NECROTIZING ENTEROCOLITIS, A RARE BUT SEVERE CONDITION WITH INSIDIOUS POSTOPERATIVE COMPLICATIONS

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Necrotizing enterocolitis, a rare but severe condition with insidious postoperative complications:

Abstract: Necrotizing enterocolitis (NEC) is one of the most frequent causes of gastrointestinal perforation in premature neonates, only few case series and reports being described in adult patients. Early in the course of the disease, superficial mucosal ulceration, sub mucosal edema and hemorrhage occur. Further progression leads to transmural necrosis leading sometimes to bowel perforation. Six cases encountered in our clinic in recent years led us to resume discussions on necrotizing enteritis, not because it is a rare disease, but due to the severe postoperative complications.

Material and methods: Our lot consisted of four stage 1 patients and two with Bell stage III NEC and severe intestinal injury, necrosis, and perforation. All of the patients were diagnosed preoperatory with other surgical conditions, like appendicitis with peritonitis, perforated duodenal ulcer or acute cholecystitis.

Results: We present to review two cases. For patients undergoing laparotomy, resection of the involved intestine mandates either enterostomy formation or primary anastomosis. An intermediate option is laparotomy with intestinal resection and delayed anastomosis 48 to 72 hours later. Because of the small number of patients in our lot, we cannot advise a certain surgical treatment, but a strategy involving bienterostomy per primam should be further analyzed. The choice of operative intervention reflects multiple variables, including age, physiologic status, institutional resources and surgeon preference based on experience. Primary peritoneal drainage for perforated NEC may help to resuscitate and treat a critically ill patient initially, and in some instances, may be definitive operative intervention.

Conclusions: Relatively rare disease, of unknown etiology and elusive pathogenesis, NEC has initial non-specific symptoms and clinical features that mimic more common surgical diseases. There is considerable controversy regarding which procedure is preferable. Currently, in the absence of rigorous evidence supporting the superiority of one approach over the other, surgical intervention depends mostly on the treating institution or the individual surgeon. Keywords: ENTEROCOLITIS, LAPAROTOMY, PERFORATION.

Necrotizing enterocolitis (NEC) is one of the most frequent causes of gastrointestinal perforation in premature neonates, only few case series and reports being described in adult patients. Relatively rare disease, of unknown etiology and elusive pathogenesis, has initial nonspecific symptoms and clinical features that mimic more common surgical diseases.

Due to the multifactorial nature of the
disease and limitations in disease models, NEC remains, above all, a problem of diagnosis and treatment, being accompanied by severe complications in evolution.

It is characterized by diffuse ulceration and necrosis of the distal small bowel and the colon, leading to perforation of the bowel, followed by high morbidity and mortality.

Although the exact etiology and pathogenesis of NEC remains elusive, it is well established that several bacterial and viral species have been associated with outbreaks of NEC (Clostridium spp., Klebsiella spp., Staphylococcus epidermidis, Escherichia coli, Rotavirus) (1). No single pathogen has been identified as causative and the ability of the microflora to colonize the epithelium and to ferment unabsorbed nutrients may be more important than the strain itself (2, 3).

Perforation seems to be caused by enteral acute ischemia due to quickly installed obstruction of the parietal microcirculation, strictly limited to the intestinal wall, without mesenteric involvement (4).

The ileum and proximal colon are the most commonly affected sites in NEC although any segment of the gastrointestinal tract can be involved including the stomach. Severity of bowel wall necrosis ranges from a small localized mucosal necrosis of a segment to transmural necrosis of the entire small intestine and colon in most severe cases (5).

In more advanced stages of NEC, pathological findings include gastrointestinal bleeding, inflammation, bacterial overgrowth, intestinal distension with multiple dilated loops of small bowel, pneumatosis-intestinalis, intestinal perforation, necrosis, hypotension, septic shock, pneumoperitoneum, and intra-abdominal fluid (6).

Six cases encountered in our clinic in recent years led us to resume discussions on necrotizing enteritis, not because it is a rare disease, but due to the severe postoperative complications (fig. 1 a, b).

**CASE REPORTS**

**First case**, a man (MI aged 39 years) was admitted to the emergency room with the diagnosis of duodenal penetrating ulcer and pancreatitis. Admission status is marked by pallor and hypotension, severe
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abdominal pain in a patient with grade II-III obesity.

First 12 hours of evolution are followed by shock, upper abdominal pain exacerbation and signs of peritoneal irritation requiring intervention (cholecystectomy for acute cholecystitis).

Due to multiple hydroaeric levels on Rx and bloating, reintervention was required for early postoperative ileus.

Fig. 2 a, b. Enterectomy with termino-terminal anastomosis

Intraoperative: ecchymotic, thin-walled intestinal loops and multiple mesenteric lymph nodes. A jejunal segment of about 30 cm of necrosis and multiple perforations, blocked by the great omentum were discovered (fig. 2 a, b).

Starting at 6 days after reoperation follows a series of other 4 reinterventions imposed by anastomotic fistulas or perforations distant to the anastomosis.

The sixth intervention unveils both anastomotic failure and necrosis of afferent intestinal loop, requiring bientoenterostomy with Pezzer and Folley catheterization of intestinal ends. Enteral nutrition is used for the next 40 days, until complete extinction of peritonitis phenomena is achieved. A new re-reintervention with reintegretion and anastomosis results in slow, favorable outcome with complete healing.

Second case also has special clinical features. Patient D.L. aged 15 years, was admitted for symptoms of acute appendicitis. Surgical intervention reveals acute catarrhal appendicitis for which appendectomy and drainage is performed. From the 5th postoperative day symptoms worsen and diffuse abdominal pain, bloating, lack of intestinal transit and fever require reintercation under the suspicion of peritonitis by cecal fistula. Laparotomy unveils generalized peritonitis with micro perforations of ileum in a 40 cm area, for which enterectomy and subsequent termino-terminal anastomosis are performed.

At the 11-day postoperative relaparotomy uncovers anastomosis failure and, in addition, an area of necrosis on jejunal loop, compelling right hemicolecctomy and cuneiform resection of jejunal necrosis. After a couple of days, a new jejunal fistula required enterectomy and reanastomosis.

DISCUSSION

The main factors responsible for the development of NEC are intestinal ischemia
and bacterial infection. Although the etiology still remains unclear, the common organisms implicated are bacteria like Klebsiella spp., E. coli, Enterobacter spp., Pseudomonas, Clostridium spp., and Staphylococcus epidermidis. It occurs most often in the areas of the bowel, where the blood flow from the major mesenteric arteries overlaps (7).

Early in the course of the disease, superficial mucosal ulceration, sub mucosal edema and hemorrhage occur. Further progression leads to transmural necrosis leading sometimes to bowel perforation. The clinical presentation include; abdominal distension, bilious vomiting and bloody diarrhea (5).

NEC can be classified clinical and radiological into 3 stages, as proposed by Bell and later modified by Walsh and Kliegman (5).

Stage 1 consists of non-specific clinical and radiological signs (fever, abdominal distension, dilated loops on X-ray and bowel thickening). Despite the presence of intestinal necrosis and perforation, pneumoperitoneum may not be observed. Stage 2 is characterized by blood in the stools, metabolic acidosis and classical signs of intestinal pneumatosis on X-ray. In stage 3, patients present with hypotension and shock, disseminated intravascular coagulation, occasionally peritonitis and sepsis.

Our lot consisted of four stage 1 patients and two with Bell stage III NEC and severe intestinal injury, necrosis, and perforation. All of the patients were diagnosed preoperatory with other surgical conditions, like appendicitis with peritonitis, perforated duodenal ulcer or acute cholecystitis.

Traditional surgical management of intestinal necrosis relies on the principles of controlling the source of peritoneal contamination by laparotomy, exploration of the intra-abdominal viscera, and excision of the involved, necrotic intestine.

Management options include: aggressive perioperative resuscitation, often including inotropic support; expedient and minimal operative behavior with attempts at limiting physiologic insult; evaluation of the entire intestine with resection of only necrotic or perforated bowel; enterostomy proximal to intestine of questionable viability; and preservation of the ileoceleal valve if possible (8).

The choice of operative intervention reflects multiple variables, including age, physiologic status, institutional resources and surgeon preference based on experience.

For patients undergoing laparotomy, resection of the involved intestine mandates either enterostomy formation or primary anastomosis. An intermediate option is laparotomy with intestinal resection and delayed anastomosis 48 to 72 hours later.

Finally, primary peritoneal drainage for perforated NEC may help to resuscitate and treat a critically ill patient initially, and in some instances, may be definitive operative intervention.

There is considerable controversy regarding which procedure is preferable. Currently, in the absence of rigorous evidence supporting the superiority of one approach over the other, surgical intervention depends mostly on the treating institution or the individual surgeon.

These are studies that suggest that the type of operation performed for perforated necrotizing enterocolitis does not influence survival or other clinically important early outcomes in preterm infants (8).

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terostomy per primam should be further analyzed.

Necrotizing enterocolitis is a severe inflammatory disorder of the intestine and a major cause of death and morbidity. In contrast to medicine improvement during the past 30 years, the mortality rate of 30 to 50 percent for patients with intestinal perforation due to necrotizing enterocolitis remains essentially unchanged (9).

REFERENCES

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