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Case Report

Bilateral peroneal neuropathy after bariatric surgery: A case report

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ABSTRACT

The popularity of bariatric surgery (BS) began to increase due to the dramatic rise in severe obesity in the past decades. Postoperative follow-up after BS is important to avoid possible medical complications. Therefore, medical complications after BS should be well-known and defined. Herein, we present a case of bilateral peroneal neuropathy (PN) developed after successful BS. The patient lost 40 kg during 16 weeks of follow-up. The foot drop developed after 18 weeks after surgery on the left side and than 24 weeks after surgery on the right side. Peroneal neuropathy-associated weight loss is usually unilateral. Bilateral PN with weight loss is uncommon. The rate of weight loss is an important risk factor for PN. This case report highlights the importance of optimal dietary after BS to control the weight loss rate and nutrient deficiency.

Keywords: Bariatric surgery; foot drop; gastric banding; peroneal neuropathy; weight loss.

The long-term success rates of non-surgical modalities for obesity such as diets, dietary supplements, and exercise programs have been quite variable. Currently, there is a considerable interest to bariatric surgery (BS) in the treatment of morbid obesity.[1,2] However, the awareness about medical complications after BS is still limited.

Peroneal neuropathy (PN) is one of the neurological complications after BS.[1] It is common in the fibular head as mononeuropathy, although PN-associated weight loss is relatively rare and usually unilateral.[3-7] In the literature, there are few cases with bilateral PN after weight loss.[8,9] The rate and amount of weight loss is a significant risk factor for the development of PN.[7] Therefore, it is of utmost importance to keep the rate of weight loss after BS under control. Herein, we report a case of bilateral PN occurred after BS.

CASE REPORT

A 30-year-old women with a long-term history of obesity underwent gastric banding surgery with 110 kg weight and 37.9 kg/m^2 Body Mass Index. She had no comorbidity, except for polycystic ovary syndrome. She was on daily 5 mg folic acid and combined B vitamin preparation (250 mg thiamine HCl [vitamin B1], 250 mg pyridoxine HCl [vitamin B6] and 1 mg cyanocobalamin [vitamin B12]) after BS. She lost 40 kg within 16 weeks during follow-up.

Eighteen weeks after BS, she had weakness on her left foot. Subsequently, 24 weeks after surgery, similar symptoms started at the right foot. She was, then, referred to our electromyography (EMG) laboratory 25 weeks after surgery. She had habitual leg crossing and weakness of bilateral ankle dorsiflexion and eversion (left: 2/5 and right: 3/5) and weakness of bilateral toe dorsiflexion (left: 0/5 and right: 1/5). The strength of other muscles was normal. She had also hypoesthesia at the dorsum of the left foot. The upper and lower extremity deep tendon reflexes were normal. The Babinski responses were bilateral flexor.

The laboratory findings were normal. There was no abnormality at glucose and lipid metabolism parameters. There were no nutritional deficiencies. The compound muscle action potential (CMAP) was recorded with a surface electrode placed on extensor digitorum brevis muscle (EDB) for deep peroneal nerve motor conduction study. The supramaximal

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Table 1. Nerve conduction studies findings of peroneal nerve

	Latency (ms)	Amplitude (mV)	Amplitude (%)	Velocity (m/sn)
Left peroneal nerve (motor)				
Ankle-extensor digitorum brevis	4	5	-6	44.4
Distal fibular head-ankle	10.3	4.7		
Proximal fibular head-distal fibular head	17.2	**0.1**	**-97**	**21.7**
Right peroneal nerve (motor)				
Ankle-extensor digitorum brevis	4.2	7.6	-11	49.1
Distal fibular head-ankle	9.7	6.8		
Proximal fibular head-distal fibular head	14.5	**2**	**-71**	**35**
Left superficial peroneal nerve (sensory)		SNAP not reported		
Right superficial peroneal nerve (sensory)	2.4	**6**		39.8

SNAP: Sensory nerve action potential; Bold numbers: Abnormal values.

stimulation was done at ankle, above, and below the fibular neck. The motor conduction velocity at the bilateral fibular head were slowed (left: 21.7 m/s, right: 35 m/s, normal: >40 m/s). The CMAP amplitude loss, which was compatible with conduction block, was observed at the bilateral fibular head (right: 97%, left: 71%). The sensory nerve action potential (SNAP) was unable to be obtained using the left superficial peroneal sensory nerve conduction study. The amplitude of SNAP was reduced in the right superficial peroneal sensory nerve conduction study. The nerve conduction study findings of both peroneal nerves are seen in Table 1. The other nerve conduction studies (median, ulnar, tibial, and sural) were within normal limits and polyneuropathy was ruled out. Abnormal spontaneous activities were observed on needle EMG of the bilateral tibialis anterior and peroneus longus muscles. Reduced recruitment of motor unit potentials were seen in tibialis anterior and peroneus longus muscle bilaterally. Needle EMG studies of the other muscles (i.e., gastrocnemius, short head of biceps femoris, and paraspinal muscles) showed normal results. Therefore, L5 radiculopathy and sciatic neuropathy were excluded. These results were compatible with conduction block and partial axonal loss of peroneal nerve at the fibular head bilaterally.

The range of motion exercise, stretching exercise, and ankle foot orthosis for assisting weakness of ankle were recommended. Electrical stimulation to tibialis anterior muscle was prescribed. However, the patient was lost to follow-up, as he preferred visiting an external center for this treatment located far distance from our center.

DISCUSSION

Woltman[10] was the first who reported the relationship between the weight loss and foot drop. Thereafter, many cases have been published about the correlation between PN and weight loss, such as diets, malnutrition, anorexia, and severe diseases including malignant diseases.[3-6,11] Peroneal neuropathy is a common mononeuropathy which accounts for 15% of all mononeuropathies in adults.[12] However, the prevalence of PN-associated weight loss has not been reported, yet. In a study, peripheral neuropathy has been shown to develop in 26 (7%) of 393 patients with BS, and only one of them was found to have PN.[13]

Peroneal neuropathy-associated weight loss is usually unilateral.[7,14] In the literature, there are few cases with bilateral PN after BS.[8,9] Although mononeuropathy was the most common peripheral neuropathy after BS (81%), polyneuropathy (8%) and radiculopathy (11%) may be also observed.[13] Therefore, the diagnosis should be considered in the differential diagnosis, particularly in patients with bilateral involvement. Cruz-Martinez et al.[12] suggested that extensive electrophysiologic study should be performed to exclude polyneuropathy. In our case, EMG helped us to rule out other differential diagnoses including polyneuropathy and radiculopathy, and it is also beneficial to follow the severity and prognosis of neuropathy.

Furthermore, mechanical factors play an important role in PN-associated weight loss. Several studies have suggested that the peroneal nerve becomes more susceptible to minor injuries, possibly due to the loss of subcutaneous tissues.[3,12] Weyns et al.[7] compared nine patients who developed foot drop after BS with a control group without PN. The authors found that rapid weight loss was a high risk for foot drop after BS, and recommended slow weight reduction after BS to avoid PN. However, mechanical factors are not the sole factors to be blamed. The etiology of PN-associated

weight loss is likely multifactorial. The metabolic factors may also involve in the development of nerve dysfunction.[3] Possible risk factors for peripheral neuropathy after BS include proportion of weight loss, accompanying gastrointestinal symptoms, not being under control of a nutritional clinic, low serum albumin and transferrin levels. In addition, in a study, sural nerve biopsies showed prominent axonal degeneration and perivascular inflammation.[2] Therefore, inflammation and altered immunity may also play a role in the pathogenesis of peroneal neuropathy after BS.[2]

Nonetheless, there is no consensus in the literature regarding the optimal treatment. Several studies indicate that surgical intervention seems successful.[5] Weyns et al.[7] suggested that surgery was indicated, if there was no evidence of functional recovery within three weeks after conduction block across the fibular head in PN. On the other hand, there are other studies in the literature regarding a good recovery with the conservative treatment (i.e., nutritional replacement, regulating the weight loss rate, and physical therapy).[14] One of the previous studies showed that 29 of 30 patients with PN-associated weight loss recovered within three weeks to three months, and only one patient needed surgical intervention and recovered at six months after surgery.[12] However, further large-scale and long-term follow-up studies are required to compare conservative and surgical treatment in this patient population.

In conclusion, physicians need to be alert about PN which develops after BS. Electromyography is a very useful tool for both diagnosis and follow-up of recovery. To prevent the development of PN after BS, the rate of weight loss should be taken under control. Most importantly, diet of patients should be organized with nutritional clinical support. Although inflammation and altered immunity may also play a role in the pathogenesis, this should be investigated in further studies.

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