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Review

Clinical Status and Extra-Intestinal Findings of Inflammatory Bowel Disease

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Abstract

Ulcerative colitis (UC) is a chronic, idiopathic inflammatory disease characterized by periods of remission and flare-ups. It begins in the rectum and progresses proximally, most commonly affecting adults aged 30–40 years. In Crohn's disease (CD), the clinical presentation can vary depending on the extent of the disease. The most common symptoms include cramping abdominal pain and diarrhea. Patients with severe disease may also experience fever and weight loss. The transmural inflammatory process can result in fibrotic strictures, leading to recurrent episodes of ileus. In cases of aggressive inflammation, the condition may lead to the development of fistulas and abscesses.

Inflammatory bowel disease (IBD) is a systemic condition that affects both the gastrointestinal system and extra-intestinal systems in many patients. Most extra-intestinal manifestations (EIMs) are associated with active episodes of intestinal inflammation. These include aphthous ulcers, type 1 peripheral arthritis, erythema nodosum (EN), and episcleritis. Other EIMs, such as ankylosing spondylitis and type 2 peripheral arthritis, occur independently of bowel disease activity.

Keywords: Inflammatory bowel disease, clinical status, extra-intestinal manifestations

INTRODUCTION

Ulcerative colitis (UC) is a chronic, idiopathic inflammatory disease that affects the colon and is most commonly seen in adults aged 30–40 years. Mucosal inflammation associated with UC, which begins in the rectum and progresses to the proximal portions of the colon, is characterized by periods of attacks and remissions. Most patients with UC present with complaints of diarrhea and blood in the feces, and up to 15% of patients may exhibit severe disease symptoms.[1] Symptoms include an increased frequency of defecation; soft, mucous, and/or bloody feces; a feeling of urgent need to defecate; mucous discharge; nocturnal defecation; incontinence; fatigue; and abdominal pain, which is usually localized in the left lower quadrant (in cases of whole colon involvement and/or toxic megacolon).

Abdominal pain in the form of cramps is more commonly seen in Crohn's disease (CD).[2] Fever and weight loss may also occur in patients with severe UC. The clinical presentation varies depending on the extent of the disease. For example, urgency and tenesmus are more common in patients with proctitis, while bloody diarrhea and abdominal pain are more pronounced in patients with pancolitis. Physical examination findings are typically normal in patients with mild disease but may include pallor, fever, hypotension, tachycardia, abdominal tenderness, and other symptoms in those with moderate to severe disease. Blood may be detected during a rectal examination.

Patients with prolonged diarrhea may also exhibit signs of peripheral edema, subcutaneous fat loss, and muscle wasting due to malnourishment. Chronic diarrhea can cause skin degradation, which may result in the development of skin tags or anal fissures.

Tympanic sounds on percussion and abdominal swelling may indicate colonic dilation, necessitating an urgent radiographic examination. Symptom severity in ulcerative colitis can range from mild disease, characterized by four or fewer defecations per day with or without blood, to severe disease, which involves severe cramps, continuous bleeding, and more than ten defecations per day.[3]

CD is characterized by transmural inflammation, and any part of the gastrointestinal system, from the oral cavity to the perianal region, may be affected. The main symptoms of CD include abdominal pain, diarrhea, fatigue, fever, and weight loss.[4] Patients with disease limited to the distal ileum typically present with lower right quadrant abdominal pain as an initial symptom.

The transmural inflammatory process in CD can lead to fibrotic strictures, often causing recurrent episodes of abdominal pain and small intestine obstructions, or, less commonly, obstructions in the colon. In cases of aggressive inflammation, this process may result in the development of fistu-

las and abscesses. Diarrhea is a common symptom caused by excessive fluid secretion from the inflamed small or large intestine, impaired fluid absorption, and bile salt malabsorption due to terminal ileum inflammation, bile salt loss, or enteroenteric fistulas. Fever occurs less frequently and may stem from the inflammatory process itself or from complications such as intestinal perforation and intra-abdominal abscesses.[5]

CD-induced transmural intestinal inflammation can also result in fistula and abscess formation. Fistulas are abnormal pathways that connect two epithelium-lined organs. For example, fistulas may connect the intestine to the bladder (enterovesical), the skin (enterocutaneous), another segment of the intestine (enteroenteric), or the vagina (enterovaginal). Enteroenteric fistulas may be asymptomatic but can sometimes cause diarrhea. Enterovesical fistulas frequently lead to recurrent urinary tract infections, while retroperitoneal fistulas can cause urethral obstructions. Enterovaginal fistulas often manifest as gas or feces exiting through the vagina. Enterocutaneous fistulas result in discharge from the skin surface.[6]

In cases of CD associated with upper gastrointestinal system (GIS) involvement, aphthous ulcers in the mouth and gingival pain may occur. Esophageal involvement may occasionally present with odynophagia or dysphagia. Patients with gastroduodenal involvement may experience upper abdominal pain, nausea, and/or vomiting, particularly after eating.[7]

EXTRA-INTESTINAL FINDINGS

Inflammatory bowel disease (IBD) is a systemic condition that can affect both the gastrointestinal system and extra-intestinal systems in many patients.[8] Extra-intestinal manifestations (EIMs) occur in 25–40% of IBD patients, and the presence of one EIM increases the risk of developing additional EIMs. Approximately 25% of individuals are diagnosed with an EIM before receiving an IBD diagnosis; however, most patients are diagnosed with EIMs after their IBD diagnosis.[9]

In a large IBD cohort study, peripheral arthritis or axial arthritis, uveitis, and primary sclerosing cholangitis were identified as the most common EIMs diagnosed prior to the onset of IBD. These EIMs were found to have a significant impact on the quality of life of IBD patients.[10]

The severity and development of EIMs, as well as their correlation with intestinal IBD activity, vary among patients. Most EIMs are associated with ongoing intestinal inflammation, including aphthous ulcers, type 1 peripheral arthritis, erythema nodosum (EN), and episcleritis. However, other EIMs, such as ankylosing spondylitis and type 2 peripheral arthritis, occur independently of intestinal disease activity (Table 1).[11]

MAIN POINTS

- Most patients with ulcerative colitis present with bloody diarrhea. Up to 15% may also experience symptoms of severe disease.
- Other symptoms in UC patients include an increased frequency of stools, soft-textured stools, mucus and/or bloody stools, an urgent need to defecate, mucus discharge, nocturnal defecation, incontinence, fatigue, and abdominal pain.
- The primary symptoms of Crohn's disease are abdominal pain, diarrhea, fatigue, fever, and weight loss. When the disease is confined to the distal ileum, patients typically first report right lower quadrant abdominal pain.
- Extra-intestinal manifestations develop in 25–40% of inflammatory bowel disease patients, and the presence of one EIM increases the risk of developing subsequent EIMs.
- Aphthous ulcers, type 1 peripheral arthritis, erythema nodosum, and episcleritis are associated with disease activity, whereas other EIMs, such as ankylosing spondylitis and type 2 peripheral arthritis, are independent of bowel disease activity.

The prevalence of EIMs has been reported to range between 6% and 47% of all IBD patients. Patients can experience multiple EIMs, with more than 20% of IBD patients reporting two different EIMs and 10% reporting three or more. EIMs can emerge either before or after an IBD diagnosis. In 26% of all patients with EIMs, the EIM was identified up to 25 months (median: 5 months) before the IBD diagnosis.[10]

MUSCULOSKELETAL SYSTEM EXTRA-INTESTINAL MANIFESTATIONS

In IBD, seronegative spondyloarthropathies are the most common EIMs. The reported prevalence of these EIMs varies depending on the clinical and/or radiological criteria applied, ranging from 6% to 46%.[12] The prevalence of arthritis in IBD decreases with increasing age.[13] Young age and colonic involvement are recognized as significant risk factors.

Peripheral arthritis typically presents as migratory arthritis and does not cause significant joint deformity. It affects 10–20% of CD patients and 5–14% of UC patients. Peripheral arthritis is categorized into two types: type 1 and type 2. Type 1 Peripheral Arthritis is seronegative, asymmetrical, and pauciarticular (usually affecting fewer than five joints). It generally occurs in conjunction with active disease. The knees are the most commonly affected joints, followed by the shoulders, hips, wrists, ankles, and elbows. Type 2 Peripheral Arthritis typically affects the metacarpophalangeal joints, is symmetrical, and is polyarticular (involving more than five joints). Unlike type 1, type 2 is not associated with intestinal disease activity. Type 2 peripheral arthritis has been found to increase the risk of uveitis.[14]

Table 1. Relationship between EIM Activity and Bowel Activity

EIM	Associated with clinical activity	Relationship with clinical activity unclear	Unrelated to clinical activity
Axial Arthropathy			X
Peripheral Arthropathy	X (Tip 1)		X (Tip 2)
Erythema Nodosum	X		
Pyoderma Gangrenosum		X	
Sweet Syndrome	X		
Oral Aphthous Ulcer	X		
Episcleritis	X		
Uveitis		X	
Primary Sclerosing Cholangitis		X	

The treatment of type 1 peripheral arthritis focuses on managing the underlying intestinal disease, whereas treatment for type 2 peripheral arthritis is symptom-based and may include rest, intra-articular steroid injections, physiotherapy, or sulfasalazine. Celecoxib, a selective COX-2 inhibitor, has been shown to be a potential treatment option and can be safely used in IBD patients, as it has not been associated with disease flare-ups.[14]

Axial arthropathies, which are less common than peripheral arthritis, include ankylosing spondylitis and sacroiliitis. These conditions are more prevalent in males and do not typically occur concurrently with gastrointestinal flare-ups. Ankylosing spondylitis is characterized by lumbar pain and stiffness, which tend to worsen in the morning or at night but improve with physical activity.

IBD-related sacroiliitis is usually bilateral and can be either symptomatic or asymptomatic. Imaging studies have shown that approximately 50% of patients with CD have asymptomatic sacroiliitis. The prevalence of clinical sacroiliitis is estimated to be around 8%, and in 3–6% of cases, it is found alongside both axial and peripheral joint disease.

While most patients experience recovery from intestinal inflammation, joint disease often persists, especially in cases involving polyarticular involvement. In such instances, a multidisciplinary approach to treatment is recommended to effectively address both disorders.[15]

DERMATOLOGICAL EXTRA-INTESTINAL MANIFESTATIONS

Cutaneous EIMshave been reported in 5–15% of IBD patients. EN and pyoderma gangrenosum (PG) are the most frequently observed skin EIMs in IBD patients.[16]

EN is characterized by red, swollen, tender nodules, approximately 1–5 cm in diameter, which are typically found on the extensor surfaces of the lower extremities, most commonly in the anterior tibial region. The prevalence of EN in IBD patients has been reported as 5–15% in CD and 2–10% in UC.[11] In a Swiss IBD cohort study, EN was observed in 6.8% of patients with inactive CD and 2.4% of those with active CD, contrasting with the common belief that EN is linked to active CD. Among UC patients, EN was seen in 2% of those with inactive disease and 4.7% of those with active inflammation.[12] Diagnosis is based on clinical evaluation, and a skin biopsy is not typically required. Since EN often occurs alongside intestinal disease activity, effective management of intestinal inflammation is essential.

PG typically begins as an erythematous pustule or nodule that rapidly develops. It can occasionally progress to deep ulcers or purulent material at the base of an ulcer that remains sterile in culture.[17] The prevalence of PG in IBD patients ranges from 0.4% to 2.6%.[18] While PG is most commonly found on the lower extremities, it can develop on any part of the body, particularly at postoperative stoma sites on the abdominal wall.[18] Pathergy, a significant physiological reaction to minor trauma or injury, is frequently observed in PG. As a result, lesion biopsy should be avoided, and diagnosis should be made clinically. PG has been reported in 1.4% of patients with inactive CD and 2.4% with active CD, as well as in 1.5% of those with inactive UC and 3% with active UC.[12] PG is more common in females than males. While the relationship between PG and intestinal disease activity remains unclear, it may improve with appropriate IBD treatment.

Acute febrile neutrophilic dermatosis (Sweet syndrome) is a rare EIM characterized by the sudden onset of tender, erythematous papules and plaques on the upper extremities, trunk, and face, accompanied by fever and leukocytosis. Sweet syndrome has been reported to develop prior to an IBD diagnosis (20% of cases), concurrently with IBD (28%), or following an IBD diagnosis (52%).[17] Similar to arthritis, it is more common in women, tends to occur alongside intestinal disease activity, and is associated with other EIMs. Management of Sweet syndrome requires the use of topical or systemic steroids in conjunction with IBD-specific medications.

ORAL PATHOLOGIES

There is ongoing debate about whether oral lesions in the upper gastrointestinal tract should be considered EIMsof IBD or simply signs of the disease. The prevalence of oral lesions in IBD patients has been reported to range from 5% to 50%.[18] Individuals with IBD frequently develop oral lesions, with young age, male gender, and CD status identified as risk factors.[18]

The classic oral lesion associated with IBD is aphthous stomatitis, which presents as painful ulcerations that can cause dysphagia or odynophagia. These lesions are typically found along the buccal and labial folds. Aphthous stomatitis has been reported in up to 25% of CD cases and around 10% of UC cases.[19] The severity of aphthous stomatitis often increases with active intestinal disease. Topical steroids are the first line of treatment, and anesthetics and anti-inflammatory medications are also used to address both the oral lesions and the intestinal disease activity.

Periodontitis, a chronic inflammatory condition characterized by swelling, bleeding of the gingiva, and destruction of bone and soft tissue, is also commonly observed in IBD patients.[20] Both aphthous stomatitis and periodontitis are generally associated with active intestinal disease, particularly in cases of perianal disease.

OCULAR EXTRA-INTESTINAL MANIFESTATIONS

Approximately 2–7% of IBD patients experience ocular problems. Episcleritis, scleritis, and anterior uveitis are the most commonly observed ocular EIMs in IBD.[21]

Episcleritis is a relatively benign condition that does not impair vision. It typically occurs concurrently with intestinal disease activity and presents with pain and redness in the eyes. Scleritis, which affects the deeper layers of the eye, is a more severe condition. If not diagnosed and treated promptly, scleritis can lead to vision problems. It often causes significant ocular discomfort, occasionally severe enough to wake the patient from sleep. Effective management of intestinal inflammation is critical in these cases.[22]

Uveitis is inflammation of the middle layer of the eye, which includes the iris, ciliary body, and choroid. Patients with uveitis often present with headaches, blurred vision, and ocular pain. In IBD, anterior uveitis is the most frequently reported form. Unlike episcleritis and scleritis, uveitis is less strongly associated with intestinal disease activity.

A Swiss IBD cohort study reported uveitis in 5.2% of patients with non-active CD, in 12% of those with active CD, in 3.5% of UC patients with non-active disease, and in 4.1% of those with active UC.[12]

HEPATOBILIARY EXTRA-INTESTINAL MANIFESTATIONS

Primary sclerosing cholangitis (PSC) is the most significant hepatobiliary EIM observed in IBD patients. Between 60% and 80% of PSC patients have underlying IBD.[23] The frequency of PSC is up to 5%, particularly in patients with UC and colonic CD.[24]

PSC is a chronic cholestatic liver disease characterized by fibrosis of the intrahepatic and extrahepatic bile ducts. Risk factors for PSC development in UC patients include pancolitis, a history of appendectomy, and male gender.[25] Additional testing for PSC should be performed in IBD patients with elevated serum gamma-glutamyl transferase (GGT) or alkaline phosphatase levels.

Histologically, PSC is defined as an inflammatory process that triggers lymphocyte infiltration of the intrahepatic and extrahepatic bile ducts, followed by fibrosis. This can lead to strictures in the large or small bile ducts, potentially progressing to liver cirrhosis, end-stage liver disease, and cholangiocarcinoma over time.[26]

More importantly, PSC is associated with a 10-fold increased risk of colorectal carcinoma in IBD patients.[27] As a result, colonoscopy should be performed at the time of PSC diagnosis and then annually thereafter. Additionally, annual ultrasound scans are recommended due to the high risk of malignancy from gallbladder polyps associated with PSC. If polyps are detected, cholecystectomy should be performed.

UNCOMMON EXTRA-INTESTINAL MANIFESTATIONS

Acute idiopathic pancreatitis is a rare EIM of CD and must be differentiated from pancreatitis caused by IBD-specific drugs, such as azathioprine or, in rare cases, 5-ASA.[28] Autoimmune pancreatitis can also occur in IBD patients, with type 2 autoimmune pancreatitis being more common in this population than in the general population.[29]

Interstitial lung disease is associated with UC, while granulomatous lung disease is more commonly linked to CD. Bronchopulmonary EIMs may develop in some UC patients even after colectomy.[30]

EIMs significantly increase the disease burden in IBD patients. A multidisciplinary team approach is essential for adequately managing EIMs and improving patients' quality of life.

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REFERENCES

1. Torres J, Billioud V, Sachar DB, Peyrin-Biroulet L, Colombel JF. Ulcerative colitis as a progressive disease: the forgotten evidence. *Inflamm Bowel Dis*. 2012;18(7):1356-1363. [CrossRef]
2. Baumgart DC, Sandborn WJ. Inflammatory bowel disease: clinical aspects and established and evolving therapies. *Lancet*. 2007;369(9573):1641-1657. [CrossRef]
3. Silverberg MS, Satsangi J, Ahmad T, et al. Toward an integrated clinical, molecular and serological classification of inflammatory bowel disease: report of a Working Party of the 2005 Montreal World Congress of Gastroenterology. *Can J Gastroenterol*. 2005;19 Suppl A:5A-36A. [CrossRef]
4. Lichtenstein GR, Loftus EV, Isaacs KL, Regueiro MD, Gerson LB, Sands BE. ACG Clinical Guideline: Management of Crohn's Disease in Adults. *Am J Gastroenterol*. 2018;113(4):481-517. Erratum in: *Am J Gastroenterol*. 2018;113(7):1101. [CrossRef]
5. Sands BE. From symptom to diagnosis: clinical distinctions among various forms of intestinal inflammation. *Gastroenterology*. 2004;126(6):1518-1532. [CrossRef]
6. Schwartz DA, Loftus EV Jr, Tremaine WJ, et al. The natural history of fistulizing Crohn's disease in Olmsted County, Minnesota. *Gastroenterology*. 2002;122(4):875-880. [CrossRef]
7. Annunziata ML, Caviglia R, Papparella LG, Cicala M. Upper gastrointestinal involvement of Crohn's disease: a prospective study on the role of upper endoscopy in the diagnostic work-up. *Dig Dis Sci*. 2012;57(6):1618-1623. [CrossRef]
8. Garber A, Regueiro M. Extraintestinal Manifestations of Inflammatory Bowel Disease: Epidemiology, Etiopathogenesis, and Management. *Curr Gastroenterol Rep*. 2019;21(7):31. [CrossRef]
9. Fine S. Extraintestinal Manifestations of Inflammatory Bowel Disease. *R I Med J (2013)*. 2022;105(9):13-19.
10. Vavricka SR, Rogler G, Gantenbein C, et al. Chronological Order of Appearance of Extraintestinal Manifestations Relative to the Time of IBD Diagnosis in the Swiss Inflammatory Bowel Disease Cohort. *Inflamm Bowel Dis*. 2015;21(8):1794-1800. [CrossRef]
11. Vavricka SR, Schoepfer A, Scharl M, Lakatos PL, Navarini A, Rogler G. Extraintestinal Manifestations of Inflammatory Bowel Disease. *Inflamm Bowel Dis*. 2015;21(8):1982-1992. [CrossRef]
12. Vavricka SR, Brun L, Ballabeni P, et al. Frequency and risk factors for extraintestinal manifestations in the Swiss inflammatory bowel disease cohort. *Am J Gastroenterol*. 2011;106(1):110-119. [CrossRef]
13. Karreman MC, Luime JJ, Hazes JMW, Weel AEAM. The Prevalence and Incidence of Axial and Peripheral Spondyloarthritis in Inflammatory Bowel Disease: A Systematic Review and Meta-analysis. *J Crohns Colitis*. 2017;11(5):631-642.
14. Orchard TR, Wordsworth BP, Jewell DP. Peripheral arthropathies in inflammatory bowel disease: their articular distribution and natural history. *Gut*. 1998;42(3):387-391. [CrossRef]
15. Rogler G, Singh A, Kavanaugh A, Rubin DT. Extraintestinal Manifestations of Inflammatory Bowel Disease: Current Concepts, Treatment, and Implications for Disease Management. *Gastroenterology*. 2021;161(4):1118-1132. [CrossRef]
16. Farhi D, Cosnes J, Zizi N, et al. Significance of erythema nodosum and pyoderma gangrenosum in inflammatory bowel diseases: a cohort study of 2402 patients. *Medicine (Baltimore)*. 2008;87(5):281-293. [CrossRef]
17. Greuter T, Navarini A, Vavricka SR. Skin Manifestations of Inflammatory Bowel Disease. *Clin Rev Allergy Immunol*. 2017;53(3):413-427. [CrossRef]
18. Ribaldone DG, Brigo S, Mangia M, Saracco GM, Astegiano M, Pellicano R. Oral Manifestations of Inflammatory Bowel Disease and the Role of Non-Invasive Surrogate Markers of Disease Activity. *Medicines (Basel)*. 2020;7(6):33. [CrossRef]
19. Thrash B, Patel M, Shah KR, Boland CR, Menter A. Cutaneous manifestations of gastrointestinal disease: part II. *J Am Acad Dermatol*. 2013;68(2):211.e1-33; quiz 244-246. [CrossRef]
20. Vavricka SR, Manser CN, Hediger S, et al. Periodontitis and gingivitis in inflammatory bowel disease: a case-control study. *Inflamm Bowel Dis*. 2013;19(13):2768-2777. [CrossRef]
21. Taleban S, Li D, Targan SR, et al. Ocular Manifestations in Inflammatory Bowel Disease Are Associated with Other Extra-intestinal Manifestations, Gender, and Genes Implicated in Other Immune-related Traits. *J Crohns Colitis*. 2016;10(1):43-49. [CrossRef]
22. Troncoso LL, Biancardi AL, de Moraes HV Jr, Zaltman C. Ophthalmic manifestations in patients with inflammatory bowel disease: A review. *World J Gastroenterol*. 2017;23(32):5836-5848. [CrossRef]
23. Rossi RE, Conte D, Massironi S. Primary sclerosing cholangitis associated with inflammatory bowel disease: an update. *Eur J Gastroenterol Hepatol*. 2016;28(2):123-131. [CrossRef]
24. Kaplan GG, Laupland KB, Butzner D, Urbanski SJ, Lee SS. The burden of large and small duct primary sclerosing cholangitis in adults and children: a population-based analysis. *Am J Gastroenterol*. 2007;102(5):1042-1049. [CrossRef]
25. Fraga M, Fournier N, Safroneeva E, et al.; Swiss IBD Cohort Study Group. Primary sclerosing cholangitis in the Swiss Inflammatory Bowel Disease Cohort Study: prevalence, risk factors, and long-term follow-up. *Eur J Gastroenterol Hepatol*. 2017;29(1):91-97. [CrossRef]
26. Dean G, Hanauer S, Levitsky J. The Role of the Intestine in the Pathogenesis of Primary Sclerosing Cholangitis: Evidence and Therapeutic Implications. *Hepatology*. 2020;72(3):1127-1138. [CrossRef]

27. Bergquist A, Ekbom A, Olsson R, et al. Hepatic and extrahepatic malignancies in primary sclerosing cholangitis. *J Hepatol*. 2002;36(3):321-327. [CrossRef]
28. Pitchumoni CS, Rubin A, Das K. Pancreatitis in inflammatory bowel diseases. *J Clin Gastroenterol*. 2010;44(4):246-253. [CrossRef]
29. Suk Lee Y, Kim NH, Hyuk Son J, et al. Type 2 Autoimmune Pancreatitis with Crohn's Disease. *Intern Med*. 2018;57(20):2957-2962. [CrossRef]
30. Eliadou E, Moleiro J, Ribaldone DG, et al.; ECCO CONFER COMMITTEE. Interstitial and Granulomatous Lung Disease in Inflammatory Bowel Disease Patients. *J Crohns Colitis*. 2020;14(4):480-489. [CrossRef]