

Cellulitis May Present As Foot Drop In A Diabetic Patient

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Citation

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Abstract

We are presenting a diabetic patient who was admitted with right hemiparesis because of a stroke and after a few days in hospital developed left foot drop. After thorough investigation, it turned out to be a complication of a smoldering cellulitis around the left ankle.

A 62-year-old Afro-American lady was transferred from another hospital where she presented complaining of right sided weakness. The night before, she went to bed without any complaints. When she woke up in the morning, she had right upper and lower extremity weakness, right side of face weakness and slurred speech. Brain CT showed acute hemorrhage in the left basal ganglia. She was on anticoagulation and received fresh frozen plasma for reversal. After a few days in hospital and some improvement in right hemiparesis she developed a left sided foot drop. She was unable to dorsiflex her ankle or extend her toes. She had decreased sensation to light touch in her first web space but did have normal sensation throughout the dorsum of her foot as well as the remainder of her leg. The skin appeared normal. She did have some generalized tenderness of her foot but no acute swelling. She did not have any palpable defect in the tendons before or behind her lateral malleolus. MRI and 3-phase bone scan revealed acute changes in the soft tissues of the left leg, ankle and foot. The findings were compatible with cellulitis. According to the "double crush" theory, diabetic neuropathy is the first crush and compression of the nerve by soft tissue edema is the second crush.

INTRODUCTION

Foot drop is defined as significant weakness of ankle and toe dorsiflexion. It can be caused by 3 general etiological categories: neurologic (peroneal nerve injury), muscular and neuromuscular (myositis, myasthenia gravis), and anatomic (tendon rupture). These categories may overlap. The peroneal nerve is susceptible to different types of injury. Some of these include nerve compression from lumbar disc herniation (e.g. L4, L5, S1), trauma to the sciatic nerve, spondylolisthesis, spinal stenosis, spinal cord injury, bone fractures (leg, vertebrae), stroke, tumor, diabetes, lacerations, gunshot wounds, or crush-type injuries. Drop foot may be found in patients with Amyotrophic Lateral Sclerosis, Multiple Sclerosis, and Parkinson's Disease. Sometimes the peroneal nerve becomes injured when stretched during hip or knee replacement surgery.

We are presenting a diabetic patient who was admitted with right hemiparesis because of a stroke and after a few days in hospital developed left foot drop. After a thorough investigation, it turned out to be a complication of a silent cellulitis around the left ankle.

CLINICAL CASE

A 62-year-old Afro-American lady was transferred from another hospital where she presented complaining of right sided weakness. The night before, she went to bed without any complaints. When she woke up in the morning, she had right upper and lower extremity weakness, right side of face weakness and slurred speech. Brain CT showed acute hemorrhage in the left basal ganglia. She received fresh frozen plasma and her INR was 2.0 on admission. During the day, she noticed mild improvement in muscle strength in the right lower extremity. She said that she had some liquids and didn't have problems swallowing. She denied trauma. No dizziness. No change in her vision. No chest pain and no shortness of breath. The lady with a history of hypertension, mitral valve repair, chronic atrial fibrillation, diabetes and hysterectomy was on Toprol XL 50 mg. daily, Coumadin 5 mg. daily, Digoxin .125 mg. daily, Demadex 20 mg. daily, Nexium 40 milligrams daily and Accupril 140 mg daily. She was transferred on Catapres-TTS-2 patch weekly and insulin sliding scale. She stated she had a stroke in 2000. She said it involved her right body and that she had to use a cane and a walker for about a month, and then the problem resolved. She was a retired health service worker. No history of

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smoking or alcohol abuse. Father's health was unknown, apparently he was deceased. Mother was alive, she had hypertension. Patient with two siblings, one dead from aneurysm of the brain, one alive with hypertension. Two children alive and well, except one with hypertension.

On physical exam, the patient was lying in bed in no acute distress. Temperature 98.9, pulse 88, respiratory rate 18, blood pressure 143/76. The lungs were clear on auscultation. Heartbeat was irregular and there was a metallic click in the mitral area. There was no edema. On neurological exam, the patient was awake and alert with some speech hesitancy and some occasional difficulty following commands. Pupils were reactive. Extraocular movements were conjugate with mild decreased right gaze. Peripheral visual fields were intact. The right corneal reflex was absent, otherwise facial sensation was intact to pin and light touch. There was mild right facial weakness. Good tongue protrusion and mobility. Good phonation except for labials. There was right facial droop. Muscle strength in right upper extremity was 0/5 and in the lower extremity 2/5. Sensation was intact. No cogwheel rigidity, tremor or spasticity. No Hoffman, Babinski or clonus. Left toe was down, right toe was equivocal. Deep tendon reflexes were 1+ in the upper extremities, absent in the lower extremities.

EKG showed atrial fibrillation with a heart rate 78 per minute, intermediate axis and no ischemic changes. Labs: white blood cells 7.1, hemoglobin 13.1, platelets 245. Hemoglobin A1c 5.3. Cardiac enzymes negative.

It was determined that she had an acute cerebral vascular accident in the left hemisphere causing right sided hemiparesis. After a few days in hospital she developed a left sided foot drop. Orthopedics were consulted. What concerns her left lower extremity she was able to perform a straight leg raise and flex and extend her knee without significant difficulty, however, she did have some generalized weakness within her left lower extremity compared to what she said her baseline is. Regarding her ankle specifically, she was unable to dorsiflex her ankle or extend her toes. However, she was able to flex her toes and plantarflex her ankle with a 4/5 strength. She also had decreased sensation to light touch in her first web space but did have normal sensation throughout the dorsum of her foot as well as the remainder of her leg. She had 1+ dorsalis pedis pulse. The overlying skin appeared normal. She did have some generalized tenderness throughout her hind foot and the dorsum of her foot. She did not have any acute swelling in her foot, ankle or lower leg. She did not have any palpable

defect in her extensor hallucis longus or extensor digitorum longus tendons or perineal tendons behind her lateral malleolus. Her Achilles tendon was in continuity and she had a negative Thompson/Simmonds' test.

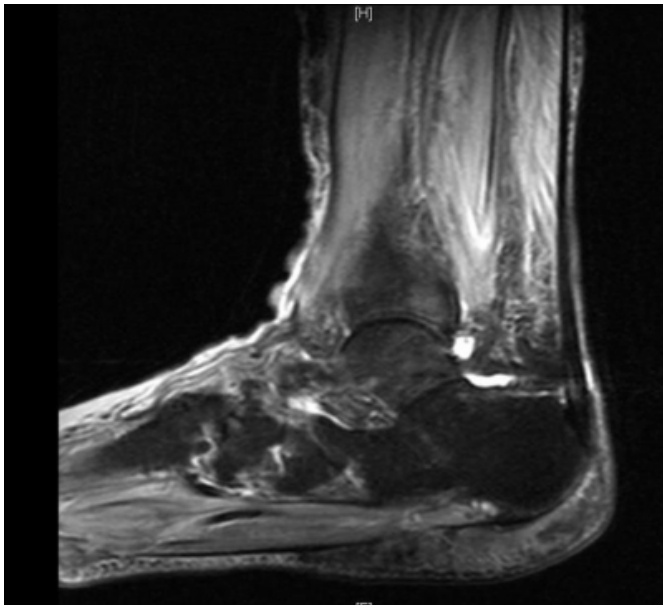
Whether foot drop was due to worsening of her stroke or to direct compression injury of the peroneal nerve while lying in bed over the last few days was unclear. It was unlikely that a traumatic rupture of any of her left lower extremity tendons caused this. It was more likely a neurological phenomenon but was it peripheral or central in nature? We did not know!

Plain X-ray imaging showed mild diffuse bony demineralization. Questionable periosteal reaction was noted adjacent to the medial malleolus although no focal acute fractures or dislocations were seen. There were mild degenerative and arthritic changes noted in the tarsus.

CT of the lower extremity with contrast showed patchy areas of stranding involving the subcutaneous fat both medial and lateral that extends to the muscle fascia. No well formed fluid collections were identified. The muscles of the calf were unremarkable. No osseous lesions were identified.

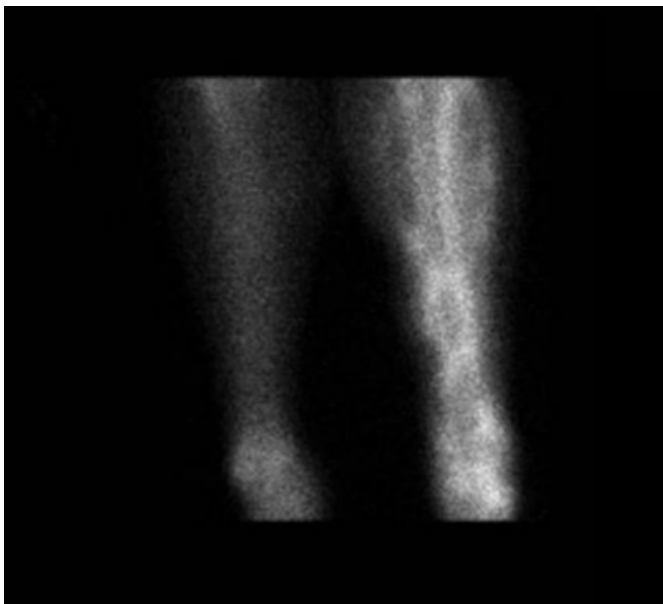
MRI of the left ankle without contrast showed heterogeneous marrow signal involving the tibia, calcaneus and to the greatest extent the talus, abnormal marrow signal along the distal soleus muscle with fluid or edema along the margins of the adjacent flexor hallucis longus tendon and diffusely increased T2 signal/edema throughout the subcutaneous tissues of the ankle and hind foot (Figure 1).

Figure 1



Left lower extremity venous doppler investigation was negative. The 3-phase bone scan showed increased flow throughout the left lower leg, ankle and foot region with a similar appearance on the blood pool images and delayed images without any real discrete focal intense abnormal areas of uptake about the left ankle to strongly suggest focal osteomyelitis - findings more compatible with cellulitis (Figure 2).

Figure 2



The patient was started on Vancomycin and quick improvement ensued.

DISCUSSION

After the sciatic nerve bifurcates at the distal third or mid thigh level, the peroneal nerve crosses laterally to curve over the posterior rim of the fibular neck to the anterior compartment of the lower leg, dividing into superficial and deep branches. The superficial branch travels between the 2 heads of the peronei and continues down between the peroneal tendon and the lateral edge of the gastrocnemius. It then branches to the ankle anterolaterally to supply sensation to the dorsum of the foot. The deep branch divides just after rounding the fibular neck. The initial branch supplies the anterior tibial muscle. Remaining branches supply the extensor digitorum longus and extensor hallucis longus and a small sensory patch at the first dorsal web space (sensation was decreased in this area in our patient).

The foot and ankle dorsiflexors include the tibialis anterior, extensor hallucis longus, and extensor digitorum longus. These muscles help the body clear the foot during swing phase and control plantar flexion of the foot on heel strike. Weakness in this group of muscles results in an equinovarus deformity. This is sometimes referred to as steppage gait, because the patient tends to walk with an exaggerated flexion of the hip and knee to prevent the toes from catching on the ground during swing phase. When walking, the force of heel strike exceeds body weight, and the direction of the ground reaction vector passes behind the ankle and knee center. This causes the foot to plantar flex and, if uncontrolled, to slap the ground. Ordinarily, eccentric lengthening of the anterior tibialis, which controls plantar flexion, absorbs the shock of heel strike. Foot drop can result if there is injury to the dorsiflexors or to any point along the neural pathways that supply them (our patient did not have any palpable defects in the tendons of these muscles).

The functional integrity of neuronal axons depends on the continued supply of trophic substances synthesized in the neuronal perikaryon and transported down the axon, known as axoplasmic flow. A laceration or a crush injury interrupt this flow. When a proximal insult affects the nerve root, it diminishes axoplasmic flow, making it more susceptible to injury. A distal lesion further compromises the flow, and clinical palsy results. This is the double crush phenomenon thought to be responsible for the increased risk of foot drop after hip replacement in a patient with preexisting spinal stenosis. The spinal stenosis causes the proximal compromise, and intraoperative stretch of the sciatic nerve provides the distal insult. This also applies to diabetic

patients with generalized neuropathy when a mild local pathologic process in the leg like cellulitis affects the peroneal nerve and leads to a foot drop. Diabetic nerves are more vulnerable to compressive injury at potential sites for entrapment₂. According to the “double crush” hypothesis, most patients remain asymptomatic despite having diabetic nerve disease. Only when the additional injury occurs (compression of the nerves at entrapment sites) will the patients become symptomatic. Applying this theory on our case, the metabolic stress of diabetes is the first crush and compression of the nerve by soft tissue edema in the leg cause the second crush.

The double-crush syndrome was initially described by Upton and McComas in 1973₃. They postulated that nonsymptomatic impairment of axoplasmic flow at more than one site along a nerve might summate to cause a symptomatic neuropathy. This was suggested by their clinical observation that the majority of their patients had a median or ulnar neuropathy associated with evidence of cervicothoracic root lesions. They also hypothesized that one of the constraints on axoplasmic flow could be a metabolic neuropathy, and this is supported by the high association of diabetes and carpal tunnel syndrome. Other researchers have since reported series of patients supporting the frequent association of a proximal and distal nerve compression syndrome, including carpal tunnel syndrome associated with cervical radiculopathy, brachial plexus compression, and diabetic neuropathy. Subsequently, MacKinnon and Dellon have expanded the description of this syndrome to include a) multiple anatomic regions along a peripheral nerve, b) multiple anatomic structures across a peripheral nerve within an anatomic region, c) superimposed on a neuropathy, and d) combinations of the above₄.

It is known that diabetic patients are at risk for developing

cellulites. Cellulitis manifests itself as swelling, redness, pain, and/or warmth. Our diabetic patient did not show any clinical sign of cellulitis or a laboratory finding pointing to an inflammatory process in the organism and it did not come to mind as a possible cause of foot drop. Diabetic patients are also at risk for strokes. The first impression was that it could be a complication of a developing stroke or the result of nerve compression in bed. The third possible cause of foot drop, tendon rupture, was unlikely since no traumatic event was reported. In addition to that, the patient had no history of muscle or neuromuscular disease. MRI revealed acute soft tissue changes around the left ankle and 3-phase bone scan showed increased flow throughout the entire left leg, ankle and foot without any discrete focal intense abnormal areas of uptake which would point to osteomyelitis. The findings were more compatible with cellulitis. Thanks to these two diagnostic investigations the cause was exposed.

CONCLUSIONS

Foot drop is a deceptively simple name for a potentially complex problem. In a diabetic patient with neuropathy and in the absence of trauma it can be a complication of a smoldering cellulitis in the leg or around the ankle. In such a case, MRI and 3-phase bone scan are the necessary diagnostic methods to exclude osteomyelitis and confirm cellulitis.

References

1. Pritchett JW, Porembski MA. Foot Drop. EMedicine, Dec 2006. Available at: <http://www.emedicine.com/orthoped/topic389.htm>
2. Upton, McComas. The double crush in nerve entrapment syndrome. *Lancet* 1973;2/7825:359-362.
3. Dellon AL, MacKinnon SE, Seiler WA. Susceptibility of the diabetic nerve to chronic compression. *Ann Plast Surg* 1988;20:117-119.
4. Dellon AL, MacKinnon SE. Chronic nerve compression model for the double crush hypothesis 10. *Ann Plast Surg* 1991;26:259-264.

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