Case Report

Popliteal Venotomy During Posterior Cruciate Ligament Reconstruction in the Setting of a Popliteal Artery Bypass Graft

Venu M. Nemani, M.D., Ph.D., Rachel M. Frank, M.D., Keith R. Reinhardt, M.D., Cecilia Pascual-Garrido, M.D., Adam B. Yanke, M.D., Mark Drakos, M.D., and Russell F. Warren, M.D.

Abstract: Injury to the vascular structures in the popliteal fossa during arthroscopic cruciate ligament reconstruction can be limb threatening or even life threatening. We present the first report, to our knowledge, of an isolated injury to a popliteal vein during arthroscopic posterior cruciate ligament reconstruction. Unfortunately, the venotomy led to cardiopulmonary arrest and flash pulmonary edema in this patient. Preoperative planning is paramount to assess risk of injury to vascular structures, which may be increased in patients who have had prior procedures on the affected knee. Furthermore, vascular surgery consultation preoperatively after a magnetic resonance angiogram or venogram and avoiding the use of epinephrine in the arthroscopy fluid should be considered when performing these higher-risk procedures.

Arthroscopic ligament reconstruction has been widely and successfully used for injuries to the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL). Although these procedures are quite safe, with an overall complication rate of 0.56%, injury to the vessels in the posterior aspect of the knee can be a devastating, albeit rare, complication. Reconstruction of the PCL, in particular, requires a thorough appreciation of normal posterior knee anatomy, because the popliteal artery typically lies 5.9 to 7.6 mm posterior to the posterior tibial cortex. Isolated injuries to the popliteal vein, however, have not been reported during PCL reconstruction, likely because of its location posterior to the artery.

We present the case of an isolated popliteal venotomy sustained during PCL reconstruction that led to intraoperative cardiopulmonary arrest and flash pulmonary edema. We believe important factors contributing to this patient’s unique course were her previous popliteal artery bypass graft causing aberrant vascular anatomy with the formation of additional dilated veins, as well as the use of arthroscopy fluid containing epinephrine. Although several reports on popliteal artery injury are available, to our knowledge, this is the first report of an isolated popliteal vein injury sustained during arthroscopic PCL reconstruction. The patient was informed that data concerning this case would be submitted anonymously for publication.

CASE REPORT

A 19-year-old woman was involved in a motorcycle accident that resulted in a right native hip dislocation and a left knee dislocation with vascular injury. Initially, she was taken to a regional medical center where, in addition to undergoing closed reduction of
her right hip and left knee, she underwent left popliteal artery bypass with a contralateral saphenous venous graft for popliteal insufficiency, as well as placement of an external fixator to maintain the reduction of the left knee. When she presented to us 4 weeks later, radiographs of the left knee showed widening of the medial compartment and vascular clips. Magnetic resonance imaging of the left knee showed complete disruption of the ACL, PCL, and medial collateral ligament, as well as a bucket-handle tear of the lateral meniscus. The patient was taken to the operating room at 6 weeks after injury for removal of the external fixator and an examination under anesthesia, which showed a 2+/1001 anterior drawer, 1+ posterior drawer, medial opening to valgus stress, and range of motion limited to 80° of flexion.

After completing 4 months of physical therapy to increase the patient’s quadriceps strength and range of motion, she was taken to the operating room to undergo a single-stage multiligament reconstruction of the ACL, PCL, and medial collateral ligament, with concomitant meniscal debridement. After uneventful induction of anesthesia with spinal anesthesia and a left femoral nerve block, the patient was positioned supine on the operating table with a well-padded tourniquet placed on the superior thigh. Of note, the tourniquet was not inflated for the procedure. Intraoperatively, during PCL stump debridement, an anomalous branch of the popliteal vein was noted to be intimately adherent to the insertion fibers of the PCL near the tibia. After complete dissection of the PCL from the tibial footprint, a 5-mm venotomy was noted in the vessel (Fig 1). There was no extravasation of blood noted from the venotomy, which was likely prevented by the fluid pressure within the knee. During preparation of the vessel for repair, the patient’s blood pressure acutely rose to 200/100 mm Hg, which was believed in part to be due to entrance of the epinephrine in the arthroscopy fluid into the systemic circulation through the venotomy. On exchange to arthroscopy fluid containing saline solution alone, as well as administration of a β-blocker, the patient had a cardiopulmonary arrest, and cardiopulmonary resuscitation (CPR) was initiated. She quickly regained her pulse but required intubation for coarse breath sounds due to flash pulmonary edema. With administration of intravenous furosemide and subsequent diuresis, her pulmonary status improved, and attention was returned to the vascular repair.

A vascular surgeon evaluated the patient, and after standard preparation and draping, a second diagnostic arthroscopy was performed and the venotomy site was easily identified. By use of a probe in the posteromedial portal for retraction of soft tissues, the popliteal venotomy site was ligated arthroscopically with 4 vascular clips (Ligaclip; Ethicon Endo-Surgery, Cincinnati, OH) without difficulty by the senior attending orthopaedic surgeon (Fig 2). At this point, the decision was made to defer multiligament reconstruction to a later date. The patient’s incisions were closed, she underwent placement of a knee immobilizer, and she was transferred with the endotracheal tube in place to the recovery room. Doppler examination postopera-
tively confirmed the presence of both posterior tibial and dorsalis pedis pulses. A chest radiograph showed flash pulmonary edema (Fig 3), which improved quickly with ongoing diuresis. The patient was extubated on postoperative day 1 and discharged home on postoperative day 3.

A magnetic resonance imaging scan performed 3 months postoperatively showed mild venous dilation of the popliteal vein distal to the knee joint line, as well as close apposition of the popliteal vein and the posterior joint capsule, without an intervening fat plane (Fig 4). Once the patient had returned to her baseline preoperative medical state, she was again scheduled to undergo completion of the multiligament reconstruction. A vascular surgeon was present during the case, given the previous complication. Knowing the proximity of the vasculature to the posterior capsule in this patient, care was taken to protect the posterior vessels with a periosteal elevator during PCL guide pin placement and tunnel drilling for the tibial insertion of the PCL graft. Of note, a tourniquet was not inflated for this procedure. The procedure was carried out without complications, and the patient was discharged home on her third postoperative day.

Four months after reconstruction, the patient had regained full knee range of motion and, on examination, showed stable collateral ligaments and a negative Lachman test. At 4 years’ follow-up, the patient had excellent range of motion from 0° to 140°. She had a 1+ posterior drawer and a negative Lachman test. She had mild degenerative changes noted in the lateral compartment on routine office radiographs.

DISCUSSION

We present the case of a popliteal venotomy sustained during PCL reconstruction that led to acute intraoperative cardiopulmonary arrest and flash pulmonary edema. This case is unique even among the most experienced orthopaedic surgeons for several reasons. First, the posterior knee anatomy in this patient was abnormal as a result of her previous popliteal artery bypass grafting procedure, which resulted in dilated anomalous popliteal veins adjacent to the posterior knee capsule. The patient’s altered anatomy put her at higher risk for such an intraoperative injury to occur during PCL tibial footprint debridement. Second, the particular course of events that occurred during the case, including the patient’s drop in blood pressure, development of pulmonary edema, need for CPR, and definitive ligature of the vein, is not commonly encountered among orthopaedic surgeons.

Although serious complications during arthroscopic knee surgery are rare, with an overall complication rate of 0.56%,1 complications can include instrument breakage, infection, hemarthrosis, iatrogenic chondral damage, and injuries to nerves, ligaments, or vessels. Damage to the popliteal artery or vein, notably, is one
of the most feared complications because it can result in both limb-threatening and life-threatening hemorrhage. The reported incidence of vascular complications after arthroscopic knee surgery is exceedingly small, with a large survey of experienced arthroscopicists reporting only 9 injuries to the popliteal vessels in greater than 375,000 procedures.\(^1,3\) However, this may be an underestimation of the true incidence, given that these data were collected retrospectively. There have been multiple published case reports in the literature of injury to the popliteal artery during arthroscopic cruciate ligament reconstruction. Makino et al.\(^4\) reported a popliteal artery laceration occurring in a 17-year-old boy during double-bundle PCL reconstruction. The vessel was repaired with an interposition saphenous vein graft, and the patient’s pulses returned. Wu et al.\(^5\) reported an acute occlusion of the popliteal artery after double-bundle PCL reconstruction. The patient was treated with close observation only because the patient refused either intra-arterial thrombolytic therapy or emergent surgical intervention. The patient’s pulses and sensation gradually returned over the course of 12 hours, and the patient did well. Janssen et al.\(^6\) reported a pseudoaneurysm formation of the popliteal artery in a patient after ACL reconstruction with bicortical tibial screw fixation. The patient had sensory deficits from nerve compression by the pseudoaneurysm, which incompletely resolved after pseudoaneurysm repair.

Despite the low incidence of popliteal vessel injury, there are indeed several steps in cruciate ligament reconstruction that place these structures at risk for damage where extra care must be taken intraopera-

<table>
<thead>
<tr>
<th>Procedure Step</th>
<th>Tips to Avoid Inadvertent Vascular Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Debridement of PCL stump from posterior tibia, manipulation of posterior capsule</td>
<td>Keep arthroscopic pump pressure to a minimum to reduce joint distension.</td>
</tr>
<tr>
<td>Advancement of guidewire for tibial tunnel placement in PCL reconstruction</td>
<td>Use a spade-tipped guidewire or a tapered drill bit; advance instrument under image intensification.</td>
</tr>
<tr>
<td>Drilling of tibial (PCL) or femoral (ACL) tunnels for cruciate ligament reconstructions</td>
<td>Advance drill under image intensification; drill tibia with knee in flexion during PCL reconstruction.</td>
</tr>
</tbody>
</table>

\(^{1,3}\) Indeed, the normal distance from the posterior margin of the PCL to the posterior joint capsule is only 1.7 mm,\(^7\) and in our case, the neurovascular structures were adhered directly to the posterior capsule because of postoperative scarring from the previous popliteal artery bypass grafting, placing the patient at higher risk for vascular complications. Several techniques can be used to avoid damage to vascular structures, such as using a spade-tipped guidewire or a tapered drill bit, advancing these instruments under image intensification, or making a posteromedial safety portal to protect the neurovascular structures directly.\(^8,9\) More recently, Ahn et al.\(^10\) have described a technique to elevate the PCL off the posterior joint capsule with a shaver introduced through the posterolateral portal and a right-angle rasp through the anteromedial portal, which resulted in a significantly increased distance between the PCL and the popliteal artery. The arthroscopic infusion pump pressure can be kept to a minimum to decrease the degree of joint distention to avoid compressing the posterior joint capsule against the popliteal vessels. Judicious use of posterior accessory portals is recommended, making sure not to place these portals too far anteriorly to avoid anterior-to-posterior positioning of instruments.\(^11\) Lastly, drilling the tibial tunnel in PCL reconstruction with the knee in increased flexion moves the popliteal artery posteriorly away from the joint capsule and should decrease the risk of vascular injury.\(^2\) Although injury to the structures in the popliteal fossa is the most concerning complication, there is also potential for injury to the saphenous and peroneal neurovascular bundles with medial and lateral incisions, respectively.\(^11\) Injury to these structures can also lead to undesirable results and must be avoided.

### TABLE 2. Key Points for Safe Arthroscopic Cruciate Ligament Reconstruction in Setting of Altered Anatomy From Prior Operations

<table>
<thead>
<tr>
<th>Procedure Step</th>
<th>Tips to Avoid Inadvertent Vascular Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider preoperative magnetic resonance angiography or formal angiography to clearly define posterior vascular anatomy.</td>
<td></td>
</tr>
<tr>
<td>Have a vascular surgeon immediately available for intraoperative assistance if vascular injury occurs.</td>
<td></td>
</tr>
<tr>
<td>Do not elevate a tourniquet to allow for prompt detection of vascular injury.</td>
<td></td>
</tr>
</tbody>
</table>
To our knowledge, the case presented is the first report in the literature of isolated popliteal vein injury during arthroscopic PCL reconstruction. It is highly unusual to have injury to the popliteal vein without injury to the artery, because the artery typically lies more anteriorly in the popliteal fossa. However, they did previously report a case of laceration to both the popliteal artery and vein during arthroscopic total synovectomy in a patient with pigmented villonodular synovitis. As stated previously, postoperative changes from the previous saphenous vein bypass likely caused development of collateral veins anteriorly adjacent to the joint capsule, which placed them at higher risk for laceration during the procedure.

In addition to the unique presentation of an isolated popliteal vein laceration, the subsequent hemodynamic instability that occurred in this patient is also not commonly encountered in the literature. In our case there was no significant bleeding associated with the venotomy, because this was prevented by the increased intra-articular pressure from the arthroscopy fluid. After the venotomy, the patient had a rapid rise in blood pressure to 200/100 mm Hg, and it is our impression that the epinephrine used in the irrigation fluid played a major role in the acute hypertension, because it gained rapid access to the venous circulation through the venotomy. After removal of the epinephrine from the irrigation solution and administration of an intravenous β-blocker, the patient then had an acute drop in blood pressure, followed by flash pulmonary edema, requiring a brief course of CPR. This unusual series of events has been described once previously in a case report by Mazzocca et al., in which an otherwise healthy patient similarly had epinephrine-induced pulmonary edema during arthroscopic knee surgery. In that case, on drilling of a tibial tunnel in preparation for meniscal allograft transplantation, the patient had a sudden onset of hypertension and tachycardia, which were subsequently treated with an intravenous β-blocker. Similar to our case, flash pulmonary edema subsequently developed in their patient. As the authors elegantly described in their report, epinephrine causes activation of α-adrenergic receptors, which in turn increases peripheral vascular resistance and thus increases blood pressure, which ultimately shunts blood from the peripheral vasculature to the pulmonary vasculature. The body responds to this by increasing cardiac output through a compensatory increase in heart rate and/or cardiac contractility. The administration of β-blockers thus becomes counterproductive because this blocks these normal compensatory mechanisms and can cause pulmonary congestion. As a result, Mazzocca et al. noted that in these situations, β-blockers should be avoided and the treatment of choice is selective α-adrenergic blockers and vasodilators, with diuretics such as furosemide used as needed for symptom control. Although the authors postulated that the combination of epinephrine in the arthroscopy fluid and its immediate access to the venous circulation once the tibial tunnel was drilled is what ultimately led to their patient’s hemodynamic instability, the role of epinephrine is less clear in our case. In our patient the flash pulmonary edema also could have been caused by acute iatrogenic hypervolemia, with the popliteal venotomy acting as a portal for a large volume of saline solution to enter the intravascular space.

Despite its low incidence, injury to the popliteal vessels can be a devastating complication of arthroscopic PCL repair. One must maintain a high index of suspicion and initiate prompt workup and treatment if injury is suspected. Patients with a history of trauma or operations involving the posterior knee should receive preoperative magnetic resonance angiography or formal angiography to clearly define the posterior knee vascular anatomy before ligamentous reconstruction. Furthermore, a vascular surgeon should be immediately available to consult regarding angiography and possible repair if vascular injury occurs intraoperatively. With regard to epinephrine in arthroscopy fluid, at our institution, its use has been discontinued in knee arthroscopies over the past 4 years since this case, without any difficulty in performing arthroscopic knee procedures.

Acknowledgment: The authors thank Dr. Mark Muller for help with initial preparation of the manuscript.

REFERENCES


