Osteoarthritis in the Anterior Cruciate Ligament-deficient Knee—Epidemiology, Biomechanics, and Effects on the Meniscus and Articular Cartilage

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Abstract

Anterior cruciate ligament (ACL) tears are one of the most commonly encountered and operated on problems in sports medicine. The natural history of the ACL-deficient knee and its association with the long-term development of osteoarthritis remains a topic of interest in the orthopaedic surgery literature. Recent long-term clinical studies have confirmed that patients with chronic ACL deficiency have an increased risk for secondary meniscal damage and chondral injury; however, a clear link between ACL deficiency and the development of osteoarthritic changes in the knee has yet to be documented. This article reviews the current understanding of the association between ACL deficiency and osteoarthritis, focusing on the biomechanics of ACL deficiency and its impact on meniscal and articular cartilage injury.

Keywords

Anterior cruciate ligament (ACL), osteoarthritis, meniscus, articular cartilage, biomechanics

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Injuries to the anterior cruciate ligament (ACL) are common, with well over 300,000 ACL ruptures occurring annually in the US.[1–3] The majority of ACL injuries are related to athletic activities and affect patients between 15 and 45 years of age.[4] Treatment options following an ACL injury include surgical reconstruction or rehabilitation without ligament reconstruction. For patients who choose non-operative management of their ACL injury, the natural history of the ACL-deficient knee and its association with the long-term development of osteoarthritis continues to be a subject for debate within orthopaedic surgical literature.

The incidence and progression of osteoarthritis following ACL rupture have yet to be clearly defined. Some authors believe that in the absence of a functioning ACL, repetitive subluxation during weight-bearing activities results in meniscal tears, articular cartilage injury, and stretching of secondary capsular and ligamentous restraints.[5–8] While recent long-term clinical studies have confirmed that patients with chronic ACL deficiency have a significantly increased risk for secondary meniscal damage and chondral injury[9–11] and that patients treated with meniscectomy have an accelerated progression of osteoarthritis,[12–14] a clear link between ACL deficiency and the development of osteoarthritic changes in the knee has yet to be documented.

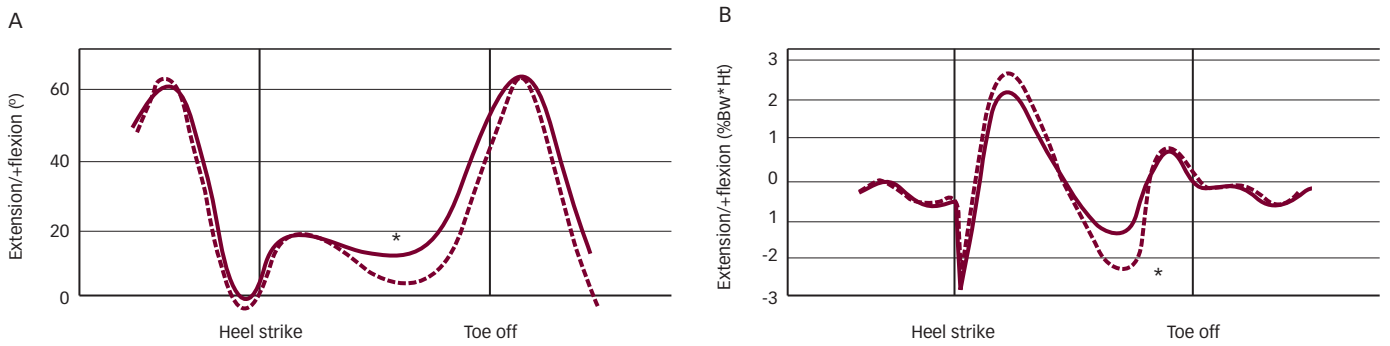
This article reviews the current understanding of the association between ACL deficiency and the development of osteoarthritic changes within the knee, focusing on the biomechanics of ACL deficiency and its impact on meniscal and articular cartilage injury.

Epidemiology

The ACL is the most commonly injured ligament in the knee, with approximately 70% of tears occurring during participation in athletic activities,[15] typically secondary to a non-contact, deceleration/pivoting mechanism.[1,15] While overall more males sustain ACL ruptures than females, when controlled for the number of male to female participants in athletics, females have been shown to have a two to eight times greater incidence of ACL injury than their male counterparts.[16] The majority of ACL ruptures occur in patients between 15 and 45 years of age, a 30-year time period that includes approximately 47% of the US population.[15] It has been estimated that one ACL rupture will occur for every 1,500 player-hours spent participating in sports such as soccer, basketball, football, and skiing,[17] equating to an annual incidence of one injury per 1,750 Americans.[15] Over 300,000 ACL reconstructions are performed each year in the US.[3] At an approximate cost of $17,000 per ACL procedure coupled with an estimated cost of $2,000 per ACL injury patient treated non-operatively, the annual cost of management of ACL injuries utilizes well over 1 billion US healthcare dollars.[15,18]

In up to 80% of cases, rupture of the ACL is associated with injury to other structures within the knee, including the menisci, collateral ligaments, and articular cartilage.[19,20] The pattern of concomitant injuries is influenced by the force and mechanism of the traumatic event.[19] Rupture of the medial collateral ligament is the most common associated injury, occurring in up to 60–70% of cases, while meniscal tears (lateral more commonly than medial) have been documented to

Figure 1: Sagittal Plane Knee Motion



*Comparison of sagittal plane knee motion (A) and sagittal plane knee moment (B) between the anterior cruciate ligament (ACL)-deficient group (solid line) and the control group (dashed line) during walking. The peaks represent the average of each group. *Significant difference (p<0.05) between the two groups. Source: Patel et al., 2003.*[33]

occur in 50% of acute ACL tears.[20] Acute chondral injuries have been reported to occur in 20% of ACL ruptures, but many authors believe that the commonly seen patterns of bone bruising, termed post-traumatic bone marrow lesions, represent occult cartilage damage occurring secondary to the compressive forces on the joint surfaces at the time of the ACL tear.[21–23]

The reported incidence of osteoarthritis following a non-operatively treated ACL injury ranges from 10 to 95% in the orthopaedic surgery literature.[9,19,20,24] This wide variation is due, in part, to the fact that most long-term follow up studies do not differentiate between isolated ACL ruptures and those occurring in association with meniscal or chondral injuries. In a series of 49 patients whose ACL injuries were managed non-operatively, Kannus and Jarvinen reported a 70% incidence of radiographic gonarthrosis eight years after injury.[25] Nebelung and Wuschech evaluated the long-term outcomes of 19 ACL-deficient athletes and found that at 20 years following the injury, 18 of 19 (95%) had evidence of severe osteoarthritis on arthroscopic examination.[9] In a clinical evaluation of 26 female soccer players who sustained complete ACL rupture managed conservatively, Lohmander et al. reported that 11 of 26 (42%) had radiographic evidence of osteoarthritis 12 years after injury.[26] In this study, no significant difference was seen in the incidence of post-traumatic degenerative changes between these non-operatively treated patients and a group of 42 players who were treated with ACL reconstruction. Von Porat et al. performed a similar evaluation of 205 male soccer players with ACL tears and reported a 78% incidence of radiographic gonarthrosis at 14 years following injury, with no difference found between those treated with operative reconstruction and those managed non-operatively.[27]

Biomechanics of Anterior Cruciate Ligament Deficiency

Anterior translation of the tibia on the femur occurs during normal knee function as a result of the force applied on the tibia by quadriceps contraction. This anterior translation is primarily restricted by an intact ACL, especially in the motion sector from 45° of flexion to full knee extension.[20,28,29] In addition to acting as the primary restraint to tibial translation in the sagittal plane, the ACL functions as a major secondary restraint to internal rotation and as a minor restraint to external rotation.[1] Tension created in the ACL coupled with the joint compressive forces occurring with weight-bearing increase the stability of the knee and optimize load transfer during physiological activity.[20]

The kinematics of the tibio-femoral articulation change following ACL rupture, particularly in the sagittal plane. Excess anterior–posterior laxity of the knee occurs, predominantly involving the lateral compartment, with anterior subluxation of the lateral tibial plateau relative to the lateral femoral condyle.[2] In a study utilizing dynamic magnetic resonance imaging (MRI) to evaluate tibio-femoral translations during knee range of motion, Logan et al. found that with ACL deficiency, the lateral tibial plateau was persistently subluxed anteriorly throughout the arc of flexion from 0 to 90° with no significant change in the medial compartment articulation.[30] This relative internal rotation of the tibia allows the posterior aspect of the lateral femoral condyle to approach the posterior limit of the tibial plateau during activity, potentially reproducing the pivot-shift phenomenon or sensation of giving way.

In response to increased anterior translation of the tibia relative to the femur, patients with an incompetent ACL attempt to cope by altering muscular forces about the knee. Gait studies have demonstrated that ACL-deficient patients use stronger contractions of their hamstrings in an attempt to pull the tibia posteriorly or limit the extent of quadriceps contraction to avoid increased anterior tibial translation (a quadriceps-avoidance gait).[2,31,32] In a review of the gait patterns of 18 ACL-deficient patients, Patel et al. reported that 72% demonstrated a significantly reduced mid-stance knee flexion angle, allowing them to reduce the demand placed on the quadriceps during the stance phase of gait.[33] Additionally, the authors found that the remaining 28% of patients tended to lean forward during ambulation, increasing their external hip flexion moment during mid-stance, similarly reducing quadriceps demand. Wexler et al. compared the gait of 30 ACL-deficient patients with 30 controls and found that patients without a functioning ACL walked with increased knee extension angles during terminal stance secondary to significantly decreased external knee flexion moments.[34]

Other gait adaptations have been identified in patients lacking a functional ACL. In their comparison of the gait patterns of 13 ACL-deficient knees, 21 ACL reconstructions, and 10 controls, Georgoulis et al. found that the ACL-deficient patients had significantly increased

tibial rotational motion during the swing phase compared with the other two patient populations.[35] Where external rotation of the tibia was observed in both the controls and the ACL reconstruction patients, those in the ACL-deficient group demonstrated a mean 10º of internal rotation during initiation of the swing phase. Based on their findings, the authors concluded that patients with ACL deficiencyΩexperience repeated episodes of rotational instability during walking.

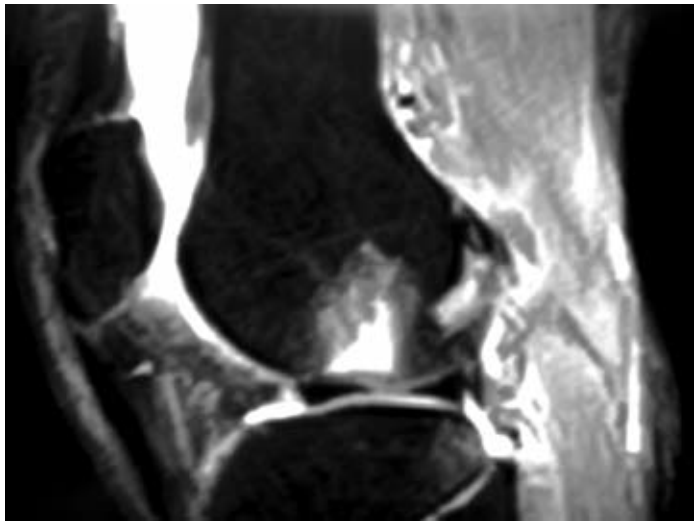
It has been postulated that the combination of excessive anterior tibial translation and rotational instability during weight-bearing activities in patients with ACL deficiency predisposes them to the development of meniscal and chondral injuries, with the end result being osteoarthritis. Abnormal translational and rotational motions following ACL injury may lead to increased shear stresses on the menisci and the application of increased weight-bearing loads to regions of the articular surface that were not functionally loaded previously (unconditioned cartilage regions).[2,36] Additionally, in contrast to the constantly changing contact points of the tibio-femoral articulation in a normal knee (rollback), the femur in an ACL-deficient knee may remain in contact with a portion of the tibial articular surface, resulting in point-loading with non-physiological loads leading to chondral damage and eventual degeneration.[20] Support for these hypotheses was reported by Andriacchi et al. in their finite element analysis of the kinematic changes associated with ACL deficiency.[36] The authors found that the relative internal rotation of the tibia secondary to ACL injury resulted in increased cartilage loss compared with intact controls, especially in the medial compartment secondary to a shift in load-bearing to thinner regions of articular cartilage.

Articular Cartilage Injuries

Acute ACL ruptures are known to be associated with chondral and subchondral damage occurring at the time of injury.[37,38] As seen on MRI studies, these lesions appear as bone bruises with abnormal signal at the terminal sulcus of the lateral femoral condyle (LFC) and the posterolateral corner of the tibial plateau (see *Figure 2*).[39–41] While these areas of bone bruising have not been described in detail histologically, they are felt to be consistent with microtrabecular injury with associated hemorrhage, edema, and inflammation.[39,40] Additionally, cartilage lesions can exist without associated bone bruising, identified both by MRI and macroscopically at the time of surgery. During knee arthroscopy, cartilage injuries can appear as chondral softening, chondral fractures, impaction lesions, or chondral flaps, and occur in the lateral compartment 70% of the time.[42,43]

The development of acute lateral compartment injury is thought to be due to lateral subluxation of the tibial articular surface impacting the LFC at the moment of the ACL tear. Compressive forces across the chondral and subchondral surfaces produce the so-called 'kissing' lesion seen on MRI.[40,41] Vellet et al.[44] reviewed 120 patients with this pattern of injury and classified them into three different types. Type 1, or the reticular pattern, involved medullary edema and comprised 70% of lesions. Typically, these lesions were found to resolve six to 12 months after injury on follow-up MRI. Type 2, or a geographical pattern of bone bruising, was defined as a localized signal with contiguity to the subjacent articular surface and was seen in 25% of acute injuries. These were noted to have persistent changes on follow-up MRI at six to 12 months in 66% of cases. Type 3 lesions (found in less than 5% of the patient cohort) were defined as disruption or depression of the normal contour of the articular surface. The long-term outcome of these persistent abnormalities remains unclear. Faber et al. noted on six-year follow-up that 60% of patients with initial bone bruises had persistent abnormalities on MRI.[43]

Figure 2: T_2-weighted Sagittal Magnetic Resonance Imaging-detected Bone Bruise in a Patient with Acute Anterior Cruciate Ligament Injury



With chronic ACL-deficient knees, further chondral injury may occur as a result of repetitive abnormal shear stresses across the cartilage surface.[45] Additionally, alterations in the biochemical environment occur in association with the ACL-injured knee and may contribute to progressive chondral degeneration. Changes in metalloproteinases, collegenases, interleukins (ILs), tumor necrosis factor-alpha (TNF-α), and keratin sulfate levels have been described.[46–48] Recently, Marks et al. found IL-1B and TNF-α levels to be elevated in knees with ACL rupture, with the extent of elevation correlating with the amount of chondral damage present.[5] Further research is needed to define the changes in the articular cartilage microenvironment following ACL injury and how these biochemical alterations affect the development of osteoarthritis.

Details on the geographical location of cartilage lesions as well as their relationship to patient age at the time of surgery have been lacking in the literature. A recent study by Slauterbeck et al. attempted to answer these questions by reviewing 1,209 consecutive patients with ACL rupture.[49] They found that 43% of patients in their cohort had femoral articular cartilage injures evident at the time of ACL reconstruction. Patients who were ≥25 years of age were more likely to have multiple cartilage lesions present (7.7 versus 1.3%), supporting similar findings previously reported by Maffulli et al.[40] For the lateral femoral condyle, a delay to surgical reconstruction was associated with a greater proportion of large defects and grade 3 lesions, while for the medial femoral condyle surgical delay was associated with the presence of larger lesions.[49] Additionally, female patients in this study had a greater percentage of grade 1 lesions (29 versus 16%), whereas male patients had a greater percentage of grade 3 and 4 lesions (49 versus 35%). The

authors also noted that higher-grade cartilage lesions were present in older male patients undergoing ACL reconstruction compared with those found in younger male patients.[49] In a similar study by O'Conner et al., the authors reported that the risk for cartilage injury was increased with surgical delay in men but not in women undergoing ACL reconstruction.[50]

Meniscus Injuries

The incidence of medial meniscal tears at the time of ACL injury varies from 25 to 45%[38,51,52] and for the lateral meniscus from 31 to 65%.[10,53,54] The frequency of meniscal injuries increases over time in the setting of an ACL-deficient knee.[55,56] The location and pattern of meniscal injury also varies between acute ACL ruptures and chronic ACL-deficient knees.[57] Acute tears occurring at the time of ACL injury are typically radial and involve the posterior horn or mid-lateral third of the lateral meniscus.[57–59] The mechanism for acute lateral meniscus tears is theorized to be associated with the common bone bruise pattern seen with ACL injuries, where a valgus moment is coupled with anterolateral subluxation of the tibia, resulting in impingement of the posterolateral tibial rim and meniscus on the anterolateral femoral condyle. This mechanism would be expected to load the posterior horn of the lateral meniscus, resulting in a possible tear.[45] The medial meniscus is an important restraint to anterior–posterior translation in the ACL-deficient knee, acting as a mechanical block or wedge. Repetitive anterior translation of the tibia in the setting of ACL deficiency may overload the medial meniscus, resulting in tears. Multiple studies have documented an increased prevalence of medial meniscus injury as the time course after an ACL injury increases.[60,61]

The pathophysiology of a meniscus tear is related to its function in both joint congruency and joint stability. A recent biomechanical model demonstrated that forces in the medial meniscus were doubled in the ACL-deficient knee compared with those seen in the intact knee when subjected to an anterior and axial force.[62] In addition to restoring anterior–posterior translation to levels similar to those present in knees with an intact ACL, ACL reconstruction reduced the forces seen within the medial meniscus. Slauterbeck et al. recently studied 1,104 patients undergoing ACL reconstruction and found 722 meniscus injuries.[49] This study demonstrated an increased risk for meniscal injury with a delay to surgical reconstruction, supporting these pathophysiological principles. In this study, patients with a surgical delay of more than two months had an increased risk for medial meniscus injury (8–19%). This corresponds to previous literature that demonstrates an increased risk for meniscus injury with chronic ACL deficiency.[37,57,61,63,64]

Slauterbeck et al. also demonstrated a decreased risk for meniscal injury in female patients compared with male patients (56 versus 71%), with males more likely to have combined medial and lateral meniscus injuries (20 versus 11%).[49] Piasecki et al. similarly found fewer meniscal injuries in female patients than male patients; however, this observation was made in a cohort of basketball players.[65] O'Connor et al. also reported a higher rate of meniscal injury in males than females, and all patients in their study had an increased risk for meniscal injury with surgical delay.[50]

The association between meniscal injury and osteoarthritis has also been documented in the literature; however, wide variations in incidence exist. In general, 50% of patients who have undergone meniscectomy are reported to have radiographic evidence of knee osteoarthritis at 10–20-year follow-up, correlating to an odds ratio of about 10.[19] While some reports suggest that these rates are lower after partial meniscectomy,[66,67] a recent meta-analysis failed to find supportive evidence.[19] The outcome appears to be worse for women, obese individuals, and those who had a lateral meniscus resection.[19] The proposed explanations for the wide variations in reported rates of osteoarthritis following meniscectomy result from differences in the injury, the individual, and the methods and scoring mechanisms used to evaluate patients.[19] With respect to the injury, a variety of articular cartilage and meniscal pathology occurs with both the acute ACL tear and chronic insufficiency. It is challenging to separate out the effect of these variables without large multicenter trials. For the individual, delineation of variations between sex and age has been attempted;[49,50,65] however, other variables that likely affect outcome including muscle strength, education, and body mass index are only rarely reported and cohorts are often underpowered to study such factors.[67–70] Finally, more detailed methods of following patients clinically with complete, validated outcomes are needed. The creation of projects such as the Multicenter Orthopaedics Outcomes Network (MOON) to evaluate factors that correlate ACL injuries and osteoarthritis will clearly have a dramatic effect on the attempt to answer these challenging questions.

Conclusion

For patients who choose non-operative management following ACL rupture, the natural history of the ACL-deficient knee and its association with the development of osteoarthritis continues to be a subject of interest within the orthopaedic surgery literature. Recent studies have attempted to more clearly define the changes in knee kinematics produced by ACL deficiency. It has been postulated that the combination of excessive anterior tibial translation and rotational instability during weight-bearing activities in patients with ACL deficiency predisposes them to the development of meniscal and chondral injuries, with the end result being osteoarthritis. Continued research is necessary to further understand the interplay between ACL deficiency, abnormal knee mechanics, the articular cartilage microenvironment following ACL rupture, and the subsequent development of gonarthrosis. ■

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