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When It's Not IBD: A Clinician's Map Through the Mimic Maze

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Abstract

Background/Objectives: Inflammatory bowel disease (IBD) may present with nonspecific symptoms and diagnosis may be challenging. Therefore, many diseases may mimic the clinical presentation, endoscopic findings, and histologic features of IBD.

Methods: In this article, we will review some common mimics of IBDs causes. For each of the mimics, we will discuss the common features and the other findings that can help to distinguish those mimics from true IBD.

Results: The diverse presentation of IBD and IBD-mimics necessitates consideration of the differential diagnosis, and reassessment of treatment in presumed IBD patients without successful clinical response.

Conclusion: The identification of IBD mimics is essential for effective management and treatment of patients with gastrointestinal symptoms. Gastroenterologists must utilize a combination of diagnostic tools to differentiate between IBD and its mimics effectively, ensuring patients receive appropriate care tailored to their actual condition.

Keywords: IBD; Colitis; Ischaemic; Diverticulosis; Radiation; Microscopic; Diversion; Infectious; NSAIDs; Checkpoint inhibitors

Abbreviations: IBD: Inflammatory Bowel Disease; CD: Crohn's Disease; UC: Ulcerative Colitis; JAK: Janus Kinase; CC: Collagenous Colitis; LC: Lymphocytic Colitis; PPI: Proton Pump Inhibitors; IELs: Intraepithelial Lymphocytes; ICI: Immune Checkpoint Inhibitor; IRAE: Immune-Related Adverse Event; GI: Gastrointestinal; CTCAE: Common Terminology Criteria for Adverse Events; EGD: Esophagogastroduodenoscopy

Introduction

A collective term "Inflammatory bowel disease (IBD)" is used for immune mediated disorders including Crohn's disease and Ulcerative colitis. However, they involve different sites of gastrointestinal tract [1]. Crohn's disease can cause destruction of any part of GI tract, but it majorly involves the ileum causing the inflammation to spread through multiple layers of GI tract which is referred to as transmural inflammation. Contrarily, in ulcerative colitis, the inflammation is restricted to the mucosal layer of colon and rectum only [2]. Clinical manifestation of both the diseases is the same, including diarrhea, bowel urgency, abdominal pain, GI bleeding, weight loss and malnutrition [3]. Similarly, the complications may involve bowel obstruction, perforation, fistula and abscess in Crohn's disease. Perforated bowel and toxic megacolon are the complications of ulcerative colitis. However, 25% of the IBD patients also tend to have extraintestinal manifestations. Relapse and remittance are a significant characteristic IBD [4].

IBD shares the global disease burden, and its incidence and prevalence are increasing worldwide; specifically in the Asian immigrants in the western countries [5]. Global Burden of Disease, Injuries and Risk factors revealed that the prevalence of IBD in the United Kingdom is 0.45% which is 449.6 per 100,000 [6] . As the etiology and pathogenesis of IBD is still unknown; hence, the nonspecific drug regimen is used to treat almost all cases of IBD either classified or Unclassified [7]. Currently, there is no definitive test considered the gold standard for diagnosing inflammatory bowel disease (IBD). Diagnosis relies on a combination of clinical evaluation, endoscopic examination, histological analysis, imaging studies, and laboratory tests. However, reaching a diagnosis can be difficult when symptoms are nonspecific or when endoscopic and histological results are inconsistent. Further complicating the diagnostic process, up to 14% of patients initially diagnosed with either Crohn's disease (CD) or ulcerative colitis (UC) experience a change in diagnosis over time-most often from UC to CD, particularly after undergoing ileal pouch-anal anastomosis [8].

Moreover, studies suggest that approximately 80% of patients initially diagnosed with IBD-unclassified (IBD-U) are eventually reclassified as having either CD or UC within eight years [9]. There are other diseases that mimic IBD as the patient symptoms can persist verily. Certain infections (like chloridoids difficile infection), drugs (like NSAIDS) and environmental factors can cause the GI disturbance and complicate the process to reach to a certain diagnosis

Discussion

Assessment and approach to IBD:

IBD encompasses two main autoimmune conditions requiring complex multi-disciplinary approach (MDT) approach towards effective management. The diagnostic journey is mainly initiated by focused history taking and clinical evaluation. This is followed by evaluation of biochemical markers including CRP to facilitate identification of active disease [10]. Moreover, the role of faecal calprotectin is worth emphasizing as a useful tool for IBD diagnosis. Faecal calprotectin is a cytosolic neutrophil protein which is released through neutrophil breakdown. This useful tool can aid in diagnosis and assessment of response to therapy [11]. In addition, stool cultures, parasite screening, and tests for Chloridoids difficile are important to exclude infectious causes. Next in line is endoscopic investigations for direct visualization of inflamed mucosa and obtaining tissue samples for diagnosis.

Ileocolonoscopic is the investigation of choice and gold standard for diagnosis of IBD. Endoscopic investigation not only serves diagnostic role, but it also helps to assess remission/ response to therapy. Typical endoscopic findings of Crohn's disease or Ulcerative Colitis help in scoring the disease (in terms of severity) to guide treatment plans [12]. Use of Video Capsule Endoscopy has revolutionized diagnosis of IBD and Crohn's in particular. Small bowel capsule evaluation for diagnosis and management of Crohn's disease helps to investigate for jejunal disease as it is a risk factor for strictures requiring surgical management. Therefore, capsule endoscopy is of great importance in this aspect of IBD. It is notable that capsule retention remains a cause of concern in this investigation type [13]. Discussing about various imaging modalities CT imaging helps in assessment of complications such as abscesses, fistulas, perforations, strictures etc., [14]. However, MR Enterography takes a lead as it is fundamental and complementary in the assessment of Crohn's disease diagnosis and follow up [15]. It helps to identify inflammatory signs i.e. mural thickening/ perivisceral oedema/ post contrast enhancement/ lymphadenopathies etc., in Crohn's disease. It is important to note that Intestinal ultrasound is gaining popularity given high sensitivity and specificity to identify intestinal inflammation/ lesions and complications e.g. strictures/ abscess/ fistulas etc., [10].

Medical management strategies include various new and emerging drugs with different mechanisms of action. Amino salicylates are small molecules- most commonly prescribed to reduce intestinal inflammation. Corticosteroids are also aimed to provide anti-inflammatory effect for short term management. Immunomodulators including azathioprine, mercaptopurine etc., are helpful in maintaining remission [16]. Amongst biologic therapies, Anti- Tumor necrosis factor α (anti-TNFα) agents (i.e., infliximab, adalimumab) are the mainstay for IBD management. Other treatment options are an exhaustive list including Anti- interleukin agents (i.e. Ustekinumab, Risankizumab etc.,), Janus Kinase (JAK) inhibitors (i.e. Tofacitinib, Upadacitinib etc.,) and Anti- adhesion agents (i.e. vedolizumab, natalizumab etc.,) which target inflammatory pathways and cellular signaling in the pathogenesis of IBD [17]. Due to concerns of disease progression and complications or an increased risk of colon cancer, follow up endoscopies are scheduled. These help to monitor patient's response to therapy and mucosal healing [18]. Ultimately, the diagnosis and evaluation of IBD is a continuous process that requires regular reassessment to reflect shifts in disease activity and how the patient responds to treatment.

IBD Mimics

Below are highlights of some of IBD mimics:

Ischaemic colitis

Ischaemic colitis occurs when the blood flow to the colon is inadequate to meet its metabolic needs, leading to mucosal damage that can progress to full-thickness (transmural) necrosis. In about 75% of cases, it affects the left side of the colon and can resemble left-sided inflammatory bowel disease (IBD), often presenting with abdominal pain and rectal bleeding. Clinicians should consider this diagnosis in patients with sudden symptom onset and known vascular risk factors. On CT imaging, segmental colitis is observed in up to 40% of cases, especially in watershed regions; a "target sign"-representing submucosal swelling between areas of enhanced mucosa and serosa-is highly indicative. Right-sided colonic involvement tends to signal more severe disease. During endoscopy, common findings include a solitary longitudinal ulcer (known as the "single-stripe sign") and darkened (dusky) mucosa, which may point to gangrenous changes. Histologically, ischaemic colitis is associated with lamina propria haemorrhage, granulation tissue with crypt abscess, and crypt destruction. In contrast to Crohn's disease, the rectum is usually spared, and the demarcation of inflammation is normally clear in ischaemic colitis [19].

Disease recurrence is uncommon in ischaemic colitis, however it is associated with worse long-term prognosis compared with IBD. In a Spanish cohort study involving 135 participants with ischaemic colitis treated medically or surgically, the overall 5-year recurrence rate was only 9.7%, but notably the 5-year mortality rate was 31% [20]. Most of these deaths were due to pre-existing comorbidities. Conversely, although approximately 80% of patients with Crohn's disease and 20% with ulcerative colitis require bowel surgery during their lifetimes, a recent Norwegian population-based longitudinal study found similar mortality rates between controls and those with IBD. This could be due to the advancements in medical therapies in IBD [21].

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Segmental colitis associated with diverticulosis

Segmental colitis associated with diverticulosis is an uncommon cause of chronic diarrhea, crampy abdominal pain (particularly in the left lower quadrant) and episodic rectal bleeding. The aetiopathogenesis and disease course are poorly understood; however, it typically affects the sigmoid, and colonic inflammation of surrounding areas of diverticula is observed. Endoscopic and histologic appearances may mimic IBD, although there is rectal sparing. Treatment is typically with ciprofloxacin or metronidazole, whereas those with persistent symptoms may respond to mesalazine or glucocorticoids [22]. SCAD and IBD mimic each other as they share indistinguishable pathological and therapeutic attributes [23]. Additionally, the histologic features of SCAD and IBD includes nonspecific chronic inflammation which makes it difficult to distinguish them [24]. However, the location of anomaly, presence of fever, leucocytosis and blood profile are the distinctive traits found in IBD and make them differ from each other [25]. Studies show that post-diverticulitis, the risk of IBD increases in patients [26]. Also, the prognosis becomes much worse, and morbidity chances are more in patients suffering from diverticulitis along with IBD as compared to IBD alone [27].

Radiation-induced colitis

Radiation-induced colitis is a recognized adverse effect of lower abdominal radiation therapy. It is associated mostly with radiotherapy for prostate and cervical cancers. Acute radiation-induced colitis happens within 3 months of treatment, whereas there may be a latent period of months or years before chronic radiation-induced colitis manifests. Up to 80% of those receiving pelvic or abdominal radiotherapy may experience acute intestinal toxicity symptoms, and half of these patients experience chronic intestinal problems. Although the advent of radiotherapy has improved cancer survival, radiation-induced colitis significantly impacts one's quality of life. Risk factors for radiation-induced colitis include long treatment duration, high radiation dose, and significant area of bowel involvement. Patient-related factors including a low body mass index (BMI), a history of IBD and previous abdominal surgery may also contribute [28].

Patients with acute radiation-induced colitis usually present with non-bloody diarrhea, abdominal pain, and nausea. These symptoms generally resolve within a few weeks of the completion of treatment. This acute phase is due to the release of reactive oxygen species during radiation, which can cause damage to the gut wall leading to intestinal inflammation and ulceration. In those with chronic radiation-induced colitis, bloody diarrhea, flatulence, abdominal pain, and weight loss are recognized symptoms. Years after the initial injury, scar tissues may accumulate within the submucosa leading to occlusive vasculitis, and atrophy of the mucosa. Treatment for radiation-induced colitis entails mainly symptomatic control, such as managing diarrhea. Bile acid sequestrants, hyperbaric oxygen therapy, and argon plasma coagulation have also been suggested as effective treatment options.

The diagnosis of radiation-induced colitis relies on the elicitation of recent or historic radiotherapy from the history. In the acute phase, endoscopic findings may include mucosal erythema, oedema, and ulceration. In the chronic phase, mucosal pallor, telangiectasia, and strictures may be seen on endoscopy. Histologically, there may be leucocyte infiltration and crypt abscess acutely, and foam cell invasion and hyaline thickening of walls of blood vessels in longstanding disease. Radiation-induced colitis is normally segmental, and the rectum is typically the most affected part of the bowel. The key to differentiating this condition from IBD is spotting telangiectasia on endoscopy and the presence of hyalinization in histology, which are both rare in IBD [29].

Microscopic colitis

First described in the 1980s, microscopic colitis is a chronic inflammatory disorder affecting the large intestine, mainly consisting of collagenous colitis (CC) and lymphocytic colitis (LC). The pathophysiology is multi-factorial, with dysregulated host response, microbiome, and genetic susceptibility all likely to be implicated. The overall incidence rate was estimated to be 11.4 cases per 100,000-person years8. It typically affects the elderly population, although 25% of those with microscopic colitis are diagnosed under the age of 45, and rare cases among children have been described in the literature8. It is associated with female sex, smoking, medications including NSAIDs and proton pump inhibitors (PPI), and autoimmune conditions particularly coeliac disease [30].

The clinical manifestations of LC and CC are very similar. An insidious onset of watery diarrhea, faecal urgency and faecal incontinence are key clinical features. As opposed to IBD, the diarrhea is not normally bloody, and abdominal pain is less common. The condition normally follows a remitting-relapsing pattern, with most people responding well to budesonide as a first-line treatment initially but frequently relapse on cessation of treatment. Other management options include antidiarrheal agents, bile acid sequestrants, biologics, and occasionally surgery. Compared to IBD, serious complications such as colonic perforation is significantly rarer in microscopic colitis. Furthermore, the risk of colorectal cancer is not increased in microscopic colitis, and some studies have even suggested that it is a protective factor [31]. A thorough history-taking identifying relevant risk factors, alongside convincing histopathological findings are essential to ascertaining a diagnosis.

The levels of faecal calprotectin, a non-specific marker of bowel inflammation, are generally lower in microscopic colitis compared to IBD. Endoscopy findings are classically normal or may show minimal non-specific findings such as patchy erythema, oedema, and petechiae; ulcers are rarely seen. The characteristic histological findings of CC include a thickened subepithelial collagenous band of ^{3}10mm, increased inflammatory infiltrate in the lamina propria dominated by mainly lymphocytes and plasma cells. Conversely, in LC the subepithelial collagenous band is not

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thickened, but there is an increased number of intraepithelial lymphocytes (IELs) exceeding 20 per 100 surface epithelial cells, and inflammatory infiltrate in the lamina propria. In both subtypes, the crypt architecture remains intact, unlike in IBD [32].

Diversion colitis

Diversion colitis is the inflammation of the segments of the colon that are diverted from the faecal stream as a result of surgery. The pathophysiology remains to be clarified, but it is thought to be due to the lack of exposure to luminal bacteria and the subsequent reduction in nutrients provided by these bacteria in the decommissioned bowel segments. Most patients with diverted bowel segments have diversion colitis, although only one-third of them will experience symptoms including rectal bleeding, abdominal pain, and tenesmus. Colitis usually develops 3 to 36 months following surgery. Having IBD may increase the risk of developing diversion colitis after bowel surgery, and reciprocally diversion colitis has been reported to trigger the development of IBD [33]. In people with diversion colitis and IBD, they are more likely to have symptoms and abnormal endoscopic findings compared to those with diversion colitis alone.

The gold standard management of diversion colitis entails restoring bowel continuity, although when this is not feasible, other treatments for instance short-chain-fatty-acid and 5-aminosalicyclic acid are also effective to varying extents. The prognosis is thought to be excellent, with clinical symptoms generally regressing upon reestablishment of the faecal stream. However, chronic histological inflammation even after restoration of the faecal stream has been described in the literature [34]. One should be vigilant about the possibility of diversion colitis as a differential diagnosis upon ascertaining previous bowel surgery in the past medical history. The endoscopic and histopathological findings of IBD and diversion colitis may closely resemble each other, together with the fact that one condition may predispose to the other, posing a significant diagnostic challenge. Endoscopy may show erythema, mucosal friability, oedema, and ulceration. The most recognized pathological finding is lymphoid follicular hyperplasia, although crypt distortion, crypt abscesses, and Paneth cell metaplasia may also be present [35].

Infectious colitis

Infectious colitis refers to the inflammation of the colon caused by infective pathogens. These pathogens can be bacterial (e.g. Shigella, Salmonella, Yersinia), viral (e.g. Cytomegalovirus, Norovirus), or parasitic (E. histolytica). It is a common cause of acute diarrhea and can be challenging to differentiate from IBD. Furthermore, IBD can be a predisposing factor for some gastrointestinal infections. The clinical manifestations vary depending on the underlying pathogen. Diarrhea, abdominal pain, tenesmus, and fever are common presenting features. The diarrhea may contain blood, as in cases caused by Campylobacter, Shigella, and E. histolytic, or non-bloody in colitis due to Norovirus. The onset of symptoms is usually sudden, with febrile illness and vomiting being prominent early clinical features generally. Moreover, some pathogens are associated with extra-intestinal manifestations, such as erythema nodosum and reactive arthritis in Yersinia, making it even more difficult to differentiate from IBD [36].

Infectious colitis should always be considered as a differential diagnosis in people presenting with a diarrheal illness. Careful history-taking should yield clues to the diagnosis, including a travel history, history of immunosuppression, and having contact with unwell persons. Microbiological investigations such as assessing faecal microscopy, culture, and sensitivity, as well as testing for parasites and cysts may be helpful in identifying the causative pathogen. Despite this, stool culture is only useful in diagnosing bacterial colitis in less than half the time. Inflammatory markers will largely be elevated in both IBD and infectious colitis, however a low albumin is more suggestive of IBD [37]. Endoscopic findings in infectious colitis are usually non-specific, showing ulceration, oedema, and erosion. Histologically, there may be inflammatory infiltration of the lamina propria and cryptitis, but preservation of the crypt architecture would favour infectious colitis over IBD as the diagnosis.

Yersinia enterocolitica infection primarily affects the terminal ileum, leading to mucosal ulcers, infiltration by neutrophils, and thickening of the ileal wall. The disease most commonly involves the distal ileum and cecum, with patients typically showing signs of small-bowel obstruction and a palpable, tender abdominal mass. Diagnosis is most accurately achieved through colonoscopy with biopsy and culture. Endoscopic findings characteristic of Yersinia include aphthoid ulcers in the cecum and terminal ileum, presenting as round or oval raised lesions with ulceration. Unlike Crohn's disease, the ulcers tend to be uniform in size and shape [38].

Mycobacterium avium-intracellular complex infection typically arises in individuals with advanced HIV or other forms of immunosuppression. It often presents as a widespread systemic illness characterized by diarrhea, abdominal pain, weight loss, fever, and malabsorption. Diagnosis is confirmed through culture of mucosal biopsy samples. Disseminated histoplasmosis may also affect the ileocecal region. Additionally, the terminal ileum can be involved in other infections such as those caused by Salmonella species and cytomegalovirus. Cases of ileitis have also been reported in Clostridium difficile infections [39].

Nonsteroidal anti-inflammatory drugs (NSAIDs)

NSAIDs can lead to various gastrointestinal complications. While they are most commonly associated with peptic ulcer disease, they can also cause inflammation and ulceration in the small intestine and colon [40]. Biopsy findings typically show signs of active, acute inflammation, although chronic changes may be seen in long-term users [41]. Prolonged NSAID use can result in small bowel strictures that resemble those seen in Crohn's disease. These strictures are characteristically smooth, symmetric, and diaphragm-like, and are considered diagnostic of chronic NSAID

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exposure. Histologically, they exhibit submucosal fibrosis and may also show chronic changes such as distortion of normal tissue architecture and pyloric metaplasia. Although these strictures most frequently affect the small intestine, the colon can also be involved [42].

Immune-based chemotherapy

Immune checkpoint inhibitor (ICI) colitis is the most frequently reported gastrointestinal (GI) immune-related adverse event (irAE). Patients with ICI colitis usually present with diarrhea of variable clinical frequency. Symptoms usually develop within a few weeks to a few months after starting therapy. In patients with more severe disease, diarrhea may be associated cramping abdominal pain, urgency, and rectal bleeding. Less typical symptoms such as fever, abdominal distension, and/or severe abdominal pain should raise suspicion for severe disease and/or possible colonic perforation or toxic megacolon [43]. The diagnosis of ICI colitis should be suspected in patients who are receiving ICI therapy and who develop diarrhea with or without bleeding or abdominal pain. The diagnosis is established clinically by assessing symptoms according to Common Terminology Criteria for Adverse Events (CTCAE) and excluding other causes (e.g., infection). The diagnosis may be further confirmed with endoscopic evaluation with biopsies. For all patients in whom ICI colitis is suspected, laboratory studies including blood tests and stool studies are obtained.

For patients with grade 2 to 4 symptoms and/or an elevated stool inflammatory marker, we typically perform a lower endoscopy (e.g., flexible sigmoidoscopy) with biopsies. While flexible sigmoidoscopy is the initial test of choice, some contributors start with an ileocolonoscopic to also assess for right-sided colitis or ileitis. Patients with persistent symptoms but with a normal flexible sigmoidoscopy or colonoscopy can subsequently be evaluated with upper esophagogastroduodenoscopy (EGD) [44]. Endoscopic and pathologic findings are similar to IBD; deep, serpiginous ulcerations may be found on endoscopy, and histologic specimens may show nonspecific acute and chronic inflammation [45]. Treatment includes early recognition, especially in a patient with the correct clinical history, ensuring that cytomegalovirus has been ruled out, and initiation of corticosteroids or, in refractory cases, infliximab. Also, discussion with oncology about the possibility of chemotherapy discontinuation would be important especially in those with severe refractory colitis [46].

Endometriosis- a mimic of IBD

Endometriosis is a chronic progressive inflammatory disease with presence of endometrial tissue outside of the uterus for example in ovaries, muscular layers of uterus, peritoneum, gastrointestinal tract and urinary tract etc. It has a global prevalence of 10%, mainly affecting premenopausal women. Various forms of endometriosis include ovarian, superficial peritoneal and deep infiltrating forms (including bowel endometriosis). Symptoms associated with Deep infiltrating endometriosis including diarrhea, intestinal cramping, bloating etc. are overlapping and frequently misdiagnosed with IBD. The most common sites of deep infiltrating endometriosis in bowels is rectosigmoid- estimated to be 65.7%, followed up by 20% involvement of ileocecal junction and 15% of cases involve rectum. Endometriosis share common Gastrointestinal symptoms along with dysmenorrhea, dyspareunia.

However, evidence depicts variation in presenting symptoms. Hence, endometriosis mimics IBD and differentiation between the two becomes clinically challenging. Ultimately, misdiagnosing and mistreating endometriosis for IBD. In addition to clinical resemblance, intestinal endometriosis and IBD share similar endoscopic and sometimes even histologic (mucosal architecture distortion and/ or inflammation like IBD) features. Histological diagnosis is considered gold standard for endometriosis. Studies show that thorough history taking and examination with diagnostic lab tests and radiological imaging are also crucial for diagnostic purposes. Gynecologists and Gastroenterologists should understand the importance of underlying differential or concomitant diagnosis of these conditions, ensuring timely referral to relevant specialties and using an MDT approach to guide in patient management [47].

Amyloidosis- a mimic of IBD

Accumulation of amyloid fibrils in the form of insoluble β-pleated sheet in extracellular space, which may cause disruption in organ function, is known as amyloidosis. Immunoglobulin-light- chain- related amyloidosis (AL) and reactive amyloidosis (AA) are the two common types that cause intestinal amyloidosis. According to research in England, incidence rate of AL amyloidosis is 3 cases per million person-years, whereas incidence of AA amyloidosis is 1 case per million person-years. In patients suffering from systemic AL amyloidosis, 3-28% are estimated to have GI tract involvement. . Highly sensitive whole body I- labelled SAP scintigraphy is useful scan for diagnosis of AA and AL amyloidosis, assessment of extent of disease/ organ involvement and monitor response to treatment [48].

However, Definitive diagnosis for amyloidosis relies on biopsy. Histologically, acellular, eosinophilic appearance on microscopy and display of characteristic apple-green birefringence under polarized light on Congo red stain are key features for amyloidosis. . It is interesting to note that there are no specific findings of amyloid deposition seen on endoscopy. Intestinal amyloidosis (primarily small intestine) mimics IBD as they share common symptoms including chronic diarrhea, malnutrition, weight loss, intestinal bleeding and ulceration, intestinal dysmotility and dysphagia, pseudo-obstruction and perforation. Hence, misleading the diagnosis. . It is imperative that early diagnosis is obtained for amyloidosis to prevent organ dysfunction and provision of timely treatment [49].

Conclusion

IBD remains an important differential diagnosis in people presenting with diarrhea and gastrointestinal symptoms. However, many conditions may mimic the clinical presentation and

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investigation findings of IBD. The prompt diagnosis and appropriate treatments are integral to effective IBD care. Conversely, misdiagnosing an IBD mimic as true IBD may lead to the delay in the treatment and worsening of the underlying condition. Whilst endoscopy plays an important role in the diagnostic work-up, it is imperative to interpret its findings in the context of the clinical presentation and histology results, recognizing that many mimics share similar endoscopic appearances with IBD.

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