Proposed Mechanism for Non-Contact Anterior Cruciate Ligament Injury

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Abstract
This case report illustrates a theory of a mechanism of injury for anterior cruciate ligament (ACL) tears. Studies show ACL injuries can occur with maximal quadriceps muscle contraction. When the timing of the foot plant is miscalculated and is a split second later than planned, the exaggerated muscle contractions cause the tibia to shift more anterior than what would have occurred had the foot struck the ground at the correct time. With anterior translation, the tibia becomes internally rotated because the shorter and downward slope of the lateral tibial plateau, and this puts the ACL in a lengthened position. At the time of foot plant and impact, the sulcus terminalis of the lateral femoral condyle contacts the posterolateral tibial plateau. I propose that the impact of the femur on the posterior part of the tibia causes a fulcrum action, pushing it further anteriorly and medially, causing the lengthened ACL to rupture. Bone bruises on the lateral aspect of the femoral condyle and the posterolateral aspect of the tibial plateau, which occur at the time of impact, can be explained by this mechanism. The fulcrum action of the femur pushing the tibia forward causing an ACL injury has not been described.

ABBREVIATIONS
ACL: Anterior Cruciate Ligament

INTRODUCTION
Several mechanisms for anterior cruciate ligament (ACL) injury have been described [1-3]. In a study that included 71 noncontact injuries, videotapes revealed that most injuries were sustained at footstrike with the knee close to full extension. The authors theorized that forceful quadriceps contraction may be a major contributor to ACL injury [1]. In two other studies of video analysis of basketball or handball players, most players were injured while in close proximity to an opponent [4,5] and the ACL injury was believed to occur milliseconds after initial ground contact [4]. In general, non-contact ACL injuries occur in sports involving jumping, landing, and pivoting where the athlete generates forces upon sudden deceleration that are greater than the ACL can accommodate [1,6]. DeMoratet al. [7], found that a 4500N, one-second quadriceps muscle contraction applied to a fixed femur with the knee in near full extension could produce enough anterior tibial translation to cause injury to the ACL. Elias et al. [8], studied the effect of the soleus and gastrocnemius muscles contraction on tibiofemoral motion. Using a knee simulator to control the quadriceps and hamstring muscle forces and hip load on the knee, the gastrocnemius and soleus muscles were each loaded, separately and together, with a 180N constant force. The investigators found that the soleus muscle forces translated the tibia posteriorly and gastrocnemius muscle force translated the tibia anteriorly, but combined forces of the gastrocnemius and soleus muscles caused anterior translation of the tibia. The location of bone bruises associated with ACL injury, typically the lateral femoral condyle and the posterolateral tibial plateau [9] may provide further understanding as to the mechanism for ACL injuries. One thought is that the ACL tears and then the translation in the joint occurs, causing impaction of the femur on the tibia [10]. This current case report describes a different proposed mechanism for non-contact ACL injury and is based on the work of others [1,7,8] and observations of our patients and data.

Proposed Mechanism for Non-Contact ACL Injury

When playing sports, athletes generate both quadriceps and hamstring muscle contraction in anticipation of changing directions or landing from a jump. The quadriceps muscle must generate greater forces than the hamstring muscles in order to absorb the landing forces and to keep the knee from being flexed too quickly. Furthermore, the gastrocnemius and soleus muscles are contracted to avoid unopposed dorsiflexion of the ankle. These muscle contractions are performed thousands of times during athletic practice and competition. When the timing of the foot plant is miscalculated, however, the foot is planted
a split second after the anticipated landing. In these instances, exaggerated and forceful quadriceps and gastrocnemius muscle contraction, which occurs in anticipation of the foot contacting the ground, causes the tibia to shift farther anteriorly than would have occurred had the foot struck the ground at the correct time. The anterior subluxation of the tibia on the femur then causes an internal rotation motion because of the downward and shorter slope of the lateral tibial plateau. At the time of foot plant and impact, the ACL is in a stretched position, which causes the sulcus terminalis of the lateral femoral condyle to come into uncontrolled contact with the posterolateral tibial plateau. We propose that, because of the downward and shorter slope of the lateral tibial plateau, the impact of the femur on the posterior part of the tibia causes a fulcrum action or “crow bar” type effect on the tibia, pushing it farther forward as the knee flexes and causing the stretched ACL to rupture.

CASE PRESENTATION

An 18 year old male was competing in motocross jumping when he lost control of his motorcycle in the air. He let go of the motorcycle and landed from a height of about 10 feet in the air. The patient stated that he knew the landing would be difficult because of the height and the angled ramp upon which he was landing. He threw his motorcycle forward before landing and tried to time his anticipated landing, which required a large quadriceps muscle contraction. The video of his injury showed that the patient’s knees were bent at about 30° of flexion and that he landed slightly off balance so that his right leg took on most of the force of the landing (Video 1). As the knee went into further flexion, the patient felt a pop and had immediate and severe pain. The physician on the scene of the injury stated that the knee was dislocated because the tibia was forward of the femur and the knee was in a subluxed locked position. With distraction of the joint, the tibia was reduced and the patient had relief of pain. He was taken to the local clinic where an arteriogram was performed, and the arteriogram was read as normal. His MRI revealed an isolated ACL tear and bone bruises on the lateral aspect of the femoral condyle and the posterolateral aspect of the tibial plateau (Figure 1). Plain radiographs also showed a dent in the lateral femoral condyle (Figure 2). The patient sought further treatment at our clinic. Arthroscopic evaluation of the joint at the time of ACL reconstruction revealed a posterior peripheral medial meniscus tear, a radial lateral meniscus tear, and a chondral fracture and dent in the lateral femoral condyle (Figure 3).

DISCUSSION

This case illustration shows the extreme of what might occur with ACL injuries involving deceleration with planting your foot to change directions or with landing from a jump. In our experience of treating over 5000 patients with ACL injuries, most ACL injuries occurred when an athlete was in close proximity to an opponent, which disrupted the injured athlete’s timing and coordination [1,4,5].

We propose that when an athlete mis-times a landing or foot-plant by planting the foot a split second after expected, the quadriceps and gastrocnemius muscle contractions, which usually occurs as part of mechanism to absorb the forces, have already occurred but without the usual joint compression forces that are present with ground contact. In the case described, the patient had applied maximal muscle forces in anticipation of his landing. At the time of actual foot plant, his tibia was so far forward that the terminal sulcus of the femur came in contact with the posterolateral tibial plateau and the compression of the lateral compartment and the lateral meniscus caused an indentation on the lateral femoral condyle. When the leg muscles contracted after the injury to pull the knee back together, the lateral meniscus got caught in the dent in the lateral femoral condyle, thus preventing the knee from fully reducing.

The dent in the lateral femoral condyle and the severity of the...
bone bruises shown on the MRI indicate that the tibia was shifted forward at the time of impact with the ground. We propose that when impact occurred, the anterior position of the femur, the blow of the femur on the posterior aspect of the tibia caused the knee to be forced into further flexion, which caused the ACL to be stretched enough to be torn. Figure 4 depicts a sequential illustration of this "crow bar" motion. This sudden shift in the position of the tibia may correspond to the feeling patients describe as "my knee gave out to the inside."

Bone bruises are seen in about 80% of MRIs following ACL injury [9,12-14]. The study by DeMorat et al. [7], found that a forceful quadriceps muscle contraction applied to an unfixed lower leg caused a large amount of elastic deformation in the ACL with a mean anterior displacement of 19.5 mm, which was enough to cause 3 complete ACL tears and 3 partial ACL tears in 13 knees studied. If a large unrestricted quadriceps muscle contraction can cause the tibia to be displaced anteriorly as much as 20 mm, then it is possible that the bone bruises typically seen on the lateral femoral condyle and the lateral tibial plateau in conjunction with ACL injuries are caused at the time of impact upon landing and deceleration with foot plant. The timing of the injury in relation to the foot plant typically is described by patients as happening slightly after foot plant, but not at the same time as foot plant. Videotape analysis studies have determined that ACL injuries mostly likely occur immediately after initial contact with the ground. Krosshaug et al. [4], estimated that the time of injury ranged from 17 to 50ms after initial ground contact. We propose that the pivot shift appearance observed by video analysis is the "crow bar" type injury we have described.

It is likely that there are several different types of mechanisms for non-contact ACL injury. This case illustrates a different type of mechanism for ACL than what has been previously proposed. Further biomechanical, animal, and clinical studies would be useful to further explain this theory; however, the clinical situation described in this report would be difficult to reproduce in the laboratory setting.

REFERENCES
