Musculoskeletal Dysfunction and Drop Foot: Diagnosis and Management Using Osteopathic Manipulative Medicine

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Drop foot can arise from various musculoskeletal or neurologic etiologic processes. The condition involves the muscles of foot dorsiflexion (tibialis anterior, extensor hallucis longus, and extensor digitorum longus) and the nerves that supply them, primarily the common peroneal nerve. Common causes of drop foot include compartment syndrome, diabetes, stroke, lumbar disc protrusions, musculoskeletal compression, myopathies, neuropathies, and peripheral nerve injuries.1-3

During the swing phase of heel-toe gait cycle, the muscles of dorsiflexion work as agonists and allow the foot to clear the ground. During heel strike of the stance phase, these muscles work as antagonists and control plantar flexion of the foot. Injury to the muscles of dorsiflexion or their nervous supply can cause drop foot and corresponding steppage gait—also known as drop-foot gait. In individuals with drop foot, the plantar flexors have no resistance and cause the foot to remain in plantar flexion during swing phase, therefore not allowing the toes to clear the ground and causing them to slap to the ground during heel strike. To avoid foot drag, an individual with steppage gait will walk with exaggerated hip and knee flexion to clear his or her affected foot from the ground during swing phase.4,5

As noted by Pritchett,6 “Peroneal neuropathy caused by compression at the fibular head is the most common compressive neuropathy in the lower extremity. Footdrop is its most notable symptom.” In the present report, we describe a patient who developed drop foot secondary to posterior fibular head dysfunction. However, unlike many published reports of drop foot,1,2,7 the condition was diagnosed and managed using osteopathic manipulative medicine.

Report of Case

A 47-year-old white man presented to the primary care office noticeably dragging his left foot. He stated that he could not lift up his left foot. He had no trauma to his left leg. However, he had driven 5 straight hours in his automatic transmission car the night before and stated that his left leg was bent for most of the car ride. On exiting the car, he noticed some numbness and tingling in his left leg. When he awoke the next morning, he was unable to raise his left foot.

The patient denied having any similar episodes in the past and denied any pain. He had not taken any pain medication for the numbness and tingling. Attempts at stretching his leg muscles gave no relief.

The patient had no history of diabetes or muscular, neurologic, or vascular problems. His medical history included hypertension and hypercholesterolemia, which were managed with lisinopril and atorvastatin calcium, respectively. The patient did not smoke tobacco and had no hypersensitivities.

Physical examination revealed a physically fit man with a pleasant disposition and stable vital signs (blood pressure, 124/78 mm Hg; body temperature, 98.2°F; heart rate, 67 beats per min; respiratory rate, 14 breaths per min). He ambulated with a steppage gait. Heart, lung, and abdomen examinations were unremarkable.

On musculoskeletal examination of the extremities, the patient had normal pulse on palpation (2+ pulses) bilaterally in the upper and lower extremities. Manual testing revealed 5/5 muscle strength throughout the right lower extremity and 1/5 muscle strength in the left lower extremity with ankle dorsiflexion and eversion. Sensation was absent to light touch.
and two-point discrimination along the dorsum of the left foot at the L5 and S1 dermatomes. Deep tendon reflexes were 1/4 at the Achilles and patellar tendons on the left lower extremity and 2/4 on the right lower extremity.

On osteopathic musculoskeletal examination, the patient had left posterior fibular head dysfunction with tenderness on palpation of the left common peroneal nerve. The patient had tight biceps femoris muscle with lateral hamstring Jones’ tender points, bilaterally. The patient also had hypertonic psoas major and minor muscles bilaterally.

The patient was diagnosed as having drop foot secondary to common peroneal nerve impingement from posterior fibular head dysfunction. With the patient in a prone position, osteopathic manipulative treatment (OMT) was applied using muscle energy for the psoas and hamstring muscles and deep articulation for the posterior fibular head.

While the fibular head was articulated, the patient noted discomfort but then stated that his foot was getting “warm and tingly.” Articulation was continued for about 1 minute. Total OMT lasted approximately 15 minutes. On re-evaluation, the patient had improved sensation along the dorsum of his foot and demonstrated left foot dorsiflexion with 4/5 motor function.

The patient was directed to use exercise band routines to strengthen the dorsiflexors and evertors of his left ankle. He was able to walk out of the office without steppage gait. The next day, he telephoned the office and stated that he had complete range of motion, strength, and sensation in his left foot.

Comment
Drop foot may occur with injury to the muscles of dorsiflexion or along the nervous pathway of these muscles. The sciatic nerve develops at the lumbrosacral plexus and travels through the greater sciatic foramen, located 80% of the time beneath the piriformis muscle, and continues along the posterior aspect of the thigh. It divides to form the common peroneal and tibial nerves above the popliteal fossa of the knee.

The common peroneal nerve travels laterally to wind over the posterior aspect of the fibular neck, dividing into the superficial and deep peroneal nerves. The superficial peroneal nerve supplies the peroneus longus and brevis muscles and provides sensation to the anterolateral aspect of the leg and the dorsum of foot. The deep peroneal nerve supplies the anterior tibial, extensor digitorum longus, and extensor hallucis longus muscles and supplies sensation to the web space between the first and second toes.

The common peroneal nerve runs a superficial course, close to the periosteum of the fibular neck for approximately 6 cm, covered by only skin and subcutaneous tissue, making it vulnerable to direct insult. For example, it could become compressed at the fibular neck after prolonged squatting. Although the patient in the present report was not squatting and denied leaning his left leg against the car door, we believe that the prolonged bent position of his left leg may have caused drop foot.

The fibula has reciprocal movements of the fibular head proximally and the stylus of the fibula distally. When the fibular head translates posteriorly, the stylus of the fibula translates anteriorly. This motion occurs when the foot is in plantar flexion. If the fibular head was maintained in a posterior position, then the ankle would be restricted in dorsiflexion. As described in the present report, the patient was unable to dorsiflex his foot, providing evidence that he had a posterior fibular head dysfunction.

The biceps femoris muscle has a long head, which originates from theischial tuberosity and sacrotuberal ligament, and a short head, which originates from the linea aspera and the lateral supracondyle of the femur. Insertion of this muscle is on the head of the fibula and the lateral condyle of the tibia. In normal functioning, the biceps femoris muscle provides flexion and lateral rotation of the leg at the knee. Hypertonicity of this muscle could place a posterior draw on the fibular head, causing the common peroneal nerve to be compressed, as described in our patient.

In the present report, muscle energy to the psoas and hamstring muscles and deep articulation to the fibular head resolved the patient’s symptoms. Therefore, the cause of this patient’s symptoms was likely secondary to his tight bicep femoris muscle, causing the fibular head to be pulled posteriorly. The posterior fibular head then caused compression of the common peroneal nerve, leading to drop foot.

Conclusion
In the present report, proper musculoskeletal examination identified areas of dysfunction, therefore avoiding the use of unnecessary diagnostic studies such as laboratory tests, radiography, magnetic resonance imaging, nerve conduction studies, and electrophysiologic tests. The patient had immediate relief of symptoms and normalization of his gait from a simple yet precise OMT technique without the need of more expensive treatment modalities, such as ankle foot orthosis, medications, or invasive treatments (eg, nerve root blocks, spinal decompression, tendon transposition). Osteopathic physicians with a strong knowledge of anatomy will be able to optimize osteopathic diagnosis and OMT, which can provide cost-effective patient care.

References


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