Title

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CLINICAL VIGNETTE

A Curious Case of Acquired Bilateral Common Peroneal Nerve Palsy

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Case

A 35-year-old male with a history of injection drug use (IDU) and chronic low back pain presented to the ED reporting bilateral lower extremity weakness and numbness. The deficits were bilateral, but greater on the left lower extremity. The patient reported his sensory deficit primarily in a boot distribution with sensory loss most prominent in his left foot. He has difficulty walking because of difficultly lifting his feet. His left foot slaps the ground when he tries to walk and he lifts his knees higher than usual to walk. The patient reports that his last IDU was months prior, but inhaled methamphetamine 2 days ago. He reports no trauma, fevers or chills, bowel incontinence, saddle anesthesia, or urinary retention. He has chronic back pain that feels like it is flaring up, but was not severe at ED presentation.

On exam, his vital signs were normal and the patient was alert and in no acute distress. His cardiopulmonary and back examinations were unremarkable. He had normal rectal tone without perianal numbness. His left lower extremity revealed 5/5 strength of hip flexors/extensors, knee flexion/extension, plantarflexion, and 4/5 dorsiflexion. Decreased sensation to pinprick was noted on left dorsal and plantar aspect of the left foot. His right lower extremity revealed similar findings of 5/5 strength of hip flexors/extensors, knee flexion/extension, plantarflexion, and 4/5 dorsiflexion. His sensation was intact in the right lower extremity aside from a mild decrease over the medial aspect of the right foot. His ambulation was limited due to left calf pain but revealed a slight left foot drop with steppage gait.

His labs were remarkable for a WBC 17, normal ESR. The remaining labs were unremarkable.

An MRI was ordered to evaluate for possible epidural abscess, diskitis, or osteomyelitis and revealed extensive areas of probable myelopathy involving multiple extensor, paravertebral and gluteus muscles. Focal T2 prolongation within the right paraspinal extensor muscles at L3-L5 represented a possible phlegmon or hematoma formation in the correct clinical setting. Other etiologies such as neoplasm were unlikely and there was no evidence of cord compression or cauda equina.

Neurosurgery evaluation confirmed no spinal cord issue with nonspecific findings on MRI suggestive of muscle inflammation with normal creatinine. He was hydrated with intravenous fluids during his ED observation period and discharged with return precautions.

The patient returned to the ED 2 days later reporting that symptoms in his right lower extremity resolved but he was not able to bear weight on his left leg due to continued weakness and was admitted for further evaluation. During his hospitalization the patient admitted that the day he developed symptoms, his roommate had found him sleeping cross-legged on the hardwood floor. The patient was examined by orthopedics and determined to have acquired bilateral common peroneal nerve palsies from sitting cross-legged for an unknown period of time.

Discussion

Common peroneal nerve palsies are caused by a variety of reasons including compression most common, transection, nerve ischemia/infarct, radiation-induced injury, inflammation, and degeneration.[1] The most common site of entrapment occurs at the fibular neck due to superficial nerve location, exit from the crural fascia, with increased vulnerability to injury.[2] Understanding the course of the peroneal nerve and its innervation helps narrow the differential and focus the physical examination. This case illustrates the importance of a comprehensive history and physical exam in the approach to the undifferentiated patient with mild back pain, bilateral foot drop, and no other signs of cauda equina or cord compression.

The common peroneal nerve starts from the posterior divisions of the L4-S2 nerve roots. It passes through the distal posterior thigh and continues posteriorly, inferior to the biceps femoris muscle and laterally to the head of the lateral gastrocnemius muscle prior to curving around the fibular neck and branching into the superficial peroneal nerve (SPN) and the deep peroneal nerve (DPN).[3] These branches innervate both motor and sensory aspects of the lateral and anterior aspects of the lower extremity.[3] Injury may occur from the sciatic nerve to the terminal branches with the most common presentation being partial or complete foot drop.[4,5] Foot drop can occur acutely or over days and weeks depending on the etiology of the disease.[5]

The clinical history should focus on obtaining a detailed account of precipitating factors such as time of onset, duration of symptoms, recent illness, direct trauma, or prolonged sitting or squatting. Careful review of past medical history should focus on the presence of active malignancies, chemotherapy,

radiation, or demyelinating diseases. Social history should include occupation. For example, Serkan et al showed how squatting in farmers who were not overweight was associated with unilateral and bilateral peroneal nerve palsies.[6]

Foot drop is the most common physical finding. Additionally, foot eversion and weakness in foot or toe dorsiflexion may suggest this pathology.[3] Assessing gait may elicit a 'foot slap' and high stepping. It's imperative to passively dorsiflex the foot in order to avoid the confounding variable of weakness in the tibialis posterior.[7] Patients usually have a normal hip range of motion and strength. It is important to perform hip flexion and abduction to unmask the peripheral nature of the disease.[1]

One clinical pearl, reported by Lezak et al, is elicitinh Tinel sign. The clinician taps along the pathway of the nerve and attempts to elicit paresthesias and tingling, especially around the fibular head, distal to the site of compression. A positive Tinel Sign can reassure the examiner of compression versus other central pathology.[3] Masakado et al reported some cases with complete absence of sensation of the anterior distal area of the leg and dorsum of the foot.[2] The loss of sensory function is more specific to the injury site being below the knee.[2]

Imaging helps rule out occult causes of injury or irritation of the peripheral nerve. Simple radiographs can rule out fractures and more detailed imaging such as MRI and CT can distinguish masses including tumor or cysts.[1,8] Ultrasound is an excellent modality to view the nerves of the lower extremity but requires more investigation.[9] Neuro-conductive studies including needle EMG can localize the site of damage but the role in the outcome of patient care is still to be determined.[2]

Treatment is mostly conservative care. A case study of 16 workers with peroneal nerve palsy from squatting fully recovered after 3-6 weeks of rest and close follow-up.[6] Surgical decompression is an option given that patients with severe motor and sensory impartment may not recover with conservative treatment alone.[9] Given the varied nature of pathology, it is recommended to consult orthopedics if conservative treatment fails.[10]

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