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Authors:

Michael C. Bond, MD, FACEP, FAAEM, Assistant Professor, Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore.

Emilie Cobert, MD, MPH, Resident Physician, Department of Emergency Medicine, University of Maryland, Baltimore.

Peer Reviewer:

Andrew D. Perron, DO, Professor and Residency Program Director, Department of Emergency Medicine, Maine Medical Center, Portland.

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Knee and Patellar Dislocations: Identifying Subtleties for Optimal Recognition and Management

Knee dislocations have the potential to result in significant morbidity and mortality if not correctly diagnosed and optimally managed. Early identification and treatment of neurovascular injury and compartment syndrome may avert disaster for the patient. Recognition of hard signs of vascular injury mandate immediate surgical exploration, whereas soft signs of vascular injury require further diagnostic evaluation. Closed reduction may be performed when emergency surgery is not indicated. Following reduction, hospital admission is recommended to monitor for neurovascular compromise.

Patellar dislocations are common, and the vast majority are managed by the ED physician. Acute surgical management is uncommon and indicated for persistent patellar subluxation and detachment of the vastus medialis obliquus muscle and medical retinaculum from the medial aspect of the patella.

The authors present a comprehensive and detailed review of the diagnosis and management of knee and patellar dislocations. Common pitfalls and diagnostic advances are reviewed and discussed to maximize patient outcome and decrease long-term functional morbidity.

—The Editor

Introduction: Definition and Classification

A knee dislocation is a complete disruption of the articulation between the tibia and femur. It is often the result of high-energy impacts from sports and motor vehicle collisions. Traumatic dislocations of the knee, while uncommon (with an incidence of 0.02%), can result in significant morbidity and mortality.[1] Young adult males are more frequently the victims of these injuries, fitting the epidemiologic profile of trauma patients in general. The incidence of knee dislocations is probably significantly underreported because many dislocations reduce spontaneously before the affected person reaches a medical facility, so the true nature of the injury is never realized. If the dislocation does not reduce spontaneously, the condition is classified as a "locked" dislocation, which carries significant risk of long-term functional, neurologic, and vascular injury or dysfunction. Therefore, prompt evaluation, stabilization, diagnostic assessment, and treatment are imperative to achieve the best functional outcome.

Multiple classification systems for knee dislocations have been devised, based on the force of the mechanism of injury (high, low, ultra-low), its acute versus chronic nature, or the number of ligaments injured. The widely accepted Schenck's classification system of knee dislocation (*see Table 1*), based on the number of ligaments torn, is designed for operative planning and comparison with similar injuries.[2] In Type I dislocations, one cruciate ligament and one collateral ligament are torn; the second cruciate and collateral ligaments remain

Executive Summary

- The most common clinical classification system for knee dislocations is based on the position of the tibia in relation to the femur. Thus, the dislocation can be classified as anterior (most common), posterior, lateral, medial, or rotary (very rare).
- About 40% of people with knee dislocations have an associated popliteal artery injury.
- Damage to the peroneal nerve has been reported in 25% to 40% of patients with knee dislocations and is more common in those with lateral and posterolateral dislocations.
- Two studies have shown that a normal ABI has an NPV of 100%. However, since intimal tears or dissections can present with normal ABIs initially but change as a thrombus forms and occludes the popliteal artery, patients with knee dislocations need to be admitted for serial examinations.

intact. In Type II dislocations, both cruciate ligaments are torn and the collateral ligaments remain intact. Type III dislocations are subdivided into M and L, based on whether the medial or lateral collateral ligament is torn. In Type III dislocations, both cruciate ligaments and one collateral ligament are torn. In Type IV dislocations, all four ligaments (both cruciate and both collateral ligaments) are torn and the posterolateral corner (PLC) is injured. In Type V dislocations, a periarticular fracture is also present.

The most common clinical classification system is based on the position of the tibia in relation to the femur. Thus, the dislocation can be classified as anterior (most common), posterior, lateral, medial, or rotary (very rare).[3] Combinations can also occur, with the most common being posterolateral.[4] This classification system is of limited value for dislocations that reduce spontaneously prior to medical evaluation. In these cases, the Schenck classification system should be used.

Table 1. Schenck Classification System for Knee Dislocations

I	Single cruciate ligament plus a collateral ligament	ACL or PCL plus a collateral
II	ACL/PCL	Collaterals intact
IIIM	ACL/PCL/MCL	LCL intact
IIIL	ACL/PCL/LCL	MCL intact
IV	ACL/PCL/MCL/LCL and PLC	
V	Fracture Dislocation	
ACL = Anterior cruciate ligament, PCL = Posterior cruciate ligament, MCL = medial cruciate ligament, LCL = lateral collateral ligament, PLC = posterolateral corner		

Anatomy and Pathophysiology

The knee joint consists of three articulations between the femur, tibia, and fibula: the tibiofemoral, patellofemoral, and proximal tibiofibular articulations. The patella, the largest sesamoid bone in the body, sits directly anterior to the joint. The patella enhances and aligns the force of the quadriceps muscle during knee extension and keeps the quadriceps force from being pulled laterally. The strong medial patellofemoral ligament (MPFL) stabilizes the patella and offsets the lateralizing force of the quadriceps muscle.

Four ligaments stabilize the knee joint. The anterior cruciate ligament (ACL) prevents forward movement of the tibia in relation to the femur, while the posterior cruciate ligament (PCL) prevents posterior movement of the tibia. The lateral collateral ligament (LCL) stabilizes the knee laterally and is the primary restraint to varus angulation, while resisting internal rotation forces. Finally, the medial collateral ligament (MCL) provides medial stability and is the primary restraint to valgus angulation. The knee also has two menisci (medial and lateral), which are fibrocartilaginous structures that act as shock absorbers between the knee and femur, reducing friction and decreasing the contact area between the bones.

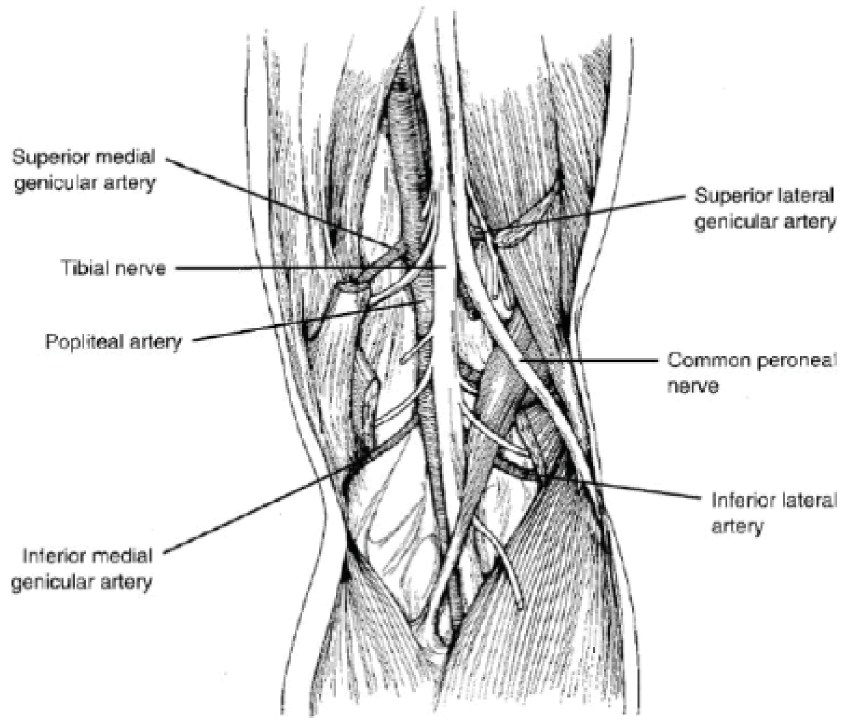
The posterolateral corner of the knee is critical to its stability, particularly in the varus and rotatory planes. The anatomy of this region is formed by the deep, middle, and superficial layers of tissue. The deep layer contributes most of the stability to this region and is composed of the popliteus muscle, the popliteofibular ligament, the LCL, the arcuate ligament, and the fabellofibular ligament.[5] The peroneal nerve travels through the deep layer. The middle layer consists of the patellar retinaculum and the patellofemoral ligament. The superficial layer consists of the lateral fascia, the iliotibial tract, and the tendon of the biceps femoris muscle. Isolated posterolateral corner injuries are rare; this region is commonly injured in conjunction with ACL or PCL tears.[6]

The popliteal artery provides the major arterial supply to the lower leg. It is tethered proximally by the adductor hiatus of the thigh and distally by the proximal soleus fascial arch. (*See Figure 1.*) The popliteal artery is susceptible to injury during dislocation by direct blow, stretch, or shearing forces. A wide range of injuries can occur to the popliteal artery (e.g., intimal flap tear, thrombosis, pseudoaneurysm, and transection). The knee has poor collateral circulation from the genicular arteries, which may be easily avulsed or thrombosed during a dislocation. As a result, damage to the popliteal artery can lead to significant morbidity.

The sciatic nerve divides into the tibial and common peroneal nerves at the distal femur. The common peroneal nerve divides into the sural cutaneous, deep peroneal, and superficial peroneal nerves as it wraps around the head of the fibula. Knee dislocations can injure any of these nerves. The peroneal nerve is at high risk for injury during knee dislocations because it has a superficial course through the popliteal fossa and runs very close to the fibular neck. The common peroneal nerve provides motor innervation to the short head of the biceps femoris. Its branch, the lateral sural cutaneous nerve, provides sensory innervation to the superior lateral side of the leg. The superficial peroneal nerve provides motor innervation to the lateral compartment of the leg and sensory innervation to the lateral aspect of the lower leg and the dorsum of the foot. The deep peroneal nerve provides motor innervation to the anterior compartment of the leg and sensory innervation to the first dorsal web space.

The tibial nerve is also at risk for injury during knee dislocations. It runs medially to the common peroneal nerve in the popliteal fossa and extends straight down distally to innervate the posterior compartment of the lower leg and the plantar aspect of the foot.

Figure 1. Anatomy of the Popliteal Space



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Knee Dislocation

A knee dislocation (*Figure 2*) is defined as a complete disruption of the joint such that the articular surfaces are no longer in contact; dislocations also include knees with multi-ligamentous injury or multi-directional instability.[7] Subluxation is similar to a dislocation in that the joint is disrupted; however, the articular surfaces remain partially intact. Knee dislocations may induce multiple ligamentous injuries, but it is possible to have a dislocation without bicruciate ligament tears. Knee dislocations are classified as anterior, posterior, medial, lateral, rotary, or a combination of these directions, which are based on the tibia's position in relation to the femur. Anterior dislocations are the most common classification, with an incidence of 31%.[8] Posterior dislocations often require greater force in the mechanism of injury.

The incidence of fracture among patients with knee dislocation is as high as 40%.[8] Ligamentous injury is variable, depending on the force and direction of the dislocation. Several studies recommend early operative repair of injured ligaments to achieve good functional outcome.[3,9]

About 40% of people with knee dislocations have an associated popliteal artery injury.[8,10] If the pulse is absent, reperfusion must be restored within 6 to 8 hours after injury to minimize the risk of limb necrosis and amputation.[3,9,10] Popliteal artery intimal tears are more common in anterior dislocations; full transection of the artery is more common in posterior dislocations. Because of the poor collateral circulation around the knee, damage to the popliteal artery can result in significant morbidity. In cases of complete ligation of the popliteal artery, necrosis has been reported in as many as 72.5% of patients.[9]

The reported incidence of neurologic injuries associated with knee dislocations ranges from 16% to 40%.[7] During a dislocation, particularly with forced varus or hyperextension stress, the fibular neck may pull against the peroneal nerve. Damage to the peroneal nerve has been reported in 25% to 40% of patients with knee dislocations and is more common in those with lateral and posterolateral dislocations.[8] The highest risk of injury to the common peroneal nerve (including complete transection) is associated with dislocations combined with disruption of the ACL, PCL, and posterolateral corner.[11] It is not surprising that dislocations resulting from forces high enough to cause multiple ligamentous and bony injuries also result in more severe nerve injuries, including complete transection.

Figure 2. Anterior Knee Dislocation

Images courtesy of Michael C. Bond, MD

Physical Examination

A careful and focused physical examination of the injured extremity must be performed. Gross inspection includes assessment for open injuries and deformity and a skin inspection for mottling and cyanosis. The examiner should compare the injured joint and limb with the uninjured leg. In all three positions, the knee and patella should be inspected and palpated for alignment, deformity, swelling, hemarthrosis, effusion, and open wounds.

Passive and active range of motion should be evaluated. The patient should actively extend the knee from a flexed position. During range of motion, the examiner should feel for crepitus, which may be a sign of cartilage injury. The examiner should also evaluate the patella during active range of motion to look for any abrupt lateral shifting.

The knee must be checked for ligamentous stability. If the history suggests the patient may have had a knee dislocation that reduced spontaneously, the only objective evidence may be the observation of multidirectional knee instability. The patient should be evaluated for anterior or posterior displacement of the tibia when stressed (anterior or posterior drawer signs) and for valgus or varus laxity when the MCL and LCL ligaments are stressed, respectively. In patients who present with a locked dislocation, expedient reduction is important to minimize vessel and nerve strain.

Extremity perfusion and viability are high priorities. The first step in the evaluation for a potential vascular injury includes observation of both hard and soft signs. Hard signs include an expanding hematoma, active hemorrhage or pulsatile bleeding, absent distal pulses, a cold or pale limb, and a palpable thrill or audible bruit.[1,12] The presence of a hard sign is an indication for immediate surgical exploration with vascular reparation. Soft signs of vascular injury include history of hemorrhage, small or non-expanding hematoma, diminished pulses, wound proximity to major vessels, poor skin color, and capillary refill time longer than 2 seconds.[1,12] The presence of a soft sign mandates further diagnostic evaluation aside from the physical examination. Vascular perfusion should be assessed manually by palpation of the dorsalis pedis (DP) and posterior tibial (PT) pulses. A retrospective review by Peck et al found that 7% of patients with blunt lower-extremity trauma had palpable pedal pulses despite significant vascular injury.[13] This observation highlights the limitations of the physical examination to detect vascular injuries. However, in that review, there was no quantification of "palpable pedal pulses." The pulses may have, in fact, been diminished or unequal. Interestingly, a retrospective cohort study showed that no patients with a normal neurovascular examination and no signs of arterial insufficiency or compartment syndrome, in whom angiography was deferred, sustained limb loss or required vascular intervention.[14] Treiman et al found that 4% of patients with a normal pulse examination after a knee dislocation had a popliteal artery injury.[15] None of them required arterial repair. The sensitivity, specificity, and negative predictive value (NPV) of the pedal pulse examination in predicting arterial injury were 85.2%, 93.2%, and 95.3%, respectively. The sensitivity, specificity, and NPV of the pedal pulse examination in detecting a popliteal artery injury requiring operative repair were 100%, 92.4%, and 100%, respectively.[15] The utility of capillary refill time (CRT) to indicate perfusion has also been scrutinized. CRT has been shown to be an inadequate clinical indicator of vascular perfusion.[16] Skin and subcutaneous tissue can remain viable with much less blood flow than is required by muscle.[10]

An ankle brachial index (ABI) is an inexpensive, noninvasive, quick test that can be done at the bedside to evaluate arterial competency in an injured extremity. Liberal use of arteriography can result in false-positive results, placing the patient at risk for contrast angiography-related complications (e.g., allergic reaction, contrast nephropathy, vessel injury, cost, and delay to diagnosis). During the past 30 years, there has been a significant shift to evaluate potential vascular injuries with soft signs of arterial injury with noninvasive testing first.

Calculation of the ABI begins with the use of Doppler ultrasound to measure the systolic blood pressure (SBP) at the ankle (the DP or PT artery). The SBP of the ankle is divided by the SBP of the ipsilateral (if uninjured) arm.[17] A value greater than 0.9 is normal, and a value less than 0.9 suggests vascular injury. Lynch and Johansen demonstrated that an ABI less than 0.9 had a sensitivity, specificity, and NPV of 87%, 97%, and 99%, respectively, for arterial disruption.[18] Investigators at a trauma center in Turkey found higher sensitivity (95%) and specificity (97%) in patients who had only soft signs of arterial injury.[12] An ABI less than 0.9 with or without soft signs warrants further investigation with arteriography.[19,20] Two studies have shown that a normal ABI has an NPV of 100%.[21,22] In these studies, the authors concluded that emergent angiography is not necessary for patients with a normal ABI. However, since intimal tears or dissections can present with normal ABIs initially but change as a thrombus forms and occludes the popliteal artery, these patients need to be admitted for serial examinations. Preexisting peripheral vascular disease can be a limitation to ABI testing and can result in low ABI values independent of trauma.

Arterial pressure indices (APIs) are an alternative noninvasive measurement. APIs are similar to ABIs except that they are calculated by dividing the distal SBP of the injured extremity by the SBP in the uninjured paired extremity, as measured by Doppler (in other words, comparing one leg with the other).[23] APIs less than 0.9 are considered abnormal and have a diagnostic accuracy up to 95%.[18] Nassoura and colleagues showed that APIs have a sensitivity, specificity, positive predictive value, and negative predictive value of 72.5%, 100%, 100%, and 96%, respectively, for clinically significant injury.[24] Johansen and associates demonstrated that a cutoff ratio of 1.0 for the API increased the negative predictive value to 99%. However, just like ABIs, this measurement can miss intimal flaps, dissections, and false aneurysms, as these injuries do not affect the flow of arterial blood.[20]

In conclusion, the physical examination of patients with knee dislocations has significant limitations in detecting arterial injury, but not in determining the need for operative repair and the risk of limb loss. Patients without indications for emergent surgery who present with abnormal pulses or neurologic deficits should undergo further diagnostic tests (i.e., Doppler ultrasound, angiography, or arteriography).[19] The decision to perform an invasive arterial diagnostic evaluation should be made in consultation with the radiologist, orthopedist, and vascular surgeon.

In a 10-year review by Branco and colleagues, patients with knee dislocations were found to be at high risk for the development of compartment syndrome[25] (more than 40% in patients with combined arterial and venous injuries required fasciotomy). Signs and symptoms of compartment syndrome include painful passive muscle stretch, swelling, pallor, diminished pulses (late sign), and hyperesthesia.[26] Clinical assessment of compartment syndrome may not always be easy because of the high association of neurologic injuries and the patient's resulting altered pain perception. Therefore, it may be necessary to measure intracompartmental pressures directly. Compartment syndrome may not be present initially, so clinical signs and symptoms must be monitored closely during a patient's emergency department (ED) and hospital stay. If markedly increased swelling, increased compartment pressures, or clinical symptoms of compartment syndrome are noted, a four-compartment fasciotomy is needed emergently. It is important for the clinician to remember that compartment syndrome can develop as a result of soft-tissue edema and does not have to be associated with a vascular injury. In the obtunded patient, consider the use of continuous or intermittent invasive compartment pressure measuring devices.

The neurologic status of the limb should be assessed, with close attention to the tibial and common peroneal nerves. Peroneal nerve injury is far more common than other nerve injuries. Findings of peroneal nerve palsy include the inability to dorsiflex the foot ("drop foot") and the presence of sensory deficits on the anterolateral leg and dorsum of the foot. Findings of tibial nerve palsy include the inability to plantar flex the foot and the presence of sensory deficits on the plantar aspect of the foot.

It is imperative that the clinician responsible for the initial neurologic examination document the findings thoroughly. This documentation is especially important for patients who require immediate operative repair and to note the presence of any neural injury that occurred prior to the patient's arrival. A lack of documentation of preoperative neurologic status can have significant medicolegal ramifications if postoperative neurologic deficits are discovered.

Niall et al showed that when the deep peroneal nerve, which supplies the anterior muscle compartment, is injured in association with traumatic knee dislocation, the prognosis for recovery is worse than in patients in whom the superficial peroneal nerve is injured.[11] In this series, only 21% of patients with injury to the common peroneal nerve achieved complete recovery. Overall, the prognosis for complete neurologic recovery ranges from 14% to 75%, which reflects an uncertain neurologic prognosis.[11]

The ligaments and osteochondral structures should be examined next. The Lachman test is the most sensitive for acute ACL rupture, and the posterior drawer test is most sensitive for acute PCL rupture.[27] MCL and LCL competency is tested with varus and valgus stress, respectively.

Imaging

Radiographic evaluation is an important step in the diagnostic evaluation. Plain radiographs can identify subluxations, dislocations, associated fractures, and effusions. As in most orthopedic evaluations, a joint above and one below the injury should also be imaged to avoid missing associated injuries. Computed tomography (CT) is playing a larger role in the evaluation of knee dislocations because of its higher sensitivity for fractures and associated proximal tibiofibular dislocations.[28] Furthermore, CT angiography, which requires venous cannulation, is safer and has fewer complications than traditional arteriography, which requires arterial cannulation.[29,30] Sufficient evidence exists to suggest that CT angiography may soon surpass arteriography as the gold standard in evaluating arterial injury. Inaba et al report that multi-slice helical CT angiography achieved 100% sensitivity and 100% specificity in diagnosing clinically significant arterial injury.[31] Another study by Soto et al found CT arteriography to have a sensitivity of 95.1% and a specificity of 98.7%.[32] Finally, magnetic resonance imaging (MRI) has higher accuracy than the clinical examination to detect the extent or site of soft-tissue injury (85% to 100% vs. 53% to 82%, respectively).[30] MRI is particularly useful to prepare the patient for surgical repair by an orthopedist, as it can delineate ligamentous and cartilaginous injuries.

Figure 3. Anterior Prosthetic Knee Dislocation

A. Pre-reduction; B. Post-reduction
Images courtesy of Michael C. Bond, MD

Treatment

Initial management of an acute knee dislocation includes recognizing and treating limb-threatening injuries and maximizing long-term joint use. Indications for emergent surgical management include an open wound, compartment syndrome, an irreducible dislocation or failed closed reduction, and a grossly unstable dislocation. Emergent operative vascular repair should be performed when any hard signs of vascular injury are noted. Consider operative repair in patients with evolving soft signs of vascular injury or diminishing ABIs or APIs.

When emergency surgery is not indicated, proceed with closed reduction. Induce procedural sedation in conscious patients, because it allows the reduction to be done with minimal pain and anxiety and it provides a good opportunity to do a thorough ligamentous examination without patient discomfort and voluntary guarding.

Reduction of a knee dislocation requires the aid of an assistant. The assistant should exert firm and constant longitudinal traction on the proximal tibia to ensure that the femur and tibia are distracted. While traction is being held, exert a force on the proximal tibia that is counter to the direction of the dislocation to cause the reduction. For a posterior dislocation, lift the proximal tibia anteriorly to reduce the joint. For an anterior dislocation, push the proximal tibia posteriorly. Avoid the application of pressure on the popliteal space during the reduction attempt, so as to prevent additional stress and traction on the popliteal artery. Many posterolateral dislocations are irreducible with closed reduction attempts because the medial condyle of the femur traps the medial capsule of the joint.[4]

After a closed reduction, recheck the ABIs or APIs. Stabilize the knee with a non-constrictive knee immobilizer. Ideally, the patient should be placed in a hinged knee brace with the knee at 15 degrees of flexion to avoid strain on the popliteal vessels; however, if a hinged brace is not available, a straight-leg knee brace is acceptable. Hospital admission is strongly recommended in light of the high rate of associated neurovascular injuries and the potential for late manifestations of neurovascular compromise. Serial ABIs and neurologic examinations should be performed hourly for up to 48 hours.[33] Any clinical deterioration of the neurovascular status, as indicated by examination or ABIs, warrants an immediate surgical evaluation or diagnostic study (e.g., Doppler ultrasound or CT angiography) because this change may be secondary to an expanding popliteal thrombosis or compartment syndrome.

Anticoagulation

The use of anticoagulation and thrombolytics improves limb salvage and is recommended in patients with traumatic popliteal thrombosis.[34,35] It is contraindicated in patients with pelvic, intra-abdominal, or head trauma. The decision to begin systemic anticoagulation should be made in conjunction with orthopedic and vascular surgery consultants.

Prosthetic Knee Dislocations

Unlike prosthetic hip dislocations, prosthetic knee dislocations are very rare. Prosthetic knee dislocations can be posterior or anterior. (*See Figure 3.*)

Posterior dislocations typically occur in the post-operative period and are usually the result of trauma that disrupts the PCL ligament. Factors that predispose a person to posterior dislocations are valgus deformity of the knee, malposition or improper selection of prosthetic components, patellar instability, and extensor mechanism dysfunction. The mechanism for this dislocation is typically flexion and external rotation of the knee when the lateral side of the knee is too loose.[36]

Anterior dislocations more commonly occur months to years after surgery and usually are not associated with trauma.[36] Many of these dislocations result from loss of integrity of the posterior cruciate ligament, which provides anteroposterior stability of the knee and assists in femoral rollback. This motion is essential for the extensor mechanism of the knee to function. Stiehl et al postulated that abnormal kinetics of the prosthetic knee allow the PCL ligament to pull the femur posteriorly in the extended knee, resulting in anterior translation of the tibia and posterior translation of the femorotibial contact point. This abnormal motion causes gradual lengthening of the PCL and the posterior capsule, allowing the tibia to sublux and eventually dislocate anteriorly.[37]

Prosthetic knee dislocations can be reduced in a similar manner to that used for native knee dislocations. These deformities should be evaluated in a similar manner to assess the patient for neurovascular injury. Some patients will require a revision of the knee prosthesis.

Figure 4. Patellar Dislocation

A. Anteroposterior view; B. Sunrise view
Reproduced from: Minkowits R, Inzerillo C, Sherman O. Patella instability. *Bull NYU Hosp Jt Dis* 2007;65(4):280-293, with permission.

Patellar Dislocation

The patellofemoral joint consists of the articulation of the patella bone with the trochlear groove of the femur. The incidence of acute patellar dislocation in adults is estimated to be 20:100,000.[38] Females are at highest risk in the second decade of life, whereas males are at highest risk in the third decade of life.[17,38]

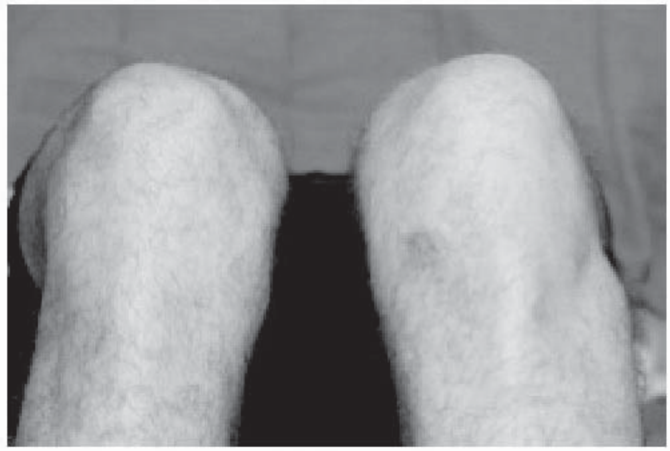
There are two types of patellar dislocation: lateral and medial. Lateral dislocations are more common. Dislocations can be caused by a direct blow or an indirect injury, with the latter being more common. Patellar dislocation from indirect injury can occur when the patient is standing with the knee flexed in a valgus position while the femur rotates internally on an externally rotated tibia, as can occur with the strong quadriceps muscle contraction of a baseball player's hind leg at the end of a swing.[38] This typically results in a lateral dislocation. A direct blow to the medial side of the patella can also force it laterally, causing a dislocation.

The medial patellofemoral ligament (MPFL) provides stability to the patellofemoral joint when the knee is between full extension (0 degrees) and 30 degrees of flexion.[17] Because it is the primary ligamentous structure preventing patella dislocation, it is also the ligamentous structure most commonly injured in acute patellar dislocations.[39] When the knee is flexed more than 30 degrees, the patella is protected from dislocation by the lateral femoral condyle and the trochlear groove.

Most acute patellar dislocations reduce spontaneously, which results in a diagnostic challenge for the clinician. The physical examination of the patella should begin with inspection. When the patient is sitting, the patella should be symmetric. Its normal position is centered within the trochlear groove and facing straight ahead. If the patella is in a high and lateral position, it is described as a "grasshopper eye," as it seems to look over the examiner's shoulder. (*See Figure 4.*) The patella is then palpated to localize areas of maximal tenderness, which suggests soft-tissue injury. Despite the diagnostic challenges with patellar dislocations, several physical examination findings are consistent with a patellofemoral dislocation: hemarthrosis (especially medially, owing to disruption of the MPFL and other medial restraints), tenderness over the medial facet of the patellar and adductor tubercle, medial ecchymosis, and a positive apprehension sign.

When acute patellar dislocation is suspected, a radiographic evaluation should be done to rule out osteochondral fragments within the joint. Anteroposterior (AP), lateral, and Merchant views should be obtained. The Merchant view is obtained by having the patient lie supine with the knee flexed to 45 degrees. The radiograph is taken with the X-ray beam aimed 30 degrees to the floor. A similar view is the sunrise view, which is obtained by having the patient lie prone with the knee acutely flexed.

Figure 5. Proximal Left Tibiofibular Joint Dislocation



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An MRI study can also be considered, as it is more sensitive for bony and cartilaginous pathology. MRI findings that are consistent with an acute patellofemoral dislocation are focal impaction injuries of the lateral femoral condyle, osteochondral injuries to the medial facet of the patella, and medial retinacular ligament injuries.[40] Associated osteochondral injuries are an indication for surgical repair; if this type of injury is missed, the patient's outcome is likely to be poor.

When the patella is in a "locked dislocation" (that is, it is still dislocated at this point in the evaluation), the clinician should attempt a closed reduction. A lateral patella dislocation can be reduced by extending the flexed knee while applying medially directed pressure to the lateral aspect of the patella. In many cases, the patella will be repositioned when the knee is fully extended or slightly hyperextended. The same technique can be used for medial dislocation, except the laterally directed pressure would be applied to the medial edge of the patella.

Indications for acute surgical management include persistent patellar subluxation and detachment of the vastus medialis obliquus muscle and medial retinaculum from the medial aspect of the patella. The ideal management (operative vs. non-operative) of primary, first-time patellar dislocations is a topic of debate. Nikku et al randomized 127 first-time lateral patellar dislocations without tibiofemoral ligamentous injuries or osteochondral fractures into two groups: closed treatment with immobilization and rehabilitation; and early operative treatment.[41] Outcome was assessed based on recurrent instability in the form of redislocation or recurrent subluxation, the need for additional operations, activity scores, and patients' opinions. Seven-year follow-up demonstrated no benefit of surgical intervention over immobilization and rehabilitation. However, surgically managed patients report significantly more sustained pain after operative repair.

Acute patellar dislocations can be managed conservatively with ice and elevation for the first several days and immobilization with a knee brace for 2 to 3 weeks. If a large hemarthrosis has formed, sterile aspiration is recommended prior to immobilization. Patients can bear weight as tolerated while the knee is in extension. Once the patient is comfortable, 6 weeks of rehabilitation is started.

Figure 6. Anteroposterior Radiograph of Left Knee Showing Lateral Displacement of Fibular Head

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Patients who experience recurrent subluxations or dislocations should be referred to an orthopedic surgeon for follow-up and consideration for surgical correction.

Proximal Tibiofibular Dislocation

The tibiofibular articulation is an inherently stable joint because it has tremendous ligamentous support, especially when the knee is in extension. Dislocation usually occurs when the knee is flexed while the foot is rotated and plantar-flexed. Isolated dislocations occur in sports that involve twisting of the knee.

Types of proximal tibiofibular dislocations include subluxation and anterolateral, posteromedial, and superior dislocations.[42] Subluxations tend to occur in pre-adolescent girls and are usually atraumatic. Anterolateral dislocations (*see Figures 5 and 6*)[43] are the most common type of this injury (85%) and usually result from a fall when the knee is flexed, the ankle is inverted, and the foot is plantar-flexed.[33] Posteromedial dislocations (10%) are usually caused by direct trauma and are associated with common peroneal nerve injury.[33] Superior dislocations (2%) are uncommon and occur with high-energy ankle injuries. Superior dislocations are always associated with superior displacement of the lateral malleolus and are often associated with a common peroneal nerve injury.

Physical examination findings may be subtle. They include prominence of the fibular head, lateral knee pain, limited knee extension, and crepitus, popping, or locking. Normal radiographic characteristics of this joint include overlap of the fibular head with the lateral margin of the lateral tibial condyle on the AP view.[33] On the lateral view, most of the fibular head should lie posterior to the posterior margin of the tibia. CT is recommended as the study of choice for suspected proximal tibiofibular dislocations because it has higher sensitivities and specificities than plain radiographs.[42] Since findings on plain radiographs are subtle, a comparison radiograph of the contralateral limb may be indicated if the diagnosis is suspected and CT is unavailable.

Posteromedial dislocations are frequently associated with peroneal nerve injuries and are often irreducible because of interposed soft tissue. Anterolateral dislocations can be treated with closed reduction. This can be done by flexing the knee, dorsiflexing and externally rotating the foot, while placing pressure over the fibular head until a "pop" is heard or felt.[44] The knee should be immobilized initially, but the patient should be encouraged to begin early mobilization. Failed reduction of an anterolateral dislocation and of most posteromedial and superior dislocations requires open reduction by an orthopedist.

Conclusion

Knee and patellar dislocations have the potential to be devastating injuries with poor neurovascular outcomes. It is the emergency clinician's goal to identify patients who require emergent surgical intervention and to cautiously observe those in whom neurovascular injury might develop. With attention to detail and an understanding of the diagnostic and therapeutic options in the emergency setting, the emergency clinician can maximize the functional outcome of patients with these injuries.

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CME/CNE Questions

1. The most common type of knee dislocation is:

   A. anterior
   B. lateral
   C. medial
   D. posterior
   E. rotary

2. The risk of a popliteal artery injury in association with a knee dislocation is as high as:

   A. 10%
   B. 25%
   C. 40%
   D. 60%
   E. 80%

3. Popliteal artery injuries that are not treated within how many hours result in amputation in as many as 86% of patients, owing to ischemic changes?

   A. 12
   B. 14
   C. 16
   D. 8
   E. 10

4. What ankle brachial index is considered normal?

   A. greater than 0.0
   B. less than 0.5
   C. greater than 0.5
   D. less than 0.9
   E. greater than 0.9

5. After a knee dislocation has been reduced, it should be immobilized with the knee at what degree of flexion?

   A. 5 degrees
   B. 10 degrees
   C. 15 degrees
   D. 25 degrees
   E. 45 degrees

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

6. The most common mechanism that causes a patellar dislocation is:
   A. a direct blow
   B. an indirect injury
7. The most common proximal tibiofibular dislocation is:
   A. an anterolateral dislocation
   B. a posteromedial dislocation
   C. a superior dislocation
8. How can lateral patella dislocations be reduced?
   A. By flexing the knee and applying lateral pressure to the medial border of the patella
   B. By flexing the knee and applying medial, pressure to the lateral border of the patella
   C. By hyperextending the knee and applying lateral to the medial border of the patella
   D. By hyperextending the knee and applying medial pressure to the lateral border of the patella
9. Which of the following is a hard sign of a vascular injury?
   A. capillary refill greater than 2 seconds
   B. diminished pulse
   C. history of hemorrhage
   D. pulsatile bleeding
   E. small or non-expanding hematoma
10. A Schenck Type IIIM knee dislocation is associated with a tear of which of the following ligaments?
   A. both cruciate ligaments and the lateral collateral ligament
   B. both cruciate ligaments and the medial collateral ligament
   C. both cruciate ligaments only
   D. one cruciate ligament and the lateral collateral ligament
   E. one cruciate ligament and the medial collateral ligament

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