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CASE REPORT

From paralytic ileus to Guillain-Barre Syndrome: A diagnostic puzzle

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ABSTRACT

Guillain-Barre Syndrome (GBS) is a rare, acute neuro-immunological disorder that affects the peripheral nerves, characterised by rapid, ascending symmetrical limb weakness and sensory deficits. Autonomic dysfunction is reported in 40-45% of GBS cases, typically manifesting in the later stages. However, it is uncommon for paralytic ileus to be the initial presenting symptom before motor and sensory deficits become apparent. We report a case of GBS in a young man who had paralytic ileus as his primary presenting complaint. The patient was managed supportively but showed minimal improvement. A subsequent neurological assessment revealed proximal myopathy of 4 limbs with generalised areflexia. A nerve conduction study showed electrophysiological evidence of diffuse sensory motor axonal neuropathy affecting the lower limb more than the upper limb. He underwent plasma exchange therapy, in his fourth week of illness. With plasma exchange, his abdominal symptoms resolved, but neurological recovery remained partial. The delayed diagnosis likely affected his outcome, highlighting the importance of early GBS recognition for timely treatment to achieve better recovery.

Keywords: *Paralytic ileus, Guillain-Barre syndrome, Diagnostic puzzle, Peripheral neuropathy, Gastrointestinal motility*

INTRODUCTION

Guillain-Barre Syndrome (GBS) is an immune-mediated neuropathy affecting the peripheral

nerves, typically presenting with ascending weakness and non-length-dependent sensory symptoms. Autonomic dysfunction, which may manifest as cardiac arrhythmias, blood pressure fluctuations, constipation, diarrhoea or ileus, and urinary retention, often occurs in the later stages of GBS when motor and sensory symptoms are already prominent (Bellanti & Rinaldi, 2024).

Paralytic ileus refers to the inability of the bowel to move its contents forward due to impaired motility, without the presence of a mechanical obstruction. Common symptoms include nausea, vomiting, abdominal distension, constipation and inability to pass flatus. This condition can result from various factors, including dysmotility of the bowel musculature, post-abdominopelvic surgery, certain medications, infections, and endocrine metabolic disturbances (Weledeii,2020).

There were literature reports of Guillain-Barre Syndrome (GBS) with predominant autonomic gastrointestinal dysfunction symptoms preceding the onset of motor weakness. Typically, in adult cases, the onset of motor weakness was reported within two weeks of the onset of the gastrointestinal symptoms. In our case, we present a young gentleman who was admitted to our centre with a one-week history of abdominal symptoms and was initially diagnosed with paralytic ileus, in which the initial neurology examination was normal. Subsequently, motor weakness was detected on day 10 of admission (day 17 from the onset of abdominal symptoms). Cerebrospinal fluid analysis and nerve conduction studies were performed and a diagnosis of Guillain-Barre Syndrome was made. In this case, it is suggested that the onset of motor symptoms following paralytic ileus may be varied and may take longer than previously reported. Therefore, it posed significant challenges in achieving the correct diagnosis.

CASE PRESENTATION

A 37-year-old gentleman was admitted to the Emergency Department with a one-week history of constipation, vomiting and intolerance to food. Over the past four days, he developed abdominal distension, and on the day of admission, he began experiencing shortness of breath. He was previously healthy, with no prior weight loss, abdominal surgery or hematochezia. However, he had an upper respiratory tract infection three weeks before admission. On examination, his vital signs were notable for a blood pressure of 118/65mmHg, heart rate of 130 beats per minute, and febrile with a temperature of 38.1 oC. He was tachypnoeic, with a respiratory rate of 34 breaths per minute, and oxygen saturation of 90% on room air. The abdomen was grossly distended with generalised tenderness but without guarding or rebound tenderness. Bowel sounds were sluggish. The lung examination was unremarkable. Muscle strength and tendon reflexes were intact and normal.

Blood investigations showed a white cell count of 25 x 103/uL with neutrophil predominance. Procalcitonin level was elevated at 32.80 ng/mL. He also had hyponatremia (sodium 114 mmol/L), hypokalemia (potassium 2.9 mmol/L) and hypocalcemia (calcium 1.97 mmol/L). Thyroid function tests were normal. All these electrolyte' abnormalities were consistent with gastrointestinal loss (vomiting) and the loss of appetite. Contrast-enhanced computed tomography (CECT) of the abdomen showed diffuse large bowel dilatation, with a maximum diameter of 8.9 cm at the transverse colon, a prominent ileocecal junction, and terminal ileum (Figure 1). There was also short segment dilatation of the ileum, measuring up to 3.9 cm likely due to obstruction, but no mass lesions were identified to account for the obstruction.

DISCUSSION

The patient was diagnosed with pneumonia

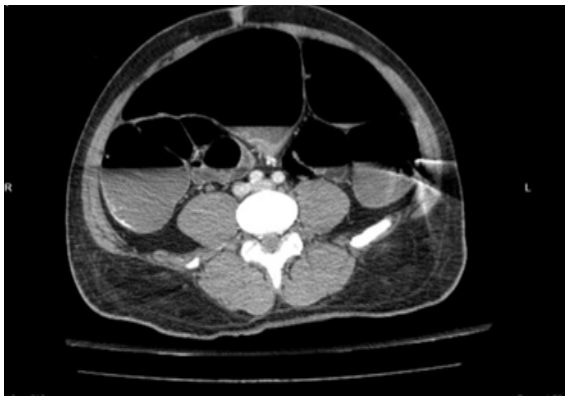

Figure 1: Contrast-enhanced abdominal computed tomography (axial view), arrows show dilated colons with air-fluid levels.

and intra-abdominal sepsis complicated by ileus, precipitated by electrolyte imbalances. He was on a high-flow nasal cannula (HFNC). Abdominal decompression was achieved with a nasogastric tube, and broad-spectrum antibiotics were initiated, along with intravenous fluids and electrolyte correction. Total parenteral nutrition was administered temporarily. The patient's electrolytes were normalised by day four of treatment. Ten days after admission, the patient was able to wean off supplemental oxygen. However, his ileus symptoms only partially resolved; he had minimal bowel output despite regular laxatives, and his abdomen remained mildly distended but non-tender. A colonoscopy was done, revealing a normal large bowel.

The patient was then allowed to eat orally. However, it was observed that he was unable to sit unassisted and fumbled while attempting to use a spoon independently. A neurological examination revealed proximal myopathy of the upper limbs, flaccid paralysis of the lower limbs, reduced sensations, and generalised areflexia. A nerve conduction study showed electrophysiological evidence of diffuse sensorimotor axonal neuropathy, with greater involvement of the lower limbs than the upper limbs. A lumbar puncture was performed, revealing a cerebrospinal fluid (CSF) cell count of 0 and elevated protein at 0.64g/L, suggestive of cytoalbuminologic dissociation. Based on these findings, a diagnosis of Guillain-Barre Syndrome (GBS), an Acute Motor Sensory Axonal Neuropathy (AMSAN) variant, was made. Ileus, tachycardia and fever were the evidence of dysautonomia in this case.

The patient exhibited no facial muscle involvement. Creatine kinase (CK) levels were normal. Both blood and stool cultures were negative. Serum ganglioside antibody testing, including anti-GM1 and anti-GQ1B, returned negative results. Additional tests showed normal levels of vitamin B12 and folate, negative HIV serology and a negative antinuclear antibody (ANA) test.

The patient received plasma exchange therapy for Guillain-Barre Syndrome (GBS) in the fourth week of his illness. A total of five sessions were completed. While this treatment led to the complete resolution of his abdominal symptoms, neurological improvement was only partial. His muscle power showed some progress, with an improvement to a Medical Research Council (MRC) scale of 4 in the upper limbs (previously 3) and 3 in the lower limbs (previously 2). There was no involvement of the respiratory or bulbar muscles, he remained on room air and was able to tolerate feeding. Upon discharge, his mobility was dependent on a wheelchair.

In our case, we were initially considering ileus due to sepsis, electrolyte imbalance and pseudo-obstruction as a result of lack of mobility. However, with standard treatment of ileus, which includes abdominal decompression, electrolytes correction, fluid replacement and total parenteral nutrition, ileus symptoms are only partially resolved. With the completion of the plasma exchange after the diagnosis of Guillain-Barre Syndrome (GBS), the abdominal symptoms completely resolved suggestive of the relation of ileus to Guillain-Barre Syndrome (GBS).

DISCUSSION

Guillain-Barre syndrome (GBS) is an immune-mediated neurological condition that affects the peripheral nerve and causes progressive paralysis of the autonomic, bulbar, and respiratory systems (Bellanti & Rinaldi, 2024).

GBS is classified into four subtypes: acute inflammatory demyelinating polyradiculoneuropathy (AIDP) which accounts for 85% of the cases, followed by, acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and lastly, Miller-Fisher syndrome (MFS) at 5% of the cases (Torres-Vásquez et al., 2020).

In addition to motor weakness, autonomic nervous system involvement can be prominent during GBS. Autonomic manifestations are thought to arise from either failure or overactivity of the sympathetic and parasympathetic systems. These symptoms may include labile blood pressure and heart rates, abnormal hemodynamic responses to medications, sweating and pupillary abnormalities, and bladder or bowel dysfunction. Notably, autonomic involvements have been associated with an increased risk of clinical deterioration, suggesting that these clinical features may indicate more severe disease progression (Chakraborty et al., 2019).

Normal physiology of gastrointestinal motility is regulated by both intrinsic and extrinsic neural control. Extrinsic parasympathetic input to the stomach, small intestine, and much of the colon are mediated by the vagus nerve, while parasympathetic innervation of the distal colon comes from sacral parasympathetic fibres. Sympathetic input to the stomach, small intestine, and colon are provided by the splanchnic and lumbar colonic nerves (Spencer & Hu, 2020).

It is believed that early-onset ileus in GBS results from an imbalance between sympathetic and parasympathetic tone due to immune-mediated damage. This is supported by autopsy findings, which have demonstrated mononuclear inflammatory cell infiltration and demyelination of somatic, parasympathetic, and sympathetic fibers (Shahrizaila et al., 2021).

Paralytic ileus in GBS is typically seen in the advanced stages of the disease. However, it is rarely reported as the initial symptoms before the onset of motor weakness Nowe et al. (2008) first reported a case involving a 74-year-old man who presented with paralytic ileus as the initial symptom, followed by paraparesis 4 days later. There was a similar case of a 34-year-old man, with symptoms of paralytic ileus and only to develop progressive limb weakness 5 days later. (Man & Fu, 2014). There were two severe GBS cases (a 28-month-old girl and a 54-year-old man) reported subsequently, both starting with bowel dysfunction. Both patients eventually required intubation and invasive mechanical ventilation. (Lee et al., 2019; Lee SH & Lee KH, 2017)) All the cases involved an episode of upper respiratory tract infection occurring 5 days to 3 weeks before the onset of GBS symptoms. From the previous literature, the onset of weakness was detected within 2 weeks of ileus symptoms and all were given intravenous immunoglobulin (IVIG) most of them achieved complete resolution of ileus and some degree of neurological recovery except the 54-year-old man who passed on. In our case, the motor weakness was detected on day 10 of admission (17 days after the onset of ileus symptoms) suggesting that the onset of motor weakness following paralytic ileus may be varied and may take longer than previously documented. As for our case, since the diagnosis of GBS is made in the 4 weeks from initial symptoms, plasma exchange therapy was initiated

CONCLUSION

In summary, although paralytic ileus is generally seen in the later stages of GBS, its occurrence as an early isolated symptom is rare but noteworthy. These cases highlighted

the need for clinicians to remain vigilant when encountering unexplained gastrointestinal dysfunction. In such scenarios, especially with a recent history of upper respiratory infection, GBS should be considered as a differential diagnosis to ensure timely and appropriate intervention to prevent irreversible neurological damage.

CONFLICT OF INTEREST

The authors do not have any conflict of interest

CONSENTS

Written informed consent was obtained from the patient to publish the case with its related pictures. A copy of the written consent is available for review by the Editor in Chief.

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