Complications Associated with Posterior Cruciate Ligament Reconstruction and Avoiding Them

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Abstract

Posterior cruciate ligament (PCL) injuries often occur as part of a multiligament injury pattern and can present a significant challenge to the treating surgeon. When PCL reconstruction is indicated, complications can arise in the intraoperative and postoperative period that lead to poor outcomes. These complications include neurovascular injury, fracture, compartment syndrome, persistent posterior laxity, motion loss, residual knee pain, osteonecrosis, and heterotopic ossification. The purpose of this review is to highlight complications associated with PCL reconstruction and strategies to avoid them.

Keywords

- posterior cruciate ligament
- posterior cruciate ligament reconstruction
- complications
- multiligament knee injury
- compartment syndrome
- heterotopic ossification
- osteonecrosis

In recent decades, outcomes following posterior cruciate ligament (PCL) reconstruction have improved with better understanding of PCL anatomy and biomechanics and new surgical techniques. However, despite these advances, PCL reconstruction remains fraught with potential complications. In a review of 92,565 arthroscopies in the American Board of Orthopaedic Surgery database, PCL reconstruction was noted for a complication rate of 20.1% compared with 9.1% for anterior cruciate ligament (ACL) reconstruction and 4.7% for all arthroscopic knee procedures.[1] In light of this, the purpose of this review is to describe risks specific to PCL reconstruction and strategies to avoid them. These complications include neurovascular injury, fracture, compartment syndrome, persistent posterior laxity, motion loss, residual knee pain, osteonecrosis, and heterotopic ossification (►**Table 1**).

Neurovascular Injury

Neurovascular injury may occur at the time of PCL injury or during PCL reconstruction. Popliteal artery injury, consisting of an intimal tear, laceration, or occlusion, occurs in up to one-third of knee dislocations.[2] If indicated, vascular assessment should be performed by physical examination of distal pulses, measuring the ankle-brachial index, or angiography.

The popliteal artery passes through the popliteal fossa in close proximity to the PCL tibial attachment and is at risk of injury during creation of the tibial tunnel. A cadaveric study simulating arthroscopic conditions found that the popliteal artery was located a mean sagittal distance of 29.1 ± 11 mm (range = 18–55 mm) from the mid-PCL, while the mean sagittal distance from the PCL fovea was a mere 9.7 ± 5 mm (range = 3–16 mm).[3] In a similar cadaveric study, the

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Table 1 Complications associated with posterior cruciate ligament reconstruction

Complication	Time period
Neurovascular injury	Intraoperative
Fracture	Intraoperative/early postoperative
Compartment syndrome	Early postoperative
Persistent posterior laxity	Postoperative
Motion loss	Postoperative
Residual knee pain	Postoperative
Osteonecrosis	Postoperative
Heterotopic ossification	Postoperative

distance between the PCL insertion and popliteal artery was 7.6 mm in the axial plane and 7.2 mm in the sagittal plane on magnetic resonance imaging (MRI) at five knee flexion angles ranging from 0 to 100 degrees.[4] In addition, a line was drawn along the projected path of a PCL tibial tunnel guide pin. The pin passed through popliteal artery in 100% of specimens at 0, 30, 45, 60, and 90 degrees, but only 60% of specimens at 100 degrees, suggesting that drilling the tibial tunnel in higher knee flexion confers some degree of protection.

Several case reports have described popliteal artery laceration and occlusion during and after PCL reconstruction.[5,6] Mechanism of injury may include direct penetration from the transtibial guidewire or reamer, manipulation of the posterior joint capsule and adjacent tissue, arterial spasm, atherosclerotic plaques, and use of a pneumatic tourniquet.[7]

There are many strategies to minimize risk of neurovascular damage during reconstruction. Surgeons must develop a knowledge of and appreciation for the complex anatomic relationships of the popliteal artery and tibial nerve in relation to the tibial PCL attachment. Adequate debridement of the footprint and mobilization of adjacent soft tissues allows for direct visualization of guidewire and reamer passage. Fluoroscopy and/or direct visualization via a posterior-medial portal can also be used to assess guidewire and reamer positioning. Other techniques include a posteromedial safety incision to facilitate direct visualization and palpation for protection of the neurovascular bundle. Additionally, arthroscopic debridement of the PCL facet and posterior capsular release allows for mobilization of the adjacent soft tissues, thereby increasing the distance of the popliteal artery from the PCL footprint.[8]

Fracture

Fracture of the tibia, femur, and patella have been reported during PCL reconstruction.[9–11] Excessively, large tunnel diameter increases risk of fracture in the femur and tibia. Care should also be taken to avoid tunnel convergence in multiligament reconstructions by employing guidelines for tunnel depth and trajectory (►**Fig. 1**).[12,13] Risk of tibial tunnel convergence is greatest between the PCL tunnel and posterior oblique ligament and superficial MCL tunnels. Risk of femoral

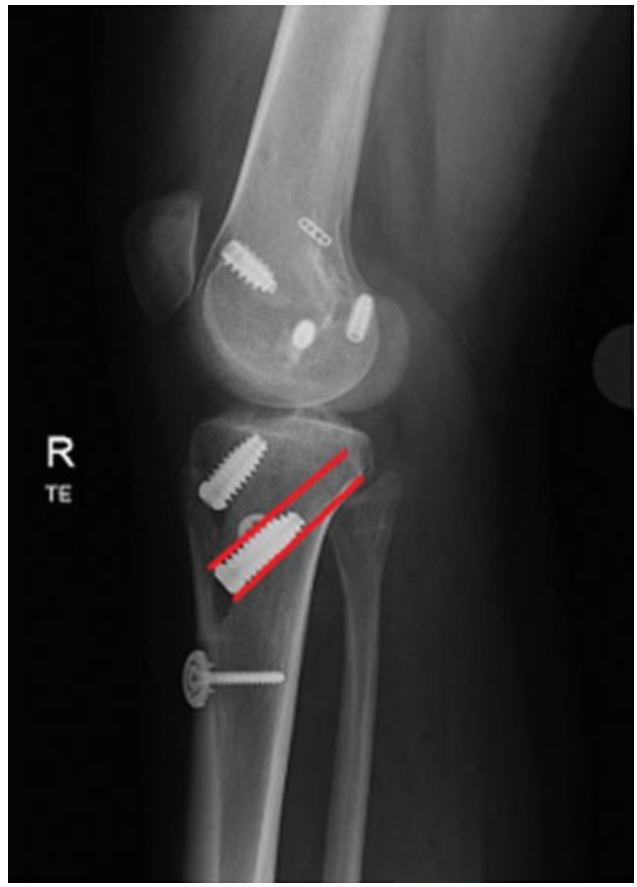

Fig. 1 Lateral knee radiograph showing appropriate tibial tunnel placement low on the posterior cruciate ligament facet to create a better lever arm.

tunnel convergence is greatest between the PCL tunnel and superficial MCL tunnel. When performing a double-bundle PCL reconstruction, an adequate bone bridge and divergent trajectory between femoral tunnels minimizes risk of fracture.

Bone-patellar tendon-bone autograft is a commonly used graft for single bundle PCL reconstruction. Risk of patella fracture during harvest has been well described in the ACL reconstruction literature, occurring in 0.2 to 2.3% of cases.[7,10,14–16] Strategies to minimize risk of patella fracture include limiting depth of bony resection to one-third of the depth of the patella, making initial cuts using a saw rather than osteotome,[17] using a curved osteotome to create round cornered plugs,[18] limiting bone plug size to no greater than 25 to 30 mm by 10 mm, and bone grafting the harvest site with either tunnel reamings or bone plug trimmings.[19]

Compartment Syndrome

Compartment syndrome can occur following a lower extremity injury or surgery. During PCL reconstruction, fluid extravasation through a defect in the joint capsule into the lower leg can increase lower extremity compartment pressures.[20] Maintaining the pump flow in a low setting or using gravity flow cane minimize the risk of excessive fluid extravasation. Improper patient positioning and padding can also contribute to the development of compartment syndrome. Positioning the

contralateral leg in a well holder in a flexed and abducted position increases pressure in the gluteal compartments and gluteal compartment syndrome has been reported during PCL reconstruction.[21] Placing the contralateral extremity flat on the operating table with the heel off the bed increases surface area of gluteal contact, thereby lowering compartment pressure.[7] Maintaining adequate mean diastolic pressure and shorter operative times also decreases risk. Postoperatively, patients deemed high risk for compartment syndrome must be monitored with serial compartment check exams. Long-acting lower extremity nerve blocks should be avoided to ensure accurate assessment of sensation, motor function, and changes in pain. Finally, vascular status of the limb must be closely monitored as delayed compartment syndrome has been reported after the rupture of a popliteal artery pseudoaneurysm following PCL reconstruction.[22]

Persistent Posterior Laxity

Persistent posterior laxity is the most common complication after PCL reconstruction, resulting from several causes including missed concurrent ligament injuries, technical errors during reconstruction, decreased tibial slope, overly aggressive physical therapy, and patient noncompliance. The most commonly missed injury is a concurrent posterolateral corner injury to the lateral collateral ligament, popliteofibular ligament, and/or popliteus tendon.[9] Unrecognized posterolateral corner injury results in increased forces on the PCL graft, which increases the risk of later graft failure.[23,24] In one series, posterolateral ligament deficiency contributed to PCL graft failure in 40% of cases undergoing revision PCL reconstruction.[25] In addition, technical errors during reconstruction, such as improper graft tension or placement of the femoral reconstruction tunnel too posterior and proximal, can lead to residual laxity. Recent evidence has shown that patients with a decreased tibial slope are at increased risk of sustaining a PCL tear.[26] Biomechanical data have shown that decreasing tibial slope results in a linear increase in forces across PCL grafts regardless of flexion angle.[27] For this reason, tibial slope should be evaluated in the revision setting and slope increasing proximal high tibial osteotomy considered. Finally, overly aggressive physical therapy can lead to graft attenuation or loss of fixation. Patient noncompliance with activity restrictions in the early postoperative period can also lead to graft failure.

Motion Loss

Compared with motion loss after ACL reconstruction, in which loss of extension is more common than loss of flexion, motion loss after PCL reconstruction more commonly results in flexion deficits. In a systematic review of outcomes following isolated and combined PCL reconstruction, loss of flexion occurred in 0 to 26.4% of patients, while loss of extension occurred in 0 to 8% of patients.[28] Causes of restricted motion can be broadly divided into three categories: soft tissue factors (e.g., suprapatellar pouch adhesions and arthrofibrosis), technical error (e.g., too distal and anterior placement of the femoral tunnel, poor isometrics of a concurrent MCL reconstruction, and improper graft tensioning), and lack of progress with rehabilitation (e.g., due to poor compliance, motivation, or an inexperienced therapist).[7,9,29] Firstline treatment of suprapatellar pouch adhesions and arthrofibrosis is nonoperative with physical therapy. If range of motion does not improve, arthroscopic lysis of adhesions and/or gentle manipulation under anesthesia can be performed beginning at approximately 12 weeks postoperatively.[30–33]

Residual Knee Pain

In the early postoperative period, pain can occur at the surgical incisions, graft harvest sites, and from symptomatic hardware.[7] Removal of hardware may be considered beginning approximately 12 months after surgery. Neuropathic pain can result from nerve injury (e.g., common peroneal or tibial nerve) at the time of the initial injury or from iatrogenic injury during reconstruction. At long-term follow-up, PCL graft failure and persistent posterior laxity can lead to patella baja, altered tibiofemoral and patellofemoral kinematics, and increased contact forces in the medial compartment and patellofemoral joint.[34] Over time, these changes manifest as anterior knee pain due to degenerative joint disease in the anteromedial tibiofemoral and patellofemoral joint, reactive synovitis, and persistent effusion. A complete history, physical exam, and imaging work-up including repeat plain radiographs, MRI, and/or stress radiographs should be completed in these cases.

Osteonecrosis

Avascular necrosis of the medial femoral condyle has been reported after PCL reconstruction.[35] In one such case, a 28-year-old professional football player sustained a combined grade III PCL and medial collateral ligament (MCL) injury. The patient underwent single bundle PCL reconstruction using a 12-mm patellar-ligament graft passed through a 12-mm tunnel in the medial femoral condyle. The graft was fixed on the femoral side with an interference screw, and the passing sutures attached to the bone plug were passed through a button secured on the supracondylar ridge of the medial femoral condyle. Concurrently, the MCL was reconstructed by using a semitendinosus autograft secured at the femoral attachment using an interference screw and soft tissue washer. The patient recovered appropriately during the immediate postoperative period and underwent removal of the MCL interference screw and washer at 7 months. After resuming unrestricted activity, he developed medial joint line pain. Radiographs and MRI revealed a clear defect over the distal medial femoral condyle articular surface consistent with avascular necrosis. The patient underwent curettage and bone grafting through a transcondylar approach and was able to return to professional football.

The etiology of medial femoral condyle avascular necrosis remains controversial, but several theories have been posited. First, the medial femoral condyle appears more prone to avascular necrosis due to a poor circulation and less collateral supply compared with the lateral condyle.[36] The medial femoral condyle has both extraosseous and intraosseous blood supply. The

extraosseous supply is provided by the superior medial genicular artery and branches of the popliteal artery. The intraosseous supply is limited to a single nutrient vessel with a broad watershed area of limited vascularity. Compared with ACL reconstruction, PCL reconstruction tunnels are positioned more distal and closer to the articular surface, perhaps compromising the tenuous vascularity of the medial femoral condyle subchondral bone. Reconstruction tunnels should be relatively vertical to avoid a horizontal tunnel, which could devascularize the subchondral bone. If performing a double-bundle reconstruction, an adequate bone bridge must be left between tunnel.[9]

Heterotopic Ossification

Heterotopic ossification of the posterolateral joint capsule is a rare complication following PCL reconstruction. Patients will often report decreased range of motion and pain over the posterolateral aspect of the knee. The etiology is thought to arise from injury to the posterolateral joint capsule at the time of the initial injury or from reaming the femoral tunnels.[7] Prophylaxis consisting of anti-inflammatory medications and low-dose radiation have not been investigated in this context.[20] Firstline treatment for mild-to-moderate cases consists of physical therapy and manipulation under anesthesia. In severe and refractory cases, lysis of adhesions, soft tissue release, and resection can be performed after maturation of the heterotopic focus, typically after at least 12 months.[37]

Conclusion

Posterior cruciate ligament injuries requiring reconstruction are rare and often occur as part of a multiligament injury pattern. Reconstruction poses several challenges owing to the close proximity of the PCL to important neurovascular structures in the popliteal fossa and technical challenges associated with executing a complex reconstruction. Complications in PCL surgery occur at a greater frequency than during ACL reconstruction and may include neurovascular injury, fracture, compartment syndrome, persistent posterior laxity, loss of motion, residual knee pain, osteonecrosis, and heterotopic ossification. Surgeons must counsel patients regarding these well-described risks and employ strategies to mitigate their effect on achieving a satisfactory outcome.

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