Necrotizing enterocolitis

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Necrotizing enterocolitis' (NEC) is a serious, life-threatening disorder in the adaptation of the digestive system of a newborn (mostly premature) to extrauterine life. It is a postnatally acquired acute disease characterized by hemorrhagic-necrotizing, ulcerating inflammation of the intestine. It typically affects the terminal ileum, cecum, and other parts of the colon. It is a sudden abdominal event with the risk of intestinal perforation and the development of peritonitis. The incidence of this disease is increasing, especially in developed countries thanks to advanced healthcare and increased survival of premature newborns.[1] NEC most commonly affects premature infants after enteral feeding has been initiated. It only affects full-term newborns in about 10% of cases. The incidence is estimated to be 6–10% NNPH below 1500 g.

The mortality rate of NEC is 10-30%.[2]

Newborns with NEC can be roughly divided into 3 groups:

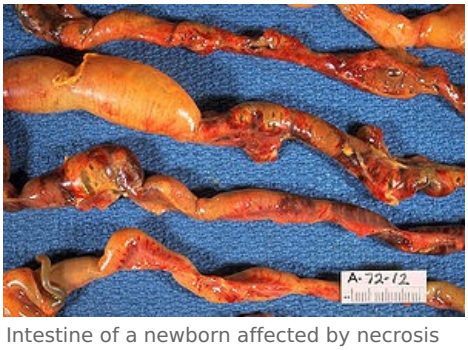
- a full-term newborn - there is almost always a significant risk factor for intestinal ischemia, typically perinatal asphyxia, or newborns with Hirschsprung's disease; NEC often appears in the first days of life;
- premature newborn (< 30 weeks of gestation) - usually has no other risk factor except prematurity;
- slightly/moderately premature newborn (30th to 36th week of gestation) - usually has a combination of risk factors: prematurity + perinatal asphyxia or intrauterine growth restriction;
  - in preterm infants, NEC typically develops in the 2nd to 3rd week of life, after enteral feeding is initiated.[3]

Risk Factors

- Prematurity and low birth weight (the intestinal mucosa is more permeable to microorganisms and toxins),
- intrauterine growth restriction/retardation (chronic fetal hypoxia, redistribution of cardiac output with a decrease in flow in the superior mesenteric artery),
- premature outflow of amniotic fluid, abruption of the placenta,
- perinatal asphyxia, low Apgar score,
- umbilical artery catheterization (intestinal vascular damage), exchange transfusion through umbilical catheters for hemolytic disease,
- infection, pathogenic bacteria,
- hypoxia and shock,
- hypothermia,
- ductus arteriosus patens (during PDA the flow decreases in the superior mesenteric artery; retrograde flow in diastole damages the vascular supply of the intestine), cyanotic heart defect,
- anemia, polycythemia (hyperviscosity syndrome), thrombocytosis,
- artificial nutrition, hyperosmolar nutrition, rapid increase in enteral nutrition doses, excessive fluid intake.[1][3]

Etiopathogenesis

The following are likely involved in the development of NEC:



Intestine of a newborn affected by necrosis and pneumatosis. Autopsy.

- **hypoxia**,
- **immaturity/prematurity**,
- **poor integrity of the intestinal mucosa** - impaired barrier function allows absorption of macromolecules and translocation of bacteria, disrupts digestion;
  - intestine of premature infants has an immature microvillous membrane with a relative lack of mucus and secretory IgA, with increased permeability of small and large molecules; further damage to the intestinal mucosa then increases the permeability of microorganisms and toxins; this process can start NEC;
  - antenatal administration of corticosteroids (induction of lung maturity) induces intestinal maturation, reduces its permeability and protects against NEC;
- **bacterial flora** - NEC is not an infectious disease, but the presence of bacteria (along with other risk factors)

is probably necessary for its development;
    - blood culture is positive in 10-50% and the finding usually correlates with stool or peritoneal fluid culture; the most frequently present are: *Escherichia coli, Klebsiella, Enterobacter* and *coagulase-negative staphylococci*;
    - sometimes NEC occurs in epidemics, then clostridia (*C. difficile, C. butyricum, C. perfringens,* further *Klebsiella spp., non-pathogenic Escherichia coli, enterotoxic E. coli, Salmonella spp., Pseudomonas aeruginosa, Rotavirus, Coronavirus*);
- **the presence of a metabolic substrate (milk) in the intestinal lumen**;
    - milk in the intestines supports the proliferation of bacteria and the production of endotoxins by Gram-negative bacteria; the bacteria then form gas and short-chain fatty acids from the unabsorbed nutrients, which can have a toxic effect on the intestinal epithelium; long-chain fatty acids and undigested casein further contribute to mucosal inflammation and damage; milk fats increase the permeability of the mucous membrane; in premature newborns, intestinal motility and digestive ability are insufficiently developed; however, excessively delaying the initiation of enteral nutrition is not recommended;
    - trophic feeding, i.e. administration of minimal enteral nutrition with parenteral nutrition, does not increase the incidence of NEC, on the contrary, it can protect against the development of NEC;
    - in newborns fed breast milk, the incidence of NEC is 7-10 times lower than in formula-fed babies;
    - the method of introduction of enteral nutrition affects the incidence of NEC.[3]

NEC can affect any part of the digestive tract. It most often affects the terminal ileum, cecum and ascending colon. This is a **transmural *disability. A typical laparotomy, histological and radiological sign is*** *pneumatosis'* or the presence of gas in the submucosa and subserosa of the intestinal wall. This gas is mainly nitrogen and hydrogen produced by bacteria. At laparotomy, the bowel is pink and discolored, often distended with areas of damaged mucosa. The first histological symptom is "coagulative necrosis of the mucosa" with the formation of microthrombi, which leads to irregular ulceration of the mucosa, swelling and bleeding. **cytokines** play an important role in the development of inflammation and intestinal damage in NEC. Levels of interleukins-1, -3, and -6, tumor necrosis factor-α (TNF-α), and platelet-activating factor (PAF) correlate with disease severity. A better understanding of the roles of individual cytokines in the future offers the potential for prophylaxis and treatment of NEC.[3]

The final stage is transmural necrosis – intestinal gangrene with perforation and peritonitis.[1]

Clinical picture

The clinical picture is very variable - from inconspicuous deterioration with non-specific signs such as lethargy, temperature instability and apneic pauses to the rapid development of shock with peritonitis and death. Children who do not have gastrointestinal symptoms (flatulence, biliary vomiting, blood in stool) have the appearance of sepsis: they are pale, marbled, and often jaundiced.[3]

Diagnosis

- Risk group of newborns, if they have impaired tolerance to enteral nutrition, a distended tummy and/or blood in the stool,
- blood count: leukocytosis/leukopenia, thrombocytopenia,
- ABR: metabolic and respiratory acidosis,
- may be present: hyperkalemia (from disintegrated erythrocytes), hypoalbuminemia (with effusion in the abdominal cavity),
- aerobic and anaerobic blood culture,
- blood in stool,
- X-ray of the abdomen (dilation of intestinal loops, ileus, uneven distribution of gas in the intestine, pneumoperitoneum) - clear evidence when gas is found in the wall of the intestine (pneumatosis intestini) and in the course of the vena portae.[1]

Modified Bell criteria[4]				
	Stage	**System symptoms**	**Abdominal symptoms**	**X-ray findings**
Suspicious NEC	**IA**	temperature instability, apnea, bradycardia, lethargy	gastric residue, abdominal distention, vomiting, stool positive for occult bleeding	normal finding or mild dilatation of intestinal loops, ileus
	IB		macroscopically bloody stool	
Proven NEC	**II**		disappearance of bowel sounds, palpation sensitivity ±	pneumatosis intestinalis
	IIB	+ mild metabolic acidosis and thrombocytopenia	clear palpable tenderness, there may be signs of phlegmon on the skin of the abdomen or a palpable abdominal mass in the right lower quadrant	gas in v. portae, ± ascites
Developed by NEC	**IIIA**	hypotension, bradycardia, respiratory failure, combined respiratory and metabolic acidosis, DIC, neutropenia	generalized peritonitis, marked pain and distension of the abdomen	clear ascites
	IIIB			pneumoperitoneum

Treatment

- Discontinuation of enteral nutrition,
- introduction of a nasogastric tube (bowel decompression),
- monitoring of vital functions, monitoring of fluid intake and output, blood pressure and diuresis,
- monitoring of the internal environment, blood count, ions,
- blood culture collection, urine K+C, sputum,
- ATB treatment (e.g. combination of ampicillin, gentamicin, metronidazole),
- regular surgical consultation,
- with the development of respiratory distress and a high position of the diaphragm, intubation and UPV.[1]

In case of failure of the conservative procedure or perforation of the intestine, surgical revision is indicated.

Complications

A complication of NEC with extensive bowel resection is **short bowel syndrome** (SBS, ``short bowel syndrome*) and subsequent **intestinal failure,* when in which the small intestine is not able to adequately absorb fluids, electrolytes and nutrients necessary for growth and development (i.e. impaired absorptive function despite the sufficient size of the intestine). Nutrition must be administered parenterally.**[5][6]

A metabolic complication of long-term parenteral nutrition is ``intestinal failure associated liver disease *(IFALD), characterized by direct hyperbilirubinemia, increased levels of transaminases, and subsequently impaired liver synthetic function (prolonged prothrombin time and INR).*[5]

Infectious complications of parenteral nutrition include catheter-associated bloodstream infection (CABSI).

DANCE

TANEC/TRAGI (*Transfusion-associated Necrotizing Enterocolitis*, *Transfusion-associated Gut Injury*) is a necrotizing enterocolitis that occurs within 48 hours after a blood transfusion.

Links

Related Articles

- Blood in newborn's stool
- Short bowel syndrome (pediatrics)
- Neonatal sepsis

External links

- V. Fridrichová: DANCE (case report) (https://www.fnbrno.cz/tragi-fridrichova/f2968)

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