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Bilateral foot drop linked to rapid intentional weight loss and long-distance walking

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ABSTRACT

There are many causes of acute onset foot drop ranging from deep fibular nerve or sciatic nerve injury caused by trauma or a compressive mass such as a neuroma, to spinal cord disorders like disc herniation causing L4-5 radiculopathy. Even brain disorders like MS, stroke or ALS can result in foot drop and various muscular dystrophies affecting the tibialis anterior muscle responsible for foot dorsiflexion and eversion. We present a case of bilateral foot drop as a complication of rapid 70 lb weight loss which was described in literature previously as "slimmer's palsy".

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KEYWORDS

fibular mononeuropathy, foot drop, weight loss, slimmer's palsy

A 56-year-old diabetic white female was referred for an electromyography (EMG) study of her legs due to bilateral foot drop. She had no family history of neuromuscular diseases. In 2015, while weighing 264 lb, she started pursuing a healthier lifestyle and by June 2016, was down to 225 lbs. She continued weight loss attempts by walking 10,000 to 15,000 steps daily and reducing her carbohydrate intake. In November 2016, she began to notice a complete right foot drop, followed in December 2016 by total left foot drop. At that point she had lost a total of 109 lb, and weighted 155 lbs. The patient did admit to crossing her legs more often since slimming down. Additionally, in her five year employment as a kindergarten teacher, she sat on low chairs while teaching, presumably putting additional pressure on the fibular nerve.

Upon initial presentation, patient's height was 5'6", weight was 168 pounds and body mass index was 27.1. Blood pressure was 176/96. There was no distal leg atrophy, fibular nerve hypertrophy or Tinel sign upon palpation at the fibular head. Scapular winging, neck or facial weakness were absent. Strength testing revealed full power except 2/5 in bilateral foot dorsiflexion, 0/5 in bilateral foot eversion, 5/5 at foot plantarflexion, and 0/5 in bilateral toe dorsiflexion. Gait was drop-foot. Deep tendon reflexes (DTR) were 2+ except for absent Achilles reflexes. Sensation was intact.

Nerve conduction studies (NCS) showed a normal left fibular motor response recorded from the extensor digitorum brevis (EDB) muscle, however the amplitude recorded from the left tibialis anterior (TA) muscle was moderately reduced (3.5 mV) with normal distal motor latency (DML) and slowing at the fibular head (31 m/s). The right fibular motor amplitude recorded from the EDB muscle was very reduced (0.9 mV), with slowing at the fibular head (38 m/s), normal DML, and the amplitude recorded from the TA muscle was moderately reduced (2.0 mV). F- waves were normal (Figure 1). Needling of the legs showed abnormal spontaneous activity (fibrillations) bilaterally in the TA and peroneus longus (PL) muscles, and no motor units recorded because of inability to activate muscles to dorsiflex or evert her feet. No myopathic recruitment pattern was observed

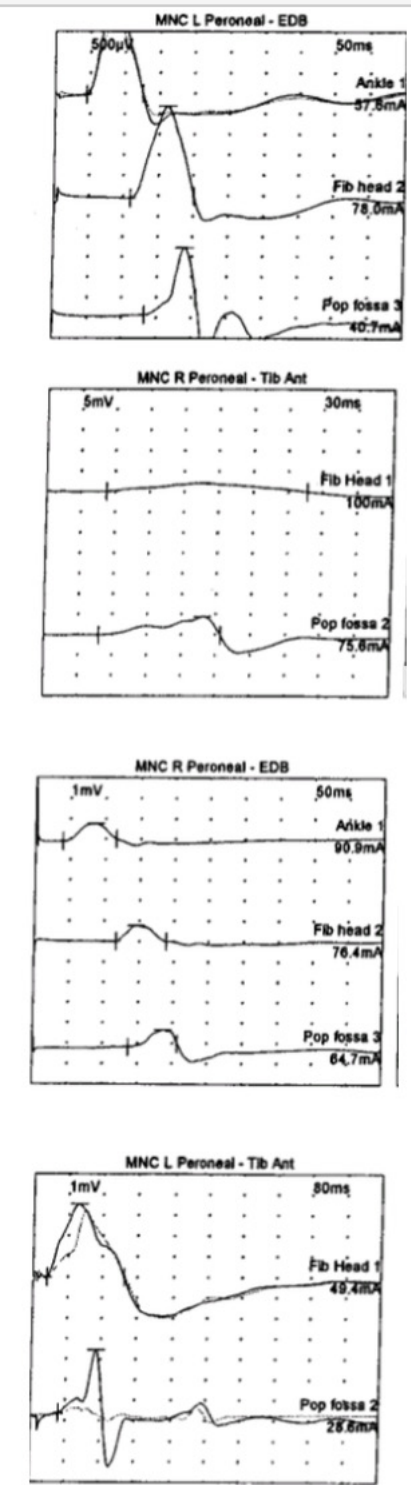


FIGURE 1. Nerve conduction study of bilateral peroneal motor nerves showing lower amplitudes on the right, which matches to where the onset of foot drop started initially.

in the rest of the leg muscles tested. She was advised to not cross her legs and decrease the daily distance walked.

Laboratory testing was unremarkable: normal CBC, A1c 5.4%, vitamin D 27, B12 321, iron 90, Ca 9.4, AST 20, ALT 15, TIBC 319, Mag 1.5. Genetic testing via Invitae blood collection kit showed no pathogenic mutations in the Comprehensive Neuromuscular Disorders Panel and Comprehensive Neuropathy Panel.

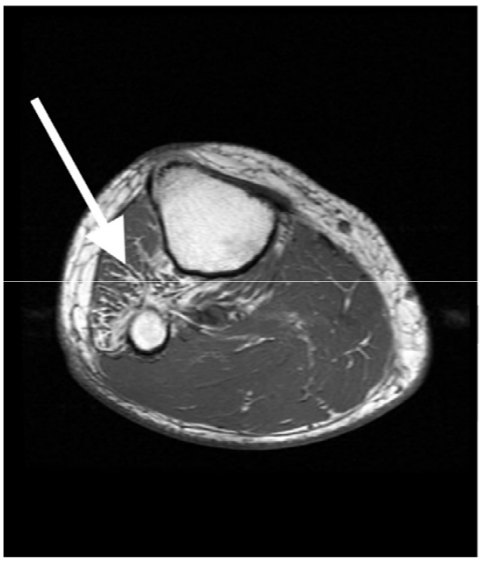
MRI of the right knee was obtained using a 1.5T machine (GE) to visualize the fibular nerve. Abnormal edema and mild fatty atrophy of the anterior compartment musculature was found with mild prominence of the right fibular nerve adjacent to the fibular head with no associated mass or abnormal enhancement (Figure 2). A complex tear of the right lateral meniscus was incidentally found and conservative management was recommended by orthopedics.

At her follow-up visit in May 2017, physical exam showed improvement in her lower extremity strength bilaterally. Foot eversion improved to 4/5 and dorsiflexion to 4/5. Sensation remained intact, but DTR at Achilles remained absent. She continued to walk with a high-steppage gait. She reported that she was able to ambulate more easily with bilateral ankle foot orthotic (AFO) braces. By October 2017, the patient had completely recovered strength bilaterally, though she did continue to wear braces when speed walking for exercise. On the morning prior to her last visit, she had walked 2 miles without pain. At her last visit, she weighed 176 pounds, had a BMI of 28.4, slightly increased from her initial visit. She was instructed to follow up as needed, to continue exercising as tolerated and to avoid sitting in low chairs.

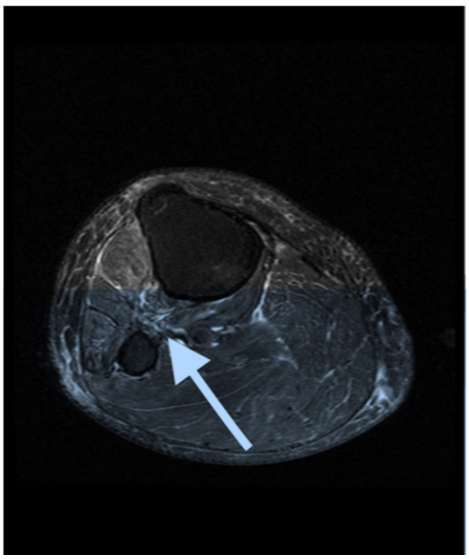
DISCUSSION

Fibular paralysis presents with toe extensor, ankle dorsiflexor and foot eversion weakness, foot drop and hypesthesia in the common fibular nerve distribution.1 The injury can be anywhere along the path of the nerve but is usually next to the

FIGURE 2. MRI of right knee without contrast.



A) Axial T1 imaging demonstrates fatty atrophy of the anterior compartment musculature.



B) Axial T2 image with fat saturation demonstrates enlargement of the peroneal nerve with muscular

fibular head. Our patient was diagnosed with "Slimmer's palsy" due to extreme weight loss resulting in atrophy of the fat pad that is normally present to cushion the fibular nerve. The problem was exacerbated by frequent crossing of her legs and sitting in low chairs. Hereditary neuropathy with predisposition to pressure palsy (HNPP) was ruled out in our patient with genetic testing, which corresponded to no prolonged DML or other demyelinating findings on the NCS. She was not showing signs of nutritional deficiency and had not taken any weight loss pills. She did not have any of the otherwise reported causes of fibular nerve paralysis like compression from lower extremity edema, frequent falls that would jeopardize the course of the fibular nerve[2], knee dislocation[3], alcoholism, chronic infection, thyrotoxicosis, paraneoplastic conditions, or vitamin B depletion.[4] Sotaniemi described ten cases of fibular nerve paralysis, nine of whom implemented a self-directed slimming regimen and all developed fibular paralysis over several days. He concluded that special care should be implemented when losing more than 10 lb per month and prescribed physical therapy; six patients recovered completely in 2-6 months.[4] The likely etiology is that the fat pad cushioning the peroneal nerve disappears in weight loss and thus exposes the nerve to potential for injury through compression, whereas before the nerve was more protected during traction and activities of daily living.

Because she achieved complete recovery, it is safe to assume that patient experienced neuropraxia, or stage 1 injury according to Sunderland's criteria, which constitutes conduction block or segmental demyelination.[3] In such cases it is important to rule out the possibility of hereditary neuropathy with liability to pressure palsies (HNPP), as Logroscino showed that it may play a role. He reported a case of post-operative foot drop after a total hip replacement with total spontaneous recovery in three months in a patient who later was found to have HNPP.[5] In Logroscino's case, ultrasound was employed to show enlargement in the fibular nerve, just as MRI demonstrated in our case.

We did MRI of the knee primarily to evaluate the region for any anatomic lesions such as adhesions, spurs, osteochondroma, ganglion cyst, synovial cyst, or schwannoma compressing the fibular nerve. We chose to focus on imaging only the right knee

region because patient's symptoms started first on the right, with right foot drop, manifesting one month earlier than left foot drop and because her nerve conductions were worse on the right. So if the MRI of right knee did not reveal peroneal nerve enlargement which would imply pathology and no other right sided structural issue was found, then by default the other side which was less affected, would not need to be imaged because MRI of left knee region would be low yield. The asymmetry in the nerve conductions is possibly reflective of a detrimental training effect whereby her right foot was the dominant leading foot while walking on the track in one direction. This may have exposed her to some imbalances in muscle group strength over the long distances she walked on a daily basis. It is curious however that during strength testing in clinic her foot dorsiflexion and eversion was not asymmetric, and rather appeared to be equally impaired bilaterally by the time she presented. This possibly correlates with the fact that serial neurophysiology tests would be best at showing the gradual improvement through remeylination of nerves and normalization timeline of nerve conduction studies. As we only performed one nerve conduction study, it appears that in this case, the reversal of nerve conduction changes on the EMG machine may lag behind objective power testing performed by the examiner.

This case highlights the importance of patient counselling, undertaking conservative management with suitable imaging and EMG surveillance to rule out fibular nerve entrapment that would require surgical decompression.

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