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The Sequelae of Drop Foot After Knee Dislocation: Evaluation and Treatment

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Although knee dislocations are relatively rare, serious complications make treatment difficult. Common peroneal nerve (CPN) palsy is a debilitating complication with an incidence reported as high as 50%. Even after successful ligament reconstruction or repair, unresolved CPN palsy is a major factor contributing to poor outcomes. CPN palsy is more common with open dislocations, rotatory dislocations, and in patients with posterolateral corner injuries. CPN palsy can be readily diagnosed clinically, although a high index of suspicion is needed. Conservative management can be appropriate in the early phase of treatment; however, for persistent nerve damage, surgery is the treatment of choice because it results in better functional outcomes. The benefits of surgical exploration in the acute setting are controversial. Neurolysis, primary nerve repair, nerve grafting, and posterior tibialis tendon transfer have all been used as surgical options. As late surgical treatment of CPN typically results in poor prognosis, prompt diagnosis and close follow-up are of paramount importance.
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Introduction

Multiligament knee injury due to knee dislocations is a complex injury often leading to concomitant neurovascular injury. The common peroneal nerve (CPN) is a common injury seen with knee dislocations. The CPN is vulnerable to injury due to its anatomical location and firm attachment to surrounding soft tissue structures about the lateral side of the knee.[1,2] The reported incidence of injury to the CPN during knee dislocation varies between 4% and 50%.[3–12] Owens et al[13] concluded that CPN injury was found in 75% of patients with knee dislocations. Multiple studies have demonstrated that CPN injuries are more prevalent with high-energy injury mechanisms (eg, motor vehicle or industrial accidents), open dislocations, and knee dislocations associated with posterior cruciate ligament and posterolateral corner (PLC) injuries, increased body mass index, and fibula fractures.[1,10,14,13,15,16] CPN injuries may range from incomplete nerve palsy, which often leads to paresthesias, to complete nerve palsy, causing motor weakness in dorsiflexion of the ankle and toes as well as foot eversion. This motor deficit often causes significant disturbances in the gait pattern, leading to the need for an orthotic or surgical intervention.

Long-term outcome studies suggest that half of patients with CPN injuries recover spontaneously.[17] Patients with injuries to multiple ligaments and persistent CPN palsy have worse functional outcomes.[16,18] Surgical treatment is required for cases with irreversible nerve injury or persistent functional deficits. Controversy exists regarding the timing and type of surgical intervention. This article discusses the anatomy, pathophysiology, and evaluation and treatment options for CPN injuries.

Anatomy

The CPN is a division of the sciatic nerve and is composed of nerve roots L4-S3. The sciatic nerve courses through the posterior thigh and divides into the tibial nerve and CPNs just proximal to the popliteal fossa and deep to the biceps femoris. The CPN continues to track distally to the PLC deep to biceps

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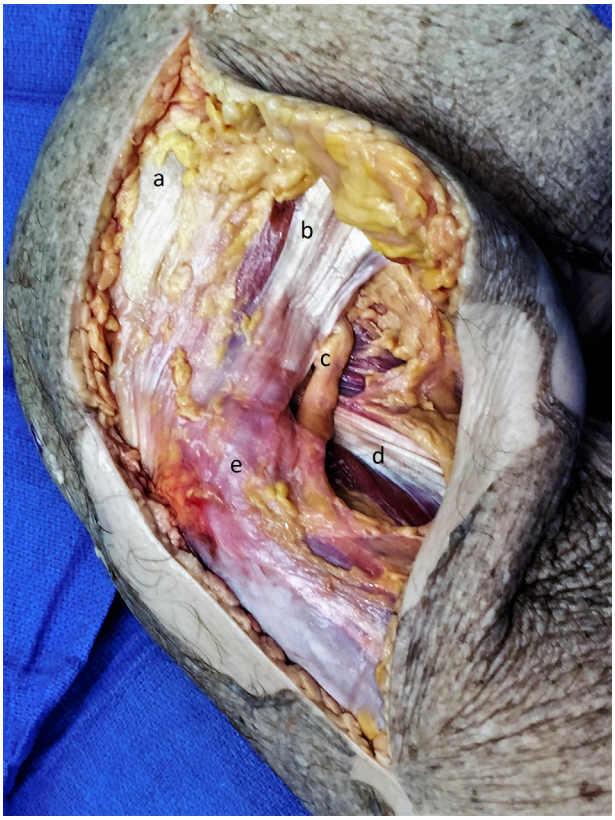

Figure 1 Anatomy of the lateral aspect of the knee. (A) Iliotibial band, (B) biceps femoris, (C) common peroneal nerve, (D) lateral head of the gastrocnemius, and (E) location of the head of the fibula.

tendon as the nerve makes its course to the lateral compartment of the leg[18] (Fig. 1). The CPN takes a turn to curve around the neck of the fibula, where it lies directly over fibular periosteum for approximately 6 cm. At this level, the nerve is protected solely by subcutaneous tissue and skin.[19]

The CPN typically divides into 3 branches. The first branch, the lateral articular nerve, innervates the inferolateral portion of the knee joint capsule and the lateral collateral ligament. Between the peroneus longus muscle belly and the proximal fibula, the nerve divides into the 2 main branches: the superficial and deep peroneal nerves. The superficial branch passes though a tunnel formed by the origin of the peroneus longus muscle and the intermuscular septum. It travels between and innervates the peroneus longus and brevis muscle, which act to evert the foot. The superficial branch also provides sensory innervation to the anterolateral aspect calf and the dorsum of the foot.

The deep peroneal nerve passes through a second fibroosseous tunnel formed by the origin of the extensor digitorum longus muscle, approximately 4 cm distal to the peroneal muscle tunnel. The deep peroneal nerve innervates the muscles of the anterior compartment of the leg: tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The tibialis anterior is the main dorsiflexor of the ankle joint. Within the foot, the deep peroneal nerve innervates the intrinsic toe extensors, the extensor digitorum brevis, and the extensor hallucis brevis. The deep branch provides sensory innervation to the first web space.[20]

Injury Mechanism and Pathoanatomy

Knee dislocations are classified by energy level of the injury or according to the anatomical location.[21] Kennedy[4] described knee dislocations as anterior, posterior, medial, lateral, or rotatory, which refers to the position of the proximal tibia in relation to the distal femur. Traction injuries to the CPN are associated with anterior and anteromedial dislocations, which result from stress varus and hyperextension forces. These stretch injuries are attributed to the firm periosteal attachment of the CPN in the region of the fibular neck. Traction mechanism injuries can range from mild stretch to complete rupture of the nerve. The posterior dislocation pattern has a higher rate incidence of injury to the neurovascular structures around the knee. According to some studies CPN injuries are 100% correlated with concomitant PLC injuries following posterior knee dislocations.[20] Among all dislocations, posterolateral mechanism is most likely to cause permanent peroneal nerve injury (Chart 1).[3,22–24]

It is important to recognize the energy and mechanism associated with knee dislocation that occurred. High-velocity mechanisms such as motor vehicle accidents, pedestrian vs motor vehicle, motorcycle accidents, and falls from a height, are more likely to have associated neurovascular injuries. An ipsilateral popliteal artery injury has a 44% association with CPN palsies.[25] Peroneal nerve injuries in traumatic knee dislocation are also common in sports injuries.[26,27] A knee dislocation with a concomitant fibula head fracture is highly indicative of a PLC injury and therefore an injury to the CPN. Additionally, patients with morbid obesity may sustain knee dislocations during daily activities, which may result in CPN palsy after these ultralow energy knee injuries.[25,27,28]

Mechanisms of nerve injury include laceration, compression, traction, and focal ischemia.[29] An injury causing elongation 15% or greater than the length of the nerve can cause disruption to both the intraneural and extraneural microvasculature. This may result in a complete failure of its blood supply.[30] Tomaino et al[31] demonstrated that a stretch injury mechanism may result in a longer overall zone of injury compared with a complete rupture. The length of trauma to an intact CPN is predictive of functional recovery.[8]

Stretch injuries may also rupture the vaso nervorum, the nutrient vessels of the nerve. Damage to these vessels may result in ischemic changes from a compressive hematoma. This bleeding causes a gradual expanding hematoma, which delays the presentation of the nerve palsy. CPN function may be normal immediately after the injury, but it then regresses over 24-48 hours. As symptoms of paresthesia and motor weakness develop, immediate surgical intervention is indicated. For these delayed presentations, surgical release likely provides immediate relief, and possibly full recovery.[32]

The CPN is more susceptible to injury during knee dislocations than other neurologic structures are for several anatomical and biologic reasons. These include the 4-6 cm-long subcutaneous course around the neck of the fibula, where the tethered anatomy of the deep and superficial branches