

Paralytic ileus as the presenting symptom for Guillain–Barré syndrome: a case report

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Abstract

Guillain–Barré syndrome (GBS) is an acute neuroimmunological disorder characterized by rapidly ascending symmetrical limb weakness, areflexia, and sensory deficits. Approximately 65% of patients with GBS present with autonomic dysfunction, which commonly occurs in advanced stages. However, paralytic ileus, a sign of gastrointestinal dysautonomia, is rare as the presenting feature in GBS before motor weakness becomes evident. We report the case of a 54-year-old man admitted to the Emergency Department with paralytic ileus as the prodromal feature in early-stage GBS. Total parenteral feeding and prokinetic use were initiated, but no clinical improvement was observed. The patient showed rapid progression to quadriplegia, which was ultimately determined to be respiratory muscle failure requiring mechanical ventilation and intensive care unit admission. He underwent 5 days of intravenous immunoglobulin therapy and muscle strength was partially improved thereafter. However, the patient's enteral nutritional support was undesirable because of persistent poor gastric emptying complicated by fungemia and profound sepsis throughout the hospital course. Finally, he died 1 month after admission. Ignorance of this unusual prodrome to GBS could result in delayed treatment, along with potential progression to life-threatening events. Early recognition of GBS and prompt immunotherapy are critical for reducing morbidity and mortality.

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Keywords

Guillain–Barre Syndrome, intestinal pseudo-obstruction, gastrointestinal motility, autonomic nervous system disease, intravenous immunoglobulin, plasma exchange, radionuclide imaging

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Introduction

Ileus is an occlusion or paralysis of the bowel preventing forward passage of the intestinal content, gas, or liquid, and can be a common reason for presentation to clinicians for care. Common manifestations of ileus include nausea and vomiting, cramps, bloating, and retention of stool and flatus. Ileus may be mechanical or paralytic. Mechanical obstruction can be due to intraluminal, intrinsic to the intestinal wall, and extrinsic obstruction, and it usually requires surgical intervention. Paralytic ileus results from dysmotility of the musculature of the bowel, but no mechanical obstruction, and it has multiple causes, including post-abdominopelvic surgery, pharmacological, infection, and endocrinometabolic factors.¹ The therapeutic goal of this condition is to treat the underlying diseases.

The syndrome of ileus with these various etiopathological and clinical features develops the same, overlappable, and pathophysiological alterations. After initial treatment for ileus and completion of diagnostic evaluation, whether the patient should have surgery performed at once or receive conservative treatment needs to be determined. Appropriate treatment depends on the timely determination of the pathogenesis and on close interdisciplinary collaboration. We describe a case of paralytic ileus as the presenting syndrome for early-stage Guillain–Barré syndrome (GBS) in a 54-year-old male patient.

Case report

A 54-year-old man was admitted to the Emergency Department with a 1-week history of abdominal fullness and obstipation.

He had no history of anticholinergic medications, trauma, or abdominal surgery, but had an upper respiratory infection 3 weeks previously.

On arrival, his vital signs were as follows: temperature, 36.8°C; pulse rate, 104 beats/minute; respiratory rate, 20 breaths/minute; and blood pressure, 138/81 mm Hg. His abdomen was distended, nontender, and tympanitic, without bowel sounds or signs of peritoneal irritation or hepatosplenomegaly. No urinary retention was observed. A neurological examination showed intact muscle strength and normal tendon tap test results. Routine blood and biochemical analyses were unremarkable. Abdominal contrast-enhanced computed tomography (CT) (Figure 1) showed a dilated small intestine and colon with multiple air–fluid levels. Gadolinium-enhanced magnetic resonance imaging of the thoracolumbar spinal cord was performed to rule out the



Figure 1. Contrast-enhanced abdominal computed tomography (axial view) image shows a dilated small bowel and colon loops associated with multiple air–fluid levels. There is no evidence of transitional zones or valvulae conniventes.

possibility of multiple sclerosis or myelopathies, and it showed no anomalies. No contrast passed through the duodenum during a 6-hour small bowel series examination (Figure 2). Gastric emptying scintigraphy (Figure 3) showed markedly delayed radiotracer emptying, which suggested paralytic ileus.

Over 3 days of total parenteral feeding and prokinetic use, the patient showed persistent severe ileus. Development of symmetrical mild paraparesis with medical research council (MRC) grade 4/5, in combination with hypoesthesia (with an impairment of light touch and pinprick over distal limbs, but no sensory level) and loss of deep tendon reflexes in the lower limbs, were observed on hospital day 2. A nerve conduction study on the same day showed demyelinating sensorimotor polyneuropathy, and cerebrospinal fluid analysis showed a normal white blood cell count ($3/\mu\text{L}$; reference: $0\text{--}5/\mu\text{L}$), and high protein

(2030 mg/L ; reference: $150\text{--}450\text{ mg/L}$) and glucose levels (7.77 mmol/L ; reference: $2.22\text{--}3.89\text{ mmol/L}$). Because of the clinical presentation and context, and the lack of electrolyte or acid–base balance abnormalities or other causes for the patient’s condition, we diagnosed him with paralytic ileus secondary to GBS. Immediate treatment with intravenous immunoglobulin (IVIG) 0.4 g/kg daily (from hospital days 2–6) was initiated.

On hospital day 3, the patient’s condition rapidly progressed to involvement of the upper extremities (MRC grade: 4/5) and flaccid quadriplegia (MRC grade: 2/5) was observed on day 4. On day 5, the patient’s condition was ultimately determined to be respiratory muscle failure requiring mechanical ventilation and admission to the intensive care unit. After 5 days of treatment, his muscle strength of the extremities partially improved. The best recovery performance was achieved with an MRC grade of 3/5 on the next day after the IVIG therapeutic

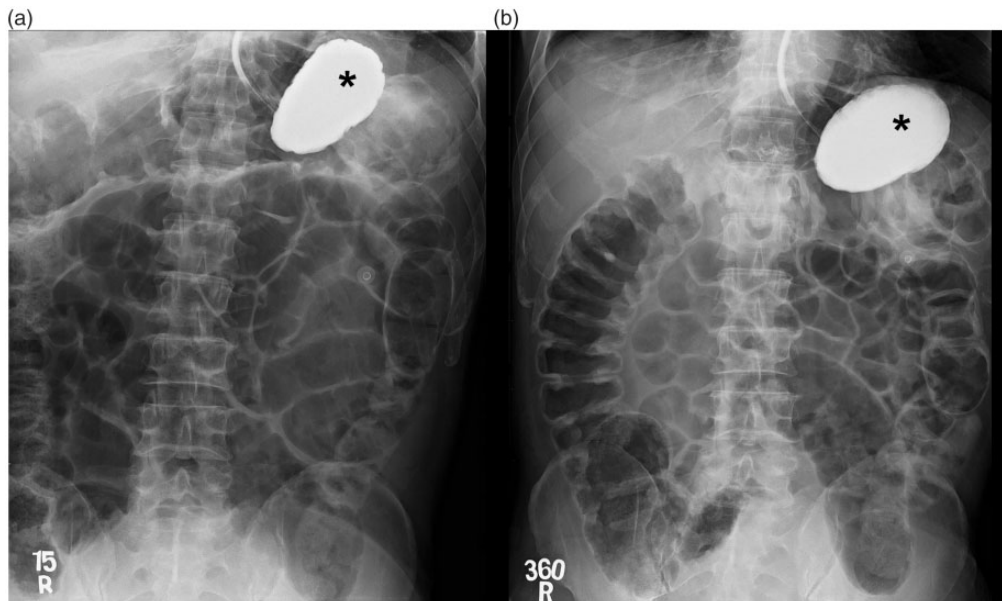


Figure 2. Small bowel series using barium sulfate contrast (asterisks). Views at 15 minutes (a) and 360 minutes (b) show diffuse distention of the small and large bowel loops. No contrast had passed through the duodenum by the end of the 6-hour examination.

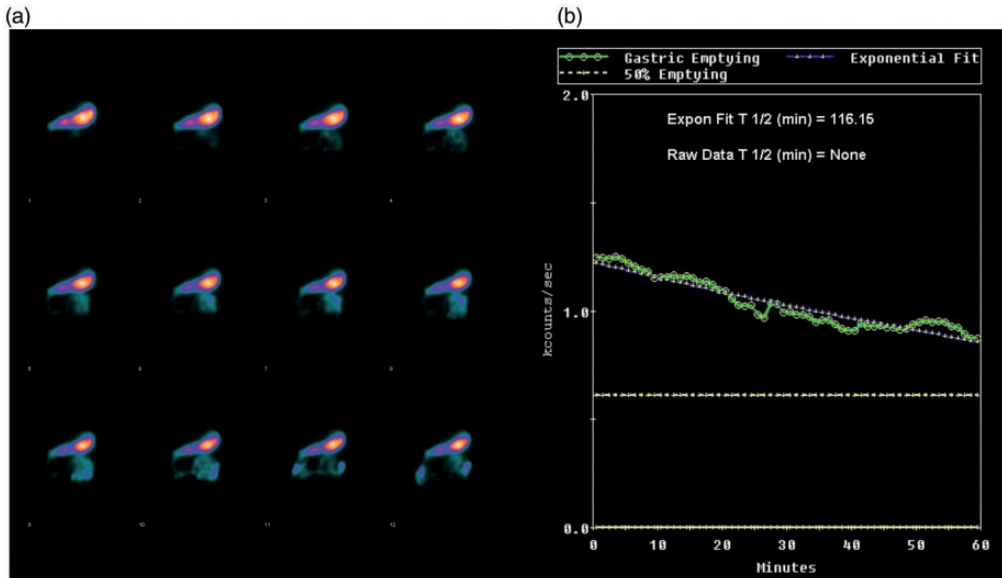


Figure 3. Gastric emptying scintigraphy with Tc-99m diethylene-triamine-pentaacetate solution. (a) Serial static anterior images that were obtained immediately after swallowing the solution (one frame/5 minutes) show radiotracer stasis mainly in the stomach at the end of the 60-minute examination. (b) Time–activity curve of the region of interest plotted for the stomach region shows a considerably delayed gastric emptying time. The half gastric emptying time ($T_{1/2}$) in the exponential fit was 116.15 minutes, which is much longer than the normal reference period (10–20 minutes).

course had finished. However, enteral feeding was undesirable because of persistent gastroparesis complicated by fungemia and profound sepsis throughout the hospital course. Finally, he died 1 month after admission.

Ethics and consent

Local Ethics Committee approval was not necessary because this was a case report and specific information about the patient was not included in the report. Written consent for publication was obtained from the patient's parents.

Discussion

GBS, which was first described by Guillain, Barré, and Strohl in 1916,² is an acute neuroimmunological disorder characterized by rapidly ascending symmetrical limb weakness with areflexia and sensory deficits.

These patients usually present a few days to 1 week after onset of symptoms. The weakness can vary from mild difficulty with walking to nearly complete paralysis of all facial, respiratory, and bulbar muscles, as well as upper and lower extremity muscles. Most patients with GBS reach the nadir within 2 weeks and experience a monophasic disease course without relapse.^{2,3}

GBS is thought to be caused by aberrant immune responses triggered by antigenic stimuli shortly after antecedent infections (often mild respiratory or gastrointestinal illness) or vaccination.⁴ This aberrant response generates autoimmune antibodies against peripheral nerves, thereby resulting in demyelination or axonal damage with subsequent conduction blockage. Diagnosis of GBS is based on the clinical picture, cytoalbuminic dissociation in cerebrospinal fluid, and electrophysiological abnormalities.^{3,4}

Generally, approximately 65% of patients with GBS show dysautonomia, which commonly occurs in advanced stages. This condition can lead to broad systemic consequences, and can manifest as cardiac dysrhythmias, fluctuation in blood pressure, or vasomotor or gastrointestinal dysfunction.^{3,4}

Ileus refers to complete or partial disruption in intestinal transit either by functional (adynamic or paralytic) or mechanical bowel obstruction.¹ In the present case, abdominopelvic CT excluded organic obstruction, and gastrointestinal functional examinations confirmed paralytic ileus. Paralytic ileus is triggered by multiple factors, including postoperative, pharmacological, or endocrinometabolic origins, or as in our case, gastrointestinal dysautonomia secondary to GBS.⁵ Diffuse gastrointestinal dysmotility may result in intestinal dilatation, increased luminal pressure, gut wall ischemia, and intestinal bacterial overgrowth. These conditions may further promote entry of viable microorganisms through an injured intestinal wall into the systemic circulation. Therapeutic goals for ileus include fluid management, early establishment of enteral nutrition, and approaches to treat the underlying causes.⁶

Paralytic ileus in GBS is mainly found in severe or advanced stages, and is thought to be caused by uncoordinated motility between sympathetic and parasympathetic bowel tone.^{3,7,8} However, GBS is rarely reported as a solely prodromal symptom before motor weakness becomes evident or in less severe cases.^{6,8} Nowe et al.⁸ described a similar case of a 74-year-old man with adynamic ileus as the presenting symptom before the first peripheral neurological signs appeared. Unlike our case, gastric emptying scintigraphy was not used for possible confirmation of paralytic ileus in this previous case. Treatment, including IVIG or plasma exchange, should be started promptly before irreversible nerve damage occurs.^{3,4}

In our case, the patient's course can be explained by delayed detection of GBS and involvement of the gastrointestinal, autonomic, and respiratory systems. There was further evolution of bacterial translocation and concomitant systemic invasive infection. These in turn can lead to a poor prognosis.

Conclusion

Paralytic ileus as the presenting feature in GBS is atypical and easily neglected because of its insidious development. Patients with ileus caused by different pathogenic mechanisms may show overlapping clinical features at their first visit to the Emergency Department, and this is a diagnostic pitfall. However, ignorance of this unusual prodrome to GBS can result in delayed treatment and potential progression to life-threatening events.³ Findings in our case are instructive in that early recognition of any atypical symptoms of GBS and prompt immunotherapy are critical for reducing morbidity and mortality.^{3,4,7}

List of abbreviations

GBS, Guillain–Barré syndrome.

CT, computed tomography.

IVIG, intravenous immunoglobulin.

Authors' contributions

KHL wrote the manuscript and was responsible for the medical care. THH consulted the relevant literature. JTL, LFL, CCS, and WCC revised the manuscript. FCY supervised all of the work. All authors read and approved the final manuscript.

Data sharing

No additional data.


Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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