Review Article

Acute Management of Traumatic Knee Dislocations for the Generalist

Robert H. Boyce, MD
Keerat Singh, MD
William T. Obremskey, MD, MPH

Abstract

Acute knee dislocations are an uncommon injury that can result in profound consequences if not recognized and managed appropriately on presentation. Patients presenting with knee pain in the setting of high- or low-energy trauma may have sustained a knee dislocation that spontaneously reduced. Prompt reduction of the dislocated knee and serial neurovascular examinations are paramount. Damage to the popliteal artery is a common associated injury that can be diagnosed on physical examination using ankle brachial indices (ABIs), CT angiography, or standard angiography. After reduction, patients with a normal pulse examination and an ABI ≥0.9 may be observed, with serial examination performed to document vascular status and monitor for compartment syndrome. Patients with asymmetric pulses or an ABI <0.9 in the presence of pulses may be treated urgently depending on the results of additional vascular imaging, and patients with absent pulses and clear signs of vascular compromise should be treated emergently. Some knee dislocations are not reducible and should be taken emergently to the operating room for an open reduction. Persistent joint subluxation or severe soft-tissue injuries after reduction require temporary external fixation before definitive repair or reconstruction of ligaments is performed.

From the Department of Orthopaedic Surgery Vanderbilt University, Nashville, TN.

Dr. Obremskey or an immediate family member serves as a board member, owner, officer, or committee member of the Orthopaedic Trauma Association and the Southeastern Fracture Consortium. Neither of the following authors nor any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Boyce and Dr. Singh.

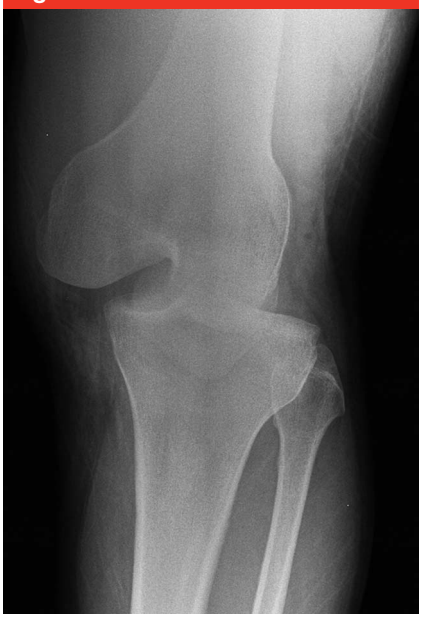
J Am Acad Orthop Surg 2015;23: 761-768

http://dx.doi.org/10.5435/JAAOS-D-14-00349

Acute knee dislocation is a rare injury that has an estimated incidence of <0.02% of orthopaedic injuries[1] or 2 to 29 injuries per million annually.[2,3] In comparison, hip fractures in young males occur at a rate of ≥100 per million annually.[4] The incidence of acute knee dislocations likely has been underreported in the literature given their propensity to self-reduce and the potential for misdiagnosis. Approximately 50% of knee dislocations spontaneously reduce before a formal evaluation.[5] Whether the patient presents with an acutely dislocated knee or an occult dislocation (ie, one that has been reduced in the field or at another hospital), a prompt and thorough evaluation is crucial.

Knee dislocation predominantly occurs in a younger population, with a male-to-female ratio of 4:1.[4] Dislocation can result from both high-energy mechanisms (eg, fall from a height, crush injury, motor vehicle collision [MVC], pedestrian versus motor vehicle) or low-energy mechanisms (eg, misstep during routine walking, martial arts kicks, trampoline fall).[6] Although it is true that half of these injuries are the result of a MVC or high-energy mechanism, approximately one third of the cases result from a lower energy mechanism, which often occurs in sports

Figure 1



AP radiograph of the knee demonstrating an anterolateral dislocation.

injuries. A population-based study of the rate of knee dislocation in Finland over an 11-year period demonstrated that 46% of dislocations were caused by a low-energy fall.[3]

There is increasing evidence that morbid obesity is a risk factor for knee dislocation.[7] Azar et al[8] reported on 17 cases of knee dislocation in patients with an average body mass index of 48 following "ultra-low-velocity" activities of daily living. Dislocations in patients who are obese have a similar and potentially higher incidence of neurovascular injury relative to high-energy dislocations.[8] Open knee dislocations represent 5% to 17% of all knee dislocations.[9] Bilateral dislocation occurs in approximately 5% of cases.[10,11] Knee dislocations associated with sports injuries have a lower incidence of neurovascular injury compared with those resulting from a MVC.[12]

The potential for limb loss or the irreversible consequences of limb ischemia is present with any knee dislocation regardless of the velocity of the mechanism of injury. According to the recent literature, injury to the popliteal artery occurs at rates ranging from 1.6%[3] to 40%.[8] It is likely that the average rate is approximately 16%.[13] A delay of >8 hours in reestablishing arterial blood flow resulted in an amputation rate of 85% in a study of 18 patients with knee dislocation and an associated injury to the popliteal artery.[14] Prolonged warm ischemia time was the primary factor in associated vascular injury and knee dislocation in the Lower Extremity Assessment Project study, which reported an amputation rate of 20%.[14] Treating orthopaedists must understand the pathophysiology, anatomy, and management options for knee dislocation to avoid suboptimal outcomes.

Pathophysiology and Classification

A knee dislocation is a disruption of the tibiofemoral articulation, which can occur in any direction and typically involves at least two ligaments (Figure 1). Knee dislocations can be described based on the direction of dislocation or the severity of ligamentous damage. Kennedy[15] categorized the dislocation based on the direction of displacement of the tibia. Five types of knee dislocations were described: anterior, posterior, medial, lateral, and rotatory. Cadaver studies have shown that an anterior dislocation is caused by a hyperextension mechanism that causes a sequential failure of the posterior capsule, posterior cruciate ligament (PCL), and sometimes the anterior cruciate ligament (ACL).[15] Both the ACL and PCL are often disrupted.[11] Posterior dislocations, the second most common type, are caused by a significant posteriorly directed force across the proximal tibia as is seen in the "dashboard" injury.[15] Lateral and medial dislocations comprise 18% and 4% of knee dislocations, respectively.[16] In rotatory dislocations, the PCL remains intact as the tibia rotates about the femur.

Some knee dislocations are reduced before evaluation, making a classification based on the direction of displacement somewhat challenging; therefore, a classification system based on the severity of ligamentous damage was developed by Schenk et al[17] and modified by others[18,19] (Table 1). KD (knee dislocation) I is a single cruciate ligament injury. KD II is bicruciate disruption only, and KD III refers to a bicruciate injury with posteromedial or posterolateral disruption. KD IV is a bicruciate injury with posterolateral and posteromedial disruption. KD V is a fracture-dislocation. Subdesignations of C and N indicate vascular and neural injuries, respectively. The direction of knee dislocation does not necessarily predict which ligaments are ruptured. Both classifications schemes are simple and can be used to guide further evaluation and management, but these schemes alone must not be relied on for clinical decision making.

Vascular Anatomy

Given the vulnerability of vessels around the knee, particular attention should be paid to the vascular anatomy. The superficial femoral artery enters the adductor hiatus between the adductor and hamstring portion of the adductor magnus muscle, where the artery is relatively constrained and continues in a posterior-inferior direction into the popliteal fossa. Here, it becomes particularly vulnerable because only the fat of the popliteal fossa cushions it from the posterior body border. It provides five geniculate branches and travels inferiorly in the tendinous arch of the

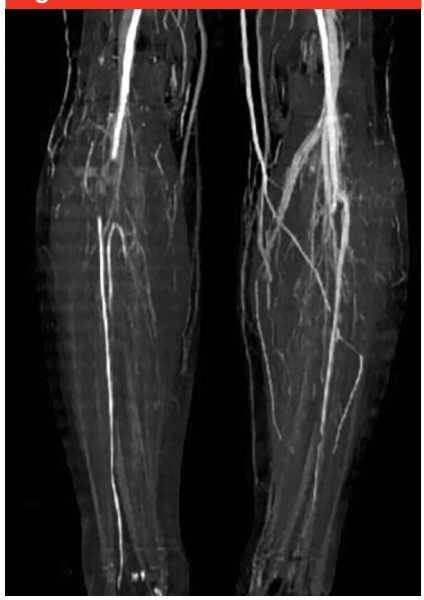
Table 1

Anatomic Knee Dislocation Classification System

Classification	Description
KD I	KD with PCL intact. Collateral ligaments may be injured.
KD II	KD with both PCL and ACL injured and collateral ligaments intact.
KD III[a]	KD with both PCL and ACL injured and one collateral ligament injured, either medial or lateral.
KD IV	KD with ACL, PCL, and both collateral ligament injury.
KD V	KD with a periarticular fracture

ACL = anterior cruciate ligament, KD = knee dislocation, PCL = posterior cruciate ligament
[a] For KD III injury, the injury is written as KD IIIM or KD IIIL, depending on whether the medial or lateral collateral ligament is damaged. The subdesignations C and N indicate the presence of vascular and neural injury, respectively (eg, KD IVCN is a knee dislocation with neurovascular compromise).
Adapted with permission from Wascher DC: High-velocity knee dislocation with vascular injury: Treatment principles. *Clin Sports Med* 2000;19(3):457-477.

Figure 2



CT angiogram of the lower extremities demonstrating the normal arterial flow around the left knee and a disruption of the flow caused by a dislocation of the right knee, with an associated popliteal artery injury.

soleus muscle, where it is relatively constrained.[20] Figure 2 shows normal arterial flow around the knee and the way that flow is disrupted with knee dislocation and injury to the popliteal artery. Although anastomoses do exist between the geniculate arteries and the anterior tibial recurrent artery, these are insufficient to maintain adequate perfusion to the leg. Rupture of the popliteal artery is known to occur at 50° of hyperextension in cadaver models.[15] In anterior dislocations, the popliteal artery suffers a traction injury that leads to intimal damage, whereas a posterior dislocation results in transection of the popliteal artery from the tibia. Forty-four percent of posterior dislocations and 39% of anterior dislocations are associated with vascular compromise.[16]

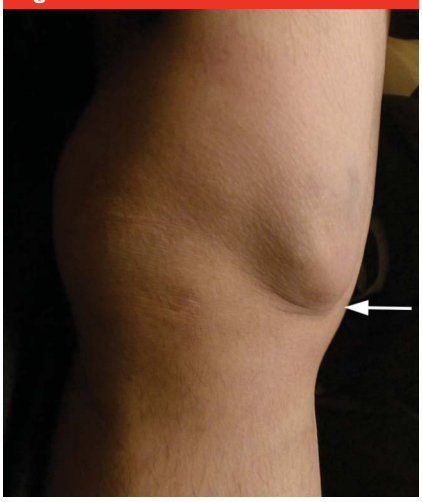
Because the potential for nerve palsy exists in the setting of an acute knee dislocation, an understanding of the perigeniculate neural anatomy is helpful. The sciatic nerve divides into the tibial nerve and the common peroneal nerve in the distal posterior thigh. The common peroneal nerve travels posteroinferior along the biceps femoris muscle and courses anteriorly at the level of the fibular neck. The tibial nerve, being lateral to the popliteal artery in the popliteal fossa, courses inferiorly posterior to the popliteus muscle and enters the deep compartment of the leg through the tendinous arch of the soleus. Peroneal nerve palsies occur in approximately 25% of knee dislocations.[21] The prognosis is generally poor, and the return of nerve function is equivocal at best, with rates averaging 50%.[22]

History and Clinical Presentation

A thorough evaluation includes a history of the time and details of the mechanism of dislocation, prior attempts at reduction, and a full medical history, with particular attention paid to anticoagulant use and any history of bleeding or clotting disorders. Any symptoms of paresthesias or sensory deficits in the leg and foot secondary to potential neurapraxia to the common peroneal and/or tibial nerve should be noted because these raise the risk of a vascular injury.

Initial evaluation may reveal gross deformity about the injured extremity. In cases of a lateral dislocation, a skin dimple is seen along the medial side of the knee joint (Figure 3). Knee swelling is not universal because the capsule is torn in knee dislocations.[23] In addition to noting hard signs of vascular injury (eg, pallor, coolness to touch, delayed capillary refill, evidence of pulsatile hematoma), a pulse examination of the foot (specifically, the dorsalis pedis and posterior tibialis vessel) is mandatory and should be documented both before and after reduction and compared with the contralateral extremity. The presence of normal pulses postreduction has historically presented a dilemma. In young patients with a popliteal artery injury, the collateral flow about the knee is robust enough to possibly sustain a normal pulse in the foot for a short time.[24] Therefore, an ankle brachial index (ABI) performed with the use of a manual blood pressure cuff, and a Doppler probe, should be obtained postreduction regardless whether the pulse examination is normal and symmetric.

Figure 3



Clinical photograph of the knee demonstrating a skin dimple (arrow) seen in the setting of an anterolateral knee dislocation.

The ABI is the ratio of the systolic blood pressure measured at the ankle to that measured at the brachial artery and is easily calculated. With the patient in the supine position, a blood pressure cuff is placed on the affected ankle above the malleoli. The ultrasound transducer is used to locate the dorsalis pedis or posterior tibial artery signal. The blood pressure cuff is inflated while listening to the Doppler signal. Once the signal has disappeared, the pressure in the cuff is slowly released until the Doppler signal can be heard again. The pressure at which the Doppler signal in the dorsalis pedis or posterior tibial artery returns is the systolic blood pressure value for the ankle. For the brachium, the blood pressure cuff is placed on the arm, and the brachial pulse is located in the antecubital fossa using the ultrasound transducer. The cuff is inflated until the Doppler signal from the brachial artery disappears. The cuff is then gently relieved of pressure until the signal in the brachial artery returns. The pressure at which the Doppler signal returns is the systolic blood pressure in the brachium. To calculate the ABI, the systolic blood pressure measured at the ankle is divided by that measured at the brachial artery. A ratio of <0.9 is considered abnormal and necessitates further investigation.[25]

After the knee is reduced, a thorough examination should be performed while the patient is still under conscious sedation because pain and guarding may limit this critical aspect of the evaluation. The injured knee should then be splinted to help maintain reduction. For subacute presentations where the knee may have spontaneously reduced or was reduced previously at an outside hospital or urgent care facility, the extremity should be fully evaluated in the same manner as an acute presentation because of the high risk of associated neurologic and vascular injury. The extremity should then be splinted for stability, and ABIs should be obtained.

Imaging

Prereduction AP and lateral radiographs of the affected knee help the orthopaedist evaluate for concomitant fracture and the direction of displacement. This assessment is sometimes difficult in patients who are morbidly obese. Once the knee is reduced, a postmanipulation AP and lateral knee radiograph should be obtained to verify alignment. If plain radiography reveals the presence of a periarticular or intra-articular fracture, a postreduction CT is recommended to fully evaluate the fracture and for preoperative planning.

MRI aids the diagnosis of ligamentous, meniscal, and articular damage and should be obtained after a successful knee reduction.[26] Such imaging will help with surgical planning and will be of higher quality than an MRI obtained postoperatively when hardware was used for fracture fixation because of the scatter from metal artifact. At times, MRI may not be feasible despite the use of MRI-compatible external fixation systems. Patients may still experience discomfort or the heating of pins, particularly if they are within the coil.[27]

Management

Joint Reduction

Knee reduction should be attempted with the patient under conscious sedation. The reduction maneuver is one that reverses the deforming force. If this cannot be done in the emergency department, the patient should be brought to the operating room for a reduction performed under anesthesia. If successful, the knee should be splinted in 20° of flexion using a construct that prevents posterior subluxation of the tibia and minimizes traction of the vasculature. Immobilization provides stability, comfort, and relaxation of soft tissues. The splint should be windowed appropriately to allow for repeated vascular examination of the foot. We believe that a plaster splint provides more stability than does a premade, commercially available knee immobilizer, particularly in patients who are obese because their leg anatomy has a shape that fits poorly in most off-the-shelf immobilizers. Immediate postreduction biplanar radiographs should be obtained to assess the quality and maintenance of the joint reduction.

Vascular Status

The risk of injury to the popliteal artery led many authors to suggest that angiography should be performed for all knee dislocations;[28] however, advances in the management of vascular injuries have challenged this dogmatic approach. A protocol of careful physical

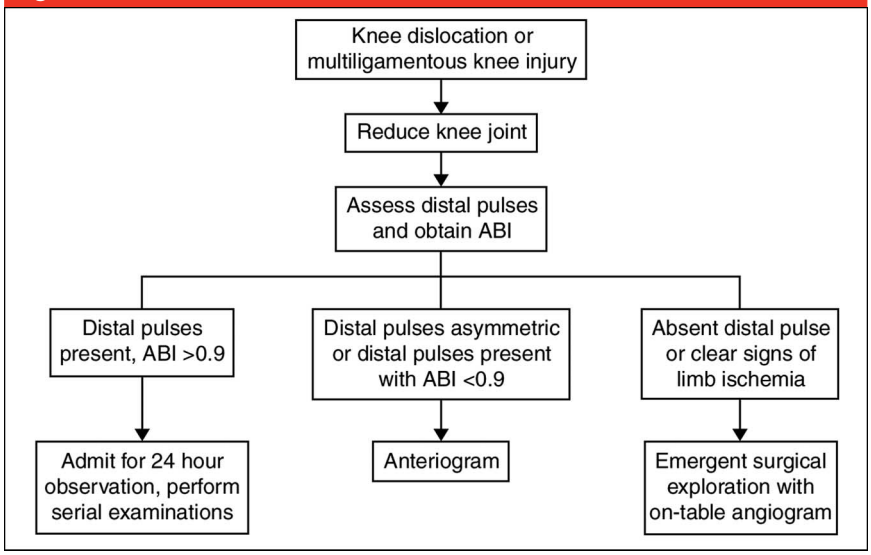
examination, ABIs, and selective angiography is well supported by the current literature.[29,30]

Subsequent studies have demonstrated that patients with normal pulses and ABIs ≥0.9 can be safely observed given the absence of evidence of lesions that require surgical repair.[29,31] One study found that a normal pulse examination has a negative predictive value of 100% for determining vascular injury.[29] In a study of 126 patients with knee dislocations, Stannard et al[31] demonstrated that physical examination alone has a sensitivity of 100% and a negative predictive value of 100% in identifying a clinically significant arterial injury. Patients were examined by the same physician every 4 hours for 24 hours after reduction.

When vascular compromise is suspected (ie, ABI <0.9, absent palpable and Doppler pulses, or other hard signs), further vascular imaging and a vascular consultation should be obtained. An angiogram performed outside the operating room can result in unnecessary delay.[30,31] Although angiography has been the standard of care, a CT angiogram (CTA) has a high sensitivity and specificity; although it is not the preferred test, CTA has the advantage of being noninvasive and requires less radiation.[32,33] If a CTA cannot be obtained or if there will be significant delay, an on-table angiogram can be performed instead.

Recently, duplex ultrasonography has emerged as an alternative to CTA or angiography for diagnosis of vascular injury. In one prospective study, ultrasonography was shown to have a sensitivity and specificity of 95% and 99%, respectively.[34] Weaknesses of duplex ultrasonography include its potential to miss small intimal tears, the operator-dependent nature of the test, and the availability of a technician; therefore, we do not recommend its routine use.[19]

Figure 4



Authors' algorithm for management of concomitant vascular injury in knee dislocation injury. (Adapted with permission from Nicandri GT, Chamberlain AM, Wahl CJ: Practical management of knee dislocations: A selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med* 2009;19 [2]:125-129.)

If a postreduction vascular examination shows absent pulses, an ABI <0.9, or hard signs of ischemia, vascular surgery consultation is imperative. A warm, pulseless extremity is deceptive and should not be accepted because the collateral flow around a significant arterial injury may maintain some perfusion for a period of time. An ABI <0.9 has been shown to have 100% positive predictive value for a vascular injury requiring repair.[25] Figure 4 illustrates our algorithm for management of a potential vascular injury associated with a knee dislocation or multiligamentous injury.

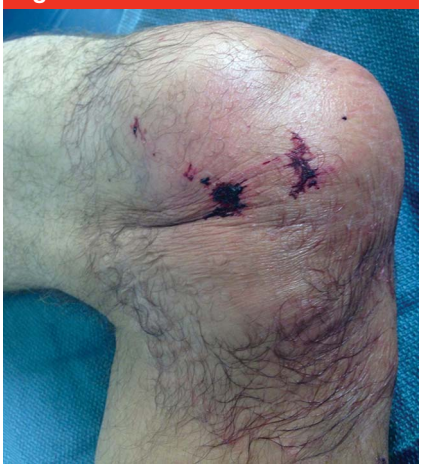
Neurologic Status

Injuries to the common peroneal nerve (CPN) are reported at a rate of 16% to 40% in patients with knee dislocations.[35] In most cases, the nature of injury is caused by stretching of the nerve as opposed to complete transection; therefore, patients with CPN palsy should be fitted with an ankle-foot orthosis and should undergo physical therapy to prevent an equinus contracture. CPN palsy alone is not an indication for immediate nerve exploration or repair unless there is an obvious transection noted through an open traumatic wound. If the CPN palsy persists at the time of definitive reconstruction of the multiligamentous knee injury, nerve exploration and neurolysis may be indicated for adherent hematoma and fibrosis.[35] If no clinical or electrical evidence of nerve continuity exists by 3 months, surgical intervention for direct or intercalary nerve repair is indicated.[36]

The Irreducible Knee

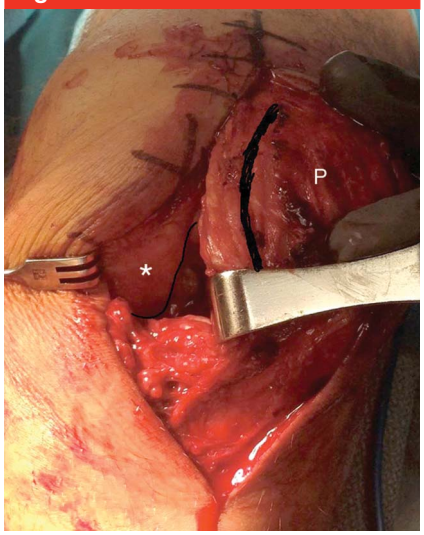
Most knee dislocations either reduce spontaneously or can be reduced with the patient under conscious sedation; however, cases of irreducible simple knee dislocations and fracture-dislocations in which one or both femoral condyles perforate the capsule and surrounding ligament architecture have been reported.[37,38]

Figure 5



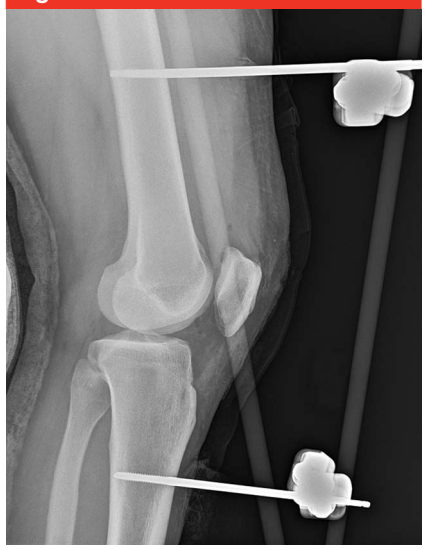
Clinical photograph demonstrating an irreducible knee dislocation, with threatened medial skin.

Figure 6



Intraoperative photograph of the knee demonstrating open reduction of a knee dislocation. The medial femoral condyle (asterisk) has perforated the retinaculum. P = patella

Figure 7



Lateral radiograph of the knee demonstrating spanning external fixation applied after reduction.

This often occurs in the setting of a lateral or anterolateral dislocation. In the event of unsuccessful reduction in the emergency department under conscious sedation, the patient should be taken urgently to the operating room for a reduction under general anesthesia. In many cases, the irreducible knee has pinched or threatened skin, particularly on the medial aspect of the knee (Figure 5).

The patient should be positioned on a radiolucent table that is compatible with angiography and fluoroscopy. If closed reduction performed under general or regional anesthesia is unsuccessful, the surgeon should be prepared to proceed to an open reduction of the knee joint. We recommend against the use of a tourniquet because this can exacerbate tissue ischemia and propagate thrombus formation in the setting of an intimal tear. A midline surgical incision with a medial parapatellar arthrotomy is useful to address the acute dislocation and later ligament reconstruction. The anatomy is only slightly distorted in most irreducible dislocations. The incision and arthrotomy are approximately the same length as those for a total knee arthroplasty except that extension into the quadriceps tendon is not required. The dislocated knee will have some distortion of the normal anatomic relationships; the patella will be tightly approximated to the lateral aspect of the trochlear groove, and the medial femoral condyle will have perforated the joint capsule (Figure 6). The key to the reduction is the use of a small retractor that is inserted between the capsule and the medial condyle to "shoehorn" the condyle back into the joint. This will produce a clear restoration of joint relationships despite multidirectional instability. If this maneuver is unsuccessful, the joint arthrotomy can be extended until the femoral condyle can be reduced. Examination of the knee and documentation after joint reduction facilitate the definitive repair. Fluoroscopy or plain radiography should be performed at this point to confirm the reduction and splinting.

External Fixation

If the knee is not able to be held in a satisfactorily reduced position, then the use of a spanning external fixator is recommended. Two pins placed anterolaterally in the femur and two pins placed anteromedially in the tibia are usually sufficient to maintain the reduction (Figure 7). Care must be taken to avoid placing the pins too close to the knee joint, which may contaminate the joint or complicate obtaining an MRI.

Postoperatively, the patient's neurovascular status should be closely examined, documented, and observed for at least 24 hours before discharge. The patient is kept non–weight bearing and wears a knee immobilizer, splint, or external fixator until definitive repair or reconstruction is performed.

Repair or Reconstruction

Definitive management of knee dislocation remains controversial, with management options ranging from completely nonsurgical to repair or reconstruction. Nonsurgical management has inferior results compared

with those of repair or reconstruction.[39] Management of some associated injuries, such as vascular injury or compartment syndrome, cannot be delayed. In the setting of compartment syndrome, a release should be done and wounds closed before any repair or reconstruction of the ligaments. Periarticular fractures may be spanned with an external fixator or stabilized acutely based on the surgeon's comfort and familiarity with the injury. Medial tibial plateau injuries that require reduction also necessitate a vascular examination because they may be fracture-dislocations. Other injuries that are not amenable to delayed reconstruction include patellar tendon tears, menisci displaced outside the joint, and periarticular avulsions of the meniscal attachments, iliotibial band, biceps femoris, or lateral collateral ligament.[40] Acute repair of these structures greatly enhances overall knee stability.

A staged reconstruction protocol has been proposed and described by several authors.[39,41] Overall results are good with delayed reconstruction; however, up to one third of knee dislocations have associated injuries that require more urgent attention. In a study of 25 patients who underwent primary repair of knee dislocations, good results were reported with early repair of all injured structures, and range of motion was similar to that achieved with delayed reconstruction, but 20% of patients required knee manipulation secondary to stiffness.[42]

Summary

Acute knee dislocations are a rare and underdiagnosed injury that can result from high- and low-energy mechanisms. A high degree of suspicion for this injury should be maintained in the setting of an occult presentation in which the patient reports substantial knee pain and has symptoms of neurovascular injury. Physical examination findings and diagnostic tools (eg, ABI, CTA, angiography) can direct treatment in a timely manner. Repeat examinations of the affected extremity are paramount. Emergent reduction should be attempted with appropriate sedation, postreduction imaging, immobilization, and repeat examinations. In the setting of irreducible dislocation, emergent intervention in the operating room must be undertaken, with a release of constraining structures. Swift treatment performed in conjunction with the general trauma and/or vascular surgeon can help improve outcomes and decrease complications, such as vascular injury, compartment syndrome, or poor function.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 19, 25, and 31 are level II studies. References 11, 12, 29, 36, 39, and 41 are level III studies. References 2-6, 8, 9, 13-16, 21-23, 28, 32, 33, 35, 40, and 42 are level IV studies. References 1, 7, 10, 17, 18, 24, 26, 27, 30, 34, 37, and 38 are level V expert opinion.

References printed in **bold type** are those published within the past 5 years.

1. Rihn JA, Groff YJ, Harner CD, Cha PS: The acutely dislocated knee: Evaluation and management. *J Am Acad Orthop Surg* 2004;12(5):334-346.
2. Peltola EK, Lindahl J, Hietaranta H, Koskinen SK: Knee dislocation in overweight patients. *AJR Am J Roentgenol* 2009;192(1):101-106.
3. **Sillanpää PJ, Kannus P, Niemi ST, Rolf C, Felländer-Tsai L, Mattila VM: Incidence of knee dislocation and concomitant vascular injury requiring surgery: A nationwide study. *J Trauma Acute Care Surg* 2014;76 (3):715-719.**
4. Icks A, Haastert B, Meyer G: Remarkable decline in hip fracture incidence in younger age groups in Germany. *Am J Public Health* 2007;97(10):1733-1734.
5. Seroyer ST, Musahl V, Harner CD: Management of the acute knee dislocation: The Pittsburgh experience. *Injury* 2008;39 (7):710-718.
6. Durbhakula SM, Das SP, Uhl RL: Traumatic anterior knee dislocation. *Orthopedics* 2002;25(10):1083-1085.
7. **McKee L, Ibrahim MS, Lawrence T, Pengas IP, Khan WS: Current concepts in acute knee dislocation: The missed diagnosis? *Open Orthop J* 2014;8:162-167.**
8. **Azar FM, Brandt JC, Miller RH III, Phillips BB: Ultra-low-velocity knee dislocations. *Am J Sports Med* 2011;39 (10):2170-2174.**
9. King JJ III, Cerynik DL, Blair JA, Harding SP, Tom JA: Surgical outcomes after traumatic open knee dislocation. *Knee Surg Sports Traumatol Arthrosc* 2009;17 (9):1027-1032.
10. Robertson A, Nutton RW, Keating JF: Dislocation of the knee. *J Bone Joint Surg Br* 2006;88(6):706-711.
11. Harner CD, Waltrip RL, Bennett CH, Francis KA, Cole B, Irrgang JJ: Surgical management of knee dislocations. *J Bone Joint Surg Am* 2004;86(2): 262-273.
12. Shelbourne KD, Klootwyk TE: Low-velocity knee dislocation with sports injuries: Treatment principles. *Clin Sports Med* 2000;19(3):443-456.
13. **Halvorson JJ, Anz A, Langfitt M, et al: Vascular injury associated with extremity trauma: Initial diagnosis and management. *J Am Acad Orthop Surg* 2011;19(8): 495-504.**
14. Patterson BM, Agel J, Swiontkowski MF, Mackenzie EJ, Bosse MJ; LEAP Study Group: Knee dislocations with vascular injury: Outcomes in the Lower Extremity Assessment Project (LEAP) Study. *J Trauma* 2007;63(4):855-858.
15. Kennedy JC: Complete dislocation of the knee joint. *J Bone Joint Surg Am* 1963;45: 889-904.
16. Green NE, Allen BL: Vascular injuries associated with dislocation of the knee. *J Bone Joint Surg Am* 1977;59(2):236-239.
17. Schenck RC Jr, Hunter RE, Ostrum RF, Perry CR: Knee dislocations. *Instr Course Lect* 1999;48:515-522.
18. Wascher DC: High-velocity knee dislocation with vascular injury: Treatment principles. *Clin Sports Med* 2000;19(3): 457-477.
19. Wascher DC, Dvirnak PC, DeCoster TA: Knee dislocation: Initial assessment and implications for treatment. *J Orthop Trauma* 1997;11(7):525-529.

20. Netter FH: *Atlas of Human Anatomy*, ed 5. Philadelphia, PA, Saunders Elsevier, 2010.

21. Niall DM, Nutton RW, Keating JF: Palsy of the common peroneal nerve after traumatic dislocation of the knee. *J Bone Joint Surg Br* 2005;87(5):664-667.

22. Bonnevialle P, Dubrana F, Galau B, et al; la Société française de chirurgie orthopédique et traumatologique: Common peroneal nerve palsy complicating knee dislocation and bicruciate ligaments tears. *Orthop Traumatol Surg Res* 2010;96(1):64-69.

23. Perron AD, Brady WJ, Sing RF: Orthopedic pitfalls in the ED: Vascular injury associated with knee dislocation. *Am J Emerg Med* 2001;19(7):583-588.

24. McCutchan JD, Gillham NR: Injury to the popliteal artery associated with dislocation of the knee: Palpable distal pulses do not negate the requirement for arteriography. *Injury* 1989;20(5):307-310.

25. Mills WJ, Barei DP, McNair P: The value of the ankle-brachial index for diagnosing arterial injury after knee dislocation: A prospective study. *J Trauma* 2004;56(6):1261-1265.

26. Howells NR, Brunton LR, Robinson J, Porteus AJ, Eldridge JD, Murray JR: Acute knee dislocation: An evidence based approach to the management of the multiligament injured knee. *Injury* 2011;42(11):1198-1204.

27. Ziran BH, Smith WR, Anglen JO, Tornetta P III: External fixation: How to make it work. *J Bone Joint Surg Am* 2007;89(7):1620-1632.

28. McCoy GF, Hannon DG, Barr RJ, Templeton J: Vascular injury associated with low-velocity dislocations of the knee. *J Bone Joint Surg Br* 1987;69(2):285-287.

29. Abou-Sayed H, Berger DL: Blunt lower-extremity trauma and popliteal artery injuries: Revisiting the case for selective arteriography. *Arch Surg* 2002;137(5):585-589.

30. Nicandri GT, Chamberlain AM, Wahl CJ: Practical management of knee dislocations: A selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med* 2009;19(2):125-129.

31. Stannard JP, Sheils TM, Lopez-Ben RR, McGwin G Jr, Robinson JT, Volgas DA: Vascular injuries in knee dislocations: The role of physical examination in determining the need for arteriography. *J Bone Joint Surg Am* 2004;86(5):910-915.

32. Peng PD, Spain DA, Tataria M, Hellinger JC, Rubin GD, Brundage SI: CT angiography effectively evaluates extremity vascular trauma. *Am Surg* 2008;74(2):103-107.

33. Gakhal MS, Sartip KA: CT angiography signs of lower extremity vascular trauma. *AJR Am J Roentgenol* 2009;193(1):W49-57.

34. Fry WR, Smith RS, Sayers DV, et al: The success of duplex ultrasonographic scanning in diagnosis of extremity vascular proximity trauma. *Arch Surg* 1993;128(12):1368-1372.

35. Mook WR, Ligh CA, Moorman CT III, Leversedge FJ: Nerve injury complicating multiligament knee injury: Current concepts and treatment algorithm. *J Am Acad Orthop Surg* 2013;21(6):343-354.

36. Garozzo D, Ferraresi S, Buffatti P: Surgical treatment of common peroneal nerve injuries: Indications and results. A series of 62 cases. *J Neurosurg Sci* 2004;48(3):105-112.

37. Braun DT, Muffly MT, Altman GT: Irreducible posterolateral knee dislocation with entrapment of the adductor magnus tendon and medial skin dimpling. *J Knee Surg* 2009;22(4):366-369.

38. Capito N, Gregory MH, Volgas D, Sherman SL: Evaluation and management of an irreducible posterolateral knee dislocation. *J Knee Surg* 2013;26(suppl 1):S136-S141.

39. Levy BA, Dajani KA, Whelan DB, et al: Decision making in the multiligament-injured knee: An evidence-based systematic review. *Arthroscopy* 2009;25(4):430-438.

40. Becker EH, Watson JD, Dreese JC: Investigation of multiligamentous knee injury patterns with associated injuries presenting at a level I trauma center. *J Orthop Trauma* 2013;27(4):226-231.

41. Levy BA, Fanelli GC, Whelan DB, et al; Knee Dislocation Study Group: Controversies in the treatment of knee dislocations and multiligament reconstruction. *J Am Acad Orthop Surg* 2009;17(4):197-206.

42. Owens BD, Neault M, Benson E, Busconi BD: Primary repair of knee dislocations: Results in 25 patients (28 knees) at a mean follow-up of four years. *J Orthop Trauma* 2007;21(2):92-96.