Acute bloody diarrhea with right sided colitis

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ABSTRACT: A 50-year-old woman presented with bloody diarrhea and radiological evidence of right sided colitis. Enterohemorrhagic *Escherichia coli* was isolated from the stool. The illness subsided spontaneously but was complicated by the development of hypoproteinemia and ascites. This disease presents a variable clinical picture but apparent 'ischemic colitis' in younger patients is especially suggestive of infection with *E coli* 0157:H7. **Can J Gastroenterol 1988; 2(1):37-40**

Key Words: *Colitis, Escherichia coli 0157:H7*

A 50-YEAR-OLD WOMAN WAS SEEN in the emergency department with abdominal pain and bloody diarrhea.

Six days earlier she had awoken with severe right lower quadrant abdominal cramps. Several hours later watery diarrhea developed with bowel movements every 1 to 2 h. Within 24 h the stools became grossly bloody. At this time she became nauseated and vomited several times.

Stool specimens for culture had been sent by her family doctor shortly after the illness began. These were reported as negative for pathogens.

Three days prior to the onset of symptoms, two family members had been acutely ill with watery diarrhea. Their illnesses resolved spontaneously. Stool cultures were not obtained.

There was no history of recent travel outside Canada. The patient had not had any animal contact, had not eaten any unusual foods and had not received antibiotics in several years.

Her previous history was unremarkable. Specifically, there was no history of liver disease or excessive alcohol intake. She was not receiving any medications.

On examination she appeared ill and was mildly dehydrated. Temperature was 36.8° C, pulse 115/min and regular and blood pressure 170/100 mmHg. Examination of the head and neck revealed no abnormalities. The chest was clear and heart sounds normal. Abdominal examination revealed no areas of tenderness, liver and spleen were not enlarged. Bowel sounds were normal but an epigastric bruit was heard. There were no stigmata of liver disease and foot pulses were normal.

LABORATORY INVESTIGATIONS

Hemoglobin was 183 g/L, hematocrit 0.53 and white cell count 20,500/mm^3 with 63% neutrophils, 28% stabs, 7% lymphocytes and 2% monocytes. Serum sodium was 130 mmol/L (normal, 135 to 145 mmol/L) and potassium 3.0 mmol/L (3.5 to 5.0

mmol/L). Serum chloride was 93 mmol/L (98 to 110 mmol/L) and bicarbonate 25.2 mmol/L (22 to 32 mmol/L). Blood urea was 4.8 mmol/L (3.0 to 6.5 mmol/L) and creatinine 72 μmol/L (40 to 130 μmol/L). Serum bilirubin was 4.8 μmol/L (less than 18 μmol/L), serum aspartate aminotransferase (AST) was 17 iu/L (less than 38 iu/L), alanine aminotransferase (ALT) was 17 iu/L (less than 38 iu/L) and alkaline phosphatase was 74 iu/L (40 to 120 iu/L). Serum albumin was 27 g/L (35 to 50 g/L). Urinalysis showed a trace of protein and no red cells or casts. Repeat stool cultures showed no growth of pathogens.

Sigmoidoscopy revealed edematous colonic mucosa without ulceration.

Following admission the patient appeared to deteriorate. She had frequent bloody bowel movements and developed abdominal distension. Because of the possibility of ischemic colitis, a barium enema was performed (Figure 1, left panel) and this showed marked irregularity of the ascending and proximal transverse colon with thumb printing. This was consistent with the diagnosis of ischemic disease. The rest of the colon and the terminal ileum were within normal limits.

Six days after admission *Escherichia coli* 0157:H7 positive for Verotoxin was isolated from the patient's stool.

The patient was treated with intravenous fluids and appropriate corrections were made to her electrolytes. The diarrhea settled spontaneously. On the 10th day of her illness the white cell count was normal. By the 12th day her bowel frequency decreased to two bloodless formed stools per day. At this point she was noted clinically to have ascites with a fluid thrill.

At this time serum albumin was 31 g/L (normal 35 to 50 g/L) and urinalysis was negative for protein. Serum bilirubin was 5 μmol/L, AST was 26 iu/L and ALT was 36 iu/L. The prothrombin time was 13 s (12 to 15 s) and partial thromboplastin time was 28 s (25 to 37 s). The ascites quickly resolved and because the patient was clinically well further investigations were not performed.
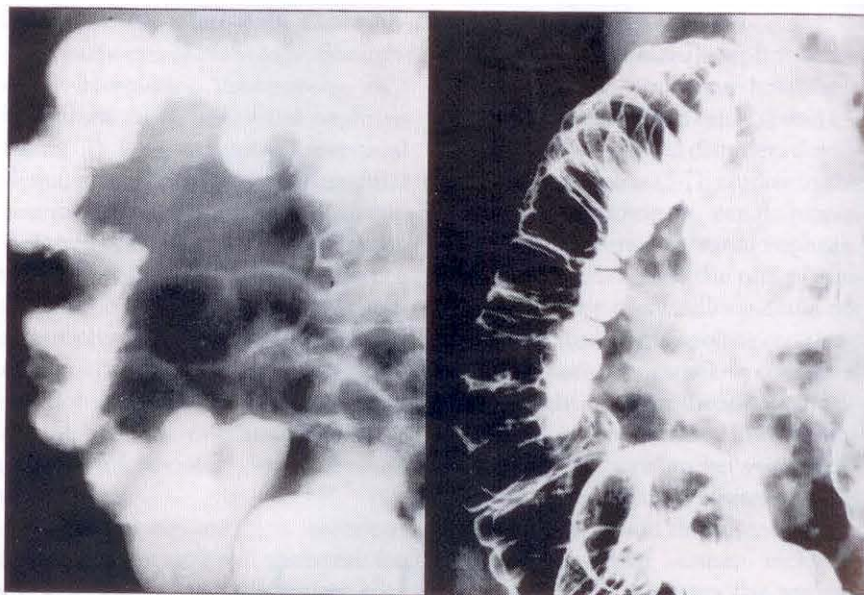
She was discharged on the 16th day

Figure 1) *Barium enema, showing right side of the colon, on ninth day of illness (left hand panel) and eight weeks later (right hand panel)*

of her illness at which time her bowel movements had returned to normal.

Eight weeks later she was asymptomatic without ascites. Serum albumin was 49 g/L. Repeat barium enema was entirely within normal limits (Figure 1, right panel).

DISCUSSION

A 50-year-old woman presented with bloody diarrhea in the absence of fever or systemic illness. The acute onset of illness with abdominal pain, watery diarrhea and later bloody diarrhea was suggestive of an infectious etiology. Two family members had already been ill. Stool cultures, however, showed no growth of the common enteric pathogens. Colonic ischemia was considered and the finding of an epigastric bruit supported this diagnosis.

Sigmoidoscopic examination showed edematous mucosa but no specific abnormalities. Barium enema showed changes in the right colon compatible with ischemic disease.

Until relatively recently the diagnosis would not have been elucidated further. The isolation of enterohemorrhagic *E coli* from the repeat stool cultures and the recognition that this organism may cause right sided colitis enabled the correct diagnosis to be made.

The recognition of *E coli* as pathogenic is attributable to Bray and Beavan (1). In the 1940s, in England, they recognized that only a fraction of childhood diarrheas were explicable on the basis of known pathogens. They postulated that *E coli* might, in some circumstances, be capable of producing diarrhea. Rabbit antiserum was prepared to *E coli* cultured from the stool of a child with diarrhea. Using the antiserum they were able to demonstrate the presence of the same organism in subsequent outbreaks of diarrhea as compared to a control population without the illness. Thus they provided evidence of the pathogenicity of certain *E coli* strains.

Since that time there has been an enormous increase in our knowledge of pathogenic *E coli*. The organism may be classified on the basis of the O (lipopolysaccharide), H (flagellar) and K (polysaccharide) antigens. Subsequent assignment to enteropathogenic, entertoxigenic, enteroinvasive, enteroadherent or enterohemorrhagic groups is clinically of little benefit because pathogenesis is not yet clear and because the organism does not always produce disease appropriate to its classification. The affected family members in the present case likely had enterohemorrhagic *E coli* but yet had a

watery diarrhea without gross blood. The ability of enterohemorrhagic *E coli* to produce watery diarrhea has been reported previously (2).

The 0157:H7 strain of *E coli* was first recognized in Irish piglets in 1970 (3) as a cause of enteritis. Since then, sporadic and clustered cases have been reported. Outbreaks have been described in homes for the aged (4) and in a day care centre (5).

The disease is characterized by an incubation period of three to nine days with a median of four days (4). The organism may be ingested in contaminated food. Hamburger from a restaurant chain was responsible for an outbreak in the United States (6). Infection via fecal oral contamination and via fomites likely occurs (5). Typically the infection begins with crampy abdominal pain followed by watery diarrhea which may become grossly bloody. There may be nausea and vomiting but fever is uncommon. Some patients have symptoms suggestive of an upper respiratory infection (6).

Laboratory evidence of inflammation may be absent. The erythrocyte sedimentation rate and white cell count may be normal or only minimally elevated (6).

Sigmoidoscopic examination may be normal or show abnormalities ranging from edema to frank ulceration (2). Colonoscopically the disease may be most marked in the proximal colon and edema, inflammation and hemorrhage have been reported (2). Barium enema may be normal or show, as in this case, changes suggestive of submucosal edema (2,6).

The disease appears to show a predilection for the right colon and may mimic ischemic disease. It seems likely in milder cases, or in cases where the patient is seen some time after the onset of the illness, that no significant lesion may be seen endoscopically or radiologically. The disease appears to resolve without residual colonic scarring.

The illness is usually self-limited with a mean duration of eight days (2) but in the elderly and very young it may be fatal. A mortality rate of 36% was reported for an outbreak in London, Ontario in 1985 (4). There is an association with hemolytic uremic syndrome a complication which carries a high mortality (2,5,7).

The organism may be found in the stools for up to 14 days (8) but is more likely to be detected if stools are collected within the first six days of illness (9).

Treatment consists of fluid and electrolyte replacement. Trimethoprim-sulfamethoxazole or ampicillin may be effective in shortening the duration and severity of illness (10). There are, however, no controlled trials of antibiotic efficacy. In the retrospective data presented by Remis and colleagues (2) the mean duration of illness in patients receiving antibiotics was 7.5 days as compared to 8.5 days in untreated patients. Of particular importance would be a trial designed to show whether antimicrobial therapy prevented the development of disease and hemolytic uremic syndrome in contacts.

The organism produces a toxin identical to that of *Shigella dysenteriae* type 1 (11). This toxin, designated Verotoxin 1, is cytotoxic for HeLa and Vero cells (a cell line derived from monkey kidney cells). A second cytotoxin, Verotoxin 2, not neutralized by antibody to the *Shiga* toxin may also be found. Some strains produce fimbriae which may be an important factor in determining virulence (12).

Verotoxin has been shown to cause platelet aggregation (13) and this may be important in the pathogenesis of the hemolytic uremic syndrome. *E coli* 0157:H7 is not the only enteric pathogen associated with hemolytic uremic syndrome. This association has also been reported for *Salmonella*, *Shigella* and *Campylobacter jejuni*.

In the present case the disease followed a relatively benign course. There was no evidence of hemolysis and renal function remained normal. The disease subsided with supportive therapy.

The case presented here is unusual as ascites has not previously been reported as a complication of the illness. The serum albumin was low possibly secondary to protein loss from the inflamed bowel. The low serum oncotic pressure due to hypoalbuminemia no doubt contributed to the ascites but does not seem sufficent to explain it. There was no clinical or laboratory evidence of hepatic or pancreatic disease and no evidence of peritonitis.

When seen in follow-up eight weeks after the onset of the illness the patient was well with a normal serum albumin and no evidence of ascites. Repeat barium enema showed resolution of the colonic abnormalities.

E coli 0157:H7 presents to the gastroenterologist in a variety of ways; as watery diarrhea, bloody diarrhea, colonic bleeding with no apparent cause or as apparent ischemic colitis. The reports of right sided 'ischemic colitis' in young adults (14) reported in the literature prior to recognition of this organism may be descriptions of enterohemorrhagic *E coli* colitis.

Microbiological laboratories should screen for this organism in all cases of bloody diarrhea when no other pathogen is found. Initial screening is based on the absent or slow fermentation of sorbitol by this strain of *E coli*. Over 90% of other strains of *E coli* ferment sorbitol. If a nonfermenting strain is found it can be serotyped to determine if it is enterohemorrhagic *E coli*.

The gastroenterologist should consider *E coli* 0157:H7 as a cause of infectious or apparent 'ischemic' colitis when cultures are negative for the common pathogens. This is especially important if barium enema or colonoscopy shows disease predominantly affecting the proximal colon.

UNE FEMME DE 50 ANS S'EST PRESENTÉE AVEC UNE DIARRHÉE SANGLAUTE et, à l'examen baryté, une colite du côté droit. Les coprocultures ont revélé la présence d'une souche de *E coli* enterohemorrhagique. Il y eu une amélioration de la maladie cependant elle fut compliquée d'hypoproteinémie et d'ascite. La présentation clinique de cette maladie est variable mais chez les jeunes la découverte d'une 'colite ischémique' suggère fortment une infection causée par *E coli* 0157:H7.

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