Aljthalin and Al-Attas
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CASE REPORT Open Access

Guillain–Barré syndrome variants: a grave complication of bariatric surgery

Raseel A. Aljthalin and Alawi A. Al-Attas*

Abstract

Background: Guillain–Barré syndrome is an acquired inflammatory disease of the peripheral nervous system. It is the most frequent cause of acute flaccid symmetrical weakness of the limbs and areflexia usually reaching its peak within a month.

Case presentation: Herein we report two post-bariatric surgery cases that presented with clinical pictures of Guillain–Barré syndrome based on their clinical and electrophysiological findings. Both patients were treated with intravenous immunoglobulin for 5 days. Both patients showed significant improvement and full recovery.

Conclusion: Guillain–Barré syndrome is a possible grave complication of bariatric surgery which needs prompt recognition and intervention.

Keywords: Guillain–Barré syndrome, Bariatric surgery, Nerve conduction studies, Intravenous immunoglobulin, Acute axonal sensory motor neuropathy

Background

Guillain–Barré syndrome (GBS) is an acquired inflammatory disease of the peripheral nervous system and the most frequent cause of acute flaccid symmetrical weakness of the limbs and areflexia usually reaching its peak within a month [1, 2]. It occurs after an infectious disease in which the immune response generates antibodies that cross-react with gangliosides at nerve membranes [3]. The diagnosis of GBS is based on the patient history and neurological, electrophysiological, and cerebrospinal fluid (CSF) examinations. Disease progression can be rapid, and most patients with GBS reach their maximum disability within 2 weeks [1]. Intravenous immunoglobulin (IVIg) and plasma exchange are proven effective treatments for GBS [3]. Herein we report two cases of young patients that underwent bariatric surgery who then presented with features of GBS.

*Correspondence: alawiattas@yahoo.com

Department of Adult Neurology, Prince Sultan Military Medical City, P.O. Box 59046, Riyadh 11525, Saudi Arabia

Case presentation

Case 1

Case 1 was a 21-year-old male smoker with morbid obesity status and post-distal gastrectomy with gastroduodenal anastomosis 4 months prior to the presentation.

His initial body mass index (BMI) and body weight were 62.1 kg/m^2 and 202 kg, respectively. However, 4 months postoperatively his BMI and body weight were 47.7 kg/m^2 and 155 kg, respectively. The patient presented with bilateral lower limb weakness involving mainly bilateral feet associated with numbness and burning sensation over the planter aspect of both feet. He also reported that he could not maintain balance for about a month. Two weeks prior to this admission, he presented to the emergency room with the same complaint, but it was much milder. He denied history of recent travel, raw milk ingestion, vaccination, family history of the same presentation, illicit drug use, fever, or history of upper respiratory tract infection. On further questioning, he denied any gastrointestinal or genitourinary symptoms.

The clinical examination revealed that his vital signs were normal, and his higher mental functions were appropriate for his age. He was awake, alert, and oriented to

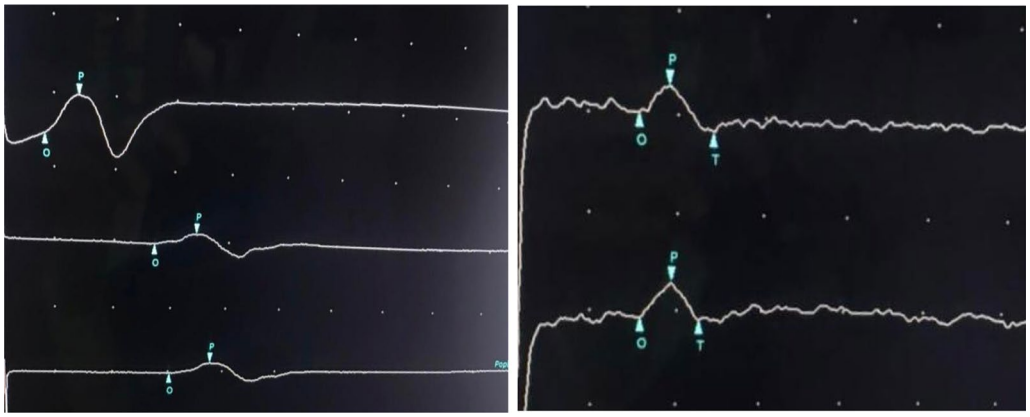

Fig. 1 Low-amplitude CMAP in bilateral tibial and peroneal nerves. Additionally, polyphasic units, a few positive sharp waves and fibrillation potentials, as well as slow wave resting potential, are present. *O*: Onset of the wave; *P*: Peak of the wave; *T*: Terminal part of the wave

time, place, and person. Examination of cranial nerves including fundus examination was normal. Motor examination revealed normal tone, power, and reflexes in both upper limbs. On the other hand, there was hypotonia of bilateral lower limbs, and the power according to the Medical Research Council (MRC) grading was 4 in both lower extremities with bilateral foot drop and weak foot eversion. Both ankle and knee reflexes were absent bilaterally. The sensory examination was intact for proprioception and vibration sense for upper and lower limbs, but there was a decrease in pinprick sensation in the lower limbs bilaterally, and no sensory level was detected. The plantar responses were downgoing bilaterally. The patient was unable to walk or maintain balance without unilateral assistance probably as a result of the lower limb weakness.

The initial diagnosis was subacute combined degeneration of the spinal cord due to recent gastric bypass surgery. However, vitamin levels including vitamin B_{12} and its metabolites like methylmalonic acid and homocysteine, vitamin B_1, vitamin D, and folate were within normal limits.

CSF showed mildly elevated protein, normal cell count, glucose, lactate, and negative culture. Moreover, serological and CSF tests for *Campylobacter jejuni*, cytomegalovirus (CMV), Epstein–Barr virus (EBV), and *Haemophilus influenzae* infection were negative. Furthermore, serum tests for anti-GQ1b and GM1 antibodies were negative.

The nerve conduction study (NCS) and electromyography (EMG) depicted in Fig. 1 showed low-amplitude compound muscle action potential (CMAP) in bilateral tibial and peroneal nerves while it was normal in bilateral median and ulnar nerves. Additionally, polyphasic units, a few positive sharp waves and fibrillation potentials, as well as slow wave resting potential, are present. Latencies and conduction velocities were normal and no conduction block was present in bilateral peroneal and posterior tibial nerves; there was no evidence of demyelination. Sensory nerve action potential (SNAP) was normal for peroneal, sural, ulnar, and median nerves.

The findings of the NCS and EMG were consistent with acute motor axonal neuropathy (AMAN). MRI of the brain and spine was unremarkable.

On the basis of the clinical, neurophysiological, and CSF findings, the case was diagnosed as GBS. The patient completed a 5-day course of IVIg, and he was referred to the rehabilitation center for an extensive rehabilitation program. Two weeks later his power and tone improved significantly, including the foot droop. He was able to walk alone with a cane and able to maintain his balance.

Case 2

Case 2 was a 23-year-old woman who was medically free: her initial BMI and body weight were 39.6 kg/m^2 and 96 kg, respectively; postoperatively her BMI and body weight were 26.71 kg/m^2 and 62 kg, respectively. She underwent laparoscopic sleeve gastrectomy without immediate complications. However, 8 months later she started to have decreased oral intake, difficulty in swallowing, and severe bilateral lower limb numbness with inability to maintain her balance with weakness involving mainly her feet with right foot drop. The weakness progressed rapidly and then ended with intubation due to desaturation and decreased level of consciousness. She was kept on minute ventilation (MV) for 21 days. She had failed one trial of extubation and ended with tracheostomy tube insertion. Prior to the presentation, she gave a history of nausea, vomiting, and diarrhea. She denied history of recent travel, raw milk ingestion, family history of the same presentation or any neurological diseases, no history of illicit drug, head trauma, fever, or history

of upper respiratory tract infection. She denied history of genitourinary symptoms or sphincter loss.

On clinical examination, she was connected to a mechanical ventilator through an endotracheal tube, but she was awake, alert, and responding to commands. Examination of her cranial nerves was normal including fundus examination. Motor examination revealed hypotonia of bilateral lower limbs, but normal upper limbs. Moreover, the power according to MRC grading was 0 in both lower extremities with right foot drop compared to the upper limbs where the power was +3 in the right upper limb and +2 in the left upper limb. Both ankle and knee reflexes were absent bilaterally. The sensory examination was intact for proprioception and vibration sense for upper and lower limbs. Furthermore, there was decreased pinprick sensation in both lower limbs bilaterally, and no sensory level was detected; cerebellar examination was limited because of severe weakness.

Vitamins all were within normal limits, including vitamin B12, and its metabolites methylmalonic acid and homocysteine, vitamins B, D, and B_6, and folate. CSF showed normal cell count, glucose, protein, lactate, and negative culture. Moreover, serological and CSF tests for *C. jejuni*, CMV, EBV, and *H. influenzae* infection were negative. Furthermore, serum tests for anti-GQ1b and GM1 antibodies were negative.

The findings of sensory NCS showed absence of SNAPs for peroneal, sural, ulnar, and median nerves. Her motor NCS revealed absent CMAP for peroneal and tibial nerves; however, for ulnar nerve and median nerve, NCS showed decreased amplitude. Latencies and motor conduction velocities were normal in bilateral peroneal and posterior tibial nerves, without evidence of demyelination. The needle electromyography examination demonstrated markedly abnormal reduction in recruitment and few positive sharp waves and fibrillation potentials were present. F-wave response was absent in the right median and both peroneal nerves; these features were consistent with GBS variant such as acute axonal sensory motor neuropathy (AMSAN). MRI of the brain and spine was unremarkable.

On the basis of the clinical, neurophysiological, and CSF findings, this case was diagnosed as GBS. The patient completed a 5-day course of IVIg. A week later she was disconnected from the mechanical ventilator, but still tracheostomized without ventilator support. She was able to raise bilateral upper and lower limbs against gravity and 2 weeks later against resistance. She was then referred to the rehabilitation center for an extensive rehabilitation program.

Discussion

The neurological complications from bariatric surgery are mainly related to deficiencies in micronutrients secondary to malabsorption after surgery. GBS is a potential delayed complication of bariatric surgery and has been reported in a number of patients who showed improvement after IVIg. Although the mechanism is still not well understood, it could be related to immune and inflammatory processes of neuronal injury, as suggested by data from sural nerve biopsies showing inflammatory cell infiltrates in patients with acute or subacute neuropathies or radiculoplexoneuropathies post bariatric surgery [4]. The suggested pathogenesis was based on the findings of axonal degeneration rather than demyelination, and normal CSF including protein levels [5]. In the literature, there were three similar cases of the axonal form of GBS in Saudi Arabia reported by Sunbol et al. The other form, acute inflammatory demyelinating polyradiculoneuropathy (AIDP) due to bariatric surgery, was reported in Pakistan by Ishaque et al. between 2015 and 2017. Their cases met the National Institutes of Health criteria for bariatric surgery and after a period of a month to 1 year postoperatively, all cases showed excellent response to IVIg with good functional recovery [6, 7]. GBS-related bariatric surgery complications can occur in a period of 6 weeks up to 2 years [5]. In a study done in the USA by Koffman et al., they reported neurological complications in 96 patients, two of whom had GBS-related bariatric surgery similar to our cases [4]. Furthermore, a cross-sectional study conducted in Saudi Arabia previously found that among 451 patients who underwent bariatric surgery, GBS axonal form was found in two patients, one of whom died from massive pulmonary embolism [8].

Conclusions

GBS is a rare, delayed complication post bariatric surgery with unknown mechanism. However, immune and inflammatory processes of neuronal injury could be implicated. Hence, it is recommended to counsel, explain, and follow up such a grave complication.

Abbreviations

AIDP: Acute inflammatory demyelinating polyradiculoneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute axonal sensory motor neuropathy; BMI: Body mass index; CSF: Cerebrospinal fluid; CMAP: Compound muscle action potential; CMV: Cytomegalovirus; EBV: Epstein–Barr virus; EMG: Electromyography; GBS: Guillain–Barré syndrome; *H. influenzae*: *Haemophilus influenzae*; IVIg: Intravenous immunoglobulin; MRC: Medical Research Council; MV: Minute ventilation; NCS: Nerve conduction study; SNAP: Sensory nerve action potential.

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Author contributions
RA: concept behind the work, collecting the case medical history and investigations and writing the manuscript. AA: drafting, editing, and revising the final manuscript, supervising the case medical history and investigations. All authors read and approved the final manuscript.

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Availability of data and materials
The corresponding author takes full responsibility for the data, has full access to all of the data, and has the right to publish any and all data separate and apart from any sponsor.

Declarations

Ethics approval and consent to participate
All procedures performed in the study were in accordance with the ethical standards of the Research and Ethical Committee of the Prince Sultan Military Medical City. However, the case report is exempt from consent.

Consent for publication
Case report is exempt from consent by the research and ethical committee.

Competing interests
The authors declare that there is no competing interest.

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