Case Report

Post-bariatric Guillain-Barré syndrome: a case report emphasizing timely recognition and intervention

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Background: Guillain-Barré syndrome (GBS), a heterogeneous group of immune-mediated peripheral neuropathies, is frequently linked to antecedent gastroenteritis or respiratory tract infections, serving as a notable etiology for the onset of acute flaccid symmetrical limb weakness accompanied by areflexia.

Case Description: This comprehensive case study meticulously documents the clinical trajectory of a 27-year-old female who, following laparoscopic sleeve gastrectomy to address obesity, progressively developed ascending symmetrical limb weakness and sensory perturbations in both lower extremities. Ultimately, this deterioration led to a profound inability to ambulate autonomously. Upon subsequent diagnostic evaluation, the presence of GBS of the axonal variant was confirmed. The therapeutic approach adopted in this case involved the initiation of intravenous immunoglobulin (IVIG) therapy, resulting in substantial and noteworthy clinical recovery. The successful outcome highlights the efficacy of timely and targeted interventions in mitigating the debilitating effects of GBS.

Conclusions: This illustrative case underscores the paramount importance of expeditious recognition and diagnostic acumen in the post-bariatric surgical milieu when GBS is suspected. The timely identification of GBS and the swift initiation of appropriate interventions are pivotal in optimizing patient outcomes and preventing the progression of potentially life-threatening sequelae. By shedding light on the intricacies of this clinical scenario, this case study contributes valuable insights into the nuanced management of GBS in the context of postoperative care, emphasizing the significance of a multidisciplinary approach to enhance patient well-being.

Keywords: Guillain-Barré syndrome (GBS); bariatric surgery; sleeve gastrectomy; case report; nutritional deficiency

Received: 23 November 2023; Accepted: 28 December 2023; Published online: 07 March 2024.

doi: 10.21037/jeccm-23-142

View this article at: https://dx.doi.org/10.21037/jeccm-23-142

Introduction

Guillain-Barré syndrome (GBS) represents an acute inflammatory demyelinating polyneuropathy typified by its hallmark features of areflexia and rapidly progressive symmetrical muscle weakness that involves all four limbs. The precise pathophysiological underpinnings remain elusive, although autoimmunity incited by viral or bacterial infections, or even surgical interventions, is postulated (1). Laparoscopic sleeve gastrectomy, a routinely performed

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bariatric procedure known for its commendable safety profile, may be complicated by nutritional deficits due to dietary non-compliance, thereby fostering neurological sequelae (2). This report documents the emergence of GBS a mere 2 months following laparoscopic sleeve gastrectomy. We present this case in accordance with the CARE reporting checklist (available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-142/rc).

Case presentation

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

A 27-year-old female with a pre-existing condition of obesity, characterized by a body mass index (BMI) of 36 kg/m^2, underwent laparoscopic sleeve gastrectomy in July 2022. The surgical procedure and immediate postoperative phase proceeded uneventfully. Subsequent observations revealed lapses in adherence to the recommended dietary regimen and prescribed vitamin supplementation.

Two months following surgery, she exhibited the gradual onset of ascending symmetrical limb weakness, accompanied by alterations in sensory perception within both lower extremities. Within a span of days, her condition deteriorated, culminating in instability and a reliance on external support for ambulation. Over the ensuing fortnight, the upper limbs became progressively engaged by the malady. Concomitant clinical manifestations encompassed diplopia, dysphagia, dyspnea, palpitations, fatigue, nausea, vomiting, dizziness, headache, and disrupted sleep patterns. The patient denied a recent history of travel, vaccination, familial antecedents of similar clinical presentations, illicit drug utilization, fever, or antecedent upper respiratory tract infections. Moreover, she had no prior occurrences of comparable symptomatology.

Upon physical examination, notable weaknesses were identified in both upper and lower limbs, with a conspicuous preponderance of distal muscle weakness. Her upper limb distal muscle power was graded at 4/5, while her lower limb distal muscle power stood at 3/5, concomitant with sensory impairment in these distal areas. The cerebellar and cranial nerve examination were normal. Her biochemical tests are listed below in *Table 1*.

Cerebrospinal fluid (CSF) analysis, performed on the first day of symptom onset, yielded unremarkable results. Neuroimaging investigations, inclusive of computed tomography (CT) brain and MRI of the brain and spine, yielded no anomalies. Nerve conduction velocity (NCV) and electromyography (EMG) examinations disclosed bilateral neuropathy, principally of the axonal variety, affecting both upper and lower limbs. Synthesizing the compendium of clinical and neurophysiological data, the definitive diagnosis of axonal GBS was arrived at.

Therapeutic intervention involved the prompt initiation of intravenous immunoglobulin (IVIG) therapy at the recommended standard dosage of 0.4 grams per kilogram per day, administered over a 5-day period. This course of treatment precipitated tangible enhancement in motor function of the upper limbs. Concurrently, physiotherapy was integrated into a holistic care regimen. Following 1 month of such care, the patient evinced remarkable improvements in both upper and lower limb functionality, culminating in her discharge to a rehabilitation facility, where she would receive continued high-dose IVIG therapy and rigorous physical rehabilitation.

Patients perspective

The critical turning point in my battle against GBS was the swift initiation of IVIG therapy. This marked the beginning

Highlight box

Key findings

- This case report highlights the importance of the timely identification and intervention in optimizing patient outcomes and averting the evolution of potentially life-threatening sequelae.

What is known and what is new?

- Guillain-Barré syndrome (GBS) is a very important cause of post-inflammatory purely motor ascending paralysis that mimics a variety of conditions.
- Post-bariatric GBS is a rare but exceedingly important consequence of bariatric surgeries which, if identified early, can dramatically decrease the mortality and morbidity associated with the disorder.

What is the implication, and what should change now?

- A heightened awareness regarding the potential diagnosis of GBS, with due diligence in excluding close differentials, is crucial for fostering timely identification and the implementation of therapeutic interventions to mitigate this debilitating complication.

Table 1 Results of the laboratory investigations

Test	Result	Normal range
Hemoglobin (g/dL)	12.5	12.0–16.0
WBC count (×10^9/L)	8.69	4.5–11.0
Blood glucose (mmol/L)	6.6	3.9–5.5
Potassium (mmol/L)	3.16	3.5–5.0
Creatinine (μmol/L)	44	60–110
Albumin (g/L)	3.18	3.9–4.9
Calcium (mmol/L)	2.34	2.2–2.6
Magnesium (mg/dL)	0.91	1.7–2.3
TSH (μIU/mL)	2.07	0.4–4.0
25-OH vitamin D (nmol/L)	81.9	30–100
CRP (mg/L)	27.4	0–5
ESR (mm/h)	20	0–20
IgG (g/L)	18.10	6.0–16.0
Lipase (U/L)	167	10–140
ALP (U/L)	38.18	42–144
RBC (×10^{12}/L)	4.37	4.5–5.5
Hct (%)	36.8	35.0–45.0
Vitamin B12 (pg/mL)	891	160–950

WBC, white blood cell; TSH, thyroid-stimulating hormone; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IgG, immunoglobulin G; ALP, alkaline phosphatase; RBC, red blood cell; Hct, hematocrit.

of a transformative process that significantly enhanced the motor function of my upper and lower limbs.

The integration of physiotherapy into my holistic care regimen played a crucial role in complementing the effects of IVIG therapy. The comprehensive approach, addressing both medical and rehabilitative aspects concurrently, contributed to the substantial improvements I experienced over the course of 1 month. The positive changes were particularly evident in the functionality of both my upper and lower limbs. After a month of dedicated care, I reached a significant milestone in my recovery journey—my discharge to a rehabilitation facility. At present, I am able to walk independently without any assistance and am in good health.

Discussion

In contemporary times, the popularity of bariatric surgeries has ascended substantially. These procedures are celebrated for their effectiveness and relative safety; however, they are not without their associated risks and potential complications. Neurological complications following bariatric surgery are estimated to afflict approximately 5% to 10% of patients (3).

Laparoscopic sleeve gastrectomy, recognized for its low morbidity and mortality rates, has gained prominence as a bariatric surgical technique. This procedure entails the surgical excision of a portion of the stomach, culminating in a reduction in its capacity, thereby constraining food intake and facilitating weight reduction. Despite the overall safety profile of laparoscopic sleeve gastrectomy, it does carry inherent risks, including the propensity for nutritional deficiencies resultant from dietary non-adherence. Such deficits have the potential to precipitate neurological complications, with GBS emerging as a rare yet rapidly progressive neurological sequelae of bariatric surgery (4).

GBS is an encompassing term denoting immune-mediated peripheral neuropathies that tend to manifest subsequent to episodes of gastroenteritis or respiratory tract infections. Patients grappling with GBS typically present with insidiously progressive muscular weakness, paresthesia, and a loss of reflexes. Weakness tends to originate within the distal lower extremities before ascending proximally (1). The diagnosis of GBS hinges on clinical features, corroborated by laboratory assessments and imaging studies. EMG and NCV investigations hold salience in the diagnostic discourse (5). The therapeutic approach to GBS revolves around a foundation of supportive care, with specific therapeutic modalities such as IVIG garnering consideration for moderate to severe cases. IVIG therapy enjoys the status of first-line treatment, though alternative options like plasmapheresis and corticosteroids have found utility (4). The clinical course of GBS mandates close vigilance, given the potential requirement for mechanical ventilation in nearly 24% of afflicted patients and the somber reality that up to 12% succumb to disease-related complications (5). Extensive investigations have explored the spectrum of axonal and demyelinating variants of GBS, informing their respective management trajectories. It is noteworthy that appropriate interventions foster complete recovery in approximately 85% of cases, with the remaining 15% potentially enduring varying degrees of sequela (5).

The specter of GBS looms prominently within the context of bariatric surgery, a notion substantiated by the extant medical literature. GBS-related bariatric surgery complications may materialize within a temporal window

extending from a mere few weeks to as long as 2 years following the index surgical event. The etiology of such post-surgical GBS remains a subject of conjecture, with speculations pertaining to the potential predisposition of these patients to autoimmune processes or latent malignancies. Furthermore, both general anesthesia and conscious sedation modalities employed during surgery have been proffered as potential triggers for peripheral radiculopathy (6). While the definitive establishment of a causal relationship remains elusive in the cases presented, the collective repository of clinical experiences underscores the imperative obligation to apprise patients deliberating bariatric surgery of the plausible risk of GBS. The data from sural nerve biopsies showed inflammatory cell infiltrates in patients with acute or subacute neuropathies or radiculo-plexo-neuropathies post-bariatric surgery. The suggested pathogenesis was based on the findings of axonal degeneration rather than demyelination, and normal CSF including protein levels. In addition, over 50% of these patients had an unknown cause of their poly-neuropathy and developed residual weakness despite vitamin supplementation (6). Therefore, further studies are warranted to reveal underlying pathophysiology and pathogenesis of GBS after bariatric surgeries.

It is worthy of note that a close differential diagnosis for post-surgical GBS encompasses neuropathies consequent to nutritional deficits, a realm to which vitamin B12, thiamine, and vitamin D deficiencies are chief contributors (7). Of these, vitamin B12 deficiency stands out as an entity capable of culminating in peripheral nerve demyelination, mirroring the clinical presentation of GBS. Thiamine deficiency portends the peril of Wernicke's encephalopathy, characterized by ataxia, ophthalmoplegia, and cognitive impairment. Vitamin D deficiency, in turn, has been implicated in the genesis of myopathy and muscle weakness (8).

In this specific case, we highlight the presentation of post-inflammatory demyelinating or axonal neuropathy, a clinical entity distinct from post-surgical neuropathy arising secondary to nutritional deficiencies. The patient's clinical profile was notably devoid of abnormalities in laboratory findings and nutritional assessments, effectively discrediting nutritional deficiencies as the precipitant of her condition. CSF analysis yielded unremarkable results. However, neurophysiological evaluation in the form of NCV studies, conjoined with her clinical symptomatology, converged in favor of a diagnosis of GBS. The subsequent clinical response to IVIG therapy provided further substantiation for this diagnostic conclusion.

Upon a comprehensive review of the existing medical literature, we identified a paucity of reported cases of GBS following bariatric surgery, with the majority of these cases showcasing an axonal form of GBS. One such illustrative case presented by Ishaque *et al.* involved a patient who experienced the acute onset of progressive weakness and paresthesia in all four limbs within 6 weeks following laparoscopic sleeve gastrectomy. The clinical and neurophysiological findings in this instance were suggestive of a demyelinating variant of GBS, prompting the initiation of IVIG therapy (8).

A parallel case reported by Najjari *et al.* encapsulated a scenario of demyelinating GBS wherein a patient developed GBS just 1 day after undergoing gastrostomy. The patient demonstrated positive EMG and NCV findings, achieving discernible improvement with IVIG treatment (7).

In an analogous vein, Nadia *et al.* delineated a case characterized by progressive lower limb weakness emerging 2 months post-laparoscopic sleeve gastrectomy. This patient was diagnosed with the acute motor axonal neuropathy (AMAN) variant of GBS, predicated on both clinical and neurophysiological parameters. The patient commenced treatment with IVIG but, unfortunately, succumbed to complications arising from paralytic ileus secondary to peritonitis (9).

A compelling parallel was evident in a case depicted by Aljthalin *et al.*, whereby a patient presented with progressive bilateral lower limb weakness 4 months subsequent to distal gastrectomy featuring gastroduodenal anastomosis. Diverging from our case, CSF analysis in this instance unveiled elevated protein levels with a normal cell count. Neurophysiological findings remained consonant with the AMAN variant of GBS. With the administration of IVIG treatment and concurrent rehabilitation, the patient achieved a trajectory of recovery (6).

Landais *et al.* contributed a case encapsulating lower limb paresis emerging 5 months following gastric bypass surgery. Laboratory assessments in this scenario disclosed deficiencies of vitamin B9 and B12. Furthermore, the patient displayed elevated levels of anti-gangliosides, anti-GM1, and anti-GD1a antibodies. Neurophysiological findings alluded to the AMAN variant. IVIG treatment engendered a rapid convalescence in this particular case (10).

Conclusions

GBS, though rare, stands as a potential consequence

of bariatric surgery, a phenomenon accentuated by its increasing prevalence with the escalating popularity of these surgical interventions. While the capacity to definitively ascribe causality between bariatric surgery and post-surgical GBS remains enigmatic, the cumulative body of scientific literature advocates for the conscientious consideration of GBS as a plausible risk in the context of patient counseling for bariatric surgery. A heightened awareness regarding the potential diagnosis of GBS, with due diligence in excluding close differentials, is crucial for fostering timely identification and the implementation of therapeutic interventions to mitigate this debilitating complication.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-142/rc

Peer Review File: Available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-142/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-142/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

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doi: 10.21037/jeccm-23-142
Cite this article as: Mahwish N, Omara EIM, Rangraze IR, Al Qaeidy A. Post-bariatric Guillain-Barré syndrome: a case report emphasizing timely recognition and intervention. J Emerg Crit Care Med 2024;8:5.