Massive Bowel Ischemia - An Exceptional Case

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Abstract: Necrotizing enterocolitis (NEC) is an acquired, multifactorial syndrome characterized by segmental, inflammatory, or ischemic necrosis of the small and large bowel. Although in adults the incidence of NEC is much lower and has lower mortality, the degree of morbidity remains high. As AMI may lead to fatal intestinal gangrene early diagnosis and treatment are paramount.

Keywords: necrotizing enterocolitis, massive bowel ischemia, intestinal gangrene, acute mesenteric ischemia.

INTRODUCTION

Necrotizing enterocolitis (NEC) is an acquired, multifactorial syndrome characterized by segmental, inflammatory, or ischemic necrosis of the small and large bowel (Fick, K. A., & Wolken, A. P. 1949). It primarily affects premature neonates of low birth weight. The incidence varies from 1 to 3 per thousand live births. Reported mortality rates are between 20% and 50% (Henry, M. C., & Moss, R. L. (2005, August). The term adult necrotizing enterocolitis (ANEC) is used to describe similar patterns of bowel necrosis in adults and is commonly known as Drambrand and Pigbel. ANEC is a rare disease and only small numbers of cases are reported in literature (Katara, A. N. ET AL., 2004).

Although AMI accounts for only about 1%–2% of gastrointestinal illnesses, the incidence has been increasing considerably (Corder, A.P., & Taylor, I. 1993; & Bjorck, M. ET AL., 1997).

Case Report

A 45 year male of average built presented to the emergency department with complaints of distention of abdomen vomiting and constipation for 3 days he was also having history of fever off and on, he was a known i.v drug abuser. On examination, he was dehydrated, tachycardia was present blood pressure was normal. On abdominal examination abdomen was distended, generalized tenderness was present. There was no masking of liver dullness. Bowel sounds were absent.

The patient was admitted, resuscitated, and investigations were sent. Total leucocyte count was raised, serum creatinine was also raised. X-ray abdomen showed multiple dilated small bowel loops. There was no free gas under the diaphragm on erect chest x-ray. Usg abdomen suggested mild free fluid in the abdomen with dilated small bowel loops having no peristalsis.

The patient was subjected to exploratory laparotomy under general anesthesia. On exploration, mild clear free fluid was present in the peritoneal cavity. The whole small bowel was dusky in color with multiple gangrenous patches and filmy thin wall (figure 1). The condition of ascending and transverse colon was also similar to the small bowel (figure 2). The stomach, duodenum and the descending, sigmoid colon and rectum was normal. Aortic pulsations were normal however; there was no pulsation in the small bowel mesenteric vessels.

Since this was a case of massive bowel ischemia no further intervention was done as the resection would lead to complete loss of small bowel. The abdomen was temporarily closed with a drain. The
The patient could not be extubated and was shifted to ICU where he expired within 24 hours.

**DISCUSSION**

Necrotizing enterocolitis (NEC) is a disease of small and large bowel that primarily affects premature neonates with low birth weight. The incidence of NEC ranges from 0.3 to 3 per thousand live births but is approximately 10% in infants weighing less than 1500g (Chung, D. H. ET AL., 2001; & Huang, F. S., & Warner, B. W. 2001) Although in adults the incidence of NEC is much lower and has lower mortality, the degree of morbidity remains high.

NEC in adults is common in the developing countries and its etiology is multifactorial. Infectious agents, inflammatory mediators and circulatory disturbance have all are included in the etiology and pathogenesis of NEC. Epidemic outbreaks of NEC point to the role of infectious agents, especially when the same organism is isolated from stool, blood and peritoneal fluid. The common organisms implicated are bacteria like Klebsiella, E. Coli, Enterobacter, Pseudomonas, Clostridia and Staphylococcus epidermidis, viruses like Corona virus, Rota virus and Entero virus and rarely, fungi like Candida albicans (Kulkarni, A., & Vigneswaran, R. 2001).

Classification of intestinal ischemia proposed by the American Gastroenterological Association in 2000 (Brandt, L. J., & Boley, S. J. 2000).

**Acute mesenteric ischemia**

a) Major arterial occlusion  
b) Minor arterial occlusion  
c) Major embolus  
d) Mesenteric venous thrombosis  
e) Splanchnic vasoconstriction (nonocclusive mesenteric ischemia)

**Chronic mesenteric ischemia or intestinal angina**

**Ischemic colitis**

As AMI may lead to fatal intestinal gangrene early diagnosis and treatment are paramount. A high index of suspicion in the setting of a compatible history and physical examination serves as the cornerstone to early diagnose mesenteric ischemia (Stoney, R. J., & Cunningham, C. G. 1993; Kaleya, R. N., & Boley, S. J. 1992; & Mansour, M. A. 1999).

The findings on a plain abdominal radiograph in AMI are also nonspecific (Stoney, R. J., & Cunningham, C. G. 1993; Hall, J. B. et al., 1998; Lund, E. C. et al., 1988; Smerud, M. J. et al., 1990; & Wolf, E. L. et al., 1992). In the early stage of the disease, 25% of patients may have normal findings on abdominal radiography.

In a case of suspected AMI with no clinical indication of exploratory laparotomy, mesenteric angiography remains only investigation of choice. Early angiography has been shown to improve survival rates (Boley, S. J. et al., 1981; Schuler, J.J.1995; & Grace, P. A. et al., 1993). Also, angiography coupled with the plain abdominal radiograph reveals the cause of abdominal pain in 25% to 40% of patients who do not have mesenteric ischemia.

Treatment remains largely supportive and consists of bowel rest, gastrointestinal decompression, fluid resuscitation and antibiotic therapy. Some patients may require correction of hypotension, respiratory dysfunction, anemia, coagulation disorders or acid-base imbalance. Surgical intervention is necessary if there is intestinal necrosis or frank perforation or when there is clinical deterioration over 12–24 hours despite intensive medical support, as evidenced by persistent or worsening metabolic acidosis, increasing ventilatory support requirement, deteriorating hematological parameters and persistent thrombocytopenia.

In our case, small bowel extending from duodenojejunal junction till ileocolic junction and ascending and transverse colon was gangrenous which is supplied by superior mesenteric artery. Also, the pulsations of the mesenteric vessels were absent indicating the possibility of superior mesenteric artery occlusion (AMI). So the resection of the affected bowel would not have been compatible with life and so was not done. The only option was bowel transplantation, the facility for which was not available at our center and the patient’s attendent was also not ready for such procedure. Thus, we closed the abdomen with drain in situ after adequate counselling of the patient’s attendents but the patient died in ICU within 24 hours.
REFERENCES