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Endoscopy for the Diagnosis of Inflammatory Bowel Disease

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Abstract

The diagnosis of inflammatory bowel disease (IBD) and the differentiation between Crohn's disease and ulcerative colitis can be challenging. Colonoscopy with ileoscopy is the useful diagnostic test for patients with suspected inflammatory bowel disease. Esophagogastroduodenoscopy, enteroscopy, and capsule endoscopy all have complementary roles to ileocolonoscopy. Endoscopy not only allows for the visualization of inflammation due to IBD but also for histological analysis, both of which can aid the in proper diagnosis and to exclude other causes of enteritis and colitis. This chapter will describe the use of endoscopy for the diagnosis of IBD.

Keywords: inflammatory bowel disease, Crohn's disease, ulcerative colitis, colonoscopy, diagnosis

1. Introduction

Colonoscopy is the most essential diagnostic tool for patients with suspected inflammatory bowel disease (IBD). Ileoscopy at the time of colonoscopy is critical to both diagnose IBD, differentiate between ulcerative colitis (UC) and Crohn's disease (CD), and to determine the extent and distribution of inflammation as this will affect prognosis and treatment. Other endoscopic modalities including esophagogastroduodenoscopy (EGD), capsule endoscopy, and enteroscopy all have a role in the diagnosis of IBD in select situations. It is critical to understand the endoscopic features and perform the endoscopy appropriately to improve the diagnostic yield and differentiate between IBD and other causes that might mimic IBD as well as differentiating CD and UC as the medical and surgical treatments can be different. This chapter will focus on the practical approach of using endoscopy to diagnose IBD.

2. Ileocolonoscopy

Colonoscopy with ileoscopy is the gold standard for the diagnosis of IBD. It allows for both direct visualization of the most commonly affected areas of bowel in patients with IBD and tissue sampling for histologic analysis. While the inflammation seen in UC is mainly limited to the colon, CD may present with inflammation anywhere from the mouth to anus. Therefore, any colonoscopy done to evaluate symptoms concerning for IBD, or less commonly if a patient is incidentally found to have colonic inflammation during colonoscopy, intubation of the terminal ileum should be attempted. If ileoscopy is successful, taking biopsies of the ileum and colon are also critical aspects of the diagnostic evaluation as this is more sensitive than visual evaluation of the mucosa to find evidence of inflammation.

2.1. Crohn's disease

Crohn's disease was initially described as regional ileitis in 1932 in which a new entity was described as being similar to UC but affecting the small intestine and leading to luminal stenosis [1]. Since then, the endoscopic features and distribution of CD has been extensively elucidated. CD can affect any part of the alimentary tract from the mouth to the anus, but the terminal ileum and colon are most commonly affected. About 29% of patients with CD have involvement of both the ileum and colon, 35% have isolated ileitis, 36% of patients have colitis, and 4% have upper gastrointestinal tract involvement at the time of diagnosis. This distribution can evolve over time during a patient's disease course and so these proportions may not stay static in a population with CD over time [2].

Findings on index ileocolonoscopy at the time of CD diagnosis vary depending on the severity of inflammation, but the distribution and pattern can be helpful in diagnosing CD. Skip lesions, areas of inflamed mucosa separated by normal appearing mucosa, is characteristic of CD [3, 4]. Rectal sparing occurs in at least 50% of patients. The inflammation is patchy and circumferential inflammation is uncommon [5, 6].

Mild inflammation presents endoscopically with erythema, granularity, altered vascular pattern, friability, and small discrete superficial and aphthous ulcers. As the inflammation progresses, deep, serpiginous, and linear ulcerations and cobblestoning develop (**Figure 1**). About one-third of patients with CD will develop a fistula over their lifetime. In perianal disease fistulas may be apparent on physical exam, and perianal fistulas are more commonly seen in patients with rectal inflammation. Endoscopically, fistula openings may be visible as small openings in the colon or ileal mucosa [7]. Strictures, perianal disease, and isolated ileitis are also indicative of but not 100% specific for CD [6, 8]. Because of the discontinuous inflammation in CD, the area immediately surrounding inflammatory patches or ulcers are more likely to have an intact vascular pattern and absent or minimal inflammation on biopsy [9].

2.2. Ulcerative colitis

The inflammation seen in ulcerative colitis begins at the anal verge and extends proximally. Ulcerative colitis always involves the rectum, but if treatment has been started prior to



Figure 1. Terminal ileitis due to Crohn's disease.

colonoscopy the rectum may be spared or there may be patchy rectal inflammation [10]. The proximal extent of inflammation varies—about 46% of patients with UC have proctosigmoiditis, 37% have left sided colitis, and 17% have pancolitis [11].

On colonoscopy the inflammation in UC is circumferential and continuous. The features vary depending on severity. Early and mild inflammation appears as erythema, edema, and abnormal vascularity. Moderate UC has a “wet sand-paper” appearance due to changes in light reflection, erosions, superficial ulcers, and friability. As the severity of inflammation progresses the ulcerations become confluent, friability worsens, and spontaneous bleeding may develop [12–14] (**Figure 2a** and **2b**). Because the inflammation is continuous, the mucosa surrounding ulcerations will usually at a minimum have a diminished vascular pattern but more commonly will show more obvious signs of inflammation [9].

Pseudopolyps are also often seen in UC and develop as a result of regenerating epithelium but can be seen in CD as well [15]. They develop in patients with more severe and extensive periods of inflammation. While pseudopolyps themselves are not at risk of malignant transformation, UC patients with pseudopolyps may have a higher incidence of colorectal cancer as a result of more severe inflammation that predisposes to pseudopolyp formation [15, 16]. Pseudopolyps do not need to be resected, but there can be difficulty in distinguishing between pseudopolyps and adenomatous tissue, in which case biopsies or resection should be performed [12].

Patients with UC may have two unique areas of inflammation that may be confused as representing CD. In patients without inflammation in the right colon, there may be a “cecal patch”, or localized inflammation around the appendiceal orifice (**Figure 3**). The prevalence of peri-appendiceal inflammation in UC is 5%. The significance of the cecal patch is uncertain, but its presence does not signify a more aggressive disease phenotype or higher colectomy rates [17]. Additionally, 10–25% of patients with pan-colonic UC have “backwash ileitis” which can be confused as representing CD. Backwash ileitis usually presents as localized, continuous, and short segment erythema in the terminal ileum without discrete ulcerations

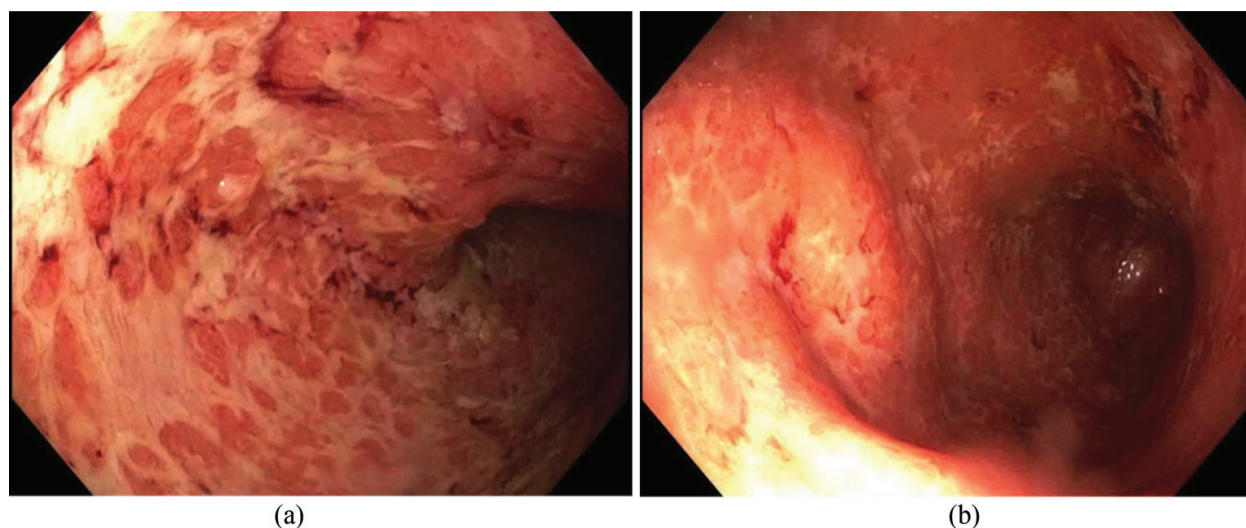


Figure 2. Severe ulcerative colitis.

or strictures and always occurs in the setting of pancolitis [18–20]. In contrast to peri-appendiceal inflammation, backwash ileitis represents a more severe disease course and increased risk for colectomy [21]. It should be noted that these observations are based on observational findings and one should not use these findings as definitive findings for distinguishing CD from UC. These findings further do not necessarily alter the medical management of IBD.

2.3. Biopsy collection

Biopsies should be obtained from both normal and abnormal appearing mucosa. A minimum of two biopsies should be taken from at least five sites throughout the colon including the rectum and terminal ileum. The biopsies should be labeled appropriately and separated so that the site of biopsy can be correlated with histology [13, 22, 23]. A full set of colonoscopic biopsies improves the diagnostic yield by histology for both CD and UC. Full biopsies may also reveal inflammation not seen well endoscopically that can affect prognosis and need for dysplasia surveillance [22]. Granulomas are present in patients with CD in at most 25% of patients at initial presentation and therefore cannot be used to differentiate between CD and UC when absent [24]. However, biopsies taken from micro-ulcers <5 mm in size and ulcer edges are more likely to demonstrate granulomas [25]. Terminal ileal biopsies are also vital in distinguishing UC from CD and for ruling out IBD mimickers.

2.4. Complications and contraindications

The complications seen in patients undergoing diagnostic ileocolonoscopy are similar to the general population. Complications include bleeding, perforation, and respiratory failure due to over sedation. It is not clear if IBD patients have an increased risk of perforation, with some studies finding no increased risk and others showing a higher rate of perforation, particularly in hospitalized IBD patients undergoing colonoscopy [26–29]. Full colonoscopy should be undertaken with caution in patients with severe inflammation, in those unable to undergo full bowel

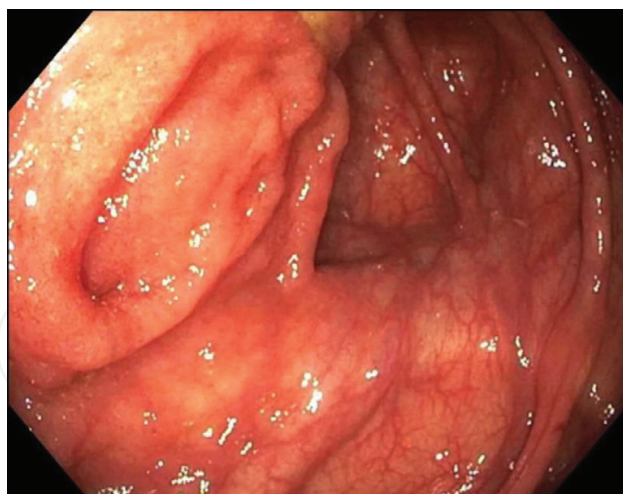


Figure 3. Peri-appendiceal inflammation.

prep because of severe symptoms, and definitely avoided in patients with toxic megacolon. In patients with severe disease and inflammation flexible sigmoidoscopy can be used for diagnosis and ruling out some infections, but sigmoidoscopy may not allow for differentiation between UC and CD. Despite these concerns, the overall rate of perforation is still very low. However, if perforation does occur it can require surgery and cause significant morbidity and even mortality and therefore caution should be taken in the presence of considerable inflammation.

3. Esophagogastroduodenoscopy

Although CD can involve any area from the mouth the anus, upper gastrointestinal (GI) tract involvement is less common than ileal or colonic inflammation. Because upper GI inflammation is often seen in patients without IBD, the prevalence is difficult to accurately determine but has been described in 13–55% of patients with IBD [30]. In terms of the distribution of upper GI tract involvement, one study found upper GI inflammation attributable to CD in the esophagus in 6.5%, upper-middle stomach in 47.8%, lower stomach in 24.6%, duodenal bulb in 31.9%, and second portion of the duodenum in 18.1% [31]. EGD is not necessary for all adult patients with suspected IBD but should be done for those with upper GI symptoms such as nausea, vomiting, and early satiety. Endoscopic evaluation of the upper GI tract can also be useful when the diagnosis is uncertain.

EGD is recommended for pediatric patients with suspected IBD at the time of initial colonoscopy. There is a significantly higher proportion of pediatric patients with indeterminate colitis compared to adults, and EGD can help distinguish between CD and UC. There can also be inflammation with granulomas on biopsy even without colonic or ileal inflammation. Additionally, children can frequently present with non-specific symptoms such as weight loss or anemia for which an EGD is warranted to evaluate for IBD as well as other causes such as celiac disease [13, 32, 33].

3.1. Mucosal appearance and distribution

Esophageal Crohn's disease can appear as scattered erosions and aphthous ulcers with mild-moderate disease. More severe esophageal inflammation due to CD appears as longitudinal ulcers and can even have a cobblestone appearance. Stricturing and fistulization of the esophagus is rare but does occur in 20 and 5%, respectively, of patients with esophageal CD [34]. Importantly, esophageal CD must be differentiated from other causes of esophageal inflammation including reflux disease, eosinophilic esophagitis, and infectious esophagitis as the medical and surgical treatment for each condition varies. Granulomas are detected in less than 25% of cases of esophageal CD and therefore the absence of granulomas cannot be used to exclude esophageal CD [31].

Gastric CD is the most commonly observed site of involvement in the upper GI tract. The endoscopic findings are relatively non-specific for CD and include erythema, aphthous or linear ulcers, and granularity most commonly in the antrum. Bamboo-joint-like appearance in the stomach, typically in the cardia and upper body, is more specific finding for CD. The bamboo-joint-like finding appears as edematous folds with fissures or linear furrows arranged transversely [35]. Notably, gastritis without ulceration is often seen in patients with UC and cannot be used to differentiate CD from UC [19].

Mucosal features of duodenal CD can also be frustratingly non-specific. Findings include erythema, edema, aphthous and longitudinal erosions and ulcerations. Duodenal CD may have protruding lesions in the second portion of the duodenum that arrange longitudinally or a notch-like appearance in the second portion of the duodenum that may be a more reliable marker of inflammation due to CD [31, 35].

3.2. Biopsy collection

A minimum of two biopsies should be taken from the esophagus, stomach, and duodenum for patients undergoing EGD for suspected IBD. Biopsies should also be taken from the stomach to rule out *Helicobacter pylori* infection depending on the patient's symptoms and endoscopic findings. More than two biopsies should be obtained from the esophagus and duodenum if there is concern for other diseases such as celiac disease or eosinophilic esophagitis to improve the diagnostic yield of the procedure and directed biopsies should be taken of any visible lesions.

4. Endoscopic evaluation of the small intestine

Evaluation of the small bowel in patients with suspected CD can be useful when the diagnosis is uncertain after ileocolonoscopy or upper endoscopy. Enteroscopy is also valuable for therapeutic benefit in the setting of small bowel strictures at the time of diagnosis and is typically guided by radiographic imaging findings. There are multiple modalities for small bowel evaluation—capsule endoscopy (CE), push enteroscopy, and antegrade (via mouth)

or retrograde (via anus) device assisted enteroscopy. The benefit of push or device assisted enteroscopy is the ability to sample tissue for histology and for therapy in the case of stricturing CD. For all afore mentioned modalities, they should be undertaken if the findings would change medical or surgical management of the patient and are not required prior to starting medical therapy.

4.1. Capsule endoscopy

Capsule endoscopy is important when the diagnosis of IBD is uncertain after EGD and colonoscopy with ileoscopy or in cases of indeterminate colitis. Capsule endoscopy is less invasive compared to standard endoscopy and allows for imaging of the entire small bowel that may not be easily reached even by device assisted enteroscopy. Additionally, CE has a similar or higher sensitivity compared to other small bowel imaging modalities such as small bowel follow through, magnetic resonance enterography (MRE), or computed tomography enterography (CTE). The main limitation of CE is the inability to obtain biopsies for histologic analysis, which can lead to diagnostic challenges as small bowel findings on CE may not be specific to IBD. An advantage of CE over small bowel imaging modalities is the ability to detect subtle inflammation that may not be seen on CTE or MRE [13, 36]. Another disadvantage of CE is that it can become retained in up to 5% of CD patients and may require enteroscopy or surgery for retrieval.

Small bowel inflammation due to IBD has a similar appearance to IBD elsewhere in the GI tract. This includes more subtle features such as erythema, granularity, loss of villi, and edema, to more prominent findings such as ulceration of varying sizes, strictures, and fistula openings [37, 38].

The main complication of capsule endoscopy is capsule retention. Because of the stricturing nature of CD, there is estimated to be a slightly higher risk of capsule retention compared to patients without CD. In patients with known or suspected strictures or with obstructive symptoms, assessment with patency capsule or alternative small bowel imaging modality (CTE or MRE) beforehand is imperative [39]. The risk of capsule retention in patients undergoing evaluation for suspected CD is lower than in patients with established CD but still occurs in about 1–2% of patients [40]. In cases of retention for longer than 2 weeks, the capsule should be retrieved. Occasionally, if a capsule is retained due to a small bowel stricture that is due at least in part to active inflammation, the capsule will eventually traverse a stricture if effective medical therapy is initiated. If unsuccessful, retrieval can be accomplished by balloon or push enteroscopy, but in some cases surgical intervention is required.

4.2. Enteroscopy

The advantage of enteroscopy over CE is the ability to obtain tissue when the etiology of small bowel inflammation is uncertain. Additionally, enteroscopy can allow for dilation of small bowel strictures that may not be reached by standard colonoscopy with ileoscopy or EGD. Push enteroscopy is a technique in which a colonoscope, typically pediatric, is advanced

to the proximal jejunum. Double and single balloon enteroscopy is more technically challenging than push enteroscopy but can be advanced past the reach of push enteroscopy. Single or double balloon enteroscopy can be done antegrade (via the mouth) or retrograde (via the anus) depending on the site of suspected disease. Double balloon enteroscopy can be effective for the diagnosis and staging of suspected small bowel CD in 30–48% of cases but is not the preferred initial test [41, 42]. Findings on enteroscopy are the same as CE, namely erythema, edema, loss of villi, ulcerations, and possibly strictures and fistula openings. The major complication rate of balloon enteroscopy is 0.72% and includes perforation, pancreatitis, aspiration, and bleeding [43]. Complication rates of push enteroscopy are similar to balloon enteroscopy [44]. It should also be noted that enteroscopy whether antegrade or retrograde may not visualize the entirety of the small intestine and typically requires general anesthesia to complete.

5. Indeterminate colitis and differentiating UC and CD

The most important aspect of ileocolonoscopy for suspected IBD is making the correct diagnosis and staging the disease as this will affect prognosis and treatment. Ileocolonoscopy can differentiate UC from CD nearly 90% of the time. Indeterminate colitis is used for a small subset of patients with colitis cannot be easily classified into UC or CD by endoscopic findings or histology [45].

The pattern and distribution of inflammation is critical for distinguishing CD and UC. UC presents with continuous inflammation and in untreated UC always involves the rectum. In CD, rectal sparing is often present and the inflammation is patchy with intervening areas of normal mucosa. However, the presence of rectal inflammation can be seen in up to 50% of patients with CD is therefore not diagnostic of UC [8]. Additionally, because of the continuous nature of the inflammation in UC, the mucosa immediately surrounding ulceration will be abnormal. This is apparent as erythema or decreased vascular pattern around ulcers in UC. In CD, the mucosa around ulcers shows a normal vascular pattern and absence of inflammation [3, 4, 19]. Central to discriminating CD from UC is ileoscopy. While backwash ileitis can be present in up to 25% of UC patients with pancolitis, the inflammation in this setting is usually mild, continuous, and shorter. It should be noted that the definition of backwash ileitis is controversial and the term was initially created prior to the era of ileo-colonoscopy and was used to describe a finding on barium enema. The presence of ulcers in the terminal ileum in a patient without right colon inflammation is specific for CD compared to UC. However, it is important to remember that there are other causes of terminal ileitis, including infection, vasculitis, malignancy, or NSAID induced inflammation [46]. Inflammation, particularly ulceration, stricturing, or fistulization of the upper GI tract or small bowel, is virtually diagnostic of CD over UC, although mild gastritis or duodenitis without ulceration can be present in patients with UC. Granulomas, if present, are also consistent with Crohn's disease, and biopsies of the ulcer edge increase the chance of finding a granuloma [25].

When a diagnosis of CD or UC cannot be made based on endoscopy, histology, and radiography, the term indeterminate colitis or IBD-unclassified is used. About 7–10% of adult patients with IBD will have indeterminate colitis. An even higher proportion of children, nearly 30%, have indeterminate colitis [32, 45]. Some of these patients will be reclassified as CD or UC as the disease evolves and defining characteristics of UC or CD develop. EGD and CE may be helpful in establishing the correct diagnosis by revealing small bowel inflammation consistent with CD in about 15% of patients with indeterminate colitis. However, a normal EGD or CE study does not rule out CD [47]. If a patient is classified as indeterminate colitis, this should not affect therapy choices or present or future endoscopic evaluation.

6. Differentiating IBD from IBD mimickers based on endoscopy

The diagnosis of IBD relies on a combination of symptoms, laboratory analysis, imaging, endoscopy, and histology. However, the endoscopic inflammation in IBD can be non-specific and due to causes other than IBD. In addition to differentiating between CD and UC and staging the extent of disease, other causes of bowel inflammation should be ruled out. This is particularly important as the treatment for IBD may lead to worsening of other conditions, particularly infection.

6.1. Infection

Infection is an important mimicker of IBD on endoscopy. Common infections such as *Clostridium difficile* and *Escherichia coli* should be ruled out with stool testing prior to colonoscopy. *Yersinia* spp. can often lead to right lower quadrant abdominal pain and fever with imaging showing ileitis and an appearance suggestive of acute appendicitis. *Salmonella*, *Actinomyces*, and *E. coli* infections can also lead to enteritis and particularly ileitis that may look like IBD [48]. Intestinal tuberculosis can lead to ulceration, nodularity, and stricturing of the terminal ileum and ileocecal valve [49].

Cytomegalovirus (CMV) infection can lead to inflammation and ulceration in any part of the gastrointestinal tract. The ulcers in CMV enteritis or colitis have been described as having a “punched-out” appearance. Biopsies can help differentiate CMV from IBD. However, many patients with IBD will have coexisting CMV and endoscopy is important to rule out concomitant CMV infection that is contributing to bowel inflammation. However, it can be sometimes challenging to determine whether CMV is an innocent bystander or an active participant in inflammation in IBD patients [50].

6.2. Vasculitis

Rarely, vasculitis can affect the bowel, typically the small intestine. Systemic lupus erythematosus, polyarteritis nodosa, Henoch-Schönlein purpura, and Behçet’s disease may all be confused with IBD. Polyarteritis nodosa frequently affects the gastrointestinal tract in up to 65%

of patients and may lead to symptoms of bowel ischemia [51]. Behçet's disease in particular can lead to discrete ulcers in the small and large bowel with normal intervening mucosa that can be confused for CD. However, the ulcers in Behçet's disease are usually fewer in number, larger, deeper, and rounder than seen in IBD [52].

6.3. Ischemia

Ischemia can lead to edema, erythema, erosions and ulcerations that can look similar to IBD. Severe ischemic colitis can lead to a dusky and even black appearance with necrosis. The inflammation is usually segmental with a sharp demarcation affected and unaffected mucosa depending on the vascular supply. The left colon is most commonly affected. An accurate history and acuity of symptoms can also help distinguish IBD from ischemic colitis [53, 54].

6.4. Segmental colitis associated diverticulosis syndrome

Segmental colitis associated diverticulosis (SCAD) can be especially difficult to distinguish from IBD. SCAD is associated with diverticulosis and most commonly affects the sigmoid colon. The rectum and right colon are typically spared. The endoscopic features of SCAD include edema, erythema, erosions, and ulcers, often with sparing of the diverticular orifices [55]. Because endoscopic and histologic features overlap with IBD, the diagnosis can be challenging but SCAD is more often found in older patients and often responds to mesalamine [56].

6.5. NSAID enteropathy

NSAIDs are the most common medication that can lead to bowel inflammation. NSAIDs can lead to "diaphragm disease" or pinhole openings due to 2–3 mm thin walled septae with normal mucosa between diaphragms. NSAIDs can also lead to erosions and ulcers not just in the stomach and duodenum but small bowel as well [48].

7. Novel techniques and future directions

This section will discuss techniques that are available or being developed but not widely utilized or have not been evaluated sufficiently to recommend that these techniques be used as standard of care.

7.1. Endoscopic ultrasound

Although still being studied, endoscopic ultrasound (EUS) is emerging as technique that can be valuable for the diagnosis of IBD and differentiation between CD and UC. In one study comparing EUS in IBD patients to healthy controls, patients with active IBD undergoing EUS had increased total wall thickness of the sigmoid colon compared to healthy controls.

Furthermore, patients with UC had increased wall thickness of the mucosa with normal submucosa and muscularis propria, whereas CD patients had increased submucosa thickness with normal mucosa thickness [57]. In addition to being used to assess bowel inflammation, EUS has a recognized role in the diagnosis and evaluation of CD related perianal disease. EUS can determine fistula anatomy with accuracy that is slightly higher than MRI (91% vs. 87%). EUS can also assess for adjacent abscesses and the degree of active inflammation which can in turn guide management [58, 59].

7.2. Endocytoscopy and endomicroscopy

Endopathology, which includes both endocytoscopy (EC) and confocal laser endomicroscopy (CLE), allows for magnification of the mucosal surface and real-time histologic assessment at the time of endoscopy. EC and CLE can be performed with stand-alone probes that are advanced through an endoscope or via probes integrated into the distal end of an endoscope. Endocytoscopy typically requires *N*-acetylcysteine for mucolysis followed by topical application of a staining agent. CLE allows for tissue magnification by illumination with a low power laser light that is reflected through a pinhole and requires either a topical agent or an intravenous fluorescence agent, usually fluorescein sodium, for adequate visualization [60, 61]. Magnification assessment by EC allows for the detection of mucosal inflammatory cells, crypt assessment, and nucleus-cytoplasm ratio, whereas CLE can assess crypt architecture, inflammatory cell infiltrate, and vessel architecture but fluorescein does not allow for nuclear visualization and assessment. Both EC and CLE have excellent correlation with histology in IBD and can diagnose inflammatory and architectural changes even if the mucosa appears normal endoscopically [62, 63]. Both EC and CLE may allow for identification of microscopic changes that can predict relapse in established IBD patients in remission, but their role in diagnosis at this time is unclear. EC and CLE are areas undergoing active investigation and do not yet have widespread applicability.

7.3. Spectroscopy

Elastic scattering spectroscopy, reflectance spectroscopy, and fluorescence spectroscopy have shown promise for the diagnosis of IBD. In addition to aiding in the diagnosis of IBD, Raman spectroscopy has evidence that shows promise for the differentiation of CD and UC. Spectroscopy in general provides a unique tissue signature that is based on the makeup of the tissue and its interaction with light and is different in normal compared to inflamed tissue. Scattering spectroscopy provides information based on the microscopic structure, whereas Raman spectroscopy and fluorescence spectroscopy provide data based on the biochemical makeup of the tissue [64–66]. Spectroscopy in general shows promise for the diagnosis of IBD but needs further evaluation.

7.4. Optical coherence tomography

Optical coherence tomography (OCT) generates a cross-sectional image of the internal microstructure by measuring back-reflected light. OCT can evaluate tissue to a depth of at the least

the muscularis propria in most patients and provides information on transmural inflammation by identifying disruption of the layered structure of the bowel wall. Such disruption on OCT can therefore help differentiate CD from UC [67, 68]. OCT also requires further study before clinical application.

8. Conclusion

The most important test for the diagnosis of IBD is colonoscopy with ileoscopy being a critical component in initial testing. Capsule endoscopy can be a useful tool when the diagnosis is uncertain and certainly in patients with disease on radiographic studies out of reach of standard ileo-colonoscopy. In addition CE has a similar or higher sensitivity compared to small bowel imaging modalities. In terms of mucosal appearance, continuous inflammation from the anal verge proximally is consistent with UC whereas discontinuous inflammation with ileitis, upper GI or other small inflammation and the presence of stricturing or fistulizing disease is diagnostic for CD over UC. However, the mucosal appearance of the inflammation is not 100% specific for either disease. Appropriate attention should be made to obtaining biopsies to increase the diagnostic yield of the procedure. At least two biopsies should be taken from five sites during ileocolonoscopy including the ileum and rectum and normal and abnormal appearing mucosa. The diagnosis of IBD relies upon a combination of history, radiography, laboratory, and endoscopic features, with ileocolonoscopy providing the most accurate and useful data.

Conflict of interest

Jeffrey Jacobs—none.

Scott Lee—none.

Notes/Thanks/Other declarations

None.

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