EJCRIM
European Journal
of Case Reports
in Internal Medicine

SEMAGLUTIDE-INDUCED ISCHEMIC COLITIS IN A MIDDLE-AGED FEMALE WITHOUT VASCULAR RISK FACTORS

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Received: 01/04/2026
Accepted: 07/04/2026
Published: 10/07/2026

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent was obtained from the patient for publication of her case and any accompanying images.
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How to cite this article: Joma M, Karam AM, Sayeh W, Sawaf B, Bseiso A, Katwala A, Alastal Y. Semaglutide-induced ischemic colitis in a middle-aged female without vascular risk factors. *EJCRIM* 2026;**13**:doi:10.12890/2026_006649

ABSTRACT

Ischemic colitis (IC) typically occurs in patients with vascular risk factors. We report a case of a 53-year-old woman without comorbidities who developed IC after initiating semaglutide. She presented with acute abdominal pain and haematochezia. Imaging, colonoscopy, and biopsy confirmed IC, while infectious workup was negative. Symptoms resolved following discontinuation of semaglutide and supportive care. This case highlights a potential association between glucagon-like peptide-1 receptor agonists and IC and underscores the importance of recognizing rare adverse effects in patients without traditional risk factors.

KEYWORDS

Ischemic colitis, semaglutide, vascular risk factors, cessation of therapy

LEARNING POINTS

- Glucagon-like peptide-1 receptor agonists (GLP-1RAs) may be associated with ischemic colitis even in patients without traditional vascular risk factors, highlighting the need for heightened clinical suspicion in atypical presentations.
- New-onset abdominal pain and haematochezia in patients receiving GLP-1RAs should prompt early evaluation for ischemic colitis, including imaging and endoscopic assessment to exclude alternative diagnoses.
- Prompt discontinuation of the suspected offending agent and supportive management can lead to complete clinical recovery, emphasizing the importance of early recognition and intervention.

INTRODUCTION

Ischemic colitis (IC) accounts for 50–60% of all gastrointestinal ischemic events, usually affecting the elderly or patients with vascular risk factors such as hypertension, diabetes mellitus, atherosclerosis, and prothrombotic disorders[1]. It results from transient hypoperfusion of the colonic wall, leading to varying degrees of mucosal injury. Clinical manifestations typically

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include a sudden-onset of abdominal pain in the left lower quadrant, followed by haematochezia[2]. While most cases are self-limited, severe forms may require hospitalization and surgical intervention.

Glucagon-like peptide-1 receptor agonists (GLP-1RAs), including semaglutide and tirzepatide, are increasingly used for glycaemic control and weight loss. These agents exert their effects through delayed gastric emptying, appetite suppression, and enhanced insulin secretion[3]. Gastrointestinal adverse effects such as nausea, vomiting, and constipation are commonly reported; however, IC remains rare and poorly understood[4]. Herein, we present a case of semaglutide-associated IC in a middle-aged female with no significant cardiovascular or thrombotic comorbidities.

CASE DESCRIPTION

A 53-year-old female with a past medical history of irritable bowel syndrome (IBS) and attention-deficit hyperactivity disorder (ADHD) presented with acute onset of crampy left lower quadrant abdominal pain and multiple episodes of frankly bloody diarrhoea. Symptoms began abruptly during the night, waking her from sleep. At presentation, she denied nausea, vomiting, fever, chills, chest pain, shortness of breath, recent travel, and exposure to infectious contacts. Her bowel habits had previously been stable and not changed from normal. The patient reported initiating weekly semaglutide 0.75 mg injections for weight management approximately 11 weeks prior to her presentation in the clinic.

On physical examination, she was afebrile and hemodynamically stable. Abdominal examination revealed mild tenderness over the left lower quadrant without rebound or guarding. Laboratory workup showed normal white blood cell count, C-reactive protein, and procalcitonin levels. Stool studies, including gastrointestinal pathogen polymerase chain reaction and Clostridioides difficile testing, were negative.

A contrast-enhanced computed tomography (CT) scan of the abdomen and pelvis revealed moderate-to-severe wall thickening of the descending colon, sigmoid colon, and rectum. Colonoscopy demonstrated diffuse erythema, friability, congestion, and ulcerations of the descending colon, and biopsies were taken *(Fig. A and B)*. Diverticulosis was noted in the sigmoid colon, and the terminal ileum was normal. Histological examination of the biopsy showed focal crypt dropout, lamina propria haemorrhage, and stromal hyalinization *(Fig. 2)*– findings consistent with IC.

Semaglutide was discontinued, and the patient was managed conservatively with bowel rest, a clear liquid diet,

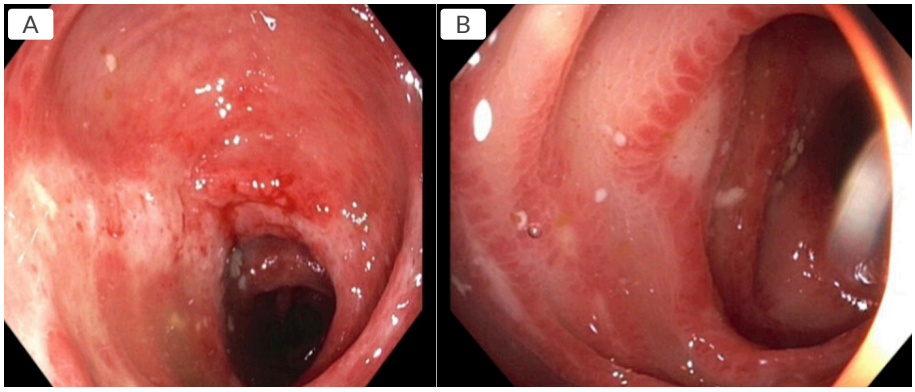


Figure 1. *Colonoscopic images demonstrating abnormal mucosal appearance in the affected colonic segment. A) Longitudinal ulcer with surrounding erythema in the descending colon (single-stripe sign); B) Scattered small ulcers in the left colon.*

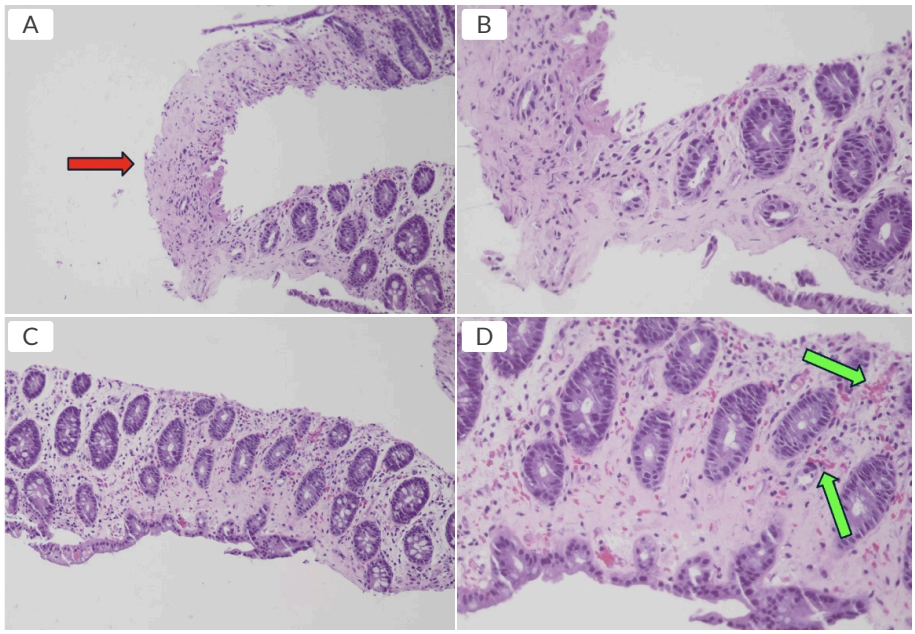


Figure 2. *Histopathologic images from colonic biopsy demonstrating microscopic features of colonic mucosal injury. A) Focal crypt dropout (arrow); B) Intermediate magnification of focal crypt dropout; C) Stromal hyalinization; D) High magnification demonstrating lamina propria hemorrhage (arrows).*

References	Agent	Demographics	Risk factors	Imaging	Treatment	Outcome
Bayless et al. (2024)[5]	Tirzepatide	62 F	HF and weight loss	CT: left colitis; colonoscopy: ulcerated mucosa	Cessation	Resolved
Wichelmann et al. (2022)[6]	Semaglutide	56 F	None	CT: left colitis; biopsy: IC	Conservative management	Recovered
Sekhon et al. (2025)[7]	Semaglutide	43 F	None	Recurrent IC; biopsy: IC	Conservative management	Relapse
Mayer et al. (2025)[8]	Semaglutide	37 F	None	CT: colonic wall thickening	Cessation	Resolved
Present case report	Semaglutide	53 F	IBS	CT: pancolitis; biopsy: crypt dropout, hyalinization	Cessation	Resolved

Table 1. *Summary of published case reports describing GLP-1 RA-associated ischemic colitis.*
Abbreviations: GLP-1 RA, glucagon-like peptide-1 receptor agonist; HF, heart failure; CT, computed tomography; PMH, past medical history; IC, ischemic colitis; IBS, irritable bowel syndrome.

intravenous fluids, and a short course of oral antibiotics. Her symptoms resolved completely, and she was discharged in stable condition with outpatient gastroenterology follow-up.

DISCUSSION

IC occurs when blood flow to the colon is reduced, resulting in mucosal ischemia and inflammation[1,2]. While the majority of IC cases are multifactorial, common contributors include hypovolemia, hypotension, vasoconstrictive medications, thromboembolic events, and impaired colonic motility. However, a subset of patients develops IC in the absence of traditional risk factors, prompting evaluation of alternative aetiologies such as pharmacologic agents.

GLP-1RAs (including semaglutide and tirzepatide) are an increasingly popular class of drugs used in controlling type 2 diabetes mellitus and obesity management. Their mechanism involves potentiating glucose-dependent insulin secretion, suppressing glucagon release, and delaying gastric emptying[3]. GLP-1RAs known physiological effects on the bowel often lead to gastrointestinal side effects, particularly constipation, which may play a role in colonic hypoperfusion. Semaglutide has also been associated with dehydration and volume contraction, both of which can exacerbate ischemic vulnerability[4]. These conclusions suggest a hypothesized causation between GLP-1RAs and IC.

Four prior case reports have described IC in the context of initiation of GLP-1RA therapy, *(Table 1)*. Bayless et al. reported IC in a 62-year-old woman following tirzepatide use[5]; the patient had heart failure and constipation, both likely contributing to ischemia. Wichelmann et al. described a 56-year-old woman who developed left-sided IC following 8 weeks of semaglutide use without other clear predisposing factors[6]. Sekhon et al. reported a 43-year-old woman who developed recurrent IC after semaglutide initiation, with symptoms reappearing upon rechallenge[7]. Mayer et al. described a 37-year-old woman with no predisposing risk factors who developed recurrent IC and only improved after cessation of semaglutide[8]

Compared to these reports, our case highlights several unique aspects. Our patient lacked predisposing risk factors such as cardiovascular, diabetic, or thrombotic comorbidities. Finally, the onset of her symptoms occurred approximately 11 weeks post-initiation, which is consistent with the delayed gastrointestinal adverse profile observed with GLP-1Ras[4].

The American College of Gastroenterology (ACG) recommends supportive care and conservative management for most cases of IC, including bowel rest, fluid resuscitation, and treatment of any underlying cause[1]. In our case, prompt discontinuation of semaglutide and conservative therapy led to full clinical recovery. Prior to discontinuation, endoscopic and histologic confirmation was done to exclude inflammatory bowel disease or other mimics.

Given the increasing utilization of GLP-1RAs in non-diabetic populations, clinicians must remain vigilant for atypical adverse effects such as IC. Patients with GLP-1RA-associated IC may require discontinuation of the medication with conservative management to improve their symptoms. Although a few case reports have suggested this association, larger retrospective and prospective studies are needed to better define the risk of ischemic colitis among patients receiving these agents and to clarify the underlying mechanisms.

We present a case of IC associated with semaglutide use in a patient without vascular or other traditional risk factors who improved after cessation of their GLP-1RA therapy. This case underscores the importance of considering GLP-1RA-induced IC in patients presenting with acute lower gastrointestinal symptoms while on GLP-1RAs. Early recognition, proper workup, discontinuation of the offending agent, and supportive care can lead to promptly favourable outcomes. As the use of GLP-1 RAs expands,

heightened awareness of rare but serious gastrointestinal complications is essential.

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