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A Rare Etiology of Bilateral Foot Drop: Weight Loss

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Case Report / Olgu Sunumu

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ABSTRACT

Excessive weight loss especially when it occurs in a short period of time can lead to some neurological problems including peroneal nerve palsy. Foot drop is the most common presentation of peroneal neuropathies and they rarely occur bilaterally at the same time. Herein, we presented a 46-year-old male patient who developed bilateral foot drop after losing 25 kgs. in two months with an intensive weight reduction diet which was advised by a doctor as a therapeutic regimen. The electromyographic study confirmed the bilateral entrapment neuropathy of the peroneal nerves at the fibular necks and these findings could not be attributed to any other factor except the weight loss. After a balanced diet and a physiotherapy programme, he recovered completely. This case highlights an uncommon but a well documented etiology of peroneal neuropathies. Being aware of this complication and close follow-up are important for the prognosis of these patients.

Keywords: foot deformities, peroneal neuropathies, weight loss

İki Yanlı Düşük Ayağın Nadir Bir Nedeni: Kilo Kaybı

ÖZ

Aşırı kilo kaybı, özellikle eğer kısa sürede oluşmuşsa, peroneal sinir hasarı da dahil olmak üzere, bazı nörolojik problemlere yol açabilir. Düşük ayak peroneal nöropatinin en sık prezantasyonudur ve nadir olarak eş zamanlı iki yanlı oluşur. Biz bu olguda, 46 yaşındaki erkek hastada, doktor tarafından tedavi amaçlı önerilen yoğun diyet nedeniyle iki ayda 25 kg. zayıfladıktan sonra iki yanlı düşük ayak gelişimini sunduk. Elektromiyografik çalışmalar, fibula başında iki yanlı peroneal sinirin tuzaklanmasını destekledi ve bu bulgular kilo kaybı haricinde başka bir faktöre bağlanamadı. Dengeli diyet ve fizyoterapi programı sonrası hasta tamamen düzeldi. Bu olgu, peroneal nöropatilerin sık görülmeyen ama iyi tanımlanmış bir sebebini vurgulamaktadır. Bu komplikasyonun farkında olmak ve hastaları yakın takip etmek, prognoz açısından önemlidir.

Anahtar kelimeler: ayak deformiteleri, peroneal nöropatiler, ağırlık kaybı

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Introduction

Obesity is a largely preventable public health problem associated with multiple chronic diseases. Fighting with obesity and successful weight reduction can reduce the comorbidities and improve the quality of life. But excessive weight reduction especially when it occurs in a short period of time can lead to some neurological complications. A relationship between peroneal nerve palsy and weight loss has been well documented (1).

Peroneal nerve palsy is the most common entrapment neuropathy in the lower extremity (2). Cruz-Martinez et al. (3) demonstrated that 20% of the peroneal neuropathies developed after marked weight loss and peroneal nerve palsy accounts for approximately 15% of all mononeuropathies in adults. The peroneal nerve is susceptible to injuries due to its superficial course at the level of the fibular head. Subcutaneous fat around the fibular head normally protects the peroneal nerve from damage. Weight loss leads to depletion of this fatty cushion and makes it more sensitive to compression (4,5).

The main presenting symptom of peroneal neuropathy is foot drop. Electrophysiologic studies can confirm, localize and grade the severity of mononeuropathies. Peroneal neuropathies rarely occur bilaterally at the same time. Bilateral involvement can be attributed to the injury mechanisms (femoral fractures, pelvic injury, squatting) or to the complications of systemic causes (2,6).

Herein we present a rare case of bilateral peroneal neuropathy which developed as an unusual complication of massive and rapid weight loss.

Case

A 46-year-old man was referred to our outpatient neurology clinic with a one-month history of bilateral foot drop. There had been no history of trauma or an unusual activity involving the legs. His medical history included new-onset diabetes mellitus (DM) and obesity with a body mass index (BMI) of 33.6 kg/m^2. Two months ago, he was diagnosed as DM type 2. Insulin, metformin and a weight loss programme were started. He reported that he had lost 25 kgs. (from 96 kgs. to 69 kgs.) during this short period and the insulin treatment was stopped. His neurological examination revealed weakness of ankle dorsiflexion on both sides with diminished sensation on the dorsum of the feet and anterolateral side of both calves. Deep tendon reflexes were normal. Tinel sign for peroneal nerve was positive bilaterally. The remainder of the neurologic examination was unremarkable. His blood count, serum biochemistry, thyroid functions and vitamin B12 levels were normal except high serum glycated hemoglobin A1c and glucose levels.

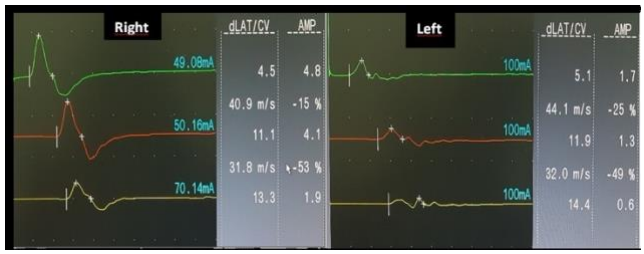

Figure 1: Peroneal motor study of both sides, recording from extensor digitorum brevis muscle and stimulating ankle, below fibular head and lateral popliteal fossa.

Nerve conduction studies were performed to localize the nerve injury. Recording from extensor digitorum brevis muscles and stimulating below fibular head and lateral popliteal fossa, there was a 53% block in the right compound muscle action potential (CMAP) amplitudes and a 49% block in the left CMAP amplitudes (Figure 1). Also, there was significant slowing of motor conduction velocities at the fibular necks of both sides. Left peroneal nerve was affected more severely than the right one. Nerve conduction studies of bilateral posterior tibial, sural, median and ulnar nerves were within the normal range. Needle electromyography (EMG) of bilateral tibialis anterior and peroneus longus muscles revealed motor unit potentials with normal amplitude, duration and phasicity, increased insertional activity, fibrillations, and sharp waves and reduced recruitment patterns. On the other hand, bilateral gastrocnemius, short heads of the biceps femoris and gluteus medius muscles were recorded as normal in needle EMG examination. So the EMG study was reported as showing bilateral entrapment neuropathy of the peroneal nerves at the fibular heads. A magnetic resonance imaging of the lumbar spine was performed and there was no

abnormality.

All other causes of foot drop were excluded and it was accepted as a complication of weight loss. A balanced diet and a physiotherapy programme were started promptly. After a follow-up period of four months, his feet drop improved satisfactorily and the control EMG found to be normal.

Discussion

Weight reduction is occasionally followed by neurological complications. In a study for assessing the clinical patterns of peripheral neuropathies occuring after bariatric surgery, it was reported that the mononeuropathies are the most frequent types, followed by the sensory-predominant polyneuropathies and the radiculoplexus neuropathies respectively (7). In the same study, 39 of the 435 patients developed mononeuropathy after bariatric surgery and two of them had peroneal neuropathy at the fibular head (7). The most common presentation of peroneal nerve palsy is foot drop. It is mostly due to compression, trauma or traction of the peroneal nerve around the knee that may result from habitual leg-crossing, squatting, expanding masses or may be seen in bed-ridden motionless patients having a constant pressure upon peroneal nerves (3,5). Since our patient had none of these risk factors, it was accepted as a complication of massive weight loss.

In addition to mechanical compression, metabolic changes also play an important role (4,8). Rapid and uncontrolled weight reduction may lead to severe metabolic deficiencies impairing the peripheral nervous system function. These may include disturbances in lipoprotein accumulation, catecholamine and hormonal activity and electrolytes (5,9). They cause intraneural oedema which leads to compression syndrome at the risky areas like anatomic tunnel regions (1). So the electromyographic study of our patient confirming the entrapment neuropathy at the fibular head is due to predisposing nature of the anatomical conditions. It was demonstrated that patients who developed peroneal neuropathy after weight loss had family members with electrodiagnostic findings suggesting hereditary neuropathy with liability to pressure palsies (HNPP) (10). So there may also be a genetic component of peroneal neuropathy after weight loss.

Both DM and widespread polyneuropathies are well-known risk factors for the development of mononeuropathies (2,11). Thaisetthawatkul et al. (11) demonstrated that increased baseline glycosylated hemoglobin is a risk factor for peripheral neuropathy after bariatric surgery. But the EMG study of our patient revealed no polyneuropathy and the temporal profile associated with the development of bilateral foot drop suggested that these mononeuropathies were due to rapid weight loss rather than DM.

Meylaerts et al. (4) examined the ultrasonographic characterization of common peroneal nerve and found that BMI was positively correlated with the transverse cross-sectional area of the fibular tunnel. They also showed that weight loss results in decrease in amounts of fat tissue around the fibular head which facilitates the chronic compression of the nerve supporting the hypothesis that the vulnerability could be increased upon severe weight loss.

Both the amount of weight loss and the time period in which weight loss achieved is significantly correlated with the foot drop (1). Our patient reported bilateral foot drop after a weight loss of 25 kgs. (26%) in two months. Loss of more than 15% of the body weight may result in peroneal neuropathy (12). The signs seem to appear only after at least two or three months of dieting (9). Sen et al. (13) demonstrated that after a mean weight loss of 50.6 kgs. in six months, 1.1% of the patients developed foot drop as a complication of laparoscopic sleeve gastrectomy (13). It was noted that if the weight loss exceeds 5 kgs. per month, special care should be taken (9). Slow weight reduction is recommended and the risk of developing neuropathies significantly lessens if the patients attends to nutritional clinics during these periods (1,7,14).

The prognosis for peroneal neuropathy after weight loss is good, especially if the underlying polyneuropathy is not severe (2). Diet modification, physical therapy and rehabilitation programmes should be the first-line treatment options. The outcome of a 68 weight loss associated peroneal neuropathy

patients reported that 78% of these patients recovered completely (5). Our patient's prognosis was also good. The severity of clinical weakness and ratio of conduction block were found to be correlated with delayed recovery function (3). Surgical intervention might be the second option in cases with severe or persistent deficits.

This case highlights a rare but a well-documented etiology of the peroneal nerve palsies. Being aware of this problem and prompt neurological and neurophysiological examination is important for the better management of these patients.

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