Review

Discovery Medicine 2026; 38(208): 1327–1342
https://doi.org/10.24976/Discov.Med.202638208.105

Inflammatory Low Back Pain Across Inflammatory Joint Diseases: Diagnostic Challenges and Evolving Treatment Strategies

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Submitted: 3 March 2026 Revised: 13 April 2026 Accepted: 22 April 2026 Published: 20 May 2026

Inflammatory low back pain (IBP) is a key clinical feature of axial spondyloarthritis (axSpA) and an important diagnostic clue in patients presenting with chronic back pain. However, IBP is neither disease-specific nor uniformly expressed across inflammatory joint diseases, and its recognition in routine clinical practice remains challenging. Significant symptom overlap with mechanical low back pain, heterogeneity of clinical presentation, limited specificity of imaging findings, and frequent misuse of classification criteria as diagnostic tools contribute to persistent diagnostic uncertainty and delay. Despite advances in imaging and therapeutics, several critical challenges remain unresolved, including the limited specificity of IBP constructs, the risk of MRI overinterpretation, and the unclear nosological status of axial psoriatic arthritis (axial PsA). Moreover, variability in treatment response across disease phenotypes highlights the need for more precise diagnostic and therapeutic frameworks. This narrative review provides a structured and critical synthesis of current evidence on IBP across inflammatory joint diseases. It emphasizes key diagnostic pitfalls, including imaging-related challenges and misapplication of classification criteria, and highlights disease-specific differences between axSpA, axial PsA, and rheumatoid arthritis. In addition, it outlines clinically relevant considerations for diagnostic evaluation and therapeutic decision-making. Future research should focus on the development of integrated diagnostic models, improved imaging interpretation strategies, and disease-specific therapeutic approaches to optimize patient outcomes.

Keywords: inflammatory back pain; axial spondyloarthritis; psoriatic arthritis; rheumatoid arthritis; biologic disease-modifying antirheumatic drugs; MRI

Introduction

Low back pain (LBP) is among the leading causes of disability worldwide and constitutes a substantial clinical and socioeconomic burden [1,2]. Although most cases are mechanical or degenerative in origin, a clinically important subset is attributable to inflammatory processes, most commonly in the context of inflammatory joint diseases (IJDs) [3]. Inflammatory low back pain (IBP) represents a cardinal manifestation of axial spondyloarthritis (axSpA) and a key feature for its early recognition; however, it may also occur in psoriatic arthritis (PsA) with axial involvement and, less frequently, in rheumatoid arthritis (RA) [4–6]. Accurate identification of IBP is therefore critical, as it directly influences diagnostic pathways, imaging strategies, therapeutic decision-making, and long-term outcomes [7].

Despite increasing awareness over the past two decades, IBP remains frequently underrecognized in both primary care and specialist settings [8,9]. Clinical overlap with mechanical LBP, heterogeneity of symptom presentation, and reliance on classification rather than diagnostic criteria contribute to substantial diagnostic delay—particularly in axSpA, where delays of several years continue to be commonly reported [10–13]. Classic IBP features, including morning stiffness, improvement with exercise, and nocturnal pain, lack absolute specificity and may be variably present across both inflammatory and non-inflammatory conditions [14,15]. These limitations are further compounded by differences in axial disease expression across IJDs and by ongoing debate regarding whether axial PsA and axSpA represent distinct or overlapping clinical entities [6,16,17].

Advances in imaging, particularly magnetic resonance imaging (MRI), have improved the early detection of inflammatory axial disease [18–22]. However, imaging findings should be interpreted with caution due to limited specificity and the potential for false-positive results [23]. These challenges are discussed in detail in subsequent sections. In parallel, the therapeutic landscape of inflammatory joint diseases has evolved rapidly with the introduction and

widespread use of biologic and targeted synthetic disease-modifying antirheumatic drugs (DMARDs), enabling earlier and more effective control of axial inflammation [24–26]. Nevertheless, treatment response may vary according to disease phenotype, extent of structural damage, and accuracy of the initial diagnosis, further underscoring the importance of precise clinical and imaging assessment [27,28].

Although multiple reviews have addressed inflammatory back pain and axial spondyloarthritis, important gaps remain in the current literature. Many publications focus predominantly on axSpA, with comparatively limited attention devoted to IBP in PsA and RA, particularly with respect to axial PsA as a distinct clinical phenotype [16,29,30]. Furthermore, several reviews adopt a largely descriptive approach, offering limited critical appraisal of diagnostic uncertainty, imaging pitfalls, and the evolving role of classification criteria in real-world clinical practice [31–33]. Recent high-impact publications have emphasized the need to reassess traditional IBP constructs in light of emerging imaging data, evolving disease concepts, and contemporary treatment strategies [34–36].

Against this background, the present narrative review aims to address the following key clinical questions:

• Why is inflammatory back pain frequently misdiagnosed or underrecognized?

• What are the limitations of current classification and imaging criteria?

• How should IBP be managed in the context of evolving targeted therapies?

• How should IBP be approached in clinical practice in the context of future research developments and multidisciplinary care?

Methods

Literature Search Strategy

This narrative review was informed by a structured literature search but did not adhere to formal systematic review methodology. A formal risk-of-bias assessment was not performed, as the aim of this review was to provide a conceptually integrative and clinically oriented synthesis rather than a quantitative evaluation of evidence.

Electronic databases, including PubMed, Scopus, and Web of Science, were searched for articles published between January 2005 and March 2025. The search strategy combined Medical Subject Headings (MeSH) and free-text terms, including “inflammatory back pain”, “axial spondyloarthritis”, “ankylosing spondylitis”, “axial psoriatic arthritis”, “psoriatic arthritis AND spine”, “rheumatoid arthritis AND low back pain”, “sacroiliitis”, “magnetic resonance imaging”, “ASAS criteria”, and “biologic DMARDs”. Boolean operators (AND/OR) were applied to optimize sensitivity and specificity.

Study selection was guided by clinical relevance, methodological robustness, and contribution to current understanding of diagnostic and therapeutic challenges in inflammatory low back pain.

Eligibility Criteria

Publications were eligible for inclusion if they met the following criteria:

(1) Original clinical studies, consensus statements, clinical guidelines, or high-impact narrative or systematic reviews;

(2) Focus on IBP, axial involvement, or low back pain in inflammatory joint diseases (including axial spondyloarthritis, psoriatic arthritis, and rheumatoid arthritis);

(3) Provision of data on epidemiology, pathophysiology, diagnostic approaches, imaging modalities, or therapeutic strategies;

(4) Publication in peer-reviewed journals in English within the predefined timeframe.

Exclusion Criteria

A total of 108 full-text articles were excluded based on the following predefined categories:

• Irrelevant topic (n = 40).

• Inappropriate population or disease focus (n = 27).

• Lack of original clinical data (n = 22).

• Duplicate or overlapping datasets (n = 9).

• Other reasons (non-English language or insufficient methodological clarity) (n = 10).

This classification is reflected in the flowchart (Fig. 1) to improve the transparency of the study selection process.

Importantly, misuse of classification criteria as diagnostic tools in routine clinical practice represents a major source of misclassification and may lead to both overdiagnosis and delayed recognition of true inflammatory disease.

Study Selection and Data Synthesis

Titles and abstracts were screened for relevance, followed by full-text assessment based on predefined inclusion and exclusion criteria. The selection process was performed by two reviewers, with discrepancies resolved through discussion and consensus. Given the heterogeneity of study designs, populations, imaging definitions, and reported outcomes, a narrative thematic synthesis was conducted. Evidence was integrated across key domains, including clinical features and classification of inflammatory back pain, pathophysiological mechanisms, imaging characteristics and limitations, disease-specific patterns across inflammatory joint diseases, and contemporary therapeutic strategies. Where applicable, levels of evidence presented in summary tables were interpreted in accordance with general principles of the GRADE (Grading of Recommendations Assessment, Development and Evaluation) approach, although a formal grading process was not systematically applied. This approach was chosen to provide a clinically meaningful and conceptually coherent synthesis

of the available evidence. The process of literature identification and selection is summarized in Fig. 1.

Definition, Classification and Diagnostic Criteria of Inflammatory Low Back Pain

Inflammatory low back pain (IBP) refers to a constellation of clinical features that reflect inflammatory involvement of the axial skeleton, particularly the sacroiliac joints and spine. IBP is not a diagnosis per se but rather a clinical construct designed to facilitate the early identification of patients with underlying inflammatory joint diseases, most notably axial spondyloarthritis (axSpA) [37–39]. The concept of IBP was introduced to distinguish inflammatory spinal pain from mechanical low back pain, particularly in younger patients presenting with chronic symptoms.

IBP represents a constellation of symptoms suggestive of inflammatory axial disease. However, no single feature or combination reliably distinguishes inflammatory from mechanical pain.

Critically, classification criteria (e.g., ASAS) were developed for research standardization rather than diagnosis. Their application in routine clinical practice without an appropriate clinical context may result in misclassification [37,38,40]. This distinction between classification and diagnosis is essential, as inappropriate reliance on criteria may lead to both overtreatment and missed diagnoses.

Pathophysiological Mechanisms of IBP

Inflammatory low back pain reflects complex interactions between innate and adaptive immune pathways. In axSpA, the interleukin-17 (IL-17)/IL-23 axis and tumor necrosis factor (TNF)-mediated inflammation play central roles, with enthesitis considered a primary lesion driving both pain and structural progression. Mechanical stress at entheses may trigger inflammatory cascades in genetically predisposed individuals [39,41]. In contrast, psoriatic arthritis demonstrates immunological heterogeneity. The lack of efficacy of IL-23 inhibitors in axial disease, despite strong effects in peripheral PsA, supports fundamental biological differences between axial PsA and axSpA.

Clinical Definition and Key Features

Several sets of clinical criteria have been proposed to characterize IBP, including those developed by Calin, Berlin, and the Assessment of SpondyloArthritis International Society (ASAS) [42–44]. Commonly cited features include an insidious onset before the age of 40–45 years, improvement with exercise but not with rest, nocturnal pain with improvement upon rising, and prolonged morning stiffness, particularly in the context of axial inflammatory disease [42–45]. Among these features, morning stiffness lasting longer than 30–60 minutes has traditionally been regarded as supportive of inflammatory pain; however, its diagnostic specificity remains limited [46].

Importantly, no single symptom or combination of symptoms reliably distinguishes IBP from mechanical low back pain in all clinical settings. Studies have shown that individual IBP features may also be reported by patients with degenerative spine disease, fibromyalgia, or nonspecific chronic low back pain [15,47]. Consequently, IBP should be interpreted within a broader clinical context rather than as a stand-alone diagnostic criterion.

Classification Criteria and Their Limitations

The ASAS classification criteria for axial spondyloarthritis, developed to facilitate early disease recognition, incorporate IBP as a key entry feature in patients with chronic back pain lasting more than three months and with symptom onset before the age of 45 years [42,44]. These criteria integrate clinical features, imaging findings, and laboratory markers, including HLA-B27 status.

Although the ASAS criteria have improved sensitivity for early axSpA, they were designed for classification rather than diagnosis—a distinction that is frequently overlooked in clinical practice [15,48,49]. Application of these criteria outside their intended research context may contribute to misclassification, particularly in populations with low disease prevalence or among individuals with nonspecific MRI findings. This limitation is especially relevant in patients with overlapping features of axial PsA or in those with chronic mechanical stress affecting the sacroiliac joints [20,21].

Evolution of IBP Criteria

The first accepted criteria for IBP were the Calin Criteria (1977), which were characterized by high sensitivity (>90%), but low specificity. They focused on 5 key symptoms: (1) insidious onset; (2) age at onset <40 years; (3) duration of back pain ≥3 mo; (4) associated with morning stiffness; and (5) improvement with exercise, with 4 of the five criteria considered mandatory for IBP.

The Berlin Criteria (2006) better define IBP and have 81% specificity and 70% sensitivity, combining five key clinical parameters—age, insidious onset, nocturnal pain, improvement with exercise, and lack of relief from rest.

In 2009, the Assessment of SpondyloArthritis International Society (ASAS) introduced new ASAS criteria for IBP, which have 77.0% sensitivity and 91.7% specificity when at least four of five parameters are present: age of onset, <40 years, insidious onset, improvement with exercise, no improvement with rest, and pain at night. The ASAS criteria integrate imaging, but carry the risk of misuse [50].

Critical issue: Classification criteria ≠ diagnostic criteria. Misapplication leads to overdiagnosis and inappropriate biologic use.

Clinical Issues in Diagnosing IBP

Inflammatory low back pain remains frequently underrecognized due to its clinical overlap with mechanical

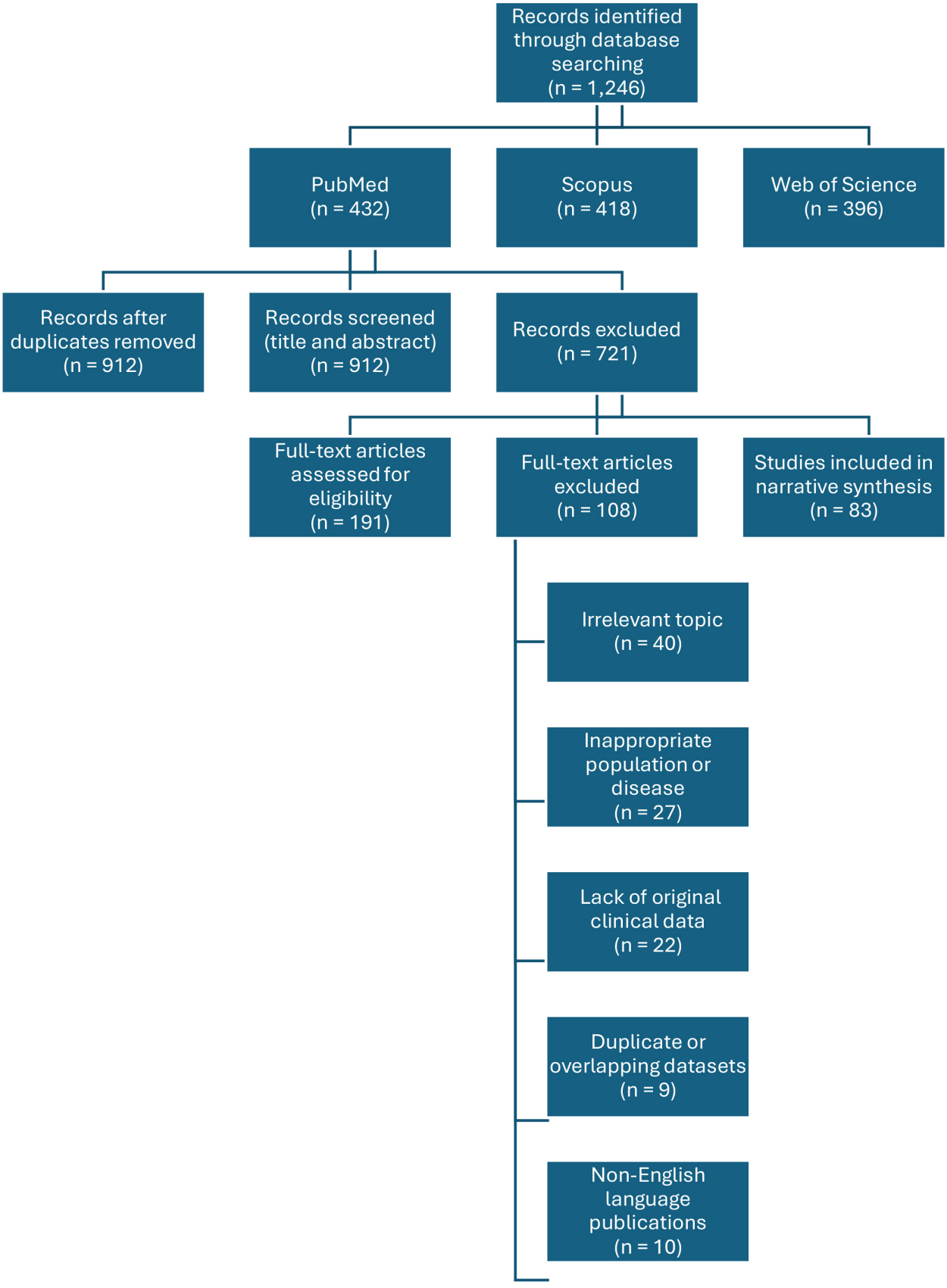

Fig. 1. Literature selection process (narrative review).

low back pain, the absence of specific biomarkers, and the variability of symptom presentation. Correct identification requires timely referral to a rheumatologist to identify or rule out a systemic autoimmune problem [6]. In addition, inappropriate use of imaging in unselected populations contributes to diagnostic uncertainty. Recent evidence in the literature suggests that imaging is only useful in a limited number of patients with certain conditions, including cancer, infection, inflammatory disease, fracture, and severe neurological deficits, which account for only 5–10%. In the remaining 90–95% of cases of LBP (called nonspecific or uncomplicated LBP), it may do more harm than good [51]. While imaging is essential in selected cases, its indiscriminate use may lead to overdiagnosis, increased healthcare costs, and unnecessary exposure to radiation. Current recommendations emphasize that imaging should be guided by clinical suspicion and the presence of red flags rather than used as a screening tool in nonspecific low back pain. IBP should be treated differently depending on the underlying disease. Non-pharmacological therapy includes exercise and patient education. The preparation of a patient's therapeutic plan includes consideration of the individual and general condition of each patient, taking into account factors such as age, gender, the presence of concomitant diseases, drug interactions, the patient's socioeconomic status, as well as the presence of certain clinical findings, such as the predominance of axial or peripheral symptoms and enthesal and extra-articular manifestations [21,52,53].

Magnetic Resonance Imaging: Advantages and Limitations

Magnetic resonance imaging (MRI) of the sacroiliac joints and spine enables visualization of active inflammatory lesions, including bone marrow edema (BME), synovitis, capsulitis, and enthesitis, before the development of structural damage detectable on conventional radiography [18,44]. The incorporation of MRI findings into the ASAS classification criteria has significantly improved sensitivity for early axSpA, particularly in patients without radiographic sacroiliitis [44,54,55].

Furthermore, variability in MRI acquisition protocols, scoring systems, and reader expertise contributes to interobserver variability and diagnostic uncertainty [56]. Although structural lesions such as erosions, fat metaplasia, and ankylosis increase diagnostic confidence, their absence does not exclude early inflammatory disease, particularly in younger patients or those with a short duration of symptoms [57]. Overreliance on MRI may lead to overdiagnosis and inappropriate initiation of biologic therapy. Conversely, patients with clinical IBP but negative MRI require longitudinal assessment rather than premature exclusion of inflammatory disease.

MRI Specificity and ASAS Lesion Definitions

The Assessment of SpondyloArthritis International Society (ASAS) has established standardized definitions for MRI lesions suggestive of sacroiliitis, with bone marrow edema serving as the key criterion for active inflammation [58–61]. These definitions have improved diagnostic consistency and facilitated research standardization. Nevertheless, the specificity of MRI findings remains a critical issue. Bone marrow edema is not pathognomonic for inflammatory disease and may be observed in a variety of non-inflammatory conditions, including mechanical stress, degenerative changes, postpartum status, and athletic activity [62]. Importantly, the specificity of MRI findings increases when bone marrow edema is deep, extensive, or present on multiple consecutive slices, particularly when accompanied by structural lesions such as erosions or fat metaplasia.

MRI Pitfalls: False Positives and the Need for Contextual Interpretation

A major challenge in the use of MRI in IBP is the risk of false-positive findings. Several studies have demonstrated that MRI lesions resembling sacroiliitis can be present in healthy individuals, particularly in physically active populations. Similarly, degenerative changes and biomechanical stress may produce imaging patterns that mimic inflammatory lesions.

Therefore, MRI findings should not be interpreted in isolation. A contextual approach integrating clinical presentation, laboratory findings, and patient characteristics is essential to avoid overdiagnosis and misclassification. This is particularly relevant in populations with low pretest probability of inflammatory disease, where the positive predictive value of MRI findings is reduced.

MRI-Negative IBP: Diagnostic and Management Considerations

A subset of patients with clinically suggestive IBP may have negative MRI findings, particularly in early disease stages. This scenario presents a diagnostic challenge and underscores the limitations of relying solely on imaging criteria. In such cases, longitudinal assessment, repeated imaging, and close clinical follow-up are recommended. Additional factors, including HLA-B27 status and response to nonsteroidal anti-inflammatory drugs (NSAIDs), may support clinical decision-making. Patients with a negative MRI result generally have a lower likelihood of progression to structural damage compared to those with a positive MRI result. Therefore, early detection of positive inflammation on MRI allows for early therapeutic intervention that can significantly reduce or prevent progression to irreversible structural damage [63–66].

Radiography and Computed Tomography

Conventional radiography remains useful for detecting established structural changes, such as erosions, scle-

Table 1. Imaging characteristics of axial spondyloarthritis and axial psoriatic arthritis.

Feature	Axial spondyloarthritis (axSpA)	Axial psoriatic arthritis (axial PsA)
Sacroiliitis pattern	Commonly bilateral (~70–80%) and relatively symmetric	Often asymmetric (~40–60%)
MRI bone marrow edema	Common (~60–80% in early disease); typically extensive	Variable (~30–60%); often less typical or patchy
Structural changes (syndesmophytes)	Thin, marginal, vertically oriented	Bulky, non-marginal, often asymmetric
HLA-B27 association	High (~70–90%); strong association	Lower (~20–50%), weaker association
Clinical expression of IBP	Typical and more homogeneous	Heterogeneous; may overlap with mechanical features

rosis, and ankylosis of the sacroiliac joints, particularly in longstanding disease [67]. However, radiographic changes often lag behind clinical symptoms by several years, which limits their utility in early diagnosis.

Computed tomography (CT) provides superior visualization of structural lesions compared with conventional radiography and may be helpful in selected cases with equivocal findings [68]. Nevertheless, the use of CT is constrained by radiation exposure and its inability to detect active inflammation, restricting its role primarily to problem-solving rather than routine assessment.

Imaging Controversies in Axial Psoriatic Arthritis

The role of imaging is particularly complex in patients with suspected axial psoriatic arthritis (axial PsA). Although axial involvement is increasingly recognized in PsA, its imaging features may differ from those observed in axSpA, thereby complicating classification and diagnosis [6,16,17]. Axial PsA often demonstrates asymmetric sacroiliitis, less pronounced or atypical syndesmophytes, and a lower frequency of HLA-B27 positivity (approximately 19–49%) compared with axSpA (typically 75–90%) [6,16]. MRI findings in axial PsA may overlap with those seen in both axSpA and degenerative spinal disease, increasing the risk of misclassification when ASAS imaging criteria are applied indiscriminately [69]. These differences have fueled ongoing debate regarding whether axial PsA represents a distinct clinical entity or a phenotypic variant within the broader spondyloarthritis spectrum.

Clinical Implications of Imaging Interpretation

In the era of early biologic intervention, imaging findings carry important therapeutic implications. Overinterpretation of nonspecific MRI lesions may lead to inappropriate initiation of long-term immunomodulatory therapy, whereas underrecognition of true inflammatory changes may delay effective treatment and contribute to structural progression [23,27].

Current recommendations emphasize that imaging findings should be interpreted as part of an integrated diagnostic approach, incorporating clinical features of IBP, laboratory markers, and disease-specific characteristics [47]. Imaging should support, rather than replace, clinical judgment (Table 1).

IBP Beyond Axial Spondyloarthritis

Although IBP is most strongly associated with axSpA, axial symptoms with inflammatory characteristics have also been described in psoriatic arthritis and, less commonly, in rheumatoid arthritis [5,6,16]. In PsA, axial involvement may present with clinical and imaging features that partially overlap with those of axSpA, yet differ with respect to sacroiliac joint symmetry, syndesmophyte morphology, and HLA-B27 association [6,16,17]. These differences challenge the universal applicability of traditional IBP constructs and underscore the need for disease-specific interpretation.

In rheumatoid arthritis, inflammatory low back pain is uncommon and typically reflects secondary mechanisms, such as cervical or lumbar spine involvement in longstanding disease, rather than primary sacroiliitis [6]. Nevertheless, its presence may further complicate the differential diagnosis, particularly in patients with coexisting degenerative spinal changes.

Taken together, these considerations underscore that IBP should be regarded as a screening and contextual clinical concept rather than a definitive diagnostic entity. Accurate assessment requires careful integration of symptom characteristics, physical examination findings, imaging results, and laboratory markers, all interpreted within the broader clinical phenotype. Overreliance on simplified criteria or isolated imaging findings carries a risk of inappropriate classification and may have significant therapeutic implications in the era of early targeted treatment (Table 2).

Disease-Specific Patterns of Inflammatory Low Back Pain in Inflammatory Joint Diseases

Although inflammatory low back pain (IBP) is most closely associated with axial spondyloarthritis (axSpA), axial symptoms with inflammatory characteristics may also occur in other inflammatory joint diseases, particularly psoriatic arthritis (PsA) and, less commonly, rheumatoid arthritis (RA). In axial PsA, symptoms are more heterogeneous, often overlapping with mechanical pain. These differences challenge the assumption that axial PsA represents simply a variant of axSpA. Recognition of disease-specific patterns

Table 2. Key differences between inflammatory and mechanical low back pain.

Feature	Inflammatory low back pain	Mechanical low back pain
Typical age at onset	Typically <40–45 years	Any age, more common >40 years
Onset	Insidious	Often acute or activity-related
Morning stiffness	Common (>30–60 min; sensitivity ~70–80%, specificity ~50–60%)	Mild or short-lived (<30 min)
Effect of exercise	Improvement Typical (~70–80%)	Often worsens
Effect of rest	No improvement	Improvement
Nocturnal pain	Common but not specific Frequent (~60–70%), improves on rising	Uncommon (<20%)
Inflammatory markers	May be elevated (~40–60%)	Typically normal
MRI findings	Bone marrow edema with/without structural lesions; interpretation requires clinical context	Degenerative changes (disc disease, osteophytes)

Note: Findings should be interpreted in a clinical context.

is essential, as the clinical significance, imaging correlates, and therapeutic implications of IBP differ across these conditions.

Axial Spondyloarthritis

Axial spondyloarthritis represents the prototypical disease in which IBP is a defining clinical feature. Chronic back pain with inflammatory characteristics often constitutes the initial manifestation, preceding radiographic changes by several years [10–12]. In axSpA, IBP is typically associated with symptom onset before the age of 45 years, prolonged morning stiffness, nocturnal pain, and improvement with physical activity [42–44].

In axSpA, IBP reflects active inflammation of the sacroiliac joints and spine and correlates variably with objective markers of disease activity, such as C-reactive protein levels and MRI-detected inflammation [18,42]. Diagnostic delay remains a major challenge, particularly in patients without radiographic sacroiliitis, contributing to delayed initiation of effective therapy and an increased risk of structural progression [10,11].

In axSpA, IBP is primarily driven by inflammation at the enthesis—the interface between tendon/ligament and bone. Mechanical stress at entheseal sites induces microdamage, which in genetically predisposed individuals (notably HLA-B27 carriers) triggers innate immune activation [70,71].

Key molecular pathways include:

• IL-17/TNF axis: Central to disease pathogenesis, promoting recruitment of neutrophils and macrophages and sustaining chronic inflammation [72].

• Enthesitis-driven inflammation: Considered the primary lesion, preceding synovitis and structural damage [73].

• Bone remodeling imbalance: Coupling of inflammation with aberrant repair leads to new bone formation (syndesmophytes), mediated by pathways such as Wnt signaling and bone morphogenetic proteins (BMPs) [74].

This mechanistic framework explains the classical IBP phenotype (morning stiffness, improvement with exercise) and the strong response to TNF and IL-17 inhibitors.

Axial Psoriatic Arthritis (Axial PsA)

Axial involvement in psoriatic arthritis is increasingly recognized but remains heterogeneous and incompletely defined [6,16,17]. IBP may be present in patients with axial PsA; however, its clinical expression often differs from that observed in axSpA. Back pain may be less stereotypical, morning stiffness may be shorter or less prominent, and symptoms may coexist with mechanical or degenerative spinal pain [16,69].

Axial involvement in PsA represents a more heterogeneous entity with overlapping but distinct mechanisms compared to axSpA. Although IL-17 remains relevant, emerging evidence suggests a more complex immunological profile:

• IL-23 pathway: Plays a dominant role in peripheral PsA, but its limited role in axial disease is reflected by the lack of efficacy of IL-23 inhibitors in axial manifestations [75].

• Tissue-specific immune responses: Differences in cytokine gradients and cellular infiltration patterns between skin, synovium, and enthesis [76].

• Structural heterogeneity: More variable bone remodeling patterns, including both erosive and proliferative changes [77].

Clinically, this translates into less typical IBP features, more frequent asymmetry, and weaker correlation between symptoms and imaging findings.

Imaging features further complicate the recognition of IBP in axial PsA. Asymmetric sacroiliitis, atypical syndesmophytes, and frequent overlap with degenerative changes may limit the applicability of traditional IBP constructs and ASAS classification criteria [6,17]. These differences support the concept that axial PsA may represent

a distinct clinical phenotype rather than a simple variant of axSpA, with important implications for diagnosis and treatment selection.

Rheumatoid Arthritis

In contrast to spondyloarthritis, inflammatory low back pain is uncommon in rheumatoid arthritis and is not considered a characteristic feature of the disease [6]. When present, low back pain is typically mechanical or degenerative. Pathophysiologically:

• Synovial-driven inflammation predominates rather than enthesitis [78].

• Cytokine profile is dominated by TNF, IL-6, and other mediators, without the same enthesis-centered mechanisms seen in axSpA [79].

• Sacroiliac joint involvement is rare, and MRI findings usually reflect degenerative rather than inflammatory changes [80].

When inflammatory spinal symptoms occur in RA, they typically reflect secondary mechanisms, such as cervical spine involvement, atlantoaxial subluxation, or inflammatory changes affecting the lumbar spine in longstanding disease [6,81].

Low back pain in RA is more frequently attributable to degenerative changes, osteoporosis-related fractures, or comorbid mechanical conditions, which may coexist with systemic inflammation [82]. Consequently, the presence of IBP-like symptoms in RA warrants careful evaluation to exclude alternative diagnoses, including concomitant spondyloarthritis or non-inflammatory spinal pathology.

Comparative Clinical Implications

These disease-specific mechanisms highlight that IBP should not be interpreted as a uniform construct across inflammatory joint diseases.

While IBP is central to the diagnosis and management of axSpA, its significance in PsA and RA is more variable and context dependent:

• In axSpA, IBP strongly reflects enthesitis-driven inflammation.

• In axial PsA, IBP is less specific and may not reliably indicate axial inflammatory activity [83].

• In RA, IBP-like symptoms are more likely to represent non-inflammatory pathology.

Failure to recognize these distinctions may lead to inappropriate application of classification criteria, misinterpretation of imaging findings, and suboptimal therapeutic decision-making (Table 3).

Therapeutic Strategies for Inflammatory Low Back Pain: Current Options and Unmet Needs

Management of inflammatory low back pain (IBP) has evolved substantially over the past two decades, in parallel with advances in the understanding of inflammatory joint diseases and the expanding availability of targeted therapies. Nevertheless, effective treatment of IBP remains closely dependent on accurate diagnosis, appropriate phenotypic classification, and timely intervention.

NSAIDs remain first-line therapy. Biologic agents, particularly TNF and IL-17 inhibitors, are highly effective in axSpA.

A key translational insight is the divergent response to IL-23 inhibition. While IL-23 inhibitors are effective in peripheral PsA, they have failed to demonstrate efficacy in axial disease. This discrepancy provides indirect evidence that axial inflammation in axSpA is not primarily IL-23-driven, further supporting disease heterogeneity.

Fig. 2 illustrates an integrated, phenotype-driven treatment decision-making pathway in inflammatory back pain, highlighting the role of disease heterogeneity and differential response to biologic therapies in guiding clinical management.

Treatment should follow a stepwise approach:

• Initial: NSAIDs and exercise.

• Escalation: biologic therapy.

• Refractory: switching or JAK inhibitors (Fig. 2).

The figure illustrates a stepwise therapeutic approach, beginning with initial management (NSAIDs and non-pharmacological interventions), followed by escalation to biologic therapies (TNF and IL-17 inhibitors) in patients with persistent disease activity and according to the disease phenotype. Subsequent steps include switching strategies and consideration of targeted synthetic DMARDs in cases of inadequate response.

Importantly, treatment decisions are guided by integrated clinical evaluation, imaging findings (particularly MRI), and disease phenotype, highlighting the need for a multidimensional approach in the management of inflammatory back pain.

Nonsteroidal Anti-Inflammatory Drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) remain the recommended first-line pharmacological therapy for IBP, particularly in axial spondyloarthritis (axSpA) [84,85]. NSAIDs provide symptomatic relief and may reduce inflammatory activity, with some evidence suggesting a potential effect on radiographic progression when used continuously in selected patients [86]. However, response to NSAIDs is heterogeneous, and a substantial proportion of patients experience incomplete symptom control or intolerance due to gastrointestinal, cardiovascular, or renal adverse effects [87].

Importantly, NSAID responsiveness, once considered a supportive feature of IBP, lacks sufficient specificity to serve as a diagnostic discriminator, as improvement may also be observed in mechanical low back pain [88]. This further underscores the need to interpret therapeutic response within a broader clinical and imaging context.

Table 3. Characteristics of inflammatory low back pain across inflammatory joint diseases.

Feature	Axial spondyloarthritis (axSpA)	Axial psoriatic arthritis (axial PsA)	Rheumatoid arthritis (RA)
Clinical IBP phenotype	Typical; high pre-test probability	Variable; lower specificity	Rare; usually non-inflammatory or degenerative
MRI pattern	Characteristic sacroiliitis with BME ± structural lesions	Atypical or mixed patterns; possible overlap with degenerative changes	Predominantly degenerative changes; inflammatory sacroiliitis uncommon
Imaging–clinical concordance	Moderate to high when criteria are met	Variable; often weaker correlation	Low; imaging findings rarely explain IBP phenotype
Response to targeted therapy	Robust response to TNF and IL-17 inhibitors	Variable response; domain-specific	Axial symptoms not a primary therapeutic target
Interpretative pitfalls	Overreliance on MRI in low-prevalence settings	Misclassification as axSpA	Misattribution of mechanical pain as inflammatory

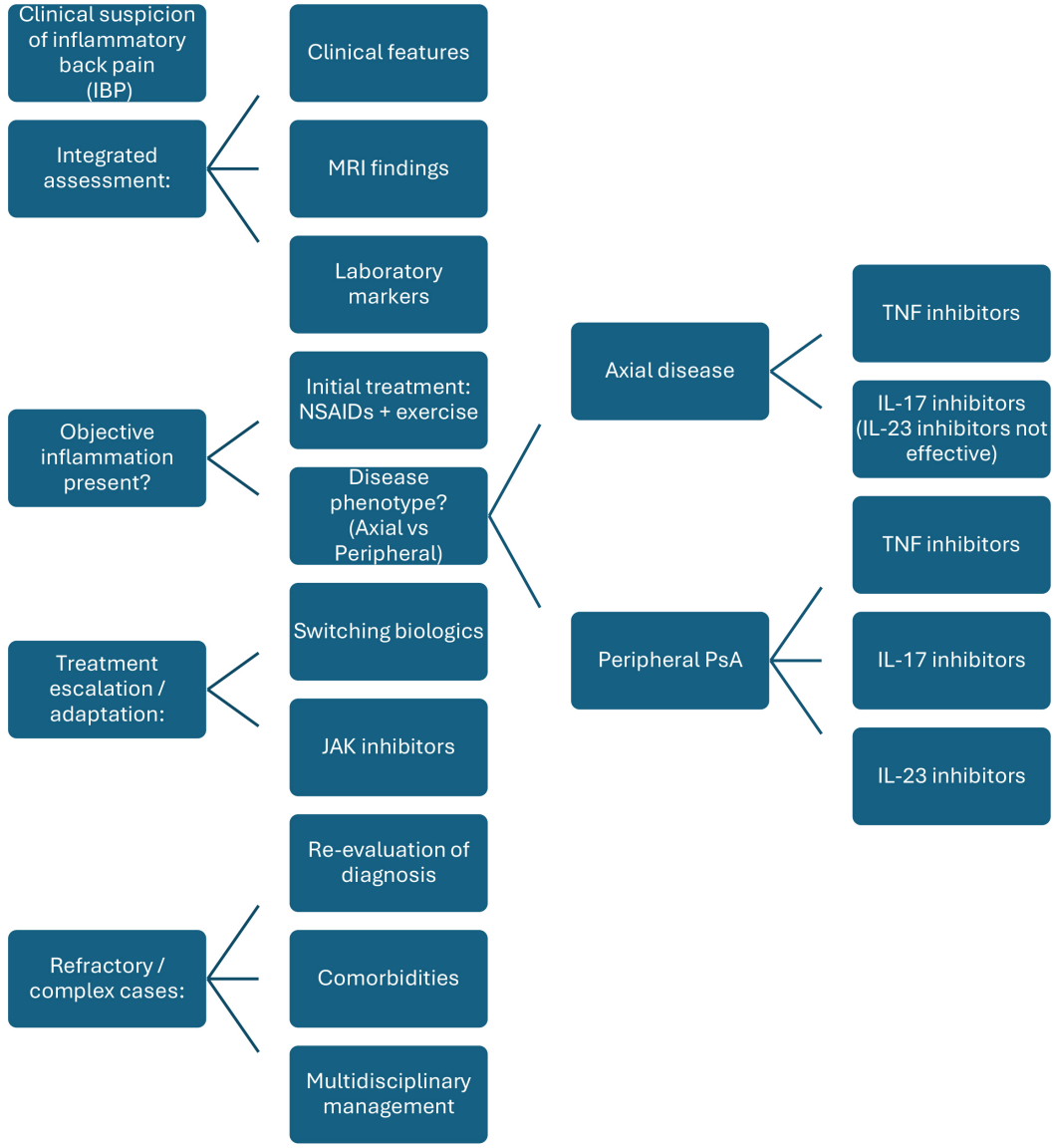

Fig. 2. Integrated treatment decision-making pathway in inflammatory back pain across inflammatory joint diseases.

Biologic and Targeted Synthetic DMARDs

The introduction of biologic disease-modifying antirheumatic drugs (bDMARDs), particularly tumor necrosis factor (TNF) inhibitors and interleukin-17 (IL-17) inhibitors, has transformed the management of IBP associated with axSpA [24–26]. These agents have demonstrated robust efficacy in reducing spinal inflammation, improving physical function, and enhancing quality of life in both radiographic and non-radiographic axSpA [26,89].

A key translational insight is the divergent response to IL-23 inhibition: while IL-23 inhibitors are effective in peripheral psoriatic disease, they have consistently failed to demonstrate efficacy in axial disease. This discrepancy supports the concept that axial inflammation is not primarily IL-23-driven and further highlights fundamental differences between axial spondyloarthritis and axial psoriatic arthritis [90,91].

Targeted synthetic DMARDs, such as Janus kinase (JAK) inhibitors, have further expanded therapeutic options, although long-term data on structural outcomes and safety in axial disease continue to evolve [92]. Treatment selection increasingly reflects individual disease characteristics, prior treatment response, comorbidities, and patient preferences.

Therapeutic Considerations in Axial Psoriatic Arthritis

Evidence guiding the treatment of IBP in axial psoriatic arthritis remains more limited than that available for axSpA. While TNF and IL-17 inhibitors are commonly used and may be effective in reducing axial symptoms, dedicated clinical trials focusing specifically on axial PsA remain scarce [93,94]. As a result, treatment strategies are often extrapolated from axSpA data, despite potential differences in underlying pathophysiology and imaging phenotypes.

This evidence gap highlights an unmet need for disease-specific studies to inform the optimal management of axial involvement in PsA and to clarify whether therapeutic responses differ meaningfully from those observed in axSpA.

Limited Role of Conventional Synthetic DMARDs and Glucocorticoids

Conventional synthetic DMARDs, such as methotrexate and sulfasalazine, have limited efficacy in axial disease and are not recommended for the treatment of IBP in axSpA [84]. Similarly, systemic glucocorticoids provide minimal and short-lived benefit in axial inflammation and are generally discouraged because of their unfavorable risk–benefit profile [95].

Local glucocorticoid injections may offer temporary symptom relief in selected cases, particularly for peripheral manifestations, but do not address underlying axial inflammation.

Non-Pharmacological and Multidisciplinary Approaches

Non-pharmacological interventions, including structured exercise programs, physical therapy, and patient education, constitute essential components of IBP management across inflammatory joint diseases [96]. Regular physical activity improves mobility, reduces pain, and complements pharmacological treatment.

Given the chronic nature of IBP and its impact on physical function, mental health, and work productivity, a multidisciplinary approach involving rheumatologists, physiotherapists, and, when appropriate, psychologists and pain specialists is increasingly recognized as best practice (Table 4).

Evidence levels (strong, moderate, limited) reflect a qualitative synthesis of available clinical trial data and guideline recommendations, interpreted in alignment with general GRADE principles (Table 5).

The elements summarized in Table 5 provide the conceptual and evidence-based framework underlying the phenotype-driven treatment decision-making pathway illustrated in Fig. 2, particularly in relation to disease heterogeneity, diagnostic uncertainty, and differential therapeutic response.

Multidisciplinary Care and Future Perspectives

Inflammatory low back pain (IBP) is a complex clinical manifestation that extends beyond pain control and requires a comprehensive, patient-centered management approach. Given its chronic course, fluctuating activity, and impact on physical function and quality of life, optimal care for patients with IBP should integrate pharmacological treatment with non-pharmacological and supportive interventions [96].

Multidisciplinary care models involving rheumatologists, physiotherapists, specialist nurses, and, when appropriate, psychologists and pain specialists have demonstrated benefits in improving functional outcomes, treatment adherence, and patient satisfaction in chronic inflammatory joint diseases [97,98]. Structured exercise programs and individualized rehabilitation strategies are particularly important in axial disease, where maintenance of spinal mobility and posture is critical to long-term outcomes [96].

Patient education and shared decision-making play a central role in the management of IBP [99]. Improved understanding of disease mechanisms, treatment goals, and the importance of sustained physical activity may enhance adherence and empower patients to actively participate in their care [100]. In addition, early referral pathways and improved collaboration between primary care physicians and rheumatologists may help reduce diagnostic delay, a persistent challenge in patients with axial inflammatory disease [10,11].

Table 4. Therapeutic strategies for inflammatory low back pain and current evidence gaps.

Therapeutic approach	Evidence in axSpA	Evidence in axial PsA	Interpretation/Level of recommendation
NSAIDs	Recommended first-line; well-established efficacy	Recommended First-line	Standard initial management
TNF inhibitors	Highly effective; preferred	Effective	Preferred option in active disease
IL-17 inhibitors	Highly effective; preferred	Effective	Preferred alternative to TNF inhibitors
IL-23 inhibitors	Not recommended for axial disease	Effective for peripheral disease; limited axial benefit	Not routinely recommended for axial involvement
JAK inhibitors	Emerging option with growing evidence	Limited but evolving data	Conditional use in selected patients
csDMARDs	Ineffective for axial disease Not recommended (axial)	May be used for peripheral manifestations only	Restricted indication (non-axial disease)

Table 5. Summary of IBP across inflammatory joint diseases—limitation, contrast, clinical significance.

Domain	Key points
Limitations	- Overlap between inflammatory and non-inflammatory back pain - Limited specificity of MRI findings in early disease - Variability in clinical presentation across diseases
Disease differences	- Axial vs peripheral dominance across SpA spectrum - Differential response to biologics (e.g., IL-23 effective in PsA but not axial SpA) - Heterogeneity in imaging patterns
Clinical significance	- Need for integrated clinical–imaging interpretation - Risk of misclassification if relying on single modality - Importance of early and accurate diagnosis
Treatment evidence gaps	- Limited data for axial PsA - Unclear positioning of JAK inhibitors in IBP - Lack of head-to-head trials - Insufficient long-term outcome data

Looking forward, several areas warrant further investigation. These include the development of more specific clinical and imaging biomarkers for IBP, refinement of classification criteria to better reflect real-world disease heterogeneity, and the need for dedicated clinical trials focusing on axial psoriatic arthritis [27,97]. Advances in imaging techniques, including quantitative MRI and artificial intelligence–assisted interpretation, may improve diagnostic accuracy but will require careful validation to avoid exacerbating current challenges related to overdiagnosis.

Unmet Needs and Future Directions

Despite significant therapeutic advances, several unmet needs persist. These include limited availability of biomarkers for predicting treatment response, uncertainty regarding optimal sequencing and switching of biologic therapies, and insufficient evidence to guide disease-specific management of axial PsA and IBP outside the axSpA spectrum [27,97]. Furthermore, early and accurate identification of patients most likely to benefit from targeted therapy remains a central challenge- Qualitative synthesis based on consistency of evidence across RCTs and guidelines; not formal GRADE

Future research should prioritize prospective, disease-specific studies integrating clinical phenotypes, imaging patterns, and treatment response to better define inflammatory low back pain beyond the axSpA paradigm [101].

Following the above findings, the following recommendations for clinical practice can be derived:

• Keep in mind that IBP is a screening construct, not a diagnosis.

• MRI findings should be interpreted clinically.

• Avoid abuse of classification criteria.

• Take into account disease-specific differences.

• Re-evaluate MRI-negative patients.

Limitations

This review has several limitations that should be acknowledged. First, it was conducted as a narrative review rather than a formal systematic review; therefore, the literature search, although structured, may not have captured all

relevant studies. Second, no formal risk-of-bias assessment was performed, which limits the ability to quantitatively appraise the quality of included evidence. Third, study selection was based on expert consensus, introducing a potential risk of selection bias.

Additionally, heterogeneity across included studies—in terms of design, populations, imaging definitions, and outcome measures—limits direct comparability and precludes meta-analytic synthesis. The interpretation of MRI findings, in particular, is influenced by evolving definitions and variability in reader expertise, which may affect generalizability.

Finally, the rapidly evolving therapeutic landscape, especially regarding targeted therapies (e.g., JAK inhibitors and IL-23 pathway agents), means that some conclusions may require future reassessment as new evidence emerges.

Conclusion

Inflammatory low back pain remains a pivotal yet clinically challenging manifestation across inflammatory joint diseases. While it is most strongly associated with axial spondyloarthritis, IBP may also occur in psoriatic arthritis and, less commonly, in rheumatoid arthritis, with important differences in clinical presentation, imaging characteristics, and therapeutic implications. IBP remains a clinically useful but imperfect construct. Diagnostic accuracy depends on integrating clinical, imaging, and biological data.

Despite substantial advances in imaging and targeted therapies, accurate recognition of IBP continues to be hindered by symptom overlap with mechanical low back pain, limited specificity of imaging findings, and inappropriate application of classification criteria in routine clinical practice. These challenges are particularly consequential in the era of early biologic intervention, where both overdiagnosis and delayed diagnosis may adversely affect long-term patient outcomes.

This narrative review underscores the need for an integrated, disease-aware diagnostic approach that combines careful clinical assessment, judicious interpretation of imaging, and consideration of disease-specific patterns and real-world clinical heterogeneity. Multidisciplinary, patient-centered management strategies, together with ongoing research aimed at refining diagnostic tools and developing tailored therapeutic approaches, are essential to improving outcomes for patients with inflammatory low back pain across the spectrum of inflammatory joint diseases.

This integrated approach is reflected in the decision-making pathway illustrated in Fig. 2, where clinical assessment, imaging interpretation, and therapeutic choices are closely interconnected. An ideal multidisciplinary workflow in the evaluation of inflammatory back pain involves a structured and iterative collaboration between specialties. Rheumatologists lead the clinical assessment and diagnostic synthesis, while radiologists provide standardized and context-aware interpretation of imaging findings, particularly MRI. Physiotherapists contribute to functional evaluation and help differentiate inflammatory from mechanical pain patterns, while additional specialists may be involved based on comorbidities. Importantly, this process is not linear but requires continuous feedback between clinical findings and imaging interpretation, particularly in cases of diagnostic uncertainty.

Future research should prioritize biomarkers, improved imaging specificity, and disease-specific treatment strategies. In the coming years, axial psoriatic arthritis (axPsA) research is expected to prioritize the development of biomarkers (e.g., matrix metalloproteinase-3, synovial CD3+ cells) to predict treatment response and refinement of MRI to differentiate axPsA from other spondyloarthritis, along with dedicated studies of axial-specific therapies for PsA involvement. The future use of artificial intelligence (AI) and machine learning (ML) for automated analysis of radiographs and MRI could have increasing potential for improving diagnostics and monitoring of treatment. On the other hand, collaboration between rheumatologists, radiologists and rehabilitation specialists is an important part, especially essential for the holistic treatment of patients with chronic autoimmune diseases.

Availability of Data and Materials

The datasets used or analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions

Conceptualization, formal analysis, editing, writing the first draft, supervision: DT; Methodology, investigation, visualization, writing: MGP. SS and SPB have collected and analyzed data and have been involved in drafting the manuscript and all authors have been involved in revising it critically for important intellectual content. The authors read and approved the final manuscript. The authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

Acknowledgment

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

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