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Management of early-onset knee osteoarthritis in athletes: current concepts

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Abstract

Early-onset knee osteoarthritis (OA) is no longer a condition of ageing joints; it is now an emerging threat to the careers of athletes in their prime. High-impact sports, repetitive microtrauma, and incomplete recovery after injury contribute to a biologically active and mechanically unstable joint environment, often years before radiographic changes appear. In this narrative review, we explore how modern sports medicine is shifting from reactive to proactive strategies, focusing on joint preservation, functional optimisation, and long-term performance sustainability. From precision diagnosis using advanced imaging and biomarkers to tailored exercise programmes, biologic injections, and targeted surgical options, the management of early-onset knee OA demands an integrated, sport-specific approach. The athlete's return to sport is not merely a clinical milestone but a biomechanical, psychological, and strategic decision. Despite growing awareness, current guidelines fail to address the complexity of early-onset OA in high-performing individuals, and robust long-term data are lacking. This review provides an evidence-based framework to delay degeneration, empower athletes, and keep performance at the centre of care.

Keywords Early-onset osteoarthritis, Sport performance, Return to sport, Knee biomechanics, Regenerative medicine, Precision medicine, Biomarkers

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Introduction

Early-onset knee osteoarthritis (OA) in athletes has emerged as a growing clinical concern [1, 2]. Traditionally considered a degenerative condition of ageing, OA is increasingly recognised as a significant issue in young, physically active individuals, particularly those engaged in high-impact sports such as football, rugby, basketball, and long-distance running [2–4]. This shift in epidemiological profile is supported by growing evidence indicating that elite and semi-professional athletes face a disproportionately high risk of developing OA in weight-bearing joints well before reaching middle age [5, 6]. The mechanisms underpinning this phenomenon are multifactorial and include acute joint injuries, chronic mechanical overload, inadequate rehabilitation, and intrinsic anatomical or genetic predispositions [7].

Although this review mainly addresses tibiofemoral early-onset knee OA, patellofemoral involvement may

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occur secondarily in athletes with pathoanatomical factors leading to maltracking or instability, underscoring the role of altered biomechanics in early degenerative pathways [8–11]. The importance of recognising and managing early-onset OA in athletes lies not only in preserving joint integrity and delaying the progression to more advanced degenerative stages, but also in maintaining athletic performance, extending career longevity, and preventing irreversible disability [12]. Unlike age-related OA, the early-onset form observed in sportspeople frequently follows a post-traumatic pattern, with structural changes emerging shortly after injuries [13, 14]. These initial events accelerate the catabolic environment within the joint, leading to early cartilage breakdown, subchondral bone remodelling, and synovial inflammation. Furthermore, early-onset OA in this context is often underdiagnosed or misclassified, as radiographic signs may lag behind symptoms, and the typical pain profile may differ from that of older sedentary individuals [15]. The high physical demands and psychological pressure to return to play may drive athletes to resume activity prematurely, often before sufficient joint healing has occurred, thereby further exacerbating the degenerative cascade [16, 17].

The current clinical challenge is therefore twofold: first, to develop reliable criteria for the early detection and monitoring of OA in young athletes, and second, to establish multidisciplinary, individualised management strategies that balance symptom control, joint preservation, and performance optimisation [18]. Although conservative approaches remain the first-line treatment, the role of regenerative medicine, biologics, and tailored physiotherapy protocols is becoming increasingly prominent [19]. At the same time, surgical options such as cartilage restoration procedures or realignment osteotomies must be critically evaluated in light of each athlete's age, sport, and functional goals [20–22].

The present narrative review synthesises and critically appraises the current evidence on the aetiology, diagnosis, and management of early-onset knee OA in athletic populations, providing a comprehensive overview of the condition and proposing informed strategies for its prevention and treatment, with the ultimate goal of preserving joint function and athletic performance over time. From a clinical perspective, this review addresses a common but underrepresented scenario in sports medicine, namely the management of athletes with early-onset knee OA who fall outside traditional OA frameworks. It provides practical guidance to support everyday decision-making on load management, rehabilitation progression, imaging use, injection timing, and return-to-sport clearance, integrating current evidence with sport-specific biomechanics and clinical reasoning. Rather than proposing rigid treatment algorithms, the review offers an athlete-centred framework that helps clinicians balance performance demands with joint preservation, thereby translating fragmented evidence into actionable principles applicable to real-world sports medicine practice.

Definition and diagnosis of early-onset knee osteoarthritis in athletes

Early-onset knee OA in athletes represents a distinct clinical entity that diverges substantially from the classical age-related form of the disease. Although senile OA typically arises as a slowly progressive degenerative condition associated with cumulative cartilage wear, subchondral bone sclerosis, and joint space narrowing in individuals beyond the fifth or sixth decade of life, the early variant observed in sports populations often develops within the second to fourth decade of life [23–25]. It is typically triggered by mechanical, biological, or traumatic factors that act on structurally compromised joints [26]. This distinction is more than chronological, as early-onset OA is frequently characterised by focal rather than diffuse cartilage degeneration, a more prominent inflammatory component in the early stages, and a history of high-impact joint loading or previous injury [27]. Moreover, the clinical course may differ significantly, with fluctuating symptoms and intermittent flares that are often poorly correlated with radiographic findings, particularly in the early phase of disease development [28].

Diagnosing early-onset OA in athletes remains challenging, given the subtlety of initial structural changes and the lack of universally accepted criteria tailored to this demographic [29]. Although the American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) criteria provide a foundation for OA diagnosis, they were initially developed for older, sedentary populations [30] (Table 1). They may not fully capture the clinical spectrum observed in young active individuals. The ACR criteria for knee OA, which combine clinical symptoms such as pain, morning stiffness, and functional limitation with signs including crepitus, bony tenderness, and joint enlargement, have high sensitivity but lower specificity in athletic cohorts [29, 31]. The EULAR criteria enhance diagnostic accuracy by incorporating age thresholds, stiffness duration, and the presence of radiographic features, yet remain biased toward degenerative profiles that are not necessarily applicable to early structural damage seen in post-traumatic or overuse conditions [30].

For this reason, advanced imaging modalities are essential in the assessment of early-onset OA in athletes [32]. Magnetic resonance imaging (MRI) plays a pivotal role, as it allows for the direct visualisation of cartilage

Table 1 Comparison of diagnostic frameworks for knee OA and athlete-specific considerations (ACR: American College of Rheumatology; EULAR: European League Against Rheumatism)

Framework	Core diagnostic elements	Strengths	Limitations in athletes	Athlete-specific considerations
ACR criteria	Knee pain combined with clinical features (age, stiffness, crepitus, bony tenderness/enlargement)	High sensitivity; easy to apply in routine practice	Developed for older, sedentary populations; low specificity for early or post-traumatic OA; limited role of imaging	Symptoms may be activity-related and intermittent; absence of typical stiffness or crepitus does not exclude early disease
EULAR criteria	Clinical symptoms integrated with age thresholds and radiographic findings	Improved diagnostic accuracy in established OA; structured approach	Reliance on radiographic changes limits applicability in pre-radiographic or early-onset OA	Structural changes may lag behind symptoms; radiographs often normal despite functional impairment
Imaging-based assessment (MRI)	Cartilage defects, bone marrow lesions, meniscal pathology, synovitis	Detection of early structural and pre-radiographic changes	Limited specificity; abnormalities may be present in asymptomatic athletes	Findings should be interpreted in relation to symptoms, load exposure, and sport demands
Athlete-specific integrated approach	Symptoms, functional performance, injury history, imaging, load tolerance	Reflects real-world sports medicine practice; supports individualised decision-making	Lack of validated thresholds; limited guideline support	Emphasis on functional impact, response to load, and risk of progression rather than radiographic severity

integrity, subchondral bone marrow lesions (BML), synovitis, meniscal damage, and early osteophyte formation [33, 34]. Unlike standard radiographs, which may appear normal or only mildly altered in the early stages, MRI can detect pre-radiographic changes that may precede symptomatic deterioration [34, 35]. T2 mapping and delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) further enable quantitative evaluation of cartilage composition, offering valuable insights into matrix degradation and early biochemical alterations [36, 37]. Meniscal extrusion, posterior root tears, and subchondral bone changes are particularly relevant in this population, as they may act as catalysts for rapid structural decline even in the absence of widespread cartilage loss [38–41]. When interpreted in conjunction with clinical findings and functional assessments, MRI can guide not only the diagnosis but also the stratification of athletes for appropriate therapeutic interventions and prognostic counselling [42].

Advanced imaging can identify early structural and compositional joint changes, but its routine use in sports medicine must be weighed against cost, access, and clinical yield. Standard MRI is usually sufficient to support management decisions when interpreted alongside symptoms and functional findings, whereas quantitative techniques, although informative in specialist or research settings, remain less accessible and their incremental impact on everyday decisions is often unclear. Imaging abnormalities may also precede symptoms, and an overly imaging-driven approach risks overdiagnosis or unnecessary restriction from sport. A pragmatic strategy is therefore to use imaging when results are likely to change clinical management, particularly load modification, rehabilitation planning, or return-to-sport decisions, rather than as a standalone marker of disease severity.

Aetiopathogenetic factors in young athletes

The development of early-onset knee OA in athletic populations is driven by a complex interplay of mechanical, biological, and individual factors that create a pathophysiological environment conducive to accelerated joint degeneration [18, 43]. Among the most well-documented contributors are acute traumatic injuries, particularly those involving the anterior cruciate ligament (ACL) and the meniscus [44]. Ruptures of the ACL represent a critical event in the natural history of knee OA, with evidence showing that over 50% of individuals develop radiographic signs of osteoarthritic changes within 10–15 years following injury, regardless of whether reconstruction is performed [45, 46]. The biomechanical consequences of ACL deficiency or graft laxity, such as increased tibiofemoral shear forces and altered joint kinematics, initiate a cascade of cartilage degradation, subchondral bone remodelling, and synovial inflammation [25, 47, 48]. Similarly, meniscal tears, particularly partial meniscectomies, significantly diminish load distribution and shock absorption, increasing peak contact stress on articular cartilage, which in turn accelerates focal cartilage wear and promotes early degenerative changes [39, 49, 50].

In addition to discrete injuries, cumulative mechanical overload plays a central role in the pathogenesis of early-onset OA in athletes involved in high-impact sports [51]. Activities such as football, basketball, and long-distance running expose the knee joint to repetitive axial compression, torsional stress, and shear forces that exceed physiological thresholds, particularly during eccentric loading phases and abrupt directional changes [2, 52]. These repetitive microtraumatic insults disrupt cartilage homeostasis, triggering an imbalance between anabolic and catabolic processes within the joint environment [27, 53]. Chondrocytes subjected to excessive mechanical stress upregulate the expression of matrix metalloproteinases, aggrecanases, and pro-inflammatory cytokines, thereby contributing to the breakdown of the extracellular matrix, chondral fibrillation, and subchondral microdamage [54]. The cumulative nature of this stress is particularly detrimental when combined with insufficient recovery time, suboptimal rehabilitation, or premature return to high-load activities, all of which are common in competitive sports settings [2, 55–57].

Individual predisposition also modulates the susceptibility to early joint degeneration. Anatomical incongruities, such as trochlear dysplasia, femoroacetabular impingement with compensatory knee loading, or varus-valgus malalignment, alter joint biomechanics and load distribution, predisposing the patient to asymmetric cartilage damage [58–60]. Furthermore, generalised ligamentous laxity and hypermobility syndromes may amplify joint instability, leading to microinstability patterns that are difficult to detect clinically yet highly deleterious over time [61, 62]. Genetic predisposition is increasingly recognised as a significant factor, with polymorphisms in genes encoding for collagen type II, interleukin-1β, and matrix-degrading enzymes shown to influence cartilage resilience and inflammatory responsiveness [63, 64]. Additionally, the concept of "sport-related obesity" has gained attention, referring to athletes with elevated body mass due to hypertrophic musculature or excessive energy intake, which nonetheless contributes to increased mechanical joint loading and metabolic dysregulation [65]. Adipose-derived cytokines such as leptin and resistin have been implicated in cartilage catabolism and synovial inflammation, further reinforcing the metabolic contribution to early-onset OA in this population [66, 67].

Clinical evaluation and outcome measures

The clinical assessment of early-onset knee OA in athletes requires a nuanced and multidimensional approach, given the unique presentation patterns in this population and the limitations of conventional diagnostic frameworks [18, 68, 69]. Pain remains the primary symptom prompting clinical evaluation, although its characteristics may differ substantially from the patterns typically observed in age-related OA [26]. In young athletic individuals, pain is often intermittent and activity-dependent, with exacerbations during high-load or impact phases rather than at rest [26, 70]. Morning stiffness, if present, is usually brief and subtle, and crepitus or joint swelling may be inconsistent [70]. Significantly, the perception of pain in athletes may be modulated by psychological resilience, competition demands, and pain-coping strategies, leading to underreporting or delayed presentation [71]. Clinical examination should assess joint line tenderness, effusion, patellofemoral crepitus, range of motion (ROM), and stability, with attention to previous surgical scars, gait abnormalities, and signs of compensatory loading [26, 72].

To quantify functional impairment and monitor disease progression, patient-reported outcome measures (PROMs) have become essential, particularly when evaluating the impact of early-onset OA in a high-demand cohort [73]. Among the most validated instruments, the Knee Injury and Osteoarthritis Outcome Score (KOOS) provides a comprehensive assessment across five subscales: pain, symptoms, activities of daily living, sports and recreation function, and knee-related QoL [74, 75]. The KOOS is especially suited for athletic populations, as it captures sport-specific limitations and has demonstrated sensitivity to change following both conservative and surgical interventions [74, 76]. The International Knee Documentation Committee (IKDC) subjective knee form is another frequently used measure, providing a composite index of symptoms, function, and ability to perform sports activities [77, 78]. While both tools have demonstrated robust psychometric properties, their interpretation in elite athletes must consider ceiling effects and baseline expectations of physical performance that may differ from those in general populations [79]. For this reason, additional assessments such as the Tegner Activity Scale or sport-specific return-to-play criteria are often integrated into clinical evaluation protocols [80].

Beyond symptomatology and functional scoring, there is growing interest in the role of biochemical markers in detecting and monitoring early joint degeneration at the molecular level [81, 82]. Cartilage oligomeric matrix protein (COMP) is one of the most extensively studied biomarkers in this context, with elevated serum levels correlating with cartilage turnover and structural damage in both symptomatic and asymptomatic individuals [83]. Urinary C-terminal cross-linking telopeptide of type II collagen (CTX-II), which reflects collagen type II degradation, has also been proposed as a sensitive marker of cartilage catabolism [84, 85]. Both markers have demonstrated associations with radiographic progression and joint space narrowing in longitudinal studies; however, their variability and lack of disease specificity currently limit their routine clinical application [86, 87]. Nevertheless, in research settings and potentially in elite sports medicine clinics, these biomarkers may serve as adjunctive tools to stratify risk, monitor response to intervention, and identify athletes at a higher likelihood of rapid OA progression [83, 87]. Their integration with imaging findings and clinical metrics could support a more personalised approach to OA management in the athletic population [83].

Conservative management

The conservative management of early-onset knee OA in athletes requires a strategic and individualised approach to reduce symptoms, preserve joint function, and continue physical activity without accelerating structural degeneration [18, 88]. A cornerstone of this approach is patient education and self-management, as highlighted by international guidelines such as those from the Osteoarthritis Research Society International (OARSI) [89, 90]. Athletes must be informed of the nature of the disease, the biomechanical and inflammatory processes involved, and the rationale behind each therapeutic modality [12, 91]. Promoting active participation in treatment fosters adherence to rehabilitation protocols, encourages early recognition of symptom exacerbation, and supports the long-term preservation of joints [92]. Educational interventions often focus on load management, joint protection techniques, neuromuscular control, and psychological coping strategies to mitigate the impact of persistent discomfort [93].

Therapeutic exercise is a central component of conservative management, with robust evidence supporting its efficacy in alleviating pain and improving function in individuals with early-onset OA [94]. Structured programmes combining aerobic training, resistance exercises, flexibility, and proprioceptive work have demonstrated clinical benefits across a range of functional outcomes [95, 96]. In athletic populations, exercise regimens must be tailored to the specific biomechanical demands of the sport and adjusted according to the stage of disease and current symptomatology [97]. The primary goal is strengthening periarticular musculature, particularly the quadriceps and hip stabilisers, to reduce aberrant loading patterns and enhance joint stability [96].

At the same time, careful attention must be paid to avoid overuse, as excessive or high-impact training may perpetuate cartilage breakdown and synovial irritation [12]. Periodisation of training load, incorporation of cross-training and recovery phases, and regular reassessment of movement patterns are essential to ensure that therapeutic exercise remains protective rather than detrimental [98].

Load modification also encompasses body mass optimisation, a relevant but often overlooked factor in athletic populations [98]. Even small reductions in body weight have been associated with meaningful improvements in pain and functional scores, particularly in athletes with elevated lean mass or metabolic dysregulation [99]. Weight management strategies should be integrated into dietary counselling and strength conditioning programmes, with an emphasis on maintaining lean muscle mass while reducing excess mechanical stress on the joint [100, 101].

Pharmacological management serves a supportive role, primarily during symptomatic flares or in conjunction with rehabilitation [93, 98, 102]. Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used for their analgesic and anti-inflammatory effects, although prolonged use carries risks of gastrointestinal, renal, and cardiovascular adverse events [102]. Paracetamol may be considered for milder symptoms but has limited efficacy in moderate to severe cases [102]. The use of opioids is discouraged, particularly in athletes, given the risk of dependency, cognitive impairment, and reduced performance capacity [102].

Intra-articular therapies provide targeted symptom relief and are frequently employed in sports medicine [103, 104]. Corticosteroid injections offer short-term pain reduction, especially in the presence of synovitis, but repeated administration may accelerate cartilage deterioration [105, 106]. Viscosupplementation with hyaluronic acid has shown mixed results, though some studies suggest benefit in athletes with low-grade OA and high functional demands [107]. Platelet-rich plasma (PRP) has emerged as a promising biological option, with several randomised trials indicating moderate improvements in pain and function, likely given its anti-inflammatory and trophic effects on synovial tissue and cartilage [108–110]. Orthobiologics are increasingly used in early knee OA, yet the evidence remains difficult to translate into consistent practice. Reported outcomes are variable, partly because studies differ in product preparation, dosing, protocols, and patient selection, which limits comparability and reproducibility. Regulatory frameworks also vary across countries, affecting standardisation, quality control, and availability, and often confining these interventions to selected settings. In addition, long-term efficacy data are limited, and short-term symptom improvement does not necessarily indicate sustained structural benefit or delayed progression. For these reasons, biologic interventions should be positioned as adjuncts within a broader management plan, supported by careful selection of candidates and realistic expectation setting.

Additional non-pharmacological tools include functional braces, which may reduce joint stress and improve proprioception, particularly during dynamic activities [98, 111, 112]. Custom orthotics and insoles may correct lower limb alignment or redistribute plantar pressures, though evidence for their efficacy in OA remains limited [93, 113]. These adjuncts should be considered as part of a comprehensive management plan based on individual biomechanics and sport-specific needs [93].

Surgical options and regenerative interventions

When conservative strategies fail to control symptoms adequately or when structural deterioration progresses despite optimal non-operative care, surgical intervention may become necessary to preserve joint integrity and restore function in young athletic patients with early-onset knee OA [114, 115]. Surgical decisions in this population are inherently complex, as the aim is not merely symptom relief but also the restoration of biomechanics and the prevention of further degeneration, with particular attention to the long-term implications of each procedure on sporting performance and future treatment options [115].

Meniscal preservation plays a pivotal role in the surgical management of early-onset OA in athletes [39]. Given the meniscus's critical function in load distribution, shock absorption, and joint stability, meniscal repair is strongly favoured over resection whenever feasible [116]. Advances in arthroscopic techniques and suture anchors have increased repair success rates, particularly for peripheral longitudinal tears in vascularised zones [117, 118]. Even chronic and complex tears may be amenable to repair in carefully selected patients [119]. Meniscal transplantation is another option for symptomatic patients with prior meniscectomy and persistent compartmental overload, although outcomes are highly dependent on precise graft sizing, alignment correction, and patient compliance [120].

In cases of mechanical axis deviation, particularly varus alignment with medial compartment overload, high tibial osteotomy (HTO) represents a well-established option to redistribute joint loading and delay the progression of unicompartmental OA [121, 122]. HTO in active patients has shown favourable results in terms of pain relief, functional recovery, and return to sports, especially when combined with biological or chondral procedures [123]. The choice between opening wedge and closing wedge

techniques is influenced by patient anatomy, surgical experience, and concomitant procedures, though both can achieve satisfactory realignment when meticulously executed [122, 124]. HTO may also create a favourable environment for cartilage repair by offloading the affected compartment and reducing mechanical stress on grafted or regenerated tissue [125, 126].

Cartilage restoration techniques constitute a major focus in the management of focal chondral lesions in the early stages of OA. Autologous matrix-induced chondrogenesis (AMIC) has gained popularity due to its one-step nature, combining microfracture with the application of a collagen membrane to stabilise the clot and support chondral regeneration [127–129]. Matrix-assisted autologous chondrocyte implantation (mACI) remains the reference standard for large or complex defects, providing superior hyaline-like repair tissue and durable results in selected athletes [130–132]. The use of synthetic scaffolds, both cell-seeded and acellular, has expanded the available options, with various biomaterials under investigation to enhance cell migration, matrix deposition, and integration with native cartilage [127, 130]. The integration of regenerative therapies with mechanical correction, such as concurrent osteotomy, is increasingly advocated to maximise the biological potential of these interventions [125].

Total knee arthroplasty (TKA) is rarely indicated in young athletes with early-onset OA [133, 134]. However, it may be considered in exceptional cases of diffuse, symptomatic, and functionally limiting disease where all other measures have failed [135]. More commonly, unicompartmental knee arthroplasty (UKA) may be considered for isolated medial or lateral disease, provided ligamentous stability and normal patellofemoral kinematics are preserved [136, 137]. However, these decisions must be weighed against the need for revision in future decades and the impact on high-impact activities [136, 138]. Shared decision-making, accurate patient selection, and precise surgical execution remain the cornerstones of successful outcomes in this challenging group [138].

Return to sport and prevention of recurrence

The return to sport (RTS) following early-onset knee OA represents a critical phase in the management of athletic patients, demanding a coordinated effort that extends beyond symptom resolution and incorporates functional recovery, biomechanical optimisation, and psychological readiness [65, 88, 123]. One of the most challenging aspects in this process is the absence of universally validated criteria specific to early-onset OA, particularly in the context of high-performance athletes whose expectations and physical demands far exceed those of the general population [139]. A comprehensive RTS protocol must be grounded in both clinical judgement and objective metrics, ensuring that joint homeostasis has been re-established before resumption of high-load or pivoting activities [88].

Education plays a foundational role in facilitating a safe and sustainable RTS [140]. Athletes must be thoroughly informed about the nature of OA, its modifiable risk factors, and the rationale behind each therapeutic step, especially concerning training load, biomechanics, and tissue adaptation [12, 91]. Equally important is the involvement of the broader team ecosystem, including coaches, physiotherapists, athletic trainers, and strength and conditioning professionals [94, 141]. These stakeholders must understand the chronic and progressive potential of OA, adopting a long-term perspective that prioritises joint health over short-term performance. This is particularly relevant in competitive environments where pressure to resume activity prematurely may compromise structural healing and increase the likelihood of recurrence or progression.

Objective RTS criteria typically encompass the restoration of joint ROM, the absence of swelling or reactive synovitis, quadriceps and hamstring strength symmetry above 90% compared to the contralateral limb, and validated functional testing, such as single-leg hop tests, Y-balance assessment, and movement quality analysis [142, 143]. Proprioceptive control and neuromuscular coordination are crucial components, especially in sports that involve directional changes, deceleration, or landing mechanics [144, 145]. Athletes must demonstrate not only physical competence but also confidence in the affected limb, which is often influenced by psychological parameters, including fear of reinjury and self-efficacy [146]. PROMs such as the ACL-RSI or Tampa Scale for Kinesiophobia may provide valuable adjuncts to guide clinical decision-making [79, 123]. Sport-specific protocols are crucial for addressing the biomechanical and physiological demands of each discipline. In football, particular emphasis is placed on rotational stability, agility under load, and dynamic valgus control during high-speed manoeuvres [52, 147]. In basketball, vertical shock absorption, posterior chain integration, and ankle-knee-hip coordination during jumps and landings are prioritised [148]. Rehabilitation and reconditioning programmes should be tailored to simulate match scenarios progressively, beginning with linear movements and low-load drills, and advancing toward reactive, unpredictable tasks under fatigue [12]. This gradual exposure enables neuromuscular reprogramming and adaptation to real-game intensity, reducing the risk of biomechanical compensation or recurrence of joint symptoms [149].

To minimise the risk of recurrence and long-term progression, long-term strategies must include regular

biomechanical screening, early recognition of overload patterns, ongoing strength and mobility work, and periodised training plans with adequate recovery [12, 52]. Athlete monitoring technologies, such as inertial sensors and load management platforms, may help identify detrimental trends and facilitate timely intervention [150]. Ultimately, a successful RTS in the context of early-onset OA is not a singular event but a dynamic process requiring continuous collaboration between the athlete and the multidisciplinary team.

Clinical framework

To enhance clinical applicability, a pragmatic decision tree summarising the key diagnostic and therapeutic steps in the management of early-onset knee OA in athletes is presented in Fig. 1. The algorithm integrates symptom patterns, functional impairment, imaging findings when clinically relevant, and sport-specific demands, providing a structured framework to support everyday decision-making in sports medicine practice.

Current guidelines and evidence-based recommendations

The management of early-onset knee OA in athletic populations remains an area of evolving clinical practice, where evidence-based recommendations are still extrapolated mainly from studies conducted on older, sedentary individuals [18]. Major international bodies, such as the American Academy of Orthopaedic Surgeons (AAOS) and the Osteoarthritis Research Society International (OARSI), have issued comprehensive guidelines for the non-operative management of knee OA [93, 143, 151]. Yet, these documents rarely differentiate protocols based on age, aetiology, or physical activity level. The AAOS guidelines provide strong support for interventions such as supervised exercise, weight loss in overweight patients, and NSAIDs, while cautiously recommending intra-articular corticosteroids and viscosupplementation based on variable evidence [151]. OARSI has adopted a more patient-centred approach, stratifying recommendations according to comorbidity profiles and resource access [143]. Still, the specific needs of high-performance or recreational athletes with early-onset OA are not directly addressed in either document. This lack of specificity is problematic, as the pathophysiological mechanisms, functional expectations, and therapeutic responses in young active patients differ significantly from those of older cohorts. The traditional outcome measures used in OA research, such as the WOMAC index or radiographic joint space narrowing, may not be sensitive enough to detect meaningful changes in athletic individuals who present with early cartilage damage but retain a high level of functional capacity [79]. Furthermore, treatment

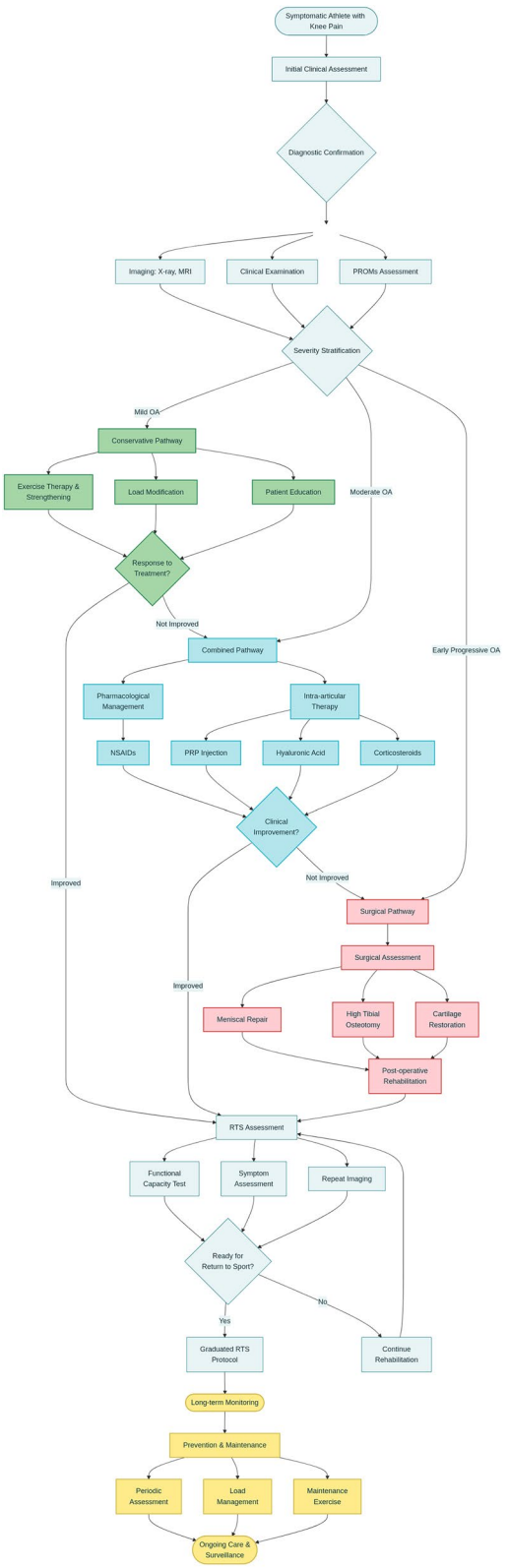

Fig. 1 The decision tree discussed in this review

priorities in athletes often extend beyond pain control to include optimisation of performance, prevention of structural deterioration, and safe RTS. These priorities require nuanced protocols that consider the biomechanics of the sport, previous injury history, and the psychological context of the patient [96, 149].

One of the most striking gaps in the literature is the paucity of high-quality randomised controlled trials focusing specifically on early-onset OA in athletic populations. Existing studies often group young and older patients or include heterogeneous OA phenotypes, limiting the external validity of their findings for sports medicine practice [26]. Moreover, most trials have short follow-up durations, which hampers the ability to evaluate disease-modifying effects of interventions or to determine whether symptom improvements translate into structural benefits over time. This limitation is particularly relevant when assessing the impact of biological treatments such as PRP or cell-based therapies, whose long-term efficacy remains uncertain despite promising short-term results [90, 105, 106, 109].

Another area of limited clarity concerns the integration of surgical and regenerative interventions within a structured treatment algorithm. While expert opinion supports early mechanical correction and chondral repair in select cases, there is a lack of standardisation regarding indications, timing, and combination strategies [122, 125, 127, 130]. Likewise, the role of neuromuscular rehabilitation, load monitoring, and sport-specific conditioning in delaying OA progression is widely acknowledged but poorly quantified in terms of evidence hierarchy [52, 144]. This evidentiary gap calls for targeted research efforts, including longitudinal studies with clearly defined athletic cohorts, sport-specific outcome measures, and imaging-based assessments of joint integrity. Developing such data would allow for the refinement of current guidelines and support a more tailored approach to managing early-onset OA in the athletic population, balancing scientific rigour with the practical realities of clinical sports medicine.

Future directions, protocol optimisation, education and research priorities

The management of early-onset knee OA in athletes stands at a critical juncture, with an expanding understanding of disease mechanisms but a persistent lack of tailored, evidence-based protocols that address the specific demands of this unique population. One of the most urgent research priorities is the development of well-designed longitudinal cohort studies that follow athletes from the time of injury or symptom onset through the subsequent stages of disease, with standardised collection of clinical, functional, imaging, and load-exposure data over time. Such cohorts would allow a more accurate definition of the natural history of early-onset knee OA in athletic populations and facilitate the identification of prognostic factors associated with progression or clinical stability. In parallel, sport-specific randomised controlled trials are needed to evaluate targeted interventions, particularly rehabilitation strategies, load-modification programmes, and selected injectable or surgical treatments, within homogeneous athletic subgroups. These trials should incorporate clinically meaningful and performance-oriented endpoints, including symptom trajectories, return-to-sport outcomes, and durability of functional improvement. Although short-term studies provide important preliminary efficacy signals, they remain insufficient to capture the fluctuating and load-dependent course of joint degeneration in athletes exposed to recurrent microtrauma and variable training demands, underscoring the need for longer follow-up and pragmatic study designs aligned with real-world sports medicine practice. Another critical area for future development lies in personalising treatment strategies based on identifiable biological and biomechanical phenotypes. The heterogeneity of early-onset OA in athletes, ranging from focal post-traumatic chondral lesions to global joint overload syndromes, calls for precision medicine approaches that go beyond the traditional one-size-fits-all model. For example, athletes with predominant synovial inflammation may derive greater benefit from biologic therapies targeting cytokine cascades. In contrast, those with isolated structural malalignment or altered kinematics may respond better to corrective surgery or neuromuscular retraining. Advanced phenotyping, incorporating machine learning algorithms and multidimensional data sources, could facilitate treatment stratification, reduce therapeutic failure, and guide shared decision-making processes in clinical settings. Technological innovation also holds significant promise for reshaping the diagnostic and monitoring landscape. Advanced imaging modalities, such as quantitative MRI, T2 mapping, and dGEMRIC, enable the early detection of biochemical cartilage changes before morphological damage becomes apparent on standard radiographs [36, 37, 41]. These tools offer the potential to track therapeutic response at a tissue level and may serve as surrogate endpoints in clinical trials. In parallel, the development of reliable and reproducible biomarkers, both systemic and local, could further refine disease staging and help identify athletes at high risk of rapid progression. Promising candidates include markers of cartilage degradation (CTX-II), synovial inflammation (IL-1β, TNF-α), and bone remodelling (osteocalcin, PINP), although their routine use remains limited by variability and lack of specificity [82, 84–87]. The incorporation of digital

health platforms, wearable technologies, and tele-rehabilitation into routine care may also transform how early-onset OA is monitored and managed. Real-time tracking of joint load, movement patterns, and adherence to therapeutic exercise could enhance individualisation of care, prevent overload episodes, and support athlete autonomy in disease self-management. Digital decision-support tools and patient-facing applications could also reinforce education, improve communication with multidisciplinary teams, and increase treatment adherence over time. Ultimately, the future of early-onset OA management in athletes depends not only on technological progress but also on a cultural shift toward preventive and proactive care models. This includes comprehensive education of athletes, coaches, and clinical teams on modifiable risk factors, early signs of degeneration, and the importance of evidence-based strategies for joint preservation [91, 92, 140]. Coordinated research efforts, interdisciplinary collaboration, and the development of sport-specific guidelines will be crucial in bridging current knowledge gaps and translating emerging science into clinically relevant practice.

Conclusion

Early-onset knee OA in athletes is a distinct clinical condition that requires management strategies different from those traditionally applied to age-related OA. Effective care should prioritise early recognition, accurate phenotyping, and individualised load management, integrating clinical findings, imaging, and functional assessment rather than relying solely on radiographic severity. For the sports physician, the practical challenge lies in balancing symptom control, joint preservation, and performance demands, particularly when guiding rehabilitation progression, injection timing, and return-to-sport decisions. This review emphasises that early-onset OA should not automatically be viewed as a contraindication to continued athletic participation, but rather as a modifiable condition that can be stabilised through structured exercise, biomechanical optimisation, and carefully selected adjunctive therapies. By translating current evidence into clinically applicable principles, this work aims to support informed decision-making in daily sports medicine practice, reduce the risk of disease progression driven by mismanaged loading or premature return to play, and ultimately help athletes maintain both joint health and long-term performance.

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Data availability
All relevant data are included in the article.

Declarations

Ethical approval and consent to participate
This study complies with ethical standards. Not applicable.

Consent for publication
Not applicable.

Competing interests
Prof. Dr. Filippo Migliorini is Deputy Editor of the European Journal of Medical Research. Luise Schäfer is a member of the Editorial Board of the European Journal of Medical Research. All other authors declare no conflict of interest.

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