CASE REPORT

Non specific jejunoileitis – a report of 8 cases

ANAND ALLADI, KANISHKA DAS, KARUNA V.*, ASHLEY J D'Cruz

Departments of Pediatric Surgery and Pathology, St John's Medical College Hospital, Bangalore 560 034

Nonspecific jejunoileitis is a nonocclusive, necrotizing inflammation of the small intestine. We treated 8 patients of jejunoileitis in a short span of 8 months. Their mean age was 8.6 years. All had acute pain in abdomen and most had hematochezia. Radiology was helpful only in diagnosis of complications of the disease. Four patients responded to conservative management; the other 4 required surgery – laparotomy and lavage in 2; and multiple laparotomies with resections in 2. One patient died due to chronic malnutrition and metabolic complications. Bowel histology was suggestive of resolving vasculitis in one patient and chronic inflammation in another patient. [Indian J Gastroenterol 2001;20:195-196]

Key words: Necrotizing enteritis, intestinal vasculitis, small intestine

Nonspecific jejunoileitis or necrotizing enteritis is a nonocclusive necrotizing inflammation predominantly involving the small intestine; its etiology and pathogenesis are not known. Clinically it is characterized by severe symptoms that are out of proportion to signs. It is self-limiting and usually responds to bowel rest and antibiotics. The more severe forms often require multiple operations, either in the early period or at a later date to manage complications like fistulae, strictures and adhesive obstruction.

We report eight patients with varying severity of disease in order to highlight the clinical spectrum of the disease and re-examine the approach to treatment.

Case Reports

From January to August 1999, eight patients (aged 4-13 years, mean 8.6; 7 boys) with necrotizing enteritis were admitted to the Department of Pediatric Surgery. We analyzed the case records of these children.

The duration of symptoms ranged from 4 to 7 days. The main presenting features were abdominal pain in all, and vomiting; frank blood in stool and fever in six. Three patients had moderate dehydration on admission. All had either hematochezia or occult blood in stool. There was no history of having any specific food preceding the illness, except in one who had had a non-vegetarian community meal.

Blood culture and stool microscopy were negative for pathogens in all cases. Peritoneal fluid culture obtained at surgery in one patient grew Enterobacter species. Abdominal radiographs were non contributory in three patients, showed thickened bowel loops in 2, and fixed loop in one. Contrast study, which was done in two patients at a later stage, revealed a stricture in one and spiculations of the mucosa in the other.

Four patients responded to conservative management in the form of bowel rest, IV fluids and antibiotics (ampicillin 100 mg/kg/day, q6h; gentamicin 5 mg/kg/day and metronidazole 7.5 mg/kg/dose, q8h). The other four were operated upon. Two patients underwent laparotomy and peritoneal lavage. One patient underwent laparotomy, re-look laparotomy after 48 hours and a third surgery for resection of post-inflammatory stricture.

The fourth patient, who was referred after a laparotomy and diagnosis of necrotizing enteritis, was explored to drain an intra-abdominal abscess and for peritoneal lavage. He was re-explored later and found to have multiple dense adhesions and sealed perforations. Resection of 100 cm of bowel was needed. Post-operatively he was started on peripheral parenteral nutrition. The patient developed ventricular arrhythmias and convulsions, which on echocardiography and CT scan of the brain were found to be due to fungal balls in the left ventricle and fungal embolus in the brain, respectively. He was started on amphotericin-B and responded in 15 days with total disappearance of the lesions. On day 60, he developed chicken pox, which was treated with acyclovir. He continued to have recurrent chest and GI infections and finally died of chronic malnutrition and metabolic complications. Serology for HIV was negative.

Microscopic examination of resected specimens revealed features suggestive of resolving vasculitis in one patient and chronic inflammation in another (Figs 1, 2)

Discussion

Necrotizing enteritis has been known for many years by many different names. In spite of large numbers reported, little is known about its etiopathogenesis. Various theories have been put forward and include Clostridium welchi type F infection, Schwartzmann phenomenon secondary to Gram-negative sepsisemia, and type III hypersensitivity reaction to intestinal pathogenesis, but none has been proved beyond doubt.

Blood and peritoneal fluid cultures in our patients
were sterile except for one who grew Enterobacter species. Histology showing edema of the submucosa and infiltration by eosinophils and mononuclear cells in the vicinity of necrotic tissue is supportive of type I reaction; whereas polymorphonuclear infiltration of perivascular tissue with evidence of vasculitis is compatible with type III reaction. Based on these microscopic features, we suggest that the necrotizing enteritis was initiated by microvascular factors/ischemia compounded by hypersensitivity reaction type I or III.

Of the 8 patients seen by us, 6 had mild disease and recovered completely. Two patients who recovered finally required prolonged bowel rest (35nd and 45 days), parental nutrition and multiple operations. One patient died due to severe nutritional depletion leading to immune suppression and multiple infections.

In conclusion, our observations reaffirm the need for a bowel-conserving approach in necrotizing enteritis, using second- or third-look laparotomy if needed to address the intra-abdominal complications. Parenteral nutritional support may assist in recovery. Secondary infections with fungi and viruses can be successfully managed.

References

Correspondence to: Dr D'Cruz, Professor and Head. E-mail: denz@venl.com
Received October 20, 2000. Received in final revised form January 22, 2001. Accepted April 13, 2001