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Biomechanical Role of Lateral Structures in Controlling Anterolateral Rotatory Laxity: The Iliotibial Tract

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Recent research, focusing on rotatory knee laxity, has intrigued the whole orthopaedic knee community. First popularized by Hughston et al, peripheral knee injuries at the time of cruciate ligament rupture have regained more and more recognition, which has led to a better understanding of these injuries. Recent research has been focused on anterolateral rotatory instability, especially regarding those structures that are responsible for the high-grade anterior subluxation of the lateral tibial plateau when damaged. Work at Imperial College London showed that the iliotibial tract (ITT) was the primary restraint to internal tibial rotation, especially the capsulo-osseous layer of the ITT, which contributed almost 25% of controlling a 5-Nm internal rotation torque at early flexion angles. However, due to the complex fiber arrangement, the functional anatomy of the ITT is difficult to understand. Thus, this article focuses on the involvement of the internal tibial rotation in restraining internal rotation and the pivot-shift phenomenon.
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The Role of the Peripheral Ligamentous Structures in Controlling Knee Laxity

At first sight, the knee joint seems like an easily understandable hinge mechanism. The central column consists of the cruciate ligaments that prevent anterior and posterior translation, whereas the collateral ligaments stabilize the medial and lateral sides. However, physiological knee kinematics is much more complicated than a simple hinge articulation. For example, the "screw home mechanism" locks the knee joint with tibial external rotation in full extension, so that we can relax our muscles for near-effortless standing. Furthermore, in flexion the knee is free to move in nearly 6 degrees-of-freedom fashion, neutralizing external forces. It gets even more complicated when looking at the interplay between the tibiofemoral, patellofemoral, and tibiofibular joints and pathologic knee kinematics (eg, after anterior cruciate ligament [ACL] rupture).

Recent research, focusing on rotatory knee laxity, has intrigued the whole orthopaedic knee community. First popularized by Hughston et al,[1] peripheral knee injuries at the time of cruciate ligament rupture have regained more and more recognition, which has led to a better understanding of these injuries. Overlooked concomitant injuries (eg, posterolateral injuries) are therefore seen as prevalent causes for failed cruciate ligament reconstruction surgery. In posterior cruciate ligament (PCL) reconstruction, it is common practice to address the peripheral injuries, whereas in ACL reconstruction—due to the perception of good results of isolated reconstructions, the previously used concomitant extra-articular procedures were abandoned, only to have current resurgence. There is still a lack of understanding of trauma mechanisms and their concomitant injuries.

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Hughston et al[1] classified rotatory knee instability and distinguished between 4 different types: anteromedial, anterolateral, posteromedial, and posterolateral. This is a really practical way of considering the impact of ligamentous injury when examining a knee. Nevertheless, it belies the true complexity of abnormal translations and rotations accompanying knee ligament injury. Rather than pure resultant translations or rotations, injuries cause "coupled" translations and rotations. For example in ACL injury, the pivot-shift phenomenon is due to combined anterior tibial translation (ATT) and internal rotation. Furthermore, the extent of these motions is unique to a patient's knee (the degree of injury and the structures injured, overall native ligamentous laxity, and articular geometry), the forces and torques applied by the examiner's hands, and the effect of gravity.

Anteromedial Rotatory Instability

Anteromedial rotatory instability (AMRI) has been described as resulting from a disruption of the medial collateral ligament (MCL) (with or without the posterior oblique ligament that limits internal rotation near knee extension) and is accentuated by a rupture of the ACL. This causes a positive abduction (valgus) stress test in 30° knee flexion and a positive anterior drawer test in external rotation. The Lachman test may create an impression of a gross straight anterior subluxation of the tibia, because both the medial and lateral tibial condyles are similarly mobile with AMRI, whereas the lateral tibial plateau moves more than the medial if the peripheral structures are intact, causing a coupled internal rotation.[2] The pivot-shift test may be positive in external rotation, as the medial tibial plateau subluxes in an anterior direction. The mobile medial tibial condyle is abnormally prominent with increased tibial external rotation, and this allows AMRI to be differentiated from a posterolateral corner injury, which leads to posterolateral rotatory instability (PLRI).

Anterolateral Rotatory Instability

Similarly to AMRI, anterolateral rotatory instability (ALRI) was described, resulting from a rupture of the anterolateral structures and is accentuated by an ACL disruption. However, the opposite seems more logical due to the more mobile lateral compartment; an isolated ACL deficiency will result in some degree of ALRI and is a prerequisite for the pathologic anterior subluxation of the lateral tibial plateau. This is accentuated by anterolateral structure disruption triggering a high-grade ALRI, which manifests as a big pivot-shift phenomenon on examination. Recently, research has been focused on ALRI, especially regarding those structures that are responsible for high-grade anterior subluxation of the lateral tibial plateau when damaged. However, recent research has not shown uniform results, culminating in a lot of confusion. The authors of this article believe that the failure of the posterior (deep) fibers of the iliotibial tract (ITT) is responsible for high-grade ALRI. In keeping with the medial side,[3] the capsular structures may only be the secondary restraint—among these is the "anterolateral ligament".[4-6]

Posterolateral Rotatory Instability

Posterolateral rotatory instability was initially described as external rotation of the tibia about the intact axis of the PCL, which is caused by a disruption of the posterolateral structures (lateral collateral ligament (LCL), popliteus complex including popliteofibular ligament, and arcuate complex). However, the term PLRI does not only indicate an isolated rotatory instability but may also include a coupled posterior translation of the center of the tibial plateau, which is usually clinically significant because it follows a PCL injury. An isolated PCL rupture does not result in PLRI, if the posterolateral structures are intact.

Posteromedial Rotatory Instability

Unfortunately, there is a lack of evidence and research regarding posteromedial rotatory instability (PMRI). Hughston et al[1] did not believe that there is PMRI, as the integrity of the PCL will hinder the internal rotatory movement of the tibia. However, recent research has been shown that lesions of the MCL and posterior oblique ligament will evenly increase external and internal rotation, respectively, in an ACL-intact laboratory test setup. Thus, similar to AMRI, in a PCL-deficient knee, there may be a high-grade PMRI if the medial structures are ruptured.

Functional Anatomy and Biomechanics of the Iliotibial Tract

The ITT is the distal extension of the fascia lata, which is formed proximally when it covers the tensor fascia latae and gluteus maximus muscle. It can, therefore, be divided into a dynamic proximal portion, tensioned by the tensor fascia latae muscle and a distal "ligament-like" static portion. The distal part of the ITT can further be divided into an anterior or superficial and posterior or deep part.[7] The lateral intermuscular septum tethers the fascia to the femur on the linea aspera in a horizontal fashion and extends distally into the "Kaplan fibers,"[8] which join the posterior or deep layer of the ITT to the lateral femur, creating a sling around the posterolateral femur and inserting on the anterolateral tibia posterior to Gerdy tubercle.

In the past, the fascia lata and its corresponding muscles have been extensively studied. Early anatomists considered the tensor fascia latae muscle and its ITT extension as acting on the knee. Vesalius for example called it the sixth muscle of the tibia. The most comprehensive research on the ITT has come from Emmanuel B. Kaplan in 1958.[8] He postulated the importance of the fascia lata and the ITT in the erect posture of man, as they are not present in animals. Furthermore, and in a change from previous thinking above, it was already concluded that the muscles (tensor fascia latae and gluteus maximus) transmit no action to the knee joint, but they have an important tensioning function on the ITT. Even in cadavers, where obviously no