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Lariat sign: An MRI finding associated with common peroneal nerve rupture

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ABSTRACT

Traumatic knee dislocation represents a catastrophic orthopedic injury with potentially devastating vascular and neurologic injuries. We report a case of common peroneal nerve rupture sustained during a knee dislocation with novel radiographic findings that we describe as a lariat sign. At the site of rupture, the distal nerve loops back on itself forming a lasso shape or lariat. This thickened nerve's abnormal course should not be misinterpreted as a vessel.

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Introduction

Frequently referred to as the “dark corner” of the knee, the posterolateral corner (PLC) consists of the lateral collateral ligament, popliteus tendon complex, popliteofibular ligament, and the posterolateral capsule [1,2]. The PLC provides resistance to varus stress and external rotation at the knee [1]. Laterally based peri-articular knee structures at risk of concomitant injury in the setting of PLC injury also include the biceps femoris tendon, lateral gastrocnemius tendon, iliotibial band, fibular head (and associate arcuate ligament), and the common peroneal nerve (CPN) [2]. PLC injuries may result in severe disability, persistent knee instability, and future articular cartilage degeneration. A further debilitating sequela of multiligamentous knee injury (MLKI) with PLC disruption includes CPN injury, which occurs in 44% of cases with PLC injury and biceps femoris tendon rupture or avulsion of the fibular head [3]. Approximately half

of these cases cannot functionally recover [3]. Previously unreported, however, is the occurrence of traumatic neurotmesis of the CPN, illustrated in this case by magnetic resonance imaging (MRI) findings of the CPN looped up and over the fibular head. We describe these findings as a “lariat” sign.

Case presentation

The patient, a 37-year-old male laborer, sustained a closed left knee dislocation while participating in the rodeo sport of steer wrestling. He was taken immediately to a community hospital, where he was evaluated for a limb-threatening vascular injury and found to have none. X-rays taken at the community hospital were negative for acute bone injury (Fig. 1A and B). Initial treatment consisted of external knee stabilization using a knee immobilizer. The patient presented to outpatient

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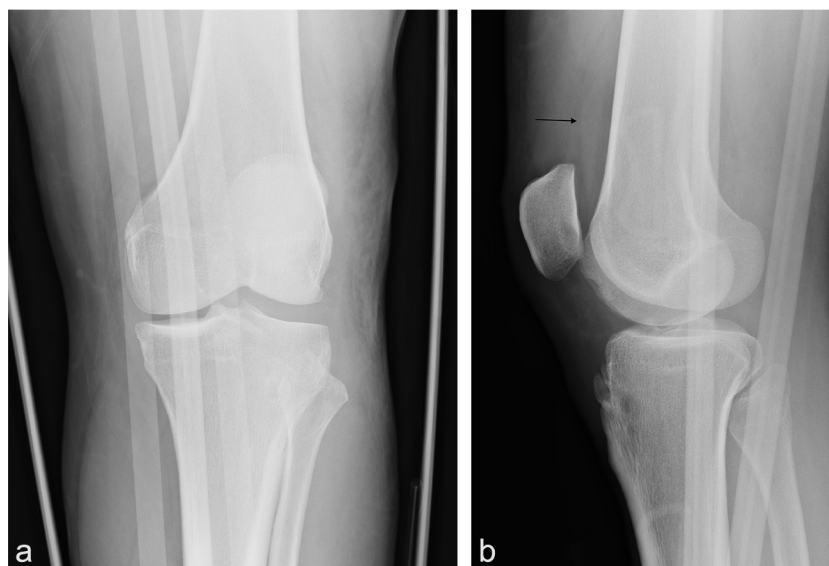


Fig. 1 – (A) Portable anterior-posterior radiograph of the left knee. External knee immobilizer splint on knee, with subcutaneous edema along the lateral knee. Otherwise unremarkable examination. (B) Portable lateral left knee radiograph. External knee immobilizer splint in place on the knee. No acute fracture. Osseous alignment normal with maintained knee joint spaces. Small knee joint effusion in the suprapatellar recess (arrow).

Orthopedic Sports Medicine clinic approximately 2 weeks after his injury. At the time of his clinic evaluation, the knee had a moderate effusion with posterolateral knee tenderness. Passive range of motion was 0° extension to 95°. The patient exhibited a positive Lachman test. Varus stress testing demonstrated grade 2 laxity at full extension and grade 3 varus at 30° of knee flexion. Valgus stress testing exhibited stability at full extension and grade 1 valgus at 30° of knee flexion. The Dial test revealed asymmetry at 30° and 90° of knee flexion compared to the uninjured limb.

On examination of his injured lower extremity motor function, the patient could not dorsiflex his left ankle or hallux, and the manual muscle test grade of the tibialis anterior muscle and extensor hallucis longus muscle was 0, indicating a drop foot. In addition, sensory function of the CPN area was absent.

MRI showed complete anterior cruciate ligament rupture. Evaluation of the PLC included injury to the lateral collateral ligament, popliteofibular ligament, popliteus tendon, distal rupture of the biceps femoris, and rupture of the CPN (Fig. 2A-C)

Examination under anesthesia revealed passive range of motion 0° extension to 130° flexion, grade 1 laxity with valgus stress at 30°, stable in full extension, grade 2 laxity with varus stress in full extension, and grade 3 laxity with varus stress in 30° of knee flexion. A positive Lachman grade 2b and grade 3 pivot shifts were noted, as were a negative posterior drawer and a positive posterolateral rotatory drawer.

The patient underwent allograft reconstruction of his anterior cruciate ligament, lateral collateral ligament, PLC allograft reconstruction, peroneal neurolysis, partial lateral meniscectomy, microfracture of the medial femoral condyle, and suture repair of the biceps femoris. The peroneal nerve was identified looped up and over the fibular head and was in discontinuity (Fig. 3). The nerve defect was measured at 8 cm and a segmental CPN reconstruction was performed in the second surgical procedure.

Discussion

MLKIs occurring in conjunction with knee dislocation represent a rare but severe orthopedic injury. CPN injury occurs in 16%-40% of patients with knee dislocation and prognosis for nerve recovery associated with MLKIs depends on the extent of disruption of the normal neural anatomy [2,4]. In our case, the patient experienced a complete CPN axonal disruption.

The CPN lies in close proximity to the PLC of the knee joint and the proximal fibula. This location places the nerve at risk of injury during varus stress, direct local trauma, and knee dislocation [2,5,6]. In the distal one-third of the thigh, the sciatic nerve bifurcates into the CPN and the tibial nerve. As the CPN exits the popliteal fossa posterior to the conjoined biceps femoris tendon and lateral head of the gastrocnemius muscle, the nerve courses distally and superficially, covered by only subcutaneous tissue and skin, before traveling lateral to the proximal fibula [2]. Vascular supply to the CPN arises from an unnamed branch of the popliteal artery within the proximal popliteal fossa. However, at the level of the knee joint, the vascular supply relies on a small vasa nervorum derived from the anterior recurrent tibial artery [7]. The tibial nerve, however, remains protected within the popliteal fossa as it courses the posterior knee and enters the deep posterior compartment of the lower leg and receives predictable vascular contributions from the popliteal and the posterior tibial arteries. As compared to the CPN, the tibial nerve is less likely to be injured during knee dislocation, which is thought to be due to the nerve's protected location and more consistent blood supply [8,9].

The superficial peroneal nerve innervates the peroneus longus and brevis muscles, which function primarily to plantarflex the first ray and evert the ankle, respectively [2].

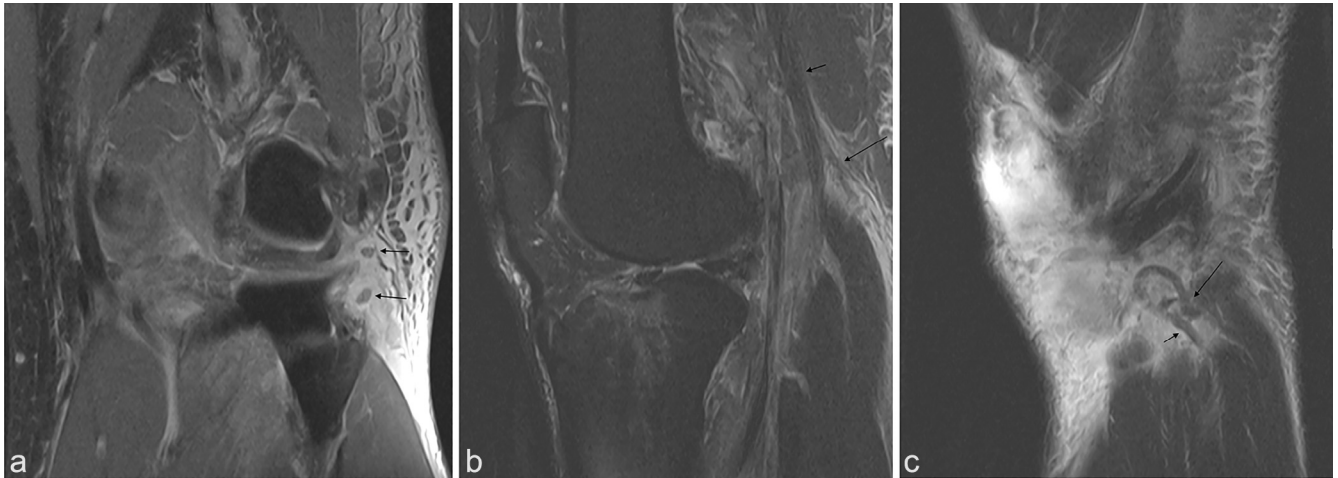


Fig. 2 – (A) Coronal left knee proton density fast spin echo fat saturation (TR: 2280; TE: 12; ST: 3; Spacing: 3.3; Siemens; Matrix: 320/0/0/240). Coronal image at the level of the fibular head including the posterior lateral femoral condyle. Marked soft tissue swelling and edema in the lateral aspect of the left knee. A redundant portion of the ruptured common peroneal nerve (distal portion) is seen in the soft tissues superior to the fibular head (arrows). **(B) Sagittal T2 turbo spin echo fat saturation (Siemens; TR: 5691.7; TE: 79; ST: 4; Spacing: 4.4; Matrix: 320/0/0/224; pixel: 0.25/0.25 mm)** Sagittal slice of the left knee just medial to the intercondylar notch. Avulsed proximal stump of the common peroneal nerve (long arrow) just distal to the branch point from the sciatic nerve (short arrow). Soft tissue edema surrounding the avulsed nerve. **(C) Sagittal T2 turbo spin echo fat saturation (Siemens; TR: 5691.7; TE: 79; ST: 4; Spacing: 4.4; Matrix: 320/0/0/224; pixel: 0.25/0.25 mm).** Sagittal slice of the left knee lateral to the fibular head. Ruptured and retracted common peroneal nerve is seen within the soft tissues of the lateral knee. The proximal segment (long arrow) of the nerve is looped back to the intact distal segment (short arrow), resulting in a “lariat” shape. TE, echo time (milliseconds); TR, repetition time (milliseconds).

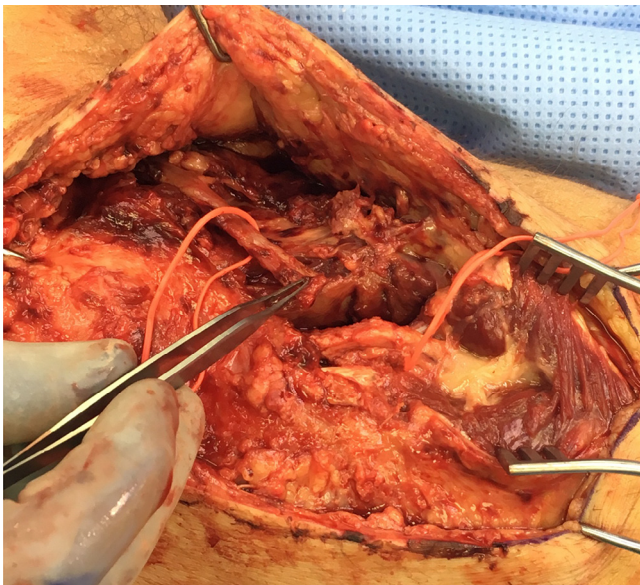


Fig. 3 – Intra-operative image of common peroneal nerve disruption; vessel loops mark proximal and distal ends of the common peroneal nerve with surgical forceps grasping the proximal edge of the nerve discontinuity. Photo provided by Jacob W. Brubacher, MD.

The deep peroneal nerve innervates 4 extrinsic muscles to dorsiflex the foot and extend the toes [2]. Injury to the CPN may result in a motor palsy involving these muscle groups. Clinical presentation involves foot drop or loss of ankle dorsiflexion, as well as a relatively unsteady gait. Additionally, terminal branches of the peroneal nerve provide sensory innervation for the dorsal foot and the first web space, and knee dislocation patients may experience sensory disturbances in these distributions.

Two nerve injury classification systems are applicable to the diagnosis and management of CPN injuries. Seddon stratified peripheral nerve injuries into 3 classes: neurapraxia (mild), axonotmesis (moderate), and neurotmesis (severe), whereas Sunderland modified that system to account for the variable outcomes of axonotmetic injuries [10,11]. MRI assists in evaluating potential ligamentous injury, detecting neurologic injury, and determining the soft-tissue response to injury. MRI allows confirmation of CPN injury and localizes the site of nerve injury. Nerve injury type may be characterized by determining surrounding fat planes, localized edema, presence of contusion, nerve fiber disruption, and encasing hematoma [12,13]. Magnetic resonance neurography involves the use of short tau inversion recovery sequences to image peripheral nerves directly and may have the potential to accurately detect the early extent of nerve lesions and monitor nerve regeneration [2,14].

MRI additionally assists in preoperative planning by identifying nerve injury requiring surgical repair or reconstruction. Early recognition of discontinuous nerve injury provides impetus for timely surgical intervention and aids surgical dissection by helping the surgeon understand the course of the injured nerve before the patient arrives in the operative suite. Measuring nerve defect before surgical intervention aids planning by ensuring appropriate allograft reconstruction options are available for the procedure.

Few evidence-based guidelines exist to navigate the management of MLKIs with concomitant nerve injury. High-grade nerve injury portends a poor likelihood of patient nerve recovery despite surgical intervention. MRI adds important clinical information, assisting with preoperative planning and surgical intervention. In the setting of traumatic knee injury or dislocation, particularly involving the PLC, vigilant scrutiny of the CPN for involvement or rupture (lariat sign) is necessary to allow prompt diagnosis and appropriate patient management.

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