Inflammatory Bowel Disease is a collective term that includes Ulcerative Colitis and Crohn’s Disease. Our goals in this lesson are to differentiate between these two diseases and also to discuss therapeutic options. This lesson provides 1.25 hours (0.125 CEUs) of credit, and is intended for pharmacists in all practice settings.

The ACPE program ID # for this lesson is 707-000-04-008-H01. Our CE Provider Registered # with CE Broker.com is 50-3170-1.

Pharmacists completing this lesson by August 31, 2007 may receive full credit.

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call toll free 1-800-323-4305. (In Illinois, Alaska and Hawaii phone 1-847-945-8050). Please write your ID Number (the number that is on the top of the mailing label) in the indicated space on the quiz page (for continuous participants only).

The objectives of this lesson are such that upon completion the participant will be able to:

1. List the etiological factors which may contribute to Crohn’s Disease (CD) and Ulcerative Colitis (UC).
2. Describe the pathology of CD and UC.
3. Discuss the clinical and the non-intestinal manifestations of inflammatory bowel disease (IBD).
4. Compare and contrast the symptoms of CD and UC.
5. Discuss the various drugs used in the management of IBD.

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Ulcerative colitis (UC) and Crohn’s disease (CD), collectively known as inflammatory bowel disease (IBD), are chronic, relapsing, inflammatory disorders of the gastrointestinal (GI) tract with overlapping clinical, epidemiologic, pathologic, immunologic, and genetic features, but without a precise course. Even though these disorders share many features, they differ from each other in certain aspects. Inflammatory bowel disease afflicts about one million individuals in the USA and results in morbidity and deterioration of the quality of life and impairment of the ability to work normally. IBD causes a number of GI and extraintestinal symptoms such as abdominal pain, malaise, rectal bleeding, diarrhea, anemia, weight loss, arthralgia, arthritis, and pallor, as well as impaired growth and sexual development in children and young adults. Due to its chronic nature and onset before age 35, treatment may be required throughout the life of the patient. In spite of the fact that remission may be achieved, at the present time there is no cure for IBD.

CROHN’S DISEASE

Crohn’s disease, also known as regional enteritis, is a chronic, relapsing, inflammatory disease of the alimentary canal. The parts most commonly involved are the distal ileum and colon, but anywhere from the mouth to the rectum may be affected. Crohn, Ginzberg and Oppenheimer, who described the disease in 1932, originally localized it to the ileum, the final section of the small intestine. There are approximately 380,000 to 400,000 Americans who are affected by the disease, and the incidence appears to be increasing. In the last twenty years, the number of cases of CD has doubled. Although it may occur at any age, it usually appears prior to the age of 35 and peaks in the 20’s. It appears that the incidence of CD is somewhat more common among women than men, in whites more than blacks or Asians, and in smokers more than in nonsmokers. The disease has a tendency to occur in certain families. Approximately 17% of patients had a positive family history of IBD. The most common familial pattern is that of siblings, a parent and a child.

ETIOLOGY

The etiology of CD is unknown, but the pattern of prevalence implies a complex interplay between factors that involve both the host and environment. These include familial or genetic, infectious, immunologic, psychologenic and dietary causes. The increased incidence among whites and the phenomenon of familial occurrence suggest a genetic predisposition to the development of the disorder. Familial clustering also may indicate exposure of the patient and other family members to similar environmental conditions. The increased prevalence of CD in monozygotic twins is an indication of the role played by genetic factors. However, no definite pattern of inheritance has been established. Environmental factors may influence the prevalence of the disease. The presence of inflammation in the alimentary canal and other clinical and pathologic manifestations has prompted the search for intestinal infectious agents. The increased intestinal bacterial count, the presence of fever and toxins and the improvement of the patient’s condition following the use of antibiotics led to the belief that infectious causes may play a role in triggering CD. In spite of these indications, there is no evidence that bacterial, fungal or viral microorganisms are involved. Immunologic factors have also been implicated. It has been theorized that individuals with CD have developed autoimmunity as a result of hyposensitization to antigens in colon cells. Theories of immunologic involvement are based on the observation that patients with CD may experience disorders such as arthritis, which
may be due to immune mechanisms. In addition, CD responds to treatment with glucocorticoids and immunologic agents such as azathioprine and cyclosporine. It has been hypothesized that CD may occur as a result of an imbalance in the production of pro-inflammatory cytokines over anti-inflammatory cytokines in the intestinal mucosa. However, extensive immunologic studies revealed no clear-cut evidence for establishing immunologic mechanisms in the etiology or pathogenesis of CD. Psychogenic factors have been suggested as a cause for CD. Stress and emotional events may cause a flare-up or exacerbation of the inflammatory process associated with CD, but do not trigger the disease. It appears that persons with CD may have a characteristic personality that makes them vulnerable to emotional stress. However, psychotherapy has been proved useless as a primary therapy, but may be useful as adjunct therapy. It has been concluded that emotional stress may aggravate symptoms of CD rather than initiating the disease. Diet has not been shown to be a causative factor.

**PATHOLOGY**

CD may affect any part of the GI tract. It is characterized by a chronic inflammation that involves all layers of the intestinal wall. The most commonly affected parts are the distal ileum and adjacent right colon. In many cases, several areas of both the small and large intestines are involved in a segmental way. The disease begins as poorly defined pathologic changes that appear granulomatous. The mucosa is hyperemic, nodular with multiple “aphoid” ulcerations. The diseased mucosa is thickened by inflammatory infiltrate. The submucosa and subserosa exhibit fibrosis. The muscularis hypertrophies. Mesenteric lymph nodes are enlarged and inflamed. At this stage of the development, the intestinal wall is edematous, but pliable. Later on, it becomes leathery, and thick with a narrowed lumen. This constriction may result in intestinal obstruction. In advanced cases, mucosal ulceration may deepen and affect the submucosa and muscularis. Fistulas, fissures and abscesses may form. It is not uncommon for certain segments of the bowel to be relatively normal. Thus, CD appears to be discontinuous. The diseased segments are often sharply demarcated from the adjacent normal regions of the bowel (skip areas); thus CD is also known as regional enteritis. Inflamed adjacent loops of the intestine may adhere to each other.

**SYMPTOMS**

In the early stages, patients experience intermittent attacks of crampy pain, diarrhea, especially at night, feeling of fullness in the lower right abdominal quadrant, slight fever, anorexia, and weight loss. Some of these symptoms may mimic those of appendicitis or intestinal obstruction. The pain intensifies after eating, but some relief may be obtained by rest and fasting. The symptoms usually begin in the early twenties, and recur sometimes every few months or even every few years, a cycle that may continue throughout the patient’s life. These episodes may undergo spontaneous remission, but recur with increasing frequency and severity. However, the symptoms may seldom or never occur. The tenderness and pain in the right lower quadrant of the abdomen is due to inflammation and obstruction. The diarrhea is usually mild and rarely more than three to five bowel movements a day. Weight loss occurs as a result of malabsorption and loss of appetite. Development of fistulas, and/or abscesses, are often responsible for fever. Rectal or intestinal bleeding as well as perforation may occur.

**Extraintestinal manifestations** (systemic or nonintestinal symptoms) may precede intestinal manifestation, or they may emerge during the course of the disease. The etiology of these manifestations remains unclear, but diagnosis can be difficult in their presence.

1. **Nutritional complications:** Because the small intestine is the main organ of absorption, many CD patients experience a varying degree of nutritional deficiencies. There may be nutritional complications such as weight loss, reduced muscle mass, electrolyte (potassium, calcium and magnesium) deficiencies, hypoalbuminemia (resulting from malabsorption and protein-loss through the damaged epithelium), anemia due to malabsorption and bleeding, bile salts deficiency leading to steatorrhea (fatty stool as a result of malabsorption), fat-soluble vitamin deficiency, and increased colonic oxalase absorption resulting in increased risk of kidney stone formation.
2. **Growth retardation**: Sexual maturation may be hindered, as well as skeletal growth retardation may take place in children who develop CD prior to puberty. However, studies of thyroid, adrenal and pituitary function revealed that hormonal deficiency is not the cause of growth suppression. It has been postulated that limiting caloric and dietary intake by young patients to avoid cramps and diarrhea may result in malnutrition, which may contribute to growth retardation.

3. **Musculoskeletal complications**: Complications affecting the joints occur in approximately 25% of the patients with CD. Arthritis, ankylosing spondylitis, and peripheral arthralgia may precede the intestinal symptoms. Any joint of the body may be affected, but the knees, ankles and wrists are the most common.

4. **Dermatologic and mucous membrane complications**: Dermatologic signs and symptoms may emerge during the course of IBD. Erythema nodosum, which heals without scarring, pyoderma gangrenosum, a lesion that appears on the trunk and causes scarring, as well as aphoid ulceration of the mouth (canker sores) may appear in 5 to 15 percent of patients.

5. **Hepatic complications**: Abnormal liver disorders such as fatty liver, cirrhosis and pericholangitis (inflammation of tissue surrounding the bile ducts) are common in IBD. Such conditions result in abnormalities in serum levels of aminotransferase and alkaline phosphatase. These complications may intensify as a result of malnourishment.

6. **Renal complications**: Obstruction uropathy, kidney stones, and fistulas in the urinary tract may be encountered.

**DIAGNOSIS**

There is no specific test for confirmation of the disease. CD should be suspected in patients who complain of chronic or nocturnal diarrhea, crampy pain, weight loss, anemia, fever, tenderness and the presence of a mass in the right lower quadrant of the abdomen, and night sweats. In cases where the intestinal symptoms are mild and insidious, attention may be mistakenly focused on diagnosis of nonintestinal complications. Thus identifying CD as the culprit becomes rather difficult. Intermittent intestinal symptoms along with varying intervals of remission may delay diagnosis. However, the symptoms may, over time, increase in frequency and intensity making diagnosis easier. Differential diagnosis that includes acute appendicitis, small bowel obstruction, UC, neoplasia and diverticular disease is essential. Characteristic radiographic findings in the bowel may reveal fistulas, constrictures, and a, “cobblestone” appearance of the mucosa. Common laboratory findings include anemia, mild leukocytosis, and elevated erythrocyte sedimentation rate. Colonoscopy is useful in determining the extent of the progress of the disease.

**TREATMENT OF CD**

There is no cure for CD. Therapy is usually directed to relieving symptoms and managing extraintestinal complications. An objective is to achieve and maintain remission that may occur during therapy or even in its absence. Since the disease is lifelong, treatment must be a continuing process. Several groups of options are utilized in the treatment of CD: immunosuppressants, aminosalicylates, corticosteroids, antimicrobials, antidiarrheals, nutrients, and surgery.

**IMMUNOSUPPRESSANT AGENTS**

These agents are utilized to reduce or prevent an immune response.

1. **Azathioprine**: Azathioprine (AZA) is an antagonist to purine metabolism and is used for its immunosuppressive properties. Its mechanism of action is unknown, but it may inhibit synthesis of RNA and DNA, and may also inhibit coenzyme formulation, resulting in inhibition of cellular metabolism. AZA is well absorbed from the GI tract, has a half-life of 5 hours, and is rapidly cleared from the bloodstream. It is metabolized in the liver to the active metabolites 6-mercaptopurine and 6–thioinosinic acid. Hematologic adverse reactions include bone marrow depression resulting in leukopenia, macrocytic anemia, and thrombocytopenia. The severity of these effects is dose dependent. Common GI side effects are nausea, vomiting, anorexia, and diarrhea. Administering the drug in divided doses and/or with meals may reduce these side effects. The risk of acquiring hepatotoxicity is higher when the drug is given in large doses (over 2.5 mg/kg). Presence of such toxicity is manifested by increased serum alkaline phosphatase, bilirubin, and ami-
notransferase. Other side effects include drug fever, chills, alopecia, arthralgia, hypersensitivity reactions, cough, mouth and lip sores. The drug is not recommended for use during pregnancy. Dosage must be individualized based on condition of the patient, response and tolerance. The usual recommended dose is 2 - 3 mg/kg daily.

2. Cyclosporine: Cyclosporine may be used to treat severely ill patients who are unresponsive to other therapies. Fungi produce this immunosuppressive agent. The drug is believed to exert its activity by inhibiting cell-mediated immune response. The exact mechanism of its immunosuppressive actions is unknown. Side effects include bleeding, tender or enlarged gums, fever, chills, vomiting, frequent urge to urinate, weakness, nephrotoxicity, paresthesias and tremor. The drug is available as a capsule or injection. When cyclosporine was administered parenterally 4 mg/kg daily for a week, it induced a positive response in 80% of patients. When given concurrently with corticosteroids, risk of seizures is increased. Consequently, these agents should be administered under strict medical supervision.

3. Methotrexate: Methotrexate is a folic acid antagonist that acts by reversibly inhibiting dihydrofolate reductase, the enzyme that reduces folic acid to tetrahydrofolic acid. This antimetabolite has shown to be capable of inducing remission when given in a dosage of 25 mg per week. The main disadvantages of methotrexate are its adverse reactions, and the contraindications for its use. The adverse effects include nausea, vomiting, diarrhea, abdominal pain, joint pain, fatigue, sores on mouth and lips, myelosuppresion, and hepatotoxicity. Because of these, patient monitoring is necessary.

4. Infliximab: Infliximab is a chimeric monoclonal antibody that may be used for active CD and fistuluous CD refractory to conventional treatment. It is administered by intravenous infusion within 4 hours of its reconstitution. Currently, all infusions are carried out in hospitals. The drug may cause invasive fungal and other opportunistic infections, demyelinating central nervous system lesions and activating latent multiple sclerosis. It should not be given to patients with congestive heart failure.

AMINOSALICYLATES

The aminosalicylates, sulfasalazine and mesalamine are the cornerstone of therapy for mild to moderate CD (the patient is ambulatory and able to take oral alimentation). There is no dehydration, high fever, abdominal tenderness, painful mass, and obstruction or weight loss of more than 10%.

1. Sulfasalazine: Sulfasalazine is composed of sulfapyridine bonded to 5-aminosalicylic acid (5-ASA). Upon intake of the drug, the enteric bacteria cause the cleavage of the 5-ASA moiety, which is responsible for the anti-inflammatory activity of the drug to be released into the intestine. About 10% to 15% of the drug’s dose is absorbed from the intestine, and the remainder is cleaved by the intestinal flora to form sulfapyridine and 5-ASA. The sulfapyridine is absorbed from the colon, while a small quantity of 5-ASA is absorbed. The exact mechanism of action of sulfasalazine in the treatment of CD is unknown. It may be due to the antibacterial activity of sulfapyridine and the anti-inflammatory action of 5-ASA. Once remission has been achieved, the usefulness of the drug in maintenance therapy is questionable. It may be given alone or in combination with other drugs. It may be administered both orally and/or rectally in the form of enema for distal disease. The adverse effects include bone marrow suppression, connective tissue disorders, hemolytic anemia, aching joints and muscles, bloody diarrhea, difficulty in swallowing, fever and chills. The usual initial dose is 3 - 4g daily. In patients who experience frequent side effects, an initial dose of 1 to 2 g daily is recommended. The usual maintenance dose is 2 g daily divided into 4 doses. Although dosage of 2 to 6 g daily achieves remission in 64 – 80% of the patients, dosages of more than 3 g daily are accompanied by increased incidence of side effects, largely due to the drug’s sulfapyridine moiety. Maintenance therapy may be continued indefinitely, but in mild cases a one-year course is appropriate. Sulfasalazine should be given after meals to reduce stomach upset. In addition, each dose should be taken with a full glass of water.

2. Mesalamine: Mesalamine is 5-aminosalicylic acid, and when taken orally, it exerts anti-inflammatory effects in the intestinal tract. The side effects caused by the sulfapyridine moiety of sulfasalazine led to the development of this sulfa-free aminosalicylic acid. It appears that larger doses of mesalamine may be given without a significant increase in adverse reactions. The mechanism of action of the drug is unclear, but it appears that its anti-inflammatory properties are due to local action rather than systemic. Although
mesalamine may be metabolized minimally to salicylic acid, this metabolite is not responsible for the drug’s pharmacologic activity. It has been postulated that mesalamine may inhibit prostaglandin and leukotriene synthesis during inflammation. Mesalamine may be administered orally and rectally (enema or suppositories). Only about 15% of a rectal dose is absorbed from the GI tract. Extensive absorption of the drug from the proximal portion of the GI tract occurs following oral administration of capsules or uncoated tablets. In order to achieve release of the medication in the lower portion of the GI tract, the drug should be given in the extended release form. Clinical improvement or remission may be attained in as many as 84% of the patients. The majority of patients who do not tolerate the adverse effects of sulfasalazine usually tolerate mesalamine even at higher doses. Rectal administration of mesalamine is usually well tolerated, but side effects such as abdominal cramps, bloody diarrhea, nausea, flatulence, rectal burning or soreness, rash and fatigue may occur. The long duration of rectal therapy, essential for positive therapeutic response, may affect compliance by the patient who may object to the long-term and inconvenient regular administration via enemas. The usual adult dose in the suppository form is 500 mg twice daily, and as an enema suspension is 4 g once daily, preferably in the evening, for up to 8 weeks or until remission is achieved. Suppositories should be retained for 1 – 3 hours or longer, if tolerated, to achieve good therapeutic effect. The dose in the capsule form is 1 g, four times daily for up to 8 weeks, whereas delayed-release tablets are administered 800 mg, three times daily, for 6 weeks.

**CORTICOSTEROIDS**

Corticosteroid therapy is utilized to suppress the inflammation in the bowel. Because of their side effects, corticosteroid use is limited in patients with moderate to severe CD. Diarrhea, abdominal pain and tenderness are reduced dramatically after initiation of therapy. Their onset of action is more rapid than that of the aminosalicylates.

1. **Prednisone** has been the mainstay drug for CD. Where there is no acceptable dosage schedule of prednisone, doses of 0.25 to 0.75 mg/kg of body weight for 8 to 12 weeks have been shown to achieve satisfactory clinical response. Once improvement takes place, the dosage is gradually reduced by 5 to 10 mg weekly until the daily dosage reaches 20 mg, and by 2.5 mg weekly thereafter. Long-term corticosteroid therapy appears to have no effect on maintaining remission. They cause intolerable side effects that include adrenocortical insufficiency, diabetes mellitus, adrenal atrophy, altered protein, fat and carbohydrate metabolism and protein catabolism, cushingoid symptoms, suppressed immune response, increased susceptibility to infection, muscle wasting, hypertension, edema, acne, impaired wound healing and peptic ulcer.

2. **Budesonide**, a newer corticosteroid with poor systemic absorption because of a 90% first-pass metabolism, is indicated for mild to moderate CD. It is associated with fewer side effects and less adrenal suppression than prednisone. It is more effective than mesalamine and is comparable to oral prednisone.

**ANTIMICROBIAL THERAPY**

Although there is no evidence that CD is caused by microbiologic agents, certain antibacterial agents are used empirically in treating mild to moderately active CD. These medications are given especially to patients who are acutely ill with fever and signs of peritoneal involvement. In some cases, they may be used concurrently with corticosteroids. **Metronidazole**, which possesses activity against anaerobic bacteria that reside in the GI tract, has been used to induce remission despite the fact that current efficacy data does not support the notion that the drug is capable of achieving remission. It has been shown that dosages of 10 mg/kg/day or 20 mg/kg/day resulted in significant improvement in ileocolitis and colitis, but remission rates were similar to those achieved with placebo treatment. Metronidazole appears to be beneficial when the perianal area is involved. Adverse effects include nausea, diarrhea, headache, dizziness, gastric distress, nephrotoxicity and anorexia. **Ciprofloxacin** in a dose of 1 g daily may cause decreased disease activity and remission. It may be used alone or in combination with metronidazole.
ANTIDIARRHEAL AGENTS

Conventional antidiarrheal drugs can provide symptomatic relief by diminishing intestinal motility. In CD, diphenoxylate, loperamide, paregoric and tincture of opium can reduce the intensity of diarrhea. Due to the chronic nature of CD, these drugs should be used with caution due to inducing narcotic dependence. Furthermore, these medications should not be used in cases of obstruction.

NUTRITIONAL TREATMENT

Nutritional therapy is used as an adjunct to provide needed vitamins, protein and minerals. This treatment is of particular value for growing children. Assessment should be based on the extent of calorie insufficiency, the presence of anorexia and weight loss.

SURGERY

When massive hemorrhage, perforation, or severe colitis are present, surgical removal of the entire colon and rectal mucosa may be required. About one third of the patients with UC will eventually undergo surgery. CD patients may require surgery especially in cases of intestinal obstruction, internal fistula and abscess, perforation and hemorrhage. Surgery in patients with CD usually is postponed as long as possible, and every attempt is made to conserve the bowel.

ULCERATIVE COLITIS

Ulcerative colitis (UC) is a chronic, inflammatory, ulcerative disease of the mucosa of the colon.

ETIOLOGY AND EPIDEMIOLOGY

CD and UC share a number of clinical, epidemiologic, immunologic, microbial and genetic factors. The exception is that the microbial etiology is more uncertain, and the inheritance factors are less distinct. It appears that UC is slightly more common among women than men. Although UC can affect patients at any age, it exhibits bimodal age distribution. A first major peak of incidence occurs at age 15 to 20, and a secondary, smaller peak, at ages 50 to 60.

PATHOLOGY

The inflammatory process in UC involves the colonic mucosa. In the majority of patients, the left side of the colon is affected, while in others, the entire colon is involved. Unlike CD, the inflammation is uniform and continuous, and the presence of normal segments of the colon is absent. The rectum is involved in the vast majority of the cases. Some clinicians believe that UC begins in the rectum or sigmoid and progresses proximally to involve the colon. Unlike CD, deeper layers of the bowel and the mesentery, as well as the regional lymph nodes are usually not involved. The colon is hyperemic, and the mucosa is dark and ulcerated and bleeds diffusely. Recurrent inflammation may lead to fibrosis underneath the mucosal epithelium, or to longitudinal retraction resulting in shortening of the colon. Degeneration changes may occur in the crypt epithelium, leading to necrosis and abscess formation.

SYMPTOMS

The major clinical manifestations of UC are rectal bleeding, bloody diarrhea, abdominal pain, weight loss and fever. The disease may begin in a subtle, mild manner, with increased urgency to defecate, mild abdominal discomfort, and the appearance of bloody stool along with mucus. Initially, the patient may relate these symptoms to other factors such as emotional stress, upper respiratory infection, or diet. However, the onset of UC symptoms may occur abruptly with sudden bloody diarrhea (4 – 5 bowel movements), low grade fever, or abdominal cramps that interfere with sleep. Rectal discharge of mucus may accompany or occur between bowel movements. Weight loss and anorexia may occur. The disease may become exacerbated and require intensive medical attention. UC patients may experience profuse diarrhea (10 – 20 bowel movements daily), fever as high as 39 degrees Centigrade, severe cramps, spasmodic
contractions of the rectum, profound weakness and pallor. The risk of developing cancer of the colon is more frequent in UC than CD.

**DIAGNOSIS**

Like CD, there is no specific diagnostic test for UC. Individuals who complain of recurring and persistent diarrhea, especially if it is bloody, perianal sepsis, abdominal pain, anorexia, and weakness should be considered candidates for UC. Sigmoidoscopy, rectal biopsy, and radiologic studies are helpful in the diagnosis of the disease. Ruling out other disorders such as hemorrhoids, colonic neoplasms, colonic diverticula, acute colitis, and amebiasis is essential in the confirmation of UC.

**TREATMENT**

Because the treatment for UC is very similar to that of CD, please review the treatment section for CD.

**COMPLICATIONS OF UC**

Complications of CD and UC can be local or systemic. Local complications such as fistulas, abscess and obstruction have been described earlier. Perforation is another potentially serious complication that may occur during the course of these diseases. The continued presence of ulceration and inflammation tends to thin the intestinal mucosa, thereby making it vulnerable to perforation or rupture. When this happens, the patient most likely will experience clinical features similar to those of peritonitis. The constant presence of inflammation may result in loss of neuromuscular tone of the bowel, resulting in colonic dilation. This dilation is more common in UC patients. Systemic complications of UC that are similar to those of CD have been discussed in the section dealing with CD.

**PROGNOSIS OF CD AND UC**

In recent years, much progress has been made in the treatment of IBD, resulting in improvement of the prognosis. While the etiology remains unknown, the use of medications discussed earlier has resulted in induction and maintenance of remission. Prognosis is usually poor in patients who develop the disease after the age of 60, in patients whose colon is totally involved, and in patients who experience toxic megacolon. After the first attack, approximately 10% of the patients experience remission that may last for several years, an additional 10% will have active colitis, and the remainder experience periods of remission and exacerbation. UC patients may develop colon cancer, in particular, if the entire colon is involved. About 33% of patients who suffer from extensive UC may eventually require surgery.

**CONCLUSION**

IBD is a chronic, inflammatory condition characterized by periods of remission and exacerbation that result in diarrhea, rectal bleeding, abdominal cramps, weight loss, and extraintestinal complications. Although surgery may be required in severe cases, pharmacotherapy with aminosalicylates, corticosteroids, immunosuppressive agents, and biologic agents are the mainstay for managing IBD. There is no cure for IBD, thus the use of medications is a life-long affair. To achieve good therapeutic results, treatment should be individualized based on the patient’s needs and tolerance to medications.

**References**

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Please fill-out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Please rate each of the following from 1 to 7. Circle your choices. (1 is the lowest rating; 7 is the highest).

1. Relevance of topic to practice. 1 2 3 4 5 6 7
2. Author’s ability to communicate. 1 2 3 4 5 6 7
3. Author’s knowledge of topic. 1 2 3 4 5 6 7
4. Appropriateness of topic. 1 2 3 4 5 6 7

5. Do you have any further comments about this lesson? ____________________________________________________________________
__________________________________________________________________________________________________________________

Please Select the Most Correct Answer

1. The peak incidence of CD is at this age:
   A. Teens
   B. Twenties
   C. Forties
   D. Sixties

2. Sulfasalazine should be taken:
   A. Before meals
   B. Only at bedtime
   C. Concurrently with methotrexate
   D. With a full glass of water

3. Which statement is incorrect about CD?
   A. The disease begins abruptly & causes violent diarrhea
   B. The disease begins as poorly defined pathologic changes in the intestine
   C. The intestinal mucosa is hyperemic
   D. Inflamed adjacent loops of the intestine may adhere to each other

4. Which of these is not considered a complication of CD?
   A. Renal complications
   B. Cardiac complications
   C. Hepatic complications
   D. Nutritional complications

5. Which statement is correct about mesalamine?
   A. Consists of a sulfapyridine moiety and 5-ASA
   B. Tolerated less than sulfasalazine
   C. It is a sulf-free amino salicylate
   D. Used only in oral dosage forms

6. Budesonide is:
   A. An aminosalicylate derivative
   B. Related to azathioprine
   C. A corticosteroid with poor systemic absorption
   D. Less effective than mesalamine

7. Which statement is correct about UC?
   A. Some believe that UC begins in the rectum
   B. UC involves only a small portion of the colon
   C. Inflammation in the colon is discontinuous
   D. Causes weight gain due to formation of edema

8. Which of these factors is not involved in causing CD?
   A. Genetic factors
   B. Infectious causes
   C. Immunologic factors
   D. Hormone imbalance

9. Which drug is not used for IBD?
   A. Metronidazole
   B. Opium tincture
   C. Ibuprofen
   D. Prednisone

10. Steatorrhea is a term meaning:
    A. Indigestion
    B. Fatty stool
    C. Increased flow of bile salts
    D. Severe diarrhea
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