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Case Report

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Unilateral foot drop following bariatric surgery: a case report

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ABSTRACT

Neurological complications following bariatric surgery (BS) are uncommon but may cause significant functional impairment if not recognized early. We report the case of a 38-year-old female who developed sudden-onset right foot drop seven months after BS, following an approximately 30-kg weight loss. Neurological examination demonstrated severe weakness of ankle dorsiflexion and foot eversion without sensory deficits. Electrophysiological studies revealed an acute-to-subacute partial demyelinating lesion of the common peroneal nerve at the fibular head with evidence of secondary axonal regeneration, whereas laboratory and imaging studies excluded metabolic and structural causes. The patient underwent a comprehensive rehabilitation program, including therapeutic exercises, neuromuscular electrical stimulation, gait training, and an ankle-foot orthosis, resulting in marked functional improvement after 15 treatment sessions. This case highlights rapid weight loss as an important risk factor for common peroneal neuropathy after BS and emphasizes the value of early electrophysiological evaluation and multidisciplinary rehabilitation in optimizing functional recovery.

Keywords: Bariatric surgery, foot drop, common peroneal neuropathy, rapid weight loss, rehabilitation

INTRODUCTION

Obesity is a chronic disease with an increasing global prevalence and is associated with numerous comorbidities, including cardiovascular disease, type 2 diabetes mellitus, hypertension, dyslipidemia, and musculoskeletal disorders.[1] Bariatric surgery (BS) is considered one of the most effective treatment options for patients with morbid obesity or inadequate weight loss despite conservative treatment, providing sustained weight reduction and improvement of obesity-related complications.[2]

Neurological complications following BS are uncommon but may cause significant functional impairment. Peripheral neuropathies are among the most frequently reported neurological complications and are associated with rapid weight loss, micronutrient deficiencies, malabsorption, inadequate nutritional intake, and intraoperative positional nerve compression.[3]

Foot drop after BS is an uncommon manifestation, most commonly resulting from common peroneal nerve compression at the fibular head due to the loss of protective adipose tissue following rapid weight loss. Prolonged surgical positioning and nutritional deficiencies may also contribute to its development.[4] If not recognized promptly, foot drop may lead to persistent motor deficits and impaired quality of life. Early neurological assessment, electrophysiological evaluation, nutritional assessment, and appropriate rehabilitation are essential for improving functional outcomes.[5]

Herein, we present a case of unilateral foot drop following BS and discuss its clinical presentation, possible pathophysiological mechanisms, diagnostic evaluation, and management in light of the current literature.

CASE REPORT

A 38-year-old female presented to the outpatient physical medicine and rehabilitation clinic with sudden-onset right foot drop that had progressively worsened over two weeks. She denied pain, numbness, paresthesia, recent trauma, or mechanical risk factors for common peroneal nerve compression. Her medical history was unremarkable except for BS performed seven months earlier for morbid obesity, after which she had lost approximately 30 kg.

Neurological examination revealed severe weakness of right ankle dorsiflexion, foot eversion, and great toe dorsiflexion (Medical Research Council [MRC] grade 1/5), while plantar flexion, sensory examination, and deep tendon reflexes were normal. Laboratory investigations, including serum glucose, thyroid function tests, vitamin B12, and folate levels, were within normal limits. Ultrasonography of the sciatic and peroneal nerves and magnetic resonance imaging of the right knee and lower leg showed no structural abnormalities or evidence of nerve entrapment. Muscle strength in the remaining lower-extremity muscle groups was normal (MRC grade 5/5).

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Electrophysiological studies demonstrated a partial demyelinating lesion of the right common peroneal nerve at the fibular head with evidence of secondary axonal regeneration, consistent with acute-to-subacute peroneal neuropathy. Based on the clinical history and diagnostic findings, rapid weight loss following BS was considered the most likely etiological factor. More specifically, right peroneal motor nerve conduction demonstrated a marked reduction in response amplitude proximal to the fibular head compared with the distal stimulation site, together with reduced conduction velocity and prolonged latency. Needle electromyography of the right tibialis anterior and peroneus longus muscles revealed 2+ positive sharp waves and fibrillation potentials at rest. During voluntary contraction, prolonged-duration, polyphasic giant motor unit potentials and a reduced recruitment pattern were observed. These findings were interpreted as a partial lesion of the right common peroneal nerve proximal to the fibular head, predominantly demyelinating in nature, with secondary axonal regeneration in the acute-to-subacute stage. Quantitative nerve conduction values were not available in the supplied report.

The patient underwent a comprehensive rehabilitation program consisting of therapeutic exercises, neuromuscular electrical stimulation, gait training, and an ankle-foot orthosis. After 15 treatment sessions, ankle dorsiflexion and great toe extension improved to MRC grade 3/5, with marked functional improvement in gait. The patient remains under regular clinical and rehabilitation follow-up. Rehabilitation was delivered five days per week for a total of 15 sessions. Treatment included ankle and foot range-of-motion exercises, strengthening exercises targeting the muscles innervated by the peroneal nerve (particularly the ankle dorsiflexors and evertors), neuromuscular electrical stimulation applied to the ankle dorsiflexors and evertors, and gait training. An ankle-foot orthosis (AFO) was used during physiotherapy and during activities of daily living, including ambulation. Following treatment, ankle eversion strength also improved from MRC grade 1/5 to 3/5.

DISCUSSION

Peroneal neuropathy is one of the most common mononeuropathies of the lower extremity and is particularly vulnerable to compression at the fibular head because of the superficial course of the common peroneal nerve.[6] Clinically, it presents with foot drop due to weakness of ankle dorsiflexion and foot eversion, while sensory symptoms may be absent in cases with predominantly motor involvement.[6,7] In our patient, the absence of pain and sensory deficits was consistent with the electrophysiological findings.

Common causes of peroneal neuropathy include trauma, knee surgery, prolonged external compression, and rapid weight loss.[7,8] In the present case, the absence of trauma, mechanical risk factors, or structural abnormalities on imaging suggested rapid postoperative weight loss as the most likely cause.

Neurological complications occur in approximately 2%-16% of patients after BS and include metabolic and compression neuropathies.[9-11] Rapid weight loss may predispose patients to common peroneal neuropathy by reducing the protective adipose tissue around the fibular head, thereby increasing susceptibility to external compression.[4,7,12,13] Our patient developed symptoms seven months after surgery following a 30-kg weight loss, consistent with the typical onset reported in the literature.[4,14] Although vitamin deficiencies are recognized causes of peripheral neuropathy after BS, normal serum vitamin levels do not exclude neuropathy, as rapid weight loss alone may contribute to nerve compression.[4,10,15] The spectrum of neurological complications after BS extends beyond isolated unilateral common peroneal mononeuropathy. Reported peripheral nervous system manifestations include bilateral peroneal neuropathy, generalized polyneuropathy, and less commonly plexopathy or radiculopathy; central neurological syndromes associated with nutritional deficiencies have also been described.[3,5,9-11,15-17] These presentations may differ in their distribution, mechanism, and time course. Nutritional and metabolic factors may contribute to more diffuse neuropathic presentations, whereas loss of protective adipose tissue and focal mechanical vulnerability may favor compressive mononeuropathy. In contrast to bilateral or generalized involvement, our patient had an isolated unilateral motor-predominant deficit, with electrodiagnostic localization to the right common peroneal nerve at the fibular head. This distinction highlights the importance of considering the full postoperative neurological differential while using the pattern of weakness and electrophysiological findings to establish localization.

Electrophysiological evaluation is the gold standard for confirming the diagnosis and determining lesion severity.[7] In our patient, nerve conduction studies and needle electromyography demonstrated an acute-to-subacute partial common peroneal nerve lesion with secondary axonal regeneration, consistent with previous reports.[12,14] Differential diagnoses, including L5 radiculopathy, sciatic neuropathy, central nervous system disorders, and hereditary or vasculitic neuropathies, were excluded based on clinical, imaging, and electrophysiological findings.[7,18]

Early rehabilitation, including an ankle-foot orthosis, strengthening exercises, neuromuscular electrical stimulation, and gait training, plays a key role in functional recovery.[7,8,17,19] In our patient, muscle strength improved from MRC grade 1/5 to 3/5 after 15 rehabilitation sessions, highlighting the importance of early diagnosis and rehabilitation in patients with foot drop following BS. In addition to ankle dorsiflexion and great toe extension, ankle eversion improved from MRC grade 1/5 to 3/5. The rehabilitation schedule comprised five sessions per week, with range-of-motion and targeted strengthening exercises, electrical stimulation of the ankle dorsiflexors and evertors, and gait training. The AFO supported mobility both during treatment and in daily activities.

CONCLUSION

Rapid weight loss following BS may increase the risk of common peroneal neuropathy, even in patients with normal serum vitamin levels. Early electrophysiological evaluation and timely multidisciplinary rehabilitation are essential for improving functional outcomes in patients presenting with foot drop after BS.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

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Author Contributions

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Editor Note

Ayşe Gülşen Doğan is a member of the Editorial Board of this journal. She was not involved in the editor assignment, reviewer selection, peer review process, or editorial decision for this manuscript. The peer review process was managed independently by another editor.

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