Toxic Megacolon Syndrome of Ulcerative Colitis *

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THE TERM toxic megacolon has been used to describe that syndrome seen in ulcerative colitis characterized by 1) a clinical picture of profound toxicity; 2) marked gaseous dilatation of the colon, and 3) vomiting and inability to eat due to the atonic dilated segment causing a paralytic type of obstruction. The diarrhea which is present does continue and is sometimes worse. This syndrome has been described in the primary fulminating type of ulcerative colitis; in the relapsing remitting type of disease; and in an acute exacerbation of the chronic type.[1,6] The mortality rate with this complication of ulcerative colitis is from 20 to 30 per cent with either medical or surgical treatment.[8]

Stenosis or stricture formation in the colon has not been a prominent feature of this syndrome.[2,5] Minute colonic perforations have been frequently described.[2] Histologically, the muscle layers of the colon are prominently involved with inflammatory and degenerative changes with apparent involvement of the myenteric plexus.[3,6]

The value of the plain roentgenogram of the abdomen in correctly assessing the clinical situation has been emphasized by several authors.[4,8] The possibility of colonic perforation from barium enema examination performed during this toxic dilatation has also been emphasized.[8]

The purpose of this communication is to report three cases of toxic megacolon associated with ulcerative colitis and to point out the usefulness of tube cecostomy in the initial management of this complication.

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Case Reports

D. D., a 32-year-old white woman, entered the U. S. Naval Hospital, Camp Lejeune, North Carolina, on August 26, 1959. She was first diagnosed and treated for ulcerative colitis in 1953. Her disease was controlled with intermittent steroid therapy until one month prior to admission, when the number of stools per day increased to ten. She lost 12 pounds and complained of intermittent nausea and vomiting. Her rheumatoid arthritis also became more symptomatic. Sigmoidoscopic examination revealed granular friable rectal mucosa. Ten days following admission, she became febrile with daily spikes of temperature as high as 40.4° C. She developed abdominal distension and bowel sounds were infrequent. She was unable to take oral feedings and vomited intermittently. Abdominal roentgenograms revealed marked gaseous distension of the colon (Fig. 1, 2). The hemoglobin was 10.5 Gm. per cent and the white blood cell count was 22,000 per cubic millimeter with a marked shift to the left. The serum protein was 4.2 Gm./100 cc. Increased doses of steroids failed to improve the patient's condition and on September 10, 1956, a subtotal colectomy and ileostomy was performed. There was fecal spillage at the time of operation and the patient died October 2, 1959, after a progressive slow deterioration from peritonitis with an ileal fistula. Examination of the colon revealed marked dilatation of the right and transverse colon and no evidence of stricture or stenosis. Microscopic examination revealed necrosis and infiltration of the muscle layers with lymphocytes and plasma cells. The mucosa was greatly ulcerated in the right and transverse colon.

W. L., a 19-year-old white man, was transferred to the U. S. Naval Hospital, Camp Lejeune, North Carolina, on September 3, 1960, with a history of rectal bleeding of nine months' duration and having had a high fever, vomiting, increased diarrhea and abdominal distension of two weeks' duration. The patient had been on increasingly large doses of steroids during the three months prior to admission. At the time of admission, the patient was toxic with a temperature of 39.7° C.

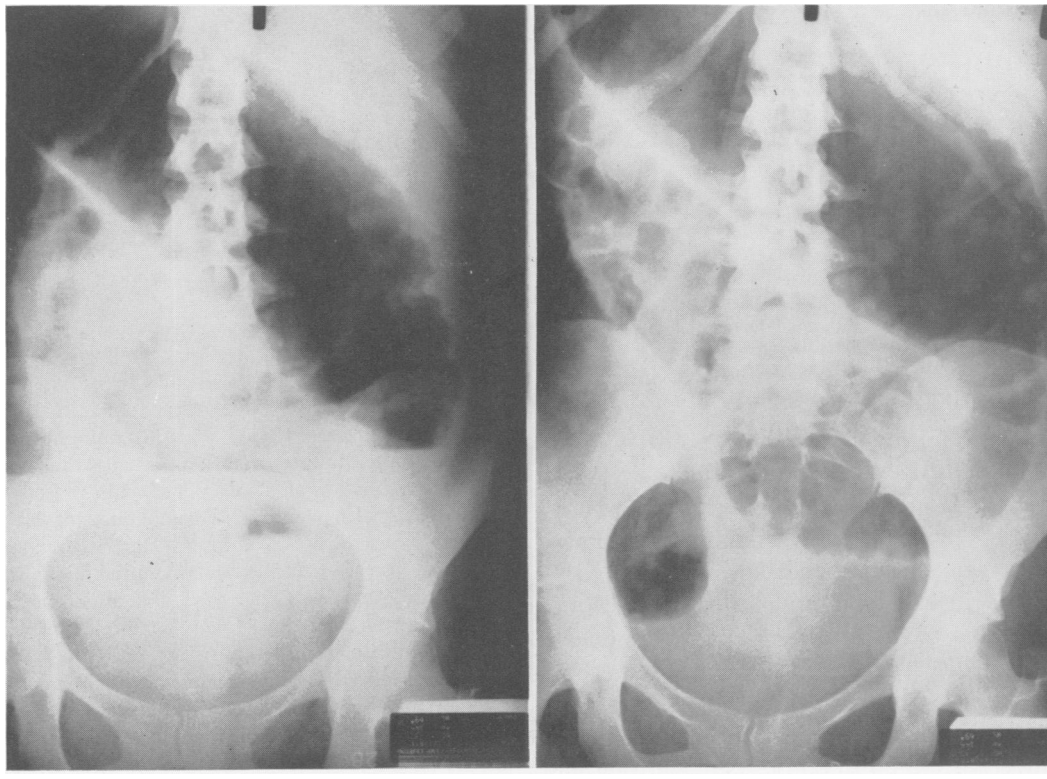

FIG. 1 (Left), FIG. 2 (Right). D. D. (Case 1).

and a pulse rate of 120 per minute. His abdomen was markedly distended with decreased peristalsis. Sigmoidoscopic examination showed friable granular rectal mucosa but no evidence of stricture. Abdominal roentgenograms (Fig. 3, 4), showed massive dilatation of the right and transverse colon. He was markedly hypo-proteinemic with a total protein of 4.7 Gm./100 cc. The hemoglobin was 11.4 Gm. per cent and the white blood cell count was 22,850 per cubic millimeter.

On the day following admission, a tube cecostomy was performed using a size 34F mushroom catheter. This was threaded to the level of the hepatic flexure and peritoneum was sutured to the cecum, prior to opening the cecum, in an effort to prevent peritoneal soiling. There was a marked clinical improvement following decompression of the dilated segment. Topical neomycin solution was given through the cecostomy tube. The patient began eating and there was gradual improvement in his nutritional status and one month later, a subtotal colectomy and ileostomy was performed. He tolerated this procedure well and his recovery was rapid and uneventful. Examination of the colon revealed no evidence of stricture. There were ulcers and polypoid hyperplasia seen grossly. Histologically, inflammatory and degenerative changes were seen in the muscle with evidence of minute perforations.

F. L., a 37-year-old white woman, was admitted to the U. S. Naval Hospital, Camp Lejeune, North Carolina, on January 15, 1961. She had a history of intermittent diarrhea of three years' duration. Multiple sigmoidoscopic and barium enema examinations had not revealed the cause of the patient's diarrhea. Two months prior to this hospitalization, she noted an increase in the number of stools to 12 per day. She had also noted a weight loss of 25 pounds. The day following admission, she developed a spiking temperature to 39.4° C. daily. She was unable to take oral nourishment and began vomiting. Her abdomen became distended. Sigmoidoscopic examination again was normal. Bowel sounds were hypoactive. The hemoglobin was 8.2 Gm. per cent, the white blood cell count 13,300 per cubic millimeter and the total serum protein was 4.4 Gm./100 cc. Abdominal roentgenograms (Fig. 5) revealed dilatation of the right, transverse and descending colon. The patient was considered for a trial of steroids but because of a large coexisting duodenal

ulcer, this was rejected. On January 25, 1961, a tube cecostomy utilizing a size 34F mushroom catheter was performed. There was marked clinical improvement with resumption of oral alimentation following this procedure. On March 6, 1961, a subtotal colectomy and ileostomy was performed. The rectal stump was preserved since it still remained free of disease. She made an uneventful recovery and resumed full activity. The colon revealed pseudo-polyps, multiple ulcerations, shortening and fibrosis down to and including a portion of the sigmoid colon. No stenotic or obstructive areas were found. Microscopic examination of the muscle layers revealed lympocytic and plasma cell infiltration with fibrosis.

Discussion

Definitive surgical therapy of ulcerative colitis requires an ileostomy with removal of the colon and rectum if the rectum is involved with the disease. Operation of this magnitude is best performed in the nontoxic patient whose nutritional status and blood volume are adequate. The technical aspects are much easier if the bowel is decompressed and cleansed.

In attempting definitive surgical treatment of ulcerative colitis during the toxic megacolon phase, one is faced with a hypoproteinemic, toxic patient whose blood volume is frequently diminished. The colon is massively distended and decompression and cleansing is impossible. Handling of the bowel at surgery is difficult or impossible without danger of massive peritoneal contamination.

With these factors in mind and with the fatal outcome of the first case presented, we resorted to the long abandoned procedure of tube cecostomy as a temporary expedient in order to more properly prepare the second and third cases with this problem. We have been impressed with the measure as an adjunct to treatment in this phase of ulcerative colitis. While not

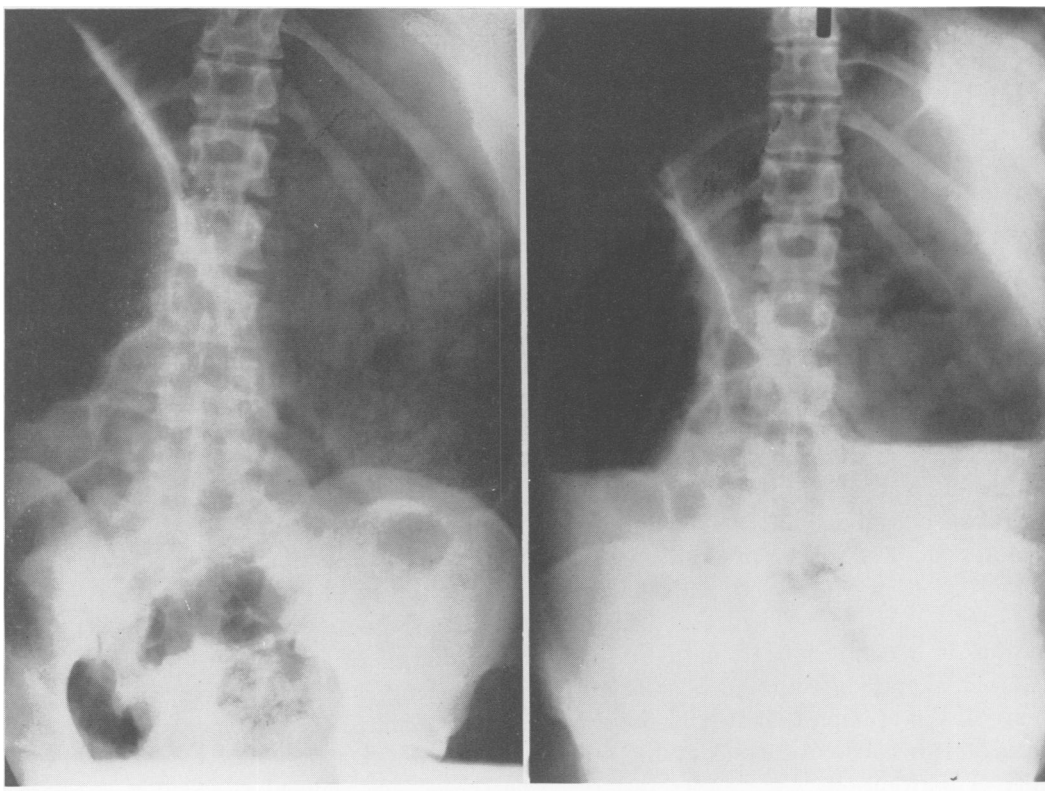

FIG. 3 (Left), FIG. 4 (Right). W. L. (Case 2).

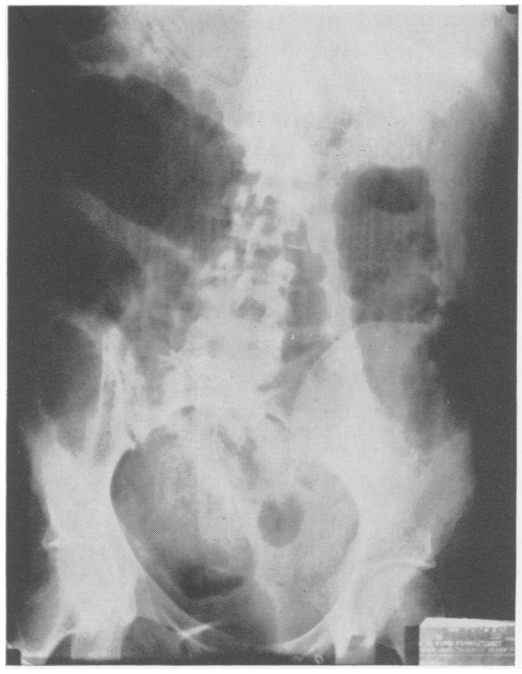

FIG. 5. F. L. (Case 3).

completely diverting the fecal stream, tube cecostomy allows for easy egress of gas and liquids and offers a pathway for topical application of antibiotic solutions. Decompression has been easily maintained in the two cases presented. It is pertinent, however, that large caliber tubes were used and they have been passed to the hepatic flexure with multiple openings throughout the intraluminal length.

It is interesting to speculate if steroid therapy may in some way be implicated in the etiology of this syndrome. However, our third case received no steroid therapy because of a coexisting large duodenal ulcer. Case one and two received increasing doses of steroids prior to operative decompression of the colon with no improvement. Turell[7] believes that colonic dilatation is a contraindication to the use of steroid therapy. Roth[6] and associates also are of the opinion that steroid therapy is hazardous because of increasing the possibility of perforation.

Lumb and associates[2] are apparently convinced that the changes of toxic megacolon are not completely reversible and because of this and the high incidence of colonic perforation they believe surgical therapy is indicated. Roth *et al.*,[6] however, have cases managed successfully by medical means alone and contend that the histological changes are reversible. They emphasize that operation should be avoided if at all possible and point to the rather high surgical mortality to support the contention.

We have considered this complication as good reason for definitive surgical treatment and have attempted to carry this out as a staged procedure and believe this is probably the treatment that will ultimately salvage the most patients. Sporadic reports of long-term remissions following cecostomy alone tempts one to remove the tube and follow the patients. Recurrence of toxic megacolon, however, in these already damaged colons is certainly quite high and argues strongly for definitive treatment.

The opportunity to examine the colon six and eight weeks following the acute stage of toxic megacolon was afforded by cases two and three and, although the microscopic changes are much less severe than case one, large numbers of chronic inflammatory cells persist in the muscle layers and certainly suggest that the histologic picture is not completely reversible.

In further support of Lumb's contention that perforation is frequently associated with this phase of ulcerative colitis, each of the three cases here presented had colonic omental adhesions which when separated at the time of definitive surgery resulted in fecal leakage.

Two of the cases presented showed maximal disease in the right, transverse and descending colon with minimal or no rectal involvement. This is somewhat at a variance with the previously reported cases. All, however, showed maximal gaseous dis-

tension in the transverse colon with less distension in the right and descending colon.

Summary

The toxic megacolon syndrome of ulcerative colitis has been reviewed and the pathogenesis described. Three such cases have been presented, two of which were treated by employing an initial tube cecostomy followed by colectomy as staged procedures. The uneventful course and the successful outcome leads us to believe that this approach offers the best opportunity to prepare the bowel for resection and to advance the patient to the best possible physiologic state prior to definitive operation.

Histologic examination of resected bowel six and eight weeks following relief of the toxic phase suggests that the tissue changes are not reversible.

The tendency toward perforation is noted and it is again emphasized that a plain film of the abdomen is sufficient roentgenographic evidence of this syndrome and barium enemas are contra-indicated.

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