ANTERIOR CRUCIATE LIGAMENT RUPTURES

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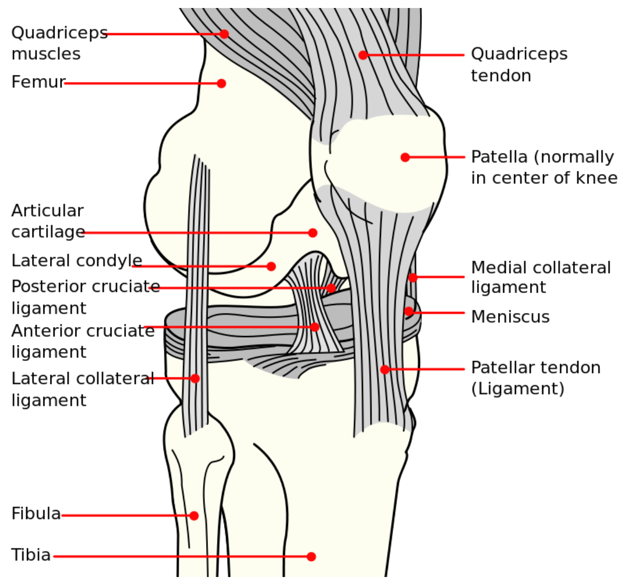
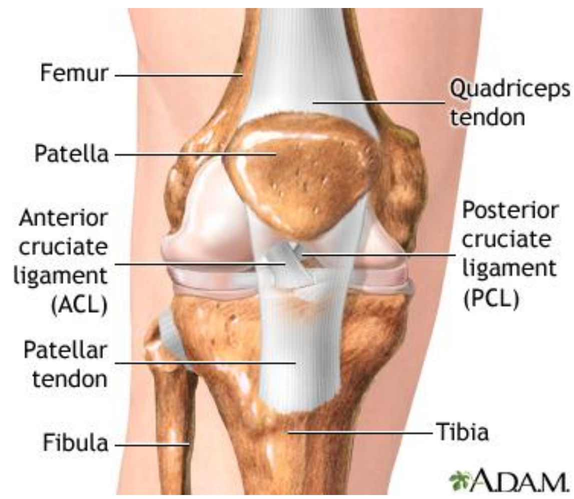
INTRODUCTION

The anterior cruciate ligament (ACL) originates at the medial wall of the lateral femoral condyle and inserts into the middle of the intercondylar area. It contributes significantly to the stabilization and kinematics of the knee joint. The femoral origin is oval and is located in the posterior aspect of the lateral femoral condyle. Therefore, it is difficult to visualize the femoral origin arthroscopically. This might be one reason for anterior malpositioning of the femoral bone tunnel during anterior cruciate ligament reconstruction. The position of the femoral origin is behind the center of rotation of the knee joint; therefore, it becomes tense when the knee is extended. The tibial insertion is oval and its center is nearly in the middle of the tibial plateau. Definite landmarks for tibial tunnel placement in anterior cruciate ligament reconstruction are the distance between the central insertion point at the intercondylar floor and the posterior cruciate ligament (7-8 mm) and the anterior horn of the lateral meniscus. The anterior cruciate ligament consists of multiple small fiber bundles. From a functional point of view, one can differentiate the anteromedial and posterolateral fiber bundles. The anteromedial fibers are tense during a greater range of motion than the posterolateral fibers. The main part of the anterior cruciate ligament consists of type I collagen-positive dense connective tissue. The longitudinal fibrils of type I collagen are divided into small bundles by thin type III collagen-positive fibrils. In the distal third, the structure of the tissue varies from the typical structure of a ligament. In this region, the structure of the tissue resembles fibrocartilage. Oval-shaped cells surrounded by a metachromatic extracellular matrix lie between the longitudinal collagen fibrils. The femoral origin and the tibial insertion have the structure of a chondral apophyseal enthesis. Near the anchoring region at the femur and tibia, there should be various mechanoreceptors, which might have an important function for the kinematics of the knee joint. The blood supply of the anterior cruciate ligament arises from the middle geniculate artery. The ligament is covered by a synovial fold where the terminal branches of the middle and the inferior geniculate artery form a periligamentous network. From the synovial sheath, the blood vessels penetrate the ligament in a horizontal direction and anastomose with a longitudinally orientated intraligamentous network. The distribution of blood vessels within the anterior cruciate ligament is not homogeneous. We detected three avascular areas within the ligament: Both fibrocartilaginous entheses of the anterior cruciate ligament are devoid of blood vessels. A third avascular zone is located in the distal zone of fibrocartilage adjacent to the roof of the intercondylar fossa (Petersen, W. et al. 2002).

The nerve supply to the ACL originates from posterior articular branches of the posterior tibial nerve, which originate from the sciatic nerve, formed by the mixed spinal nerves leaving the L5-S4 levels of the spinal cord. They contain 3 nerve fibre classes, such as mechanoreceptive sensory afferents (pressure), nociceptive sensory afferents (pain), and sympathetic efferent vasomotor fibres. Although the majority of fibers appear to have a vasomotor function, some fibers may serve a proprioceptive or sensory function (Arnoczky, S. 1983; Fromm, B. & Kummer, W., 1994)





RISK FACTORS

Several risk factors are associated with ACL injury. Such as gender, hormone, previous ACL injury (Smith et al., 2011), high risk sports like wakeboarding (Starr & Sanders, 2012).

Gender

Female athletes have been identified at increased risk of injuring their ACL during certain sports, with reported injury rates that are 3.5 times greater for basketball and 2.67 times greater for soccer when compared with male athletes who participate in these sports at similar levels of play.

Hormonal Risk Factors

Female sex hormone concentrations change over the course of the menstrual cycle, and the pattern of change may not be consistent from cycle to cycle. One rationale for studying sex hormones is based on the research that has identified estrogen and progesterone receptor sites on the ACL, and this has introduced the hypothesis that female sex hormones have an effect on the metabolism (synthesis and cleavage of matrix components), composition, and biomechanical properties of the ACL. Several studies have shown that female athletes have a higher incidence of ACL injury than male athletes in certain sports. It has been proposed that this is due to differences in physical conditioning, muscular strength, and neuromuscular control. Other suggested causes include differences in pelvis and lower extremity (leg) alignment, increased looseness in ligaments, and the effects of estrogen on ligament properties.

Previous ACL Injury

Previous ACL reconstruction is a risk factor for ACL injury in several prospective studies, both in the contralateral knee and for reinjury of the ACL graft.

High risk sports

The prevalence of ACL tears in this data set, 42.3%, is the highest reported in the literature for wakeboarding and one of the highest for any sport. The main mechanism of injury appears to involve axial compression while one lands in a provocative position; it is not related to a rotational force created by fixed bindings. The injury should be surgically repaired to effectively continue the sport. Further study is needed to determine if wakeboarding represents a high-risk sport for ACL injury.

ACL RUPTURE Mechanisms

In soccer one of the most common knee injuries is the anterior cruciate ligament (ACL) tear, which usually occurs through non-contact mechanisms. Female soccer players are at higher risk of sustaining non-contact ACL injuries than male soccer players. A good understanding of ACL loading mechanisms is the basis for a good understanding of the mechanisms of non-contact ACL injuries, which in turn is essential for identifying risk factors and developing prevention strategies. Current literature demonstrates that sagittal plane biomechanical factors, such as small knee flexion angle, great posterior ground reaction force and great quadriceps muscle force, are the major ACL loading mechanisms. A great posterior ground reaction force may be associated with a great quadriceps muscle force, which would cause great anterior draw force at the knee. A small knee flexion is associated with a large patella tendon-tibia shaft angle and ACL elevation angle, which would result in great ACL loading. Current literature also demonstrates that the ACL is not the major structure of bearing knee valgus-varus moment and internal-external rotation loadings. Knee valgus-varus moment and internal-external rotation moment alone are not likely to result in isolated ACL injuries without injuring other knee structures (Yu and Garrett 2007).

Mechanically, ACL injury occurs when an excessive tension force is applied on the ACL. A non-contact ACL injury occurs when a person themselves generates great forces or moments at the knee that apply excessive loading on the ACL. Therefore, an understanding of the mechanisms of ACL loading during active human movements is crucial for understanding the mechanisms and risk factors for non-contact ACL injuries.

Complete ACL rupture can induce other pathological knee conditions including knee instability, damage to menisci and the chondral surface, and osteoarthritis. Studies have repeatedly shown that patients with complete ACL rupture have chronic knee instability and secondary damage to menisci and chondral surfaces (Finsterbush, A. et al. 1990).

Markolf et al. (1995) also investigated the effects of anterior shear force at the proximal end of the tibia and knee valgus, varus, internal rotation and external rotation moments on the ACL loading of cadaver knees. The results of the study also showed that ACL loading due to the combined knee varus and internal rotation moment loading was greater than that due to either knee varus moment loading or internal rotation moment loading alone, and that the ACL loading due to combined knee valgus and external rotation moment loading was lower than that due to either knee valgus or external rotation moment loading alone. Finally, the results of this study showed that the ACL loading due to the anterior shear force and knee valgus, varus and internal rotation moments increased as the knee flexion angle decreased.

Quadriceps muscles are the major contributor to the anterior shear force at the proximal end of the tibia through the patella tendon.

In summary, the current literature clearly suggests that sagittal plane biomechanics are the major mechanism of ACL loading. Decreased knee flexion angle and increased quadriceps muscle force and posterior ground reaction force causing an increased knee extension moment are requirements for increased ACL loading. Although the external knee valgus moment has been demonstrated to be associated with ACL injuries, the current literature contains no evidence that knee valgus-varus and internal-external rotation moments can produce non-contact ACL injuries in and of themselves without these high sagittal plane forces.

DIOGNOSIS METHODS

The Lachman test is a clinical best test used to diagnose injury of the anterior cruciate ligament (ACL). It is recognized as reliable, sensitive, and usually superior to the anterior drawer test.

PATHOPHYSIOLOGY

ACL Ruptures About half of all injuries to the anterior cruciate ligament occur along with damage to other structures in the knee, such as articular cartilage, meniscus, or other ligaments. Injured ligaments are considered "sprains" and are graded on a severity scale. There are three grades refer to escalating severity of injury. Grade one is least detrimental and three showing greatest impact(Orthoinfo.aaos.org, 2014).

Grade 1 Sprains. The ligament is mildly damaged in a Grade 1 Sprain. It has been slightly stretched, but is still able to help keep the knee joint stable. There is no tear in connective tissue.

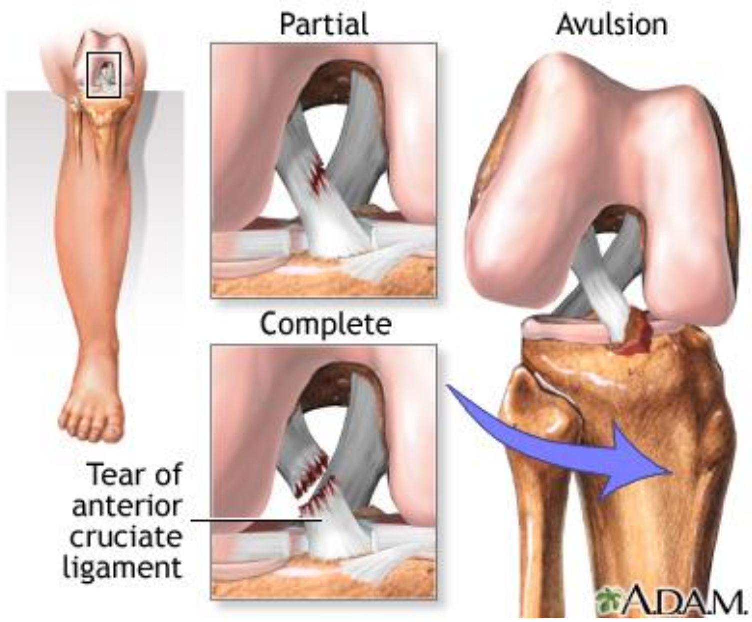
Grade 2 Sprains. A Grade 2 Sprain stretches the ligament to the point where it becomes loose. This is often referred to as a partial tear of the ligament. Fibers of ligament have been severed, allow joint to retain some stability but knee more prone to buckling.

Grade 3 Sprains. This type of sprain is most commonly referred to as a complete tear of the ligament. The ligament has been split into two pieces, and the knee joint is unstable.

Partial tears of the anterior cruciate ligament are rare; most ACL injuries are complete or near complete tears.

The anterior cruciate ligament can be injured in several ways:

- Changing direction rapidly
- Stopping suddenly
- Slowing down while running
- Landing from a jump incorrectly
- Direct contact or collision, such as a football tackle



An injury to the anterior cruciate ligament (ACL) may be described as a partial tear, complete tear or an avulsion (tearing away) from the bone attachments that form the knee.

SIGNS AND SYMPTOMS

When you injure your anterior cruciate ligament, you might hear a "popping" noise and you may feel your knee give out from under you. Other typical symptoms include:
Pain with swelling. Within 24 hours, your knee will swell. If ignored, the swelling and pain may resolve on its own. However, if you attempt to return to sports, your knee will probably be unstable and you risk causing further damage to the cushioning cartilage (meniscus) of your knee. Loss of full range of motion, tenderness along the joint line, and discomfort while walking.

TREATMENT

Treatment for an ACL tear will vary depending upon the patient's individual needs. For example, the young athlete involved in agility sports will most likely require surgery to safely return to sports. The less active, usually older, individual may be able to return to a quieter lifestyle without surgery(Orthoinfo.aaos.org, 2014).

Nonsurgical Treatment

A torn ACL will not heal without surgery. But nonsurgical treatment may be effective for patients who are elderly or have a very low activity level. If the overall stability of the knee is intact, your doctor may recommend simple, nonsurgical options.

It is highly recommend by a doctor that a brace to protect your knee from instability. To further protect your knee, you may be given crutches to keep you from putting weight on your leg.

Surgical Treatment: Surgery for anterior cruciate ligament (ACL) injuries involves reconstructing or repairing the ACL.

- Most ACL tears cannot be sutured (stitched) back together. To surgically repair the ACL and restore knee stability, the ligament must be reconstructed. Your doctor will replace your torn ligament with a tissue graft. This graft acts as a scaffolding for a new ligament to grow on.
- Grafts can be obtained from several sources. Often they are taken from the patellar tendon, which runs between the kneecap and the shinbone. Hamstring tendons at the back of the thigh are a common source of grafts. Sometimes a quadriceps tendon, which runs from the kneecap into the thigh, is used. Finally, cadaver graft (allograft) can be used.
- There are advantages and disadvantages to all graft sources. You should discuss graft choices with your own orthopaedic surgeon to help determine which is best for you.
- Because the regrowth takes time, it may be six months or more before an athlete can return to sports after surgery.

Unless ACL reconstruction is treatment for a combined ligament injury, it is usually not done right away. This delay gives the inflammation a chance to resolve, and allows a return of motion before surgery. Performing an ACL reconstruction too early greatly increases the risk of arthrofibrosis, or scar forming in the joint, which would risk a loss of knee motion.

REHABLIOTATION

Whether your treatment involves surgery or not, rehabilitation plays a vital role in getting you back to your daily activities. A physical therapy program will help you regain knee strength and motion. If you have surgery, physical therapy first focuses on returning motion to the joint and surrounding muscles. This is followed by a strengthening program designed to protect the new ligament. This strengthening gradually increases the stress across the ligament. The final phase of rehabilitation is aimed at a functional return tailored for the athlete's sport.

As the swelling goes down, a careful rehabilitation program is started. Specific exercises will restore function to your knee and strengthen the leg muscles that support it, e.g. physical therapy.

REFERENCES

1. Arnoczky, P. 1983. Anatomy of the anterior cruciate ligament. Clin Orthop Relat Res. 172:19-25.
2. Finsterbush, A, Frankl U, et al. 1990. Secondary damage to the knee after isolated injury of the anterior cruciate ligament. Am J Sports Med 18(5):475–479.
3. Fromm, B., & Kummer, W. 1994. Nerve supply of anterior cruciate ligaments and of cryopreserved anterior cruciate ligament allografts: a new method for the differentiation of the nervous tissues. Knee Surgery, Sports Traumatology, Arthroscopy, 2(2): 118-122.
4. Markolf, K. L, Burchfield D M, et al. 1995. Combined knee loading states that generate high anterior cruciate ligament forces. J Orthop Res 13: 930–935
5. Orthoinfo.aaos.org. Anterior Cruciate Ligament (ACL) Injuries-OrthoInfo - AAOS [Internet]. 2014. Available from: http://orthoinfo.aaos.org/topic.cfm?topic=a00549
6. Petersen W. and Tillmann B. 2002. Anatomy and function of the anterior cruciate ligament. Orthopade 31(8):710-718.
7. Smith, H., et al. 2012. Risk Factors for Anterior Cruciate Ligament Injury. A Review of the Literature---Part 2: Hormonal, Genetic, Cognitive Function, Previous Injury, and Extrinsic Risk Factors. Sports Health. 4(2): 155–161.
8. Starr, H.M and Sanders, B. 2012. Anterior cruciate ligament injuries in wakeboarding: prevalence and observations on injury mechanism. Sports Health. 4(4):328-332.
9. Yu, B. and Garret, W. E. 2007. Mechanisms of non-contact ACL injuries. Br J Sports Med. Aug 2007; 41(Suppl 1): i47–i51.