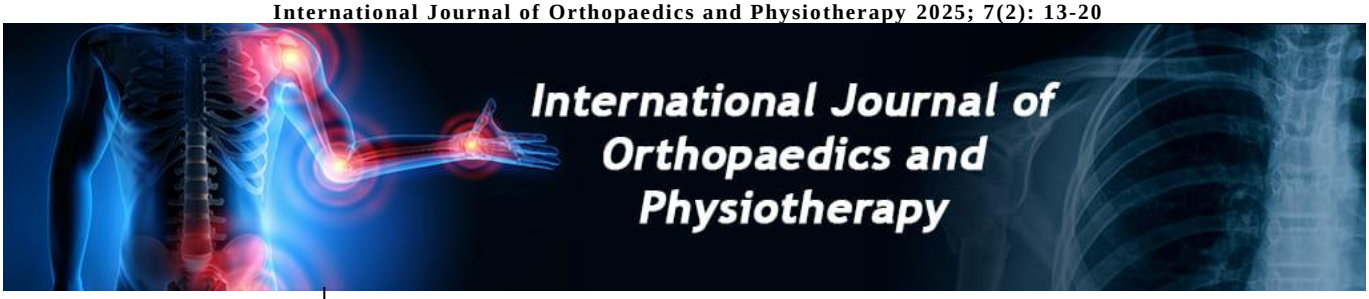

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Hip and knee osteoarthritis: Differential pain phenotypes and evidence based management strategies for orthopedic surgeons

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Abstract
Osteoarthritis (OA) of the hip and knee represents a major cause of chronic musculoskeletal pain and functional limitation worldwide. Despite shared degenerative mechanisms, these two joint disorders differ significantly in their pathophysiology, pain phenotypes, neurophysiological processes, biomechanical alterations, and therapeutic responses. This article provided a comprehensive analysis of these differences, emphasizing the implications for personalized pain management. Knee OA often presents with greater central sensitization and psychosocial comorbidity, requiring multifactorial treatment strategies, whereas hip OA demonstrates a more linear relationship between structural damage and pain. The discussion also highlighted the relevance of advanced neuroimaging, phenotypic pain classification, and targeted interventional techniques such as genicular nerve radiofrequency and robotic-assisted arthroplasty. Conservative approaches, including tailored exercise programs and weight management, remain central to care, while pharmacological and surgical interventions must be adapted to each patient's unique clinical profile. A structured, evidence-informed framework is proposed to support clinical decision-making in pain medicine, aiming to enhance outcomes and quality of life for OA patients.

Keywords: Osteoarthritis-related pain, pain phenotyping, neurophysiological mechanisms, hip and knee joint disorders, personalized therapeutic strategies

Introduction
Although hip and knee osteoarthritis (OA) are frequently grouped under a unified clinical framework, this common approach fails to reflect fundamental differences in their biomechanical behavior, symptom profiles, and treatment responses. In clinical practice, scientific literature, and among many pain specialists, there is a persistent assumption that these conditions share similar progression and therapeutic thresholds. However, equating them may obscure important distinctions and delay appropriate interventions particularly in cases of hip OA, which often requires earlier surgical consideration.

The objective of this article is to clarify the distinct clinical, pathophysiological, and therapeutic features of hip and knee OA, with special emphasis on their differential pain phenotypes. By synthesizing recent evidence, we aim to propose a multidimensional, phenotype-informed framework that can better guide individualized clinical decision-making in pain medicine (Hall *et al.*, 2019; Lespasio *et al.*, 2017) [16, 21].

Knee OA typically evolves in a way that allows for prolonged conservative treatment. Interventions such as physical therapy, weight loss, and pharmacological management can offer sustained symptom relief, often postponing the need for surgery. In contrast, hip OA is usually more structurally and functionally limiting in its early stages. Hip replacement surgery is often indicated earlier and is not constrained by patient age. The decision for arthroplasty is based on three key clinical factors: the severity of pain, degree of joint mobility restriction, and impairment of walking ability.

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The anatomical and biomechanical roles of these joints also diverge substantially. The hip, as a central axial joint, is crucial for maintaining spinopelvic balance and coordinating lower limb mechanics. Its dysfunction affects the entire kinetic chain and can lead to referred pain or biomechanical stress in adjacent regions, including the lumbar spine and ipsilateral knee. Indeed, hip OA may present with symptoms that mimic lumbar spine disorders, often confusing clinicians. Conversely, knee pathology rarely causes significant functional compromise of the hip, reinforcing the hip's dominant role in the biomechanics of the lower limb.

From a pathophysiological standpoint, knee OA frequently exhibits a discrepancy between imaging findings and the patient's reported pain levels. Pain may persist or fluctuate independently of structural degeneration, suggesting a strong component of central and peripheral sensitization. Collins *et al.* (2023) [6] observed that nearly 50% of patients with KOA experience chronic pain exacerbations over time. Neuroimaging data corroborate these findings, indicating altered activity in pain-processing brain regions and dysfunction in descending pain modulation systems (Guillot *et al.*, 2022; Zhou *et al.*, 2023) [15, 43].

Hip OA, on the other hand, tends to display a clearer structure-symptom correlation. Radiological features such as bone marrow lesions, labral tears, and subchondral cysts correlate strongly with pain and functional limitations (Metcalfe *et al.*, 2019; Fang *et al.*, 2024) [27, 9]. This concordance facilitates more straightforward clinical reasoning and timely surgical planning (Lespasio *et al.*, 2018; Pollard *et al.*, 2019) [22, 33].

Psychosocial and systemic factors modulate the experience of OA-related pain, especially in knee OA. Psychological comorbidities such as anxiety and depression, along with poor sleep quality and sarcopenic obesity, have been associated with heightened symptom burden, reduced function, and increased fall risk (Vitaloni *et al.*, 2019; Ekediegwu *et al.*, 2022; Godziuk *et al.*, 2022) [39, 8, 14]. These elements contribute to the complex clinical presentation in KOA, often compounding the dissociation between structural severity and symptom intensity.

Recent findings advocate for a paradigm shift from purely structural models to personalized, phenotype-driven treatment approaches. Neuropathic-like pain features, increasingly documented in both knee and hip OA, highlight the relevance of central sensitization and the need for nuanced diagnostic criteria (Blikman *et al.*, 2018; Pagé *et al.*, 2021) [5, 31]. Integrating clinical phenotyping, imaging biomarkers, and patient-reported outcomes is essential to selecting appropriate interventions ranging from conservative measures to interventional or surgical procedures.

Hip and knee OA represent distinct pathological and clinical entities that require differentiated approaches in diagnosis and treatment. While KOA often permits a longer course of conservative management, HOA frequently necessitates earlier surgical intervention due to its strong correlation between structural pathology and symptom severity. Understanding these differences is fundamental to optimizing pain management and improving functional outcomes through tailored, evidence-based strategies.

2. Phenotypic Characterization of Pain in Hip and Knee Osteoarthritis: Osteoarthritis (OA) of the hip and knee constitutes a major cause of chronic musculoskeletal disability worldwide. While both forms share common pathophysiological mechanisms such as cartilage degeneration, osteophyte formation, and synovial inflammation the phenotypic expression of pain differs considerably, influenced by biomechanical, neurophysiological, metabolic, and psychosocial factors (Hall *et al.*, 2019; Lespasio *et al.*, 2017) [16, 21]. This phenotypic variability underlines the importance of individualized clinical evaluation to improve therapeutic decision-making.

In knee osteoarthritis (KOA), pain is notably heterogeneous and frequently poorly correlated with structural severity observed through imaging techniques (MacKay *et al.*, 2022) [23]. Collins *et al.* (2023) [6] report that approximately 48% of KOA patients experience long-term exacerbations of pain, often exceeding two years in duration. These findings suggest that KOA pain often reflects not only mechanical overload but also central and peripheral sensitization. Fernández-de-las-Peñas *et al.* (2023) [10] further delineate three main pain phenotypes in KOA: mechanical, inflammatory, and neuropathic. The mechanical subtype is related to joint loading, whereas the inflammatory type presents with rest pain and stiffness. The neuropathic phenotype includes symptoms such as allodynia and paresthesias, associated with central sensitization in advanced stages of the disease.

The clinical complexity of KOA is compounded by systemic conditions such as sarcopenic obesity, which has been shown to intensify pain and reduce functional capacity independent of radiographic severity (Ekediegwu *et al.*, 2022; Godziuk *et al.*, 2022) [8, 14]. These metabolic-inflammatory interactions contribute to the amplification of nociceptive responses and point to the relevance of incorporating body composition and muscle function into the phenotypic classification of OA-related pain. Moreover, psychological variables such as anxiety and kinesiophobia further mediate pain chronicity and disability, requiring biopsychosocial frameworks for comprehensive management (Vitaloni *et al.*, 2019) [39].

In hip osteoarthritis (HOA), although similar phenotypic classifications are applicable, the relationship between structural joint damage and pain intensity tends to be more direct. According to Fang *et al.* (2024) [9], MRI findings such as bone marrow lesions, subchondral cysts, and synovitis correlate significantly with reported pain severity. Metcalfe *et al.* (2019) [27] add that clinical signs, including groin pain radiating to the knee and pain on internal rotation, are highly specific for HOA and serve as key diagnostic indicators. These structural-clinical correlations differentiate HOA from KOA, where pain severity often remains unexplained by imaging.

HOA pain is also modulated by non-mechanical factors. Lespasio *et al.* (2018) [22] emphasize the influence of genetic, hormonal, and biomechanical components on the persistence and intensity of symptoms. Patients with altered gait mechanics, pelvic instability, and muscle imbalance frequently present with refractory pain, even in the early stages of joint degeneration. Furthermore, Pagé *et al.* (2021) [31] document the presence of neuropathic-like features and central sensitization in HOA, particularly in chronic pain scenarios, although less commonly than in KOA.

Both KOA and HOA exhibit overlapping pain mechanisms, including peripheral and central sensitization, neuroinflammation, and psychoemotional comorbidities (Blikman *et al.*, 2018; Guillot *et al.*, 2022) [5, 15]. However, KOA tends to show a higher dissociation between structural findings and clinical presentation, making diagnosis and treatment more complex.In contrast, HOA usually presents a more consistent pattern of pain distribution and structural damage, often facilitating earlier surgical intervention (Whitaker *et al.*, 2025; Bahl *et al.*, 2018) [41, 3]. These

differences have direct implications for prognosis, therapeutic response, and patient education.

The phenotypic characterization of pain in hip and knee osteoarthritis reveals both convergence and divergence in etiological mechanisms and clinical expression. While mechanical, inflammatory, and neuropathic phenotypes occur in both conditions, their prevalence, structural correlation, and impact on function differ. As such, individualized phenotypic assessment integrating clinical examination, imaging data, and patient-reported measures is essential for guiding personalized, multidisciplinary interventions (Hunter *et al.*, 2024; Mandl *et al.*, 2019) [17, 24]. This approach not only refines diagnostic accuracy but also improves long-term outcomes in OA-related pain management.

3. Neurophysiological Mechanisms of Pain in Hip and Knee Osteoarthritis: Pain in osteoarthritis (OA), particularly of the knee and hip, transcends the notion of being purely nociceptive and mechanically driven. While early stages of the disease are typically characterized by mechanical wear and biochemical inflammation, the persistence and intensification of symptoms are increasingly understood as consequences of complex neurophysiological processes involving peripheral sensitization, central sensitization, and maladaptive neuroplasticity (Khella *et al.*, 2021) [20]. This multifaceted pathophysiology challenges traditional biomechanical paradigms and necessitates a deeper understanding of nociceptive system dynamics.

Peripheral sensitization is initiated by structural damage, such as cartilage degradation, osteophyte formation, and synovitis, leading to activation of joint nociceptors and subsequent release of inflammatory mediators, including prostaglandins, interleukins (e.g., IL-1β), and tumor necrosis factor-alpha (TNF-α). These agents lower the activation threshold of nociceptive neurons, enhancing pain transmission from the periphery (Jang, Lee & Ju, 2021; Khella *et al.*, 2021) [18, 20]. The clinical result is localized hyperalgesia and pain during joint movement or loading. In hip OA, these mechanisms are similarly activated, with matrix degradation exposing type II collagen and aggravating nociceptive signaling through immune-mediated responses (Jang, Lee & Ju, 2021) [18].

However, persistent peripheral input leads to sensitization of central neural circuits. Central sensitization involves functional and structural changes in the spinal cord and brain that result in pain amplification, reduced pain thresholds, and the extension of pain beyond the original site of injury (Corriero *et al.*, 2024) [7]. Neurophysiological changes include increased excitability of spinal dorsal horn neurons, disinhibition of descending pain-modulatory pathways, and astrocytic and microglial activation. Fernández-de-las-Peñas *et al.* (2023) [10] emphasize that central sensitization occurs in more than one-third of individuals with advanced OA and is a significant predictor of persistent postoperative pain, even after joint arthroplasty.

Neuroimaging studies offer robust support for the central processing hypothesis. Functional MRI reveals altered connectivity among the insula, anterior cingulate cortex, prefrontal cortex, and thalamus key regions involved in sensory-discriminative and affective-emotional components of pain (Zhou *et al.*, 2023) [43]. These central changes reflect cortical reorganization, which contributes not only to pain chronification but also to the inefficacy of treatments aimed solely at the joint periphery. In hip OA, Guillot *et al.* (2022) [15] demonstrated that decreased functional connectivity in pain-processing brain areas correlates with greater pain intensity, independent of structural damage, underscoring the neuroplastic adaptation of the brain in chronic pain conditions.

Bone marrow lesions (BMLs) are another critical component in the neurophysiology of OA pain. Identified via MRI, BMLs are thought to represent areas of subchondral bone ischemia, edema, and microfracture, and they have been implicated as independent pain generators (Fang *et al.*, 2024) [9]. Their presence has been strongly associated with pain severity in both hip and knee OA, even when radiographic assessments indicate minimal structural damage. This discrepancy between radiological findings and pain experience further highlights the inadequacy of imaging alone in explaining symptom burden and supports the integration of functional diagnostic tools.

In addition to neurobiological mechanisms, psychological factors such as depression, anxiety, and catastrophizing have a substantial influence on pain processing. These variables interact with the neuroendocrine and limbic systems, amplifying pain perception and diminishing the analgesic response to both pharmacological and non-pharmacological treatments (Hunter *et al.*, 2024) [17]. Vitaloni *et al.* (2019) [39] report that quality of life metrics including sleep quality, emotional well-being, and cognitive appraisal of pain strongly correlate with functional outcomes in OA patients, underscoring the relevance of psychosocial dimensions. Incorporating these domains into clinical assessment tools enables more precise phenotyping and therapeutic targeting.

Moreover, the interplay between systemic inflammation, neuroimmune responses, and gut microbiota has been explored as a contributing factor in OA-related pain. Corriero *et al.* (2024) [7] suggest that microbial dysbiosis may exacerbate systemic inflammatory pathways, influencing both peripheral and central sensitization mechanisms. Although this is an emerging field, it aligns with growing evidence that chronic OA pain is modulated not only by local joint pathology but also by systemic and behavioral factors that interact within a biopsychosocial framework.

Importantly, traditional analgesic strategies that focus solely on reducing peripheral inflammation such as NSAIDs or intra-articular corticosteroids are often insufficient in patients with dominant central pain mechanisms. Novel approaches, including centrally acting neuromodulators (e.g., duloxetine), cognitive-behavioral therapy, exercise-based desensitization, and neurostimulation techniques, have demonstrated promise in addressing these central drivers of pain (Fernández-de-las-Peñas *et al.*, 2023; Zhou *et al.*, 2023) [10, 43]. These modalities reinforce the need for a multidimensional approach to OA management, tailored to the individual's pain phenotype.

The neurophysiological mechanisms of pain in hip and knee OA involve a cascade that begins with peripheral nociceptive activation and progresses to central sensitization and neuroplastic adaptation. These processes are further modulated by psychological, systemic, and environmental influences, making OA pain a multifactorial phenomenon. Understanding the interplay of these components is crucial for the development of effective, individualized treatment

strategies that transcend structural correction and address the full spectrum of pain pathophysiology in osteoarthritis.

4. Biomechanical Alterations in Hip and Knee Osteoarthritis: Biomechanical alterations are not merely consequences but active contributors to the initiation and progression of osteoarthritis (OA), particularly in load-bearing joints such as the knee and hip. These alterations encompass deviations in joint alignment, muscular dysfunction, and changes in gait dynamics, all of which interact with inflammatory, degenerative, and neurosensory mechanisms to influence disease outcomes (Aljehani *et al.*, 2022; Gademan *et al.*, 2016) [2, 12].

In knee osteoarthritis (KOA), one of the most clinically significant biomechanical factors is frontal plane malalignment, especially varus deformity. Varus alignment leads to excessive loading of the medial compartment, creating a disproportionate mechanical burden that accelerates cartilage degradation, subchondral bone sclerosis, and meniscal extrusion (Aljehani *et al.*, 2022) [2]. Even mild deviations from neutral alignment can substantially increase the external knee adduction moment (EKAM), which is strongly associated with pain severity, disease progression, and functional limitation (Gademan *et al.*, 2016) [12].

The mechanical overloading of the knee is compounded by neuromuscular deficits, particularly in the quadriceps and hamstring muscle groups. Weakness in these dynamic stabilizers disrupt the distribution of joint forces and diminishes proprioceptive feedback, both of which are critical for maintaining joint integrity during gait and other functional tasks (Wu *et al.*, 2024) [42]. Furthermore, sarcopenia defined as the age-related loss of muscle mass and strength further compromises shock absorption and load control in older adults with KOA, aggravating joint stress during basic movements such as stair climbing or sit-to-stand transitions (Godziuk *et al.*, 2022) [14].

The biomechanical consequences of unilateral joint replacement also warrant attention. Patients undergoing total knee arthroplasty (TKA) frequently display compensatory gait patterns that overload the contralateral knee, thereby accelerating OA progression in the uninvolved limb (Aljehani *et al.*, 2022) [2]. This interlimb compensation highlights the systemic nature of biomechanical adaptation and supports the need for bilateral assessment and targeted interventions even when only one joint is surgically treated.

Computational biomechanical models have provided valuable insights into the distribution of stress and strain across articular surfaces. Gardiner *et al.* (2016) [13] demonstrated that postural deviations and altered joint geometry can result in repeated microtrauma in already vulnerable regions. These repetitive insults not only exacerbate local tissue degeneration but may also trigger inflammatory responses that reinforce the cycle of mechanical breakdown and biochemical inflammation. Such findings underscore the value of early biomechanical correction to delay or prevent irreversible structural damage.

In hip osteoarthritis (HOA), biomechanical alterations present distinct patterns but similarly contribute to the progressive loss of joint function. Patients with HOA often exhibit shortened step length, decreased gait speed, and increased double-limb support time, all of which are markers of impaired joint mechanics and neuromuscular coordination (Bahl *et al.*, 2018) [3]. These changes correlate with both symptom severity and radiographic findings, although functional impairment often precedes substantial imaging evidence, suggesting a strong neuromechanical component to early-stage HOA (Lespasio *et al.*, 2018) [22].

Importantly, biomechanical recovery following total hip arthroplasty is not always complete. Bahl *et al.* (2018) [3] reports that patients may continue to show asymmetrical loading, pelvic instability, and altered hip kinematics long after surgery. This residual dysfunction highlights the need for extended rehabilitation protocols that emphasize neuromuscular retraining, not just range of motion or strength recovery. Furthermore, persistent muscle imbalance especially between the hip abductors, adductors, and flexors can lead to inefficient movement strategies, increased joint loads, and compensatory stress on adjacent joints (Lespasio *et al.*, 2018; Sueyoshi *et al.*, 2024) [22, 35].

Postural compensation in HOA can also involve the lumbar spine and contralateral lower limb. Pelvic tilt, excessive lumbar lordosis, and trunk lean are frequently observed strategies to reduce hip loading, but these adaptations may lead to secondary problems such as back pain or contralateral joint degeneration (Sueyoshi *et al.*, 2024) [35]. These systemic interactions reaffirm that OA should be conceptualized as a condition affecting the entire kinetic chain, rather than an isolated joint disease.

The integration of biomechanical assessments into the clinical management of OA enables a more individualized approach. Gait analysis, strength testing, and joint loading assessments can inform tailored interventions, including physiotherapy, orthotic devices, and surgical alignment correction. According to Gademan *et al.* (2016) [12], biomechanical modeling can even be used to identify optimal candidates for joint-preserving interventions and predict outcomes following total joint replacement. This is especially relevant in early-stage OA, where conservative biomechanical strategies may substantially alter disease trajectories.

Biomechanical alterations in hip and knee OA are fundamental not only to the expression of symptoms but to the very mechanisms of disease progression. These alterations interact dynamically with neuromuscular, inflammatory, and psychological components, requiring integrated, multidisciplinary treatment strategies. Addressing biomechanical dysfunction through both preventive and rehabilitative approaches holds promise for improving quality of life and delaying structural deterioration in OA populations.

5. Pharmacological Management of Hip and Knee Osteoarthritis: Pain management is a cornerstone in the pharmacological treatment of hip and knee osteoarthritis (OA), aiming not only to reduce symptoms but also to improve joint function and quality of life. However, the heterogeneity of pain phenotypes ranging from nociceptive and inflammatory to neuropathic and centrally sensitized pain demands a tailored and multifaceted pharmacologic approach. In this context, pharmacological interventions must address both the peripheral and central mechanisms underlying chronic OA pain.

Non-steroidal anti-inflammatory drugs (NSAIDs) remain the most prescribed medications for OA-related pain, especially in early or moderate stages of the disease. They exert their analgesic and anti-inflammatory effects by inhibiting cyclooxygenase enzymes and reducing

prostaglandin synthesis. Nevertheless, their long-term use is limited by the risk of gastrointestinal, cardiovascular, and renal complications, particularly in elderly populations with comorbidities (MACKAY *et al.*, 2022) [23]. Given their risk profile, NSAIDs should be used at the lowest effective dose and for the shortest possible duration, particularly when pain is clearly linked to mechanical overload and synovitis.

In patients who do not respond adequately to NSAIDs or who have contraindications, weak opioids such as tramadol may be considered. However, recent evidence suggests that opioids offer no clear advantage over NSAIDs in terms of pain-related functional outcomes and are associated with higher rates of adverse events, including dependence, sedation, and falls (Krebs *et al.*, 2018). Therefore, opioid therapy should be limited to carefully selected, refractory cases and always integrated into a broader pain management plan (Mandl *et al.*, 2019) [24]. This reinforces the principle that controlling pain does not necessarily require escalating analgesic potency but rather understanding the dominant pain mechanism in each patient.

Intra-articular corticosteroid injections, such as triamcinolone, have traditionally been used to manage acute exacerbations of pain, particularly when associated with synovial inflammation. These injections can lead to significant short-term pain relief by suppressing local inflammatory responses. However, repeated injections raise concerns about potential chondrotoxicity and acceleration of joint degeneration (Mackay *et al.*, 2022) [23]. According to Lespasio *et al.* (2017) [21], corticosteroid injections should be used judiciously and in conjunction with non-pharmacologic strategies, especially in advanced OA, where structural damage may limit their efficacy and safety.

To optimize local drug delivery and reduce systemic toxicity, novel formulations such as liposome-based corticosteroids have emerged. These systems allow for extended intra-articular release, maintaining therapeutic drug concentrations for longer periods (Mitsou; Klein, 2025) [28]. Such innovations are particularly relevant for pain control in patients with recurrent flares or persistent low-grade inflammation, potentially reducing the frequency of injections and mitigating systemic side effects.

The exploration of biological and regenerative therapies is also advancing in the field of OA pain management. Therapies targeting proinflammatory cytokines, including interleukin-1β and TNF-α, have shown promise in experimental settings but are not yet established in clinical practice. Furthermore, intra-articular administration of mesenchymal stem cells (MSCs), proposed for their immunomodulatory and regenerative properties, has yielded mixed results. In a randomized, double-blind, placebo-controlled study, Zhou *et al.* (2023) [43] reported no significant differences in pain reduction between MSC and placebo groups after six months, suggesting the need for further investigation regarding optimal dosing, patient selection, and long-term outcomes.

Emerging nanotechnological strategies, such as drug-loaded nanoparticles and intelligent intra-articular vectors, have the potential to revolutionize pain control in OA. These delivery systems aim to enhance retention within the joint cavity and achieve controlled, sustained release of analgesic and anti-inflammatory agents (Khella *et al.*, 2021) [20]. While still under investigation, such approaches are particularly attractive for patients with contraindications to systemic pharmacotherapy or who exhibit fluctuating patterns of pain related to inflammatory bursts.

Pharmacological pain control in OA must go beyond a symptomatic approach and instead integrate phenotypic characterization of pain, patient comorbidities, and individual response to therapies. NSAIDs continue to be the foundation of analgesic treatment, with corticosteroids and tramadol serving as adjuncts in selected cases. Innovative strategies, including liposomal formulations, biologics, and nanotechnologies, offer future potential but require further clinical validation. Effective pain management in OA is not solely about suppressing nociceptive input but also about modulating central mechanisms and improving overall patient function.

6. Conservative Therapies in Hip and Knee Osteoarthritis: Conservative therapies are fundamental in the management of osteoarthritis (OA) of the hip and knee, particularly in early and moderate stages of the disease. These interventions not only postpone surgical needs but also provide multidimensional benefits including pain relief, functional restoration, and enhancement of quality of life (Hunter *et al.*, 2024; Vitaloni *et al.*, 2019) [17, 39]. Tailoring these strategies to individual clinical phenotypes, comorbidities, and biomechanical profiles is essential to optimizing outcomes (Ekediegwu *et al.*, 2022) [8].

In knee OA, conservative treatment frequently includes structured physical therapy aimed at strengthening the quadriceps and hamstring muscles, improving joint stability and proprioception, and enhancing gait mechanics (Bahl *et al.*, 2018) [3]. Adjunctive use of laterally wedged insoles and medial arch supports contributes to realigning load distribution and mitigating medial compartment degeneration, especially in patients with varus alignment (Jindasakchai *et al.*, 2023; Aljehani *et al.*, 2022) [19, 2]. Pain modulation strategies such as pain neuroscience education and neuromuscular re-education are integrated into multidisciplinary rehabilitation protocols (Blikman *et al.*, 2018; Guillot *et al.*, 2022) [5, 15]

In contrast, hip OA demands a more nuanced approach given its central biomechanical role and stronger correlation between structural degeneration and symptomatology (Fang *et al.*, 2024; Hall *et al.*, 2019) [9, 16]. Exercise therapy prioritizes gluteal and pelvic stabilizer activation, as gait alterations and pelvic asymmetry are common (Bahl *et al.*, 2018; Berteau, 2024) [3, 4]. While orthotic interventions are less relevant, targeted joint mobilization, neuromuscular re-education, and aquatic therapy may benefit patients with movement-related pain or altered posture.

Intra-articular viscosupplementation with high molecular weight hyaluronic acid has demonstrated variable efficacy in both joints but is more commonly applied in knee OA due to accessibility and anatomical feasibility (Patel *et al.*, 2024; Altman *et al.*, 2016) [32]. For hip OA, although technically more complex, ultrasound-guided HA injections may offer pain relief and functional improvement in selected cases (Mackey *et al.*, 2022) [23]. Corticosteroid injections, while effective short-term, must be used cautiously due to their potential chondral toxicity, especially in younger or more active individuals (Mandl *et al.*, 2019; Mackey *et al.*, 2022) [24, 23].

Interventional pain management plays a pivotal role in OA refractory to conventional therapy. In knee OA, genicular nerve blocks and radiofrequency ablation (RFA) have

proven effective in disrupting nociceptive signaling, with cooled and pulsed RFA showing durable benefits in reducing pain and improving function (Soetjahjo *et al.*, 2024; Zhou *et al.*, 2023) [34, 43]. In hip OA, the pericapsular nerve group (PENG) block targets articular branches of the femoral, obturator, and accessory obturator nerves, offering perioperative and chronic pain control. PENG has demonstrated efficacy not only in surgical settings but also as part of conservative management strategies (Girón-Arango *et al.*, 2018) [46].

Weight management remains a cornerstone across both conditions, as excess body weight contributes to mechanical overload and systemic inflammation (Wu *et al.*, 2024; Godziuk *et al.*, 2022) [42, 14]. Psychological support, pain neuroscience education, and cognitive behavioral therapy are critical, especially in knee OA, where psychosocial factors such as kinesiophobia and catastrophizing are closely linked to disability and poor adherence to therapy (Blikman *et al.*, 2018; Ekediegwu *et al.*, 2022) [5, 8]. Technological advancements, such as wearable biofeedback systems and remote monitoring tools, are being integrated into rehabilitation programs to enhance adherence and personalize interventions (Vaidya *et al.*, 2024) [37].

In summary, although hip and knee OA share common therapeutic goals, their conservative management must be differentiated. Knee OA generally responds well to structured rehabilitation and minimally invasive pain procedures, allowing for prolonged non-surgical management. Hip OA, on the other hand, often necessitates earlier escalation of care due to its biomechanical centrality and stronger structure-symptom correlation. A joint-specific, phenotype-guided, and interdisciplinary approach remains fundamental to optimize outcomes in both conditions.

7. Surgical Treatments for Osteoarthritis: An Expanded Technical Discussion: Surgical interventions are essential in managing advanced and treatment-resistant osteoarthritis (OA), particularly when conservative and pharmacological strategies have been exhausted. Total knee arthroplasty (TKA) represents the gold standard for end-stage knee OA, providing substantial pain relief and functional restoration. The decision between TKA and unicompartmental knee arthroplasty (UKA) should be based not only on radiographic criteria but also on patient symptomatology, axial alignment, and compartmental degeneration. The Knee Osteoarthritis Grading System (KOGS) has proven to be a useful tool for surgical decision-making, improving selection accuracy and clinical outcomes (Oosthuizen *et al.*, 2019) [30].

However, surgical success is not guaranteed. Approximately 30% of patients undergoing TKA report persistent postoperative pain one year after surgery. Chronic pain following arthroplasty has been linked to central sensitization mechanisms, pain catastrophizing, and psychological comorbidities such as anxiety and depression. These elements are predictive of unfavorable surgical outcomes and should be identified preoperatively through comprehensive clinical assessments (Fernández-DE-LAS-Peñas *et al.*, 2023; Collins *et al.*, 2023) [10, 6]. Moreover, pre-existing neuropathic pain components may contribute to the persistence of pain and reduced functional gains (Blikman *et al.*, 2018) [5].

Minimally invasive procedures, such as genicular nerve radiofrequency ablation, have emerged as viable alternatives for patients who are not surgical candidates. This technique offers significant pain relief for up to six months with a favorable safety profile and minimal complications, positioning it as a palliative option in advanced OA management (Soetjahjo *et al.*, 2024) [34]. Additionally, such interventions may delay the need for joint replacement in certain populations with multimorbidity or surgical contraindications.

Technological innovations have revolutionized surgical approaches to joint replacement. Robotic-assisted arthroplasty and intraoperative navigation systems allow for enhanced precision in prosthetic alignment and bone preservation. These advances have been associated with improved biomechanical outcomes, reduced operative times, and higher levels of postoperative patient satisfaction (Vaidya *et al.*, 2024) [37]. Furthermore, accurate implant positioning reduces wear and revision rates, thereby improving long-term success.

In cases of hip OA, total hip arthroplasty (Tha) remains the most effective surgical treatment. It is indicated primarily when pain and functional limitation significantly impair quality of life. Lespasio *et al.* (2018) [22] emphasize the necessity of comprehensive clinical evaluation to determine the appropriateness of THA, while Aandersson *et al.* (2022) [1] highlight the correlation between postoperative outcomes and patient-reported measures of function and satisfaction. Nonetheless, biomechanical alterations in gait may persist long after surgery. Bahl *et al.* (2018) [3] observed that many patients continue to exhibit asymmetries and reduced neuromuscular control one year postoperatively, reinforcing the need for long-term, individualized rehabilitation.

Several factors influence the postoperative trajectory, including baseline functional status, presence of sarcopenic obesity, and adherence to physiotherapy protocols (Godziuk *et al.*, 2022; Zonnenberg *et al.*, 2017) [14, 45]. Structured multidisciplinary rehabilitation integrating physical therapy, psychological support, and patient education is crucial to optimize recovery and minimize chronic pain and functional decline (Vitaloni *et al.*, 2019; Ekediegwu *et al.*, 2022) [39, 8].

Thus, surgical treatment for osteoarthritis should not be viewed solely as a mechanical solution, but rather as part of a comprehensive management pathway. Proper patient selection, prehabilitation, technological integration, and robust postoperative support are essential components for maximizing functional recovery and improving quality of life for individuals affected by this debilitating condition.

8. Conclusion: Hip and knee osteoarthritis (OA), while sharing common degenerative pathways, present with distinct pain phenotypes, biomechanical dynamics, and neurophysiological mechanisms. These differences have profound implications for diagnostic precision, prognostic stratification, and therapeutic targeting in the context of contemporary pain medicine. Knee OA is more frequently characterized by a dissociation between structural severity and symptom burden, a higher prevalence of central sensitization, and greater psychosocial comorbidity, which collectively demand a multidimensional and individualized therapeutic approach (Collins *et al.*, 2023; Fernández-de-las-Peñas *et al.*, 2023; Vitaloni *et al.*, 2019) [6, 10, 39]. In contrast, hip OA typically shows a closer correlation between imaging findings and pain intensity, allowing for earlier and

more predictable responses to surgical interventions (Fang *et al.*, 2024; Metcalfe *et al.*, 2019; Lespasio *et al.*, 2018) [9, 27, 22].

Understanding the neurophysiological basis of OA pain particularly the roles of peripheral and central sensitization, neuroinflammation, and maladaptive neuroplasticityis critical to optimizing pain management strategies. Functional neuroimaging studies and clinical phenotyping provide valuable insights into the complexity of chronic OA pain, reinforcing the need to move beyond traditional structural and pharmacologic paradigms (Guillot *et al.*, 2022; Zhou *et al.*, 2023; Corriero *et al.*, 2024) [15, 43, 7]. Moreover, emerging evidence supports the use of individualized pharmacologic regimens, neuromodulatory agents, and targeted interventions such as genicular nerve radiofrequency for refractory cases (Soetjahjo *et al.*, 2024; Mautner *et al.*, 2023) [34, 26].

Conservative therapies, including supervised exercise, biomechanical optimization, weight management, and psychosocial interventions, remain foundational across both OA types.

These approaches not only mitigate symptoms but also target the underlying contributors to chronic pain and joint dysfunction (Hunter *et al.*, 2024; Ekediegwu *et al.*, 2022; Jindasakchai *et al.*, 2023) [17, 8, 19]. Surgical options, particularly total joint arthroplasty, are effective for advanced disease, but success depends on accurate patient selection, management of central pain mechanisms, and long-term rehabilitation strategies (Oosthuizen *et al.*, 2019; Vaidya *et al.*, 2024; Bahl *et al.*, 2018) [30, 37, 3].

Ultimately, the management of hip and knee OA must be anchored in a comprehensive framework that integrates structural findings, pain phenotypes, neurophysiological mechanisms, and patient-reported experiences. This personalized approach allows for the optimization of conservative, pharmacological, interventional, and surgical strategiesthereby enhancing functional recovery, minimizing chronic pain, and improving quality of life for individuals living with osteoarthritis.

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