Management of Multiligament knee injuries (mlki's) with concomitant fractures and neurovascular injuries- A descriptive review

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Abstract

MLKI'S are on the rise due to increasing number of Road traffic accidents (RTA). MLKI's are often challenging injuries to treat especially when they are associated with periarticular fractures and/or neurovascular deficits. These additional knee injuries can affect the surgical timing of ligament injuries and also can affect the outcome. The existing literature lacks clear-cut guidelines regarding approach and management of MLKI's with these associated injuries. This descriptive review is an attempt to highlight key concepts from the existing literature, along with our experience in treating MLKI's with these associated injuries and formulate protocols that could help clinicians in their day to day practice. **Keywords:** MLKI, KDV, Periarticular fractures, Neurovascular injuries, Vascular assessment, CPN palsy, Staged reconstruction.

Introduction

There is a surge in Multiligamentous knee injuries (MLKI's) in the recent past due to increasing number of road traffic accidents (RTA). MLKI's are defined as injuries those involve at least two of the four major ligaments of the knee (anterior cruciate ligament [ACL], posterior cruciate ligament [PCL], posteromedial corner [PMC] including the medial collateral ligament [MCL], and posterolateral corner [PLC] including the lateral collateral ligament [LCL]) [1]. Most of these would be associated with a knee dislocation either apparent at the time of presentation or spontaneously reduced [2]. MLKI's are often challenging injuries to treat especially when they are associated with periarticular fractures and/or neurovascular deficits. MLKI's are commonly associated with tibial plateau fractures with medial tibial plateau fractures being more commonly associated with PLC injuries whilst lateral tibial plateau fractures commonly associated with MCL tears and to a lesser extent with ACL tears [3]. The overall incidence of vascular injury with MLKI's requiring surgical intervention is around 5.6% and about 14-40% of MLKI's are associated with CPN palsy [4, 5]. The existing literature lacks clear-cut

guidelines regarding approach andmanagement of MLKI's with these associated injuries. The purpose of this review is to discuss a systematic approach to MLKI's with these associated injuries inorder to aid in the better assessment and management of MLKI's especially when associated with periarticular fractures and /or neurovascular injuries.

Pathoanatomy

All MLKI's are considered as spontaneously reduced knee dislocations unless proved otherwise as up to 50% of these dislocations are spontaneously reduced [6]. These are most commonly due to high energy trauma (RTA) & to a lesser extent due to low velocity trauma (collision sports) & Ultra low velocity trauma (morbidly obese) [7]. The most common combination of ligamentous injuries to occur is KD III-M constituting 52.4% of the injuries and KD III-L comprising 28.1% [2]. It is important to understand that the medial structures not only include the MCL but also the posteromedial capsule, the posterior oblique ligament (POL), the oblique popliteal ligament(OPL), semimembranosus & the posterior horn of medial meniscus constituting the posteromedial corner (PMC)

while the lateral collateral ligament (LCL), popliteofibular ligament, popliteus tendon, and the posterolateral capsule constitute the posterolateral corner (PLC). These are important soft tissue stabilizers of the knee & injury to any of these corners with cruciate ligament injury can potentially render the joint unstable.

Fracture dislocations of the knee are relatively rare injuries that involve disruption of ligamentous structures of the knee joint, along with associated periarticular fractures. As these disrupting forces traverse through the joint patterns of ligamentous and bony injuries that occur are mainly governed by the magnitude of the trauma, the position of the leg at the time of injury, and the quality of bone sustaining the injury [8,7]. These fractures can considerably alter joint geometry and make proper tunnel placement difficult while planning a single stage reconstruction on the other hand fixing the fractures first can result in hardware interference during ligament reconstruction in the subsequent stage.

MLKI's are particularly susceptible for vascular injuries due to tight adherence of the vessel at the adductor hiatus proximally and the soleus arch distally. This vascular tether makes popliteal vascular bundle vulnerable to



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Table 1: Classification of MLKI/Knee dislocation(12)	
TYPE	DESCRIPTION
KD I	Knee dislocation with either ACL or PCL intact
KD II	Bicruciate injury with collateral intact
KD III	Bicruciate injury with MCL or LCL intact
	KD3M: ACL+PCL+MCL injury
	KD3L: ACL+PCL+LCL injury
KD IV	Bicruciate injury with both collaterals injured
KD V	Periarticular fracture dislocation
(Suffixes C & N are used for vascular & nerve deficits respectively)	

n, thrombosis, dissection, intima disruption, pseudoaneurysm of the popliteal vascular bundle. Direct vessel compression and full thickness tears are seen more with posterior knee dislocations while anterior dislocations are more associated with traction type of injury causing more of intimal or intimal-media tears [9].

As far as association of nerve injury with MLKI is concerned, this review mainly focusses on CPN palsy, as it is the most commonly involved nerve in MLKI's. CPN is constrained mainly at two points at the knee, proximally as it winds around the fibular neck and secondly at the intermuscular septum. These anatomical constraints gives little room for the nerve to accommodate extreme joint positions especially seen with MLKI's [10, 11]. With extreme varus stresses at the knee, fibular neck can act as distraction pulley, resulting in different degrees of tractional injury [4].

Classification

The most commonly used classification for MLKI is the Schenk Anatomical classification (Fig.1). Periarticular fractures with MLKI's fits into the KDV subgroup, withthe suffix 'C' & 'N' denoting associated vascular and neurological injuries respectively [12](Table



1). Contrary to the belief that medial sided injuries are more associated with neurovascular injuries, recent studies demonstrate a higher incidence with KD-IIIL type of MLKI's [2].

Management

Mlki's with periarticular fractures:

Traditionally, a staged approach is advocated were in the fractures are fixed first & ligamentous injuries are reconstructed later after fracture healing with or without hardware removal [13]. Initially, detailed evaluation including plain radiographs, CT and MRI are necessary for a thorough understanding of the bony/ligamentous injuries. For undisplaced fractures/rim fractures/low energy fractures immobilization in a long knee brace and open reduction internal fixation (ORIF)/closed reduction internal fixation(CRIF) within 5-7 days is recommended. We can also consider reconstruction of associated ligament injuries at the same setting after an intraoperative evaluation for ligament laxity after fixing the fractures. For displaced high energy fractures with or without joint subluxation, reduction of the joint & stabilization with an external fixator maybe necessary until the soft tissue edema settles. After 5-7 days, Ex-Fix removal and ORIF of the fracture is recommended. As most of these combined injuries may result in stiffness eventually, it's important to reassess these injuries at regular intervals. At 8-12 weeks of follow-up, if the patient has only stiffness, we encourage range of

motion(ROM) exercises whilst symptoms of giving way would necessitate a clinical and radiographic stress testing in order to plan a ligament reconstruction. This concept of staged reconstruction still remains controversial with recent studies suggesting favorable outcomes even with a single stage approach with ORIF & ligament reconstruction at the same setting [8]. An algorithm followed in our institute while dealing with KD-V injuryis described in Table 1.

Ourexperience

Case Example:

A 28-year-old male patient sustained a KD-V injury after a Road traffic accident (RTA). Initial evaluation

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showed an anteromedial fracture of the tibial plateau with knee subluxation with no neurovascular deficits (fig. 2 a). MRI showed a PCL & MCL tear (fig. 2 d). We performed a staged treatment wherein we first fixed the periarticular fracture and stabilized the knee while the ligaments were reconstructed in the second stage. The tibial condyle depressed fracture was elevated & fixed using four 3.5mm cortical screws using a medial approach & knee was stabilized using an external fixator (Ex-Fix) in 20 degrees of flexion (fig. 2 e). External fixator was removed at 6 weeks and patient was encouraged knee Range of motion (ROM) exercises. Patient followed up only after 5 months post injury with knee instability and pain. Clinical evaluation & stress x-rays showed a posterior sag (fig. 2 f) and a medial opening on valgus stress. We performed an arthroscopic PCL reconstruction using contralateral semitendinosus & gracilis graft after removing the posterior cortical screws in order to drill the tibial tunnel & MCL reconstruction using ipsilateral semitendinosus autograft (fig.2 g).

Mlki's with vascular injury

In a MLKI, vascularity is of utmost priority, hence a methodical approach should be followed during initial assessment as missing a popliteal artery injury could be potentially disastrous. A systematic approach to physical examination by Nicandri et al. in a MLKI is a useful protocol which recommends that a ABI> 0.9 and a palpable peripheral pulse rules out any need for further diagnostic work-up for detecting vascular injury [14]. ABI testing has also proved to be effective in previous studies with a specificity of 80-100% and a sensitivity of 95-100% in detecting surgically appropriate vascular injuries [15]. Maslaris et al. have demonstrated the use of CT angiography (CTA) instead of a more invasive arteriogram if the patient has any abnormality on physical examination. Additionally, their protocol includes soft signs of vascular injury(SSVI), hard signs of vascular injury (HSVI) with poor prognostic factors and specific recommendations for each category [16]. Other investigators also highlight the advantage of using an MRI angiogram that would also help in identifying ligament injuries apart from evaluating the vasculature, which may be beneficial in a MLKI scenario [17]. An algorithm followed in our institute for MLKI's with vascular injury is shown in Fig. 3. Once a significant vascular injury is detected, vascular repair for short segmental injuries and



Figure 2: 28-year-old male patient with KD-V injury.

(a, b, c): CT cuts of the proximal tibia showing a depressed anteromedial fracture of the proximal tibia with knee subluxation. (d) MRI cuts showing PCL & MCL tear with undisplaced ACL avulsion. (e) Immediate post-operative x-ray after ORIF of tibial fracture and Ex-Fix application. (f) Posterior sag at 5 months follow up. (g) After Arthroscopic PCL & MCL reconstruction



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mental lesions is advised [4]. Following the repair/bypass grafting, the joint should be maintained in reduction at least for a period of 8-12 weeks by using a hinged external fixator [18]. A prophylactic fasciotomy may also be indicated after vascular repair to prevent secondary injury due to vascular reperfusion [4].

Functional outcomes following vascular repair in MLKI are not very encouraging. The outcomes following Vascular injuries in MLKI was first cited in the LEAP study by Patterson & colleagues [19] who reported moderate to high level of disability at 2 years of follow-up with almost 20% patients presenting to the hospital with limb ischemia requiring an amputation. Sanders et al. concluded that MLKI patients undergoing bypass grafting following vascular injury have a significantly lower functional scores than patients without vascular involvement [20].

Case Example:

A 38-year-old male patient presented to us after an RTA with anterior dislocation of right knee with absent pulses and CPN palsy (fig. 4 a). Patient was immediately taken to the operating room; knee was reduced and a spanning Ex-Fix was applied. Plastic surgery intervention was sought for vascular repair. Patient was turned to prone position, a generous skin incision was given from distal thigh to proximal leg & the tight fascial compartments were released. Popliteal neurovascular bundle was exposed and popliteal artery was found to be thrombosed for about 10cm. Thrombotic segment was excised and a repair was performed using reversed interposition great saphenous vein graft using 9-0 ethilon (fig. 4 b). Total ischemia time was 5 hours when vascularity was completely restored. SSG cover was done in 5 days. Ex-fix was removed at 2 months & an MRI was done that showed a Proximal tear of ACL & PCL with LCL injury (fig. 4 c). Doppler evaluation showed an intact repaired popliteal artery showing pulsatile flow (fig. 4 d). As the knee was stiff with ROM ranging only 0-30 degrees despite physiotherapy exercises for at least one month, we decided only to perform an arthroscopic arthrolysis to improve the ROM. At his last follow up 8 months post injury, patient had up to 120 degrees knee ROM with a stable knee and a partially recovered foot drop (fig. 4 e & f).

MLKI's with concomitant neurological injuries



Figure 4: 38-year-old patient with KD-III L injury post anterior knee dislocation with popliteal artery thrombosis & CPN palsy. (a) Radiograph at initial presentation (b) thrombosed segment of the popliteal artery (c) Harvested great saphenous vein graft (d) completed bypass graft repair, (e) immediate radiograph after popliteal artery repair and spanning Ex-Fix application (f) MRI after Ex-Fix removal showing ACL, PCL injury with thickened LCL suggestive of injury (g) Arterial doppler before arthroscopic arthrolysis showing normal flow and spectral pattern (h) Skin scarring over the grafted site after popliteal artery repair at 8 months follow up (i) 120 degrees of knee ROM



Initial assessment of neurological injuries should include a full motor examination as per Medical Research Council Grading (MRC) & sensory examination of the lower extremity. A high index of suspicion must be observed in obtunded patients, patients with high risk of neurological injury like obesity, ipsilateral fibular neck fracture, high velocity injury & associated vascular involvement, where in a thorough neurological assessment may not be possible [21]. Though nearly all patients have neurological deficits at initial presentation, a small subset may develop deficits after 24-48 hours after injury due to a developing hematoma leading to nerve compression and would definitely improve after surgical decompression [22].

Electrodiagnostic studies have an important diagnostic and a prognostic role in nerve injuries. These tests are more beneficial at 4-6 weeks when abnormal activity can be detected in denervated muscles on EMG, which can also be repeated at 4-6 weekly interval to prognosticate these injures and also to recommend if any further intervention is required to regain function [23]. Ultrasound and routinely sequenced MRI can be employed to add additional information to the diagnosis like location of the injury, compression by hematoma or scar tissue, neurotmesis or axonotmesis & gap length between cut ends of the nerve. However, accuracy of these modalities is still limited & interpretation may be solely operator dependent [24].

The goal in treatment of a patient with CPN palsy in a MLKI is to restore ankle dorsiflexion. In these cases, ligament reconstruction may be carried out first while nerve deficits should be followed up closely with timely clinical and electrodiagnostic testing at regular intervals. Splinting of the affected limb with an Ankle Foot Orthosis (AFO) to prevent

plantarflexion contracture is required while waiting for nerve recovery. Neurolysis is indicated in only those patients who are planned for a lateral sided repair/reconstruction [5]. If a neurotmesis is detected during neurolysis, direct nerve repair can be attempted or the damaged segment could be resected, tagging the nerve ends for future nerve grafting or even an early posterior tibialis tendon transfer (PTTT) can be considered. Nil clinical improvement or improvement in NCS/EMG, or a partially recovered nerve function with MRC <3/5 at 6 months follow-up, is an indication for PTTT. The PTTT can be transferred to the dorsum and can be attached to the medial cuneiform/ tibialis anterior tendon. PTTT provides good dorsiflexion at ankle, though the strength of dorsiflexion may be reduced as compared to the normal side. A useful algorithm in managing CPN palsy associated with MLKI's is outlined in fig. 5.

Our experience

Case Example:

A 25-year-old male patient came to us with an anteriorly dislocated knee and wound over the posterior aspect of distal thigh with signs of common peroneal nerve palsy with no vascular deficits (fig. 6 a). He underwent emergency reduction and stabilization of the knee using a spanning Ex-Fix & debridement of the wound (fig. 6 b). Four days later he underwent split thickness skin grafting (SSG) at the wound area. He underwent Ex-fix removal 2 weeks later and was advised knee ROM exercises. MRI post Ex-Fix removal indicated ACL, PCL



Figure 6: 25-year-old patient with KD-III L injury with associated CPN palsy and wound over posterior aspect of distal thigh.

(a) Radiograph at initial presentation, (b) immediate post-reduction radiograph, (c) MRI after Ex-Fix removal showing ACL and PCL injury with femoral sided LCL injury(blue arrow), (d) Post PCL & ACL reconstruction & LCL repair, (e) 6 months follow-up clinical profile with a good ROM, blue arrow showing the healed scar employed for neurolysis & active dorsiflexion of the left ankle during gait

tear with LCL femoral sided injury (fig. 6 c). As the knee remained stiff even after 2 weeks of physiotherapy, we undertook arthroscopic arthrolysis to gain 120 degrees of flexion on table and reconstructed the ACL and PCL using Bilateral semitendinosus & gracilis autografts & performed a neurolysis of the CPN while doing LCL avulsion repair using bio screws (fig. 6 d). At 6 months follow-up patient had a stable knee, with good ROM & a fully recovered CPN palsy (fig. 6 e).

Conclusions

MLKI's are challenging injuries to manage especially when they are associated with concomitant periarticular fractures and/or neurovascular injuries. A systematic approach should be followed in the diagnosis and treatment of these associated injuries which could improve the clinical outcome in thisscenario. A single stage/a staged reconstruction can be employed for periarticular fractures associated with MLKI's depending on type of fracture and joint stability. A methodical vascular assessment should be carried out in a case of MLKI to rule out vascular injury, and a delayed reconstruction may be planned. In cases of MLKI's associated with nerve deficits specially a CPN palsy, ligament reconstruction may be carried out first while nerve deficits should be followed up closely with timely clinical and electrodiagnostic testing at regular intervals, and a PTTT can be considered as a salvage option if nerve function does not improve even after 6 months.

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Conflict of Interest: NIL Source of Support: NIL

How to Cite this Article

Sundararajan S R, D'souza T, Rajagopal R, Rajasekaran S | Management of Multiligament knee injuries(mlki's) with concomitant fractures and neurovascular injuries- A descriptive review | Asian Journal of Arthroscopy | January-April 2020; 5(1): 9-13.