Peer Review File

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Reviewer A Comments

The authors report a case in which the patient presented GBS 2 months after SG. The literature shows the occurrence of GBS triggered by surgical treatments, but evidently after orthopedic surgeries and mainly in cases of infection, which could trigger the immune response. It is not clear if the authors' hypothesis is regarding the cause and effect of SG and GBS or a simple association. The nutritional deficit would not have a direct association with the triggering of the immunological cascade, even more with just 2 months of surgery that does not cause many vitamin and nutritional deficits. therefore, it seems to us to be an association rather than cause and effect. I suggest adding a limitation paragraph.

Reply - As advised by the respected reviewer, we have added a limitation paragraph number 4

GBS is a rare, delayed complication post bariatric surgery. 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 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, it is important to note that over 50% of the patients had an unknown cause of their poly-neuropathy and developed residual weakness despite vitamin supplementation.
Therefore, further studies need to be done to reveal underlying pathophysiology and pathogenesis of GBS after bariatric surgeries.

Reviewer B Comments

How did you make the diagnosis of GBS? The typical timeline for the disease (<4 weeks) does not match your reported symptoms onset (>8 weeks). Also you reported normal CSF profile (no albuminocytologic dissociation), normal spine MRI (no enhancement of the cauda and conus medullaris). Also very non-specific NCS/EMG findings. You reported diplopia, dysphagia .. etc with normal cranial nerves examination (how would you explain that with normal brain MRI?).

Reply - Paragraph 6 added in the text

The identification of Guillain-Barré Syndrome (GBS) predominantly relies on the characteristic clinical manifestation of sudden-onset ascending motor weakness accompanied by areflexia, and is substantiated by positive results in nerve conduction velocity (NCV), electromyography (EMG), and cerebrospinal fluid (CSF) analyses. In our specific case, the diagnosis of GBS was primarily established through the distinctive clinical features observed, as well as the supportive NCV findings, further reinforced by the patient's swift and positive response to immunoglobulin treatment.

The onset of symptoms in our patient was abrupt, with a usual persistence lasting for a duration of one to two weeks. Our analysis focuses on the initiation and duration of symptoms, independent of the specific timing in relation to the surgical procedure.

The central diagnostic significance of MRI in Guillain-Barré Syndrome (GBS) is not definitively established, as it is not incorporated into the Asbury criteria for GBS diagnosis. Nonetheless, imaging studies are employed to eliminate potential organic lesions affecting the central nervous system (CNS) or peripheral nervous system (PNS), such as transverse myelitis and compressive causes of polyradiculopathy.

In our specific case, a non-contrast MRI was performed to exclude other compressive myelopathies or demyelination in the brain, specifically to rule out conditions such as Multiple Sclerosis. It is essential to note that non-contrast sequences typically yield normal results in such instances.

Diplopia and dysphagia observed in Guillain-Barré Syndrome (GBS) primarily result from muscle weakness rather than any compressive pathologies, thereby leading to predominantly normal findings in MRI for a significant proportion of cases.