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"Disorders of the GI Tract"   January 2018

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CREDITS FOR 2017.
THEY SHOULD BE IN YOUR cpeMONITOR OR CE BROKER ACCOUNT. It is difficult to believe, but this lesson begins the 40th year that we have been continuously providing CE PRN. Thank you to all of our participants for your continued confidence and loyalty.

Pharmacy has changed, but has it really? The mechanics are different, as are many drugs and, of course, the names of pharmaceutical manufacturers and "players in our profession." Yet, the ultimate goal of pharmacy, pretty much, remains the same---providing important drug and healthcare information to patients. In this lesson, we review IBD. We primarily discuss CD & UC. The goal is to provide updated information that can improve communications with patients.

This lesson is intended for pharmacists & technicians in all practice settings. **The program ID # for this lesson is 707-000-18-001-H01-P for pharmacists, and 707-000-18-001-H01-T for technicians.**

Participants completing this lesson by December 31, 2021 may receive full credit. Release date for this lesson is January 1, 2018. 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 1-847-945-8050. **Please write your name, NABP eProfile (CPE Monitor®) ID Number & birthdate (MM/DD) in the indicated space on the quiz page.**

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

For Pharmacists:

1. Compare & contrast Crohn's Disease (CD) & Ulcerative Colitis (CD).
2. Describe causes of Inflammatory Bowel Disease (IBD).
3. Discuss treating IBD.
4. Describe the role of diet in the management of IBD.
5. Differentiate between remission & relapse of IBD, & how both occur.

For Technicians:

1. Discuss treating IBD.
2. Describe the role of diet in the management of IBD.
3. Differentiate between remission & relapse of IBD, & how both occur.

INTRODUCTION

The digestive tract, especially that part which consists of the intestines, is susceptible to abnormal inflammatory processes that affect the intestinal mucosa. The presence of such inflammation can disrupt the normal function of the intestines leading to malabsorption and abdominal symptoms (ranging from mild to severe) that not only may affect the quality of the patient's life but sometimes can lead to threatening complications.

There are a number of inflammatory diseases that affect the gastrointestinal tract and are grouped under the title of inflammatory bowel disease (IBD). **The most common conditions are Crohn's Disease (CD) and Ulcerative Colitis (UC).** It is estimated that the number of IBD cases in 2009 among individuals 25 years and older was about 1,171,000 (approximately 565,000 for CD and 593,000 for UC). The number of cases is on the increase. CD and UC are chronic, relapsing, inflammatory disorders of the digestive tract with overlapping clinical, epidemiological, pathological, immunologic and genetic features. That being said, there are some differences that exist between the two diseases: location of the inflammation in the intestines; the status of the inflammatory process in the intestinal lining; the depth of inflammation involved within the intestinal mucosa; and course of remission and relapse.

1. The inflammatory process in CD can impact any place in the entire GI tract, from the mouth to the anus. In severe cases, mouth ulcers may appear. In UC, it is usually confined to the large intestine, mainly the colon and/or rectum.

2. CD mostly develops at the last part of the small intestine and the beginning of the large intestine. UC may begin in the rectum and move upward through the large intestine.

3. In CD the patches or lesions in the mucosa appear scattered and have a cobblestone appearance; whereas, UC areas look unbroken and without interruption.

4. CD affects most layers of the mucosa of the small intestine. The submucosa and subserosa exhibit fibrosis and the lining appears coarse and uneven due mainly to ulceration and thickening of the mucosa. The inflammatory process results in an edematous lining. Such changes may cause the intestinal lumen to become narrower leading to obstruction and adhesion at the loops of the intestine. In UC the inflammation involves the innermost tissue of the large intestine.

5. Incidence of remission and relapse is more common in CD than with UC.

6. The intestinal microbial flora issues and inheritance factors are less distinct with UC.

7. Although UC can affect people at any age, it exhibits bimodal (having two maxima) age distributions. A first major peak of incidence occurs at ages 15 to 20 and a secondary peak at ages 50 to 60.

In spite of these differences, it is not easy to differentiate between both.

Both conditions share many symptoms. However, there are some that are characteristic of each disease.

1. Rectal mucus and/or bleeding are more common in patients with UC.

2. Patients who suffer from CD experience constant mild to moderate cramping. UC triggers sporadic pain particularly during bowel movement. Patients often have an urge to defecate but without results.

3. Unlike CD, UC patients rarely develop perianal fistulas and fissures.

OVERVIEW OF IBD

CD is an idiopathic, chronic, relapsing inflammatory disease that affects the lining of the digestive tract. The disease was first described in 1932 by Crohn and his associates Ginsburg and Oppenheimer. At first, it was thought that the disease affected only the ileum (third portion of the small intestine, between the jejunum and cecum). Nowadays it is confirmed that it appears at the end of the last section of the ileum and the beginning of the large intestine (colon). However, any part of the gastrointestinal tract can be affected.

UC is a chronic bowel disease that affects the innermost layer of the colon. It is characterized by diffuse inflammation and tiny ulcers covered with thick fluid (mucus) produced by the intestinal mucosa.

Causes of IBD are unknown, but it appears that there is a complex interplay between factors that involve familial history of the disease and the environment. Stress and diet have been thought to complicate IBD. While there is no evidence to support this assumption, it is believed that these two factors are considered to be aggravating factors. Microbial, environmental, socio-economical, genetic and immunological factors play a role in contributing to the pathogenesis of IBD. Normal intestinal microbial flora appear to be harmless, and can assist in the digestive process. Any disturbances in the makeup of the flora can trigger GI problems. Under normal circumstances, the flora is not attacked by the body's immune system. In IBD patients, for unknown reasons, the immune system may mistakenly consider the normal microbial flora as invaders. As a result, immune system cells from the blood travel to the intestine to get rid of the flora and in the process the immune system invades the intestinal cells causing an inflammatory reaction known as autoimmunity. The resultant inflammation causes damage to the intestinal lining and triggers characteristic IBD symptoms. The inflammation may last for months, years or throughout life.

People living in urban areas in industrialized countries are more prone to develop IBD than those who live in rural and underdeveloped countries. Reasons that may be responsible for this risk factor are lifestyle and diet. In addition, people who live in the northern hemisphere are more susceptible to IBD where the climate is colder.

Individuals whose parents or close relatives have IBD are more prone to development of disease. About 5% - 20% of IBD patients have a parent, sibling, or child who suffers from the disease. Those patients are 10 times more at risk if they have a relative other than a sister or brother, but about 30 times greater if the relative is a parent or sibling. A number of genes have been connected to IBD. However, carrying these genes will not necessarily result in the development of IBD. The risk of genetic effect is greater in CD than UC.

IBD may occur at any age but it is encountered mostly in late adolescence and early adulthood. The incidence peaks between the ages of 15 and 30. About 20% to 30% of cases are diagnosed before age 20.

By and large, the incidence of IBD is equal in both men and women. In pediatric patients the incidence is more common among boys than girls. Even though IBD occurs among all ethnicities, it is most common among Caucasians.

TREATMENT OF IBD

There is no cure for IBD. Since the disease is a lifelong condition, treatment must be a continuing process. There are a variety of options that can be utilized to control symptoms and cause remission. Maintenance of such remissions can be achieved provided the patient is compliant with the recommendations of the healthcare professional.

The main goals of IBD therapy are:

1. Controlling of inflammation and slowing its progression.

2. Achieving symptomatic relief.

3. Maintaining and achieving remission (a period of time during which there is absence or reducing of symptoms) and avert relapse (recurrence of symptoms after a period of improvement).

4. Preventing complications.

5. Improving the patient's quality of life.

The main measure that may help achieve the treatment's goals is to control the inflammation that is responsible for triggering IBD and its signs and symptoms. Furthermore, controlling or stopping the inflammatory process may result in long-term remission. Successful treatment will minimize or prevent.

The use of medication as well as surgery are the main approaches to IBD therapy.

DRUG THERAPY

There are two approaches for IBD drug therapy:

1. The regimen begins with mild drugs. Depending on the outcomes, a stronger drug may follow in case the patient's response is not satisfactory (step-up approach).

2. The second approach is to begin the therapy with administering stronger drugs (top-down).

Several approaches are utilized in therapy:

1. Inmunosuppresants

2. Anti-inflammatory agents

3. Antimicrobial drugs

4. Antidiarrheals

5. Diet

IMMUNOSUPPRESSANTS

Immunosuppressants indirectly reduce inflammation, but their main action is to reduce or prevent immune responses that cause the release of inflammatory chemicals, such as cytokines, in the intestinal mucosa. These agents may be more effective when given with other IBD medications.

Azathioprine is a purine metabolism antagonist. Its exact mechanism of action is unknown. It may inhibit synthesis of RNA and DNA and may inhibit coenzyme formulation resulting in inhibition of cellular metabolism. The drug is metabolized in the liver to the active metabolite 6-mercaptopurine and 6-thioinosine acid. Azathioprine possesses potentially serious adverse effects which include bone marrow depression resulting in leukopenia, macrocytic anemia

and thrombocytopenia. Severity of these side effects are dose-dependent. Other side effects include nausea, diarrhea, anorexia, vomiting, and lowered resistance to infection. Blood tests should be regularly performed. Hepatotoxicity may occur at higher doses. Doses should be individualized based on the patient's condition, response and tolerance.

Cyclosporine, which is produced by fungi, is mostly used for treating seriously ill UC patients who are unresponsive to other therapies. It is believed that the drug exerts its action by inhibiting cell-mediated immune response. Adverse effects such as bleeding, tender or enlarged gums, fever, chills, vomiting and frequent urge for urination may be encountered. There is a potential for more serious side effects such as nephron and hepatotoxicities, seizures, and fatal infections. It is intended for short-term use because of its toxic potential as well as the availability of safer drugs.

Methotrexate is a folic acid antagonist, and is used primarily to treat cancer, psoriasis and rheumatoid arthritis. It is also used to treat CD in patients who do not respond to other therapies. It is capable of inducing remission when given by injections of 25 mg once a week. It possesses side effects, some of which are severe, and include nausea, vomiting, diarrhea, joint pain, fatigue, sores on the mouth and lips, bone marrow suppression, liver damage and potentially life-threatening pneumonia. Monitoring the patient is necessary.

Adalimumab is a tumor necrosis factor, formerly TNF alpha, inhibitor. TNF is a leukocyte-derived cytotoxin protein that causes necrosis (death) of tumor cells. It is a multifunctional cytokine with pro-inflammatory actions. Inhibition of TNF can result in reducing inflammation in autoimmune inflammatory disease such as IBD, psoriasis and rheumatoid arthritis. Adalimumab inhibits TNF by binding to its receptors resulting in reduction of the inflammation. Adalimumab was the first fully human monoclonal antibody drug that was approved by the FDA in December 2002. Originally developed for treating rheumatoid arthritis, the medication is given subcutaneously once every other week. Side effects include allergic reaction and rare cases of hepatotoxicity, serious blood disorders, and cardiac failure. Since TNF is a component of the immune system, its suppression by adalimumab can encourage the activation of latent infections such as tuberculosis, Hepatitis B, as well as opportunistic viral, bacterial and fungal infections. Rare cases of lymphoma have been reported. As a result, the FDA advised physicians to carefully monitor patients taking adalimumab.

Infliximab is a chimeric monoclonal antibody that contains constant human domain and mouse variable domains. Chimeric antibodies are easier and cheaper to make than fully humanized antibodies. The drug is a TNF inhibitor and was approved by the FDA for treating IBD as well as other autoimmune diseases in August 1998. It is administered as IV infusion at 6 to 8-week intervals. Like all TNF inhibitors it cannot be given orally as it will be destroyed by the gastric fluid. In addition to its use in IBD, it is employed for closure of perianal fistulas in CD. Its adverse effects are similar to other TNF inhibitors.

Certolizumab pegol is a fragment of a monoclonal antibody specific to TNF and was approved by the FDA in April 2008 for treating moderate to severe CD in patients who did not respond positively to standard treatments. Side effects include stuffy nose, sinus pain, diarrhea, and flu-like symptoms. Certolizumab is given by injection.

Golimumab is a monoclone produced by transgenic mice immunized with human TNF. It is a TNF inhibitor and was approved by the FDA in May 2013 to treat moderate to severe UC, especially refractory cases. Like other TNFs, it is administered by injection. Adverse effects are similar to the other TNFs.

Natalizumab and vedolizumab are monoclonal antibody drugs that inhibit the cell adhesion molecule integrin from binding to other cells in the intestinal mucosa. This group of medications is known as selective adhesion molecule inhibitors α - 4 integrins. Natalizumab was approved by the FDA in January 2008 for inducing and maintenance of remission in moderate to severe IBD. It has been shown that the drug is effective in increasing the rate of remission and its maintenance. It is recommended for patients who do not respond to TNF inhibitors. The main side effects include progressive multifocal leukoencephalopathy (a rare and usually fatal viral infection that causes damage or inflammation of the white matter of the brain). Due to causing this condition the natalizumab was temporarily withdrawn from the market. Currently, it is back on the market but with a special restricted program. Other side effects include headache, fatigue, nausea, and maybe exacerbation of CD in a small number of patients.

Vedolizumab has a mechanism of action and use similar to natalizumab. It was approved by the FDA in May 2014 for treating moderate to severe IBD (CD and UC). It has similar adverse effects like those of natalizumab, except there is no risk for causing brain infections.

ANTIINFLAMMATORY AGENTS

Corticosteroids are potent, fast-acting anti-inflammatory medications that have been in use in the treatment of IBD for decades. Patients feel relieved within days following the onset of the corticosteroids therapy. However, due to their serious adverse effects, they are employed for short-term use, in particular during flare-ups, to achieve remission, after which the therapy is changed to other medications to maintain remission. Corticosteroids are rarely used for maintenance therapy. In addition to being anti-inflammatory drugs, they are also immunosuppressive agents. As such they diminish the tendency of the immune system to make the IBD patient prone to infections. Moreover, the dose should be in the lowest possible effective amount and shortest duration. Corticosteroids may be given orally, intravenously, or by a rectal form. Methylprednisolone, prednisone, and budesonide are used. The anti-inflammatory medications are usually used initially in step-up therapy.

Aminosalicylates. Sulfasalazines are used to treat mild to moderate cases of UC and some CD patients whose inflammation is affecting the colon. Other members of the group such as mesalamine, balsalazide and osalazine are available for oral use or as enema and suppository. Sulfasalazine is composed of sulfapyridine bonded to 5-aminosalicylic acid (5-ASA). Upon intake of the drug, the enteric bacterial flora causes the cleavage of the 5-ASA moiety, which is responsible for the anti-inflammatory activity once released in the intestine. Maintenance therapy may be continued indefinitely, but in mild cases, one-year course may be adequate. Adverse effects of the aminosalicylates include headache, GI distress, nausea, vomiting, oligospermia (low sperm count), difficult breathing, connective tissue disorders, pain in the joints and muscles and increased sensitivity to sunlight.

ANTIMICROBIALS

These may be used in case of fever. Such medications may assist in controlling the amount of drainage that may occur in UC and to enhance healing of fistulas. It is believed that the use of antibacterials may check the presence of harmful intestinal flora and may reduce the response of the intestinal immune system.

ANTIDIARRHEALS

These drugs can provide symptomatic relief by decreasing intestinal motility and cramps. Medications such as loperamide can reduce the frequency of diarrhea. Antidiarrheals are recommended in mild cases. In mild to moderate situations antibiotics and aminosalicylate may be used. However, in severe flare-ups corticosteroids may be given for a short period to improve the intensity of the symptoms followed by immunosuppressants or biologicals such as TFNs.

DIET

Diet is useful as an adjunct therapy to provide balance and nutrition containing adequate amounts of vitamins, minerals, protein, fat and carbohydrates. The intestinal inflammation caused by IBD tends to retard absorption from the intestine, and, as a result, malnutrition may occur. There is no need to pass up entire food groups. Only items that aggravate the symptoms should be avoided. Assessment should be based on the extent of caloric insufficiency, the presence of anorexia and weight loss, as well as the presence or absence of flare-ups. There is no known specific diet that prevents or treats IBD. Likewise, there is no known rule that indicates that certain foods can always act as flare-up aggravators, symptom controllers or remission initiators. What works for one patient with IBD may not work for others. Diet and eating plans should be individualized. Therefore, the patient should maintain a diary to keep track of the foods that act as offending triggers. That being said, it is important that a healthy diet be continued to help maintain general good health. The patients and their families should be aware of the following:

1. IBD can result in appetite loss due to abdominal cramps, nausea and weakness.

2. In fighting IBD, the body spends considerable energy.

3. The inflammatory process that affects the intestinal mucosa make nutrient absorption deficient and may lead to malnutrition. This deficiency may lead to growth-suppression in children and teenagers.

Recommended foods during remission:

- Drink plenty of water (up to 10 glasses a day). Alcoholic and caffeinated beverages should be avoided as they may cause dehydration.

- Lean meat such as skinless chicken, fish, in particular salmon due to rich content in omega-3 fatty acids.

- Fruits and vegetables should be an essential part of diet. However, if raw vegetables trigger a flare-up, then they should be lightly cooked. Applesauce and bananas are good substitutes for raw fruits.

- Cheese is a good source of calcium, especially Swiss and cheddar that have reduced lactose. It should be kept in mind that some patients may find such products difficult to digest. Greasy foods that contain cheese may lead to diarrhea and flatulence. Likewise, fried foods should be avoided.

- Foods high in fiber like cereal, whole wheat and high grain should be avoided. Instead, refined cereals such as cream of wheat; dry cereal such as Special K; corn flakes; and rice cereal are suggested.

- Liquid nutritional formulas can boost energy level and provide needed nutrients.

- Moderation regarding sweets.

SURGERY

This option may be needed if CD fails to respond to medications. CD surgery may not result in curing the disease. About 60% to 70% of CD patients may require further surgery to treat complications such as intestinal blockage, thickening and narrowing of the intestinal lumen, perforation of the intestine, removal of a portion of the damaged intestine, bleeding and presence of abscess. CD symptoms may return following surgery and appear in a different area of the intestine. About 30% of CD patients will experience re-emergence of the symptoms within 3 years and in about 60% in 10 years. About 50% of patients who have undergone surgery may require at least one additional surgical procedure. The surgeries involved include: bowel resection or removal of the diseased portion, or strictureoplasty, a procedure to open up a narrowed area of the small intestine.

UC can be cured via surgery which involves removal of the colon and the rectum. Surgery can reduce the risk of causing colon cancer. Colectomy involves removal of the colon and connecting the small intestine with the rectum where stool can be passed normally. Proctocolectomy is performed where both the colon and rectum are removed and the last portion of the small intestine is passed through a hole in the abdomen known as a stoma. Waste is emptied into an ostomy bag worn around the waist. Ileal pouch anal anastomosis (IPAA) involves removal of the colon and rectum and creating a new rectum called a J-pouch out of the small intestine. Advantage of this procedure is there is no need to have an ostomy bag. The patient will be able to have normal waste removal. The surgery is completed in two stages about two months apart. Risks of surgery include infection and deficiency in absorbing nutrients and drugs due to the shortened intestine.

REMISSION AND RELAPSE

Remission is a period of time during which a disease or its symptoms diminish or disappear. Relapse indicates the return or recurrence of a past disease. Flare-ups of IBD symptoms may appear during treatment indicating that the disease is active. In spite of treatment, these flare-ups may remain for weeks or longer. They may vary in intensity ranging from mild abdominal pain and diarrhea to bleeding, fever, or constriction of intestinal lumen leading to obstruction. Once the flare-ups subside, the patient may experience a period in which the symptoms may disappear or are not sensed. When this happens, the patient is said to be in remission. During this period, the damaged or inflamed mucosa of the digestive tract begins the healing process resulting in abatement of inflammation. Depending on the severity of the condition, the period of remission may last for weeks to years. It is difficult to predict the timing of return of the flare-ups as the cycle of relapse and remission does not follow an identical pattern or course in all patients. Severity of the IBD symptoms, general health of the individual, as well as the presence of flare-up triggers, play a role in the cycle. The precise cause of relapse is unknown in spite of patient adherence to the same therapy that resulted in remission.

There are a number of factors that may trigger relapse:

- Emotional or everyday life stress
- Non-compliance with therapy
- Intake of NSAIDs and antibiotics that may case changes in the microbial intestinal flora
- Smoking
- Diet

SUMMARY

IBD is a common chronic disorder that affects the digestive tract. The precise cause is unknown but there are several factors that may lead to CD and UC. IBD can occur at any age but mostly in individuals at the ages between 15 and 20. There are differences and similarities between CD and UC symptoms. There is no cure for IBD but there are measures, including medications, that can control symptoms and may lead to remission. Treatment may include immunosuppressants, anti-inflammatory agents, antimicrobials, antidiarrheals, diet or surgery. There is no known diet that prevents or treats IBD. However, there are certain foods that may aggravate IBD symptoms.

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LESSON EVALUATION

Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?
    Compare & contrast CD & UC   YES NO
    Describe causes of IBD   YES NO
    Discuss treating IBD   YES NO
    Describe role of diet in managing IBD   YES NO
    Differentiate between remission & relapse of IBD   YES NO

2. Was the program independent & non-commercial?   YES NO

                         Low Relevance   Very Relevant
3. Relevance of topic   1   2   3   4   5   6   7

4. What did you like most about this lesson?_____

5. What did you like least about this lesson?_____

January, 2018. Vol 40 #1. "Pharmacy Perspective: Disorders of the GI Tract"

Please Mark the Correct Answer(s)

1. **Which statement is TRUE about IBD?**
    A. Caused by bacterial infection.
    B. CD affects only lower part of colon.
    C. CD can affect any part of GI tract.
    D. Lesions in GI tract are cobblestone shaped.

2. **Which of these is FALSE?**
    A. UC is a transient inflammatory disorder.
    B. It is uncommon for UC patients to develop perianal fistulas.
    C. Family history of IBD plays a role in causing CD & HC.
    D. Incidence of IBD peaks between the ages of 15 & 20.

3. **Which of these is NOT considered a goal in the management of IBD?**
    A. Inflammation control.
    B. Increasing patient's weight.
    C. Achieving remission.
    D. Prevention of complications.

4. **Cyclosporine is an:**
    A. Analgesic.
    B. Antidiarrheal medication.
    C. Antibacterial agent.
    D. Immunosuppressant drug.

5. **TNF inhibitors act by:**
    A. Binding to receptors of TNF.
    B. Enhancing biotransformation of TNF.
    C. Delay absorption of TNF.
    D. Rendering TNF insoluble.

6. **Which of these is not a TNF ?**
    A. Adalimumab.
    B. Sulfasalazine.
    C. Infliximab.
    D. Certolizumab.

7. **A side effect of adalimumab is:**
    A. Elevation of blood pressure.
    B. Bipolar depression.
    C. Activation of latent infections such as TB.
    D. Activation of peptic ulcer.

8. **Which of these may cause progressive multifocal leukoencephalopathy?**
    A. Natalizumab.
    B. Metronidazole.
    C. Golimumab.
    D. Loperamide.

9. **IBD:**
    A. Is contagious.
    B. Is limited to males.
    C. Is cured by medications.
    D. Tends to retard absorption of nutrients from intestines.

10. **Diet:**
    A. Leads to long-term remission.
    B. Can lead to IBD cure.
    C. Is useful as an adjunct.
    D. Leads to flare-ups regardless of type.
    E. None of these.