inhibitors. Glucagon-like peptide 2 and its analog, teduglutide, may increase intestinal adaptation.

Carbohydrate Malabsorption

Carbohydrates can be classified as monosaccharides (glucose, fructose), disaccharides (lactose, sucrose), oligosaccharides (maltodextrose), or polyols (sorbitol, mannitol). These short-chain carbohydrates are osmotically active and can lead to increased luminal water retention and gas production through colonic fermentation. These actions can cause gastrointestinal symptoms, including gas, bloating, and diarrhea.

Lactose malabsorption is commonly due to loss of the brush-border lactase enzyme in adolescence or adulthood. Fructose malabsorption can also cause gastrointestinal symptoms, such as bloating and diarrhea. Although both lactose and fructose breath tests are available, testing is often not required because symptoms subside with elimination of the sugar from the diet and recur with ingestion of the sugar.

KEY POINTS

- Small intestinal bacterial overgrowth is caused by impaired motility, strictures, or blind loops and is treated with antibiotics.
- Short bowel syndrome is defined by loss of functional small bowel, with resultant loss of absorptive area leading to maldigestion, malabsorption, and malnutrition.
- Lactose malabsorption is common; symptoms occur when lactose is ingested and subside with elimination of lactose from the diet.

Inflammatory Bowel Disease

IBD is an idiopathic chronic inflammatory condition of the gut that includes ulcerative colitis and Crohn disease. Microscopic colitis is considered a type of IBD with distinct clinical and pathologic features. The pathogenesis of IBD likely involves host genetic predisposition and abnormal immunologic responses to endogenous gut bacteria.

Risk Factors

The primary risk factor for IBD is family history, with a risk of approximately 10% for first-degree relatives of affected patients. Individuals of Ashkenazi Jewish descent have increased risk for IBD. Tobacco smoking increases the risk for Crohn disease and is protective for ulcerative colitis.

IBD has a bimodal age presentation: an initial peak incidence in the second to fourth decades of life followed by a less prominent second peak in the seventh and eighth decades.

Clinical Manifestations

Ulcerative Colitis

The major symptoms of ulcerative colitis include diarrhea, abdominal discomfort, rectal bleeding, and tenesmus; symptoms vary by disease extent and severity. Symptoms typically have an insidious onset and often have been present for weeks or months by the time the patient seeks care, although ulcerative colitis may present acutely, mimicking infectious or colonic ischemia.

Rectal inflammation (proctitis) causes frequent defecatory urges and passage of small liquid stools containing mucus and blood. Although bloody diarrhea is considered the hallmark presentation of ulcerative colitis, diarrhea is not always present. Patients with proctitis or proctosigmoiditis may have constipation. Abdominal pain is usually not a prominent symptom of ulcerative colitis; however, most patients with active disease experience vague lower-abdominal discomfort relieved with defecation. Physical examination in patients with mild or moderate ulcerative colitis is usually normal but may reveal mild lower-abdominal discomfort over the affected colonic segment. Fever, nausea, vomiting, or severe abdominal pain indicates a severe attack or complication, such as superimposed infection or toxic megacolon.

Crohn Disease

The clinical presentation of Crohn disease may be subtle and varies by location and severity of inflammation along the gut axis as well as the presence of intestinal complications (e.g., abscess, stricture, or fistula). Abdominal pain is a more common symptom of Crohn disease than of ulcerative colitis. The ileocecal area is the bowel segment most commonly affected by Crohn disease; disease in this area often presents insidiously with mild diarrhea and abdominal cramping. Abdominal examination may reveal fullness or a tender mass in the right hypogastrium. Occasionally, the main presenting symptom is acute right-lower-quadrant pain, mimicking appendicitis. Tenesmus is less common in Crohn colitis than in ulcerative colitis because the rectum is often less inflamed than other colonic segments. Perianal disease is a common presentation of Crohn disease, with anal fissures, ulcers, and stenosis.

Fistulae, a frequent manifestation of the transmural nature of Crohn disease, consist of abnormal connections between two epithelial surfaces (perianal, enteroenteric, enterocutaneous, rectovaginal, enterovesical). Drainage of fecal material from fistulae may lead to passage of feces through the vagina, urethra, or skin. Intra-abdominal abscesses may form; the classic presentation is spiking fevers and focal abdominal tenderness, which may be masked by glucocorticoid use. Strictures may result from fibrosis or severe inflammatory luminal narrowing and may occur in any segment of the gastrointestinal tract, although the terminal ileum is the most common site. Patients with intestinal strictures often initially present with colicky postprandial abdominal pain and bloating that may progress to complete intestinal obstruction.

Table 19 summarizes the features of ulcerative colitis and Crohn disease.

Extraintestinal Manifestations

Inflammatory conditions involving extraintestinal structures may occur with IBD. These manifestations are categorized as

TABLE 19. Features of Ulcerative Colitis and Crohn Disease

Feature	Ulcerative Colitis	Crohn Disease
Depth of inflammation	Mucosa	Transmural
Pattern of disease	Contiguous and symmetric	Skips areas and asymmetric
Location	Colorectum	Mouth to anus
Rectal involvement	Nearly 100%	Less common
Ileal disease	Backwash ileitis (15%)	Common
Fistulas, abscess, and strictures	Rare	Common
Perianal disease	Rare	Common
Granulomas	Unlikely	In approximately 30%
Overt rectal bleeding	Common	Less common
Tobacco use	Protective	Exacerbates

associated with active bowel disease or independent of bowel inflammation. An extraintestinal manifestation occurs in up to 30% of patients with IBD at some time during the disease course. Peripheral arthritis is the most common manifestation (see MKSAP 19 Rheumatology).

The two most common dermatologic manifestations are erythema nodosum and pyoderma gangrenosum, both of which usually correspond to underlying IBD activity. Erythema nodosum most commonly presents as single or multiple tender nodules on extensor surfaces of the lower extremities (**Figure 18**). It usually resolves spontaneously over 4 to 6 weeks. Pyoderma gangrenosum typically presents with an exquisitely tender papule, pustule, or nodule that rapidly develops into an exudative ulcer with undermined and violaceous borders (**Figure 19**). The most frequent location is the lower leg; however, pyoderma gangrenosum is also frequently associated with a stoma site (peristomal) after ostomy placement. It is diagnosed after exclusion of other causes of ulceration. Management of pyoderma gangrenosum includes local wound care, immunosuppressants, and avoiding trauma and surgical debridement because of pathergy (i.e., it occurs at sites of trauma).

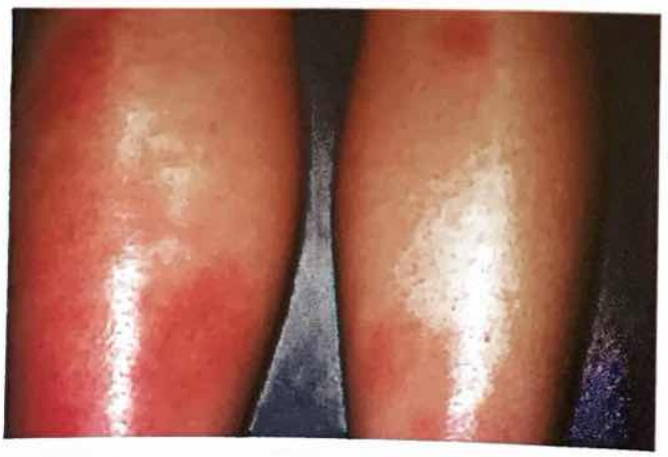

FIGURE 18. Erythema nodosum, a manifestation of inflammatory bowel disease, typically appears as ill-defined erythema overlying subcutaneous, tender nodules most commonly symmetrically located on the anterior shins.

Ocular IBD manifestations include episcleritis and uveitis. Episcleritis is more common and consists of injection of the sclera and conjunctiva. It does not affect visual acuity and is associated with active bowel disease. Uveitis presents with headache, blurred vision, and photophobia. This ocular emergency threatens vision and requires immediate referral to an ophthalmologist. See MKSAP 19 General Internal Medicine 2 for discussion of episcleritis and uveitis. Primary sclerosing cholangitis is the major liver manifestation of IBD, occurring in 5% of patients. Patients most often present with isolated elevations of serum alkaline phosphatase. The liver disease is typically progressive and independent of IBD outcome. See Disorders of the Liver for discussion of primary sclerosing cholangitis.

Diagnosis

Diagnosis of IBD relies on integration of clinical presentation, endoscopic appearance, histologic assessment of mucosal biopsy specimens, radiologic features, and exclusion of

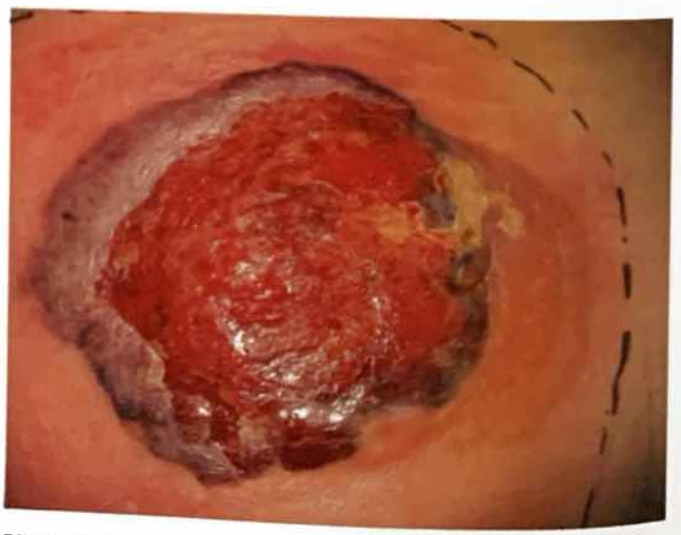

FIGURE 19. Pyoderma gangrenosum, a manifestation of inflammatory bowel disease, typically begins as a small pustule or red nodule that rapidly expands into a painful, exudative wet ulcer with an edematous, infiltrated, actively inflamed border. The border is characteristically violaceous with an edge that overhangs the ulcer.

infectious enteropathogens. IBD should be considered in any patient with chronic or bloody diarrhea. It is paramount to exclude infection, particularly with *C. difficile* and Shiga toxin-producing *E. coli*, by stool tests, especially in patients with acute symptom onset. An elevated fecal calprotectin level may help differentiate IBD from IBS. Antibody tests (e.g., anti-saccharomyces cerevisiae antibodies and perinuclear antineutrophil cytoplasmic antibodies) are available, but their role in diagnosis is undefined; they should not replace endoscopy or biopsy. Laboratory testing helps assess disease activity. Common findings include anemia, thrombocytosis, leukocytosis, and hypoalbuminemia. Iron deficiency anemia often develops from chronic blood loss. Persistently abnormal serum alkaline phosphatase levels should prompt investigation for primary sclerosing cholangitis.

Endoscopy (sigmoidoscopy or colonoscopy) with biopsy is needed to diagnose IBD. Colonoscopy is most commonly used to assess disease extent and severity. At presentation, 50% of patients with ulcerative colitis have disease limited to the rectum and sigmoid (proctosigmoiditis), 20% have left-sided disease (to the splenic flexure), and 30% present with pancolitis (to the cecum). Endoscopic findings range from decreased vascular pattern with erythema and edema in mild disease to large and deep ulcerations in severe disease. Histopathology shows features of chronic colitis, with distorted and branching colonic crypts along with crypt abscesses.

Distribution pattern differs between Crohn disease and ulcerative colitis: 50% of patients have ileocolonic disease; 30%, isolated small bowel disease; and 20%, colonic disease. A minority of patients have isolated upper gastrointestinal tract or perianal disease without small-bowel or colonic inflammation. The earliest endoscopic findings of Crohn disease include aphthous ulcers, which can coalesce to form stellate ulcers, and a "cobblestone" mucosal appearance. A characteristic mucosal feature of Crohn disease is the "skip lesion," consisting of affected areas separated by normal mucosa. Granulomatous inflammation is characteristic of Crohn disease but is uncommonly found on mucosal biopsy specimens. Histopathology in small intestinal Crohn disease shows chronic jejunitis or ileitis, and Crohn colitis has histology similar to that of ulcerative colitis, except for granulomas.

Imaging establishes the location, extent, and severity of IBD. Patients with a severe attack of IBD require plain abdominal radiography to assess for a dilated colon (indicating evolving toxic megacolon) or small-bowel obstruction (**Figure 20**). CT or magnetic resonance enterography provides information about location and severity of small-bowel disease and presence of complicating fistula, abscess, or stricture. Video capsule endoscopy is highly sensitive for detecting small inflammatory lesions of the intestine but is not commonly required.

Treatment

Goals of IBD therapy are to induce and maintain remission and prevent disease- and treatment-related complications. Remission is defined by endoscopic healing of bowel mucosa rather than symptom resolution alone. Four categories of drugs are used: 5-aminosalicylates, glucocorticoids, immunomodulators, and biologics. Stratification based on clinical severity is important in guiding IBD management. There are no validated or consensus definitions of mild, moderate, or severe IBD. For this synopsis, mild ulcerative colitis is defined as fewer than four bowel movements per day, mild to moderate rectal bleeding, no constitutional symptoms, and low overall inflammatory burden. Severe ulcerative colitis is marked by more than six bloody stools per day plus signs of systemic toxicity (e.g., fever, tachycardia, and leukocytosis). Mild Crohn disease is characterized in patients who are ambulatory and are eating and drinking normally, with weight loss less than 10% and no disease-related complications, although patients may have diarrhea and abdominal pain. Patients with severe Crohn disease may be cachectic, with significant weight loss, and may have disease-related complications. These patients are often hospitalized. Patients with moderate disease lie between the extremes.

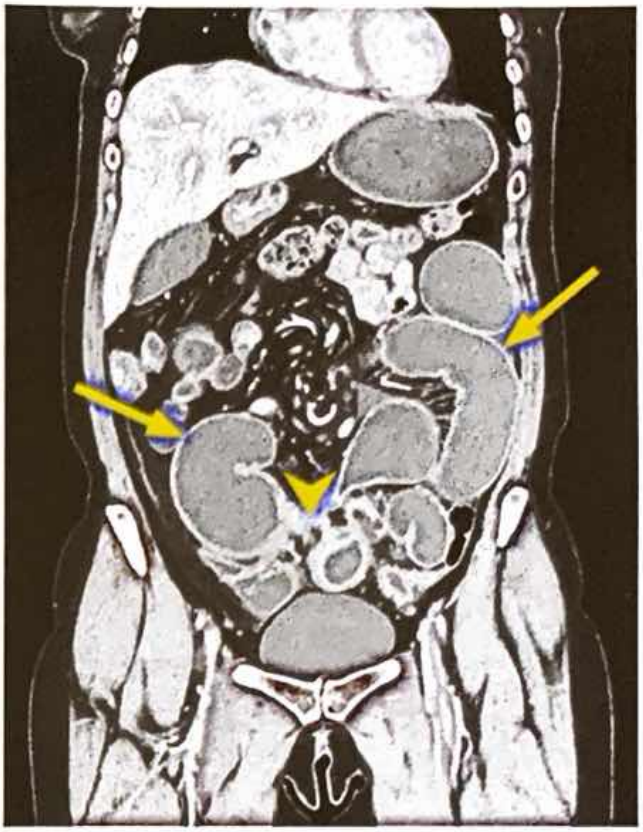

FIGURE 20. CT scan of the abdomen and pelvis in a patient with Crohn disease, showing small-bowel obstruction with dilated loops of small intestine (*arrows*) and matted loops of bowel (*arrowhead*) in the pelvis.

Patients with IBD have markedly increased risk for venous thromboembolism. Hospitalized patients with IBD should receive venous thromboembolism prophylaxis with subcutaneous heparin unless massive gastrointestinal bleeding is present. In such cases, mechanical prophylaxis should be used.

TABLE 20. Medical Treatment of Inflammatory Bowel Disease

Disease Activity	Ulcerative Colitis	Crohn Disease
Mild	Oral and topical 5-ASAs[a]	Sulfasalazine for colitis[a]
	Steroid suppository and enema[b]	Budesonide for ileocolonic disease[b]
	Multimatrix budesonide	
Moderate	Oral and topical 5-ASAs[a]	Oral and intravenous glucocorticoids[b]
	Azathioprine/6-mercaptopurine[c]	Budesonide for ileocolonic disease[b]
	Multimatrix budesonide and oral glucocorticoids[b]	Azathioprine/6-mercaptopurine[c]
	Biologic agents (infliximab, adalimumab, golimumab, vedolizumab, ustekinumab)[a]	Methotrexate[c]
	Tofacitinib[a]	Biologic agents (infliximab, adalimumab, certolizumab, vedolizumab, natalizumab, ustekinumab)[a]
Severe	Oral or intravenous glucocorticoids[b]	Oral or intravenous glucocorticoids[b]
	Cyclosporine[b]	Biologic agents (infliximab, adalimumab, certolizumab, vedolizumab, natalizumab, ustekinumab)[a]
	Biologic agents (infliximab, adalimumab, golimumab, vedolizumab, ustekinumab)[a]	
	Tofacitinib[a]	

5-ASA = 5-aminosalicylate.

[a]Remission and maintenance.

[b]Remission.

[c]Maintenance, glucocorticoid-sparing.

Table 20 summarizes medical treatment of ulcerative colitis and Crohn disease.

Pharmacotherapy for Ulcerative Colitis

5-Aminosalicylates

5-Aminosalicylates (5-ASAs) are believed to have an anti-inflammatory mechanism of action. Unconjugated 5-ASA (mesalamine) is rapidly absorbed in the jejunum, allowing only 20% of the drug to reach the ileum and colon. Oral 5-ASA is modified to reduce upper gastrointestinal absorption and enhance drug delivery to the colon (olsalazine, balsalazide).

The main therapeutic 5-ASAs are sulfasalazine, olsalazine, balsalazide, and delayed- and controlled-release mesalamine. Sulfasalazine has the most adverse effects, including fever, rash, nausea, and headache, and requires concomitant folate supplementation. Sulfasalazine is not considered first-line therapy but is a reasonable choice in patients already taking it who are in remission or have prominent arthritic symptoms.

5-ASAs are the mainstay of treatment of mild to moderate ulcerative colitis, with a dose-dependent response when used to induce remission. Patients with proctitis should receive 5-ASA suppositories. Patients with left-sided disease should receive 5-ASA enemas. In mild to moderate ulcerative colitis, combined 5-ASA therapy (oral and topical) is superior for inducing remission compared with oral or topical therapies alone. 5-ASAs are effective in maintaining remission.

Glucocorticoids

Oral and intravenous glucocorticoids are commonly used to treat moderate to severe flares of ulcerative colitis and can induce remission. However, glucocorticoids are not effective for maintenance therapy and have significant adverse effects. Multimatrix budesonide is a colonic delivery system that allows directed therapy throughout the colon with fewer systemic side effects given its high first-pass hepatic metabolism. It induces remission in mild to moderate ulcerative colitis unresponsive to 5-ASAs and in moderate to severe disease.

Immunomodulators

Thiopurines (azathioprine and mercaptopurine [6-MP]) are immunomodulators used as glucocorticoid-sparing agents in ulcerative colitis. They have a slow onset of action (2-3 months), and patients require a tapering glucocorticoid regimen to bridge the interval until thiopurines take effect. Thiopurines are no more effective than placebo in inducing remission. Thiopurine methyltransferase, a key enzyme in the metabolism of azathioprine and 6-MP, exhibits a population polymorphism. Before initiation of thiopurines, testing for the *TPMT* genotype or phenotype (enzyme activity) is recommended to prevent bone marrow toxicity by identifying individuals with low or absent *TPMT* enzyme activity. However, all patients taking thiopurines require monitoring with complete blood counts and liver chemistry testing because 70% of patients who develop leukopenia while using these agents do not have *TPMT* mutations.

Azathioprine and 6-MP are effective in maintaining remission in ulcerative colitis, should be considered in glucocorticoid-dependent patients, and can be combined with biologic agents. This includes patients who require two courses of glucocorticoids for induction of remission within 1 year and patients who require intravenous glucocorticoids for acute disease flare. The immunomodulator methotrexate is not effective in ulcerative colitis.

Biologic Agents

Tumor necrosis factor (TNF)-α, a proinflammatory cytokine, plays a critical role in the pathogenesis of IBD. The anti-TNF agents infliximab, adalimumab, and golimumab induce and maintain remission in moderate to severe ulcerative colitis, with infliximab being the preferred initial agent. Early use of biologics is appropriate after failure of 5-ASA treatment. Infliximab is administered by intravenous infusion; adalimumab and golimumab are given subcutaneously. Combination therapy with biologics and immunomodulators (thiopurines or methotrexate) is more efficacious than monotherapy with either agent in achieving glucocorticoid-free remission and mucosal healing. Before initiating anti-TNF agents, patients should undergo testing for latent tuberculosis because of an increased risk for tuberculosis reactivation during therapy. Those with latent tuberculosis should receive treatment before or with anti-TNF therapy initiation. Patients should also be assessed for chronic hepatitis B virus infection before starting anti-TNF therapy and receive treatment if needed.

Ustekinumab, a monoclonal antibody that blocks the biologic activity of interleukin-12 and -23 by inhibiting receptors for these cytokines on T cells, is effective in inducing and maintaining remission in patients with moderate to severe ulcerative colitis. Vedolizumab is also effective in inducing and maintaining remission in moderate to severe ulcerative colitis as well as in inducing remission when anti-TNF agents have failed. As with vedolizumab, the small-molecule Janus kinase inhibitor tofacitinib is effective as primary therapy in inducing and maintaining remission in moderate to severe ulcerative colitis and as secondary therapy when anti-TNF agents have failed.

Cyclosporine

Cyclosporine is an immunosuppressive used as an induction agent in select hospitalized patients with severe ulcerative colitis that is unresponsive to intravenous glucocorticoids. It acts as a potential bridge therapy to a slower-onset immunomodulatory or biologic agent.

Pharmacotherapy for Crohn Disease

5-Aminosalicylates

5-ASAs are not efficacious in small-bowel Crohn disease, but sulfasalazine is effective for remission and maintenance therapy for mild to moderate colonic Crohn disease.

Glucocorticoids

Oral and intravenous glucocorticoids are commonly used to treat moderate to severe flares of Crohn disease and are effective in inducing remission. However, as with ulcerative colitis, glucocorticoids are not effective for maintenance therapy. Controlled-release budesonide is effective in inducing remission in mild to moderate ileocolonic Crohn disease.

Immunomodulators

Thiopurines are also used in Crohn disease as steroid-sparing therapy but are not effective in inducing remission. Methotrexate should be considered for alleviating signs and symptoms of steroid-dependent Crohn disease and for maintaining remission. Side effects of methotrexate include hepatotoxicity and interstitial pneumonitis, which can manifest with cough and dyspnea of insidious onset; concomitant folate administration is also necessary.

Biologic Agents

The anti-TNF agents infliximab, adalimumab, and certolizumab treat moderate to severe Crohn disease resistant to glucocorticoids or immunomodulators. Combination therapy with infliximab and azathioprine is more efficacious than monotherapy with either agent alone in achieving glucocorticoid-free remission and mucosal healing. Increasing evidence supports biologic agents early in the disease.

The antiadhesion agents vedolizumab and natalizumab are effective in inducing and maintaining remission of moderate to severe Crohn disease. Natalizumab, however, should be used to maintain natalizumab-induced remission of Crohn disease only if serum antibody to JC virus is negative because of risk for progressive multifocal leukoencephalopathy (a central nervous system demyelinating infection caused by reactivation of the JC virus). Testing for anti-JC virus antibody should be repeated every 6 months and treatment stopped if the result is positive. Ustekinumab, a monoclonal antibody that blocks the biologic activity of interleukin-12 and -23 by inhibiting receptors for these cytokines on T cells, is also efficacious in severe Crohn disease, including when anti-TNF therapies prove ineffective.

Medical Therapy for Fistulizing Disease

Fistulizing Crohn disease is difficult to manage. The most appropriate therapy requires expert evaluation and coordination of care between internal medicine and surgery. As part of multimodality therapy, metronidazole and ciprofloxacin may be effective in simple perianal fistula. Infliximab can be effective for perianal, enterocutaneous, and rectovaginal fistulae, and tacrolimus can be effective for perianal and cutaneous fistulae. The combination of infliximab and antibiotics is more effective than infliximab alone for perianal fistulae.

Surgery

In patients with ulcerative colitis, total proctocolectomy with end-ileostomy or ileal pouch-anal anastomosis is performed for medically refractory disease, toxic megacolon, or carcinoma.

Indications for surgery in Crohn disease include medically refractory fistula, fibrotic stricture with obstructive symptoms, symptoms refractory to medical therapy, and cancer. The guiding principle of surgery in Crohn disease is preservation of bowel length and function because disease frequently recurs after segmental resection. Patients with Crohn disease who undergo surgery require aggressive anti-TNF and/or immunomodulator treatment to decrease the rate of postoperative disease recurrence.

IBD Management in Pregnancy

Most medications used to treat IBD are safe in pregnancy. Methotrexate must be stopped at least 3 months before conception because of its teratogenic effects. Biologic agents and thiopurines are considered low risk during pregnancy and breastfeeding and should be continued to maintain disease remission. Although data on tofacitinib in pregnancy are limited, it should be avoided in pregnancy. See MKSAP 19 Rheumatology for a list of biologic agents and associated risks posed in pregnancy.

Health Care Considerations

Patients with IBD have increased risk for vaccine-preventable illnesses. Inactivated vaccines can be safely administered to all patients with IBD, regardless of immunosuppression. Patients with IBD should receive the annual influenza vaccine as well as the COVID-19 vaccine, the 13-valent pneumococcal conjugate vaccine, and the 23-valent pneumococcal polysaccharide vaccine. Ideally, all immunizatons should occur before immunosuppressive therapy begins. Hepatitis B virus vaccination should be considered, especially if biologic therapy is likely to be required. The safety of administering live vaccines to patients with IBD depends on the level of immunosuppression. Certain live vaccines (e.g., measles or herpes zoster) can be given to patients with low-level immunosuppression. Low-level immunosuppression refers to receipt of less than 20 mg of systemic glucocorticoids per day; methotrexate, 0.4 mg/kg or less per week; and azathioprine (≤3 mg/kg per day) or 6-MP (≤1.5 mg/kg per day). Patients receiving anti-TNFs should not be given live vaccines (e.g., measles, mumps, rubella; varicella; and herpes zoster). See MKSAP 19 General Internal Medicine 2 for discussion of vaccination strategies.

All patients with IBD should be encouraged to stop smoking. Smoking increases Crohn disease activity and the risk for extraintestinal manifestations. Smoking cessation may temporarily worsen ulcerative colitis, but overall health benefits exceed this risk. Patients with IBD are at increased risk for metabolic bone disease due to glucocorticoid use and diminished vitamin D and calcium absorption. Bone mineral density testing is recommended in all patients starting oral glucocorticoid therapy. Otherwise, patients with IBD should be screened for osteoporosis based on established guidelines for the general population. See MKSAP 19 General Internal Medicine 2 for discussion of screening for osteoporosis. Patients with IBD should avoid using NSAIDs when possible because these drugs can exacerbate disease activity. Depression and anxiety are more common in patients with IBD than the general population and are associated with medication nonadherence. Therefore, screening for depression and anxiety is recommended in patients with IBD.

Patients with IBD have increased risk for colorectal, cervical, and skin cancers. Some immunosuppressive treatments also increase the risk for cancer, although this risk is outweighed by the protective effect of mucosal healing and resultant reduced risk for cancers associated with chronic mucosal inflammation. Long-standing colorectal inflammation increases cancer risk. In patients with ulcerative colitis with disease proximal to the sigmoid colon (or beyond the rectum, according to some guidelines) or Crohn disease involving more than one third of the colon, surveillance colonoscopy should be done 8 years after diagnosis and every 1 to 3 years thereafter. Primary sclerosing cholangitis increases the risk for colorectal cancer; surveillance colonoscopy should begin at diagnosis and occur yearly thereafter.

Women with IBD have an increased risk for cervical dysplasia; this risk is greater in women using immunosuppressive therapy. Women with IBD receiving immunosuppressive therapy should undergo Pap testing annually and receive human papillomavirus vaccination according to established guidelines.

Patients with IBD also have an increased risk for melanoma and nonmelanoma skin cancers. Most of the risk is associated with specific treatments, such as anti-TNF agents (melanoma) and immunomodulators (nonmelanoma); however, evidence suggests that the increased risk for melanoma is independent of treatment. Patients with ulcerative colitis and Crohn disease should undergo yearly melanoma screening. In addition, patients receiving immunomodulators (azathioprine and 6-MP) should be screened for nonmelanoma squamous cell cancer while using these agents. All patients with IBD should use sunscreen, wear protective clothing, avoid tanning beds, and undergo dermatologic surveillance.

KEY POINTS

- Combined oral and topical 5-aminosalicylate (ASA) therapy is superior for inducing remission in mild to moderate ulcerative colitis; 5-ASAs are effective in maintaining remission.
- The anti-tumor necrosis factor agents can induce and maintain remission in moderate to severe inflammatory bowel disease.
- Azathioprine and 6-mercaptopurine are used in inflammatory bowel disease as steroid-sparing therapy; methotrexate can be used in Crohn disease.
- Patients with inflammatory bowel disease have increased risk for colorectal, cervical, and skin cancers.

Constipation

Constipation is common, affecting 20% of the general population. Constipation can present with symptoms such as infrequent, difficult, or incomplete defecation. It can be acute or chronic and secondary or idiopathic. Medications are the most common cause of secondary constipation; other causes include mechanical obstruction, systemic illnesses, altered physiologic states, and psychosocial conditions (**Table 21**).

Once secondary causes have been excluded, chronic constipation is considered functional (idiopathic). Functional constipation is subcategorized as slow transit, normal transit,