Therapeutic and Diagnostic Colonoscopy in Nonobstructive Colonic Dilatation

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Cecal perforation has been well established as a consequence of mechanical obstruction of the distal colon and has been estimated to occur in 1.5% to 7% of patients with colon obstruction. Perforation of the cecum also occurs in cases of nonobstructive colonic dilatation (NCD). Although the incidence is unknown, the mortality rate is nearly 50%. Over an eight-year period, 44 patients (mean age 59 years) underwent 52 colonoscopic examinations for presumed NCD. Twelve patients (27%) developed NCD while convalescing from a recent operation and 29 patients (66%) had major systemic disorders that preceded the development of NCD. Medical treatment for an average of 2.6 days was uniformly unsuccessful. Mean cecal diameter prior to colonoscopy was 12.8 cm (range 9.5 to 17 cm). Based on radiographic or clinical criteria, 38 patients (86%) were successfully decompressed on the initial colonoscopic examination; mean cecal diameter decreased to 8.7 cm ($p < 0.01$). Perforation of the cecum during colonoscopy occurred in one patient (2%) who survived. Fourteen patients died; six deaths were attributed solely to the patient's underlying disease, and eight deaths occurred in patients who underwent operation. In summary, colonoscopy is a safe and effective therapeutic and diagnostic tool in cases of massive cecal dilatation. It should be considered before cecostomy in patients without radiographic evidence of pneumoperitoneum or clinical signs of peritoneal irritation.

ADYNAMIC ILEUS of the colon (Ogilvie's syndrome), pseudo-obstruction, and nonobstructive colonic dilatation (NCD) are all descriptive terms referring to a clinical entity in which the signs and symptoms of colonic obstruction are present without evidence of mechanical obstruction. An isolated segment, usually the cecum, or the entire length of the intra-abdominal colon may be effected. Chronic dilatation may result in acute symptomatology but rarely is life-threatening. Conversely, acute untreated NCD may result in cecal rupture with an attendant mortality rate of nearly 50%.[1] Decompressive tube cecostomy has been advocated by some but is not without risk; this procedure has been associated with a mortality rate of up to 20%.[2] In 1977 Kukora and Dent reported the successful endoscopic decompression of the colon in five patients with massive NCD using a flexible fiberoptic colonoscope.[3] More recently, Bernton described a colonoscopic technique to introduce and advance an intestinal tube through the anus into the cecum in order to treat recurrent cecal dilatation.[4]

This review examines the etiologies of NCD, determines the risk to the patients of developing significant complications, and evaluates the role of colonoscopic decompression in this condition.

Materials and Methods

The case records of 44 of patients in whom the diagnosis of NCD was made on clinical and radiographic grounds were reviewed for the eight-year period between 1974 and June 1982. Clinical and radiographic features, management, and outcome were assessed. All colonoscopic examinations were performed by or directly supervised by one of the authors. Comparisons among data were made and tested for statistical significance using the Student's t-test.

Results

Forty-four patients (31 men and 13 women) with a mean age of 59 years (range 25 to 89 years) underwent flexible fiberoptic colonoscopy for presumed NCD.

Twelve of the 44 patients (27%) developed NCD while convalescing from a recent operation. Twenty-nine patients (66%) had significant systemic illnesses that preceded the development of NCD (Table 1). Three additional patients were diagnosed as having acquired megacolon. Narcotic analgesics were implicated in 26 cases of NCD, but discontinuation of the drugs did not effect the clinical course in any of these cases. Serum electrolyte abnormalities (hypokalemia in seven patients, hy-

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pochloremia in one patient, and hypercalcemia in one patient) were noted in nine cases.

Abdominal distension was present in all patients, and in 26 cases palpation of the abdomen revealed diffuse tenderness. The distention developed gradually over an average time of 3.5 days. Bowel sounds were reported as normal, hyperactive, and hypoactive in 18, 6, and 20 patients, respectively. Despite increasing abdominal and colonic distension, 30 patients continued to pass watery stool that was negative for occult blood. The remaining 14 patients stopped passing flatus and stool. White blood cell count elevation ($>12{,}000/mm^3$) was observed in 26 cases and fever (38.5 C) was noted in nine cases.

Abdominal radiographs demonstated colonic dilatation in all cases. In 30 cases the dilatation was segmental, and in the remaining 14 cases the entire intra-abdominal colon was dilated. The line of gas demarcation occurred at the splenic flexure, the sigmoid colon and the hepatic flexure in 15, 8, and 7 cases, respectively. Air was present in the small bowel in 27 cases. Air-fluid levels in the colon were observed infrequently. Mean cecal diameter measured 12.8 cm (range 9.5 to 17 cm) in patients prior to colonoscopy. No significant difference in cecal diameter was noted in patients with or without small bowel distension.

Prior to colonoscopy, patients were treated uniformly with intravenous fluids and nasogastric tube decompression. In 22 cases, rectal tubes were inserted and gentle tap water enemas were administered. The mean interval between diagnosis of NCD and treatment with colonoscopy was 2.6 days. None of the patients exhibited clinical or radiologic improvement prior to colonoscopy. The interval between diagnosis and treatment was not significantly different for those patients undergoing successful *vs.* failed colonoscopic decompression.

Fifty-two colonoscopic examinations were performed on 44 patients. Thirty-six patients underwent a single colonoscopy and eight patients required a second colonoscopy. Successful decompression was defined as passage of the colonoscope to a point in the colon proximal to the sigmoid flexure and radiographic evidence of decreased cecal diameter following colonoscopy.

Colonoscopic decompression was successful initially in 32 patients (73%). The mean cecal diameter decreased significantly ($p < 0.01$) from 12.7 cm to 8.7 cm after colonoscopy. (Figs. 1 and 2). The signs and symptoms of abdominal distension resolved, and normal bowel function returned within two to five days following colonoscopy. Following initial successful colonoscopy, four of the 32 patients (13%) underwent abdominal operations for conditions unrelated to colon dilatation. Five patients developed recurrent dilatation within ten days of the initial procedure, and required a second endoscopic decompression. Four of these patients had a successful repeat decompression; however, one patient subsequently required operative treatment. A fifth patient underwent unsuccessful repeat colonoscopy and later required operation two weeks after the initial successful examination (Table 2).

Of the 12 patients with unsuccessful initial decompression, nine had radiographic evidence that cecal diameter was unchanged following colonoscopy (mean diameter precolonoscopy 13 cm and mean diameter postcolonoscopy 12.3 cm); but six patients were noted to have decreased abdominal distension to palpation and

TABLE 1. *Associated Disorders in 41 Patients with NCD*

Previous operation		12
Pelvic	7	
Abdominal	3	
Other	2	
Systemic Illnesses		47
CNS disorders	9	
Pneumonia	7	
MI or CHF	6	
Renal failure	5	
GI bleeding	5	
Metastatic cancer	5	
Liver failure	4	
Diabetes	2	
Sepsis	2	
Multiple trauma	2	

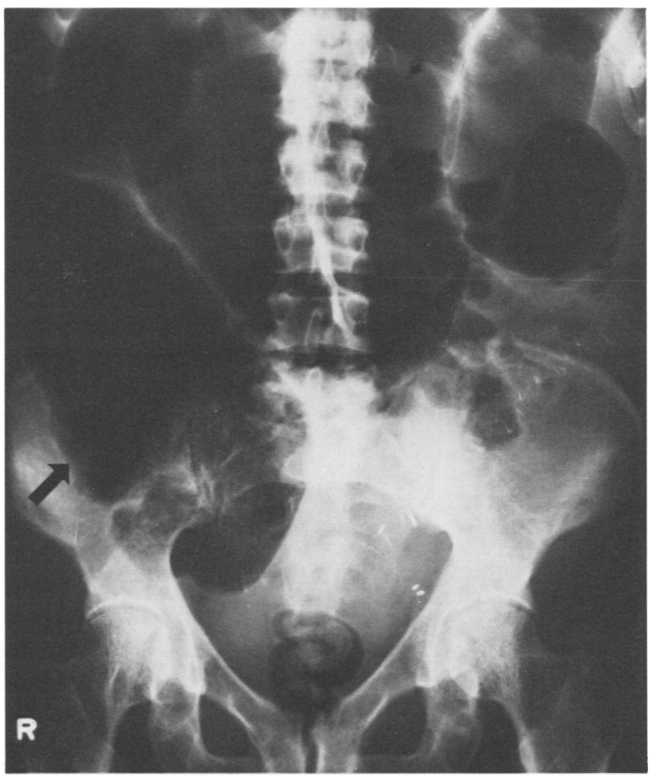

FIG. 1. Abdominal roentenogram demonstrating massive cecal dilatation (arrows) prior to colonoscopy.

FIG. 2. Abdominal roentenogram showing marked reduction in cecal diameter following successful colonoscopic decompression.

to measurement of abdominal girth. Three patients remained distended despite successful passage of the colonscope to the ascending colon, the hepatic flexure, and the splenic flexure in separate cases. One patient was noted to have necrotic mucosa in the sigmoid colon. The colonoscopy was terminated, and the patient was taken immediately to the operating room where a left colectomy was performed. In another case, solid fecal material was encountered and prevented passage of the colonscope. This patient underwent successful decompression the following day after cleansing enemas had been administered. A third critically ill patient sustained a cardiac arrest during colonoscopy, but was successfully resuscitated. Three patients with initially unsuccessful decompression required repeat colonoscopy for persistent symptoms, and in two cases the decompression was successful. The remaining patient underwent tube cecostomy one day following colonoscopy. Three other patients with unsuccessful initial decompressions became asymptomatic, resolved their colonic dilatation, and had return of normal gastrointestinal function without operation or repeat colonoscopy. Following an initial unsuccessful decompression, six patients underwent operation for conditions related to colon dilatation (Table 2).

TABLE 2. *Operations Performed in 13 Patients*

Initially Successful Decompression (N = 32)			
Rescope			Operation
Successful	Unsuccessful		Sigmoid colostomy (D)
Subtotal colectomy (D)	Cecostomy (S)		Ileostomy (S)
			Exploratory laparotomy (D)
			Cecostomy & enterolysis (S)
Initially Unsuccessful Decompression (N = 12)			
Rescope			Operation
Successful	Unsuccessful		Left colectomy (D)
	Cecostomy (D)		Ileocolectomy (D)
			Cecectomy (S)
			Exteriorazation of cecum (S)
			Cecostomy, 2 (D) (D)

(D) = died; (S) = survived.

Fourteen patients (32%) in this series died. Six deaths occurred in patients undergoing initially unsuccessful decompressions, and eight occurred in patients with successful initial decompression (Table 3). Only one death (2%) was related to a perforation of the cecum. Six deaths were attributed solely to the patient's underlying disease and eight deaths occurred in patients who underwent operation (three in the initially successful group and five in the unsuccessful group) (Table 2). Autopsies were performed in all patients who had not undergone operation and in all cases the colon appeared "normal."

Discussion

The underlying causes of adynamic ileus of the colon are unknown. Ogilvie speculated that the sympathetic nervous supply to the colon was inhibited while others have theorized that an imbalance between the parasympathetic and sympathetic innervation may cause a loss of normal spike and motor activity of the colon, especially in response to distension.[5,6] The etiology of this presumed neuronal imbalance also remains unclear. A variety of conditions have been reported in patients with NCD and appear to precede the clinical development of colonic distension. These include extra abdominal trauma (including surgery), congestive heart failure, pancreatitis, sepsis, renal failure, and electrolyte imbalance.[7,8] These processes are nonspecific and have also been associated with adynamic ileus of the small intestine.

Cecal perforation or rupture has been well established as a consequence of mechanical obstruction of the distal colon and has been estimated to occur in 1.5 to 7% of patients with colon obstruction.[9,10] Perforation of the cecum also occurs in cases of NCD, but the incidence is unknown and the mortality rate is high.[1,2] Most of the experience with cecal perforation in cases of NCD has developed from single case reports or small series. In

1973 Wojtalik reported four cases of NCD with cecal necrosis or perforation; three deaths occurred in this group.[1] Nearly 80% of previously reported cases of NCD underwent laparotomy with a preoperative diagnosis of mechanical obstruction and nearly 75% of the patients who underwent operation had decompression of the colon performed, usually by tube cecostomy. The mortality rate for this procedure was nearly 40 to 45%.

Heschl in 1880 first described the stages of progressive colonic distention to rupture in segments of bowel with distal obstruction.[11] He noted that splitting of the taenia occurred first followed by tears through the outer and the inner muscular layers and finally by perforation of the mucosa. These observations concerned only the effects of tension defined as force per unit length tangential to the bowel wall tending to create and distract a longitudinal split in the serosa of the colon wall. The tension developed equals the product of the radius of the bowel lumen and the transmural or the intraluminal pressure. As the bowel fills with air, elongation also occurs. Kozarek evaluated closed segments of sigmoid, descending, transverse, and ascending colon as well as cecum in human cadavers.[12] He observed that the pressure required to cause perforation ranged from 120 mmHg in the cecum to 202 mmHg in the sigmoid colon. Integrity of the bowel wall is also a function of wall viability. Luminal distension has been shown to result in progressive mural ischemia across the wall. The mucosa along the antimesenteric border is the most susceptable. In animal studies, VanZwalenburg observed that increasing luminal pressure from 30 to 130 mmHg caused gradual cessation of capillary, venous, and finally arterial circulation in the bowel wall. However, similar correlations in human beings have not been established.[13]

Lowman later attempted to define a group of patients, based on radiographic determination of cecal size, who appeared to be at risk for developing cecal perforation.[14] Controls consisted of patients who had undergone barium contrast studies of the colon. In each case, the greatest cecal diameter achieved during distension (not produced in a controlled fashion) with barium and air was measured on a prone film. The size of the distended cecum ranged from 3 to 10.5 cm. In 97% of cases, the cecal diameter was less than 9 cm. Thus, 9 cm was empirically determined to be the upper limit of normal for cecal diameter. This figure was then used for comparison with cases in which distension was caused by distal obstruction. It was hoped that this diameter would predict impending perforation of the cecum. The size of the cecum noted in 19 cases of distension secondary to obstruction ranged from 9.0 to 16.3 cm (mean 11.2 cm). Of these patients, 37% were noted to have cecal perforations (mean cecal diameter of 10.9 cm) at the time of operation. Wangensteen has estimated experimentally that the intraluminal pressure necessary to produce cecal perforation is 26 cm H_2O but clinical studies to date have not demonstrated any correlation between intraluminal pressure, wall tension, and luminal diameter with the likelihood or incidence of cecal perforation.[15] In the past it has been presumed that the risk of cecal perforation in cases of NCD is similar to that observed in cases of mechanical obstruction and that an aggressive approach should be taken in cases of massive dilatation (>12 cm). In the authors' experience, however, perforation occurred in only two of 44 cases (4.5%), and in both cases the patients underwent successful operation and survived. Impending cecal perforation was indentified in three cases (mean cecal diameter 13.5 cm) by colonoscopic visualization of mucosal necrosis or ulceration. In each instance, the patient underwent urgent operation before perforation had occurred.

TABLE 3. *Causes of Death in 14 Patients Undergoing Colonoscopic Decompression for NCD*

Successful Decompression (N = 8)	
Sepsis	2
Pneumonia	2
Liver failure	1
Stroke	1
Pulmonary embolus	1
Small bowel infarction	1
Unsuccessful Decompression (N = 6)	
Sepsis	3
Hyperkalemia & renal failure	1
Liver failure	1
Cecal perforation	1

The clinical features of NCD are not different from those observed in mechanical obstruction of the colon. The patient is usually greater than 50 years of age and more likely to be a male. The predominant clinical feature is abdominal distension that develops gradually over three to four days. The patient may complain of nausea and vomiting. Concurrently, the patient frequently stops passing flatus and stool. The distended abdomen may be soft to palpation but generally is tense. Bowel sounds are variably present and, if present, may be hyperactive or hypoactive. Abdominal tenderness, slight elevations of WBC, and temperature elevation without bowel perforation were additional findings regularly observed in this series.

Plain abdominal radiographs may be useful to localize the segment of colonic dilatation but cannot be used alone to exclude mechanical causes of obstruction. Cecal diameter can be determined easily and the presence or absence of pneumoperitoneum noted. An abrupt cut-off in the gas pattern may occur at any level in the colon. Air-fluid levels in the colon occur infrequently and the bowel wall usually appears thin.[2,16] The differential diagnosis includes dilatation secondary to mesenteric ischemia as well as mechanical obstruction due to volvulus or neoplasm. Distension of the small bowel coexistant with large bowel distension does not necessarily indicate an incompetent ileocecal valve and retrograde decompression of the colon. The distension may be due to air swallowing or peritoneal irritation. It is not valid to assume that patients with this finding are at less risk for developing cecal perforation or rupture.[14]

In uncomplicated cases initial management (for cecal diameter < 9 cm) has included conservative measures, such as correction of fluid and electrolyte abnormalities, nasogastric decompression to prevent further entry of air into the intestine, cessation of narcotic administration, and treatment of the associated systemic conditions.[2,17,18] Operative tube cecostomy has been advocated when the degree of dilatation is massive (>12 cm).[1,2] A variety of drugs have been used to promote intestinal peristalsis without success.[19-21] Endoscopically placed rectal tubes may be useful if pancolonic dilatation or isolated sigmoid dilatation is present. Cleansing enemas can be administered through the rectal tube to dislodge solid stool prior to colonoscopy. If a nonoperative approach is employed, serial abdominal radiographs should be obtained frequently to assess absolute cecal size as well as incremental changes in cecal size and the abdomen should be examined serially for signs of peritoneal irritation.

Medical management is usually begun when the cecum exceeds 9 cm in diameter. In several series, nonoperative and nonendoscopic treatment has resulted in resolution of NCD over two to 14 days.[17,18,22] In these series, however, the colon was not massively dilated (>12 cm). In the authors' experience, medical management was employed for an average of 2.6 days without success, as determined by cecal size and clinical symptoms. In addition, the mean cecal diameter observed in these patients was 12.8 cm, leading to speculation that the risk of perforation or mural necrosis was probably too high to continue conservative treatment.

The concept of nonoperative decompression for cecal dilatation was initiated by Euphrat who suggested maneuvering a Seldinger catheter under fluoroscopic control past areas of kinking and spasm in the colon.[23] Ghazi and others have reported the successful reduction of a sigmoid volvulus using the colonoscope.[24] Since the authors initial report of the use of colonoscopy in the treatment of NCD, others have also successfully employed this technique.[17]

Colonoscopy did not significantly delay operation in any patient and was useful for identifying impending perforation in three patients. Perforation of the cecum was attributed directly to colonoscopy in only one patient who successfully underwent operation and ultimately survived. The frequency of colon perforation during diagnostic colonoscopy has been reported to range between 0.2% and 2%.[25] Many of these perforations occur in the rectosigmoid and appear to be related to direct pressure of the endoscope on the wall. Mean and maximal intraluminal pressures achieved during diagnostic colonoscopy had been determined to be less than 25 mmHg and 100 mmHg, respectively. Colonoscopy in case of NCD is tedious and must be performed by an experienced, perseverant endoscopist. The average time for the procedure in this series was 45 minutes. For obvious reasons air insufflation must be kept to a minimum and usually is not necessary. Frequent irrigation with small volumes (50 cc) of saline through the suction channel of the scope is necessary to maintain visibility and channel patency. It is not always necessary to reach the cecum with the colonoscope in order to effect successful colonic decompression, especially if the colon is dilated beyond the hepatic flexure. In 19 of the 32 cases (59%) of successful endoscopic decompression, the scope was passed to the level of the cecum. In 11 other cases successful decompression was performed by passage of the scope to the level of the hepatic flexure only, and in two remaining cases in which the entire intra-abdominal was dilated, the scope was introduced only to the level of the splenic flexure. Upon withdrawal of the endoscope, intermittent suction should be applied until the colonic lumen collapses. The colonscope is removed slowly, in 4- to 5-cm increments, and the tip of the scope is kept in the center of the lumen to permit decompression of gas and liquid stool through the suction channel and to prevent trapping of the mucosa in the suction tip. Frequent irrigation is usually necessary to maintain visibility. Passage of the instrument into the ascending colon ensures that mechanical obstruction is not the cause of the colonic dilatation. In most cases, the bowel mucosa can be adequately visualized with colonic irrigation to determine the presence of mucosal necrosis or ulceration. Barium enema examination is not necessary when colonoscopy is performed and may be dangerous in cases of suspected NCD. Decompressive colonoscopy can be performed at the bedside if necessary or in an endoscopy suite.

The success of colonic decompression is usually based on radiographic evidence of decreased cecal diameter. In nine of the patients in whom cecal size did not decrease following colonoscopy, six were noted clinically to have decreased abdominal distension. It appears that the intraluminal pressure and thus the wall tension were reduced without actually changing cecal diameter. The bowel may also decrease in length before a change in diameter is observed. Even after tube cecostomy, it is not uncommon for cecal diameter to remain unchanged for four to five days. Based on clinical criteria alone, the success rate using colonoscopic decompression for NCD was 86%.

In summary, the authors continue to feel that colonoscopy is a relatively safe and effective therapeutic and diagnostic tool in patients with NCD. Nasogastric suction and rectal tube decompression are generally not successful in patients with cecal diameters greater than 9 cm. Although the risk of cecal perforation is unknown in patients with NCD, it appears that if the cecal diameter exceeds 12 cm, colonoscopy should be considered as an urgent form of management. If an experienced colonoscopist is not available, tube cecostomy should be performed. Patients who are not successfully decompressed by colonoscopy should also undergo tube cecostomy.

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