Peroneal Nerve Compression Secondary to an Anomalous Biceps Femoris Muscle in an Adolescent Athlete

Kevin M. Kaplan, MD, Abhay Patel, MD, and Drew A. Stein, MD

Abstract

Common peroneal nerve compression is a well-recognized entity that can cause severe debilitating clinical manifestations. The current literature describes numerous locations and mechanisms of compression, including both structural and systemic causes. Anatomical variants should be considered part of the differential diagnosis in peroneal nerve impingement. We present the case of a 14-year-old basketball player with footdrop secondary to compression of the common peroneal nerve from an accessory biceps femoris muscle, which was treated by neurolysis. In addition, we review the systematic workup of patients with nerve compression.

The common peroneal nerve is a peripheral nerve in the lower extremity susceptible to entrapment secondary to its tortuous path and relationship to the fibular head.[1,2] Determining the etiology and exact anatomical site of nerve compression can present a considerable challenge. Other conditions must be excluded in order to make the proper diagnosis.[3] Symptoms can occur after exercise, can develop gradually after a period of training, or can have an insidious onset.

We present the case of a 14-year-old basketball player who developed a compressive neuropathy of the common peroneal nerve secondary to an accessory biceps femoris muscle.

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> "...the diagnosis of an accessory biceps femoris muscle should be part of the differential diagnosis for footdrop."

Case Report

A 14-year-old basketball player presented with an 8-month history of increasing right foot weakness and numbness associated with dull leg pain. Over the course of several months, symptoms progressed, precluded him from participating in athletics, and eventually led to difficulty ambulating. The majority of numbness was localized to the dorsum of the foot, and the patient was unable to actively dorsiflex the ankle. He denied any history of trauma and medical problems and any recent weight loss, fevers, rashes, other illnesses, back pain, and bladder and bowel disturbances.

On physical examination, the patient ambulated with a right lower extremity steppage gait. Ankle dorsiflexion and extensor hallucis longus strengths were 2/5, and he was unable to stand on the right heel. Ankle eversion strength was 3/5, with all other muscle groups 5/5. Sensation was significantly decreased at the first dorsal web space and the dorsal lateral foot. Pulses were intact, and all reflexes were 2+ with negative clonus and Babinski reflexes bilaterally. The patient had no lumbar tenderness and had a negative straight leg raise bilaterally.

On the basis of the clinical history and physical examination, the patient was diagnosed with right lower extremity footdrop with weakness and decreased sensation in the distribution of the common peroneal nerve. X-rays of the knee and lumbar spine were negative. Lumbar spine magnetic resonance imaging (MRI), performed to exclude spine pathology, showed no spinal stenosis, nerve root impingement, or intraspinal pathology. Electromyography (EMG) and a nerve conduction study subsequently indicated severe right common peroneal nerve dysfunction. No response was elicited 0.5 cm proximal to the fibular head; however, there were normal distal latency, conduction velocity, and only mildly diminished amplitude below the level of the fibular head.

Right knee MRI, used to evaluate for etiologies of common peroneal nerve compression, showed that the biceps femoris muscle was prominent

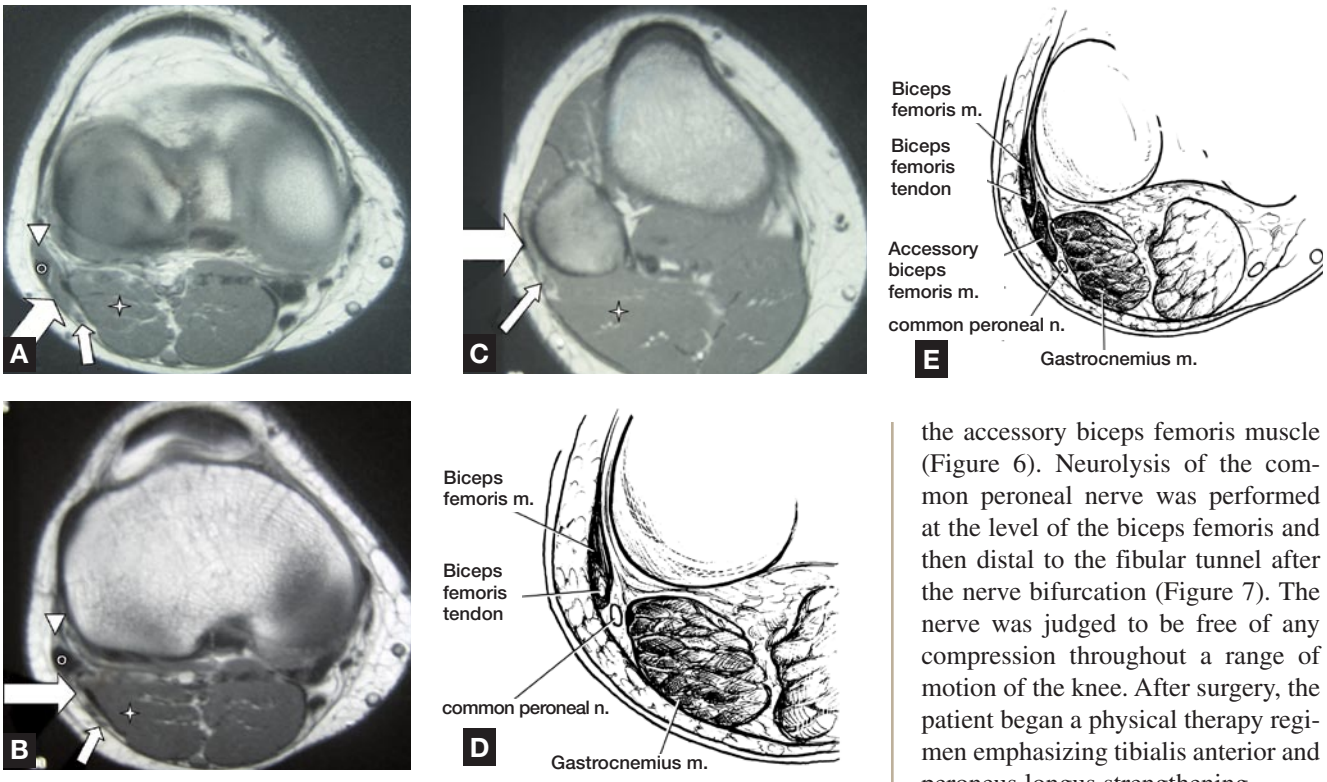

Figure 1. Axial magnetic resonance imaging shows a prominent biceps femoris muscle: (A,B) Large white arrow points to an anomalous accessory biceps femoris posterior to the tendon. As the biceps femoris normally approaches the level of the fibula, no muscle should be located posteriorly to the tendon. (C) More distal cut on which the nerve should appear traversing anterior to the fibula but remains posterior secondary to the accessory muscle. Legend for Parts A-C: small white arrow points to compression of the common peroneal nerve; arrowhead indicates the true biceps muscle (white circle indicates the biceps tendon); and asterisk marks the lateral head of the gastrocnemius. (D,E) Normal cross-section and cross-section with an accessory biceps femoris, respectively. Illustrations by NYU Hospital for Joint Diseases Graphics Department.

close to its fibular insertion and contained an accessory muscle (Figure 1). The peroneal tendons demonstrated increased uptake secondary to edema, which is the result of nerve compression (Figure 2). The signal intensity, which may be related to loss of vasomotion and vascular responses to vasoactive substances, occurred secondary to muscle denervation. The common peroneal nerve was compressed because of its anomalous course around the accessory biceps femoris and was not allowed to assume its normal anatomical position in relation to the fibula until distally in the leg (Figure 1). On the basis of MRI and clinical findings, common peroneal nerve release and neurolysis were indicated. Before surgery, the patient was placed in a custom-made ankle-foot orthosis.

In the operating room, the patient received general anesthesia and was placed in the lateral decubitus position. Under tourniquet, a 6-cm incision was made over the fibular head along the course of the biceps femoris muscle (Figure 3). A blunt dissection technique was used to identify and then incise the fascia of the biceps femoris, after which the common peroneal nerve was identified (Figure 4). Subsequently, the accessory biceps femoris muscle was identified close to its distal insertion at the fibular head (Figure 5). The common peroneal nerve was followed distally until its bifurcation and was noted to be compressed by the accessory biceps femoris muscle (Figure 6). Neurolysis of the common peroneal nerve was performed at the level of the biceps femoris and then distal to the fibular tunnel after the nerve bifurcation (Figure 7). The nerve was judged to be free of any compression throughout a range of motion of the knee. After surgery, the patient began a physical therapy regimen emphasizing tibialis anterior and peroneus longus strengthening.

During the first 2 weeks after surgery, the patient's symptoms improved significantly. By 2 months after surgery, the patient had regained full ankle dorsiflexion and ankle eversion strength compared with the contralateral side. He was able to stand on his heel and perform a single-leg stand without any difficulty. He also displayed a normal heel–toe gait pattern. Furthermore, the paresthesias and complaints of leg pain resolved. A second EMG nerve conduction study showed substantial improvement in nerve conduction and only minimal neurogenic dysfunction of the common peroneal nerve at the level of the fibular head. The distal latency and amplitude returned to normal, and only a mildly delayed conduction velocity across the fibular head was observed. With the significant improvement in the EMG nerve conduction studies and an essentially normal neurologic examination, the patient was allowed to discontinue physical therapy and return to sports as tolerated. At 1-year follow-up, the patient had no complaints and a normal physical examination and had returned to previous levels of athletic activity.

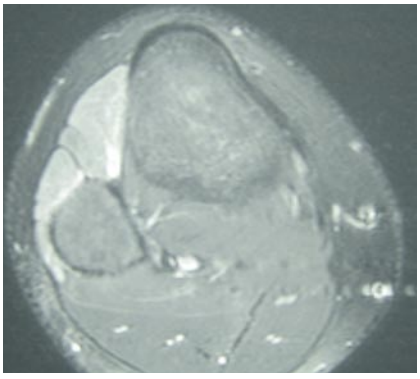

Figure 2. Axial magnetic resonance imaging shows increased uptake in the peroneal muscles consistent with compression of the common peroneal nerve.

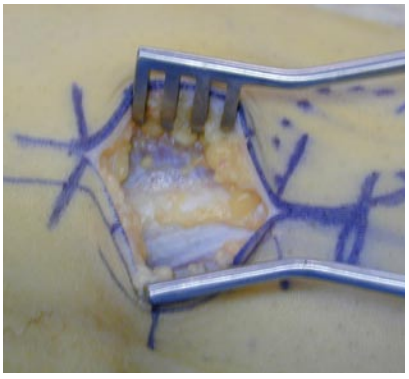

Figure 4. Incision with underlying fascia and identification of common peroneal nerve.

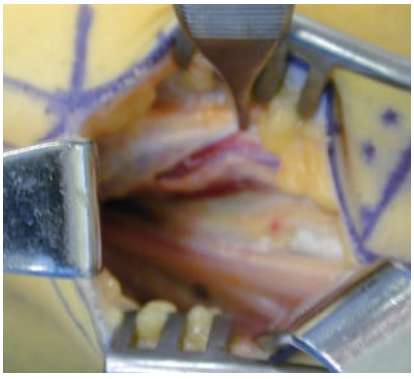

Figure 6. Release of compression at site of accessory biceps femoris muscle.

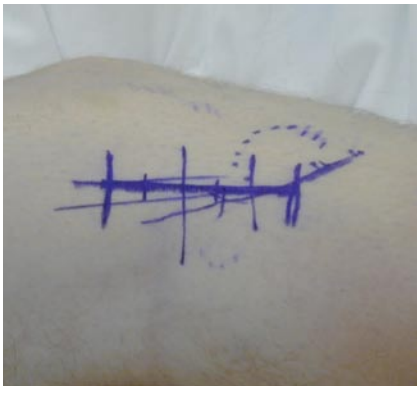

Figure 3. Marking of incision.

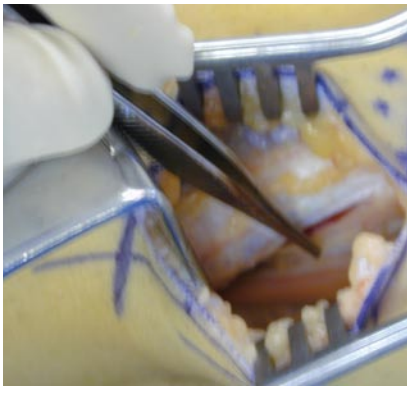

Figure 5. Identification of accessory biceps femoris muscle at end of forceps.

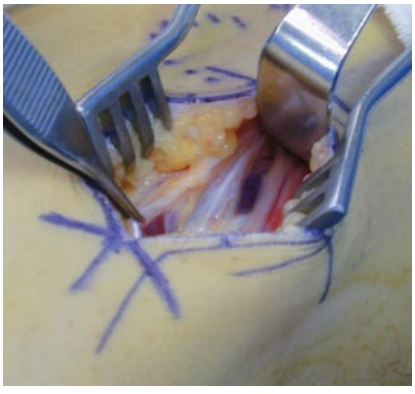

Figure 7. Visualization of bifurcation of the common peroneal nerve.

Discussion

The common peroneal nerve originates as a branch of the sciatic nerve and has contributions from nerve roots L4 to S2. As it travels down the thigh, the nerve supplies the short head of the biceps femoris along its medial side and then crosses, posterior to lateral, superficial to the head of the gastrocnemius to become subcutaneous behind the head of the fibula. The nerve then pierces the posterior intermuscular septum and ultimately splits into superficial and deep components.

Nerve entrapment as classified by Lundborg and Dahlin[4] has 3 stages. In stage I, patients have rest pain and paresthesias that become worse at night. Stage II, as seen in our patient, is characterized by continuous nerve compression that causes increasing numbness, intermittent weakness, and paresthesias. Stage III presents with constant pain, evidence of muscular atrophy, and irreversible sensory loss.

Compression or entrapment can occur in various locations, but certain anatomical sites are commonly associated with clinical symptoms. As defined by Hirose and McGarvey,[5] entrapment occurs in areas of unyielding compression. There may be external and internal forces working to create such compression. The differential diagnosis should include direct compression from a space-occupying lesion, any systemic process that could increase total body fluid, metabolic conditions, and inflammatory diseases. In addition, other causes of pain in this nerve distribution, such as lumbar radiculopathy, must be ruled out before any surgical intervention. Nerve compression secondary to a compartment syndrome must also be in the differential diagnosis, noting that neurologic changes from a compartment syndrome are typically late in the clinical course.

Several causes of common peroneal compression have been described in the literature. Traumatic peroneal palsy can occur secondary to supracondylar fracture, knee dislocations, and proximal tibiofibular fractures. Causes of atraumatic nerve palsy include presence of ganglion cysts at the level of the fibular head and a large fabella, which could compress the nerve.[6,7] Mechanical irritation of the nerve can also arise from instability of the proximal tibiofibular joint or even from pretibial myxedema in untreated hyperthyroidism.[8-10]

Gloobe and Chain[11] described the fibular fibrous arch as a prominent

anatomical site for common peroneal nerve compression. The fibrous arch consists of a deep portion formed by the deep aponeurosis of the peroneus longus muscle and a superficial portion attaching from the soleus to the aponeurosis of the peroneus longus.[12,13] Fabre and Piton[14] recommended that both the superficial and deep portions of this fibrous arch must be released in order to completely relieve compression of the common peroneal nerve. Leach and Purnell[15] described increased intracompartmental pressure, caused by peroneus longus muscle swelling, as being responsible for common peroneal nerve entrapment in long-distance runners. This is analogous to an exertional compartment syndrome isolated to the lateral compartment.

Nerve compression can also be characterized as dynamic if symptoms occur after repeated flexion and extension of the knee during sporting activities.[8,15] Common peroneal nerve compression has been attributed to dynamic compression within the fibrous arch by the well-developed surrounding muscles.[14] Hypertrophy of the accessory biceps femoris muscle may contribute to this mechanism in a previously asymptomatic patient. Although numerous etiologies of common peroneal nerve compression have been described, we have found no reports of compression secondary to an accessory biceps femoris muscle. As young athletes are becoming more competitive and developing at an earlier age, the diagnosis of an accessory biceps femoris muscle should be part of the differential diagnosis for footdrop.

Conclusions

In terms of management, patients diagnosed with this anomaly may be sent for a trial of physical therapy, depending on symptom severity. If conservative measures fail, patients may need surgical management. Neurolysis may resolve compression on the nerve; however, complete release of the accessory muscle can be performed for further decompression without sacrificing function of the true biceps femoris muscle. It is important to recognize the multitude of etiologies of peripheral nerve entrapment, as not all will be treated surgically. Our patient had direct compression from an accessory biceps femoris muscle and had complete recovery after successful neurolysis.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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This paper will be judged for the Resident Writer's Award.