Turk J Phys Med Rehab 2026;72(x):i-iv
DOI: 10.5606/tftrd.2026.16616
Available online at www.turkishjournalpmr.com

Case Report

From obesity to foot drop: A rare but reversible complication of bariatric surgery

Demet Ferahman, Elif Sena Özkaya, Ümit Yalçın

Department of Physical Medicine and Rehabilitation, Başakşehir Çam and Sakura City Hospital, İstanbul, Türkiye

ABSTRACT

Peroneal neuropathy is one of the rarest yet disabling complications that could occur after rapid weight loss following a bariatric surgery. Herein, we reported a 23-year-old female patient who developed foot drop after losing 88% of her excess weight within six months after having laparoscopic sleeve gastrectomy. Electrophysiological studies showed total common peroneal nerve involvement. Within three months, the patient showed a full functional and electrophysiological recovery after receiving conservative treatment. This case emphasizes the significance of early diagnosis, as well as the potential role of corticosteroids in individualized, nonsurgical management of peroneal neuropathy in bariatric patients.

Keywords: Bariatric surgery, foot drop, neurological complications, peroneal neuropathy, weight loss.

Obesity is one of the significant health issues in the world. As obesity increases, the number of bariatric surgery procedures increase. Even after a successful bariatric surgery, various musculoskeletal and neurological complications may develop due to rapid weight loss, micronutrient deficiencies, and hormonal changes.[1,2] Musculoskeletal complications may include bone fragility, sarcopenia, increased fracture risk, and peripheral nerve injury caused by mechanical unloading and lack of nutrition. Although malabsorptive procedures are more commonly linked, even restrictive surgical methods, such as sleeve gastrectomy, have been associated with musculoskeletal complications.[1]

Among these complications, peripheral neuropathy is the most common, with peroneal neuropathy being one of them. Clinically presenting as foot drop, this complication can lead to significant limitations in functioning if not diagnosed early and managed rapidly.[3]

In this case report, we discussed a female patient who lost 30 kg within six months following laparoscopic sleeve gastrectomy (LSG) and later showed foot drop. After a thorough evaluation, the patient underwent multidisciplinary conservative treatment, including corticosteroid therapy, vitamin supplementation, physical therapy, and orthotic support, resulting in rapid functional and electrophysiological recovery. This case is significant and different from previously reported cases due to the early signs of reinnervation, rapid functional recovery, and the successful outcome accomplished with personalized conservative treatments, distinguishing it from previously reported cases. This case report aimed to emphasize that, although rare, peroneal neuropathy is one of the potential complications of bariatric surgery and to draw attention to the significance of early diagnosis and appropriate treatment.

CASE REPORT

A 23-year-old female patient who was dragging her foot while walking and having complaints of weakness in the left foot presented to the clinic.

Corresponding author: Demet Ferahman, MD. Başakşehir Çam ve Sakura Şehir Hastanesi, Fiziksel Tıp ve Rehabilitasyon Kliniği, 34480 Başakşehir, İstanbul, Türkiye.
E-mail: demetferahman@gmail.com

Received: March 12, 2025 **Accepted:** July 22, 2025 **Published online:** December 19, 2025

Cite this article as: Ferahman D, Özkaya ES, Yalçın Ü. From obesity to foot drop: A rare but reversible complication of bariatric surgery. Turk J Phys Med Rehab 2026;72(x):i-iv. doi: 10.5606/tftrd.2026.16616.

The medical history revealed that the patient lost 30 kg (initial weight: 105 kg; weight after six months: 75 kg) after undergoing LSG six months ago. The initial body mass index was 43.7, which decreased to 31.2 after weight loss. The patient had no known medical condition and was not on any regular medication.

On physical examination, muscle strength was 0/5 in the dorsiflexors of the left foot and hallux, while other muscle groups had full strength. Sensory examination revealed dysesthesia on the dorsal aspect of the left foot compared to the right side. Laboratory findings were as follows: calcium, 10.19 mg/dL; magnesium, 2.11 mg/dL; vitamin D, 15 ng/mL; folic acid, 6.24 ng/mL; ferritin, 28 ng/mL; and vitamin B12, 179 pg/mL.

Electromyography (EMG) was performed three weeks after symptom onset to investigate the etiology of foot drop. Nerve conduction studies revealed normal sensory conduction in the left sural nerve and normal motor conduction in the tibial nerve. However, motor conduction of the left common peroneal nerve showed normal responses at the ankle level, but no response was obtained at the fibular head level. Additionally, sensory conduction in the superficial peroneal nerve was absent. Needle EMG showed abnormal spontaneous activity in the left peroneus longus and extensor hallucis longus muscles, with no motor unit potential or interference pattern. In the tibialis anterior muscle, small amplitude polyphasic motor unit potentials with severe reduction were observed along with abnormal spontaneous activity. The lateral head of the gastrocnemius and the short head of the biceps femoris muscles had normal findings on needle EMG. The electrophysiological findings were interpreted as consistent with total involvement of both the deep and superficial branches of the left common peroneal nerve, as evidenced by the absence of the superficial peroneal sensory nerve action potential and the inability to obtain compound muscle action potential from the fibular head during common peroneal motor conduction studies. To rule out knee pathologies that could cause nerve compression, magnetic resonance imaging of the knee was performed, which revealed no abnormalities.

After the diagnosis of peroneal neuropathy, the patient was immediately started on intramuscular cyanocobalamin and oral vitamin D supplementation,

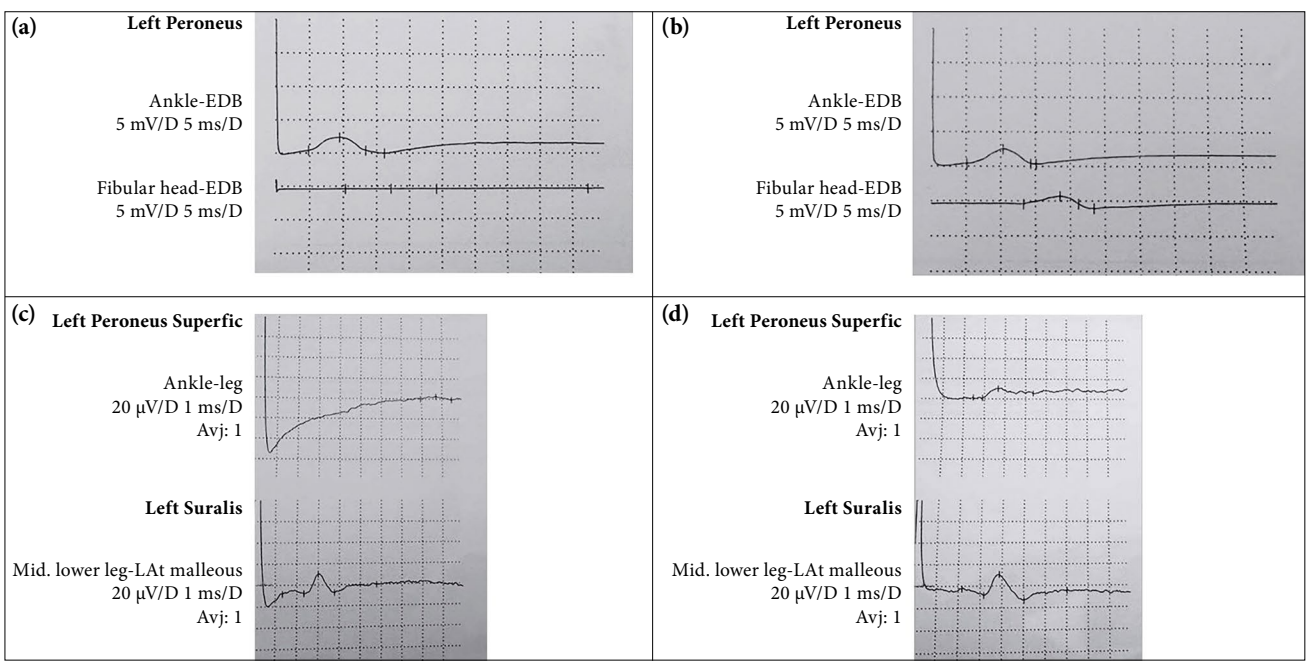

Figure 1. Electromyography findings of the patient. **(a)** In the left lower extremity peroneal nerve motor conduction study, low compound muscle action potential amplitudes are observed at both the ankle and fibular head stimulation sites, with conduction block detected at the fibular head level. **(b)** Three months after treatment, motor conduction study shows improvement in the conduction block. **(c)** The left peroneus superficialis sensory conduction study shows no sensory nerve action potential before treatment. **(d)** A sensory nerve action potential response observed after treatment, indicating recovery in sensory conduction. These findings demonstrate motor and sensory improvement in peroneal nerve dysfunction.

along with daily 24 mg oral prednisolone therapy. In three weeks, the corticosteroid dose was gradually decreased and discontinued. Physical therapy was applied to the patient, which included exercises such as stretching and strengthening of the ankle and toe dorsiflexors, as well as neuromuscular electrical stimulation applied to the tibialis anterior and extensor hallucis longus muscles. As the patient was not able to perform ankle dorsiflexion while walking, she was prescribed an ankle-foot orthosis, which aimed to improve functioning.

After three months of conservative treatment, including physical therapy, ankle-foot orthosis, and administration of vitamin supplements and oral corticosteroids, the patient achieved complete clinical and electrophysiological recovery. At the end of the treatment period, the patient had regained full ankle dorsiflexion strength, resumed independent ambulation without assistive devices, and returned to normal daily activities. The patient further underwent nerve conduction studies, which confirmed that reinnervation occurred, with substantial improvement in compound motor and sensory nerve action potentials (Figure 1). We did not observe any residual neurological deficits when we further evaluated her after three months. A written informed consent for publication was obtained from the patient.

DISCUSSION

This case is notable because the patient showed a rapid and complete functional and electrophysiological recovery after applying a conservative treatment strategy, which included corticosteroid therapy. The features that distinguish this case from previously reported cases of peroneal neuropathy following bariatric surgery are *(i)* early reinnervation findings, *(ii)* significant motor improvement within a short period, and *(iii)* the integration of corticosteroids. Peroneal neuropathy after bariatric surgery is believed to result mainly from two mechanisms: *(i)* mechanical compression of the nerve due to the loss of adipose tissue around the fibular head and *(ii)* nutritional deficiencies leading to neuropathy. Although rare, foot drop should be considered as an important complication in patients experiencing rapid postoperative weight loss, requiring early recognition and personalized conservative treatment.

Studies show that patients who lose more weight rapidly than expected within the first six months after bariatric surgery are at higher risk for peroneal nerve compression and foot drop. In a study by Şen et al.,[4] all patients who developed peroneal neuropathy lost more than 80% of their excess weight within the first six months. Similarly, studies by Weyns et al.[5] and Thaisetthawatkul et al.[6] reported that patients with peroneal neuropathy lost an average of 45 kg over eight months. Our patient lost 30 kg in six months, which corresponded to approximately 88% of her excess weight, placing her in the high-risk group for neuropathic complications.

Patients who undergo LSG may experience both rapid fat loss and malnutrition-related vitamin deficiencies. Loss of adipose tissue around the fibular head typically shows as a mononeuropathy affecting only the peroneal nerve.[7] In contrast, vitamin deficiency-related neuropathies tend to present as polyneuropathy, affecting multiple nerves in addition to the peroneal nerve.[8] However, cases with isolated foot drop accompanied by significant vitamin deficiency can also occur, as observed in our patient. Evaluation with EMG is crucial in distinguishing between polyneuropathy and mononeuropathy in such cases. If EMG findings confirm sensorimotor axonal polyneuropathy, intravenous or intramuscular vitamin supplementation should be prioritized, as the prognosis is typically worse compared to mononeuropathy.[9]

According to the literature, in cases where a nerve conduction block is detected at the fibular head and clinical improvement is not observed within three weeks, decompression surgery or nerve grafting has been recommended to relieve nerve compression.[10] However, some researchers argue that surgical intervention is unnecessary, and that vitamin supplementation and physiotherapy alone may be sufficient.[11-13] In our case, the patient was managed conservatively for three months, and at the end of the follow-up, both clinical findings and EMG results showed complete resolution.

In addition to physical therapy and vitamin supplementation, systemic corticosteroid therapy was initiated promptly after the diagnosis was confirmed. Although corticosteroids are not part of the standard treatment protocol for all peripheral neuropathies, they have been empirically used in selected cases. In noninflammatory entrapment neuropathies, such as peroneal neuropathy, recent studies suggest that corticosteroids may help reduce nerve edema, stabilize the blood-nerve barrier, and promote early

nerve regeneration. A systematic review by Couch et al.[14] demonstrated that corticosteroids may play a neuroprotective and regenerative role in both compressive and noncompressive neuropathies. Similarly, Wang et al.[15] showed that corticosteroids accelerated both motor and sensory recovery in iatrogenic nerve injuries.

In conclusion, peroneal neuropathy due to rapid weight loss following bariatric surgery is a rare but clinically significant complication that can substantially impair patients' quality of life. This case highlights the critical importance of early diagnosis and the effectiveness of conservative treatment strategies, including vitamin supplementation, corticosteroid therapy, and structured physical rehabilitation. Complete functional and electrophysiological recovery was achieved within three months through timely and individualized management. Although similar cases have been reported in the literature, this case is particularly noteworthy for the rapid and complete recovery achieved through a well-structured conservative approach that included corticosteroids. The early diagnosis, timely initiation of multimodal conservative treatment, and detailed documentation of follow-up outcomes highlight the clinical value of this case report and reinforce the importance of individualized, nonsurgical management strategies in bariatric patients at risk of peroneal neuropathy.

Acknowledgment: The authors acknowledge the use of ChatGPT (OpenAI, San Francisco, CA, USA) to improve the readability and language quality of the manuscript during the writing process. This technology was used under human supervision and control, and the final content was thoroughly reviewed and edited by the authors to ensure accuracy, originality, and adherence to ethical standards. No part of the data analysis or scientific interpretation was conducted using AI tools.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, design: D.F.; Analysis and/or interpretation, critical review, literature review: D.F., E.S.Ö.; Writing the article: D.F, Ü.Y.; Control/supervision: Ü.Y.; Data collection and/or processing: E.S.Ö.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Holanda N, Crispim N, Carlos I, Moura T, Nóbrega E, Bandeira F. Musculoskeletal effects of obesity and bariatric surgery - a narrative review. Arch Endocrinol Metab 2022;66:621-32. doi: 10.20945/2359-3997000000551.
2. Koffman BM, Greenfield LJ, Ali II, Pirzada NA. Neurologic complications after surgery for obesity. Muscle Nerve 2006;33:166-76. doi: 10.1002/mus.20394.
3. Bowley MP, Doughty CT. Entrapment neuropathies of the lower extremity. Med Clin North Am 2019;103:371-82. doi: 10.1016/j.mcna.2018.10.013.
4. Şen O, Karaca FC, Türkçapar A. Neurological complication after laparoscopic sleeve gastrectomy: Foot drop. Obes Surg 2020;30:957-60. doi: 10.1007/s11695-019-04285-6.
5. Weyns FJ, Beckers F, Vanormelingen L, Vandersteen M, Niville E. Foot drop as a complication of weight loss after bariatric surgery: Is it preventable? Obes Surg 2007;17:1209-12. doi: 10.1007/s11695-007-9203-2.
6. Thaisetthawatkul P, Collazo-Clavell ML, Sarr MG, Norell JE, Dyck PJ. A controlled study of peripheral neuropathy after bariatric surgery. Neurology 2004;63:1462-70. doi: 10.1212/01.wnl.0000142038.43946.06.
7. Zhen C, Bautista T, Shabbir A. Peroneal palsy after bariatric surgery. Saudi J Laparosc 2016;1:9-12.
8. Aghili R, Malek M, Tanha K, Mottaghi A. The effect of bariatric surgery on peripheral polyneuropathy: A systematic review and meta-analysis. Obes Surg 2019;29:3010-20. doi: 10.1007/s11695-019-04004-1.
9. Lin IC, Lin YL. Peripheral polyneuropathy after bariatric surgery for morbid obesity. J Family Community Med 2011;18:162-4. doi: 10.4103/2230-8229.90020.
10. Milants C, Lempereur S, Dubuisson A. Bilateral peroneal neuropathy following bariatric surgery. Neurochirurgie 2013;59:50-2. doi: 10.1016/j.neuchi.2012.09.004.
11. Karadag YS, Saka M, Oztekin N, Ak F. An underestimated complication of obesity management: bilateral peroneal neuropathy. J Neurol Sci Turk 2013;30:801-4.
12. Prado MP. Developed foot drop as a complication of bariatric surgery: Case reports. Einstein (Sao Paulo) 2010;8:232-4. doi: 10.1590/S1679-45082010RC1474.
13. Ramos-Leví AM, Matías-Guiu JA, Guerrero A, Sánchez-Pernaute A, Rubio MA. Peroneal palsy after bariatric surgery; is nerve decompresion always necessary? Nutr Hosp 2013;28:1330-2. doi: 10.3305/nh.2013.28.4.6495.
14. Couch B, Hayward D, Baum G, Sakthiyendran NA, Harder J, Hernandez EJ, et al. A systematic review of steroid use in peripheral nerve pathologies and treatment. Front Neurol 2024;15:1434429. doi: 10.3389/fneur.2024.1434429.
15. Wang T, Zhao J, Yuan D. Successful surgical management of a ruptured popliteal artery aneurysm with acute common peroneal nerve neuropathy: A rare case. Vascular 2021;29:256-9. doi: 10.1177/1708538120950870.