The posterior cruciate ligament (PCL) is twice as strong as the anterior cruciate ligament (Kennedy et al., 1976), and some consider it the prime stabilizer of the knee. Yet, in contrast to the vast amounts of literature on ACL injuries, relatively little attention has been paid to the PCL. This may be attributed to the fact that PCL tears are relatively uncommon. They account for only 5 to 20% of all knee ligament injuries.

Recently, however, reports of PCL injuries have been increasing. Data from the NFL draft physical examinations have consistently revealed that 2% of the athletes examined have an isolated posterior laxity (Parolie & Bergfeld, 1986). This may suggest improved diagnostic techniques including physical exam, magnetic resonance imaging, and arthroscopy.

The PCL is intra-articular and extra-synovial. It averages 38 mm in length and 13 mm in width (Girgis et al., 1975) and attaches anterolaterally on the medial femoral condyle. Its tibial attachment is in a depression in the posterior tibia between the two tibial plateaus about 1 cm below the tibial surface.

The PCL is augmented by two additional meniscofemoral ligaments: The anterior one, or ligament of Humphrey, can be 1/3 the size of the PCL and is found in 36% of knees, while the posterior one, or ligament of Wrisberg, can be 1/2 the diameter of the PCL and is found in 35% of knees (Van Dommelen & Fowler, 1989). Some researchers have concluded that the meniscofemoral ligaments contribute significantly to knee stability with isolated PCL injury.

Mechanisms of PCL Injury

The PCL may be injured through several mechanisms, the most common being an anterior-to-posterior directed blow. This occurs in sports in which the flexed knee strikes the ground. If the foot is plantar-flexed, force is transmitted through the tibial tubercle, injuring the PCL. This is the same mechanism commonly seen in auto accidents whereby the knee strikes the dashboard.

These mechanisms primarily produce isolated tears because the posterior capsule is lax and the taut PCL is in a relatively horizontal position. Forced hyperflexion may also result in PCL rupture, owing to the fact that the anterolateral bundle tightens with flexion.

Other mechanisms of PCL
injury are more likely to result in combined ligamentous injuries about the knee. Forced hyperextension will result in ACL rupture, followed by PCL rupture. The PCL may be injured with severe valgus or varus stress. The collateral ligaments are the primary restraints to these types of forces, but the PCL may be injured following disruption of one of the collaterals. This helps explain why PCL injuries are commonly associated with injuries to the posterolateral ligament complex.

Following an acute isolated PCL injury, the athlete most often has a mild hemarthrosis and complains of minimal posterior knee pain. There may be an abrasion along the anterior aspect of the knee. The injured knee’s range of motion is usually limited from approximately 10 to 60° (Loos et al., 1981).

Acutely, the athlete will walk with a slightly flexed knee to avoid terminal extension and external rotation. It is not uncommon for the athlete to report “something just doesn’t feel right.” Giving way, chronic pain and recurrent effusion may be seen in chronic PCL injuries and can be secondary to degenerative change.

The increase in posterior translation is twice as large at 90° than at 30° with an isolated PCL injury. Thus the posterior drawer sign is the best way to assess PCL integrity. In addition to displacement, quality of endpoint should be noted. Isolated PCL injury will result in approximately 10 mm or less of posterior translation.

With intact meniscofemoral ligaments and internal rotation of the tibia, posterior translation should decrease significantly when performing this test (Photo 1).

Other clinical exams may include a posterior sag sign, valgus/varus instability, and an active quadriceps test. Magnetic resonance imaging, x-ray, exam under anesthesia, and arthroscopy are other diagnostic techniques that may be valuable.

Management Protocol

Indications for surgical management of the PCL-deficient knee is dependent on the results of the exam and the outcome of conservative care.

Isolated PCL tears with posterior translation less than 15 mm are rarely reconstructed because there is no one procedure that has provided consistently excellent results (Hughston & Degehardt, 1982). Surgery is indicated, however, if there is an acute avulsion of the tibial insertion, combined ligament instabilities, loss of secondary restraints (>15 mm of posterior translation), or failed rehabilitation.

Regardless of the management protocol selected, athletes sustaining a PCL injury must be counseled about the significance of the deficit. Discussion should emphasize that the knee will never be normal again, there is an increased risk of medial joint arthritis, and that, to date, no one can accurately predict the clinical course of the injury.

In a recent long-term review of PCL injuries that did not undergo surgery, however, Cross and Powell (1984) found that 86% of the patients who injured their PCL in sports had good to excellent results, while only 8% had good results following auto accidents or falls.

Rehabilitating the athlete with an acute PCL injury involves minimizing the acute symptoms of pain, effusion, and decreased range of motion.

Intermittent application of ice packs, compression, anti-inflammatory medication, and possible knee immobilization with crutch-assisted gait are indicated the first week following injury. As pain permits, knee range of motion and quadriceps muscle tone should be emphasized. Closed kinetic chain exercising of
the lower extremity such as step-ups, leg presses, and stair climbing are recommended, as opposed to open chain activities that would increase the force across the patellofemoral joint (Photo 2).

Inclusion of a proprioceptive, running, and agility program is a prerequisite for return to sports. Most well-motivated athletes may return to preinjury performance levels at 4 to 6 weeks postinjury.

The most important component of rehabilitation is quadriceps emphasis; the hamstrings should not be activated until a full range of motion is attained and acute symptoms are under control. Return to play is based on a full active range of motion, quadriceps strength equal to the uninvolved side (Parolie & Bergfeld, 1986), satisfactory muscular endurance, proprioceptive competence, and confidence in activities associated with the sport.

There is little relationship between isolated posterior instability and the ability to return to sport. An athletic therapist who, through physical exam, determines the degree of instability and integrity of secondary restraints, constructs a sound rehabilitation program, and counsels the athlete on a lifelong commitment to maintenance of the involved knee can anticipate an excellent result.

References


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