Review Article

The Anterolateral Complex and Anterolateral Ligament of the Knee

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Abstract

The anterolateral structures of the knee have recently garnered considerable interest regarding their role in rotatory knee instability related to anterior cruciate ligament tears. Isolated anterior cruciate ligament reconstruction may not always restore rotatory stability of the knee. In these patients, additional procedures, such as lateral reconstruction or tenodesis, may be indicated. The anatomy of the anterolateral structures of the knee has been well described. Histologic and anatomic studies have reported conflicting findings regarding the presence of a discrete ligament. The biomechanical role of the anterolateral capsule in restraining internal tibial rotation has been described as negligible. The existing body of research on the anterolateral knee structures provides insight into the composition of the anterolateral complex of the knee.

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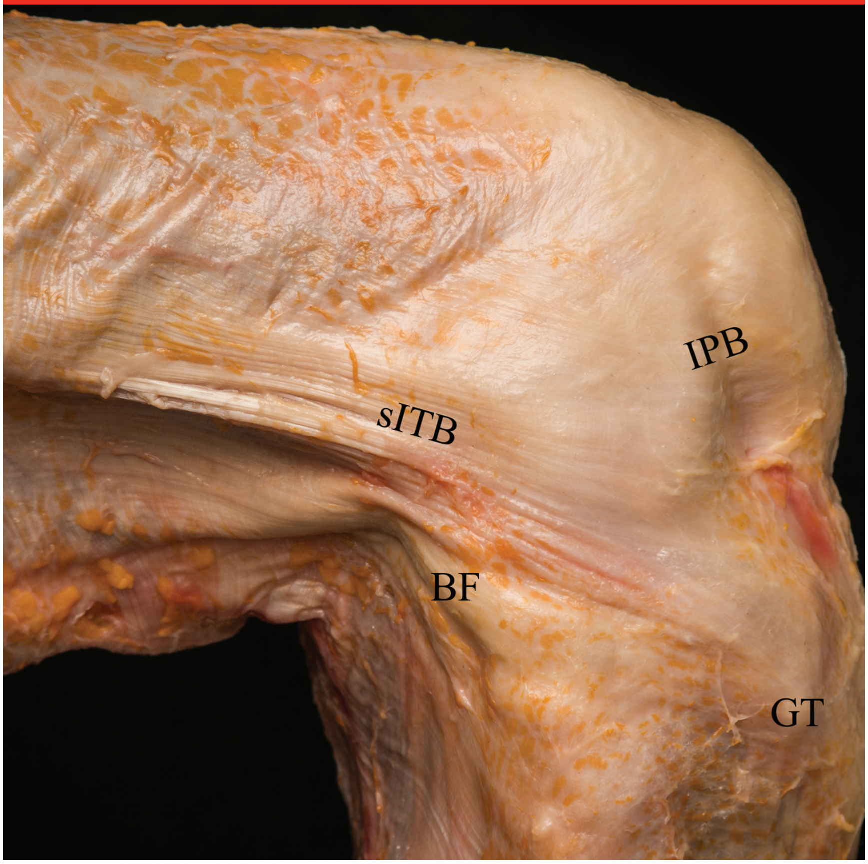
Anterior cruciate ligament (ACL) injuries often result in anterolateral rotatory instability (ALRI). The management of ACL injuries has evolved over the past several decades in an effort to address ALRI. Treatment typically consisted of repair or reconstruction in the 1950s to 1970s, then shifted to more extra-articular procedures in the 1980s, and returned to primarily intra-articular procedures in the 1990s.[1] During the past two decades, surgeons have performed predominantly intra-articular arthroscopic ACL reconstruction with an emphasis on restoring the anatomy and biomechanics of the native ACL.[1] Despite improved outcomes, some patients continue to have ALRI (eg, positive pivot shift test) even after anatomic ACL reconstruction, resulting in inferior patient-reported outcomes.[2]

Recently, scientific studies have suggested that a discrete structure termed the anterolateral ligament (ALL) may play an important role in restraint of internal tibial rotation.[3,4] These reports have piqued scientific interest in the anterolateral aspect of the knee, and the clinical problem of persistent ALRI after ACL reconstruction has been widely studied. Whereas extra-articular tenodeses and their principles are well understood, ALL reconstruction has not been fully investigated. Scientific articles describing the anatomy and biomechanical role of the ALL have reported conflicting findings.[3-11] Therefore, before proposing new surgical techniques, surgeons should consider the complex anatomy, function, and role of the structures of the anterolateral aspect of the knee.

Anatomy of the Anterolateral Complex

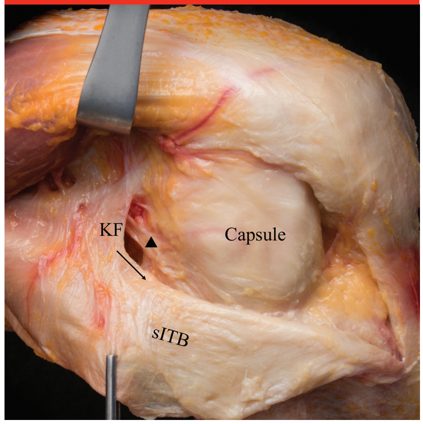
The layer-by-layer anatomy of the lateral side of the knee is complex and has not been as well defined as that of the medial side.[12] This complexity may be attributable in part to the

Figure 1



Photograph showing anatomic dissection of a fresh-frozen cadaver knee. The superficial iliotibial band (sITB), which inserts on the Gerdy tubercle (GT), shows a folding of the posterior fibers at 90° of flexion. Posteriorly, the sITB blends with the fascia of the biceps femoris tendon (BF). Anteriorly, fibers run to the lateral aspect of the patella and patellar tendon (iliopatellar band [IPB]).

Figure 2



Photograph showing anatomic dissection of a fresh-frozen cadaver knee. Posterior reflection of the superficial iliotibial band (sITB) reveals its firm insertion on the distal femoral metaphysis (Kaplan fibers [KF]). The KF are in close proximity to the branches of the superior genicular artery. The arrow shows the course of the deep iliotibial band merging with the sITB. Deep to the KF runs the capsulo-osseous layer of the iliotibial band (arrowhead). This layer blends distally to the sITB. In this specimen, no capsular thickening that would suggest the presence of a mid-third capsular ligament is visible.

distalization of the fibula during early embryonic development of the knee joint, which results in two capsular layers.[12] The superficial layer encompasses the lateral collateral ligament (LCL), whereas the deep layer lies medial to it. The superficial and deep layers merge anterior to the LCL.[12] The layers of the iliotibial band (ITB)[8,12,13] combine with the anterolateral joint capsule to form the anterolateral complex of the knee.

Superficial ITB

The dense, aponeurosis-like superficial ITB inserts distally at the Gerdy tubercle[8,13,14] (Figure 1). Its anteriormost fibers run distally in a curved fashion and insert on the lateral aspect of the patella and patellar tendon to form the iliopatellar band.[8,13] The posterior fibers reinforce the fascia of the biceps femoris muscle.[8] The superficial ITB proximally has a continuous attachment to the linea aspera of the lateral femur via the lateral intermuscular septum.[15]

Middle and Deep Layer of the ITB

The middle layer of the ITB is contiguous with the superficial ITB. It is characterized by an alignment of fibers from lateral-proximal to medial-distal that differs from the vertically aligned fibers of the superficial ITB.[13]

The deep layer of the ITB is located posterior and medial to the superficial ITB. It originates distal to the lateral intermuscular septum and combines with the superficial ITB distal to the lateral femoral epicondyle.[8,13,16] The superficial ITB also includes the Kaplan fibers, which play an important role in knee stability[5,14] (Figure 2). The Kaplan fibers are close to branches of the superior genicular vessels.[14] Therefore, in patients with rotatory knee injuries, hemorrhage in this area can be observed on MRI[14] (Figure 3). The superficial and deep ITB between the Kaplan fibers proximally and the distal insertion on the Gerdy tubercle, as well as the capsulo-osseous layer, form a functional unit that may contribute to rotatory knee stability by restraining internal tibial rotation, especially in knee flexion angles >30°.[5,14]

The Capsulo-osseous Layer of the ITB

The capsulo-osseous layer represents the deepest and most medial portion of the ITB (Figure 4). Proximally, this layer is contiguous with the fascia of the plantaris and the lateral gastrocnemius muscle, and it has some diminutive bony attachments in the area of the lateral femoral epicondyle. It runs deep and slightly posterior to the superficial ITB before merging with it distally and inserting posterior to the Gerdy tubercle.[13,14,16] The posterior fibers of the capsulo-osseous layer blend with the muscle fibers of the short head of the biceps femoris muscle. Distally, the fascia of the biceps femoris reinforces this layer of the ITB.[13] Some authors have suggested that the historically described capsulo-osseous layer is synonymous with the newly described ALL, although no consensus exists.[8]

The Anterolateral Capsule

The lateral joint capsule consists of a superficial layer and a deep layer, described by their orientation relative the LCL. These layers merge into one confluent layer anterior to the LCL.[12] Hughston et al[17] were the first to describe the lateral capsular ligaments of the knee, distinguishing between the anterior capsular ligament, the mid-third capsular ligament, and the posterolateral arcuate complex. Whereas the anterior capsular ligament is thin and has no femoral attachment, the mid-third capsular ligament represents a capsular thickening with discrete femoral and tibial bony insertions. The mid-third capsular ligament also has a firm attachment to the lateral meniscus.

Figure 3

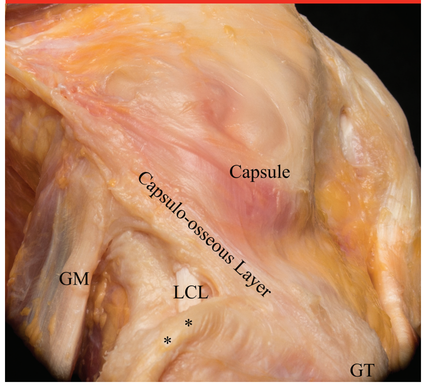
Coronal T2-weighted MRI demonstrating an anterior cruciate ligament injury. The arrow indicates a subtle hemorrhage in the area of the Kaplan fiber insertion.

These attachments form the meniscofemoral and meniscotibial ligaments, also known as the coronary ligament.[12,16] The anatomic descriptions of the mid-third capsular ligament and the ALL have considerable overlap, and some authors consider them to be the same structure.[18] Recent reports of the ALL have described separate layers deep and superficial to the LCL, analogous with the deep and superficial layers of the anterolateral capsule.[18]

Previous Detection of the ALL

Considering the comprehensive anatomic descriptions of the different layers of the ITB as well as the joint capsule, we suggest that the ALL likely refers either to the mid-third capsular ligament,[18] the capsulo-osseous layer of the ITB,[8,9,19] or a combination of both[7] (Figure 5). Because the different layers merge distally, the conflicting findings regarding its femoral origin, morphology, and course may have resulted from variation in dissection protocols and cadaver preservation methods. Furthermore, a critical

Figure 4



Photograph showing anatomic dissection of a fresh-frozen cadaver knee. After removal of the iliotibial band and sharp incision of the fascia of the biceps femoris tendon (asterisks), including excision of the superficial lamina of the lateral capsule, the capsulo-osseous layer of the iliotibial band becomes visible. GM = gastrocnemius muscle, GT = Gerdy tubercle, LCL = lateral collateral ligament

review of the literature demonstrates that investigations of embalmed specimens[7,19] have identified a different structure than those using fresh-frozen cadaver knees have found.[8,9] For example, Claes et al[7] found that the ALL inserted at the lateral epicondyle in all specimens (embalmed). Daggett et al[19] similarly found that most specimens, also embalmed, had insertions at or slightly posterior to the lateral epicondyle. However, Vieira et al[8] analyzed fresh-frozen specimens and reported that the ALL inserted in the supraepicondylar region. Dodds et al[9] described the origin as proximal and posterior to the epicondyle in fresh-frozen specimens.

Imaging of the Anterolateral Complex

Appropriate management of injuries depends on proper diagnosis. Although the anatomy of the anterolateral complex has been well

Figure 5

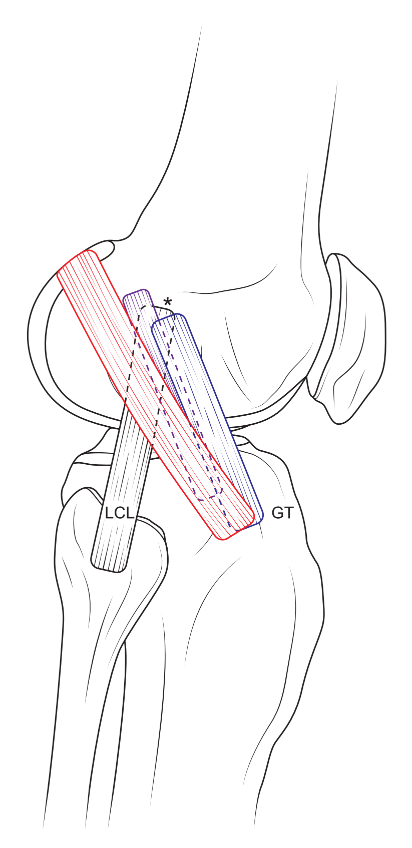


Illustration demonstrating the considerable overlap between anterolateral knee structures. The colored outlines show the approximate locations of the mid-third capsular ligament (blue), the capsulo-osseous layer (red), and the anterolateral ligament (purple), relative to the lateral collateral ligament (LCL), lateral femoral epicondyle (asterisk), and the Gerdy tubercle (GT). The capsulo-osseous layer is depicted from its proximal attachments with the lateral gastrocnemius, to its distal insertion, which spans from the posterior aspect of the GT to a region just anterior to the fibular head. The midpoint of its attachment is approximately halfway between the GT and the fibular head. The mid-third capsular ligament is depicted from its origin slightly anterior to the lateral epicondyle (anterior to the LCL, at the confluence of the superficial and deep capsule), to its insertion just posterior to the GT. The anterolateral ligament is depicted from its proximal insertion near the lateral epicondyle to its distal insertion posterior to the GT.

described, few studies have investigated imaging findings related to anterolateral complex injury. For soft-tissue imaging, we recommend the use of functional real-time ultrasonography and MRI.

Ultrasonography

Because of the proposed extra-articular location of the ALL in many studies, some authors have postulated that it should be visible on ultrasonography (Figure 6). However, visualization of the ALL is challenging, especially when the knee is fully extended.[20] With increased knee flexion angles and internal tibial rotation, Cianca et al[20] reported increased conspicuity of a ligamentous structure in the expected location.

In ultrasonographic studies followed by anatomic dissection, authors have reported conflicting results.[21,22] One study reported that ultrasonography had a sensitivity of 100% in visualization of the ALL,[22] with the ability to identify both the origin and the insertion of the ALL. In a different study, the ALL was visualized in only 60% of the specimens.[21] In that study, the average distances between the origin and insertion points of the ALL, as determined by ultrasonography and anatomic dissection, were 10.9 mm and 12.5 mm, respectively. The authors concluded that ultrasonography was inadequate to isolate the ALL from surrounding structures, and neither were the origin and insertion of the ALL consistently demonstrated on ultrasonography.[21]

Oshima et al[23] investigated anterolateral structures in nine healthy participants using real-time virtual ultrasonography, which combines MRI images with real-time ultrasonography. Using this modality, the authors visualized the femoral and tibial insertions in seven of the participants; the meniscal portions could not be visualized in any of the participants.

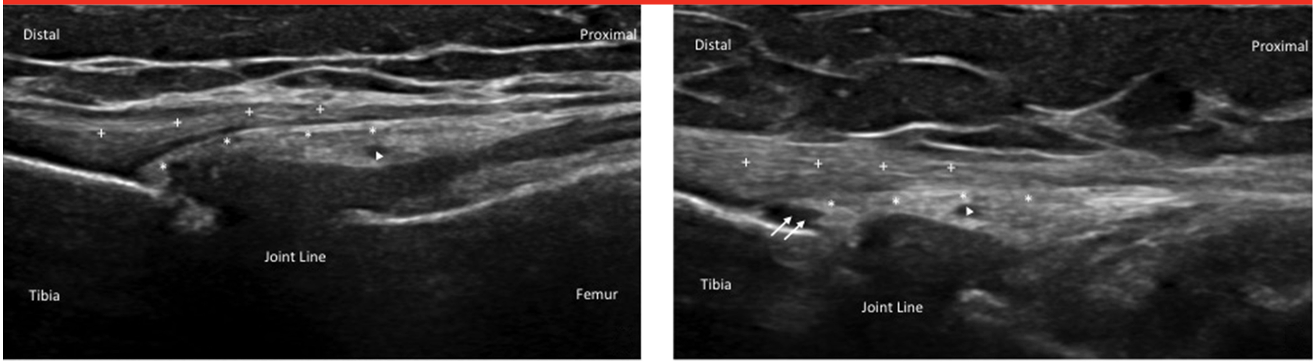
Magnetic Resonance Imaging

With the use of MRI, the ALL was identified in 51% to 100% of patients with ACL injuries in two studies.[24,25] However, in a different study, visualization of the entire ALL was reported to be as low as 11%.[26] In the study by Hartigan et al,[24] two musculoskeletal radiologists visualized the ALL on 1.5-Tesla MRI in 100% of the patients. However, one reviewer found the ALL to be torn in 26% of the patients, whereas the second reviewer found injuries in 62% of the patients, demonstrating poor inter- and intrarater reliability. In a different study, MRI findings were verified by anatomic dissection, demonstrating a sensitivity of 100% in detecting the ALL using 1.5-Tesla MRI.[27]

More consistent with previous anatomic descriptions of the anterolateral aspect of the knee, some authors have described a sheetlike structure connecting the distal femur to the proximal tibia that was not clearly separable from the LCL and the ITB on MRI.[28] Therefore, those authors recommended to forgo attempting to distinguish the ALL on routine MRI.[28] Dombrowski et al[29] investigated the morphologic appearance of the anterolateral capsule on marked cadavers using 3-Tesla MRI, with vitamin E markers placed along the course of any capsular thickening or along the expected location of the ALL. They found capsular thickening in 4 of 10 dissections, whereas MRI revealed 2- to 4-mm thickening in the mid-third capsular portion in 3 of 10 cadaver knees, demonstrating inter-specimen variability.

More recently, attempts have been made to grade anterolateral capsular injuries on MRI. In a study of patients with ACL injuries, anterolateral capsular injuries visible on MRI were re-

Figure 6



A **B**

Sonograms (long-axis view) demonstrating the anterolateral complex of the knee. The iliotibial band is marked with plus signs, whereas the anterolateral capsule is indicated with asterisks. The arrowhead marks the inferior genicular artery. **A,** Intact anterolateral capsule. The distal insertions of both the iliotibial band and the capsule are in close proximity to each other. **B,** Distal capsular injury (arrows).

ported in 51% of the patients.[30] However, high-grade injuries (complete disruption) were observed in <4%.

Biomechanical Findings

The soft-tissue and bony structures of the knee work in tandem to provide knee stability within physiologic limits. Recent biomechanical studies have focused on anterolateral soft tissues and their role in rotatory knee stability.

Numerous recent studies have investigated the influence of the ALL in ACL-deficient knees.[3-6,31] However, many of these studies refer to varying anatomic structures when describing the ALL. This heterogeneity has led to inconsistent results, and possibly overestimation or underestimation of the contribution of each structure to knee kinematics. Furthermore, the investigated injury scenarios often do not represent the injury pattern commonly associated with ACL tears. For example, few studies have evaluated anterolateral structures with a preserved ITB.[5,10,11] However, the ITB provides crucial contributions to rotatory knee stability.[5] For example, when the Kaplan fibers and the capsulo-osseous layer are sectioned, thereby disrupting the functional unit of the ITB between the distal femur and the proximal tibia, internal tibial rotation increases substantially throughout the range of motion.[5] In fact, sectioning the ALL in the presence of a preserved ITB does not result in increased rotatory instability.[5,10,11]

Contribution of the ALL

Studies that investigated the specific role of the ALL found the greatest increase in internal tibial rotation at 60° of knee flexion in ALL-deficient knees.[3] However, the maximum difference in internal tibial rotation during a simulated pivot shift between ALL-intact and ALL-deficient knees (with a reconstructed ACL) was only 2.7°.[3] When internal tibial rotation was compared between ALL-deficient and ALL-intact knees (with ACL deficiency) during a simulated pivot shift test, sectioning of the ALL resulted in a maximum increase of 3.3° at 45° of knee flexion.[31] At lower flexion angles where the pivot shift injury most often occurs, the contributing role of the ALL was even smaller.[3,31] This finding has been confirmed by recent studies demonstrating that the role of the ALL is negligible at physiologic ranges of tibial translation.[6,10,11]

The results of these biomechanical studies suggest that anterolateral capsular and extra-capsular soft tissues do contribute to rotatory instability. However, the ITB plays a relatively large role, and the contribution of the ALL alone is unlikely to account for large changes in anterolateral rotatory knee stability.

Effect of Anterolateral Injuries

From a biomechanical perspective, injuries to anterolateral structures have been associated with an increased pivot shift, especially in the presence of ACL deficiency. In vitro robotic studies investigating anterolateral capsular injuries demonstrated a higher degree of internal tibial rotation when the ALL or anterolateral capsule was deficient than when it was intact.[3,4] However, some authors have suggested that the ALL or anterolateral capsule plays only a minor role and engages only at nonphysiologic ranges of tibial translation during a simulated pivot shift test.[5,6,10,11] In a recent study of patients with ACL tears, participants with additional anterolateral capsular injuries had a greater pivot shift.[30] However, in that study, the presence of capsular thickening (ie, mid-third capsular ligament) influenced the pivot shift test, and the inclusion of patients with additional meniscal tears may have affected the results.

Management of Anterolateral Injuries

No consensus exists on the best treatment strategy for anterolateral knee injuries. Additional studies are needed to determine which of these injuries heal and which injury characteristics may affect rotatory knee stability if left untreated. A variety of surgical procedures has been described, although most are variations of lateral extra-articular reconstruction or lateral extra-articular tenodesis (LET). Different femoral fixation points, grafts, and fixation angles have been described.[32] The current evidence is based on heterogeneous clinical and in vitro studies.

Biomechanically, LET or ALL reconstruction decreases internal tibial rotation.[33] However, ALL reconstruction can result in overconstraint of internal tibial rotation at knee flexion angles >30°, regardless of the fixation angle.[33] Performing LET restrains internal tibial rotation and results in greater external tibial rotation compared with that of ACL-intact or reconstructed knees.[34] In vivo kinematic studies have found that anatomic ACL reconstruction results in a more externally rotated tibia compared with that of healthy knees.[35] This additional forced external rotation might cause abnormal knee kinematics with potentially detrimental long-term results. However, some clinical studies with long-term follow-up did not find a higher incidence of osteoarthritic changes when LET was performed in addition to ACL reconstruction.[36,37] Ferretti et al[36] reported that 51% of patients with isolated ACL reconstruction had Kellgren-Lawrence grade 2 or higher arthritis at 10-year follow-up, compared with 14% of patients who underwent ACL reconstruction combined with LET. Yamaguchi et al,[38] however, reported moderate or severe osteoarthritis in 71% of patients at a mean of 24 years after LET and ACL reconstruction, compared with 16% of patients who underwent isolated ACL reconstruction.

At least one study has shown that adding LET to ACL reconstruction may reduce the incidence of positive pivot shift postoperatively.[32] However, a separate study reported persistent pivot shift in 8.4% of patients even after combined ALL and ACL reconstruction.[39] No studies have demonstrated improved objective or subjective outcomes of one procedure compared with the others.[32,36]

A recent panel of surgeons revealed the use of a wide variety of treatment algorithms to determine when to combine lateral tenodesis or reconstruction procedures with standard ACL reconstruction.[40] The surgeons reported performing lateral extra-articular procedures in conjunction with primary ACL reconstruction 5% to 80% of the time, and this rate was higher in patients undergoing revision surgery. Although precise indications varied among the surgeons, most recommended adding extra-articular procedures in young, active patients with pathologic rotatory laxity and imaging suggestive of anterolateral complex injury. Most of the surgeons also suggested considering lateral extra-articular procedures in patients requiring revision after a previously well-performed ACL reconstruction when no additional reason for failure is identified. Surgical techniques varied and included the modified Lemaire procedure, the Marcacci and MacIntosh techniques, and the more recently described ALL reconstruction. However, the surgeons highlighted the importance of addressing the ACL deficiency as well as injuries to other secondary stabilizers, such as the menisci and collateral ligaments.[40]

Summary

The anterolateral complex of the knee consists of the ITB with its deep components, such as the capsulo-osseous layer and the Kaplan fibers, and the anterolateral capsule, including the mid-third capsular ligament. Although this ligament was only recently described as the ALL, the mid-third capsular ligament and the capsulo-osseous layer have been well documented in the orthopaedic literature.

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References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, reference 2 is a level II study. References 30, 32, 36, and 38 are level III studies. References 37 and 39 are level IV studies.

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