Quick Review: IBD - Ulcerative Colitis

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Abstract

Ulcerative Colitis is one form of chronic idiopathic illness characterized (primarily) by gastrointestinal symptoms. UC, along with Crohn's Disease (Regional Enteritis), comprise the syndrome referred to as Inflammatory Bowel Disease. IBD is the major cause of chronic intestinal inflammation in North America with a usual onset in late childhood and adolescence; in general, the clinical presentation of IBD is characterized by unpredictable remissions and exacerbations.

IN GENERAL

The two subtypes of IBD can be quickly compared in the summary table below:

Figure 1

	Ulcerative Colitis	Crohn's Disease
Rectal Bleeding:	common	sometimes
Rectum Involved:	always	sometimes
Ileum Involved:	"backwash"	common
Abdominal Mass:	not usually	common
Perianal Disease:	not usually	common
Strictures:	not usually	common
Fistulas:	not usually	common
"Skip" Lesions:	not usually	common
Depth Involved:	MURAL	TRANSMURAL
Crypt Abscesses:	common	not usually
Granulomas:	not usually	common
Cancer Risk:	3% - 1st decade, then 20% per decade after	slightly increased

The division between the types of IBD can be, in some cases, quite difficult to draw ! Crohn's is, most classically, a nonspecific, chronic, transmural, inflammatory disease that most commonly affects the distal ileum and colon - but may occur in any part of the gastrointestinal tract from the mouth to the anus.

Ulcerative Colitis is also a nonspecific, inflammatory and ulcerative disease - usually arising in the rectum and distal colon then spreading through the mucosa (with direct extension) proximally to the ileum.

UC is characterized by recurrent bloody diarrhea associated with mucosal inflammation.

It has an estimated incidence of 3 - 15/ 100,000 and a Prevalance of 40 - 225/ 100,000.

Approximately 20% of cases begin in adolescence or childhood (with the peak ranging from 15 - 25 years of age); there is a bimodal distribution to it's incidence with a second peak occurring at ages 50 - 70.

ETIOLOGY

Etiology of UC is unknown and the familial tendency is slightly less than that seen in Regional Enteritis.

1. UC usually begins in the rectosigmoid and extends proximally; however, there is a subtype that is milder in degree which is limited to the rectum (Ulcerative Proctitis).

2. In young patients, the entire colon is more often involved than in adults.

3. Pathologic change begins with degeneration of the mucosal epithelium (specifically the Reticulin Fibers), occlusion of the subepithelial capillaries, and progressive infiltration of the lamina propria

with white blood cells; all types invade but there is a predominance of PMN's.

4. Eventually, Crypt Abscesses, epithelial necrosis, and mucosal ulcerations develop.

SIGNS & SYMPTOMS

Clinically, the signs and symptoms of U.C. are fairly constant:

attacks of bloody diarrhea varying in intensity and duration but with intermittent asymptomatic periods.

The ususal attack is gradual in onset (though it may be explosive and fulminant with sudden violent diarrhea, high fever, signs of peritonitis, and toxemia).

The initial symptoms involve an increased urgency and frequency to defecate.

The diarrhea contains fresh blood and mucus which worsens as the disease progresses.

Lower abdominal cramps which worsen just before defecation are commonly seen.

When the disease and ulcerations are limited to the rectum, the feces may be hard and dry but as the process moves proximally the stools become looser and the pt may experience 15 - 20 bowel movements per day.

This later stage usually is accompanied by severe cramps and tenesmus throughout all hours of the day.

The stools may be watery and contain pus, blood, and mucus.

Associated signs and symptoms include: malaise, fever, anemia, anorexia, weight loss, leukocytosis, hypoalbuminemia, and an elevated ESR.

On physical exam, the abdomen is usually tender to palpation - particularly on the Left side and hyperactive bowel sounds can be auscultated.

DIAGNOSIS

Diagnosis is firmly established by Colonoscopy with Biopsy and pathologic confirmation of the above-mentioned microanatomical changes. The mucosa will appear as “friable” and dark-red; a “spongy” surface that is dotted with tiny ulcerations can be seen through the scope. In children, this examination must be undertaken with great care because of the risk of Toxic Megacolon. Plain films of the abdomen or barium contrast studies can not be reliably relied upon in establishing a diagnosis.

Differential diagnosis must include an infectious cause before concluding that it is idiopathic UC. Stool cultures for Salmonella, Shigella, and Campylobacter must be obtained;

- The presence of Entamoeba should be excluded by direct examination of still-warm stool specimens.
- Prior history of antibiotic use should trigger a stool assay for difficile toxin. In the male (especially of homosexual orientation), infectious proctitis should be ruled out (gonorrhea, herpes, chlamydia).
- In women using oral contraceptives, the physician should consider the possibility of “contraceptive-induced colitis” which resolves completely after stopping the medication.
- In the elderly, ischemic colitis and colon cancer should be ruled out. Most importantly, and possibly the most difficult to rule out, is the other main component of IBD: Crohn's Disease which is best done via biopsy.

PROGNOSIS

Prognosis usually is dependent on the age of onset.

In most cases beginning during childhood, the process is severe - both in activity and extent of involvement. Usually, the course is chronic with repeated exacerbations and remissions, but complete recovery after the initial attack can occur in up to 10% of cases. In another 10%, an attack may become fatal with a rapid onset of hemorrhage - perforation - sepsis - and toxemia. Nearly one-third of all patients with extensive U.C. ultimately require surgery; total proctocolectomy is permanently curative and, when performed in time, restores both life expectancy and quality of life to normal !

There is currently no curative medical treatment for UC, but medications can reduce the activity of the inflammation and the frequency of recurrence.

Supportive treatment is important to both the patient and the family.

A full and variable diet should be instituted with folate and iron supplementation. If the pt is seriously malnourished or

has sustained a lengthy period of time without nutritional support, TPN should be considered (as it was in our pt). Sulfasalazine is used to reduce inflammation (50 - 75 mg/kg/24 hrs) and side effects are uncommon. However, it does interfere with folate absorption so pts should receive supplemental folate. Corticosteroids are most effective in treating in the treatment of active disease. Enema dosing is available but for more severe disease oral Prednisone (1 - 2 mg/kg/24 hrs) should be used; even IV dosage can be used for extreme cases. Once started, steroids must be tapered and not stopped abruptly. Newer medications (cyclosporine, azathioprine, and 6-mercaptopurine) are also utilized with varying success.

Since the disease can be cured by surgical resection of the entire colon, it must be considered in long-term treatment.

The common indications of operative treatment of a child with U.C. are severe acute disease that does not respond to at least 2 wk of intensive treatment and prolonged or debilitating symptoms, particularly if there is growth or maturational delay.

Prophylactic proctocolectomy should be recommended for extensive disease - especially for patients less than 15 yr of age.

COMPLICATIONS

- Hemorrhage (which is the most common local complication),
- Toxic Megacolon (which develops from the loss of muscular tone and subsequent colonic dilation of the Transverse Segment to exceed 6 cm),
- Perforation
- Peritonitis
- Sepsis

The risk of colon cancer is a major consideration especially with long-standing disease. Such patients should be closely monitored for the early warning signs of carcinoma.

Extracolonic complications include: Arthritis, Ankylosing Spondylitis, Posterior Uveitis, Erythema Nodosum, Pyoderma Gangrenosum, and in children - severely retarded growth and development. In patients that started experiencing symptoms at a young age, the risk of Sclerosing Cholangitis is now becoming recognized.

References

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