DIAGNOSIS AND MANAGEMENT OF
INFLAMMATORY BOWEL DISEASE

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A manuscript submitted in partial fulfillment of
the requirements for the degree of
MASTER OF NURSING

WASHINGTON STATE UNIVERSITY
College of Nursing
Intercollegiate Center For Nursing Education
October 1999
To the faculty of Washington State University:

The members of the committee appointed to examine the ICNE Research requirements and manuscript of Janette M. Worley find it satisfactory and recommend that it be accepted.

[Signatures]

Chair

[Signatures]
ACKNOWLEDGMENTS

Without the daily support and encouragement from family and friends, my dream of obtaining a Master’s in Nursing and becoming a Family Nurse Practitioner would not have been possible. I would like to extend a special thank you and acknowledgment to the following people:

To:

Dr. Lorra Schumann, my graduate advisor and manuscript chair:
Your expertise in didactic and clinical practice is invaluable, but it is your compassion and your love of teaching that makes you so extraordinary. I will never forget you.

Dr. Billie Severtson and Linda Torretta ARNP:
Thank you for your support and time as you served on my manuscript committee.

Margaret Ruby:
Without you, I’d be lost in an ocean of paperwork and correspondence. Thank you for always making an extra effort.

I also want to extend my deepest gratitude and affection to

Harry Bray MD, my mentor:
Your professional guidance and encouragement is always appreciated;

and most urgently, to my family:

Keith, my wonderful husband and my two darling boys Alex and Andrew,
thank you for your tolerance in sharing my heart and mind for so long.
Presenting complaints of gastrointestinal (GI) symptoms are becoming more and more common in the primary care settings. GI complaints account for approximately 20-25% of all primary care visits. The purpose of this article is two-fold: 1) to provide a better understanding of Inflammatory Bowel Disease and 2) to provide the practitioner with criteria to accurately diagnose and manage IBD.
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DEDICATION

This manuscript is dedicated to my mother, Joan O. Quill. Your strength and unconditional faith continue to inspire me.
Presenting complaints of gastrointestinal (GI) symptoms are becoming more and more common in the primary care settings. GI complaints account for approximately 20-25% of all primary care visits. The purpose of this article is two-fold: 1) to provide a better understanding of Inflammatory Bowel Disease and 2) to provide the practitioner with criteria to accurately diagnose and manage IBD.

A Case Study:

CJ, an 18 year old girl diagnosed with viral gastroenteritis returns to clinic 48 hours later for re-evaluation. CJ continues to have diarrhea (8-12 stools/day). Right lower quadrant (RLQ) pain (mild 48 hours ago) has markedly increased. The patient states that the abdominal pain and diarrhea started “3 days ago and has just gotten worse”, additionally she states that now she feels very weak. CJ denies any history of lactose intolerance, or recent use of antibiotics. She denies contact with known TB patients and denies recent travel.

Physical exam: BP 136/88 (lying) 120/76 (sitting); P. 90; R.14; T. 99.6; height: 5 ft 1 inch; weight: 103 lbs. Patient reported 6 lb. weight loss over the last month without dieting. Abdominal assessment revealed RLQ pain with tenderness and guarding. The liver was non palpable. Bilateral tender nodules (2cm) were palpated on anterior lower legs. Initial labs showed microcytic anemia, hypoproteinemia and leukocytosis. Stool was negative for ova and parasites. Abdominal CT for perforated appendix was negative.

Inflammatory Bowel Disease (IBD) is the most likely diagnosis for this patient. IBD in broad terms, refers to any disease involving various degrees of inflammation in either the large or small intestines, usually chronic in duration, characterized by symptomatic flare-ups and remissions. Crohn’s Disease (CD) and Ulcerative Colitis (UC) are the major forms of IBD (Coulson, 1994).

EPIDEMIOLOGY:

Often Crohn’s Disease and Ulcerative Colitis are lumped under the inclusive term “IBD” because they can be clinically similar. It’s worth noting that in 5%-10% of patients, a clear separation may not be possible (Tanka & Riddell, 1990; Ehrhardt, 1996). Both CD and UC affect approximately 5 out of every
100,000 people (Cooke, 1991). These diseases affect both men and women, although women tend to have a slightly higher occurrence rate (Katz, 1994).

The onset of IBD most commonly occurs in early adulthood, between the ages of 15-40 yrs. (Cox, 1995). However, clinicians are seeing an increase in the incidence of late onset IBD (Fleischer, Grimm & Friedman, 1994; Eisen, Schultz, Washington, Burton, Sidhu-Malik & Wilson, 1993). Late onset patients present similarly, although the symptoms tend to be less severe (Fleischer, Grimm & Friedman, 1994).

Both Crohn’s Disease and Ulcerative Colitis have historically been more prevalent in industrialized western cultures (Cox, 1995). Inflammatory bowel disease tends to cluster in families. Kirshner (1991), reports that Ulcerative Colitis is more frequent among families with Ulcerative Colitis probands. Crohn’s Disease occurs more frequently among Crohn’s Disease probands. However, the two diseases intermingle in at least 25% of families. The risk of developing Crohn’s Disease is far greater for smokers than non-smokers, while the risk of developing Ulcerative Colitis is greater in non-smokers.

**ETIOLOGY:**

The etiology of Inflammatory Bowel Disease (Crohn’s Disease and Ulcerative Colitis) is still unknown. Theories of etiologies include: environmental, immunologic and genetic. IBD is thought to result from an abnormal immune response from an environmental trigger, in people who are genetically susceptible to either Crohn’s Disease or Ulcerative Colitis. An immune mediated component in IBD seems likely due to the fact that immune-related, systemic complications of the disease respond well to immunosuppressive therapy (Kirshner, 1991).

Research has established that neither Crohn’s Disease nor Ulcerative Colitis are conventional genetic diseases; there is no known chromosomal defect, gene mutation or inheritable protein responsible for IBD (Kirshner, 1991). Gene clusters thought to be responsible for IBD immune sequences include: HLA (chromosome 6), complement components (chromosome 6 and 19), T-cell antigen receptors (chromosome 7 and 14) and immunoglobulin heavy and light chain markers (chromosome 2 and 14) (Bouma, et al., 1997). Many studies have linked instances of familial reoccurrences of both CD and UC, strongly suggesting a genetic susceptibility in the pathogenesis of the disease. Although, the actual cause of
IBD is unknown, it is most likely a result of multiple etiologic mechanisms: genetics, individual immune systems, and the environment (Kirsner, 1989).

**PATHOPHYSIOLOGY:**

The small bowel and colon are primarily known for the processes of digestion and reabsorption, not necessarily their immune functions. "The intestinal mucosa has two major and largely exclusive functions: nutritional uptake and host defense" (Bischoff, 1996, p443). Mucosal layers must be selectively semi-permeable while at the same time provide a physical barrier against environmental threats (Tlaskalova-Hogenova, et al., 1995). Similar to the skin, the intestinal lining is a barrier to toxins. The gastrointestinal tract is a major pathway of entry for external antigens.

The innermost layer of intestine is a mucosal layer lined with epithelial cells. The epithelial cells of the mucosa have a rapid turnover rate of about 4-5 days. When irritated, these cells can shed more quickly and cell replacement can fall behind. The intestinal mucosa contains different types of immune cells. When a foreign substance (antigen) gains access to the mucosa, and is recognized, the epithelial cells (from mucosal lining) break down the foreign material for T-lymphocytes processing. B-lymphocytes produce antibodies that bind to and neutralize the antigen. Macrophages and mast cells surround the invading antigen, destroy it and remove harmful particles.

When the mucosal barrier breaks down, harmful antigens can penetrate into deep layers of the intestinal wall causing an inflammatory response. Increased blood flow transports white blood cells and plasma cells to the inflamed area. Edema occurs causing tissue damage. If the inflammation continues (i.e. excess epithelial permeability, over-reacting epithelium) ulcerations can result. Researchers have not determined which environmental triggers are likely to cause these chain of events, but it is certain to be an area of further interest and study (Kirshner, 1991).

Genetics will also be an area of further study. Every individual has a genetically controlled regulatory mechanism that governs the interactions of B-cells, T-cells, helper cells and suppressor cells, thus the body's immune function can be argued to be genetically mediated. Research has shown that the strongest risk factor for developing IBD is having a relative with the disease (Cottone, et al., 1997). It seems reasonable that genetic influences, either protective or susceptible, play an important role in
Inflammatory Bowel Disease. The gene mechanisms responsible for susceptibility to either Crohn’s Disease or Ulcerative Colitis may someday be identified. This could lead to the development of gene markers that could positively identify Inflammatory Bowel Disease.

**CLINICAL MANIFESTATIONS:**

The clinical manifestations of Crohn’s Disease and Ulcerative Colitis can be similar. While some characteristics provide diagnostic clues to aid in determining CD or UC, there are cases in which the location and the severity of the disease causes some overlap. Table 1 shows clinical characteristics of Crohn’s Disease and Ulcerative Colitis. (McQuaid, 1999).

**DIAGNOSTIC TESTING:**

When dealing with the constitutional complaints of pain, diarrhea, weakness: a differential diagnosis may seem virtually endless. However, the insidious onset of these symptoms allows the clinician to narrow the possibilities. Persistent or increasing complaint of RLQ pain and bloody diarrhea indicate the need for further testing. Without a detailed history, an initial diagnosis of viral gastroenteritis may be justified.

First priority should be to rule out appendicitis, which is warranted with a complaint of sudden onset of increasing RLQ pain. Often appendicitis can mimic bowel disease (McQuaid, 1999). In approximately 25% of teenagers and young adults with Crohn’s Disease, the initial presentation mimics that of acute appendicitis (McQuaid, 1999). Even with a negative result, a computed tomography (CT) scan would be of less harm than a perforated appendix.

Initial labs should include white blood cell count (WBC) with differential, complete blood count (CBC), hematocrit and hemoglobin, erythrocyte sedimentation rate, serum albumin and electrolytes. Inflammatory bowel disease may give results of microcytic anemia, leukocytosis, hypoproteinemia or hypematremia. A stool specimen should be collected and screened for ova and parasites (O&P). A positive O&P or stool culture may indicate an infectious process, such as Yersinia enterocolitis, Salmonella, Amebiasis, and Giardia lamblia (Fleischer, et al, 1994). A negative O&P or stool culture allows the clinician to rule out infectious diseases.
As a rule, patients under the age of 40, presenting with bloody diarrhea and pain should be evaluated for Inflammatory Bowel Disease and referred for a colonoscopy (Macrae & Bhathal, 1997). When a patient presents with a change in bowel habits, bloody diarrhea and pain; malignancy should be included in the differential and ruled out accordingly. Less commonly seen are intestinal tuberculosis, intestinal lymphoma, and undiagnosed human immunodeficiency syndrome (AIDS) (Farmer, 1990; Abel, Chiu, Russell & Volpe, 1990). These diseases can present similarly with constitutional symptoms (fever, pain, diarrhea, and weight loss).

**DIFFERENTIAL DIAGNOSIS OF IBD:**

Often the symptoms of Inflammatory Bowel Disease are indicative of location and severity and help to differentiate Crohn’s Disease or Ulcerative Colitis. When the disease is confined to the ileum, diarrhea tends to be moderate to severe, stools are often loose and watery. When the disease is in the colon, urgency and incontinence are frequent and rectal bleeding is not uncommon. Low grade fever is primarily due to the inflammatory process and thus seen in both Crohn’s Disease and Ulcerative Colitis.

Abdominal pain complaints may vary due to physiologic origin. RLQ pain is commonly due to ileocecal inflammation. Non-specific lower abdominal cramping usually means there is some colon involvement. Intermittent, periumbilical pain is most common just prior to evacuation of the bowel.

Distinguishing between Crohn’s Disease and Ulcerative Colitis can be a challenge. Crohn’s Disease can affect any area of the GI tract from mouth to anus. The surface of the inflamed bowel usually has a “cobblestone” appearance. This inflammation leads to the development of fissures/crevices (that develop around areas of submucosal edema), patches of full-thickness lesions and areas of normal appearing tissue.

Ulcerative Colitis manifests as a continuous mucosal erosion that is restricted to the colon, occasionally involving the ileum as “backwash ileitis” (Katz, 1994). Crohn’s Disease, when localized in the colon, can be easily confused with Ulcerative Colitis. Accurate diagnosis is made with a detailed patient history, laboratory abnormalities, diagnostic tests, and mucosal biopsies.

Ultrasonography has been used to detect mucosal thickening, however, research using ultrasonography to detect IBD has shown limited reliability as a diagnostic test (Lim, Ko, Lee, Lim & Kim,
The most common diagnostic tests to confirm a diagnosis of IBD are barium enema and endoscopy. A barium enema is used to show characteristic changes in the outline of the bowel wall. A barium enema is helpful in identifying full thickness lesions (Crohn’s Disease), but may not show superficial erosions, edema or friability (Ulcerative Colitis). Often, if a diagnosis of Crohn’s Disease is suspected an upper gastrointestinal series (with a small bowel follow-through) and a colonoscopy must be obtained. A colonoscopy provides complete visualization of the intestinal wall and allows the endoscopist to take serial biopsies. Biopsies usually provide the information necessary to determine whether Crohn’s Disease or Ulcerative Colitis is the cause of the problem and show the extent of the disease (Macrae & Bhathal, 1997).

Studies have been conducted to establish the cost effectiveness of a flexible sigmoidoscopy compared to a total colonoscopy for the initial evaluation of Inflammatory Bowel Disease (Deutsch & Olson, 1997). Flexible sigmoidoscopy allows examination of the distal 60-65 cm of the colon. Consequently, it does not require sedation (in most cases) making it less costly; however it may not give adequate information. Colonoscopy, requiring sedation (in most cases) is more expensive, but provides valuable information regarding the extent of the disease. Deutsch and Olson (1997) concluded that colonoscopy would be the more cost effective exam for physicians choosing to establish the extent of Crohn’s Disease or Ulcerative Colitis. Both barium enema and colonoscopy are somewhat similar in that they both require a bowel preparation. Each procedure can also cause considerable discomfort; however conscious sedation is now being used routinely for colonoscopies, decreasing the pain associated with this type of procedure. These exams may be contraindicated during severe flare-ups due to the risk of perforation.

**COMPLICATIONS:**

**Growth failure**

In children and adolescents, growth failure is the most common complication of both Crohn’s Disease and Ulcerative Colitis (McCarthy, 1997). Growth failure is seen more frequently in Crohn’s Disease (Kirsner, 1991). In adults, an unexplained weight loss (10% or more over 1 month) is typical of IBD (Kirsner, 1991).
Malnutrition

As a result of the chronic inflammation seen in IBD, malnutrition is also a common complication. Iron deficiency anemia is seen in both Crohn’s Disease and Ulcerative Colitis as a consequence of inadequate intake, malabsorption and mucosal injury with subsequent blood loss (Israel & Kleinman, 1994). Megaloblastic anemia is due to a deficiency in vitamin B\textsubscript{12} or folic acid (Binder, 1997).

Hypoproteinemia or the enteric loss of albumin occurs in virtually all patients with intestinal inflammation. In both Crohn’s Disease and Ulcerative Colitis, proteins leak out of ruptured capillaries and into tissues at the site of inflammation. Consequently, children with active IBD should be encouraged to increase their protein consumption to 125% of the recommended daily allowance (RDA) for their age group (Israel & Kleinman 1994). Other potential malabsorption losses include: calcium, magnesium, and potassium deficiencies (manifested by bone pain, fractures, paraesthesia or tetany). A vitamin K deficiency should be suspected with any signs of bleeding tendency (ecchymosis, melena, hematuria). While growth failure and malnutrition are systemic complications of IBD, other complications depend on the location and severity of the disease.

Abscess:

Abscesses can occur resulting from the microscopic loss of intestinal barrier function. They are manifested by a tender abdominal mass, fever and leukocytosis. An abscess should be confirmed with CT then treated with broad spectrum antibiotics. Drainage and/or surgical removal is usually necessary (McQuaid, 1999).

Carcinoma:

Patients with Crohn’s Disease (especially colonic involvement) and Ulcerative Colitis are at increased risk for developing colon cancer (Rubio & Befrits, 1997; Itzkowitz, 1997). Hastings and Weber (1993) report that although colon cancer occurs less frequently in Crohn’s Disease than in Ulcerative Colitis, Crohn’s patients are still five times more likely to develop colon cancer than in age matched controls. Most cancers occur in the inflamed segments of the bowel, particularly colorectal and at the site
of strictures and fistulas (Tanaka & Riddell; 1990). The American Cancer Society (1998) recommends routine colonoscopy screenings for patients with either Crohn’s Disease or Ulcerative Colitis every 2 years, beginning 8-10 years after initial diagnosis. “In patients who have had colitis for more than 10 years the risk of developing colon cancer increases approximately 0.5-1.0% per year.” (McQuaid, 1997, p. 593).

Fistulas:

Fistulas between loops of bowel, occur frequently in Crohn’s disease. Externally, they can manifest as perianal discomfort with tracts in the skin. Treatment including sitz baths and oral metronidazole (Flagyl) are helpful (Israel & Kleinman, 1994). Internally, these abnormal passageways between loops of the bowel can be asymptomatic and require no surgery. More often, fistulas bypass large portions of small bowel (reducing absorptive surface area) and contribute to malnutrition problems. The consequent re-routing of intestinal contents through fistulas can cause stasis and promote bacterial overgrowth. Most symptomatic fistulas require surgical removal, if a course of mercaptopurine (Purinethol) fails (McQuaid, 1999).

Hemorrhage:

The massive bleeding that can occur in Ulcerative Colitis is rarely seen with Crohn’s Disease. Signs and symptoms of hemorrhage are related to hypovolemic shock (rapid thready pulse, hypotension, syncope, cool-clammy skin, pallor) and usually require immediate surgical intervention (McQuaid, 1999).

Obstruction/Strictures:

Bowel obstruction and strictures (often at the terminal ileum) are a result of active inflammation or fibrous scarring within the lumen of the bowel and are more commonly seen in Crohn’s Disease. Patients with confirmed obstruction or severe stricture (usually presenting with nausea and vomiting) should be started on IV fluids and nasogastric suction. These patients usually require surgical removal of the obstructed bowel or balloon dilation of the stricture (McQuaid, 1999).
Perforation:

Perforation of the bowel is a complication more frequently associated with Crohn’s Disease than Ulcerative Colitis. Patients presenting with rigid, quiet abdomen and rebound tenderness should have an upright abdominal x-ray to confirm perforation (air under the diaphragm). Broad spectrum antibiotics (e.g., ticarcillin/clavulanate or (Timentin) 3 grams every 4-6 hours IV) and emergency surgery are needed to prevent peritonitis (McQuaid, 1999).

Toxic Megacolon:

Toxic megacolon, usually only a complication of Ulcerative Colitis, is characterized by abdominal distention with tenderness and colonic dilation greater than 6 cm. (McQuaid, 1999). Serial abdominal x-rays are needed to show further dilation or ischemia. These patients also require broad spectrum antibiotics, IV fluids and nasogastric suctioning.

EXTRAINTESTINAL MANIFESTATIONS:

Extraintestinal features are not “true” complications of IBD, since they are not a direct result of the disease, rather they are usually seen in association with both Crohn’s Disease and Ulcerative Colitis. These features are sometimes so subtle, they go undiagnosed until a severe episode of IBD occurs. “It is increasingly apparent that few organs escape recruitment when IBD is chronic or progressive” (Balan, LaRusso 1995; p643). Extraintestinal manifestations include: spondylitis and arthritis (joints), iritis and episcleritis (eye), erythema nodosum and pyoderma gangrenosum (skin), hemolytic anemia and thrombocytopenic purpura. Most extraintestinal features occurring with either Crohn’s or Ulcerative Colitis have immune mechanisms as a major etiology.

MANAGEMENT:

Inflammatory bowel disease is a chronic, lifelong illness characterized by periodic exacerbations’ and remissions. There is no definitive treatments or “cures” (with the exception of colectomy for Ulcerative Colitis). Therapy should include: supportive measures, drug therapy, nutrition maintenance and surgical treatment, if necessary.
Supportive Measures:

The patient with either Crohn’s Disease or Ulcerative Colitis requires emotional support. As with any chronic, “incurable” disease, the patient is at risk for depression. A positive clinician/patient relationship is crucial for successful long-term care. Symptomatic medications such as antidiarrheals (Loperamide or (Imodium) 2-4 mg QID PRN) and antispasmodics (Propantheline or (Pro-Banthine) 15 mg a.c.) are helpful and should be offered to increase “bowel normalcy”. However, the patient needs to be aware that these medications could increase the risk of bowel obstruction and should be discontinued if obstruction is suspected.

Transdermal nicotine patches have shown to be useful in prolonging remissions of Ulcerative Colitis. Topical administration of nicotine (delayed release oral capsule, enemas, foam) is the preferred route to deliver nicotine to the colon having fewer adverse effects than transdermal patches or intravenous administration (Compton et al., 1997).

Drug Therapy:

Pharmacologic intervention is used not only to reduce symptoms, but more importantly to reduce inflammation and suppress the immune response. Treatment with 5-Aminosalicylic Acid (5-ASA) is the mainstay therapy for both Crohn’s Disease and Ulcerative Colitis. It is largely unabsorbed in the body, and acts topically within the colon. For this reason, 5-ASA is available in many forms and prescribed according to infected area of the bowel.

Sulfasalazine (Azulfidine 1.5-2 grams/day achieved gradually) is a combination of Sulfapyridamine and 5-ASA. Colonic bacteria breaks this medication down allowing 5-ASA to work topically within the colon. Because sulfasalazine is broken down in the colon, this medication is not effective in the small intestines.

Mesalamine (Pentasa 1.25 g TID/ Asacol 800 mg TID) is a form of 5-ASA enterically coated with a pH sensitive resin. This allows release of 5-ASA, enabling it to work topically in the small bowel.

Enema and Suppository preparations of 5-ASA (Rowasa) provide high concentrations of 5-ASA to the distal colon with minimal side effects. To maximize their effectiveness, these forms of 5-ASA are administered before sleep.
Corticosteroids dramatically reduce symptomatology in both Crohn’s Disease and Ulcerative Colitis, but do not treat underlying disease. Short-term use of corticosteroids is usually limited to acute exacerbations. Corticosteroids are available in a variety of formulations (IV, oral, suppository, enema, and foams). For acute flare-ups oral Prednisone 40-60 mg/day for 2-3 weeks is usually effective, and should be tapered then discontinued. Although tapering schedules vary, a decrease of 10 mg/day in weekly intervals is recommended.

Cyclosporine (Sandimmune) is reserved for severe cases of Ulcerative Colitis. This immunosuppressant is often used with transplant patients to reduce the likelihood of organ rejection. Cyclosporine has been shown to beneficial to patients with severe steroid resistant colitis. However, due to multiple, serious toxicities, cyclosporine should only be administered with diligent, unremitting monitoring (Robinson, 1997). Monitoring should include BUN, serum creatinine, liver function tests, bilirubin, amylase and lipase.

6-Mercaptopurine (Purinethol 50mg/day) or Azthriprine (Imuran 100mg/day) are less toxic than cyclosporine, but are slow acting immunosuppressants and therefore not indicated for acute illness. For patients using chronic steroid therapy, immunosuppressant therapy should be considered. 6-Mercaptopurine permits reduction or elimination of steroids in over 75% of patients (McQuaid, 1999). 6-Mercaptopurine has also been shown to heal fistulas in 30-40% of patients. (McQuaid, 1999).

Antibiotic treatment has been used empirically for years. Studies have shown that combining antibiotic therapies (clarithromycin, ciprofloxin and metronidazole) with conventional steroid treatment can increase efficacy and decrease the inflammation of IBD (Robinson, 1997). Antibiotics are most commonly used to treat fistula and abscess formation seen with Crohn’s Disease.

Several pilot studies using methotrexate (25 mg/week IM, SQ or PO) are showing promise for future treatment of Crohn’s Disease (Feagan, 1995). Other areas that are under investigation include treatment with acemann (a derivative of aloe vera) to reduce signs and symptoms of activity and heparin therapy to reduce extraintestinal manifestations of IBD (Robinson, 1997).
Nutrition:

There is no standard diet that corrects IBD. The primary care provider and patient must work as a team to develop an individualized diet that produces a satisfactory outcome. A “food diary” is often helpful to determine individual responses to certain food types. Intolerant foods can vary greatly per individual. According to studies conducted in England, longer remissions were achieved when patients eliminated certain foods, particularly intolerant to each individual. The exclusion of these foods enabled 51 of 77 patients to remain in remission for up to 51 months; with an annual relapse rate of less than 10% per year (Jones, 1985). To date, these studies have not been replicated.

Controversy over a fiber-rich diet vs. a low fiber diet continues (Rossner, 1993). Nair and Mayberry (1995) report that the average Western diet contains about 11g of fiber/day, while the recommended intake should be 20-35 g/day. High fiber diets have water retention capabilities which increase bulk and soften the stool. High fiber stool lubricates the colon and ultimately reduces fecal transit time. A patient with strictly colonic involvement may benefit from fiber supplementation (Mcquaid, 1998). However, for the majority of IBD patients, a low fiber/residue diet is usually favored. Fiber (including nuts, popcorn, raw vegetables and fruit) arriving at the colon largely undigested can precipitate abdominal cramping, irritate the intestinal mucosa or create an obstruction at the site of a stricture.

Meeting nutritional requirements is especially important for patients with inflammatory bowel disease. Usually the nutritional needs of the patient can be met by oral intake alone (with a multivitamin supplement). However, when gastrointestinal symptoms become active, eating sufficient quantities of food may become difficult.

Israel & Kleinman (1994) report, “the prevalence of lactose intolerance in children with IBD is not increased when compared with healthy age-matched, control children with chronic recurrent abdominal pain” (p 101). Therefore, children should not be restricted from lactose unless they exhibit symptoms of intolerance. Lactose (the sugar molecule in milk products) may arrive at the intestines undigested, causing gas, bloating and diarrhea. For these patients “Lactaide” or avoidance of milk products may be necessary.

A low fat diet with medium-chain triglyceride oil (cooking and salad dressing) is usually recommended. Patients report frequent small meals produce fewer symptoms than the customary three large meals a day.
Panza and Porro, (1985), suggests that both Crohn’s Disease and Ulcerative Colitis positively correlated with high intake of refined carbohydrates. The researchers also suggested that a vegetarian diet reduced the risk of developing inflammation of the bowel to between 0.4-0.7 in Crohn’s Disease and 0.3-0.4 in Ulcerative Colitis.

Enteral feeding delivers liquid formulas directly into the intestines. Formulas should include elemental diets (Vivonex). Enteral feedings greatly reduce gastrointestinal irritation and can induce remissions.

Total parenteral nutrition (TPN) is used during active disease for patients with severe malnutrition. This method of nutrition allows the intestines time to rest and recover.

**Surgery:**

At some point in time the majority of patients with inflammatory bowel disease will require surgery (Kirshner, 1991). In Ulcerative Colitis, a surgical resection (colectomy) can eliminate further symptomatology, indefinitely. In Crohn’s Disease, surgery is only palliative; a surgical resection may help temporarily, but given the spreading nature of the disease, it is likely to flare-up in another area of the gastrointestinal tract, usually at the site of anastomosis. Other indications for surgery, which were mentioned earlier, include: abscesses, enteric fistulas, massive bleeding, obstruction or severe stricture.

**SUMMARY:**

The chronic, unpredictability of inflammatory bowel disease makes it difficult for patients to cope. In fact several studies quoted by Cox (1995) found that the majority of IBD patients, even the one’s who considered themselves “well”, experienced some impairment in quality-of-life satisfaction. Early detection of IBD is essential in developing patient confidence and providing motivation for cooperation in treatment. Irvine (1997) conducted a study dealing with the quality-of-life issues with IBD and concluded that despite impairments, most patients with IBD overcome the obstacles imposed by their illness and manage to remain productive members of society.

During the early stages of the disease, similar management (with anti-inflammatory drugs) makes differentiating between Crohn’s Disease and Ulcerative Colitis unnecessary. Situations that require
differentiation include: right sided pain or tenderness, steatorrhea, nutritional deficiencies, or a palpable mass (Macrae & Bhathal, 1997).

Although IBD continues to be of unknown etiology, recent advances and further study in the areas of the immune system, genetics and environmental influences may provide helpful treatment options in the future. For now, the clinician/patient goal must be to maintain adequate nutrition, promote healing, treat complications, and maintain an optimal lifestyle.
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<tr>
<td>Methylprednisolone Hydrocortisone</td>
<td>0.8-1.6 mg/kg PO 100 mg BID Enema 80 mg BID Foam</td>
<td>local anti-inflammatory</td>
</tr>
<tr>
<td>IMMUNOSUPPRESSANTS 6-Mercaptopurine</td>
<td>1-1.5 mg/kg PO</td>
<td>suppresses immune response antagonizes purine metabolism</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>1.5-2.0 mg/kg PO</td>
<td></td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>4-6 mg/kg PO 2-4 mg/kg IV</td>
<td>inhibits interleukin-2 (inhibiting T-cell activity)</td>
</tr>
<tr>
<td>ANTIMICROBIALS Metronidazole</td>
<td>10-20 mg/kg PO/IV</td>
<td>Treatment of intra-abdominal infections</td>
</tr>
<tr>
<td>Ciprofloxin</td>
<td>20-30 mg/kg PO</td>
<td></td>
</tr>
</tbody>
</table>
References


