INTRODUCTION

Ulcerative colitis is characterized by a diffuse, continuous inflammatory process usually limited to the superficial mucosa of the colon. Crohn disease entails a more focal, transmural inflammation that can affect the colon either alone or accompanied by small bowel involvement; both have the potential for severe, fulminating, or toxic colitis (1).

Since the original classification was published by Truelove and Witts in 1955, severe, acute ulcerative colitis has been defined by the presence of six or more bloody bowel movements per day associated with temperature above 37.5°C, heart rate greater than 90 beats/min, hemoglobin below 10.5 g/dL, and/or an erythrocyte sedimentation rate (ESR) greater than 30 mm/hr (2). It has long been recognized that these criteria are indications for hospitalization and intravenous corticosteroid therapy (3,4). The term fulminant colitis has been used to distinguish the most severe classification of colitis with progression of mucosal inflammation into deeper layers of the colon wall. Patients have increased bloody bowel movements to at least 10 per day, anemia requiring transfusion, abdominal distention and tenderness, and may develop circular muscle paralysis precipitating dilatation of the colon (2).

Toxic megacolon is a potential complication of fulminant colitis. Its original definition by Jalan et al. (5) requires radiologic evidence of colon dilation (>6 cm) and three of the following:
- Profound tachycardia (heart rate >120 beats/min)
- Fever (>38.5°C)
- Anemia
- Leukocytosis (total white blood cell count >10,500 cells/μL)

In addition, the patient must have at least one of the following: dehydration, altered mental status, hypotension, or electrolyte disturbances (5) (Table 129.1). Toxic megacolon is a medical emergency requiring more intense and combined medical and surgical management (6) and should be distinguished from megacolon without systemic toxicity, which can occur with Hirschsprung disease or intestinal pseudo-obstruction.

INCIDENCE

Acute severe colitis (as defined above) requiring hospitalization is estimated to occur in about 15% of patients with ulcerative colitis (7). Of these patients that are hospitalized, the colectomy rate is lower on first admission (20%) and increases with subsequent admissions. The lifetime risk of toxic megacolon was about 1% to 5% in earlier studies, but this risk is now thought to be lower secondary to better management of severe colitis (8). Toxic megacolon is more common in extensive colitis than in proctitis or proctosigmoiditis (9). However, limited right- or left-sided segmental colitis (5,10) has been associated with toxic megacolon (9,11).

MORTALITY

Mortality from severe colitis is less than 2%, with a colectomy rate of about 30% (2,3). Although mortality in early series was as high as 25%, reaching 50% if colonic perforation occurred, early recognition and management of toxic megacolon has substantially lowered mortality to below 15% (5) generally and, in experienced centers, usually below 2% (12). Factors associated with increased mortality include age older than 40 years, the presence of colonic perforation, and delay of surgery (5). Colonic perforation, whether free or localized, is the greatest risk factor leading to increased morbidity or death.

ETIOLOGY

Though it was formerly a complication predominately of ulcerative colitis, more recently there has been a shift in epidemiology toward infectious causes of toxic megacolon due to the increase in Clostridium difficile infection (13,14). Toxic megacolon has also been described with other idiopathic and infectious colitis, including Crohn disease (15), pseudomembranous colitis secondary to methotrexate, Kaposi sarcoma (8), amebic colitis (16), and other infections (Shigella, Salmonella, Campylobacter, Yersinia, Chagas disease, cryptosporidium, and cytomegalovirus [CMV]) (14,18). Other rare causes of toxic megacolon include ischemic colitis (19), collagenous colitis, and obstructive colorectal cancer (14). Certain drug therapies have been implicated as precipitating factors in the development of toxic megacolon as well as diagnostic tests including barium enema and colonoscopy (Table 129.2).

PATHOPHYSIOLOGY

The exact mechanism of dilation in toxic megacolon is unknown. The depth of inflammation correlates with the extent of dilation. Inflammation is characterized by infiltration with neutrophils, lymphocytes, histiocytes, and plasma cells with preservation of submucosal and myenteric nerve plexuses (20) arguing against a neuropathic process. One proposed mechanism is the overproduction of inflammatory mediators that inhibit smooth muscle tone, such as nitric oxide. Nitric
Damage to muscle cells from proteolytic enzymes from neutrocyte enema resulted in decreased colonic distention (22). Direct inhibition of the propria of patients with toxic megacolon when compared to colitis controls without dilation (21). Inhibition of this oxide synthase (NOS) was found to be increased in the muscularis propria of patients with toxic megacolon when compared to colitis controls without dilation (21). Inhibition of this enzyme using a nonselective reversible inhibitor administered by enema resulted in decreased colonic distention (22). Direct damage to muscle cells from proteolytic enzymes from neutrophils has also been implicated (8).

### TABLE 129.1 Determination of Severity of Bowel Disease: Clinical/Laboratory Findings

<table>
<thead>
<tr>
<th>Disease Severity</th>
<th>Severe Disease</th>
<th>Fulminant Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stools (number/day)</td>
<td>&gt;6</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Blood in stool</td>
<td>Frequent</td>
<td>Continuous</td>
</tr>
<tr>
<td>Temperature</td>
<td>&gt;37.5°C</td>
<td>&gt;37.5°C</td>
</tr>
<tr>
<td>Pulse</td>
<td>&gt;90 bpm</td>
<td>&gt;90 bpm</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>&lt;75% of normal</td>
<td>Transfusion required</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate</td>
<td>Colon wall edema</td>
<td>Dilated colon</td>
</tr>
<tr>
<td>Radiographic features</td>
<td>Thumb printing</td>
<td>Abdominal tenderness and tenderness</td>
</tr>
<tr>
<td>Clinical examination</td>
<td>Abdominal tenderness</td>
<td>Abdominal distention and tenderness</td>
</tr>
</tbody>
</table>

### TABLE 129.2 Etiology of Toxic Megacolon

**Inflammatory bowel disease**
- Crohn disease
- Collagenous colitis

**Infectious causes**
- Clostridium difficile
- Shigella
- Salmonella
- Campylobacter
- Yersinia
- Chagas disease
- Cryptosporidium
- Cryptosporidiosis
- Rotavirus
- Aspergillus
- Entamoeba
- AIDS-related Kaposi sarcoma

**Drugs**
- Methotrexate
- Chemotherapy agents

**Inhibitors of colonic motility**
- Diphenoxylate atropine sulfate (Lomotil)
- Loperamide
- Narcotics
- Anticholinergics

**Other causes**
- Electrolyte and pH disturbances, hypokalemia
- Barium enema
- Colonoscopy
- Ischemic colitis
- Obstructive colorectal cancer
- Acute ischemia of the colon due to sigmoid stenosis related to diverticulitis
- Behçet disease
- Hemolytic-uremic syndrome (HUS) caused by Escherichia coli O15

### DIAGNOSIS

#### Clinical Presentation

Patients with severe and fulminant colitis present with symptoms of rectal urgency, continuous bloody diarrhea, abdominal pain and distention, fevers, weight loss, and dehydration (23). On physical examination, one may find fever, tachycardia, abdominal tenderness and mild distention, tympany, and decreased bowel sounds (24).

The presentation of toxic megacolon typically evolves with progressive diarrhea, bloody stools, cramping abdominal pain, and abdominal distention. Impaired consciousness and lethargy may be present (5). Occasionally, in chronically treated patients, a paradoxical decrease in stool frequency with passage of only bloody discharge or bloody membranes may be an ominous sign. On physical examination, peritoneal irritation, including rebound tenderness and abdominal guarding, represent transmural inflammation with serosal involvement, even in the absence of free perforation. Conversely, peritoneal signs may be minimal or absent in elderly patients or those receiving high-dose or prolonged corticosteroid therapy. In such patients, loss of hepatic dullness may be the first clinical indication of colonic perforation. Mental status changes, including confusion, agitation, and apathy, are occasionally noted (25).

#### Diagnostic Workup

Laboratory abnormalities in severe and fulminant ulcerative colitis include leukocytosis, anemia (hemococoncentration must be taken into account), hypoalbuminemia, hypokalemia, hyponatremia, and elevated sedimentation rate and C-reactive protein (CRP). The degree of metabolic alkalosis correlates with the severity of colitis (26). Stool analysis for ova and protozoa, C. difficile, Escherichia coli O157:H7, Campylobacter, Salmonella, and Shigella should be performed as part of the diagnostic workup (26,27).

A plain abdominal radiograph can determine the extent of ulcerative colitis by the absence of fecal material distal to the margin of disease and the presence of air outlining normal haustrations proximal to the disease margin (28,29). Radiologic features of fulminant colitis include wall thickening, with islands of edematous mucosa surrounded by deep ulcerations (24,30,31). In patients with severe colitis, small bowel ileus may herald toxic megacolon (32,33) and is a bad prognostic sign for medical success (12). Plain films of the abdomen are usually sufficient radiographic studies to evaluate toxic megacolon and may reveal loss of haustration, shortening of the colon, mucosal thumbprinting, with segmental or total colonic dilatation ranging from 6 to 15 cm (24,34,35). Maximal dilatation typically involves the transverse or right colon with dilation of the sigmoid and rectum occurring rarely (36). Free peritoneal air indicates perforation, though retroperitoneal tracking of air from a colonic perforation may produce subcutaneous emphysema and pneumomediastinum without pneumoperitoneum (37). Although not all patients require computed tomography (CT), it may be more reliable than plain film radiographs in evaluating the length and severity of colitis and the presence of complications such as abdominal abscess and perforations (38,39). Findings that differentiate toxic megacolon from severe acute colitis on CT include...
segmental colonic wall thinning, air-filled colonic distention more than 6 cm, abnormal haustral pattern, and nodular pseudopolyps (36).

Endoscopic evaluation in the setting of toxic megacolon is contraindicated due to risk of perforation. However, a limited proctoscopic examination or flexible sigmoidoscopy with minimal air insufflation may be performed safely in severe colitis to evaluate the mucosa for pseudomembranes or ischemia (29). More extensive endoscopic examinations (40) are generally contraindicated due to the risk of perforation or inducing toxic megacolon. However, they have been performed safely in some experienced centers (26). Examination generally shows extensive ulceration with friable, bleeding mucosa. In rare instances, however, such as with rectal enema therapy or in the setting of Crohn disease, the rectum may be normal. In patients whose initial presentation of inflammatory bowel disease (IBD) is severe colitis, biopsies should be performed to evaluate for Crohn disease and to rule out acute self-limited colitis. In those with an exacerbation of known diagnosis, biopsies can help to exclude CMV infection (17).

If performed, the presence of severe colitis (deep penetrating diagnosis, biopsies can help to exclude CMV infection (17). Examination generally shows extensive ulceration with friable, bleeding mucosa. In rare instances, however, such as with rectal enema therapy or in the setting of Crohn disease, the rectum may be normal. In patients whose initial presentation of inflammatory bowel disease (IBD) is severe colitis, biopsies should be performed to evaluate for Crohn disease and to rule out acute self-limited colitis. In those with an exacerbation of known diagnosis, biopsies can help to exclude CMV infection (17). If performed, the presence of severe colitis (deep penetrating diagnosis, biopsies can help to exclude CMV infection (17).

**TREATMENT**

Few medical emergencies require as close cooperation between medical and surgical personnel as does fulminant colitis. A team approach with early management and continuous assessment by both groups is vital not only to determine whether surgery is indicated, but also to support critically ill patients preoperatively and postoperatively. Early recognition and institution of therapy by an experienced team can alter the outcome of this life-threatening illness (3,4,26,27).

**Medical Treatment**

Resuscitative measures in fulminant colitis and toxic megacolon are paramount and should include vigorous fluid, electrolyte, and blood replacement to maintain the serum hematocrit at approximately 30%. The goal of fluid replacement should be to restore previous losses and continue replenishing those that are ongoing from diarrhea, fever, and third spacing of fluids (26). Although severe hypokalemia may not be present, total body potassium depletion is common and may be exacerbated by glucocorticoids such that resuscitative measures should include adequate potassium replacement (26). Parenteral nutritional support in attempts to correct malnutrition and electrolyte and acid–base balance—including replenishment of phosphate, calcium, and magnesium—should be initiated. Despite the fact that bowel rest is an ineffective primary therapy for severe colitis, oral intake of fluids should be discontinued in fulminant colitis, or once colonic dilatation is recognized (42). IBD is associated with an increased risk of arterial and venous thromboembolism (43) and prophylaxis should not be withheld due to concern for bleeding. Anticholinergic and narcotic agents should be discontinued immediately. In the presence of small bowel ileus a nasogastric tube may be placed, however this is not helpful for colonic decompression (44). Patient repositioning from front to back or prone knee–elbow position may redistribute colonic air and assist in decompression (45,46). Serial abdominal plain films should be obtained every 12 hours initially and then daily as the patient improves (8). Rarely, patients with dilatation in the absence of toxic signs or symptoms may benefit from rectal tube decompression.

Aminosaliclyates, commonly used for maintenance therapy and the treatment of mild-to-moderate ulcerative colitis, have no role in the treatment of fulminant colitis (3,4,26,27). Their limited activity on superficial inflammation is insufficient to abort or control the transmural disease, and potential adverse effects—nausea, vomiting, or worsening colitis—may confuse the clinical picture. These drugs should be withheld until the patient has recovered and resumed a normal diet.

Corticosteroids are the mainstay of treatment of fulminant colitis and should be started if there is no obvious infection (47,48). There is no general agreement regarding which corticosteroid preparation or dose should be given. Several preparations and dosages have been used and are listed in Table 129.3. Approximately 75% of patients respond to corticosteroids in the setting of severe fulminant colitis (29,47,49) and less than half fail to achieve remission (27). Mechanisms of steroid resistance have not been fully elucidated (30) but the presence of hypoalbuminemia, high CRP, short duration of illness, and prior corticosteroid use are predictors of medical failure (51,52). In addition, ex-smokers have a worse prognosis (53).

The most critical assessment in the setting of fulminant colitis is the response to therapy within the first 5 days (29). Short-term prognosis to corticosteroids in severe disease can be predicted as early as 24 hours. Various prediction tools have been developed to estimate the risk of colorectal surgery or need for second-line medical therapy (54). The Travis (Oxford) index found that stool frequency greater than eight per day or three to eight per day and a CRP more than 45 mg/L on day 3 of IV corticosteroids had a positive predictive value of 85% for patients requiring colectomy (29). The Ho (Scottish) index, which assigns points (0 to 9) for stool frequency, colonic dilation, and albumin level at day three of IV corticosteroids, has not been validated prospectively but has 85% sensitivity and 75% specificity in predicting nonresponse to medical therapy with a cutoff of 4 or more (55). Continuation of intravenous corticosteroids beyond 7 to 10 days does not provide any additional benefits (47,56) and may increase morbidity and surgical risks (57).

Generally, most patients with IBD will have been receiving corticosteroids before toxic megacolon developed, in which

---

**TABLE 129.3 Medication Dosages**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Corticosteroids</strong></td>
<td></td>
</tr>
<tr>
<td>Prednisolone sodium phosphate: 25 mg intravenously every 6 hrs IV</td>
<td></td>
</tr>
<tr>
<td>Hydrocortisone: United States—100 mg every 6 hrs IV, with total daily dose of 400 mg</td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone: United States—6–15 mg every 6 hrs IV, with total daily dose of 40–80 mg; Europe—1 mg/kg</td>
<td></td>
</tr>
<tr>
<td><strong>Calcineurin inhibitors</strong></td>
<td></td>
</tr>
<tr>
<td>Ciclosporine: 2 mg/kg IV</td>
<td></td>
</tr>
<tr>
<td>• Oral conversion = double the IV dose for twice-daily oral administration (e.g., if IV dose is 100 mg/24 hr, the oral dose would be 200 mg twice daily)</td>
<td></td>
</tr>
<tr>
<td><strong>Anti-TNF agent</strong></td>
<td></td>
</tr>
<tr>
<td>Infliximab: 5 mg/kg IV at weeks 0, 2, 6</td>
<td></td>
</tr>
<tr>
<td>• Alternatively, higher induction doses have been used including 10 mg/kg at weeks 0, 1, 2</td>
<td></td>
</tr>
</tbody>
</table>
case they should be continued. There is no evidence that corticosteroids precipitate or worsen outcome in toxic megacolon. Similar to therapy for fulminant colitis, augmented doses of corticosteroids should be administered in view of the additional stress of the toxic state. There is concern that corticosteroids could mask signs of perforation or peritonitis, so close monitoring is necessary. In cases of toxic megacolon caused by infectious etiologies, corticosteroids should not be used. Just as in fulminant colitis, there is no consensus regarding the corticosteroid preparation for treatment in toxic megacolon.

Broad-spectrum antibiotics, with adequate gram-negative and anaerobic coverage, should be administered without delay once transmural inflammation or toxic megacolon is suspected (13). Antibiotics should be continued until the patient stabilizes over several days to a week or through the initial postoperative period. Whether antibiotics help avert progression of toxic megacolon is not known.

Patients who improve, as evidenced by restoration of formed bowel movements with the absence of bleeding and ability to pass flatus without using the toilet, are then transitioned to oral prednisone. They may be discharged from the hospital when tolerating a low-residue diet with formed stools without blood or rectal urgency; premature discharge is doomed to failure and readmission. Aminosalicylates are added as a maintenance therapy once patients are tolerating oral steroids and a full diet. The long-term prognosis after hospitalizations for severe colitis requiring corticosteroid therapy is not as promising as once considered (58–60).

Patients who fail to respond to IV corticosteroids require salvage medical therapy or surgery. Cyclosporin A has been used for 20 years, but anti–tumor necrosis factor (anti-TNF) agents such as infliximab are gaining favor when corticosteroids fail. Other agents that have been mentioned in case reports include oral tacrolimus (61), adalimumab, hyperbaric oxygen therapy, and leukocytapheresis (62).

Cyclosporin, a calcineurin inhibitor, administered as a continuous IV infusion, either alone (63) or in combination with corticosteroids, has been effective in treating severe ulcerative colitis (56). Immediate response rates up to 85% to 92% have been reported (64) and, similar to corticosteroids, failure to improve—as defined by having eight or more stools per day or persistence of CRP elevation after 3 days of cyclosporin—is predictive of the need for colectomy (29). Careful daily monitoring for serious side effects of nephrotoxicity, infection, and seizures must be carried out when using cyclosporin (65). Once patients have responded to intravenous cyclosporin with achievement of a clinical remission (defined previously) they are transitioned to oral cyclosporin (see Table 129.3). Patients receiving a combination of corticosteroids and cyclosporin should receive Pneumocystis prophylaxis with sulfamethoxazole-trimethoprim three times weekly (66). Forty to fifty percent of patients treated with intravenous cyclosporin experience long-term remission (66–69). Improved outcomes are reported for patients who have been transitioned to oral cyclosporin with the addition of 6-MP or azathioprine (66,70).

Infliximab, a chimeric anti-TNF monoclonal antibody, has been shown to be effective as outpatient therapy for patients with moderate to severe ulcerative colitis (71). Though cyclosporin was traditionally the preferred salvage agent used in fulminant colitis, more experience with infliximab in this setting has proven it to be as effective and possibly a safer option as it does not require the same therapeutic monitoring (72–75). One open-label randomized trial of adults with severe acute colitis who did not respond to 5 days of IV corticosteroids found that patients who received IV infliximab had similar response rates on day 7 (85%) and treatment failure rates at day 98 (54% vs. 60%, p = 0.52) as those who received IV cyclosporin (73). Additionally, no difference has been found in long-term colectomy rates (74). Furthermore, it can be continued as a maintenance drug whereas cyclosporin is eventually stopped and the patient typically is maintained on immunomodulators such as azathioprine or 6-MP. An accelerated dosing strategy whereby infliximab is given more frequent then the standard outpatient induction of 5 mg/kg at week 0, 2, and 6 may further improve outcomes and lower colectomy rates from 40% (with standard dosing) to 7% (76).

There have been no head to head trials comparing cyclosporin versus infliximab as salvage therapy for fulminant colitis. Limited data exist on the safety and effectiveness of sequential use of one drug after failure of the first. One small study including 19 patients found a 40% remission rate for patients treated with infliximab after failing cyclosporin and a 33% remission rate for patients treated with cyclosporin after failing infliximab. However, sepsis and death was reported in one patient and serious infection remains a significant concern for sequential use of salvage medical therapy (77).

Other anti-TNF agents have been used only in case reports with some success. A new biologic, vedolizumab, which is a humanized immunoglobulin G1 monoclonal antibody to α4β7 anti-integrin, approved to treat moderate-to-severe ulcerative colitis, has not been studied as salvage therapy in the case of fulminant colitis not responding to corticosteroids (78,79).

Surgical Management

Indications for surgery in fulminant colitis include clinical deterioration with massive hemorrhage, perforation, peritonitis, or failure to respond to medical therapy (3,4,26,27). Evidence of colonic perforation is an unequivocal indication for emergent surgery and is associated with severe complications, including peritonitis, extreme fluid and electrolyte imbalance, and hemodynamic instability. Other indications for emergent surgery precluding protracted medical management include signs of septic shock, multiorgan dysfunction (26,80), and imminent transverse colon rupture (diameter >12 cm) (13).

The type of operation is dependent on the clinical condition of the patient and the experience of the surgeon (26,27,81,82,83). Most surgeons prefer a laparoscopic or conventional subtotal/total colectomy with ileostomy, leaving the rectosigmoid as a mucous fistula or the rectum alone, using a Hartmann procedure (84). This approach has the advantages of limiting the lengthy pelvic dissection in acutely ill patients while allowing for the option of a subsequent restorative, sphincter-saving procedure (ileoanal anastomosis) that is typically performed months later to minimize the risk of complications including anastomotic leak (85,86). A one-stage procedure that cures ulcerative colitis without the need for a second operation is appropriate for older patients or those not desiring restorative ileal pouch–anal anastomosis. Rarely, “blow-hole” colotomies may be useful in highly selected individuals with poor operative prognoses (87,88).
In the case of megacolon, special consideration must be paid to prevent iatrogenic perforation and fecal spillage during colectomy (89).

The 30-day operative mortality rate after colectomy was found in a large systematic review to have decreased over time from 10% in the 1970s to 1.8% more recently, with complication rates of 20% for small bowel obstruction/ileus, 18.6% wound infection, 17.8% intra-abdominal abscess, 9.1% septicaemia, 6.7% rectal stump dehiscence, and 6.3% ostomy problems (39,89,90).

Managing Pregnant Women with Inflammatory Bowel Disease

The treatment of fulminant colitis and toxic megacolon in the pregnant woman is similar to the nonfemale gendered, nonpregnant patient. Continued severe illness poses a greater risk to the fetus than the medical or surgical intervention (91). Diagnostic modalities of ultrasound and magnetic resonance imaging (MRI) are safe and can be used for detection of abscess or colonic wall thickening and dilation. Low-dose radiographs (<5 rad) have minimal risk to fetus (92).

Flexible sigmoidoscopy is a valuable tool to assess the severity of disease and anatomic extent of disease. It is considered a safe procedure to perform when indicated (93). Full colonoscopy is rarely indicated. Polyethylene glycol solution has not been studied in pregnancy; thus, fetal outcomes are unknown. Generally, oral bowel preparations are not recommended and, if a full colonoscopy is necessary, tap water enemas are recommended (94). The indications for surgery—medically refractory severe colitis, obstruction, perforation, and intractable bleeding—are the same as in the nonpregnant IBD patient. Surgery for an acute indication in the setting of pregnancy carries a high risk of fetal loss (95), but case reports of deliveries of healthy infants following colectomy for fulminant colitis are reported (96,97). There is no evidence that therapeutic abortion improves the outcome of fulminant colitis, so it is not indicated. A team approach with early management and continuous assessment by the obstetrician, surgeon, and gastroenterologist is vital for the patient.

CONTROVERSIES

Although the medical management of fulminant colitis is similar to that for toxic megacolon, the absence of acute colonic dilatation may permit delay of surgical intervention. The timing of surgical intervention in these less urgent cases requires experienced clinical judgment as the delay of operative therapy may promote higher mortality due to protracted immunosuppression and malnourishment of the patient (8,89). Meanwhile, early intervention to reduce mortality must be balanced against the potential for intensive medical management to control the inflammatory process and complications, thereby potentially preventing the psychosocial and medical stigmata of colectomy. Generally, in the absence of colonic dilatation, medical management may be continued for 5 to 7 days in a further attempt to reverse transmural inflammation, as long as the patient is stable and improving. Patients with fulminant colitis who do not begin to respond to the intensive intravenous steroid regimen, as described above, should be referred to a center experienced in cyclosporin or anti-TNF therapy or undergo colectomy.

Key Points

Presentation
• Severe colitis = 6 or more bloody BMs, fever, tachycardia, anemia, elevated ESR
• Fulminant colitis = 10 or more bloody BMs, anemia requiring transfusion, abdominal distention
• Toxic megacolon = colon diameter more than 6 cm, tachycardia, fever, anemia, leukocytosis, dehydration, altered mental status, hypotension, electrolyte disturbance
• Most common causes are infection (C. difficile) and IBD

Diagnosis
• WBC, Hgb, albumin, potassium, sodium, ESR, CRP, stool culture, C. difficile testing
• Serial daily or BID plain radiograph versus CT
• Limited endoscopic evaluation (contraindicated for toxic megacolon) with biopsies to evaluate for CMV

Treatment and Outcome
• Team approach to treatment, including medical and surgical personnel
• Intravenous fluid and electrolyte resuscitation
• Blood transfusions to maintain hematocrit at about 30%
• Supplemental parenteral nutrition
• Bowel rest in the presence of vomiting, abdominal pain, or colonic dilatation
• Repositioning maneuvers (colonic dilatation)
• Specific treatments for infections
• Intravenous corticosteroids for inflammatory bowel disease
• Cyclosporin or infliximab in selected patients not responding to intravenous corticosteroids after 3 to 7 days
• Broad-spectrum antibiotics if toxic

Surgical Indications
• Failed medical therapy
• Progressive dilatation or toxicity
• Shock or multiorgan dysfunction
• Persistent hemorrhage
• Evidence of perforation

References