



OPEN ACCESS

EDITED BY

Monjur Ahmed,
Thomas Jefferson University, United States

REVIEWED BY

Sudhakar Ramamoorthy,
All India Institute of Medical Sciences,
Mangalagiri, India
Kristen Rumer,
Mayo Clinic, United States

*CORRESPONDENCE

Xuefeng Tang
✉ txfaty@163.com

RECEIVED 28 July 2025

ACCEPTED 21 October 2025

PUBLISHED 10 November 2025

CITATION

Wang W, Deng X, Jiang X, Yang M and
Tang X (2025) Case Report: Idiopathic
myointimal hyperplasia of mesenteric veins
mimicking inflammatory bowel disease: a
case report with literature review.
Front. Med. 12:1674469.
doi: 10.3389/fmed.2025.1674469

COPYRIGHT

© 2025 Wang, Deng, Jiang, Yang and Tang.
This is an open-access article distributed
under the terms of the [Creative Commons
Attribution License \(CC BY\)](#). The use,
distribution or reproduction in other forums is
permitted, provided the original author(s) and
the copyright owner(s) are credited and that
the original publication in this journal is cited,
in accordance with accepted academic
practice. No use, distribution or reproduction
is permitted which does not comply with
these terms.

Case Report: Idiopathic myointimal hyperplasia of mesenteric veins mimicking inflammatory bowel disease: a case report with literature review

Wei Wang^{1,2}, Xue Deng^{1,2}, Xin Jiang², Mengxue Yang^{1,2} and
Xuefeng Tang^{1,2*}

¹Chongqing Medical University, Chongqing, China, ²Department of Pathology, The Chongqing General Hospital, Chongqing, China

Background: Idiopathic myointimal hyperplasia of the mesenteric veins (IMH MV) is a rare and poorly understood disease, typically affecting the rectosigmoid colon of young, otherwise healthy men. Clinically, it is often mistaken for inflammatory bowel disease, as biopsies show ischemic mucosal changes without classic inflammatory features. Surgical resection is both diagnostic and curative, although the etiology of IMH MV remains unclear.

Case presentation: We report the case of a female patient with IMH MV involving the right hemicolon, concomitant with *Clostridium difficile* infection. Her symptoms persisted despite targeted treatment for *C. difficile*. She subsequently underwent a laparoscopic right hemicolectomy, which revealed mesenteric vein occlusion due to myointimal hyperplasia, confirmed by elastin staining and desmin immunohistochemistry. Histopathological examination established the diagnosis of IMH MV. The patient recovered well postoperatively, with no recurrence observed during follow-up.

Conclusion: This is the first documented case of IMH MV involving the right hemicolon and complicated by *Clostridium difficile* infection. In addition, we reviewed 82 previously reported cases from 1991 to 2024, highlighting the clinical, imaging, and pathological characteristics of IMH MV. Recognition of this rare entity is essential to avoid unnecessary pharmacotherapy, prevent misdiagnosis as inflammatory bowel disease, and facilitate timely surgical management.

KEYWORDS

idiopathic myointimal hyperplasia of mesenteric veins, *Clostridium difficile* infection, inflammatory bowel disease, idiopathic mesenteric phleboscrosis, vessels with arterial-type wall thickening of uncertain significance

1 Introduction

Idiopathic Myointimal Hyperplasia of the Mesenteric Veins (IMH MV) is a rare and poorly understood condition that presents a diagnostic challenge for clinicians and pathologists. It is classified as an ischemic bowel disease characterized by venous occlusion due to smooth muscle proliferation in the tunica intima of the mesenteric veins, without the presence of a thrombus (1). IMH MV is often misdiagnosed as inflammatory bowel disease (IBD)

(Supplementary Table S1), as definitive diagnosis of bowel ischemia and venous thrombotic disease relies on pathological changes that are not distinguishable through preoperative radiological or clinical assessments. Consequently, a definitive diagnosis can only be achieved postoperatively, since biopsies are unable to differentiate ischemic abnormalities from those associated with known IBD manifestations (2). Since the first case was reported in the United States in 1991, approximately 81 cases of IMH MV have been documented in the literature, with the present case representing the 82nd reported instance. Majority of these cases ultimately end up with different degrees of bowel resection (3).

Idiopathic myointimal hyperplasia of the mesenteric veins mostly involves the thickening of small and medium-sized mesenteric veins with the hallmark manifestation of intimal smooth muscle proliferation resulting in luminal occlusion and mucosal ischemic changes (2). Pathologists may miss the diagnosis unless elastin stains are performed, as the affected veins can easily be mistaken for arteries. The etiology of IMH MV remains unclear. One hypothesis suggests that the disease may result from an arteriovenous fistula. Another Hypothesis proposes that IMH MV represents the end stage of 'phlebitis,' as lesions resembling IMH MV have been observed in cases of lymphocytic, granulomatous, and necrotizing phlebitis (4, 5).

We herein describe the case of a female patient with idiopathic myointimal hyperplasia of the mesenteric veins (IMH MV) at right colon with *Clostridium difficile* infection (CDI).

2 Case presentation

A 68-year-old female patient, with a history of chronic abdominal pain, was admitted to the hospital with chief complaint of severe abdominal pain for the past 2 months. The abdominal pain initially started in the left hypochondrium as a vague discomfort, but later migrated and localized as a persistent distending pain in the lower abdomen, accompanied by vomiting, bloody stools, and low-grade fever. There was history of loss of appetite. She has consumed only porridge in the past 2 months. There was no history of joint pain, skin lesions, chest pain, cough, or contact with contaminated water. One month prior, she was admitted with a diagnosis of hypertensive heart disease, grade 3 hypertension, abnormal coagulation function, ischemic enteropathy, and chronic atrophic gastritis. She has previously undergone resection of a lung abscess. She self-reports an allergy to penicillins and abdominal pain after taking aspirin. No significant family history.

The physical examination revealed tenderness in the upper middle abdomen, left flank, and lower abdomen, more prominent in the upper middle and lower abdomen. No rebound tenderness or palpable masses were noted. Normal bowel sounds. The following Laboratory findings were reported: CRP = 20.51 mg/L, Hg = 106 g/L, D-Dimer = 0.78 mg/L, VIII factor activity = 267.90%, TP = 63.7 g/L, ALB = 31.8 g/L, positive fecal occult blood test, and *Clostridium difficile* antigen test.

A colonoscopy revealed congested and inflamed mucosa in the colon (Figures 1D–I). Salt and pepper appearance was seen in the region. The transverse colon exhibited a whitish appearance with loss of vasculature. Biopsy findings revealed a non-specific ulcer.

Computed tomography angiography (CTA) of the abdomen showed mixed plaques in the abdominal aorta and at the origin of the bilateral common iliac arteries (Figure 1C). Computed tomography (CT) scan of the abdomen results showed a swollen colon wall with diminished enhancement and surrounding exudate that was suggestive of ischemic bowel disease (Figures 1A,B). Based on the presentation and investigative findings, she was diagnosed with a suspected case of ischemic bowel disease and was started on scopolamine hydrobromide for spasm and pain relief, fasting, omeprazole for gastric protection, and atorvastatin calcium tablets to stabilize plaques. The patient's abdominal pain slightly subsided, but intermittent abdominal pain persisted, primarily around the navel, with slight relief after activity or oxygen inhalation. The stools were yellow and pasty. Papaverine was additionally used to relieve spasm and improve intestinal blood supply, while mesalazine was administered to repair the intestinal mucosa. Metronidazole tablets were added to treat the infection, but the patient experienced nausea and acid reflux after administration. Additionally, no pseudomembranous enteritis was found in the colonoscopy, and there was no mucus or pseudomembranes in the stool. Metronidazole was discontinued after 1 week. A conservative management approach was followed, but the symptoms did not resolve. A decision was made to proceed with a laparoscopic right hemicolectomy.

Postoperative pathology revealed the removal of 1 cm of ileum and 48 cm of colon. A local intestinal stricture was present 41 cm from the ileal resection margin, with a length of approximately 2 cm. The intestinal mucosa at this site was grayish-brown with granular hyperplasia. An ulcer was observed 28 cm from the ileocecal valve, adjacent to the stricture, measuring 10 × 3 cm. At this site, the intestinal mucosal folds were absent and the intestinal wall was hardened. The remaining proximal colon exhibited multiple scattered ulcers with a maximum diameter of 1–3 cm. The mesenteric fat at the lesion site was hyperplastic and encircled the intestinal wall, with the stricture being most prominent. Three lymph nodes were found around the intestine, with a maximum diameter of 0.2–0.3 cm (Figure 2).

2.1 Integrated histopathology description

Histopathological examination revealed that the intestinal mucosa and submucosa were replaced by fibrous tissue, with prominent proliferation of small- to medium-sized veins, some extending from the lamina propria to the serosa. The mucosal villi were absent at the stenotic segments, while the crypt architecture was largely preserved, without significant distortion or branching. Inflammatory infiltrates were present but limited, predominantly composed of neutrophils, with minimal lymphoplasmacytic component. Notably, there was no basal plasmacytosis, crypt abscesses, or Paneth cell metaplasia, features that typically indicate chronic inflammatory bowel disease, supporting the exclusion of IBD. The hyperplastic veins showed extensive intimal proliferation, resulting in slit-like lumina, with thickened walls and protruding endothelial cells. Capillaries in the affected areas also exhibited slit-like lumina and thickened walls. No histopathological features characteristic of *Clostridium difficile* infection, such as pseudomembranes, mucosal necrosis, or prominent exudates, were observed.

Abbreviations: IMH MV, Idiopathic myointimal hyperplasia of mesenteric veins; CDI, *Clostridium difficile* infection; IBD, Inflammatory bowel disease; UC, Ulcerative colitis; CD, Crohn's disease; IC, Ischemic colitis; IMP, idiopathic mesenteric phlebosclerosis; CT, computed tomography; CTA, computed tomography angiography.

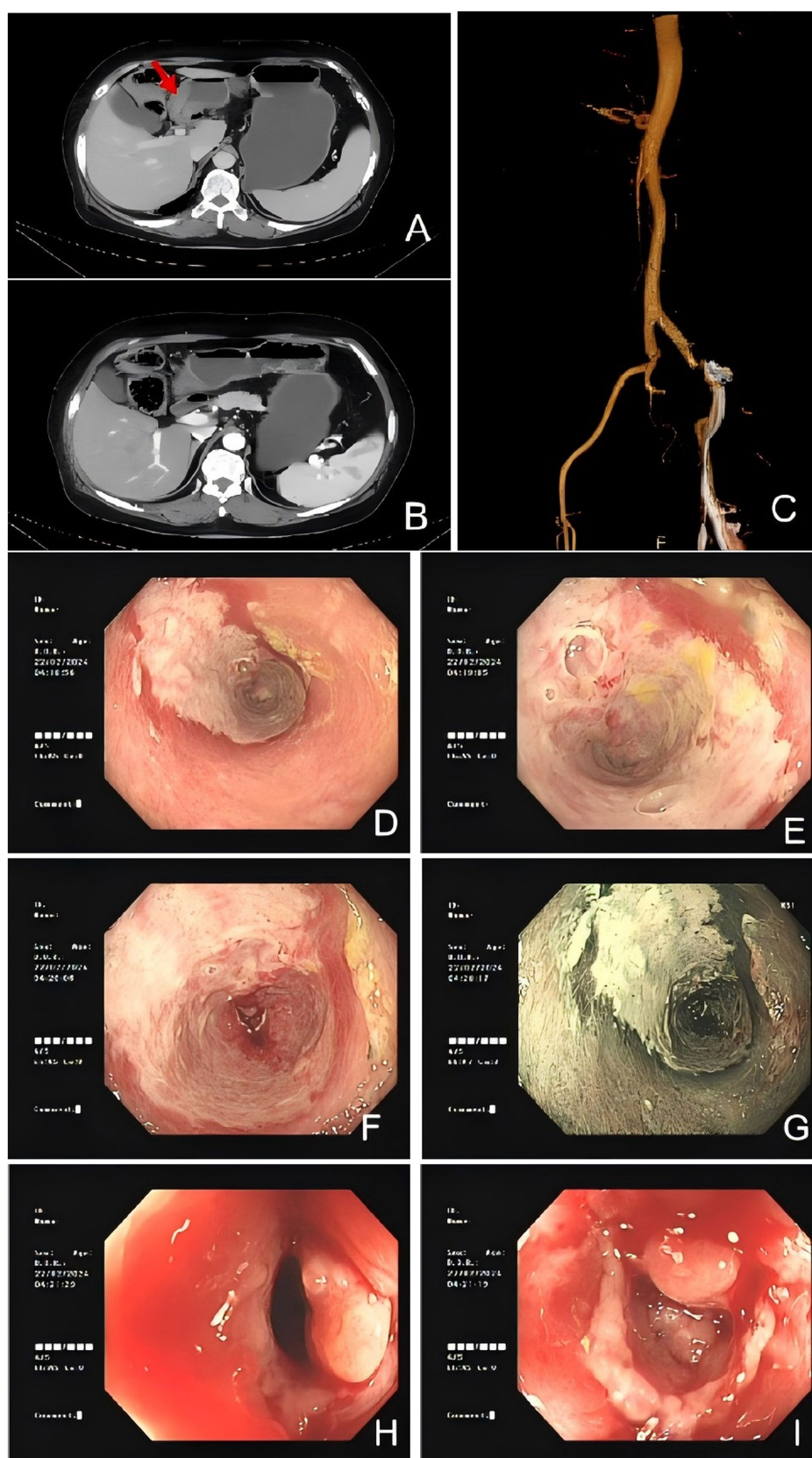


FIGURE 1

Colonic imaging and computed tomography suggested IMH MV. (A,B) Axial CT images showing thickened colonic wall with edema (red arrowhead) and no mesenteric artery stenosis. (C) Abdominal CTA revealed mixed plaques at the origin of the abdominal aorta and bilateral common iliac arteries, suggesting the possibility of mesenteric microthrombosis. Flexible sigmoidoscopy images demonstrated severely inflamed mucosa (D–J) with significant luminal narrowing (H,I).

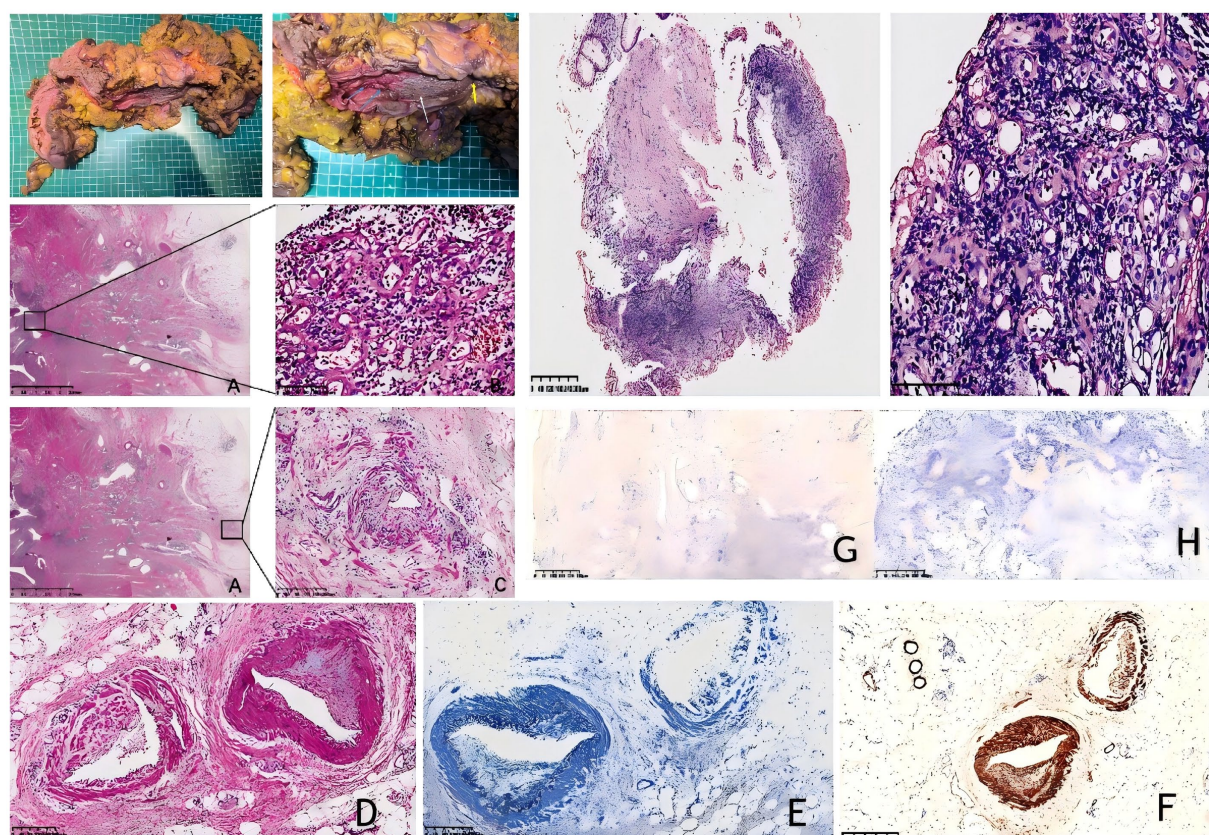


FIGURE 2

(A–D) Hematoxylin–eosin (H&E) staining of the resected surgical specimen (original magnification x200); (E) elastic fiber staining (200x magnification); (F) immunostaining for smooth muscle actin shows the endoluminal nature of the venous proliferation. (200x magnification); (G) EBER *in situ* hybridization (ISH) test. (200x magnification); (H) CMV staining is negative. (I, J) Surgical resection specimen from IMH MV. Large ulcer (white arrow); scattered ulcers (blue arrow); stenosis (yellow arrow); (K–M) Hematoxylin–eosin (H&E) staining of the mucosal biopsy (original magnification x200) shows ischemic-type changes, including epithelial sloughing, ulceration, and atrophy, with dense neutrophilic infiltration and prominent capillary proliferation but without perivascular hyalinization. The muscularis mucosae is thickened with associated fibrosis.

Immunohistochemical staining with Desmin confirmed that the smooth muscle in the venous intima was proliferating in a semicircular pattern. Concurrently, Verhoeff–Van Gieson (VVG) elastic fiber staining demonstrated noticeable intimal thickening in the arteriole-type vessels. No vasculitis was present; EBER RNA and CMV expression were negative, excluding EBV colitis and CMV colitis (Figure 2). No evidence of thrombosis or malignancy was observed. The final diagnosis was idiopathic myointimal hyperplasia of the mesenteric veins (IMH MV). Follow-up over 1 year after surgery, she was doing well, without issue.

We reviewed the patient's previous biopsy specimens and noted dense neutrophilic infiltration of the mucosal layer with prominent capillary proliferation but without perivascular hyalinization. The muscularis mucosae was thickened with associated fibrosis and lacked features of chronic active inflammation, such as lymphoplasmacytic infiltrates, basement membrane disruption, or crypt abscesses. Although these findings were not diagnostic, they did not support inflammatory bowel disease or *Clostridium difficile* infection (Figures 2K–M).

3 Discussion

Idiopathic myointimal hyperplasia is a rare cause of intestinal ischemia. Compared to other inflammatory conditions of the gastrointestinal tract, IMH MV is not caused by arterial thromboembolism,

venous thrombus or vasculitis, and its etiology remains poorly understood.

The most common symptoms reported in previous cases include abdominal pain, perforation, and hematochezia (Supplementary Table S1). The patient was admitted with chief complaint of severe abdominal pain. Patients with *C. difficile* enteritis generally present with diarrhea, crampy abdominal pain, and leukocytosis (6). Given that the patient presented with *Clostridium difficile* infection (CDI) and the clinical manifestations of IMH MV and pseudomembranous colitis caused by CDI were similar, the histological findings were distinctly different. The former often results in mucosal changes due to non-specific ischemic injury, rather than a toxin-mediated inflammatory process, with the absence of pseudomembrane formation and interstitial necrosis (7). CDI may lead to microvascular dysfunction and thrombosis of the superior mesenteric artery (8, 9) and affect the development of ischemic bowel disease (10). Ischemic colitis may be a complication of CDI (11). As a rare cause of ischemic bowel disease, CDI may offer insight into the pathogenesis of IMH MV. This case is notable as the first reported instance of IMH MV combined with CDI. CT imaging of our patient revealed focal colonic wall thickening and submucosal edema with preserved mesenteric arterial patency. However, the characteristic radiographic findings of IMH MV described in previous reports—enlarged and tortuous pericolic vessels with

rich, dilated peripheral veins—were not observed in this case (Figure 1). This absence may reflect the early stage and limited extent of venous involvement in our patient, as well as differences in imaging protocol compared with prior studies (Table 1).

Our patient has hypertension and hypertensive heart disease, consistent with previous reports noting cardiovascular risk factors in many IMH MV cases (4, 12–14). IMH MV is characterized by myointimal hyperplasia leading to mesenteric vein occlusion.

TABLE 1 Clinical characteristics of all reported cases of IMH MV to date.

	Authors, year	Age(y)/Sex	Affected site	Clinical impression	Indication for surgery	Time to surgery	Follow-up
1	Current case	68/F	Right hemicolon	IC		2 months	
2	López Morales et al. (29)	37/M	Rectum to terminal ileum	CD	Abdominal pain	7 months	Died
3	Shah et al. (30)	24/F	Rectum to descending colon	IC	Abdominal pain/perforation		
4–15	Kim et al. (28)	Mean 66 (range 58–77/11M and 1F)	Rectosigmoid (n = 9), rectum to descending colon (n = 2) ileum to transverse colon (n = 1)	IC (n = 4); UC (n = 1), non-specific colitis (n = 1), CMV colitis (n = 1), Idiopathic Phleboscleritis colitis (n = 1), IMH MV (n = 3)		Mean 3 months (range 1–8 months)	Mean 29 months (range 2–125 months)
16	Wong et al. (31)	72/M	Sigmoid to descending colon	IC	Abdominal pain		
17	Xie and Xu (32)	21/F	Rectosigmoid	IBD	Massive hematochezia	20 days	2 yr
18	Ansari et al. (33)	63/M	Sigmoid to descending colon	Entamoeba histolytica infection	Abdominal pain	>2 months	5 yr
19	Fang et al. (34)	21/F	Rectosigmoid	IBD	Hematochezia and perforation	2 months	1 yr
20	Yamada et al. (1)	81/F	Terminal ileum	Adhesive intestinal obstruction	Bowel obstruction		32 mo
21	Almumtini et al. (35)	55/M	Rectum to distal transverse	IBD	Perforation	1 yr	
22	Wu et al. (36)	53/M	Rectum to descending colon	UC	Persisting symptoms	3 months	3 months
23	Martin et al. (37)	63/M	Sigmoid to descending colon	IC/IBD	Persisting symptoms	5 months	2 months
24	Chudy-Onwugaie et al. (38)	54/M	Transverse colon	CMV colitis	Persisting symptoms	4 months	
25–32	Anderson et al. (39)	Median 62.5 (range 22–75)/6M and 2F	Sigmoid (n = 8)	IBD (n = 3)			
33	Louie et al. (40)	57/M	Small bowel		Abdominal pain		
34	Gonai et al. (41)	68/M	Sigmoid to descending colon	Mesenteric panniculitis	Persisting symptoms		
35–44	Yantiss et al. (3)	Mean 68 (range 25–83/9M and 1F)	Sigmoid to descending colon (n = 7), descending colon (n = 1), sigmoid colon (n = 2)	IC/IBD (n = 1), IBD (n = 7); IC (n = 2)	Perforation (n = 5), obstruction and refractory colitic symptoms		
45	Song and Shroff. (2)	59/M	Sigmoid to ileum	CD	Persisting symptoms	30 yr	2 wk

(Continued)

TABLE 1 (Continued)

	Authors, year	Age(y)/Sex	Affected site	Clinical impression	Indication for surgery	Time to surgery	Follow-up
46	Yang et al. (42)	44/M	Rectosigmoid	UC	Persisting symptoms	4 wk	
47	Patel et al. (43)	65/M	Sigmoid to descending colon		Perforation	1.5 Months	
48	Guadagno et al. (44)	59/F	Ileum	CD	Multiple ileal neuroendocrine tumors	6 months	3 months
49	Costa et al. (45)	47/M	Rectosigmoid	IC/IBD	Persistent symptoms	9 months	
50	Cauchois et al. (46)	48/M	Rectum	IBD		3 months	
51	Yun et al. (4)	64/M	Rectum to distal transverse	UC	Hematochezia	2 yr	6 months
52	Wangenstein et al. (47)	62/F	Rectosigmoid	UC	Persistent symptoms	2 months	1.5 yr
53	Abbott et al. (48)	58/M	Rectum to descending colon	IC/IBD	Persistent symptoms		
54	Sahara et al. (12)	76/M	Rectosigmoid	IC/IBD	Persistent symptoms	1 yr	3 months
55	Laskaratos et al. (49)	62/F	Ileum	IBD	Perforation and hematochezia		
56	Zijlstra et al. (50)	62/M	Rectum to descending colon		Acute abdomen		2 yr
57	Korenblit et al. (51)	59/M	Rectosigmoid	IC	Persistent symptoms	1 month	3 months
58	Feo et al. (52)	75/F	Rectosigmoid	IC	Persistent symptoms	6 months	
59	Lanitis et al. (53)	81/M	Terminal ileum		Appendiceal mucocoele and pseudomyxoma peritonei	6 months	
60	Korenblit et al. (51)	62/M	Entire colon (rectal sparing)	UC	Hematochezia	18 months	
61	Chiang et al. (54)	60/M	Rectosigmoid	UC	Persistent symptoms	2 months	4 months
62	Garciaos et al.	32/M	Rectum to descending colon	primary pneumatosis intestinalis	Abdominal pain and hematochezia	3 months	24 months
63	Kao et al. (55)	38/M	Rectosigmoid	IBD	Perforation	5 months	18 months
64	Savoie and Abrams (56)	22/M	Rectosigmoid	IBD	Abdominal pain and hematochezia		10 months
65	Bryant et al. (14)	42/F	Jejunum	/			
66	Abu-Alfa et al. (5)	58/M	Sigmoid	IC/IBD	Abdominal pain and hematochezia	1 yr	
67–70	Genta and Haggit et al. (57)	Mean 40 (range 25–67)/4 M	Sigmoid ($n = 1$), Sigmoid to descending colon ($n = 1$), Rectosigmoid ($n = 2$)	UC ($n = 2$), CD ($n = 1$); Stricture ($n = 1$)	Bowel obstruction ($n = 1$), toxic megacolon ($n = 1$), abdominal pain and hematochezia ($n = 2$)	Mean 3 months (range 1–6 months)	Mean 3.5 yr. range 1–7 yr
71	Li et al. (58)	64/M	Ileum	IBD	Bowel obstruction	6 months	1 yr
72	Shi et al. (59)	25/M	Small bowel	CD	Bowel obstruction	10 yr	5 months
73	Noujaim et al. (62)	66/M	Rectum to descending con	IBD	Persistent symptoms	1 month	Died

(Continued)

TABLE 1 (Continued)

	Authors, year	Age(y)/Sex	Affected site	Clinical impression	Indication for surgery	Time to surgery	Follow-up
74	Morimura et al. (60)	44/M	Left hemicolon		Intestinal ischemia and necrosis	1 month	1 yr
75	Kawasaki et al. (27)	71/M	Rectosigmoid	IMH MV		4 months	3 months
76–79	ROBERT M et al.	Median 38 (range 38–67)/4 M	Rectosigmoid		Persistent symptoms	Mean 1 months (range 1 months–1 yr)	
80–81	Bhatt et al. (61)	82,59/2 M	Sigmoid to descending colon		Intestinal ischemia and necrosis	3 months	7 yr
82	Huanhuan Xie	21/F	Rectosigmoid		Persistent symptoms	2 weeks	1 yr

IMH MV, idiopathic myointimal hyperplasia of mesenteric veins; IBD, inflammatory bowel disease; M, male; F, female; yr, year.

Experimental studies have shown that elevated blood pressure can induce intimal thickening and endothelial changes in small vessels (15–26), which may suggest a potential role of hypertension in the pathogenesis of IMH MV. Further research is warranted to clarify this association.

The absence of concrete histopathological criteria for a definitive diagnosis of IMH MV makes biopsy-based diagnosis challenging for pathologists, leading to the early initiation of treatment with anti-inflammatory drugs rather than surgery, which, at present, remains the only effective treatment for this condition and has been reported to be completely curative. However, only six cases of IMH MV have been diagnosed preoperatively (27–29). Arteriolized capillary, subendothelial fibrin deposits, and perivascular hyalinization are the most recent specific pathological features that facilitate the identification of IMH MV in mucosal biopsy samples (1). Biopsy specimens from this patient were reviewed to summarize findings indicating the absence of the muscular layer in the intestinal wall, a gap-like appearance of the intestinal wall due to capillary hyperplasia, thickening of the wall, and prominent endothelial cells. Notably, while the mesenteric vein exhibited characteristic smooth muscle proliferation in the intima (consistent with IMH MV pathology), the arterial changes presented a diagnostic paradox: although these vessels retained their intimal architecture, significant non-myointimal thickening was observed, which differed fundamentally from the venous pathology. we describe these as vessels with arterial-type wall thickening of uncertain significance, confirmed by immunohistochemistry to be non-myointimal in nature (Figure 2). This histologic feature has not been documented in prior IMH MV cases and warrants particular attention, as it may represent (1) a previously unrecognized disease variant, (2) concurrent vascular pathology, or (3) a broader disease spectrum or secondary vascular remodeling, given that small-vessel involvement of this type has not been reported in the existing literature. The presence of definitive venous pathology meets the current diagnostic criteria for IMH MV, while the observed alterations in other small vessels may indicate secondary vascular remodeling, a coexisting process, or a previously unrecognized disease spectrum that requires further investigation.

Currently, all cases are diagnosed based on pathological results, and the lack of methods for early diagnosis means that the diagnosis of IMH MV is often delayed. The clinical manifestations and

endoscopic findings are nonspecific and closely resemble those of IBD and IMP. Similar imaging manifestations have also been observed, with IMP showing more similarity in this regard. Below, we summarize the differential diagnosis of IMH MV, IBD and IMP (Table 2). In particular, IBD, with nearly 53.1% of IMH MV cases being diagnosed as IBD before surgery (Supplementary Table S2), will be subdivided into the two subtypes of IBD, namely UC and CD, to differentiate them from IMH MV. This approach can enhance clinicians' understanding of the disease and improve the diagnostic rate.

4 Conclusion

This case report describes the first documented instance of Idiopathic Myointimal Hyperplasia of the Mesenteric Veins (IMH MV) with right colonic involvement complicated by *Clostridium difficile* infection in a female patient, and also provides the inaugural description of small vessels exhibiting arterial-type wall thickening of uncertain significance in IMH MV.

Histopathological analysis demonstrated a paradoxical coexistence of characteristic venous myointimal hyperplasia and a subset of small vessels showing arterial-type wall thickening that distinctly lacked Desmin-positive smooth muscle cell proliferation—an essential feature of the venous lesions—thus creating a diagnostic paradox given the established venocentric pathology of IMH MV. Clinicians should maintain a high index of suspicion for IMH MV when encountering venocentric myointimal hyperplasia in biopsy specimens from elderly patients with suspected inflammatory bowel disease or distal colorectal ischemia, with the caveat that concurrent arterial-type wall thickening of uncertain significance should prompt thorough clinicopathological correlation to exclude alternative vasculopathies.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary material, further inquiries can be directed to the corresponding author/s.

TABLE 2 Differential diagnosis of IMH MV.

	IMH MV	UC	CD	IMP	MAD/AVD
Onset age	Older (mean age 58 years old)	Younger (20–40 years old)	Younger (18–35 years old)	Older (30–86 years old)	Younger (variable, often <50) Usually younger adults (variable, often <50)
Clinical features	Abdominal pain > hematochezia > diarrhea, complicated with intestinal bleeding and perforation	Hematochezia > diarrhea > abdominal pain	Diarrhea > abdominal pain > weight loss, perianal involvement and extraintestinal manifestations are common	Abdominal pain > nausea or vomiting > diarrhea, abdominal distension and constipation and hematochezia	Chronic abdominal pain, GI bleeding; may have ischemic symptoms
Sites of involvement	Rectosigmoid and descending colon, rarely in small intestine	Rectosigmoid, develops from distal to proximal colon	Terminal ileum and ileocecum > colon > rectum > small intestine > upper digestive tract	Right hemicolon, the ascending colon is most susceptible	Variable, can involve small intestine and colon
Skipped lesions	No	No	Yes	No	Yes
Ulcers	Non-specific ulcers	Superficial ulcers	Longitudinal ulcers, cobblestone appearance and aphthous ulcers	Non-specific ulcers	ischemic-type ulcers; may resemble Crohn's disease
Histopathology	Intima and media smooth muscle proliferation	Crypt abscess, cryptitis, superficial ulcers, and plasma cells increase in the basal layer	Non-caseating granuloma	Mucosa fibrosis, hyalinoid degeneration, the venous wall fibrotic thickening, calcification,	Arteriovenous malformations: thickened arterial and venous walls, abnormal capillary connections; mixed arterial and venous involvement
Treatment	Surgery, no response to medication	Response to 5-Aminosalicylic acid, steroid, immunosuppressant or biologic agents	Response to 5-Aminosalicylic acid, steroid, immunosuppressant or biologic agents	stopping the use of herbs and symptomatic management	Variable; sometimes endovascular intervention or surgery needed
Recurrence post operation	No	Yes	Yes	No	Possible, depending on completeness of resection or persistence of vascular malformation

IMH MV, idiopathic myointimal hyperplasia of mesenteric veins; UC, Ulcerative colitis; CD, Crohn's disease; IMP, idiopathic mesenteric phlebosclerosis; MAD/AVD, Mesenteric Arteriovenous Dysplasia/Vasculopathy.

Ethics statement

The studies involving humans were approved by Medical Ethics Committee of Chongqing People's Hospital. The studies were conducted in accordance with the local legislation and institutional requirements. The human samples used in this study were acquired

from a by- product of routine care or industry. Written informed consent for participation was not required from the participants or the participants' legal guardians/next of kin in accordance with the national legislation and institutional requirements. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Written informed consent was obtained from the participants for the publication of this case report.

Author contributions

WW: Conceptualization, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. XD: Investigation, Resources, Writing – review & editing. XJ: Formal analysis, Visualization, Writing – review & editing. MY: Methodology, Supervision, Writing – review & editing. XT: Investigation, Validation, Writing – review & editing.

Funding

The author(s) declare that financial support was received for the research and/or publication of this article. XT is currently receiving grants (2022TIAD-KPX0243 and 2023MSXM034) from Organization Technological Innovation and Application Development of Chongqing. XJ is currently receiving a grant (2024MSXM162) from the project of the Chongqing Municipal Health Commission.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

References

1. Yamada K, Hiraki M, Tanaka T, Mori D, Tanaka F, Manabe T, et al. A case of idiopathic myointimal hyperplasia of the mesenteric veins presenting with small bowel obstruction. *Surg Case Rep*. (2021) 7:17. doi: 10.1186/s40792-020-01100-8
2. Song SJ, Shroff SG. Idiopathic Myointimal hyperplasia of mesenteric veins of the ileum and Colon in a patient with Crohn's disease: a case report and brief review of the literature. *Case Rep Pathol*. (2017) 2017:1–6. doi: 10.1155/2017/6793031
3. Yantiss RK, Cui I, Panarelli NC, Jessurun J. Idiopathic myointimal hyperplasia of mesenteric veins: an uncommon cause of ischemic colitis with distinct mucosal features. *Am J Surg Pathol*. (2017) 41:1657–65. doi: 10.1097/PAS.0000000000000905
4. Yun SJ, Nam DH, Kim J, Ryu JK, Lee SH. The radiologic diagnosis of idiopathic myointimal hyperplasia of mesenteric veins with a novel presentation: case report and literature review. *Clin Imaging*. (2016) 40:870–4. doi: 10.1016/j.clinimag.2015.12.017
5. Abu-Alfa AK, Ayer U, West AB. Mucosal biopsy findings and venous abnormalities in idiopathic myointimal hyperplasia of the mesenteric veins. *Am J Surg Pathol*. (1996) 20:1271–8. doi: 10.1097/0000478-199610000-00014
6. Killeen S, Martin ST, Hyland J, O'Connell PR, Winter DC. *Clostridium difficile* enteritis: a new role for an old foe. *Surgon*. (2014) 12:256–62. doi: 10.1016/j.surgon.2014.01.008
7. Bramdev A. Pseudomembranous colitis. A clinicopathological review of 7 cases. *S Afr Med J*. (1989) 76:221–3.
8. Kurose I, Pothoulakis C, LaMont JT, Anderson DC, Paulson JC, Miyasaka M, et al. *Clostridium difficile* toxin a-induced microvascular dysfunction. Role of histamine. *J Clin Invest*. (1994) 94:1919–26. doi: 10.1172/JCI117542
9. Mastroianni A, Vangeli V, Mauro MV, Manfredi R, Greco S. Upper mesenteric artery thrombosis as a complication of *Clostridium difficile* infection. *Am J Emerg Med*. (2023) 70:181–2. doi: 10.1016/j.ajem.2023.02.002
10. Adejumo AC, Akanbi O, Pani L. Among inpatients, ischemic bowel disease predisposes to *Clostridium difficile* infection with concomitant higher mortality and worse outcomes. *Eur J Gastroenterol Hepatol*. (2019) 31:109–15. doi: 10.1097/MEG.0000000000001273
11. Ionescu EM, Curte A-M, Olteanu AO, Preda CM, Tieranu I, Klimko A, et al. Rare clinical association between *Clostridioides difficile* infection and ischemic colitis: case report and literature review. *Medicina (Kaunas)*. (2021) 57:705. doi: 10.3390/medicina57070705

Generative AI statement

The authors declare that no Gen AI was used in the creation of this manuscript.

Any alternative text (alt text) provided alongside figures in this article has been generated by Frontiers with the support of artificial intelligence and reasonable efforts have been made to ensure accuracy, including review by the authors wherever possible. If you identify any issues, please contact us.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

Supplementary material

The Supplementary material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fmed.2025.1674469/full#supplementary-material>

12. Sahara K, Yamada R, Fujiwara T, Koizumi K, Horiguchi S, Hishima T, et al. Idiopathic myointimal hyperplasia of mesenteric veins: rare case of ischemic colitis mimicking inflammatory bowel disease. *Dig Endosc*. (2015) 27:768–71. doi: 10.1111/den.12470

13. García-Castellanos R, López R, de Vega VM, Ojanguren I, Piñol M, Boix J, et al. Idiopathic myointimal hyperplasia of mesenteric veins and pneumatosis intestinalis: a previously unreported association. *J Crohns Colitis*. (2011) 5:239–44. doi: 10.1016/j.crohns.2010.12.003

14. Bryant J. Unexpected sudden death during propranolol therapy in a patient with mild mesenteric venous myointimal hyperplasia. *J Forensic Sci*. (1998) 43:905–7. doi: 10.1520/JFS14328J

15. Chobanian AV. 1989 Corcoran lecture: adaptive and maladaptive responses of the arterial wall to hypertension. *Hypertension*. (1990) 15:666–74. doi: 10.1161/01.hyp.15.6.666

16. Bobik A, Campbell JH. Vascular derived growth factors: cell biology, pathophysiology, and pharmacology. *Pharmacol Rev*. (1993) 45:1–42. doi: 10.1016/S0031-6997(25)00451-X

17. Chobanian AV. The influence of hypertension and other hemodynamic factors in atherogenesis. *Prog Cardiovasc Dis*. (1983) 26:177–96. doi: 10.1016/0033-0620(83)90005-1

18. Goldby FS, Beilin LJ. How an acute rise in arterial pressure damages arterioles. Electron microscopic changes during angiotensin infusion. *Cardiovasc Res*. (1972) 6:569–84. doi: 10.1093/cvr/6.5.569

19. Schwartz SM, Heimark RL, Majesky MW. Developmental mechanisms underlying pathology of arteries. *Physiol Rev*. (1990) 70:1177–209. doi: 10.1152/physrev.1990.70.4.1177

20. Robertson AL, Khairallah PA. Arterial endothelial permeability and vascular disease. The “trap door” effect. *Exp Mol Pathol*. (1973) 18:241–60. doi: 10.1016/0014-4800(73)90022-1

21. Pulmonary Hypertension | Pulmonary Medicine | JAMA | JAMA Network. (2012). Available online at: <https://jamanetwork.com/journals/jama/article-abstract/1367450> (Accessed February 19, 2025)

22. Tuder RM, Marecki JC, Richter A, Fijalkowska I, Flores S. Pathology of pulmonary hypertension. *Clin Chest Med.* (2007) 28:23–42. doi: 10.1016/j.ccm.2006.11.010
23. Leopold JA. Pulmonary venous Remodeling in pulmonary hypertension: the veins take Center stage. *Circulation.* (2018) 137:1811–3. doi: 10.1161/CIRCULATIONAHA.118.033013
24. Wijeratne DT, Lajkosz K, Brogly SB, Loughheed MD, Jiang L, Housin A, et al. Increasing incidence and prevalence of World Health Organization groups 1 to 4 pulmonary hypertension: a population-based cohort study in Ontario, Canada. *Circ Cardiovasc Qual Outcomes.* (2018) 11:e003973. doi: 10.1161/CIRCOUTCOMES.117.003973
25. Humbert M, Kovacs G, Hoeper MM, Badagliacca R, Berger RMF, Brida M, et al. 2022 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J.* (2022) 43:3618–731. doi: 10.1093/eurheartj/ehac237
26. Fayyaz AU, Edwards WD, Maleszewski JJ, Konik EA, DuBrock HM, Borlaug BA, et al. Global pulmonary vascular Remodeling in pulmonary hypertension associated with heart failure and preserved or reduced ejection fraction. *Circulation.* (2018) 137:1796–810. doi: 10.1161/CIRCULATIONAHA.117.031608
27. Kawasaki K, Kawatoko S, Torisu T, Mizuuchi Y, Iura T, Ohtani H, et al. Idiopathic myointimal hyperplasia of mesenteric veins depicted by barium enema examination, and conventional and magnifying colonoscopy. *Clin J Gastroenterol.* (2022) 15:734–9. doi: 10.1007/s12328-022-01647-z
28. Kim S-W, Ho Park S, Hyoung Park S, Sik Yoon Y, Kim J. Idiopathic Myointimal hyperplasia of the mesenteric veins is a peculiar venous ischemia that may be diagnosed before surgery. *Dis Colon Rectum.* (2022) 65:e707–17. doi: 10.1097/DCR.0000000000002072
29. López Morales P, González Valverde FM, Giménez Francés C, Pastor Quirante F, Albarracín Marín-Blázquez A. Idiopathic myointimal hyperplasia of the mesenteric veins, an uncommon cause of intestinal ischemia. *Rev Esp Enferm Dig.* (2022) 114:368–9. doi: 10.17235/reed.2022.8654/2022
30. Shah YB, Lee D, Khaddash TS. Endovascular approach in the management of idiopathic myointimal hyperplasia of the inferior mesenteric vein. *CVIR Endovasc.* (2021) 4:88. doi: 10.1186/s42155-021-00272-0
31. Wong R, Westerveld D, Yeo H, Jessurun J, Jesudian A. Ischemic colitis from idiopathic myointimal hyperplasia of the mesenteric veins in a post-liver transplant patient. *ACG Case Rep J.* (2021) 8:e00692. doi: 10.14309/crj.0000000000000692
32. Xie H, Xu X. Radiological and clinical findings of idiopathic myointimal hyperplasia of mesenteric veins: case report. *Medicine (Baltimore).* (2021) 100:e27574. doi: 10.1097/MD.00000000000027574
33. Al Ansari A, Ahmed S, Mansour E, Abass MA. Idiopathic myointimal hyperplasia of the mesenteric veins. *J Surg Case Rep.* (2021) 2021:rjaa453. doi: 10.1093/jscr/rjaa453
34. Fang S, Song Y, Zhang C, Wang L. Efficacy and safety of vedolizumab for pediatric with inflammatory bowel disease: a systematic review. *BMC Pediatr.* (2022) 22:175. doi: 10.1186/s12887-022-03229-x
35. Almumtin A, Al Sulais E, Elhag MA. Idiopathic Myointimal hyperplasia of mesenteric veins (IMH MV) with two spontaneous bowel perforations: a case report and literature review. *Int J Surg Case Rep.* (2021) 83:106022. doi: 10.1016/j.ijscr.2021.106022
36. Kelly Wu W, Tombazzi CR, Howe CF, Kendall MA, Walton DB, Washington MK, et al. Idiopathic Myointimal hyperplasia of the mesenteric veins: a rare imitator of inflammatory bowel disease. *Am Surg.* (2023) 89:1141–3. doi: 10.1177/0003134820973390
37. Martin FC, Yang LS, Fehily SR, D'Souza B, Lim A, McKelvie PA. Idiopathic myointimal hyperplasia of the mesenteric veins: case report and review of the literature. *JGH Open.* (2020) 4:345–50. doi: 10.1002/jgh.3.12297
38. Chudy-Onwugaje K, Ali O, Umoren M. Idiopathic Myointimal hyperplasia of the mesenteric veins of the Colon. *Clin Gastroenterol Hepatol.* (2020) 18:A19–20. doi: 10.1016/j.cgh.2019.07.030
39. Anderson B, Smyrk TC, Graham RP, Lightner A, Sweetser S. Idiopathic myointimal hyperplasia is a distinct cause of chronic colon ischaemia. *Color Dis.* (2019) 21:1073–8. doi: 10.1111/codi.14685
40. Louie CY, DiMaio MA, Charville GW, Berry GJ, Longacre TA. Gastrointestinal tract vasculopathy: clinicopathology and description of a possible “new entity” with protean features. *Am J Surg Pathol.* (2018) 42:866–76. doi: 10.1097/PAS.0000000000001060
41. Gonai T, Toya Y, Nakamura S, Kawasaki K, Yanai S, Fujita Y, et al. Gastrointestinal: idiopathic myointimal hyperplasia of mesenteric veins. *J Gastroenterol Hepatol.* (2018) 33:1939. doi: 10.1111/jgh.14384
42. Yang KH, Kwon TH, Park KS, Kim ES, Cho KB, Baek SK, et al. Idiopathic Myointimal hyperplasia of mesenteric veins. *Korean J Gastroenterol.* (2016) 67:54–7. doi: 10.4166/kjg.2016.67.1.54
43. Patel AD, Schneider Y, Saumoy M, Maltz C, Yeo H, Jessurun J, et al. Idiopathic Myointimal hyperplasia of the mesenteric veins. *ACG Case Rep J.* (2016) 3:e84. doi: 10.14309/crj.2016.57
44. Guadagno E, Del Basso De Caro M, Del Prete E, D'Armiento FP, Campione S. Coexistence of multiple ileal neuroendocrine tumors and idiopathic myointimal hyperplasia of mesenteric veins: coincidence or consequence? Case report and review of literature. *Int J Surg Pathol.* (2016) 24:627–30. doi: 10.1177/1066896916642289
45. Costa MN, Saiote J, Pinheiro MJ, Duarte P, Bentes T, Oliveira MF, et al. Segmental colitis caused by idiopathic myointimal hyperplasia of mesenteric veins. *Rev Esp Enferm Dig.* (2016) 108:821–6. doi: 10.17235/reed.2016.4051/2015
46. Cauchois A, Desfourneaux V, Kammerer-Jacquet S-F, Bouguen G, Rioux-Leclercq N, Henno S. A case of idiopathic myointimal hyperplasia of mesenteric veins. *Ann Pathol.* (2016) 36:415–9. doi: 10.1016/j.annpat.2016.09.002
47. Wangenstein KJ, Fogt F, Kann BR, Osterman MT. Idiopathic Myointimal hyperplasia of the mesenteric veins diagnosed preoperatively. *J Clin Gastroenterol.* (2015) 49:491–4. doi: 10.1097/MCG.0000000000000290
48. Abbott S, Hewett P, Cooper J, Ruszkiewicz A. Idiopathic myointimal hyperplasia of the mesenteric veins: a rare differential to be considered in idiopathic colitis. *ANZ J Surg.* (2018) 88:242–3. doi: 10.1111/ans.13210
49. Laskaratos F-M, Hamilton M, Novelli M, Shepherd N, Jones G, Lawrence C, et al. A rare cause of abdominal pain, diarrhoea and GI bleeding. Idiopathic myointimal hyperplasia of the mesenteric veins (IMH MV). *Gut.* (2015) 64:214–350. doi: 10.1136/gutjnl-2014-308319
50. Zijlstra M, Tjhi-Wensing JWMA, van Dijk MAAM, Wegdam JA. Idiopathic myointimal hyperplasia of mesenteric veins: an unusual cause of diarrhoea. *Ned Tijdschr Geneesk.* (2014) 158:A7752
51. Korenblit J, Burkart A, Frankel R, Klinge M, Greenbau L, Goldstein S, et al. Refractory pancolitis: a novel presentation of idiopathic myointimal hyperplasia of mesenteric veins. *Gastroenterol Hepatol (N Y).* (2012) 8:696–700.
52. Feo L, Cheeyandira A, Schaffzin DM. Idiopathic myointimal hyperplasia of mesenteric veins in the elderly. *Int J Color Dis.* (2013) 28:433–4. doi: 10.1007/s00384-012-1480-0
53. Lanitis S, Kontovounisios C, Karaliotis C. An extremely rare small bowel lesion associated with refractory ascites. Idiopathic myointimal hyperplasia of mesenteric veins of the small bowel associated with appendiceal mucocoele and pseudomyxoma peritonei. *Gastroenterology.* (2012) 142:e5–7. doi: 10.1053/j.gastro.2011.11.052
54. Chiang C-K, Lee C-L, Huang C-S, Huang S-H, Wu C-H. A rare cause of ischemic proctosigmoiditis: idiopathic myointimal hyperplasia of mesenteric veins. *Endoscopy.* (2012) 44:E54–5. doi: 10.1055/s-0031-1291529
55. Kao PC, Vecchio JA, Hyman NH, West AB, Blaszyk H. Idiopathic myointimal hyperplasia of mesenteric veins: a rare mimic of idiopathic inflammatory bowel disease. *J Clin Gastroenterol.* (2005) 39:704–8. doi: 10.1097/00004836-200509000-00011
56. Savoie LM, Abrams AV. Refractory proctosigmoiditis caused by myointimal hyperplasia of mesenteric veins: report of a case. *Dis Colon Rectum.* (1999) 42:1093–6. doi: 10.1007/BF02236711
57. Genta RM, Haggitt RC. Idiopathic myointimal hyperplasia of mesenteric veins. *Gastroenterology.* (1991) 101:533–9. doi: 10.1016/0016-5085(91)90035-j
58. Li H, Shu H, Zhang H, Cui M, Gao Y, Tian F. Idiopathic Myointimal hyperplasia of the mesenteric veins: a case report and scoping review of previously reported cases from clinical features to treatment. *Front Med (Lausanne).* (2022) 9:855335. doi: 10.3389/fmed.2022.855335
59. Shi Q, Chen D, Wu H, Wang Y, Liu S, Sun Q. *Chin J Inflamm Bowel Dis.* (2022) 6:275–7. doi: 10.3760/cma.j.cn101480-20211203-00098
60. Morimura F, Edo H, Niwa T, Sugiura H, Suyama Y, Okazaki S, et al. Idiopathic myointimal hyperplasia of mesenteric veins: radiological evaluation using CT angiography. *BJR|Case Reports.* (2024) 10:uaad009. doi: 10.1093/bjrcr/uaad009
61. Bhatt H, Moreira RK, Shawki SF, Rumer KK. Atypical presentation of a rare disorder; idiopathic myointimal hyperplasia of mesenteric veins (IMH MV): report of two cases. *Int J Surg Case Rep.* (2023) 111:108839. doi: 10.1016/j.ijscr.2023.108839
62. Noujaim MG, Tang H, Kalisz K, Iranzad N, Wild D. Ischemic colitis due to idiopathic myointimal hyperplasia of the mesenteric veins. *ACG Case Rep J.* (2023) 10:e01125. doi: 10.14309/crj.0000000000001125