The Anterolateral Ligament is Not the Whole Story: Reconsidering the Form and Function of the Anterolateral Knee and its Contribution to Rotatory Knee Instability

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Summary: The heterogeneity of available cadaveric, histologic, and radiographic results related to the anterolateral ligament (ALL) does not support its existence as a discrete anatomic structure. Moreover, focusing narrowly on the ALL in isolation, what has previously been referred to as "ALL myopia," obscures a thorough appreciation for the stability contributions of both capsular and extracapsular structures. We consider injury to the soft tissues of the anterolateral knee–the anterolateral complex—just one component of what is frequently found to be a spectrum of pathology observed in the rotationally unstable, anterior cruciate ligament (ACL)-deficient knee. Increased lateral tibial slope, meniscal root tears, and "ramp" lesions of the medial meniscocapsular junction have all been implicated in persistent rotatory knee instability, and the restoration of rotational stability requires a stepwise approach to the assessment of each of these entities. Through an appreciation for the multifactorial nature of rotatory knee instability, surgeons will be better equipped to perform durable ACL reconstructions that maximize the likelihood of optimal clinical outcomes for patients. The purposes of this review are to provide an update on the relevant anatomy of the anterolateral knee soft tissues and to explain the multifactorial nature of rotatory knee instability in the setting of ACL deficiency.

Key Words: anterolateral capsule—anterolateral ligament—anterolateral knee—rotatory knee instability.

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The pathoanatomy of anterior cruciate ligament (ACL)-deficiency typically involves injuries to multiple structures. The existence and, perhaps more importantly, the functional significance of the anterolateral ligament (ALL) remains a matter of the debate, and a thorough understanding of rotatory knee instability necessitates an appreciation for the dynamic interplay of multiple factors. In addition to injuries of the anterolateral capsule, a growing body of literature has elucidated the destabilizing effects of meniscal pathology and increased lateral tibial slope. Moreover, patient-specific factors such as generalized ligamentous instability, chronicity, type of sport, age, and sex may play a larger role in knee instability than previously thought.

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The authors declare that they have nothing to disclose.

For reprint requests, or additional information and guidance on the techniques described in the article, please contact Volker Musahl, MD, at musahlv@upmc.edu or by mail at Department of Orthopedic Surgery, Center for Sports Medicine, University of Pittsburgh, 3200 S Water Street, Pittsburgh, PA 15203. You may inquire whether the author(s) will agree to phone conferences and/or visits regarding these techniques.

ANTEROLATERAL KNEE ANATOMY RECONSIDERED

Although previous authors have asserted the existence of the ALL,[1–9] growing bodies of anatomic and radiographic data have cast doubt on descriptions of a discrete, pericapsular structure. Dombrowski et al[10] reported on the results of macroscopic dissection, magnetic resonance imaging (MRI), and histologic analysis of 10 fresh-frozen specimens. None of the 10 specimens were observed to have a discrete lateral capsular ligament, and a palpable macroscopic thickening of the lateral capsule was identified in only 4 of 10 specimens. Furthermore, these authors observed a substantial amount of anatomic variability when evaluating the lateral capsular knee structures with MRI. In only 3 of 10 specimens, there was a discrete thickening of the central third of the lateral capsule observed, and in 1 specimen did gross dissection findings match those appreciated with MRI. Histologically, the lateral capsular structures demonstrated a transitional microscopic appearance between that of the aligned collagen fibrils of the fibular collateral ligament (FCL) and randomly oriented cell nuclei dispersed throughout the disorganized extracellular matrix of the capsular tissues (Fig. 1). Shea et al[11] have reported a similar amount of macroscopic variability in dissection of pediatric specimens aged below 5 years with only 1 of 8 cases showing evidence of a discrete ligamentous structure. Although somewhat less variation in the presence of an ALL has been observed among slightly older specimens (9 of 14 specimens; median age, 8 y), considerable variance has been noted with respect to the anatomy of the ligament's femoral origin.[12] Even in a comparably sized cadaveric study, the results of which have been used to argue "unequivocally" on behalf of the ALL's existence, Stijak et al[7] reported a discrete thickening of the lateral capsule in only 50% of specimens. The inconsistency of these results does not convincingly support the description of the ALL as a pericapsular ligament.

The relevant anterolateral soft tissues of the knee are comprised of the iliotibial band (ITB) and the anterolateral joint capsule. The results of a recent analysis have elucidated several key anatomic relationships and provided an alternative to previous descriptions of the anterolateral knee soft tissues through dissection of 20 fresh-frozen specimens. Rather than truncating the ITB proximally and reflecting it en bloc inferiorly, Herbst et al[13] performed layer-by-layer study of the subtleties of the ITB and the intimate associated adjacent knee structures by reflecting the superficial ITB inferiorly, thus preserving Kaplan's fibers.[5] As a result, these authors described the anterolateral complex (ALC) as a series of discernible layers, focusing especially on the femoral and tibial attachments of the middle and deep layers. The macroscopic structure of the anterolateral capsule was observed as 2 distinct layers: the superficial layer encompassing the FCL, and the deep lamina coursing deep to the FCL. It is within this deep

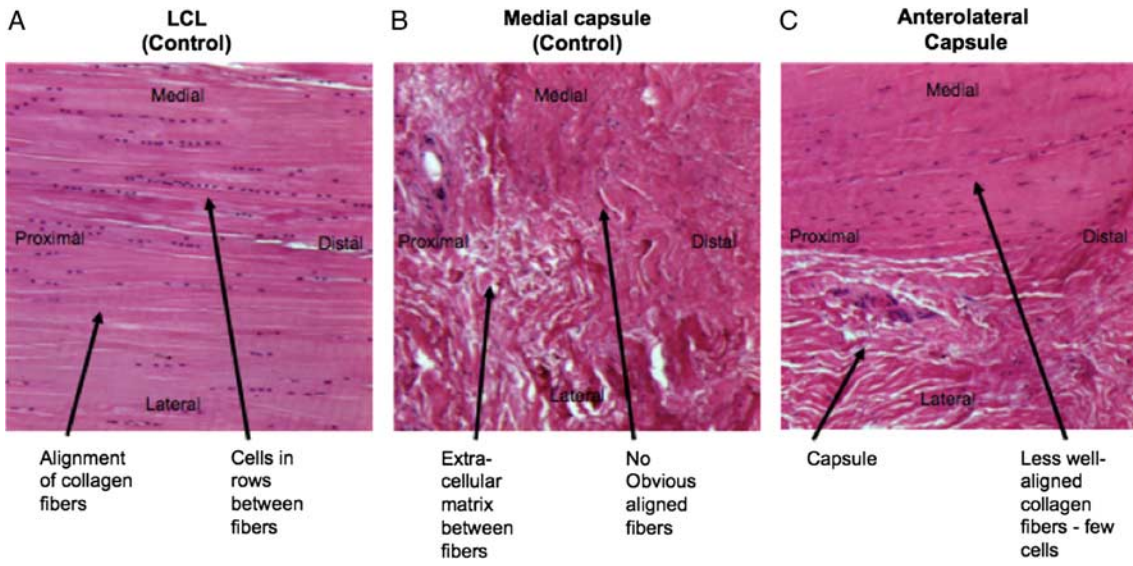

FIGURE 1. Histologic representation of aligned collagen fibrils of the fibular collateral ligament (A) versus randomly oriented cell nuclei (B) and disorganized extracellular matrix of the anterolateral capsule (C) (Reference: Dombrowski et al[10]. LCL indicates lateral collateral ligament. full color online

layer that Hughston et al[14,15] described a capsular thickening as the mid-third lateral capsular ligament. It is entirely plausible that the inconsistency with which multiple authors[7,16] have described the ALL is attributable to discrepancies in dissection techniques (proximal sectioning of the ITB with inferior reflection en bloc) and the consequences of suboptimal methods of specimen fixation (fresh-frozen vs. embalmed specimens).

The nuances of the ITB's relationship to the anterolateral knee is also worthy of special focus. The deep ITB has been observed to connect proximally to the femoral metaphysis just distal to the lateral intermuscular septum through what have previously been referred to as Kaplan's fibers (Fig. 2).[17,18] The most medial part of the ITB, the capsule-osseous layer has been consistently found to converge distally with the superficial and deep ITB slightly posterior to Gerdy's tubercle. This refined anatomic description of the ITB should inform future studies as to how best to address discrete injuries to the ALC.

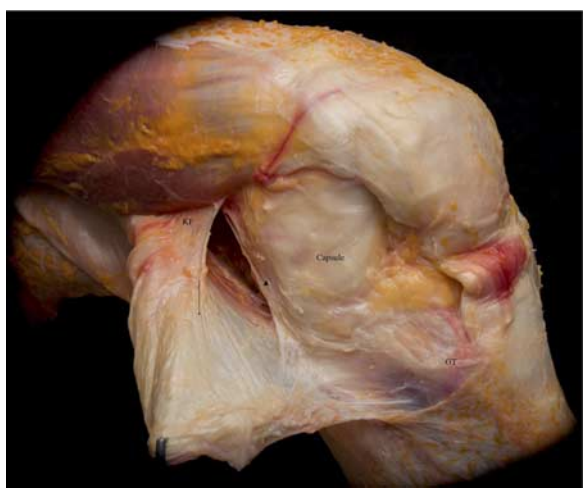

FIGURE 2. Anatomy of the anterolateral capsule with the ITB dissected layer by layer. Black arrow: deep ITB; black arrowhead: deep capsulosseous layer. GT indicates Gerdy tubercle; ITB, iliotibial band; KF, Kaplan fibers (Reference: Herbst et al[13]). full color online

FUNCTION OF THE ALC

The results of several biomechanical studies corroborate the existence of an ALC rather than the ALL. In addition, these data suggest that the ALL does not bear loads of sufficient magnitude to be considered an important stabilizing structure against rotatory knee instability. Among 14 specimens, Saiegh et al[16] identified an ALL in 6 (43%) cases. No changes in the magnitude of tibiofemoral translation or rotation were observed after transecting the ALL, while maintaining the integrity of the ITB in the setting of ACL deficiency. Guenther et al[19] applied a combined internal rotation and valgus load to fresh-frozen cadaveric knees and demonstrated that the anterolateral capsule carries negligible forces in any longitudinal direction, as would be expected in the case of a discrete ligamentous structure. Rather, these authors likened the anterolateral capsule to a sheet of tissue (eg, glenohumeral ligaments), capable of transmitting larger forces perpendicular to its longitudinal axis (Fig. 3). Perhaps equally important, the anterolateral capsule was found to bare significantly smaller forces across all tested knee flexion angles than that of ACL and FCL, further diminishing the role of the ALL. Noyes et al[20] observed similar results, with sectioning of the ALL in the setting of ACL deficiency resulting in only minimal increases in lateral compartment translation during pivot-shift testing.

Similarly, several biomechanical studies have examined the role that the ITB may play in rotational knee stability. Rahnemai-Azar et al[21] demonstrated that the ITB maintained significantly higher ultimate load (almost 50% higher) and stiffness (nearly 3-folds higher) compared with the anterolateral capsule. Kittl and colleagues scrutinized the contribution of the ITB to rotational knee stability by testing pivot-shift kinematics in ACL-intact and ACL-deficient knees. These authors showed that both the superficial and deep layers of the ITB provided

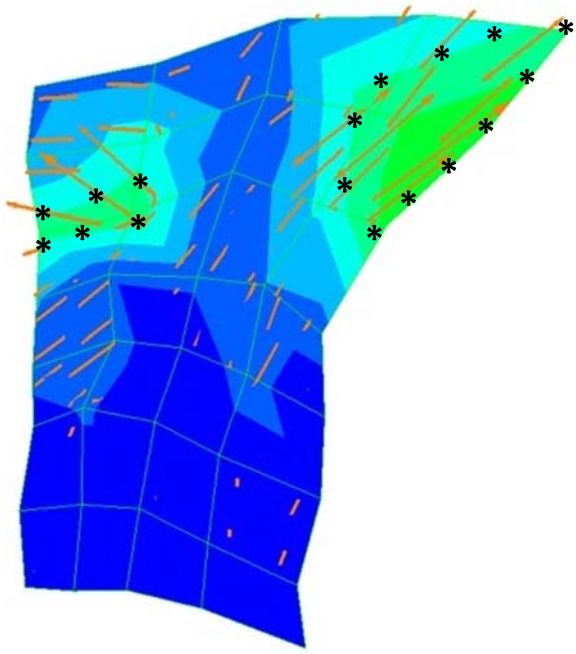

FIGURE 3. Sample fringe plot (manuscript in progress) showing strain distribution in the anterolateral capsule in response to 134 N anterior tibial load at 30 degrees of knee flexion. The regions outlined by asterisks represent areas with higher maximum principal strain magnitudes. The arrows represent the maximum principal strain vectors. This is in concordance with published data from Guenther et al[19] that shows this area acts like a tissue sheet rather than a ligament. full color online

significant restraint to internal rotation in the ACL-intact and ACL-deficient knees, whereas the other anterolateral structures provided much smaller contributions unlikely to be of clinical significance.[22]

Previously referred to as the "true knee anterolateral ligament," the capsule-osseous layer of the ITB and its relationship with the ACL is best thought of as a functional unit shaped like inverted horseshoe sling draped around the posterior femoral condyle (Fig. 4).[23,24] This anatomic and biomechanical paradigm posits that ITB, rather than either a discrete ALL or capsular thickening, is the primary restraint to anterolateral tibial subluxation during the pivot-shift test. The evolution of our understanding of the form and function of the ALC will guide future efforts to determine whether lateral-sided augmentation procedures are needed in the setting of ACL deficiency. In this scenario, it will be important to establish which procedure, between ALL reconstruction and extra-articular tenodesis, more closely restores rotational knee stability.

BEYOND THE ALC: OTHER CONTRIBUTIONS TO INSTABILITY IN THE ACL-DEFCIENT KNEE

The importance of the ALC notwithstanding, a discussion of the injuries commonly observed in conjunction with ACL tears is essential to any understanding of rotatory knee instability in the ACL-deficient knee. As is always the case, a thorough physical examination, close scrutiny of advanced imaging studies, and a methodical approach to diagnostic arthroscopy are of critical importance to the appreciation of associated injuries, which may contribute to high-grade rotatory instability. The role of the menisci as important secondary

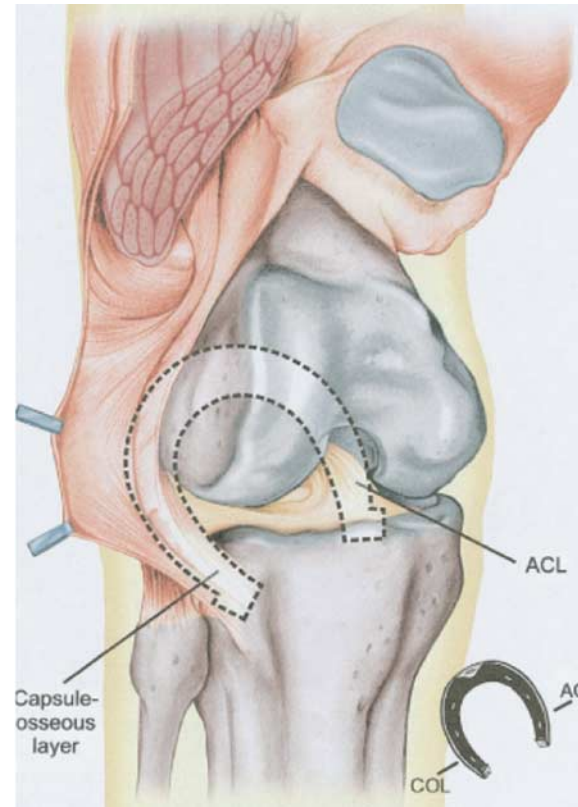

FIGURE 4. The COL of the iliotibial band and ACL work synergistically in an inverted horseshoe sling draped around the posterior femoral condyle to prevent anterolateral subluxation during the pivot-shift test (Reference: Vieira et al[24]). ACL indicates anterior cruciate ligament; COL, capsule-osseous layer. full color online

stabilizers of the ACL is well documented.[25–29] In addition, consideration must be given to patient-specific factors, which may increase the likelihood of ACL rupture, graft failure, and/or persistent rotational instability.

Posteromedial Meniscocapsular Injuries

Posteromedial meniscocapsular tears (so called "ramp" lesions) have been described as a subset of meniscal injuries observed in 9% to 40% of cases of ACL tears.[30–34] These lesions represent disruption of the attachment between the junction of the meniscotibial ligament, joint capsule, and posterior horn of the medial meniscus. Sonnery-Cottet and colleagues have further classified ramp lesions based upon the location of the meniscal tear and the corresponding degree of mobility at arthroscopic probing: type 1 lesion involves tear of the capsule posterior to the meniscotibial ligament and low mobility. Type 2 lesion is a partial, vertical-superior meniscal tear anterior to the meniscotibial ligament with low mobility. Type 3 lesion is a partial, vertical-inferior meniscal tear anterior to the meniscotibial ligament with high mobility. A type 4 lesion is a complete vertical meniscal tear anterior to the meniscotibial ligament with very high mobility. Type 5 is a "double lesion" involving 2, complete vertical meniscal tears anterior to the meniscotibial ligament.[33,35] In addition, a membrane-like tissue that can easily obscure adequate visualization of the lesion does not infrequently cover these derangements of the meniscocapsular interface. The utility of

MRI in detecting ramp lesions remains a matter of debate in the literature as a wide range of sensitivities (48% to 85%) have been reported by multiple authors.[36,37] These facts underscore the necessity for a systematic approach to careful arthroscopic inspection of the posteromedial compartment. Although the clinical consequences of "missing" these "hidden" injuries remain poorly defined,[38,39] compelling cadaveric data suggest that ramp lesions may play an important role as secondary sources of laxity in the ACL-deficient knee. Stephen and colleagues observed significantly increased anterior and rotational instability after sectioning the posteromedial meniscocapsular junction. Moreover, these instabilities were not restored after ACL reconstruction alone, perhaps underscoring the pitfall of neglecting ramp lesion repair.[40] Peltier et al[41] showed similar effects of detaching the meniscotibial ligament on anterior tibial translation and rotation at all tested knee flexion angles. In light of the documented prevalence of these lesions in association with ACL tears and the potential deleterious consequences of neglecting these injuries, close scrutiny of preoperative imaging combined with a complete diagnostic arthroscopic examination of all relevant structures is advised.

Lateral Meniscal Root Tears (LMRT)

LMRT have been found to occur in conjunction with an ACL tear 8% to 14% of patients.[42–45] Similar to the demonstrated effects of sectioning the meniscotibial ligament and simulated lateral menisectomy,[27,28] LMRT been also been shown to further destabilize the ACL-deficient knee. Shybut et al[46] demonstrated that a LMRT coupled with a meniscofemoral lesion accentuated the pivot-shift phenomenon in the ACL-deficient knee. Frank and colleagues reported similar results with LMRT without a meniscofemoral lesion in ACL-deficient state, observing increased amounts of internal rotation with knee flexion angles between 75 and 90 degrees. Moreover, increases in anterior tibia translation at 30 degrees of knee flexion during pivot-shift testing were observed compared with the isolated ACL deficiency in isolation.[47]

These biomechanical results have been corroborated by clinical data pertaining to patients with concomitant LMRT and ACL injuries. In a retrospective review of 74 cases found to have a high-grade pivot-shift (grades II and III), Song and colleagues found that the proportion of patients with a complete LMRT in the high-grade pivot group was significantly larger than the low-grade pivot-shift group (68.8% vs. 39.1%, respectively; $P = 0.017$). In addition, LMRT were significantly associated with a high-grade pivot-shift in noncontact ACL injuries (odds ratio, 4.044; 95% confidence interval, 1.125-14.534; $P = 0.032$).[48]

Tibial Slope

The osseous morphology of the proximal tibial may also be a predisposing factor for ACL injury and recalcitrant instability. Although the observation that an increased posterior tibial slope has been associated with ACL tears is not a new concept,[49–53] recent literature has more precisely defined the relationship between increased lateral tibial slope and rotatory knee instability. Using a quantitative measurement of pivot-shift using digital image analysis, Rahnemai-Azar et al[54] showed that patients with "high-grade rotatory laxity" had a significantly increased tibial slope of 9.3 ± 3.4 degrees compared with 6.1 ± 3.7 degrees ($P < 0.05$). Dare et al[55] observed significant increases in lateral tibial slope in ACL-injured pediatric patients (mean age, 14.8 y) compared with uninjured controls (5.7 ± 2.4 vs. 3.4 ± 1.7 degrees, $P < 0.001$). Similarly, a recent retrospective review of 90 collegiate football players with ACL tears revealed that increased lateral tibial slope (odds ratio, 1.32; 95% confidence interval, 1.15-1.78) was the sole, independent predictor of ACL injury.[56] In light of these clinical observations in the ACL-deficient state, more research is needed to elucidate the relationship between increased lateral tibial slope and residual rotational instability following ACL reconstruction.

Patient-specific Factors: Generalized Laxity and Sex

Generalized hypermobility may also predispose patients to rotatory knee instability manifesting as either primary ACL tear or retear in the setting of a previous ACL reconstruction.[57–60] Recently, Larson et al[61] reported on a prospective series of patients screened for hypermobility before undergoing ACL reconstruction. Significantly higher failure rates were observed among hypermobile patients compared with nonhypermobile patients (24.4% vs. 7.7%; $P = 0.006$.) Even among patients who did not experience graft failure, the hypermobile group performed worse on assessments of overall satisfaction and functional status as represented in multiple patient-reported outcome measures. Interestingly, KT-1000 measurements as represented with side-to-side differences were not different among groups (hypermobile group, 1.6 mm vs. nonhypermobile group, 1.0 mm; $P = 0.124$), perhaps suggesting that hypermobility may have a more pronounced effect on rotatory instability rather than anterior-posterior translational motion.

In addition, increased rates of ACL rupture among women have been reported in the literature,[62,63] and this fact has been attributed to a myriad of factors, including bony morphology, lower extremity coronal alignment, and neuromuscular activation patterns.[64–67] Supplementing existing descriptions of these sex-specific factors, Pfeiffer et al[68] have shown that female athletes reliably demonstrated greater lateral compartment anterior translation compared with that of men in quantitative assessments of the pivot-shift phenomenon (median, 1.6; range, 0.3 to 4.9 vs. median, 1.1; range, 0.1 to 7.1, respectively; $P < 0.005$). These data are the first of their kind and suggest that women may have increased amounts of rotatory knee instability at baseline.

CONCLUSIONS

The successful treatment of rotatory knee instability requires a thorough appreciation of the contributions of both intracapsular and extracapsular structures. Future research is best focused not on one anatomic structure—the existence of which is not uniformly supported by available anatomic and biomechanical data—but rather on a stepwise approach that recognizes the multifactorial nature of instability in the ACL-deficient knee.

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