a sitting posture and with postural alterations, it must be considered. Furthermore, because both are linked to postural issues, it's probable that SIJ discomfort is closely linked to facet joint disorders. Finally, it is vital to keep in mind that SIJ discomfort might be an indication of rheumatoid arthritis. The presence of articular effusion and inflammation on an MRI scan (particularly if bilateral) might alert the doctor to the possibility of this illness.

10. Lumbar Spinal Stenosis

Congenital lumbar spinal stenosis (CLSS) or acquired lumbar spinal stenosis (ALSS) or both can be detected by inflammatory/scar tissue following spine surgery or, even if there has been no prior surgery, by disc herniation, ligament thickening, or articular process hypertrophy [71]. The majority of LSS cases are degenerative, meaning they are caused by changes in the spine as people age [72]. A gradual narrowing of the central spinal canal and lateral recesses, as well as compression of neurovascular structures, characterizes LSS [73]. The typical lumbar spinal canal measures 15 to 27 millimetres in diameter. Even if a stenosis with a diameter of 12 mm or less in certain cases might be symptomatic, we can define lumbar stenosis as a spinal canal diameter of less than 10 mm. The usual foraminal height is 20 to 23 mm, with 15 mm or less indicating probable foraminal stenosis [74]. In adults over the age of 65 years, degenerative LSS is the most prevalent reason for spine surgery [73]. Midline back discomfort, radiculopathy with neurologic claudication, motor weakness, paresthesia, and sensory nerve dysfunction are the most common symptoms of lumbar stenosis [75]. Depending on the kind of LSS, symptoms may have a variable distribution. In a non-dermatomal distribution, if the LSS is central, the region between the facet joints may be involved, and discomfort may be bilateral. Because particular nerves are pinched in lateral recess stenosis, symptoms are frequently detected dermatomally, suggesting unilateral radiculopathy [76]. Prolonged standing or lumbar extension might increase the pain, whereas trunk flexion, sitting, stooping, or reclining can help. As the problem worsens, sitting and lying down become less efficient at relieving pain, and rest pain or a neurogenic bladder can occur in extreme cases [76], [77]. The characteristic symptom of LSS is neurogenic claudication pain, which is produced by venous congestion and hypertension around nerve roots. Standing erect and walking downhill aggravate pain, but laying supine, rather than prone, sitting, squatting, and lumbar flexion, alleviates it [78], [79].

A combination of history, physical examination, and imaging is used to diagnose LSS [75]. Age, radiating leg discomfort increased by standing or walking, and the lack of pain when seated are the most relevant data from the history [80]. A positive "stoop test" [79], [80], which involves asking the

patient to walk quickly, may demonstrate a favorable gait and posture afterward. Patients may have sensory symptoms first, and then motor problems as the pain worsens and symptoms may ease if they adopt a stooped posture [80]. Patients may experience similar alleviation if they sit in a chair that is leaned forward [81].

The use of magnetic resonance imaging (MRI) to confirm the diagnosis of LSS is advised since it allows for better evaluation of the spinal canal and the anatomic interaction between spinal and neural components [80]. Untreated LSS has an unknown natural path. According to the clinical recommendations of the North American Spine Society (NASS), the natural course is favourable in a third to half of individuals with clinically mild to severe LSS [82]. Other studies have found that the illness worsens in approximately a third of patients and improves in about a third, with the majority of patients remaining unaltered for up to 8 years [83]-[85].

11. Discogenic Pain

CLBP is thought to be caused by disc degeneration (DD) in 39 % of cases [86]. Its symptoms are atypical, axial, and without radicular radiation, and they occur without deformity or instability of the spine. DD is frequently used to rule out other kinds of CLBP. It is defined pathologically by the degeneration of the NP matrix inside the disc, as well as radial and concentric cracks in the AF [87].

Despite multiple recent breakthroughs, the major question remains how inflammation is triggered and maintained in order to cause CLBP. The development of neurons capable of communicating pain deep into the annular structures might be one cause [88]. Another theory involves the action of pro-inflammatory cytokines (IL-1beta, IL-6, and IL-8) and matrix degrading enzymes (MMP-1, MMP-3, and MMP-13) on a class of molecules known as damage associated molecular patterns (DAMPs), which include hyaluronic acid and fibronectin fragments, to stimulate sterile disc inflammation [87]. Also, hypoxic settings may promote subclinical anaerobic bacterial infection, which may play a role in the development of discogenic pain [88].

Changes in the endplates and vertebral bone marrow, such as edema in the vertebral bodies, can be detected by MRI imaging. Clinical research has shown that amoxicillin-clavulanate improves the symptoms of certain people with LBP [88], [89]. Furthermore, because advanced glycation end products (AGEs) stimulate catabolism and promote inflammation, diabetes raises the chance of developing painful DD [90].

The use of an MRI to determine if a disc is bothersome is inconclusive [91]. Provocation discography [92] seeks to mimic a patient's discomfort by injecting contrast into the disc during live fluoroscopy and imaging it with CT to reveal any related morphological abnormalities.

The clinical value of discography and its diagnostic accuracy, on the other hand, is a source of debate due to its low specificity. Aside from the reported consequences of discitis, cerebral damage, visceral injury, and dye responses [93], needle puncture of the lumbar disc has been shown to cause rapid MRI-documented DD. The process is likely complex, involving needle-induced structural damage, pressurisation, and contrast medium toxicity [94].

12. Conclusion

One of the most prevalent symptoms and ailments that people to seek medical help is low back pain. Back pain has substantial epidemiological and economic consequences, which are predicted to worsen as a result of a mix of changing attitudes and expectations, medical therapy approaches, and social assistance.

As a result, LBP must always be treated as a complicated condition in which a proper identification of pain generators is required prior to beginning any treatment. All of the current recommendations emphasize the need of a multimodal and multidisciplinary approach in determining a plan to tackle the problem rather than only relieve symptomatic pain. Finally, diligent monitoring is required to adjust our therapeutic approach to changing CLBP clinical symptoms.

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