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Li TJ. Integrating hepatitis C care into human immunodeficiency virus clinics in the direct-acting antiviral era: From biological disadvantage to structural opportunity. *World J Gastroenterol* 2026; 32(36): 118426 [DOI: [10.3748/wjg.118426](https://doi.org/10.3748/wjg.118426)]

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ORIGINAL ARTICLE

Retrospective Cohort Study

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Anti-glutamic acid decarboxylase autoimmunity presenting as chronic intestinal pseudo-obstruction with dysautonomia and late-onset autoimmune diabetes: A case report

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Abstract

BACKGROUND

Chronic intestinal pseudo-obstruction is a rare disorder characterized by recurrent symptoms of intestinal obstruction in the absence of a mechanical cause. Although uncommon, it is associated with significant morbidity and may be life-threatening. Emerging evidence suggests that autoimmune mechanisms, including anti-glutamic acid decarboxylase (anti-GAD) antibodies, may contribute to gastrointestinal dysmotility.

CASE SUMMARY

A 69-year-old man presented with recurrent episodes of small bowel obstruction, progressive weight loss, and newly diagnosed insulin-dependent diabetes mellitus. Comprehensive investigations excluded structural, infectious, metabolic, and neoplastic causes. Imaging demonstrated dilated bowel loops without a clear transition point. Surgical exploration revealed an atonic small bowel without mechanical obstruction. Markedly elevated anti-GAD antibody levels, together with dysautonomia and proximal myopathy, supported an autoimmune etiology. The patient showed significant clinical improvement following treatment with corticosteroids, intravenous immunoglobulin, and rituximab, with resolution of gastrointestinal symptoms and normalization of glycemic control.

CONCLUSION

Autoimmune etiologies should be considered in cases of unexplained chronic intestinal pseudo-obstruction. Anti-GAD antibodies may aid diagnosis, and early initiation of immunotherapy can improve clinical outcomes and reduce unnecessary surgical intervention.

Key Words: Chronic intestinal pseudo-obstruction; Anti-glutamic acid decarboxylase antibodies; Autoimmune dysmotility; Enteric neuropathy; Intravenous immunoglobulin; Rituximab; Case report

Core Tip: Chronic intestinal pseudo-obstruction is a rare and diagnostically challenging disorder that may be associated with autoimmune mechanisms. This case highlights anti-glutamic acid decarboxylase antibody-associated dysautonomia presenting with gastrointestinal dysmotility and evolving insulin-dependent diabetes mellitus. It underscores the importance of considering autoimmune etiologies in unexplained chronic intestinal pseudo-obstruction and demonstrates the potential benefit of immunomodulatory therapy, including corticosteroids, intravenous immunoglobulin, and rituximab, in achieving sustained clinical improvement.

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INTRODUCTION

Chronic intestinal pseudo-obstruction (CIP) is a rare condition characterized by relapsing symptoms and signs of intestinal obstruction in the absence of a mechanical cause. It results from impaired intestinal motility, leading to inadequate nutrition and progressive weight loss. The underlying etiology, progression, and spectrum of associated disorders remain unclear.

Impaired intestinal motility in CIP results from myopathic, neuropathic, or combined disorders of the gastrointestinal tract[1]. This syndrome was first described in 1958[2], the absence of structural lesions in open laparotomies in patients clinically presenting with intestinal obstruction. Approximately two decades later, the term “chronic intestinal pseudo-obstruction” was introduced, coinciding with the advent of central venous nutrition in the 1970s, which enabled survival in patients unable to maintain adequate enteral nutrition[3].

CIP has traditionally been associated with underlying systemic diseases; however, an increasing number of cases of primary (idiopathic) CIP have been described over time. In Japan, for instance, Iida *et al*[4] reported a prevalence of approximately 1:100000, with a female-to-male ratio of 2:1. This disease entity typically remains unrecognized for prolonged periods before an accurate diagnosis is established. During this time, patients often undergo extensive and repeated investigations and may be subjected to unnecessary surgical interventions[5]. Management is primarily aimed at maintaining nutritional status and improving symptoms through pharmacological therapy and, when necessary, endoscopic or surgical procedures.

Small-bowel peristalsis results from complex interactions between smooth muscle cells and the enteric nervous system. Normal motor function relies on coordinated smooth muscle contractions and the pacemaker activity of the interstitial cells of Cajal, both of which operate in close integration with intrinsic enteric neural pathways and extrinsic sympathetic and parasympathetic inputs. Failure of smooth muscle coordination may result from a variety of abnormalities affecting these components, either in isolation or in combination, leading to impaired intestinal motility[6].

The clinical presentation of CIP is markedly heterogeneous, depending on the underlying cause and whether involvement is segmental or diffuse, with symptom severity often fluctuating[3,7,8]. The onset is usually insidious, with symptoms progressively worsening and often preceding the initial sub-occlusive episode by several years. Clinical manifestations may closely mimic those of mechanical intestinal obstruction. The most common symptoms are abdominal pain and distension (80%), nausea and vomiting (75%), constipation (40%), and diarrhea (20%). Nausea and vomiting predominate when dysfunction involves the upper gastrointestinal tract, whereas diffuse abdominal pain, distension, and constipation suggest a primarily distal involvement[6].

There is no established diagnostic consensus for this rare syndrome; however, three key elements are generally considered essential for the diagnosis of CIP: (1) Clinical suspicion of the disorder; (2) Radiological evidence of small-bowel dilatation; and (3) Exclusion of mechanical obstruction[6,9,10].

Intestinal manometry can assist in distinguishing mechanical from functional causes of sub-occlusion by evaluating gastrointestinal motor activity. It also enables classification of the underlying dysmotility as myogenic or neurogenic, depending on whether the dysfunction arises from abnormalities of the smooth muscle or impaired enteric innervation. Full-thickness intestinal biopsies have traditionally been recommended to obtain adequate tissue for diagnostic evaluation in patients with CIP. However, the clinical utility of this approach is limited by the risk of postoperative deterioration in gastrointestinal motility, either directly related to surgical intervention or secondary to adhesion formation.

We report the case of a 69-year-old man who presented with features of CIP, in whom further evaluation revealed a high titer of anti-glutamic acid decarboxylase (anti-GAD) antibodies.

CASE PRESENTATION

Chief complaints

A 69-year-old man presented with abdominal pain, persistent nausea and vomiting, and absence of bowel movements for 1 week.

History of present illness

In May 2023, the patient presented 1 day after discharge from another hospital with a 17-day history of abdominal pain, nausea, and vomiting, accompanied by absence of bowel movements for 1 week and significant unintentional weight loss from 92 kg to 63 kg.

History of past illness

The patient had a history of chronic rhinosinusitis and osteoarthritis, with no prior history of gastrointestinal disease. He had been diagnosed recently with insulin-dependent diabetes mellitus (DM).

Personal and family history

The patient had no significant family history of gastrointestinal or autoimmune diseases. He was a non-smoker and did not consume alcohol. There was no history of relevant occupational or environmental exposure.

Physical examination upon admission

On examination, the patient appeared cachectic and clinically dehydrated. His abdomen was distended and diffusely tender, with sluggish bowel sounds. There were no signs of peritonism, and no palpable masses or organomegaly were identified.

Laboratory examinations

Initial laboratory investigations revealed a hemoglobin level of 10.8 g/dL (normal range: 13-17 g/dL), a white blood cell count of $10 \times 10^9/L$ (normal range: $4-10 \times 10^9/L$), and an elevated C-reactive protein level of 72.6 mg/L (normal range: 0-5 mg/L). Liver function tests demonstrated mildly elevated cholestatic enzymes. Viral hepatitis serology and autoimmune liver screening were negative. Arterial blood gas analysis revealed metabolic alkalosis, with a pH of 7.50 and a bicarbonate level of 32 mmol/L (normal range: 23-29 mmol/L), and a normal lactate level of 1.4 mmol/L. Glycemic assessment demonstrated a markedly elevated glycated hemoglobin of 99 mmol/mol at initial presentation (Table 1). Serological testing showed markedly elevated anti-GAD antibodies (250 IU/mL; normal range: < 17 IU/mL, negative; > 17 IU/mL, positive), while anti-neuronal antibodies were negative (Table 2).

Imaging examinations

Plain abdominal radiography demonstrated dilated small-bowel loops without evidence of a clear transition point (Figure 1). Contrast-enhanced computed tomography of the abdomen and pelvis confirmed diffuse small-bowel dilatation without evidence of a transition point or obstructing lesion (Figure 2). Magnetic resonance enterography performed subsequently showed no focal abnormality or obstructing lesion.

MULTIDISCIPLINARY EXPERT CONSULTATION

A multidisciplinary team involving gastroenterology, surgery, and neurology was engaged in the patient's care. Surgical evaluation, including exploratory laparoscopy converted to mini-laparotomy, excluded mechanical obstruction. Gastroenterological assessment raised suspicion of autoimmune gastrointestinal dysmotility in the context of persistent symptoms and the absence of structural pathology. Neurological evaluation supported an autoimmune etiology, particularly in view of autonomic dysfunction and the presence of anti-GAD antibodies.

FINAL DIAGNOSIS

Anti-GAD antibody-associated autoimmune gastrointestinal dysmotility presenting as CIP, with associated autonomic dysfunction and insulin-dependent DM.

TREATMENT

Following a gastroenterology consultation, the patient was initially treated for suspected autoimmune gastrointestinal dysmotility. He received a monitored neostigmine infusion, which resulted in clinical improvement. He was subsequently transitioned to oral pyridostigmine; however, dose escalation was limited by autonomic dysfunction (sinus tachycardia) and proximal myopathy, manifesting as muscle weakness and wasting.

Table 1 Laboratory results

Test	May 2023	September 2025	Units	Reference range
Urea	4.8	6.3	mmol/L	2.5-7.8
Sodium	138	139	mmol/L	135-145
Potassium	3.1 (decline)	4.5	mmol/L	3.5-5.3
Creatinine	103	62	μmol/L	59-104
eGFR	64.4	> 90	mL/minute/1.73 m ²	-
ALT	38	30	U/L	5-41
Total bilirubin	8.3	7.9	μmol/L	2-21
ALP	268 (increase)	105	U/L	5-41
GGT	182 (increase)	15	U/L	10-71
Total protein	74	66	g/L	60-80
Albumin	32 (decline)	45	g/L	35-50
CRP	72.6 (increase)	3.8	mg/L	0-5
TSH	1.910	-	mIU/L	0.27-4.2
WBC	10.0	6.2	× 10 ⁹ /L	4.0-10.0
RBC	3.64 (decline)	5.07	× 10 ⁹ /L	4.50-5.50
Hb	10.8 (decline)	15.2	g/dL	13-17
HCT	0.32	0.46	L/L	0.40-0.50
MCV	86.5	91.3	fL	83-101
Platelets	189	191	× 10 ⁹ /L	150-400
INR	1.0	1.0	-	-
HbA1c	90	-	mmol/mol	-

ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate; GGT: Gamma-glutamyl transferase; Hb: Hemoglobin; HbA1c: Glycated hemoglobin; HCT: Hematocrit; INR: International normalized ratio; MCV: Mean corpuscular volume; RBC: Red blood cells; TSH: Thyroid-stimulating hormone; WBC: White blood cells.

Table 2 Anti-glutamic acid decarboxylase antibody levels

Antibodies	April 2023	August 2023	March 2024	March 2025
Anti-GAD antibodies (IU/mL)	255	44.0	16.0	9.0
Anti-neutrophil antibodies	Negative	Not tested	Not tested	Not tested

Anti-GAD: Anti-glutamic acid decarboxylase.

Owing to persistent symptoms, treatment was switched to immunomodulatory therapy with intravenous immunoglobulins (IVIG). The patient received three cycles of IVIG between June 2023 and August 2023. To maintain optimal nutritional status during this period, he required intermittent nasogastric feeding and total parenteral nutrition. Following clinical improvement and discharge, his maintenance regimen included pyridostigmine (60 mg twice daily), tapering doses of prednisolone, prucalopride (4 mg once daily), lactulose, and insulin for persistent hyperglycemia.

In August 2023, following a relapse related to medication non-compliance, the patient was readmitted and treated with intravenous methylprednisolone (pulse therapy) and an additional cycle of IVIG. One month later, rituximab therapy was commenced (initial dose of 500 mg, followed by 1 g 2 weeks later). Based on the sustained clinical response, maintenance therapy with rituximab was administered every 6 months. Over a 10-month follow-up period, prucalopride, insulin, and pyridostigmine were successfully discontinued as symptoms resolved and anti-GAD antibody levels normalized.

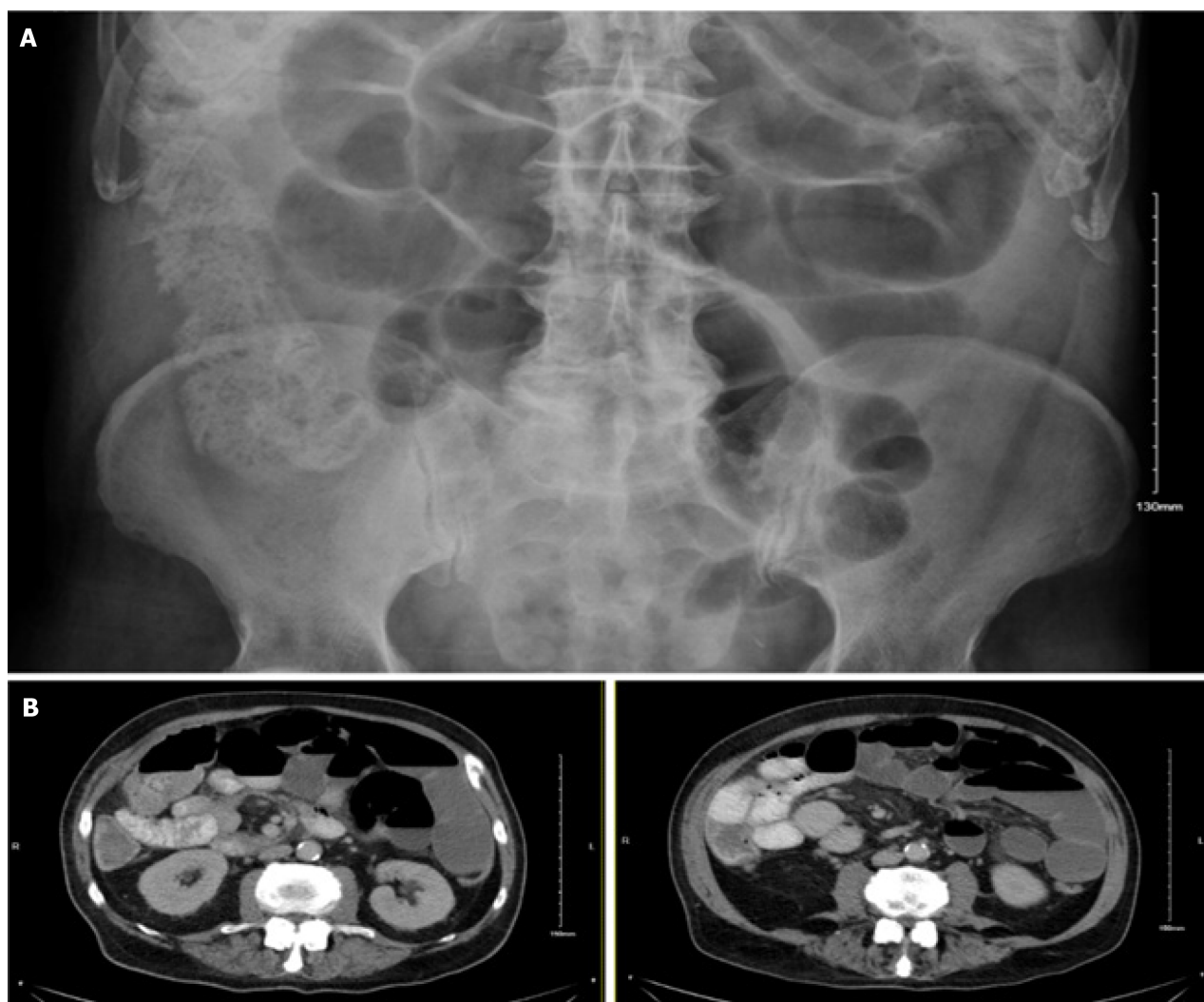


Figure 1 Abdominal radiography and computed tomography findings. A: Abdominal radiograph showing a bowel gas pattern within normal limits, with evidence of fecal loading; B: Contrast-enhanced computed tomography of the abdomen and pelvis demonstrating mild dilatation of the jejunum with fluid-filled loops and associated fecalization of the ileum. The distal ileum and ileocaecal valve are of normal caliber. The large bowel is relatively decompressed, with no intra-abdominal free fluid or air.

OUTCOME AND FOLLOW-UP

Over the subsequent 10 months of regular follow-up, the patient remained clinically well, with restoration of normal bowel function and no further evidence of autonomic dysfunction. Prucalopride, insulin, and pyridostigmine were successfully discontinued as symptoms resolved and anti-GAD antibody levels normalized (Table 2). At the 18-month follow-up, the anti-GAD antibody titer remained low (9 IU/mL), and glycated hemoglobin normalized, allowing discontinuation of insulin therapy. The patient remained asymptomatic with stable bowel habits (Figure 3). He continues on maintenance therapy with rituximab administered every 6 months. The chronological treatment course and clinical response are summarized in Figure 4.

DISCUSSION

CIP may arise secondary to numerous well-defined pathological conditions or may occur without an identifiable cause. The underlying mechanisms are typically neurogenic or myogenic in nature. Myogenic CIP is associated with degeneration and fibrosis of the muscularis propria and is also known as visceral myopathy. Neurogenic CIP is further classified into inflammatory and degenerative neuropathies. Inflammatory neuropathies are characterized by a significant immune-mediated inflammatory response within the myenteric plexus, whereas degenerative neuropathies involve processes such as altered calcium signaling, mitochondrial dysfunction, and production of free radicals[11]. CIP may also occur secondary to systemic conditions, including DM, hypothyroidism, lupus erythematosus, primary systemic sclerosis, Hirschsprung disease, and neoplasia[5].

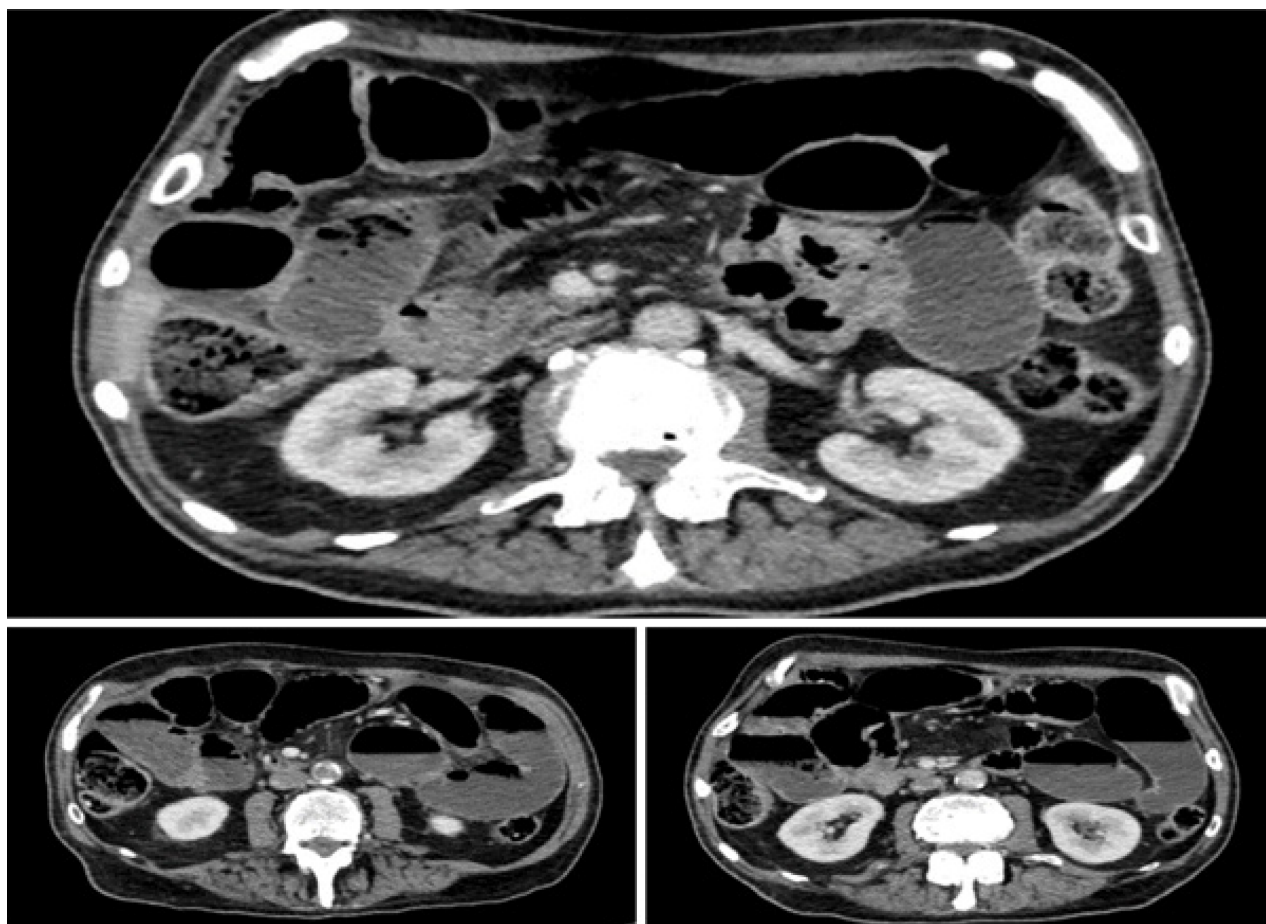


Figure 2 Computed tomography demonstrating small-bowel dilatation. Contrast-enhanced computed tomography showing multiple distended small-bowel loops measuring up to 4.3 cm in diameter, with a transition point in the right hemipelvis. The large bowel is of normal caliber, with fecal loading in the caecum and evidence of small-bowel fecalization.

GAD is an intracellular enzyme responsible for catalyzing the conversion of glutamate to gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the central nervous system. Antibodies against GAD have been implicated in several neurological disorders, including stiff-person syndrome, cerebellar ataxia, and limbic encephalitis, which are thought to arise from impaired GABAergic signaling[12]. Furthermore, anti-GAD antibodies are frequently used as diagnostic markers for autoimmune forms of DM, namely type 1 DM and latent autoimmune diabetes in adults [13]. The diagnosis of both these neurological disorders and autoimmune DM relies on the detection of high serum titers of anti-GAD antibodies and/or their presence in the cerebrospinal fluid.

The role of anti-GAD antibodies in enteric nervous system dysfunction remains unclear. Current evidence does not support a direct pathogenic effect; instead, anti-GAD positivity likely reflects an underlying immune-mediated process. In this context, it should be interpreted as a marker of autoimmune dysmotility rather than definitive evidence of antibody-mediated neuronal injury.

Anti-GAD antibody positivity has been shown to cluster in families with type 1 DM or latent autoimmune diabetes in adults, as well as in certain nondiabetic syndromes. However, the pathophysiological role of anti-GAD antibodies in nondiabetic conditions remains unclear. Furthermore, anti-GAD positivity is an independent predictor of DM regardless of family history, with the risk further increasing at higher antibody titers[14].

Autoimmune dysautonomia, manifesting in this case as orthostatic hypotension and sinus dysrhythmia, represents part of the spectrum of anti-GAD antibody-associated disorders[15]. Although the exact pathogenic role of anti-GAD antibodies remains unclear, they have been associated with autonomic nervous system dysfunction, potentially through disruption of GABA-mediated neurotransmission.

Anti-GAD antibodies are, however, rarely detected in patients with predominant enteric dysmotility, including irritable bowel syndrome, enteric dysmotility, slow-transit constipation, achalasia, and CIP[16,17]. Dhamija *et al*[18] noticed that gastrointestinal dysmotility in anti-GAD antibody-positive patients more commonly occurs in association with other autoimmune diseases, such as hypothyroidism, lupus erythematosus, and myasthenia gravis. In contrast to the typical presentation of anti-GAD antibody-associated disorders such as stiff person syndrome or stiff limb syndrome[15, 19], the present case was characterized by severe weight loss, profound muscle weakness, and proximal myopathy. These findings may suggest involvement of the peripheral nervous system rather than the central nervous system.

Immune-mediated CIP is an uncommon yet increasingly recognized etiology of severe gastrointestinal motility, in which disease-modifying immunotherapy may influence clinical outcomes. Rituximab, a monoclonal antibody targeting

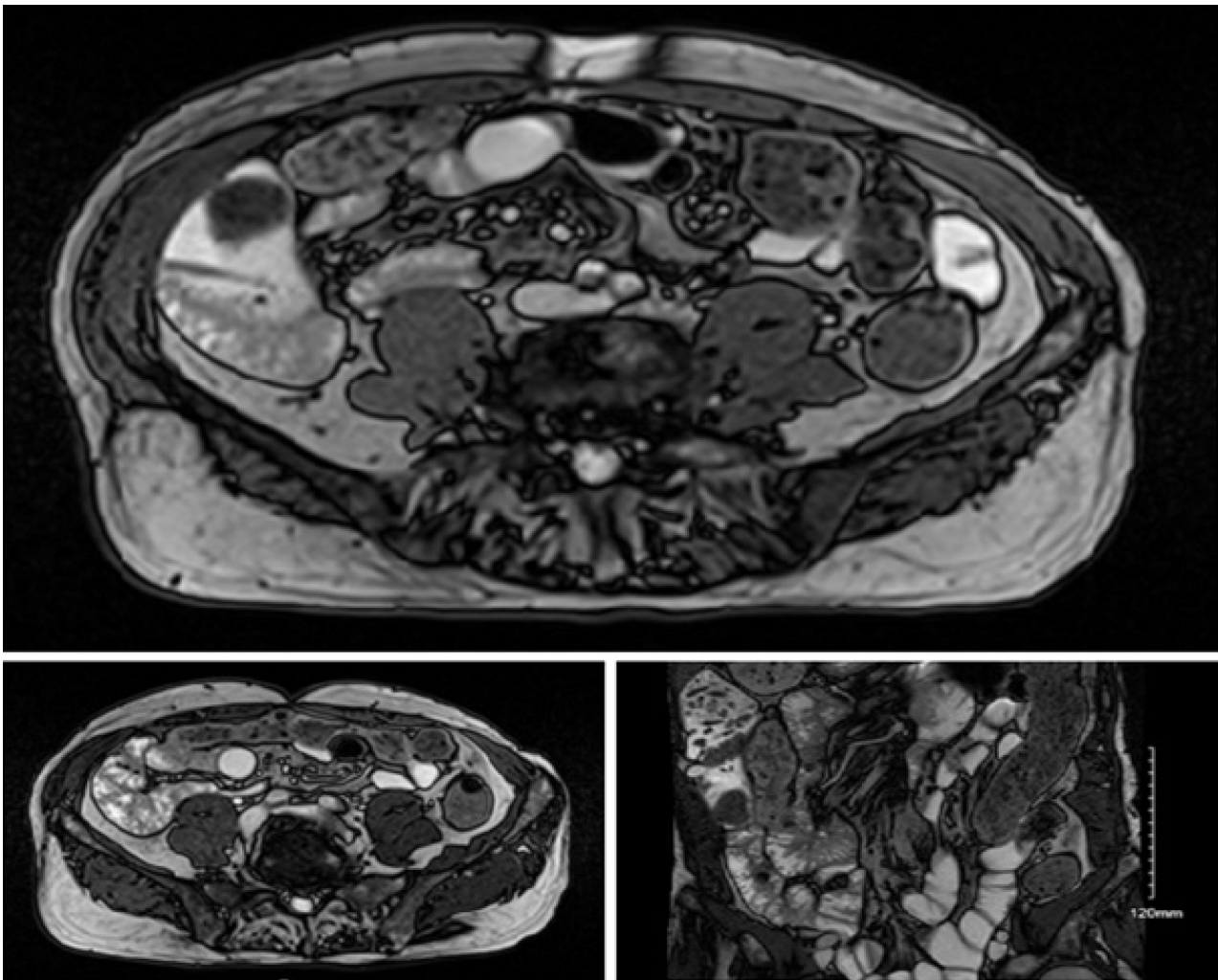


Figure 3 Magnetic resonance imaging of the small bowel. Magnetic resonance image demonstrating feces throughout the colon and fluid-filled small-bowel loops distributed throughout the abdomen. No focal small-bowel abnormality is identified.

Treatment timeline

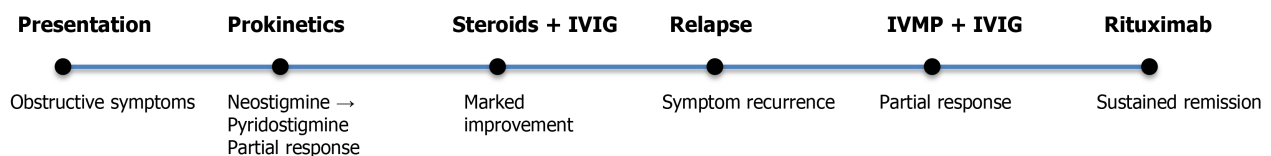


Figure 4 Treatment timeline. Timeline illustrating the clinical course and therapeutic interventions, including initial presentation with obstructive symptoms, partial response to prokinetic therapy, marked improvement following corticosteroids and intravenous immunoglobulins, subsequent relapse, and sustained remission after treatment with intravenous methylprednisolone, intravenous immunoglobulins, and rituximab. IVIG: Intravenous immunoglobulins; IVMP: Intravenous methylprednisolone.

CD20-positive B cells, has been proposed as a treatment option in refractory cases, reflecting accumulating evidence for B-cell-driven immune mechanisms contributing to enteric neuronal dysfunction. Vilaseca *et al*[20] have recently outlined novel immunotherapeutic strategies in immune-mediated CIP, reporting symptomatic improvement following rituximab in selected patients, particularly those with detectable neuronal autoantibodies, including sustained benefit in cases unresponsive to standard therapy. While evidence specific to anti-GAD antibody-associated CIP remains scarce, data from neurological disorders provide mechanistic support for B-cell depletion in anti-GAD-mediated diseases.

A systematic review evaluating rituximab in anti-GAD antibody-positive stiff person syndrome demonstrated clinical improvement in a proportion of patients, reinforcing the role of humoral autoimmunity and the potential utility of B-cell-targeted treatment[21]. Collectively, these observations indicate that rituximab may be a rational therapeutic consideration in carefully selected patients with immune-mediated CIP, particularly in refractory cases supported by serological evidence of antibody-associated pathology.

A limitation of this report is the absence of formal autonomic nervous system assessment and objective gastrointestinal motility studies, such as antroduodenal manometry or transit testing, which could have provided additional physiological confirmation of dysautonomia and enteric dysfunction. These investigations were not performed owing to limited availability and the patient's clinical condition at the time of evaluation. Nevertheless, the diagnosis was supported by the comprehensive exclusion of mechanical, metabolic, infectious, and structural causes, together with high-titer anti-GAD antibodies and a sustained clinical response to immunomodulatory therapy.

Diagnostic laparoscopy may be warranted in selected cases of suspected intestinal pseudo-obstruction when a mechanical cause cannot be confidently excluded, particularly in the presence of persistent symptoms despite appropriate conservative management and inconclusive imaging findings. In this case, surgical exploration was instrumental in excluding a structural etiology and supporting the diagnosis of CIP.

CONCLUSION

In conclusion, the finding of an atonic small bowel with preserved proximal peristalsis likely reflects segmental involvement of the enteric nervous system[3,7,8]. Radiological evidence of dilated bowel loops in the absence of mechanical obstruction meets the diagnostic criteria for CIP[6,9,10]. Moreover, the development of latent-onset autoimmune diabetes in association with the high titers of anti-GAD antibodies supports a unifying diagnosis of anti-GAD antibody-associated CIP. Treatment with methylprednisolone, IVIG, and rituximab resulted in progressive clinical improvement and marked symptom resolution, allowing discontinuation of prokinetic agents and insulin therapy.

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