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Sequence of Bone and Soft Tissue Damage and Correlation to Radiographic Measures in a Novel Cadaveric Posterolateral Corner Injury Model

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Abstract

The purpose of this study is to observe anatomical structure damage to the knee following a posterolateral corner injury and to describe a possible correlation between damaged structures and physical exam and radiographic findings. The physical exam techniques traditionally used to assess this injury include the varus stress test, the posterior drawer test, and the recurvatum test. In this study, we will use radiography to assess bony landmarks of the knee at rest and undergoing these tests, both before and after induction of a posterolateral corner injury in a cadaveric lower limb. This will better classify the structures damaged in varying degrees of posterolateral corner injury. The information will inform surgeons of expected damage to the bone and soft tissue structures of the knee, based on their physical exam and radiographic findings, in order to better prepare them for surgical repair of the joint. We also propose a novel mechanism by which to induce a posterolateral corner injury in cadaveric lower extremities, which can be used in future research.

Key Words: posterolateral corner, varus stress, orthopaedics, recurvatum angle, cadaveric model

Introduction/Literature Review

The posterolateral corner (PLC) structures of the knee are responsible for the primary restraint to varus forces on the joint, along with posterolateral rotation of the tibia relative to the femur.[1] [2] The three main stabilizing components of the PLC are the lateral collateral ligament (LCL), popliteus tendon (PLT), and popliteofibular ligament (PFL).[3] The posterolateral capsule of the knee joint is an additional static stabilizer of the knee, along with the LCL and PFL, while the popliteus tendon and muscle act as dynamic stabilizers with the iliotibial band and biceps femoris tendon and muscle.[4] Assessment of the degree of posterolateral corner injury can be made radiographically by assessing gapping of the lateral tibial plateau and lateral femoral condyle upon varus stress, with roughly 4.0 millimeters (mm) of additional gapping compared to a healthy knee indicating a grade-III posterolateral corner injury.[5] [6] [7] In addition to ligamentous and capsular injury, common peroneal nerve (CPN) damage has been observed in posterolateral corner injuries, particularly when avulsion fractures of the fibular head or damage to the biceps tendon are involved, as the anatomical location of this nerve is intimately related to these structures.[8] An ordering of expected structural damage correlating to injury severity has not been established to our knowledge, but we aim to describe this possible association between structural damage and injury severity, based on varus stress test and recurvatum test physical exam findings. One common mechanism of injury (MOI) to the PLC is a direct blow to the anteromedial portion of the knee. Similar hyperextension and varus stress can be induced through non-contact mechanisms that will also cause PLC injury.[9] We propose an *in vitro* model that replicates the MOI for PLC injury in full cadaveric lower extremities, adapting a similar principle as has been applied previously in a lateral patella injury cadaveric model.[10]

Hypothesis

There will be a correlation between physical exam findings and injury severity in posterolateral corner injuries, as well as a sequential progression of damage to structures that remains consistent between each joint tested.

Methods

Context/Protocol

Twelve total fresh frozen cadaveric lower limbs were used in this study in order to establish statistically significant data. Exclusion criteria for limbs included any prior knee surgery, artificial joints, screws or plates in the knee, or any previously sustained knee injury. The limbs studied were from cadavers donated to Miami Valley Hospital for educational and research purposes with prior consent by all donors. IRB approval was not required for this study, which was approved by the Human Investigation and Research Committee at Miami Valley Hospital. To radiographically assess the bony landmarks of the knee joint, radiographic images were taken with a C-arm, as described below.

To induce a posterolateral corner injury, we proposed a novel varus-hyperextension injury model. The limbs were placed across the gap between two surgical tables, resting on surgical foam blocks or towels at the distal tibia to allow for dynamic movement and to avoid damage to the ankle joint. The limb was laterally rotated by bumping the contralateral hip with a foam block, until a level surface was created between the medial border of the patella and the medial epicondyle of the femur. A padded strap was placed across the joint space between the distal femur and tibial plateau. Weight was gradually added to this strap until a posterolateral corner injury of the limb was induced. Injury severity was varied from joint to joint, based on

degree of hyperextension and audible tissue damage, in order to assess a range of injury severities and to draw correlations between physical exam findings and structures damaged.

Data Collection

Cadaveric limbs received were measured for the following anatomic dimensions: circumference of femoral condyle, circumference of tibial plateau, and circumference of middle calf. Posterior drawer tests were performed to confirm that the posterior cruciate ligament (PCL) of each limb was intact. X-ray images of the knee were acquired using a C-Arm. Six images were saved both before and after injury induction. Images included anteroposterior images at full extension with the limb relaxed and with the limb under varus and valgus stress, lateral images at full extension with the limb relaxed and undergoing a recurvatum test, and lateral images at 90 degrees with the limb relaxed and undergoing a posterior drawer test. Twelve total images were saved for each knee joint. Following collection of radiographic images, dissection of the joint was performed to document visual observations and note what ligamentous structures were injured in the posterolateral corner and elsewhere. Other injured structures or signs of injury such as bony avulsion, rupture of the joint capsule, and presence of bone fragments were noted. Specific structures observed during dissection included biceps tendon, CPN, ACL, PCL, LCL, popliteus tendon, popliteus muscle, popliteofibular ligament, lateral gastrocnemius insertion, and the joint capsule.

Data Analysis

All images were uploaded onto PACs software for analysis and measurement. Lateral joint space under varus stress radiography was assessed both pre-injury and post-injury by measuring the perpendicular distance between the most distal lateral femoral condyle and the corresponding tibial plateau on anteroposterior images. The difference between these

measurements before and after injury induction was named the lateral joint space "gapping". Hyperextension angle was measured on each cadaver before and after injury by assessing the lateral radiograph during the recurvatum test. Recurvatum angle (RA) was measured as the angle between the femoral medullary canal and the tibial medullary canal.

Results

A total of twelve unique cadaveric lower extremities (specimens) were put through our varus-hyperextension injury model. Two specimens were not found to have any structures injured, so data for theses specimens was not included to allow for accurate comparison of pre-injury and post-injury data. Four other specimens were found to have tibial plateau fractures on fluoroscopic imaging as a result of the model. The stress radiography data was excluded for all specimens with a fracture as mobility through the fracture site would confound any measurements made on stress radiography.

Results for all specimens are outlined in Table 1. These results include structures that were found to be damaged or compromised in each specimen, change in lateral joint space on varus stress AP radiography post-injury compared to pre-injury ("gapping"), and change in recurvatum angle on hyperextension stress lateral radiography post-injury compared to pre-injury. Six out of twelve specimens were included in final data analysis. Post-injury measurements were found to be statistically significantly different from pre-injury measurements on paired t-test for lateral joint space under varus stress (Pre-injury mean: 7.78 mm, Post-injury mean: 13.6 mm, Mean difference: 5.80 mm, 95%CI: [2.44 mm – 9.16 mm], $p = 0.0068$). Post-injury measurements were found to be statistically significantly different from pre-injury measurements on paired t-test for recurvatum angle under hyperextension stress (Pre-injury

mean: 3.88°, Post-injury mean: 20.58°, Mean difference: 16.7°, 95%CI: [0.513° - 32.9°], p = 0.0453. Frequencies of tears found on dissection in each individual anatomic structure are summarized in Table 2.

Table 1.

Limb ID	Torn Structures Post-Model	Lateral "gapping" (mm)	Δ Recurvatum Angle (°, positive is more hyperextension)
002	Posterior capsule, PFL	4.87	-2.00
003	Posterior capsule, PFL, LCL, LGT	8.10	20.4
005	Posterior capsule	5.90	9.10
006	Posterior capsule, PFL, LCL, LGT, BFT	10.6	44.2
009	Posterior capsule, PFL, LCL	1.60	14.5
010	Posterior capsule, PFL, LCL	3.70	14.0
	Mean (95%CI)	5.80 (2.44 – 9.16)	16.7 (0.513 – 32.9)

a. popliteus tendon (PLT), popliteofibular ligament (PFL), lateral collateral ligament (LCL), lateral gastrocnemius tendon (LGT), and biceps femoris tendon (BFT)

Table 2.

	No. of Tears	Damage Rate (6 Total Specimens)
Posterior Capsule	6	100%
Popliteofibular ligament	5	83%
Lateral collateral ligament	4	67%
Lateral gastrocnemius tendon	2	33%
Biceps Femoris Tendon	1	17%
Popliteus tendon	0	0%
Common Peroneal Nerve	0	0%

Discussion/Conclusion

Our proposed injury model successfully induced a posterolateral corner injury in six cadaveric specimens, and as hypothesized, these injuries correlated to increases in radiographic measurements of lateral gapping of the knee joint on varus stress and recurvatum angle of the knee on hyperextension. We were also correct in our hypothesis that specimens with the most structural damage following injury mechanism would show the greatest increases in these radiographic measurements. As seen in Table 1, Specimen 003 and Specimen 006 had the greatest increases in lateral gapping and recurvatum angle and were the only two specimens with posterior capsule, PFL, LCL, and lateral gastrocnemius tendon injuries, with Specimen 006 showing greater increases in these parameters, potentially due to the addition of a biceps femoris tendon injury.

Posterolateral corner injury to the knee remains a difficult topic to study and diagnose, due to its relatively rare incidence and significant heterogeneity in injury patterns. Even in our cadaveric varus-hyperextension model in a controlled laboratory setting, a variety of anatomic structures were injured, which illustrates the multiple factors that contribute to posterolateral corner stability. Interestingly, our model showed that 100% of injured specimens had capsular tears, as evidenced by a capsular load test. Similarly, LaPrade et. al. showed that 75% of patients with clinical posterolateral instability had tears of the capsulo-osseous layer of the iliotibial band and 58% had other capsular injury.[5] A high prevalence of capsular injury both clinically and in our model illustrates the important contribution of the capsule to posterolateral knee stability. One of our specimens illustrated this point particularly well, as the posterior capsule was the only torn structure, yet the knee was found to have lateral gapping of 5.9 mm. This is in contradiction to previous literature which has shown that lateral gapping greater than 4.0 mm is predictive of

complete disruption of the LCL and PFL.[5 6 7] These previous studies did not evaluate capsular contribution to stability, which likely explains this discrepancy.

The 100% tear rate of the posterior capsule in our varus-hyperextension model provides insight into high energy multi-ligamentous knee injuries. As our model involves only posterolateral translational forces, it illustrates that the posterior capsule is key to the prevention of posterolateral translation of the tibia when the knee is in extension. It has been well documented that the posterior cruciate ligament is a key stabilizer to this motion in flexion.[2 3] Our model highlights the posterior capsule contribution to posterior sagittal stability as well. It is reasonable to conclude, based on our results, that the posterior capsule is the first structure to fail, leading to progression of PLC injury from deep to superficial.

Limitations of this study are certainly present. First, our varus-hyperextension model only successfully induced PLC injury in the absence of fracture in 50% of specimens. The high rate of tibial plateau fracture from the model (33%) is likely the result of anatomic variation between specimens. Specimens that suffered tibial plateau fracture had smaller pre-injury recurvatum angles compared to those that suffered purely soft tissue injury (-0.3° vs 4.9°). We hypothesize that flexion contractures present in specimens before injury contributed to a specimen being more susceptible to tibial plateau fracture. We recommend that any reproduction of these methods uses cadavers without flexion contractures to maximize the efficiency of the model. Another limitation was that two out of twelve specimens were found to have no structural damage upon dissection, suggesting that PLC injury can be difficult to diagnose on physical exam or gross observations alone. This should motivate future studies to improve clinical diagnostic techniques. Additionally, our mechanism models only translational force through the knee joint, whereas typical PLC injuries involve complex forces in three dimensions, including

rotational forces. This model is the only cadaveric model of PLC injury described to date, but it is a simplified illustration of a dynamic, complex system. Finally, our results are drawn from a small sample size, partially due to the availability of donors and other resources, and partially due to the issues encountered with our model. With the proposed optimizations in place, repeating this protocol with a larger sample size may help to uncover more definitive correlations between structural damage and radiographic measurements in PLC injuries.

Future research can continue to improve our cadaveric varus-hyperextension model, possibly finding a more dynamic way to load the joint, or a more consistent model to use that will avoid the issues of fractures and uninjured limbs. Avoiding the use of limbs with flexion contractures, as discussed earlier, will be a good strategy to improve this model. Regarding future *in vivo* research, we of course cannot obtain pre-injury radiographic measurements for all patients who present with PLC injuries. However, we have seen that the accepted value of 4.0 mm of lateral gapping indicating severe PLC injury may not be as accurate as once thought. Compiling more data with each PLC injury that presents clinically may help to describe or discover patterns that can assist clinicians with accurate diagnosis and improve patient outcomes.

In conclusion, a novel *in vitro* varus-hyperextension model is described which can successfully induce posterolateral corner injury. The other purpose of this research was to describe a possible correlation between damaged structures and physical exam and radiographic findings, which was accomplished within the limitations of our small sample size. The model successfully induced PLC injury without fracture in 50% of specimens, and the posterior capsule was always the first structure to be damaged, with additional structural injury correlating to larger changes in instability measurements on stress radiographs.

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