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CHRONIC LOW BACK PAIN: A REVIEW OF DIAGNOSTIC ALGORITHMS, PATHOPHYSIOLOGICAL MECHANISMS, AND EVIDENCE-BASED MANAGEMENT IN PRIMARY CARE

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ABSTRACT

Background and Objectives: This narrative review aims to evaluate the systemic challenges in managing Chronic Low Back Pain (CLBP) and establish an evidence-based operational framework for primary care.
Methods: Utilizing data synthesized from landmark epidemiological reports, functional neuroimaging studies, and guidelines from the National Institute for Health and Care Excellence (NICE) and the American College of Physicians (ACP), the analysis focused on diagnostic triage, routine imaging stewardship, and the neurobiology of pain chronicity.
Findings: Findings indicate that the traditional biomedical model drives an "iatrogenic paradox," where over-medicalization and premature imaging induce a nocebo-driven labeling effect, worsening long-term disability. True chronicity is predominantly mediated by central sensitization, nociplastic pathways, and psychosocial yellow flags rather than structural spinal abnormalities. Consequently, clinical assessment must prioritize a "three-bucket" diagnostic triage and validated risk stratification over incidental pathoanatomical findings.
Conclusions: Addressing the escalating global burden of CLBP requires a decisive paradigm shift toward a biopsychosocial, stepped-care model. Primary care physicians must act as rigorous gatekeepers against low-value medicalization, instead prioritizing patient education, early reassurance, and active, tailored exercise therapy. Future strategies should emphasize clinical phenotyping and implementation science to effectively bridge the gap between research evidence and routine primary care practice.

KEYWORDS

Chronic Low Back Pain, Biopsychosocial Model, Iatrogenic Paradox, Evidence-Based Medicine, Rehabilitation, Primary Care

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List of Abbreviations: ACP – American College of Physicians; CBT – Cognitive-Behavioral Therapy; CES – Cauda Equina Syndrome; CLBP – Chronic Low Back Pain; CNS – Central Nervous System; CS – Central Sensitization; fMRI – Functional Magnetic Resonance Imaging; GDP – Gross Domestic Product; LBP – Low Back Pain; MRI – Magnetic Resonance Imaging; NICE – National Institute for Health and Care Excellence; NSAIDs – Non-steroidal anti-inflammatory drugs; NS-LBP – Non-specific low back pain; PCP – Primary Care Physician; QST – Quantitative Sensory Testing; SBST – STarT Back Screening Tool; SNRI – Serotonin–norepinephrine reuptake inhibitor.

1. Introduction: The Global Burden and the Iatrogenic Paradox

Chronic Low Back Pain (CLBP), clinically defined as axial or radicular pain persisting for 12 weeks or longer, represents the primary global cause of years lived with disability (YLDs), posing an unprecedented challenge to modern public health systems [1]. The magnitude of this epidemiological trajectory is comprehensively documented by the Global Burden of Disease Study 2021, which estimated that low back pain affected approximately 619 million individuals globally in 2020. Driven by global demographic shifts, rapid urbanization, and an aging population, this figure is projected to surpass 843 million cases by 2050 [1, 2]. The socioeconomic repercussions of CLBP are commensurately staggering, characterized by a profound disparity between direct healthcare expenditures and even more substantial indirect societal costs. In the United States alone, spending on low back and neck pain reached an estimated $134.5 billion in 2016, representing the highest healthcare expenditure among 154 clinical conditions analyzed in a comprehensive national registry [3]. However, the true economic rationale for clinical intervention is primarily driven by indirect costs related to lost labor productivity. CLBP is globally recognized as the leading cause of work absenteeism and presenteeism. Systematic reviews indicate that in industrialized nations, indirect costs—encompassing lost wages, premature retirement, and disability payments—account for approximately 80% to 90% of the total financial burden of the disease [2, 4]. These productivity losses drain billions from national Gross Domestic Products (GDP) annually, positioning CLBP not merely as an individual clinical issue, but as a major threat to macroeconomic stability and a primary driver of long-term labor market exit [2, 4, 5].

1.1. The Research Problem Despite this exponential increase in healthcare expenditure, the widespread proliferation of advanced diagnostic imaging, and the rising frequency of interventional spinal procedures, disability rates related to LBP continue to rise unabated [2, 6]. This clinical discrepancy highlights a profound "iatrogenic paradox": the intensification of biomedical medical care—characterized by the routine prescription of opioids, unnecessary early advanced neuroimaging, and elective spinal fusion surgeries—has paradoxically correlated with worsening long-term disability outcomes rather than improvement, a crisis heavily emphasized by the Lancet Low Back Pain Series [6]. This trajectory suggests a fundamental failure of the prevailing, deterministic biomedical model, which inappropriately seeks to identify and surgically or pharmacologically correct specific isolated anatomical defects.

1.2. Rationale and Objectives Consequently, modern evidence-based guidelines necessitate a decisive paradigm shift in frontline clinical practice: moving away from a curative, pathoanatomical "fix-it" approach toward a rehabilitative, biopsychosocial model focused on self-management, functional restoration, and the mitigation of complex psychosocial risk factors [6, 7, 8]. Within this framework, the Primary Care Physician (PCP) must adopt a central role as a rigorous gatekeeper against low-value, potentially harmful medicalization. However, a significant gap remains in translating these high-level guideline recommendations into actionable, day-to-day primary care workflows. Therefore, the objective of this narrative review is to synthesize contemporary clinical data to address this implementation gap. Specifically, this study aims to:

- Evaluate the clinical and diagnostic efficacy of the "three-bucket" triage model and imaging stewardship in primary care.
- Analyze the neurobiological and psychosocial mechanisms driving pain chronicity, specifically central sensitization and "yellow flags".
- Establish a clinically actionable, evidence-based stepped-care hierarchical matrix to guide PCPs in selecting high-value non-pharmacological and pharmacological interventions.

2. Methodology

This study utilizes a comprehensive, structured narrative review design to synthesize, critically evaluate, and integrate contemporary clinical practice guidelines, landmark epidemiological reports, and high-quality clinical trials regarding the management of Chronic Low Back Pain (CLBP) in primary care settings. To ensure academic rigor, the review process was divided into three distinct phases: literature strategy, selection screening, and data synthesis.

2.1. Search Strategy and Data Sources A systematic literature search was executed across major biomedical databases—including PubMed, MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials (CENTRAL)—from database inception through January 2026. The search architecture utilized combinations of Medical Subject Headings (MeSH) terms and free-text keywords, structured via Boolean operators (AND, OR). The primary search strings included:

- "chronic low back pain" OR "LBP"
- "biopsychosocial model" OR "yellow flags"
- "central sensitization" OR "nociplastic pain"
- "imaging stewardship" OR "labeling effect" OR "overmedicalization"
- "stepped-care" OR "stratified care" OR "primary care guidelines"

2.2. Inclusion and Screening Criteria

The resulting literature was screened based on strict clinical relevance to frontline primary care medicine. Prioritization was given to:

1. Clinical Guidelines: Consensus statements and clinical practice guidelines from major international bodies, specifically the National Institute for Health and Care Excellence (NICE, NG59) and the American College of Physicians (ACP).
2. Epidemiological Data: Large-scale global reports, such as the Global Burden of Disease Study.
3. Mechanistic Evidence: Functional magnetic resonance imaging (fMRI) and neuroimaging studies investigating central nervous system processing and nociplastic pathways.
4. Clinical Trials: Randomized controlled trials (RCTs) and high-quality systematic reviews evaluating the long-term clinical and functional efficacy of non-pharmacological, pharmacological, and interventional therapies.

2.3. Data Extraction and Thematic Synthesis

Data were systematically extracted and evaluated using a qualitative thematic synthesis technique. The analytical framework focused on three core clinical areas: the diagnostic performance and accuracy of the "Three-Bucket Triage" model, the predictive validity of clustered red flags versus isolated signs, and the psychological and functional impact of the iatrogenic labeling effect. The analysis specifically cross-examined the pathophysiological discordance between objective structural imaging abnormalities and subjective clinical symptom presentation to map the neurobiological and psychosocial mechanisms of pain chronicity. Finally, these synthesized findings were integrated to construct a clinically actionable, six-step hierarchical matrix (detailed in Results), directly mapping patient risk profiles and symptom severity to high-value primary care interventions.

3. Results:

3.1 Diagnostic Architecture: Triage, Risk Stratification, and Imaging Stewardship The initial assessment of LBP in primary care must be structured to prioritize rapid risk stratification over definitive pathoanatomical diagnosis, which is often elusive. Contemporary guidelines from bodies such as the National Institute for Health and Care Excellence (NICE NG59, 2016) and the American College of Physicians (ACP, 2017) advocate for a pragmatic "three-bucket" diagnostic triage [7, 8]. The vast majority of cases, estimated at 85–90%, are classified as non-specific low back pain (NS-LBP). By definition, NS-LBP includes patients where the nociceptive source cannot be reliably attributed to a specific spinal structure (e.g., nerve root, vertebral body, or infection) [7]. Management for this group is focused on patient education, promotion of active coping strategies and risk factors modification.

The second category, accounting for approximately 5–10% of patients, comprises radicular pain and radiculopathy. It is characterized by pain radiating into the lower limb, most commonly in accordance with the L4, L5, or S1 dermatomal distribution, and is usually associated with compression of a spinal nerve root. Radiculopathy is distinguished by the presence of objective neurological deficits (sensory loss, motor weakness, or reflex changes) [9]. The third and the rarest group, representing less than 1% of LBP cases, includes specific spinal pathology. This category encompasses serious, potentially life-threatening or time-sensitive conditions that require urgent diagnostic evaluation and treatment.It includes serious, time-sensitive etiologies such as vertebral fracture, malignancy, infection (discitis/osteomyelitis) or Cauda Equina Syndrome (CES) [8, 10].

Table 1. Three-Bucket Diagnostic Triage Model for Low Back Pain

Category	Estimated prevalence	Defining clinical characteristics	Management focus	Imaging recommendations
Non-specific low back pain (NS-LBP)	85-90%	No reliable attribution to a specific spinal structure; localized lumbar pain with or without non-dermatomal radiation; absence of objective neurological deficits	Patient education, encouragement of activity and modification of risk factors	Not recommended routinely; reserved for cases with clinical aggravation or red flag features
Radicular pain / radiculopathy	5-10%	Lower limb pain radiating into the lower limb, most commonly in accordance with the L4, L5, or S1 dermatomal distribution; radiculopathy defined by objective neurological deficits (sensory, motor, or reflex abnormalities)	Targeted conservative management and close monitoring of neurological status	Considered in the presence of persistent or progressive neurological deficits
Specific spinal pathology	<1%	Clinical features suggestive of serious pathology (e.g. fracture, malignancy, infection, cauda equina syndrome)	Urgent diagnostic evaluation and pathology-specific intervention	Indicated urgently, guided by the suspected underlying condition

Table 1. description: The table outlines a pragmatic classification framework for low back pain, detailing the relative frequency of each diagnostic category, their defining clinical characteristics, and corresponding management strategies. Non-specific low back pain constitutes the majority of presentations and typically does not warrant routine imaging. Radicular pain or radiculopathy occurs less frequently and requires focused treatment with imaging reserved for selected cases, whereas specific spinal disorders are uncommon but demand prompt diagnostic assessment and urgent imaging.

A critical component of the clinical examination is screening for serious spinal pathology through the assessment of “Red Flags.” Recent systematic reviews of the "Red Flags" caution that many individual red flags have low specificity, leading to high false-positive rates that contribute to unnecessary patient’s anxiety and imaging [10]. For this reason, red flags should be interpreted as clusters of signs and risk factors that collectively increase the likelihood of serious disease [10, 11]. Particularly important is CES, which constitutes a true surgical emergency requiring immediate investigation and decompression. Its most reliable features include new-onset bladder dysfunction (urinary retention or incontinence), saddle anesthesia involving the perineal region, and rapidly progressive or severe bilateral neurological deficits[10]. In the context of suspected malignancy, the strongest predictive factor is a prior history of cancer (especially breast, lung, prostate, renal and thyroid malignancies). Additional factors include unexplained weight loss, age over 50 years and severe pain that fails to improve after at least four weeks of appropriately delivered conservative management. The risk of vertebral fracture is increased in the presence of significant trauma, prolonged systemic corticosteroid use and advanced age, typically over 70 years [11]. One of the most robustly evidence-based recommendations in contemporary LBP management is to avoid routine imaging—such as plain radiography, magnetic resonance imaging, or computed tomography—in patients with NS-LBP who show no signs of neurological progression or red flags [7, 8]. This recommendation is grounded in two key scientific observations. First, there is a poor correlation between structural abnormalities identified on imaging and the presence of pain. High-quality systematic reviews, such as the seminal work by Brinjikji et al. (2015), demonstrate an exceptionally high prevalence of degenerative changes in asymptomatic individuals. For example, disc degeneration is present in 37% of asymptomatic 20-year-olds and 96% of asymptomatic 80-year-olds, and disc bulges are found in 30% of asymptomatic 20-year-olds [12]. This data confirms that LBP is frequently

unrelated to the structural abnormalities seen on imaging. Second, early and unnecessary imaging promotes the detection of Iatrogenic Harm and can cause The Labeling Effect. It means that Early imaging often leads to the diagnosis of "incidentalomas" (findings unrelated to the current pain). When patients are informed of "degenerative disc disease," it induces a nocebo effect, fostering fear-avoidance beliefs, catastrophizing, and the perception of a fragile spine, which subsequently drives increased disability and unnecessary referrals for specialist consultations and surgical interventions [6,13].

3.2 Pathophysiology of Chronicity: The Biopsychosocial Axis

Understanding the etiology of CLBP requires recognizing that pain persistence beyond expected tissue healing time (three months) is rarely attributable to ongoing peripheral tissue damage. Instead, chronic pain is driven by a complex interplay of neurobiological and psychosocial factors. In CLBP, the nervous system frequently undergoes maladaptive neuroplastic changes referred to central sensitization (CS).CS refers to the amplification of neural signaling within the central nervous system (CNS), characterized by hyperexcitability of spinal and supraspinal neurons. This phenomenon leads to pain hypersensitivity, which is clinically expressed as hyperalgesia—an exaggerated pain response to noxious stimuli—and allodynia, in which pain is elicited by normally non-noxious stimuli such as light touch [14]. As a consequence, the chronic pain experienced by many individuals with CLBP is increasingly classified as nociplastic pain, defined as pain arising from altered nociceptive processing despite the absence of clear evidence of actual or threatened tissue damage. Functional magnetic resonance imaging (fMRI) studies in patients with CLBP have demonstrated a shift in neural activity from primary sensory-discriminative pathways, particularly within the somatosensory cortex, toward emotional-affective brain circuits, including the prefrontal cortex, amygdala, and anterior cingulate cortex. This neurofunctional reorganization provides a mechanistic explanation for the high comorbidity of CLBP with mood disorders such as anxiety and depression and confirms that chronic pain is a disorder of the entire CNS, not just the periphery [15]. Importantly, the most potent predictors of the transition from acute low back pain to chronic, disabling CLBP are not biomechanical or structural factors but psychosocial variables, commonly known as “yellow flags”. These include fear-avoidance beliefs, characterized by the conviction that movement is dangerous and will result in catastrophic spinal damage; pain catastrophizing, defined as an exaggerated negative cognitive and emotional response to actual or anticipated pain involving rumination, magnification, and feelings of helplessness; and emotional distress, such as elevated levels of anxiety, depression, and stress, which can lower pain thresholds through dysregulation of descending pain modulatory pathways [16]. To facilitate the clinical identification of these prognostic factors, the STarT Back Screening Tool (SBST) is recommended for use in primary care settings [13]. This validated nine-item questionnaire efficiently stratifies patients into low-, medium-, or high-risk categories for persistent disability, allowing the PCP to direct the patient towards the appropriate intensity of care (simple advice vs. psychologically informed therapy) [13].

3.3 Evidence-Based Therapeutic Strategy: A Stepped-Care Approach

The contemporary management of CLBP should be based on a stepped-care approach, in which treatment intensity is progressively escalated in patients who fail to respond to initial interventions [2,7,8]. This model emphasizes the use of effective, low-risk strategies as first-line therapy, while reserving more invasive or specialized treatments for carefully selected individuals [5,6]. Clinical decision-making should be guided not only by symptom severity but also by psychosocial risk stratification, as psychosocial factors play a central role in the development of persistent disability [13, 16]. Independent of risk profile, the foundation of CLBP management is active, non-pharmacological care [7,8]. Patient education and reassurance represent a critical first step in treatment [2,6]. Clinicians should aim to de-threaten the pain experience by explaining that the spine is a robust structure and that pain does not necessarily indicate ongoing tissue damage [2,9]. Patients should be reassured that the prognosis of non-specific low back pain is generally favorable [7,9]. Such clear and positive messaging is essential for reducing fear-avoidance beliefs, which are strongly associated with pain chronicity [16]. Prolonged bed rest should be actively discouraged, and patients should be advised to remain physically active and continue their usual daily activities within tolerable limits [7,8]. Exercise therapy constitutes the most consistently supported non-invasive intervention for CLBP [17]. Systematic reviews and clinical guidelines demonstrate moderate but sustained improvements in pain and function with structured exercise programs [8,17]. Importantly, no single form of exercise has been shown to be universally superior. Motor control exercises, Pilates-based programs, general strengthening, and aerobic training appear to provide comparable benefits [17]. Consequently, exercise selection should prioritize patient preference, functional limitations, and long-term adherence rather than rigid adherence to a specific protocol [7,8]. The therapeutic effects of exercise are likely mediated through multiple mechanisms, including activation of

descending inhibitory pain pathways and exercise-induced hypoalgesia, as well as psychological effects such as graded exposure to movement and increased self-efficacy [16,18]. Current recommendations support combining aerobic conditioning with trunk muscle strengthening and flexibility exercises, tailored to individual functional deficits [8,17,18]. In patients identified as being at high risk for persistent disability, particularly those with prominent psychosocial factors such as fear of movement or pain catastrophizing, exercise therapy alone may be insufficient. In this subgroup, psychologically informed physical therapy should be considered [13,16]. This approach integrates cognitive-behavioral principles into physical rehabilitation, directly addressing maladaptive beliefs and avoidance behaviors [16]. For individuals with severe or long-standing disability, multidisciplinary biopsychosocial rehabilitation programs involving coordinated care from physiotherapists, psychologists, and physicians have demonstrated superiority over standard care in reducing long-term disability and improving functional outcomes [19].

Table 2. Stepped-care approach to chronic low back pain management

Step	Intervention	Indication / Target Patients	Key Evidence / Notes
1	Education & reassurance	All patients with non-specific CLBP	Reduces fear-avoidance beliefs, improves prognosis [2, 7]
2	Structured exercise therapy	Patients without high psychosocial risk	Moderate improvements in pain and function; various exercise types effective [8, 17]
3	Psychologically informed physical therapy	High psychosocial risk (fear of movement, catastrophizing)	Integrates CBT into rehabilitation, reduces disability [13, 16]
4	Multidisciplinary biopsychosocial rehabilitation	Severe or long-standing disability	Superior to standard care in improving function and reducing long-term disability [19]
5	Adjunct pharmacological therapy	Patients requiring symptom relief to engage in rehabilitation	NSAIDs preferred; duloxetine in central sensitization; avoid long-term opioids [8,14,20,22]
6	Interventional/surgical procedures	Selected patients with refractory symptoms or structural pathology	Epidural injections for radiculopathy; surgery only with clinical-radiological concordance [23, 24, 25]

Table 2 description: Table 2 illustrates a stepped-care approach for chronic low back pain management, ranging from low-risk interventions (education and exercise) to more specialized or invasive treatments. It also provides guidance on patient selection and summarizes key supporting evidence for each step.

Pharmacological treatment should be regarded as an adjunct to active rehabilitation rather than a primary therapeutic strategy [8, 20]. The main role of analgesic medication is to provide sufficient symptom relief to allow engagement in exercise and functional restoration. Among non-opioid analgesics, non-steroidal anti-inflammatory drugs remain the preferred first-line pharmacological option due to their established efficacy. Their use should be individualized, taking into account gastrointestinal and cardiovascular risk factors, and limited to the lowest effective dose for the shortest possible duration [20]. In selected patients with CLBP, particularly those with features suggestive of central sensitization, serotonin–norepinephrine reuptake inhibitors such as duloxetine may be considered. Clinical trials indicate modest reductions in pain intensity, likely mediated by enhancement of descending inhibitory pain mechanisms [14]. In contrast, paracetamol is no longer recommended for the management of low back pain, as high-quality randomized controlled trials have demonstrated no meaningful benefit compared with placebo [21]. Muscle relaxants may provide short-term relief during acute exacerbations but are limited by adverse effects, particularly sedation, and should therefore be used cautiously [20].

Table 3. Pharmacological options in chronic low back pain

Drug / Class	Indication	Efficacy	Key Limitations / Notes
NSAIDs	First-line for pain relief	Established moderate efficacy	Risk: GI, cardiovascular; use lowest effective dose for shortest duration [20]
Duloxetine (SNRI)	Central sensitization, neuropathic features	Modest pain reduction	Consider for selected patients; may enhance descending inhibitory pathways [14]
Muscle relaxants	Acute exacerbations	Short-term relief	Sedation, dizziness; caution in elderly [20]
Paracetamol	Previously used	Not recommended	No meaningful benefit over placebo [21]
Opioids	Rare, refractory cases	Minimal long-term benefit	High risk: dependence, overdose, opioid-induced hyperalgesia [22]

Table 3 description: Table 3 summarizes the pharmacological treatment options for chronic low back pain, including indications, efficacy, and limitations of each drug. It highlights first-line therapies and notes medications suitable only for selected patients, based on current evidence.

The use of opioids in CLBP warrants particular caution. Chronic opioid therapy for non-cancer pain has been identified as a major contributor to the opioid epidemic. Current guidelines strongly advise against initiating opioids for CLBP, except in rare and carefully selected cases. Long-term studies indicate minimal analgesic benefit, often below clinically meaningful thresholds, with no sustained improvement in function. In contrast, opioid therapy is associated with substantial risks, including dependence, overdose, and opioid-induced hyperalgesia, a paradoxical increase in pain sensitivity with prolonged use [22]. Interventional and surgical treatments should be reserved for a small subset of patients who have failed comprehensive, high-

quality non-surgical management [7,23,25]. Epidural steroid injections may provide short-term pain relief in patients with radicular symptoms and can occasionally facilitate participation in rehabilitation. However, systematic reviews have not demonstrated significant long-term benefits in pain or function, and these procedures are not indicated for non-specific axial low back pain. Facet joint interventions, such as medial branch radiofrequency denervation, may be considered in carefully selected patients with facet-mediated pain confirmed by diagnostic blocks, although the durability of treatment effects remains variable [23]. Surgical intervention should be considered only when clear clinical–radiological concordance is present. Decompressive procedures, including discectomy or laminectomy, may be appropriate for patients with severe, persistent radiculopathy or symptomatic spinal stenosis accompanied by neurological deficits that do not respond to prolonged conservative treatment [24]. In contrast, lumbar fusion for non-specific CLBP remains highly controversial. Multiple randomized controlled trials have failed to demonstrate superiority over intensive non-operative, cognitive-functional rehabilitation. As a result, spinal fusion should be limited to cases with clear structural instability, such as high-grade spondylolisthesis, or significant spinal deformity [25].

4. Discussion

The synthesis of contemporary evidence evaluated in this review underscores a critical paradigm shift in musculoskeletal medicine: the management of Chronic Low Back Pain (CLBP) must fundamentally transition from a traditional, pathoanatomical biomedical model to a resilient, systems-oriented biopsychosocial discipline. The clinical significance of our findings lies in the stark documentation of the "iatrogenic paradox," wherein routine, low-value medicalization—specifically premature advanced neuroimaging, speculative interventional procedures, and liberal opioid prescribing—directly correlates with worsening, rather than improving, long-term disability rates [2, 6]. Our analysis of the diagnostic architecture reinforces the necessity of the "three-bucket" triage model. By definitively isolating non-specific low back pain (NS-LBP), which constitutes 85–90% of presentations, from radicular syndromes and serious specific spinal pathologies (<1%), primary care physicians (PCPs) can effectively eliminate unnecessary diagnostic pathways [7, 8]. The empirical validation of imaging stewardship highlighted in this study exposes the clinical dangers of the "sensitivity-specificity paradox" inherent in modern spinal imaging. As demonstrated by Brinjikji et al. (2015), structural degenerative changes (e.g., disc bulges and facet arthropathy) are ubiquitous, normative features of the aging human spine, occurring with high frequency in completely asymptomatic populations [12]. Consequently, when a PCP routinely orders early MRIs or X-rays for non-specific LBP, these "incidentalomas" are often misattributed as the definitive source of pain. This induces a profound iatrogenic nocebo-driven labeling effect, cementing fear-avoidance beliefs, catastrophizing, and the perception of a fragile spinal structure in the patient's mind, which ultimately drives prolonged disability and low-value surgical interventions [6, 13]. A key contribution of this review is the detailed mapping of the pathophysiological shift from acute nociception to chronic nociplastic pain. Functional neuroimaging (fMRI) data convincingly demonstrate that as LBP transitions into chronicity, neural activity migrates away from classic sensory-discriminative regions toward emotional-affective cortical circuits [15]. This neurofunctional reorganization explains why true pain chronicity is predominantly mediated by central sensitization (CS) and psychosocial "yellow flags" rather than ongoing peripheral tissue damage [14, 16]. Clinically, this means that screening tools like the STarT Back Screening Tool (SBST) are not merely psychological adjuncts, but essential neurological risk-stratification instruments. Identifying fear-avoidance, catastrophizing, and emotional distress early allows the PCP to bypass standard, ineffective physical therapy and fast-track high-risk patients toward psychologically informed rehabilitation or multidisciplinary care [13, 19]. However, translating these robust, evidence-based recommendations into routine primary care workflows presents substantial systemic and logistical challenges. In day-to-day clinical practice, PCPs frequently face severe time constraints, where a brief, high-throughput consultation favors the rapid writing of an NSAID or opioid prescription, or the ordering of an MRI, over lengthier, complex counseling regarding active self-management and reassurance. Furthermore, wide gaps remain in implementation science; many healthcare systems lack integrated, interdisciplinary networks that connect primary care clinics directly with psychologists and specialized physiotherapists, rendering the deployment of multidisciplinary biopsychosocial rehabilitation (MBR) logistically unfeasible for the average practitioner [2].

4.1. Strengths and Limitations

The primary strength of this narrative review is its holistic synthesis of multidisciplinary data—uniting large-scale epidemiological reports, high-level clinical guidelines (NICE, ACP), and sophisticated functional neuroimaging insights—into a single, actionable stepped-care matrix for primary care practitioners. By bridging the gap between basic neuroscience (central sensitization) and clinical utility (pharmacological and interventional stewardship), this study provides a clear operational framework. Conversely, a key limitation inherent in the narrative review design is the potential for selection bias, as a systematic meta-analysis of individual therapeutic modalities was not performed. Additionally, while the recommended stepped-care model is highly robust in industrialized healthcare systems, its generalizability and cost-effectiveness in low-resource settings—where access to advanced non-pharmacological interventions, such as psychologically informed physical therapy or MBR, is severely restricted—warrants further empirical investigation.

4.2 Future Directions: Precision and Implementation Science

Current gaps in the management of CLBP are not primarily due to a lack of evidence, but rather to difficulties in translating existing knowledge into routine clinical practice. Bridging this gap requires focused efforts in two complementary areas: phenotyping and precision medicine, and implementation science. Phenotyping and precision medicine represent the first critical area. Moving beyond the broad and often uninformative diagnosis of NS-LBP, patients should be biologically characterized according to their predominant pain mechanisms—nociceptive, neuropathic, or nociplastic—using quantitative sensory testing (QST) and, where feasible, functional brain imaging [14]. This mechanism-based approach facilitates the identification of patients who are most likely to respond to specific interventions, such as serotonin-norepinephrine reuptake inhibitors (SNRIs) for nociplastic pain or targeted nerve blocks for neuropathic pain. By aligning treatment strategies with underlying pain mechanisms, clinicians can enhance therapeutic efficacy and reduce the reliance on generalized trial-and-error management. Implementation science constitutes the second essential area. Developing scalable strategies to integrate evidence-based care models, including the SBST-stratified approach, into everyday primary care is crucial for ensuring consistent delivery of effective treatments. Addressing socioeconomic and logistical barriers to structured physical and psychological rehabilitation is a key component of this effort. By focusing on practical implementation, evidence-based interventions can reach the patients who stand to benefit most, ultimately narrowing the gap between research and clinical practice [2].

5. Conclusions

Addressing the escalating global burden of Chronic Low Back Pain (CLBP) requires a decisive paradigm shift away from the traditional, structural-based biomedical approach toward an integrated, biopsychosocial, stepped-care model. Frontline clinical assessment must prioritize rapid risk stratification and the "three-bucket" diagnostic triage over incidental, low-value pathoanatomical findings. Evidence-based stewardship demonstrates that routine, early imaging in non-specific low back pain does not improve clinical outcomes; rather, it triggers a harmful iatrogenic "labeling effect" and a nocebo-driven cycle of fear-avoidance and catastrophizing that accelerates functional decline. True pain chronicity is predominantly driven by maladaptive neuroplastic changes within the central nervous system—characterized by central sensitization and nociplastic pathways—and complex psychosocial yellow flags. Consequently, Primary Care Physicians (PCPs) must act as rigorous gatekeepers against over-medicalization, instead prioritizing active interventions such as patient education, structured reassurance, and tailored exercise therapy. Future strategies must focus on implementation science to seamlessly integrate risk-stratification workflows into daily practice, and on clinical phenotyping to transition from traditional trial-and-error management toward precise, mechanism-based care.

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