Case Report

An Underestimated Complication of Obesity Management: Bilateral Peroneal Neuropathy

Yesim SUCULLU KARADAG, Mustafa SAKA, Nese OZTEKIN, Fikri AK

Ankara Numune Education and Research Hospital Neurology Clinic, Turkey

Summary

A 36 years old male patient developed bilateral foot drop after starting an intensive weight loss programme. Diagnosis of bilateral peroneal nerve palsy was confirmed using nerve conduction studies and electromyography. After physical therapy and a diet modification, his symptoms resolved in a few months. Weight loss is a known risk factor for the development of neuropathy at the fibular head because of fat loss previously protecting the common peroneal nerve against compression. However bilateral involvement of peroneal nerves is very rare. Physicians should keep in mind peroneal neuropathy as a complication of weight loss. In this case, diet modification and physical therapy might be a reasonable method before surgery.

Key words: Bilateral Peroneal Neuropathy, Obesity, complication of weight loss, Electromyography

INTRODUCTION

Peroneal neuropathy, the most common mononeuropathy in the lower extremity, results in clinical complaint of weakness of the ankle dorsiflexor and evertor muscles. Although it may also be seen in a wide variety of other clinical conditions such as sciatic mononeuropathy, lumbosacral plexopathy, or a lumbar (L5) radiculopathy, foot drop is the most common presentation of peroneal neuropathy. Peroneal palsy is commonly due to compression or traction of the peroneal nerve around the knee. Nutritional deficiencies, vasculitic syndromes, malignancies, infections, trauma and defects about anatomical structures are the main etiologic causes of peroneal neuropathy. Weight loss is also accepted as a risk factor for peroneal nerve palsy.
Peroneal nerve palsies seldom occur bilaterally at the same time. Numerous case reports with bilateral involvement regarding injury mechanisms (squatting, pelvic injury, femoral fractures) have been published. Herein a case of bilateral peroneal neuropathy manifested by sequential foot drop, which occurred after extreme weight loss is presented.

**CASE PRESENTATION**

A 36 year-old-man was referred to our clinic for bilateral foot drop with sensory loss on the lateral aspect of his legs and feet. He reported weakness in his lower extremities with loss of dorsiflexion in his ankles for one month. He had no positive finding in questioning about a hereditary compression neuropathy. But query about the etiologic factors of foot drop revealed that he had started an intensive weight loss programme five months ago and his complaints started after this programme. By the end of 5th month, the patient had lost 25 kgs (from 109 kgs to 84 kgs). He had no concomitant disease. Blood count, serum biochemistry, thyroid functions and vitamin B12 levels were in normal range. Patient's examination revealed weakness of feet dorsiflexion/ eversion (2/5) and diminished sensation on the dorsum of the feet and anterolateral side of both calves.

Nerve conduction studies were used for localization of the site of the nerve injury. Recording from extensor digitorum brevis muscles and stimulating from below and above fibular heads, there was a 70% block in compound muscle action potential (CMAP) amplitudes. Also there was a significant drop in motor conduction velocities across the fibular heads. Right peroneal nerve was affected more severely. Needle electromyography (EMG) of bilateral tibialis anterior and peroneus longus muscles revealed motor unit potentials of normal amplitude, duration, and phasicity; increased insertional activity, fibrillations, and sharp waves and reduced recruitments. Conduction studies of bilateral sural, posterior tibial, ulnar and median nerves were in normal range. Needle EMG of bilateral gastrocnemius, short head of biceps femoris and gluteus medius muscles were normal. Denervation potentials were not observed in examining lumbar paraspinal muscles. After these procedures all other causes of foot drop were excluded and isolated partial lesion of bilateral (right>left) common peroneal nerves was diagnosed.

He was also examined by the orthopedist. His lumbar spinal MRI and bilateral crural MRI were in normal ranges.

After the etiologic investigation, a diet modification was done. Also a physical therapy and rehabilitation programme consist of 30 sessions was started. In each session 20 minutes of hotpack, 5 minutes of ultrasound, 15 minutes of electrical stimulation and active assistive ranges of motion exercises were performed. In addition to foot orthosis, correct positions of ankles were described. By the end of 6th month, there was significant clinical and electrophysiological improvement.

**DISCUSSION**

Obesity is one of the most important and disturbing global epidemic condition that affects humans, according to World Health Organization. While fighting with obesity neurologic problems might occur. In the literature, there are many case reports of peroneal neuropathy after weight loss, anorexia nervosa, and bariatric surgery for morbid obesity.

There are several proposed mechanisms regarding peroneal neuropathy after weight loss. Since peroneal nerve is quite superficial at fibular neck and fat tissue around the nerve protects the common peroneal nerve against compression, weight loss more than 15% of body weight increase the susceptibility of peroneal nerve injury. It was noted that 20% of 150 cases of peroneal neuropathy were associated with dieting and weight loss. Our patient had lost approximately 25 kgs
Peripheral nerves glide relative to their surrounding tissues during movement. Loss of fat tissue causes impairment of nerve movement in longitudinal and transverse directions. Impaired nerve excursion at one site may increase neural strain at distant sites and causes nerve disorders especially at the entrapment areas.

Local trauma and compression are the most common causes of nerve entrapment. Also defects about anatomic structures may create angulation, compression, and stretching of the nerve. Since our patient had neither any anatomic defect nor history of trauma, prolonged kneeling or squatting; bilateral peroneal neuropathy in this case could be accepted as a complication of intensive weight loss programme.

Because of bilateral involvement, one can assume a double crush phenomenon due to increased susceptibility to compression neuropathy. Although the mechanism for increased susceptibility has not been fully elucidated, change in lipoprotein and catecholamine metabolism and increase in hormonal activity after weight loss may be suspected pathogenetic mechanism of this type of peroneal neuropathy. Metabolic influences on a nerve cell leads to decreased nerve cell function and this may cause mononeuropathy or polyneuropathy. It is well known that widespread polyneuropathy is a risk factor for mononeuropathy at common sites of entrapment, for example; peroneal neuropathy at the fibular head. Therefore, we propose that regardless of the origin of the susceptibility, weight loss is likely a risk factor for the development of the superimposed peroneal neuropathy.

The prognosis for peroneal neuropathy after weight loss is good particularly if any underlying polyneuropathy is not severe. Non surgical approach as diet modification, physical therapy and rehabilitation programmes are the first line treatment options. Rapid replacement of nutritional and metabolic support, gain weight with dietary programmes are vital in treatment of peroneal neuropathy caused after weight loss. By conservative approach a successful improvement could be seen as in our patient. Surgical therapy might be the second option in disabling cases especially in patients with bilateral involvement. Our patient's prognosis was good, bilateral motor conduction velocities of distal peroneal nerves were increased and velocities at the fibula neck segments were improved.

As a conclusion physicians should keep in mind peroneal neuropathy as a complication of weight loss programmes. In this case, prognosis is good and diet modification in addition to physical therapy might be the first choice of treatment.

Correspondence to:
Yeşim Sücüllü Karadağ
E-mail: yesimkaradag@yahoo.com

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