Original Articles

An Evidence-Based Approach to Multi-Ligamentous Knee Injuries

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Multi ligament knee injuries (MLKIs) are highly complex injuries with associated complications and often present with difficult management strategies. MLKIs may affect the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), medial collateral ligament (or posteromedial corner (PMC)), and lateral collateral ligament (or posterolateral corner (PLC)) in addition to other structures including the menisci, common peroneal nerve, and popliteal artery. MLKIs are highly associated with the male sex and are commonly seen in high-velocity motor vehicle accidents and low-velocity sports injuries. Given the multiple planes of movement in the knee and various primary and secondary stabilizers throughout those planes, there is great heterogeneity in an injury pattern and most involve the ACL and PCL. Initial evaluation of this injury includes assessment of lower extremity sensation, distal pulses, and ankle-brachial index (ABI). If vascular compromise is suspected, computed tomography angiography (CTA) or magnetic resonance angiography (MRA) are indicated to evaluate the vasculature. As opposed to CTA, MRA offers visualization of the soft-tissue structures that are commonly damaged in MLKIs. Initial management typically includes closed reduction of the knee with subsequent external fixation. Classification systems guide initial assessments; however, further management is unclear and leads the surgical team to decide the best, individualized management option for each patient. As a result, optimal surgical and postoperative treatment options remain complicated, and clinical outcomes remain difficult to predict. The purpose of this review is to consolidate the most up-to-date practices of the diagnostic workup, management, and treatment of MLKIs.

INTRODUCTION

Multi ligament knee injuries (MLKIs) are highly variable injuries with multiple complications and present with difficult management strategies.¹⁻³ MLKI is defined as a complete injury to two or more of the four major ligaments of the knee: the anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), medial collateral ligament (MCL) (and posteromedial corner (PMC)), and lateral collateral ligament (LCL) (and posterolateral corner (PLC)).^{1,3} These injuries are often associated with knee dislocations (KD) and neurovascular injury, most commonly affecting the popliteal artery (PA) and common peroneal nerve (CPN).^{1,3-6} The Schenck classification system is most com-

monly used to define MLKIs, which includes the number of involved ligaments and type of $KD.^{1,3,7}$

Assessment of the MLKI consists of MRI for static soft tissue injury in addition to radiography to demonstrate the dynamic consequences of the injury.^{3,5,7} Neurovascular assessment is essential in MLKI as approximately 40% of MLKIs and KDs have some degree of CPN palsy or vascular injury.^{8–10} Prompt physical examination and anklebrachial index (ABI) followed by arteriography in suspected vascular compromise should be completed as a missed vascular injury can be catastrophic.¹¹ Vascular injury must be treated immediately to prevent further loss of function and sequelae of further complications.

Operative treatment has been reported to produce significantly better patient outcomes than non-operative

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 Lmf82@georgetown.edu treatment.^{1–4} However, there is no consensus on the optimal timing of surgery (acute versus staged), reconstruction versus repair, graft choice, and other factors such as pre-operative rehabilitation. Consequently, there is currently no standardized treatment algorithm for MLKIs. The aim of this article is to summarize and provide an updated review of the current techniques and strategies utilized for MLKI management.

EPIDEMIOLOGY

MLKIs are heterogeneous and broadly described. The incidence of MLKIs ranges from 0.02-0.2% of all orthopedic injuries; however, recent reviews of the literature suggest this to be an under-representation.^{1,3,4} Factors such as spontaneous reduction in up to 50% of patients with a KD result in delayed or missed diagnoses.^{7,12} Additionally, there is an estimated 4:1 male to female predominance of injury across all MLKIs,² and young men aged 15 to 29 years old are at risk of MLKI from low-velocity sporting injuries, which constitute approximately 33% of all MLKIs.^{2,4,7} However, the majority of MLKI injuries result from high-velocity impacts such as motor vehicle accidents or falls from great heights.^{2–4,7} Obesity has been shown to be positively correlated with an increased risk of neurovascular injury and greater complication rates after an MLKI.^{2,3,7,13}

PATHOPHYSIOLOGY / INJURY PATTERN

BIOMECHANICS

The biomechanics of the knee can be defined by the planes of movement: internal and external rotation in the axial plane, anterior and posterior translation in the sagittal plane, and varus and valgus in the coronal plane.^{5,14,15} The internal rotational motion of the knee is primarily restrained by the posterior oblique ligament (POL) of the PMC throughout 0-30° of flexion and during full extension, while the superficial MCL (sMCL) is the primary restraint during increased angles of flexion and thought to be complementary to the POL.¹⁴⁻¹⁶ External rotation is primarily restrained by the structures of the PLC, with secondary restraint provided by the POL and other extra-articular structures.^{15–17} Anterior and posterior motion is well understood, with primary anterior restraint provided by the ACL and primary posterior restraint by the PCL.^{15,18} Both the ACL and PCL consist of similar anterior and posterior bundles that function in a codominant manner.^{5,15} Varus angulation is primarily restrained by the LCL, and valgus angulation is primarily restrained by the MCL.¹⁵

In non-pathological states, the primary restraints control stability; however, during MLKI, secondary structures play a larger role. For example, in PCL deficient knees, the POL provides important stability against posterior tibial translation. Biomechanical studies have also reported that severing the MCL can increase posterior tibial translation by 350% in PCL deficient knees.^{15,16} Understanding the biomechanical consequences of injuring primary and secondary stabilizers is crucial in the management of MLKIs.

PATHOPHYSIOLOGY & INJURY PATTERN

As primary and secondary stabilizers give the knee stability, disruption of these stabilizers through a variety of mechanisms leads to various patterns of MLKI. Dislocations occur most frequently in the knee due to anterior and posterior displacements, making up 30-40% and 22-33% of all KDs, respectively.^{5,12,15} The most common mechanism of an anterior displacement KD results from high-velocity trauma leading to hyperextension injury to the ACL and/or PCL.^{5,15} In general, hyperextension injuries lead to a higher incidence of vascular damage compared to other injury mechanism.^{5,15}

As the largest intra-articular ligament, the PCL plays a central role in all degrees of flexion about the knee. The majority of posterior displacements are characterized by injury to the PCL, with an estimated 79-87% of all knee dislocations having an associated PCL injury.^{5,19} The PCL is most often damaged in high energy traumatic events when direct impact to the proximal anterior tibia occurs in a flexed knee (dashboard injury), with a mid-substance PCL tear in the majority of cases.^{5,15,19} Overall, the most common pattern of MLKI is a complete injury to both cruciate ligaments with accompanying injury to the MCL or PLC.^{7,15}

Injury of the medial knee joint involves several stabilizing structures, and the MCL is the major medial stabilizer affected. The superficial MCL provides primary restraint against valgus stress, while the deep MCL provides secondary stabilization. As a result, complete injury to the MCL results in medial instability.^{6,15} In MLKI, MCL injury is often accompanied by damage to the ACL, PMC, or medial meniscus.^{5,20} Aside from the common high-velocity trauma, the MCL is often injured by low-velocity impacts during sports when acute valgus stress is applied to the knee.^{6,17,20} When PMC damage accompanies an MCL injury, instability in internal rotation arises as well as unrestrained posteromedial translation of the knee.^{5,6,15}

Lateral instability, like medial instability, arises from damage to several structures. The LCL plays a primary role in resisting external rotation and varus stress. It can be damaged by any combination of rotational, external, varus, and translational forces. Therefore, the LCL is rarely damaged in isolation.^{5,6,15} The PLC is commonly injured with the LCL, ACL, and PCL, and, when damaged, results in external rotational instability.^{21–24} Damage to the PLC is often caused by trauma to the anteromedial knee in the posterolateral direction that occurs in high or low-velocity settings.^{23–25}

MLKIs are comprised of a variety of injury patterns which introduces challenges in creating a standardized management protocol for these injuries.

CLINICAL PRESENTATION

VASCULAR

In the setting of MLKI with a knee dislocation, the clinical presentation often involves neurovascular compromise in addition to periarticular soft tissue injuries.^{8,26,27} Vascular compromise is reported in 3.3-80% of MLKIs and can be potentially limb-threatening if not identified and managed

promptly.^{8,10,27} While previous studies have reported the incidence of vascular injury in knee dislocations and MKLIs the be 1.6%-64%,²⁸ a recent systematic review in 2014 by Medina et al reported an estimated frequency of 18%.²⁹ MLKIs with suspected vascular injury (e.g. Ankle-Brachial Index <0.9) should be investigated with angiography, however, routine use of angiogram is in the setting of a normal ankle-brachial index and physical exam without a history of knee dislocation is not recommended for every case.^{3,4,30}

NERVE INJURY

The most commonly injured nerve associated with MLKI is the CPN. Incidence of CPN injury has been reported to occur in 8.2% to 40% of MLKI cases, with the highest incidence in lateral or posterolateral injuries.^{31,32} A retrospective study by Kahan et al. reported that MLKI involving the PLC was more likely to involve a CPN injury than mechanims not involving the PLC.³³ Additionally, MLKI in the setting of KD reported a higher incidence of nerve injury (38%) compared to a non-dislocated knee group (14%).³⁴

PERIARTICULAR SOFT TISSUE INJURY

Periarticular soft tissue injuries are characterized by injury to the meniscus or articular cartilage, with reported incidences of 55% and 48%, respectively.³² While it is not unusual for cases to include a tear of both the medial and lateral menisci, the lateral meniscus is the most commonly affected structure.³¹ Periarticular soft tissue injuries have been associated with a worse prognosis in patients with MLKI and should be addressed concurrently during management of the MLKI.³² A retrospective study with a minimum of two year follow-up reported significantly inferior International Knee Documentation Committee (IKDC) scores in patients who initially presented with meniscal or cartilage damage.³⁵

Fractures have also been reported in 10-20% of MLKI with KD, including tibial plateau and avulsion fractures such that advanced imaging and a high index of suspicion is paramount in these injuries.³⁶

DIAGNOSIS

PHYSICAL EXAM

MLKIs typically occur via a high-energy mechanism such that a thorough physical examination is essential to recognize the presence of any potentially life-threatening injuries. Burrus et al. reported that 27% of life-threatening injuries occurred concurrently with high-velocity knee dislocations.³⁷ Patients who present with polytrauma and knee injuries were demonstrated to be associated with a higher rate of severe injuries than those without knee injuries; therefore, a patient presenting with MLKI or knee dislocation should be assessed using the Adult Trauma Life Support principles.^{37,38} Recent algorithm-based protocols suggest reducing a dislocated knee as the first step in addressing MLKI or KD. Although 50% of cases spontaneously reduce, it is critical to identify and reduce a KD as it has been reported to prevent morbidity.^{30,36,38} In addition, a thorough neurovascular exam should be completed and

monitored closely.³⁸ Vascular injury must be identified and treated as soon as possible as an emergency as data has demonstrated delayed treatment of vascular injury increased the probability of compartment syndrome and/or amputation by 20%.,^{30,36,38} A study analyzing 25 cases of knee dislocation reported that 80% of vascular injuries treated more than eight hours from the time of injury resulted in limb amputation.³⁹

In cases of vascular compromise, the absence of pedal pulses has a sensitivity of 79% and specificity of 91%, whereas an ABI score of <0.9 has been demonstrated to have a sensitivity, specificity, and positive predictive value of 100%.³⁸ Consequently, pulse examination, as well as ABI measurement, must be performed in all patients presenting with MLKI. If distal pulses are absent following reduction of KD or if there are any signs of limb ischemia, surgical exploration with or without imaging is recommended.^{30,38} In the presence of distal pulses and ABI score >0.9, 24-hour observation with serial examinations may be done. Patients with asymmetric or present distal pulses with an ABI score <0.9 need to be emergently evaluated with subsequent imaging studies.^{30,38}

IMAGING

In the diagnostic workup for suspected vascular compromise in the presence of MLKI, CT angiography (CTA) has proven to be reliable; however, MRA has less exposure to radiation and has the benefit of simultaneously evaluating ligament damage.^{30,37} Vascular surgeons are, however, often more experienced in identifying vascular injury via CTA, such that teamwork and commination with the vascular surgeon are paramount in ordering their preferred imaging.

Characterization of the knee injury is pursued through a variety of imaging modalities. Due to the propensity for KD to spontaneously reduce, plain radiographs may appear normal and may not be effective in diagnosing ligament injuries. For instance, proximal tibiofibular joint injuries are missed in 9% of MLKI diagnosed with anterior-posterior (AP) radiographs.³⁶ Unrecognized injury to this joint could lead to chronic lateral knee pain, CPN injury, and instability of PLC repairs.⁴⁰

When an MRI is not readily available, stress radiography is an option that is relatively simple to obtain and can be performed at most hospitals and clinics. Stress radiographs are more effective than non-stress radiographs in identifying damage to ligaments and have been reported to be useful in preoperative planning.^{37,41} However, satisfactory stress radiographs require specific technical experience and are a painful examination for most patients in the acute setting, and could provide further injury to the neurovascular structures if already injured.⁴¹

MRI, when available, is the most effective imaging modality to identify MLKI and associated meniscal and chondral injuries. A review article by Porrino et al. illustrates the advantages of preoperative MRI in the setting of MLKI and KD, including the ability to perform preoperative planning and to increase postoperative functional outcomes.³⁶ A retrospective study by Goiney et al. demonstrated the importance of preoperative MRI imaging in de-

termining the most appropriate surgical treatment option for PCL injuries intraoperatively.⁴²

CLASSIFICATIONS

There is currently no classification system dedicated specifically to describing MLKIs; however, there are numerous systems for KD. One way to classify KDs is by describing the mechanism of injury in terms of its energy, known as the Energy of Injury Classification. It is divided into 1) high-energy KD, 2) low-energy KD, and 3) ultra-low-velocity KD.⁴³ High-energy KDs commonly involve motor vehicle collisions, falls from heights over 10 feet, and in polytrauma patients.⁴⁴ Low-energy KDs commonly occur in athletes and falls from a height of five to ten feet.^{45,46} Ultra-low-velocity KDs is a relatively new entity and are traditionally described in obese patients with a BMI greater than 48.⁴³

The most common anatomic classification of KDs in the Schenck Classification.^{30,43} The grading system outlines MLKI associated with ligament involvement from KD I - KD V. Each number value indicates the number of ligaments involved and KD V is characterized by MLKI with periarticular fracture. KD III refers to an injury to both cruciate ligaments and can be further classified with an "M" for medical injury and "L" for lateral injuries.^{43,47}

TREATMENT

Initial management of MLKI in the setting of KD includes reduction and immobilization to ensure perfusion to the limb and preservation of soft tissue.³⁰ Immobilization can be accomplished by not only non-invasive techniques such as bracing or splinting but also external fixation (EF) or internal fixation.⁴⁸ External fixation has been favorable in providing stability and monitoring of neurovascular and soft tissue status and can be done quickly such that it is the most common initial surgical treatment for joint stabilization.⁴⁸ Long term immobilization from external fixation may lead to arthrofibrosis, and as a result, some authors have described hinged external fixation in patients when definitive surgery may be delayed or in cases in which ligament reconstruction is contraindicated.⁴⁸

In general, operative management of MLKI has shown greater functional and clinical outcomes than non-operative treatment.^{38,49} Operative management has reported higher rates of return to work and return to sport without a difference in mean knee ROM when compared to nonoperative management.^{38,50} Given the superior outcomes of operative management, non-operative management is reserved for poor surgical candidates.

There are many factors that contribute to the timing of surgery, including neurovascular status at presentation, knee instability, and overall patient health.⁴⁹ The timing of surgery from the initial injury can be characterized as acute, staged, or delayed. Acute reconstruction/repair is typically defined as surgery within three weeks of injury, with the benefit of establishing normal knee kinematics; however, it has also been associated with the risk of arthrofibrosis and stiffness.³⁸ Staged surgery involves acute reconstruction of extra-articular medial and lateral structures, followed by delayed cruciate ligament reconstruction more than three weeks after the primary surgery. Early studies reported that delayed reconstruction may provide better postoperative ROM of the knee and allow of healing of extra-articular soft tissue structures that may heal non-surgically.^{51–54} However, more recent data on improved surgical techniques and aggressive rehabilitation has favored acute and staged surgery over delayed.^{38,49}

SURGICAL REPAIR

In general, surgical repair of the ligaments about the knee is reserved for cases in the acute setting. Surgical repair attempted after three weeks is more technically challenging due to poor tissue quality and difficulty defining the soft tissue planes.⁸

MCL

The MCL is one of the most common ligaments injured during MLKIs. The literature surrounding the best surgical option for these injuries is conflicting and highly debated.^{55,56} Some surgeons prefer to treat grade I (sprain) and grade II (partial tear) MCL injuries non-operatively; however, higher grade tears with ongoing laxity or instability should be treated surgically.⁵⁷ A recent systematic review by DeLong et al. described suture-only repairs to be the most commonly reported technique (49.5%), followed by Staples (12.1%) and suture anchors (11.2%).⁵⁸ Other studies have also recently described using an internal bracing repair technique.^{48,55} The benefit of internal bracing is that it allows for early rehabilitation and potentially faster recovery.^{48,55}

LCL

The LCL is one of the three components that make up the posterolateral corner (PLC) and acts to provide passive stabilization of the lateral knee against varus stress.⁵⁹ As such, is it common for LCL injuries to occur concomitantly with ACL tears and is relatively rare in isolation. One study has also demonstrated a failure to address PLC injuries in the setting of ACL reconstructions may increase the graft failure rate.⁶⁰ Similar to MCL injuries, grade I and grade II LCL tears are treated non-operatively, while grade III LCL tears may be surgically managed.⁶¹ Historically, LCL reconstruction was favored over repair techniques due to higher reported failure rates with primary repair.⁶² However, recent advancements in suture augmentation techniques have led to increased popularity amongst surgeons of repairing the ligament. Vermeijden et al. describe a technique to repair LCL tears, either in isolation or with PLC injuries, using suture augmentation.⁶⁰ The benefit associated with repair include immediate mobilization following surgery as well as improved functional outcomes and shorter rehabilitation times in a carefully selected patient population.^{60,63}

ACL

While ACL reconstruction has historically been the mainstay treatment option, repair of the ACL with augmentation has recently demonstrated acceptable outcomes in select patients.^{8,64} To determine if the ACL is possible to repair, the ligament is visualized through standard anterior medial and lateral arthroscopic portals.^{65,66} If repair is indicated in both ACL and PCL, the ACL repair is prioritized as this may help to facilitate PCL repair.⁶⁶ Excellent clinical outcomes following repair have been reported in patients with tears of the proximal region of the ACL, as opposed to mid-sub-stance tears.⁶⁷

PCL

Surgical repair of the PCL is not recommended as a systematic review by Levy et al. concluded that PCL repairs result in worse clinical outcomes when compared to PCL reconstructions.⁶⁸ However, a more recent study had demonstrated good outcomes following PCL repairs in cases of bony avulsions.⁸

SURGICAL RECONSTRUCTION

While non-operative and operative treatment are both options for MLKIs, surgical reconstruction has been associated with the most reliable and consistent positive patient-reported outcomes.⁶⁹ There are a variety of reconstruction options available depending on the ligament(s) affected, including autograft, allograft, or synthetic grafts. Graft choice is based on the number of ligaments affected, graft availability, surgeon preference, and the surgical technique chosen.⁸ Autograft options include hamstring (gracilis and semitendinosus) tendon, bone-patella tendon-bone (BTB), and quadriceps tendon (with or without distal bone block).⁷⁰ The grafts can be harvested from the ipsilateral or contralateral knee, with some surgeons advocating for contralateral knee harvest to avoid additional morbidity to the injured knee.⁸ Allograft options include Achilles tendon, extensor mechanism (quadriceps tendon, patella, patellar tendon), BTB, and tibialis anterior tendon.⁷⁰

ACL

Reconstruction of the ACL is the standard of care for treating injuries of this ligament in the setting of MLKI.⁷¹ There are a variety of different options regarding autologous and allogenic grafts that have demonstrated biomechanical reliability.⁷² However, there are significant controversies regarding graft choice and reconstruction methods.

PCL

Similar to ACL reconstructions, there is no consensus on optimal PCL reconstruction. The two main techniques are the tibial inlay and the transtibial approach.⁶⁹ One disadvantage of the inlay technique is that the approach is performed from the posterior aspect of the knee, and patient positioning may need to be altered during the surgery depending on which other ligaments may be affected.⁷³ The use of allograft and autograft has been reported in the literature; however, there is not a demonstrated superior graft choice.^{69,74,75}

MCL

While most isolated medial knee injuries can be managed non-operatively, grade III MCL tears are candidates for surgical reconstruction. In addition, an MCL tear in the setting of MLKI with demonstrated valgus laxity should be addressed at the time of surgery.⁸ A systematic review by Kovachevich et al. concluded that both MCL repair and reconstruction yielded satisfactory clinical outcomes.⁷⁶ However, certain characteristics such as mid-substance tears are poorer candidates for repair and may require reconstruction. Anatomic reconstruction of the MCL involves creating a distal tibial tunnel and fixing the MCL graft within the tunnel.⁷⁷

PLC/LCL

A recent systematic review reported superior outcomes following PLC reconstruction (9% failure rate) as compared to repair (38% failure rate).⁷⁸ There exists both anatomic and nonanatomic reconstruction techniques. The nonanatomic reconstruction techniques include Larson's technique (fibular based) or tibial based two-tailed reconstruction.^{78,79} Recent biomechanical evidence has demonstrated anatomic reconstructions may improve load sharing characteristics and reduce the risk of graft failure.⁸

If injured, the LCL should also be addressed during PLC reconstruction. Multiple techniques to reconstruct the LCL have been described including the PT-LCL reconstruction technique, Warren technique, and the Larson technique.⁵⁷ The LCL graft may be constructed from anterior tibialis or semitendinosus tendon allograft.⁶⁹

Postoperative rehabilitation is an important factor for recovery from a PLC injury. Following a PLC reconstruction, patients often begin immediate range of motion on the operative knee to prevent arthrofibrosis and stiffness.⁶⁹ However, this must be balanced with overworking the knee and preventing proper healing.⁶⁹

OUTCOMES & PROGNOSIS

With the advent of novel technologies, it is critical to understand the prognosis and outcomes of these new MLKI surgical techniques. Bakshi et al. studied National Football League athletes and the rate of return to play following an MLKI demonstrating players with concomitant ACL and MCL injuries were more likely to return to sport than players with concomitant ACL and PCL/LCL tears.⁸⁰

The type of graft used in reconstructions is an important factor that may affect clinical outcomes. Billières et al. conducted a case series evaluating postoperative laxity of patients with different MLKIs who underwent single stage reconstructions with allograft for all injured ligaments.⁸¹ At 24 months follow-up, the authors observed that MLK reconstruction using fresh-frozen allografts resulted in 95% (19/20) return to sport at the same level, while only 5% (1/20) underwent reoperation for arthroscopic arthrolysis.⁸¹ Additionally, Lee et al. used a combination of allografts and autografts for reconstruction, specifically allografts to reconstruct the PCL, PLC, and PMC, while using mostly quadriceps autografts for ACL reconstructions. The authors

reported a similar return to work rate at 94% (31/33) as well as a similar complication rate of 9.5%.⁸² Graft choice should be guided by the number of ligaments affected, availability of grafts, and surgeon preference.

Vascular compromise and subsequent limb amputation are potential devastating complications associated with MLKI in the setting of KD.⁸³ Sanders et al. compared Lysholm and IKDC scores between patients with MLKI and vascular injury to controls with MLKIs and no vascular injury.⁸³ The results demonstrated lower mean postoperative scores for both Lysholm and IKDC scores in the vascular cohort as compared to controls (62.5 versus 86.4 and 59.7 versus 83.8, respectively).⁸³

CONCLUSION

MLKIs are complex and severe injuries that may involve multiple ligaments, fractures, nerve damage, and vascular injury. Although MLKIs present with diverse injury patterns and clinical presentations, surgeons have to be able to distinguish these injuries in both the acute and delayed setting to determine the best management. Recognizing limb and life-threatening injuries that are common with MLKIs is paramount as these injuries are associated with a high morbidity in both the acute and chronic setting.

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