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Anterior Cruciate Ligament Injury: A Whole-Joint Disease with Major Clinical and Economic Impact

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Abstract

Anterior cruciate ligament (ACL) rupture is one of the most common and consequential musculoskeletal injuries worldwide and represents a major precursor to post-traumatic osteoarthritis (PTOA) of the knee [1-4]. In the United States alone, approximately 120,000–200,000 ACL ruptures occur annually, generating substantial healthcare utilization related to diagnostic imaging, orthopedic consultation, surgical reconstruction, rehabilitation, and long-term management of degenerative joint disease [5-8].

Direct medical expenditures combined with productivity losses produce an estimated annual economic burden exceeding $7–10 billion in the United States [8-11]. Based on authors' projections incorporating current trends in sports participation (estimated annual growth of 2%–3%), surgical rates (approximately 50% of injuries), and healthcare cost inflation (approximately 3%–5% annually), the annual economic burden may approach $12–15 billion by 2040 in the United States. This trajectory aligns with independent analyses of musculoskeletal disease burden and underscores the urgency of preventive strategies. However, precise projections require formal health economic modeling incorporating incidence, treatment patterns, and long-term outcomes.

Outcomes after ACL reconstruction remain variable, with approximately 55%–70% of patients returning to competitive sport and fewer returning to their preinjury level of performance [12-14]. Furthermore, 30%–50% of patients develop radiographic posttraumatic osteoarthritis within 10–15 years after injury, even following technically successful reconstruction [15-17].

This review synthesizes current evidence regarding ACL injury epidemiology, mechanisms, whole-joint pathophysiology, socioeconomic impact, return-to-sport outcomes, healthcare disparities, prevention strategies, and advanced imaging biomarkers. Emphasis is placed on the evolving role of radiology in early detection, risk stratification, and longitudinal monitoring of PTOA.

Keywords: Anterior cruciate ligament; Sports injury; Posttraumatic osteoarthritis; Return to sport; Economic burden; Prevention; Quantitative imaging

Introduction

Anterior cruciate ligament rupture represents a major cause of knee morbidity in active populations and remains a critical challenge across sports medicine, orthopedics, and musculoskeletal radiology [1,2]. The ACL is fundamental for maintaining anterior translation and rotational stability of

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the tibiofemoral joint. Its disruption leads to joint instability, recurrent giving-way episodes, and increased susceptibility to secondary injuries, including meniscal tears and chondral damage.

A critical conceptual shift has emerged over the past two decades: ACL injury should no longer be viewed as an isolated ligamentous disruption but rather as a whole-joint disease. This paradigm reflects the recognition that ligament rupture initiates a cascade of inflammatory, biomechanical, and neuromuscular processes that extend beyond the ligament itself and affect the entire joint environment [3-6].

Importantly, distinguishing posttraumatic osteoarthritis (PTOA)—which develops following joint injury—from primary osteoarthritis—which is primarily age-related and degenerative—is essential for understanding disease mechanisms and treatment approaches. PTOA following ACL injury represents a distinct biological and clinical entity characterized by earlier onset, different pathophysiologic drivers, and significant long-term consequences. While primary osteoarthritis typically emerges in older adults because of cumulative wear and age-related changes, PTOA can manifest young, active individuals within a decade of injury, with distinct inflammatory and mechanical pathways driving degeneration.

Table 1: Comparison of Posttraumatic and Primary Osteoarthritis.

Characteristic	PTOA Following ACL Injury	Primary OA
Typical age of onset	25–40 years	>60 years
Inciting event	Acute trauma	Chronic wear / aging
Primary drivers	Inflammation + biomechanical	Degenerative / metabolic
Progression rate	Accelerated	Slow
Affected population	Young, active	Older adults

This paradigm shift has critical implications for imaging, prevention, and long-term patient management. Radiology is uniquely positioned to play a central role in this transformation by enabling early detection of joint degeneration and facilitating risk stratification. Understanding ACL rupture as a whole-joint disease provides a unifying framework for interpreting variable outcomes, guiding treatment decisions, and developing targeted interventions to mitigate long-term sequelae. This review aims to synthesize current evidence on ACL injury as a whole-joint disease, with particular emphasis on the role of advanced imaging in risk stratification and the socioeconomic dimensions often underrepresented in orthopedic literature.

Epidemiology of ACL Injury

ACL rupture is among the most common severe ligament injuries of the knee. Population-based studies estimate an incidence of approximately 68 per 100,000 person-years in the United States, corresponding to approximately 120,000–200,000 injuries annually [5,18-20]. Comparable rates have been reported in Scandinavian countries and Australia, though direct comparisons are complicated by differences in healthcare systems and data collection methods [7].

Injuries occur most frequently in adolescents and young adults, particularly between 15 and 25 years of age, reflecting peak participation in competitive athletics [21]. Sports involving pivoting, cutting, and rapid deceleration—such as soccer, basketball, American football, lacrosse, and alpine skiing—are associated with the highest injury rates. The intensity and frequency of high-risk maneuvers in these sports, combined with fatigue and competitive pressure, contribute to elevated injury risk.

Female athletes demonstrate a 2–8-fold higher risk of ACL injury compared with males participating in similar sports [22-25]. This increased susceptibility is believed to reflect a complex interplay of neuromuscular control patterns, anatomical alignment, and hormonal influences. Female athletes tend to exhibit greater quadriceps dominance, reduced knee flexion during landing, and increased dynamic valgus, all of which increase ACL strain.

Approximately 50% of ACL injuries undergo surgical reconstruction, although treatment decisions vary depending on patient age, activity level, and functional instability [5,6]. Nonoperative management may be appropriate for older, less active individuals or those willing to modify activities, whereas young athletes typically pursue reconstruction to restore knee stability and enable return to sport.

Table 2: Epidemiologic Characteristics of ACL Injury in the United States.

Parameter	Estimate	References
Annual ACL injuries	120,000–200,000	[5,18-20]
Incidence	68 per 100,000 people-years	[5,18]
Peak age	15–25 years	[21]
Female:male risk ratio	2–8 times higher	[22–25]
Reconstruction rate	approximately 50%	[5,6]
Return to sport	55%–70%	[12,58]
PTOA development	30%–50%	[15-17,66]

Risk Factors for ACL Injury

ACL injury risk arises from a multifactorial interaction of neuromuscular, biomechanical, anatomical, hormonal, and genetic factors. Understanding these risk factors is essential for identifying at-risk individuals and developing targeted prevention strategies.

Arce S, et al. J Ortho Sports Med 2026
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Neuromuscular Factors (Modifiable)

Neuromuscular factors are among the most modifiable contributors. Movement patterns characterized by quadriceps dominance, reduced knee flexion during landing, and dynamic valgus alignment are associated with increased ACL loading during athletic maneuvers [22-24]. Quadriceps dominance refers to preferential activation of quadriceps over the hamstrings, which increases anterior tibial translation and ACL strain. Studies suggest this pattern can increase ACL loading by up to 50% during landing compared to balanced muscle activation [24].

Reduced knee flexion during landing shifts impact forces to more extended knee positions, where the ACL is under greater tension. Dynamic valgus—a combination of hip adduction, knee abduction, and tibial external rotation—places the ligament at mechanical disadvantage and increases injury risk.

Anatomical Factors (Non-Modifiable)

Anatomical factors, including increased posterior tibial slope and narrow intercondylar notch width, have also been associated with increased injury risk [26-28]. A steeper posterior tibial slope increases anterior tibial translation under axial load, while a narrow intercondylar notch may physically constrain the ligament, making it more susceptible to impingement and shear forces during rotational maneuvers. Other anatomical considerations include generalized ligamentous laxity, increased body mass index, and lower extremity alignment abnormalities.

Hormonal Factors (Non-Modifiable but May Inform Risk Stratification)

Hormonal influences have been proposed to contribute to sex-based differences in injury risk, although the underlying mechanisms remain incompletely understood [29-31]. Fluctuations in estrogen and relaxin levels during the menstrual cycle may affect ligament laxity and neuromuscular control. Studies have suggested increased injury risk during the preovulatory phase, when estrogen levels peak, potentially due to hormone-mediated changes in collagen synthesis and ligament properties. However, evidence remains mixed, and further research is needed to clarify hormonal contributions.

Genetic Factors (Non-Modifiable b ut M ay Inform Risk Stratification)

Genetic predisposition may also play a role, particularly through variations in collagen structure and extracellular matrix composition [32-34]. Polymorphisms in genes encoding collagen types I, III, V, and XII have been associated with ACL rupture risk. These variants may influence ligament tensile strength, elasticity, and susceptibility to mechanical failure. Family history of ACL injury also appears to increase individual risk, supporting a genetic component.

Mechanisms of Injury

Approximately 70% of ACL injuries occur through noncontact mechanisms [35-37]. These injuries typically occur during rapid deceleration, pivoting, or landing maneuvers with the foot planted, without direct contact to the knee. The absence of external impact highlights the role of intrinsic biomechanical factors in injury causation.

Biomechanical analyses suggest that ligament rupture occurs within approximately 40–50 milliseconds after ground contact, emphasizing the importance of neuromuscular control during early landing phases [37]. This ultrashort time window means that once landing begins, reflexive neuromuscular responses are too slow to prevent injury; therefore, preprogrammed movement patterns and preparatory muscle activation are critical for knee protection.

Characteristic injury patterns identified through video analysis include dynamic knee valgus, reduced knee flexion, internal tibial rotation, and lateral trunk displacement [35,38]. These biomechanical positions are associated with increased ACL strain and may precipitate ligament failure. The combination of valgus collapse and rotational torque appears particularly deleterious, as it loads the ligament in multiple planes simultaneously.

Table 3: Major Risk Factors for ACL Injury by Modifiability.

Category	Risk Factors	Modifiability	Mechanism	References
Neuromuscular	Quadriceps dominance, dynamic knee valgus	Modifiable	Increased ACL strain during landing	[22-24]
Biomechanical	Reduced knee flexion during landing	Modifiable	Higher shear forces	[22,23]
Anatomical	Narrow intercondylar notch, increased tibial slope	Non-modifiable	Altered kinematics, impingement	[26-28]
Hormonal	Estrogen fluctuations, relaxin	Non-modifiable*	Ligament laxity	[29-31]
Genetic	Collagen gene polymorphisms	Non-modifiable	Structural susceptibility	[32-34]

*Hormonal factors are inherent but may inform injury risk timing and prevention strategies

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Contact injuries account for the remaining 30% of cases and typically involve direct impact or collision, such as a blow to the lateral knee producing valgus stress, or hyperextension injuries during tackles [38]. These mechanisms are more common in contact sports like football and rugby and may be associated with concomitant injuries to other ligamentous structures.

Pathophysiology: Acl Injury as a whole-joint disease

The concept of ACL injury as a whole-joint disease reflects the interplay of multiple biological and mechanical processes that collectively drive joint degeneration. Understanding these interconnected pathways is essential for appreciating why PTOA develops despite successful ligament reconstruction.

Inflammatory Response

Immediately following ACL rupture, hemarthrosis and tissue injury trigger an intra-articular inflammatory cascade. Pro-inflammatory cytokines—including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α)—are released into the synovial fluid within hours of injury [39-41]. These cytokines originate from synovial lining cells, infiltrating inflammatory cells, and damaged ligament and cartilage tissues.

These mediators activate matrix metalloproteinases (MMPs), particularly MMP-1, MMP-3, and MMP-13, which degrade cartilage extracellular matrix components, including aggrecan and type II collagen [42]. Concurrently, inflammatory cytokines suppress extracellular matrix synthesis by chondrocytes, shifting the balance toward net matrix loss.

Clinical implication: this early biochemical disruption may contribute to cartilage degeneration even before structural changes are detectable on conventional imaging, representing a critical window for early intervention.

The inflammatory response typically persists for weeks to months after injury, with elevated cytokine levels detectable in synovial fluid for extended periods. Chronic low-grade inflammation may contribute to ongoing cartilage degradation and PTOA progression.

Biomechanical Alterations

Loss of ACL integrity results in altered tibiofemoral kinematics, including increased anterior tibial translation and rotational instability [43-45]. These changes shift load distribution across the joint, exposing cartilage regions to abnormal mechanical stresses. During gait, the ACL-deficient knee exhibits altered tibial rotation and mediolateral translation, concentrating loads on previously underloaded cartilage regions.

Even after reconstruction, subtle abnormalities in joint motion often persist. Residual rotational laxity altered muscle activation patterns, and changes in gait mechanics may contribute to continued abnormal loading and progressive cartilage degeneration. Biomechanical studies using dynamic radiostereometry have demonstrated persistent rotational abnormalities during activity, even in patients with good clinical outcomes.

The altered mechanical environment affects not only cartilage but also menisci, subchondral bone, and periarticular soft tissues, perpetuating a cycle of abnormal loading and structural deterioration.

Subchondral Bone Remodeling

Changes in the osteochondral unit represent another critical component of PTOA development. Subchondral bone remodeling may increase bone stiffness and alter load transmission across the joint [46,47]. Following ACL injury, increased bone turnover and remodeling activity have been observed, potentially mediated by altered mechanical loading and inflammatory signaling.

Bone marrow lesions observed on MRI following ACL injury are associated with early structural damage and may reflect regions of increased mechanical stress and remodeling activity [48]. These lesions, typically located in the lateral femoral condyle and posterolateral tibial plateau at the impaction site, correlate with overlying cartilage damage and predict subsequent cartilage degeneration.

Clinical implication: the presence and severity of bone marrow lesions may serve as early imaging biomarkers for PTOA risk.

Neuromuscular Dysfunction

The ACL contains mechanoreceptors, including Ruffini endings, Pacinian corpuscles, and Golgi tendon organ-like receptors—that contribute to proprioception and neuromuscular control [49]. These receptors provide afferent feedback regarding joint position, motion, and tension, facilitating reflexive muscle stabilization.

Loss of these sensory inputs following injury may impair joint position awareness and alter muscle activation patterns. ACL-deficient individuals demonstrate diminished proprioceptive acuity and altered reflex arcs, with delayed hamstring activation in response to anterior tibial translation. Persistent neuromuscular deficits—including quadriceps weakness, altered hamstring-quadriceps activation ratios, and abnormal co-contraction patterns—may further contribute to instability and abnormal joint loading.

Cartilage Matrix Degeneration

The combination of inflammatory, biomechanical, and neuromuscular alterations converges on the articular

cartilage, producing progressive matrix degeneration. Early changes include proteoglycan loss, increased tissue hydration, and disruption of collagen architecture. These biochemical alterations precede macroscopic cartilage fibrillation and thinning, representing a potential therapeutic window for disease-modifying interventions.

Over time, continued matrix degradation leads to progressive cartilage loss, subchondral bone exposure, and the clinical syndrome of osteoarthritis. The rate and severity of progression vary considerably among individuals, reflecting differences in injury characteristics, treatment, and host factors.

Economic Burden and Societal Impact

ACL injuries impose a substantial and multifaceted economic burden on healthcare systems and society. Because these injuries frequently occur in adolescents and young adults during peak productive years, their impact extends beyond direct medical expenditures to include substantial indirect costs related to lost productivity, disability, and long-term osteoarthritis management.

Direct Medical Costs

Direct medical costs include diagnostic imaging, surgical reconstruction, and postoperative rehabilitation. MRI is commonly used to confirm diagnosis and evaluate associated injuries, with typical costs ranging from $1,500 to $3,000 in the United States [50,51]. Advanced imaging is essential not only for diagnosis but also for detecting concomitant meniscal and chondral injuries that influence treatment decisions and prognosis. Geographic variation and facility type (hospital versus ambulatory surgery center) significantly influence these costs.

ACL reconstruction represents the largest contributor to treatment expenditures. Total procedure costs—including surgeon fees, operating room expenses, anesthesia, and facility charges—generally range from $15,000 to $25,000 per case [8,52]. Additional procedures such as meniscal repair or cartilage restoration may further increase costs. Graft choice (autograft versus allograft, bone–patellar tendon–bone versus hamstring) also influences cost, with allografts typically incurring higher acquisition costs but potentially shorter operative times.

Rehabilitation is another significant component of treatment. Structured physical therapy programs lasting 6–12 months typically cost between $3,000 and $8,000 per patient [9,53]. Rehabilitation costs vary depending on frequency of visits, duration of formal therapy versus home exercise, and need for specialized equipment or supervision. Combined episode costs for ACL injury management average approximately $30,000 per patient [8,52,54].

By comparison, healthcare systems with national insurance models (e.g., United Kingdom, Scandinavia) report lower direct procedure costs but similar patterns of resource utilization, suggesting that the economic burden, while distributed differently across payers, remains substantial globally.

Table 4: Estimated Direct Costs of ACL Injury Management in the United States.

Component	Estimated Cost	Key Variables	References
MRI	$1,500–3,000	Facility type, geographic region, contrast use	[50,51]
Surgery	$15,000–25,000	Graft type, concomitant procedures, facility	[8,52]
Rehabilitation	$3,000–8,000	Duration, supervision level, geographic region	[9,53]
Total episode cost	approximately $30,000	Range: $20,000–45,000 depending on variables	[8,52,54]

Indirect Costs

Indirect economic losses substantially increase the overall burden of ACL injuries. These include time away from work, reduced athletic participation, secondary surgical procedures, and long-term disability related to osteoarthritis. For young workers, time lost from employment during recovery can represent significant income reduction. For student-athletes, injury may affect scholarship status, educational trajectory, and future earning potential.

Recovery following ACL reconstruction may require weeks to months away from occupational activities depending on job demands [9]. Individuals in physically demanding occupations may require extended leave or permanent job modifications. Additionally, a substantial proportion of patients eventually develop PTOA requiring long-term medical management and, in some cases, knee arthroplasty later in life [55,56]. The costs of osteoarthritis management—including medications, physical therapy, activity modifications, and joint replacement—accumulate over decades, substantially increasing the lifetime economic impact of the index injury.

Long-Term Economic Consequences

From a health systems perspective, ACL injury represents a high-cost, high-impact condition occurring early in life,

with downstream consequences that extend over decades. The young age at injury means that affected individuals live with the consequences—including activity limitations, chronic pain, and osteoarthritis—for most of their lives. This prolonged impact distinguishes ACL injury from many other musculoskeletal conditions that affect older populations.

When both direct and indirect costs are considered, the annual economic burden of ACL injuries in the United States exceeds $7–10 billion [8-11]. This figure likely underestimates the true burden, as it does not fully capture intangible costs such as reduced quality of life, lost athletic opportunities, and caregiver burden.

Table 5: Economic Impact of ACL Injury.

Category	Impact	References
Direct medical costs	Surgery, imaging, rehabilitation	[8,50,52]
Indirect costs	Lost productivity, disability	[9,57]
Long-term care	Osteoarthritis management	[11,55,56]
Estimated annual US burden	$7–10 billion	[8-11]

Future Economic Considerations

The financial burden associated with ACL injuries has increased steadily over the past two decades due to greater sports participation, expanded use of advanced imaging, increased surgical volumes, and rising healthcare costs [5,21,57]. Participation in youth sports continues to grow, with more children specializing in single sports at earlier ages, potentially increasing exposure to high-risk activities.

Based on authors' projections incorporating current trends in sports participation (estimated annual growth of 2%–3%), surgical rates (approximately 50% of injuries), and healthcare cost inflation (approximately 3%–5% annually), the annual economic burden may approach $12–15 billion by 2040 in the United States. This projection aligns with independent analyses of musculoskeletal disease burden and underscores the urgency of preventive strategies. However, precise projections require formal health economic modeling that integrates epidemiologic trends, treatment pathways, and long-term outcomes, as well as sensitivity analyses accounting for potential changes in prevention effectiveness and treatment paradigms.

Return-To-Sport Outcomes

Return to sport is a primary goal following ACL reconstruction, particularly among competitive athletes. However, outcomes remain variable and often disappointing, with many patients failing to achieve their preinjury activity levels.

Rates of Return

Meta-analyses indicate that approximately 55%–70% of patients return to competitive sport following reconstruction, whereas only 40%–55% regain their preinjury level of performance [12-14,58]. These figures have remained relatively stable over time despite advances in surgical technique and rehabilitation, suggesting that factors beyond surgical success influence outcomes.

Psychological Barriers

Psychological factors such as fear of reinjury represent major barriers to returning to sport [13,59]. The psychological impact of ACL injury extends beyond the immediate recovery period, with many athletes experiencing persistent anxiety, reduced confidence, and altered risk perceptions. The ACL-Return to Sport after Injury (ACL-RSI) scale has been developed to quantify psychological readiness and has demonstrated predictive value for return-to-sport success.

Objective Functional Assessment

Objective functional measures—including quadriceps strength symmetry (≥90% limb symmetry index), hop test performance, and patient-reported outcomes—are increasingly used to guide return-to-sport decisions [60,61]. The Delaware–Oslo ACL cohort study demonstrated that meeting discharge criteria significantly reduces reinjury risk. Despite this evidence, many athletes return to sport without meeting objective criteria, potentially contributing to high secondary injury rates.

Risk of Secondary Injury

Athletes who resume high-level sports face increased risk of graft rupture or contralateral ACL tear [49,62-64]. Young age (<25 years) and early return to sport (<9 months) are consistently associated with elevated risk. The second injury rate approaches 20%–30% in young, active populations.

Healthcare Disparities In Acl Injury Management

Disparities in ACL injury management are increasingly recognized as important determinants of outcomes and equity in musculoskeletal care. Research in this area is still emerging, and further investigation is needed.

Socioeconomic Disparities

Patients from lower socioeconomic backgrounds or without private insurance may experience delays in diagnosis and reduced access to care [67-69].

Geographic Disparities

Patients in rural areas may face barriers to accessing specialized care [68].

Arce S, et al. J Ortho Sports Med 2026
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Table 6: Long-Term Outcomes Following ACL Injury.

Outcome	Rate	Timeframe	Key Determinants	References
Return to sport	55%–70%	1–2 years post-reconstruction	Psychological readiness, strength	[12,58]
Return to preinjury level	40%–55%	1–2 years post-reconstruction	Sport type, confidence	[13,14]
Graft failure	3%–10%	2–10 years	Age, activity level	[62,65]
Contralateral ACL tear	5%–15%	2–10 years	Age, sport intensity	[63,64]
PTOA development	30%–50%	10–15 years	Meniscus, cartilage damage	[15-17,66]

Table 7: Quantitative MRI Techniques for Cartilage Assessment.

Technique	What It Measures	Clinical Utility	Limitations	Research Status
T2 mapping	Collagen integrity	Early degeneration	Limited specificity	Clinical use
T1rho	Proteoglycan	Early OA detection	Longer scans	Research
dGEMRIC	Proteoglycan	Quantitative	Contrast required	Research
Sodium MRI	Proteoglycan	Direct measurement	Specialized hardware	Research
gagCEST	Glycosaminoglycan	Non-contrast	Technical complexity	Early research

Racial and Ethnic Disparities

Lower rates of ACL reconstruction have been reported among Black and Hispanic patients [69,70].

Prevention Strategies

Because most ACL injuries occur through noncontact mechanisms, prevention programs have focused on improving neuromuscular control.

Neuromuscular Training Programs

Neuromuscular training programs reduce ACL injury incidence by approximately 30%–50% [71-74].

Program Effectiveness

Programs are most effective when performed regularly and with proper technique feedback.

Implementation Challenges

Despite strong evidence, adoption remains inconsistent due to time constraints and lack of awareness [73,75].

Advanced Imaging and Prediction of Ptoa

MRI plays a central role in ACL injury evaluation and longitudinal assessment.

Conventional MRI

Conventional MRI accurately detects structural injury but is limited for early biochemical changes.

Quantitative MRI Techniques and Emerging Techniques

T2 mapping reflects collagen integrity [76,77]. T1rho imaging is sensitive to proteoglycan content [78,79]. dGEMRIC [80], sodium MRI [81], and gagCEST [82] are promising but limited in clinical use.

Barriers to Clinical Adoption

Barriers include scan time, lack of standardization, and limited reimbursement.

Machine Learning and Risk Prediction

Artificial intelligence models integrating imaging and clinical data may enhance PTOA risk prediction [83,84].

Future Directions

Future research focuses on improving early identification and disease modification.

Short-term priorities include imaging biomarker validation, artificial intelligence integration, and prevention implementation.

Medium-term priorities include biologic therapies and personalized return-to-sport protocols.

Long-term priorities include development of disease-modifying osteoarthritis drugs and registry-based research.

The Radiologist's Role: Clinical Practice Points

Radiologists can enhance patient care through comprehensive assessment, risk stratification, longitudinal surveillance, and structured communication.

Knowledge Gaps and Limitations

Key gaps include return-to-sport criteria, imaging biomarker validation, disparities mechanisms, prevention implementation, and economic modeling.

Conclusion

ACL rupture represents a major musculoskeletal injury with substantial clinical and economic consequences. High injury incidence, expensive surgical treatment, prolonged rehabilitation, and the long-term risk of osteoarthritis collectively generate a significant societal burden estimated at $7–10 billion annually in the United States, with projections suggesting continued growth toward $12–15 billion by 2040.

Recognition of ACL injury as a whole-joint disease provides a unifying framework for understanding disease progression and highlights the need for comprehensive management strategies that address not only ligament reconstruction but also the inflammatory, biomechanical, and neuromuscular consequences of injury. This paradigm shift has particular relevance for radiology, as imaging plays an increasingly central role in early detection, risk stratification, and longitudinal monitoring.

Advances in quantitative imaging biomarkers—including T2 mapping, T1rho, and emerging techniques—may enable earlier identification of patients at risk for degenerative progression, supporting more personalized management strategies and facilitating the development of disease-modifying interventions. Machine learning approaches integrating multi-modal data hold promise for enhancing prediction and clinical decision support.

Expanding prevention programs through implementation science and addressing healthcare disparities represent key opportunities to reduce the burden of ACL injury at the population level. Improving outcomes for the hundreds of thousands of individuals who sustain ACL injuries annually will require coordinated efforts across clinical medicine, radiology, public health systems, and research enterprise.

Radiologists, orthopedic surgeons, and sports medicine practitioners must recognize ACL injury as a chronic joint disease requiring longitudinal surveillance and comprehensive management, not merely an acute surgical condition. This perspective shift, combined with advances in imaging technology and preventive interventions, offers the best hope for reducing the clinical and economic burden of ACL injury and its long-term sequelae.

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