

CLINICAL AND MORPHOLOGICAL PICTURE OF INTESTINE PERFORATION IN
NECROTIC ENTEROCOLITIS

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Annotation: Reasons and morphogenesis of gastro-intestinal perforations in newborns are introduced in this article. Clinical and morphological distinctions between perforated NEK and isolated gastro-intestinal perforations are discussed. Morphogenesis of gastro-intestinal perforations in newborns is a basis for prescribe correct medical tactic and choice of surgical method.

Key words: newborns, intestinal perforations, necrotizing enterocolitis, morphology.

NEKROTİK ENTEROKOLITA ICHAK PERFORATSIYASINING
KLINIK- MORFOLOGIK KO'RINISHI

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Annotatsiya: Maqolada nekrotik enterokolit (NEK) bilan tug'ilgan chaqaloqlarda ichak perforatsiyasining sabablari va morfogenezi yoritilgan. Nekrotik enterokolitdagi ichak perforatsiyalari va me'da ichak yo'llaridagi spontan perforatsiyalar o'rtasida klinik va morfologik farqlar mavjudligiga e'tibor qaratiladi. NEK bilan tug'ilgan chaqaloqlarda ichak perforatsiyasining morfogenezi va uning to'g'ri davolash taktikasini belgilash hamda jarrohlik amaliyotini turini aniqlash uchun asos bo'lib xizmat qiladi.

Kalit so'zlari: yangi tug'ilgan chaqaloq, chala tug'ilgan chaqaloqlar, nekrotik enterokolit, xavf omillari, morfologiya.

КЛИНИКО-МОРФОЛОГИЧЕСКАЯ КАРТИНА ПЕРФОРАЦИИ КИШЕЧНИКА ПРИ
НЕКРОТИЧЕСКОМ ЭНТЕРОКОЛИТЕ

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Аннотация: В статье освещаются причины и морфогенез перфораций кишечника у новорожденных детей с некротическим энтероколитом (НЭК). Обращает внимание на то, что между перфоративными перфорациями при некротическом энтероколите и спонтанными перфорациями имеются клинические и морфологические отличия. Морфогенез перфораций у новорожденных с НЭК является основой для назначения правильной лечебной тактики и определения вида хирургического вмешательства.

Ключевые слова: новорожденный, недоношенные дети, некротический энтероколит, факторы риска, морфология.

Relevance: Necrotizing enterocolitis (NEK, ICD-10 code - R77) is a severe disease of infancy, which is characterized by inflammation of the intestinal wall and its subsequent necrosis [1, 2, 7].

Currently, the world has accumulated extensive experience in the treatment of newborns with peritonitis associated with intestinal perforation of various etiologies [1, 3]. However, despite certain achievements in the study of intestinal perforation in newborns with NEK, this problem

cannot be considered fully studied. This applies, in particular, to the issues of etiopathogenesis and morphogenesis of the development of this pathology, which is highly likely to lead to negative consequences due to the occurrence of life-threatening complications [5].

Thelander in 1939 described 16 cases of gastric perforation, 30 cases of duodenal perforation, and 39 cases of small and large bowel perforation in infants with necrotizing enterocolitis (NEC). The first case of successful treatment of an infant born with ileal perforation at 35 weeks of gestation was described by Agerty in 1943 [2].

The advances and successes of modern neonatology and neonatal surgery have increased the survival rate of such infants, but the problem is further complicated by the fact that the total number of colonic perforations in infants born with NEC, especially against the background of tissue dysplasia, has a clear upward trend [4, 5, 7]. Along with the increase in intestinal perforations caused by NEC, there is an increase in the number of spontaneous perforations of the small and large bowel [5, 6, 7].

In modern pediatric surgery, spontaneous perforation in newborns is considered by many researchers as an independent nosological entity [4, 5, 7]. However, some authors reject this idea and explain the mechanism of intestinal perforation by an excessively high level of cavity pressure, which leads to mechanical rupture of the wall of the hollow organ [6].

Thus, despite the long history of the study of NEC in newborns and the presence of special publications on this topic, the literature does not contain generally accepted views on the etiopathogenesis and morphogenesis of intestinal perforation and terminology for this pathology. This fact confirms the need to study intestinal perforation in newborns with NEC not only according to the clinical manifestations of pathology, but also based on the data of histological studies.

According to the literature, perforations associated with NEC account for approximately 53% of all perforations of the gastrointestinal tract (GIT), and spontaneous perforations account for 27%. However, many mechanisms of the pathological process in NEC and spontaneous perforation of the gastrointestinal tract in newborns remain unstudied, which indicates the lack of a generally accepted concept of the pathogenesis of these conditions to date [1, 4, 8], therefore, determining the nature of the developmental morphogenesis of this pathology remains a priority in solving this problem.

This will allow the introduction into clinical practice of pathogenetic treatment methods that ensure a positive outcome of the disease in infants born with intestinal perforation.

Research material and methods. During 2020-2024, 59 newborns with intestinal perforations of various etiologies were under our observation. Children were divided into 2 groups:

Group 1 - 39 (66.10%) patients with perforating NEC;

Group 2 - 20 (33.90%) infants born with spontaneous perforation.

The object of morphological research was operational and section materials. Perforations had different macroscopic appearances. Spontaneous perforation was in the form of limited perforation of the stomach or intestinal wall with a local pathological process. Perforating intestine associated with NEC had extensive inflammatory and necrotic lesions involving large areas of all layers of the intestinal wall in the pathological process.

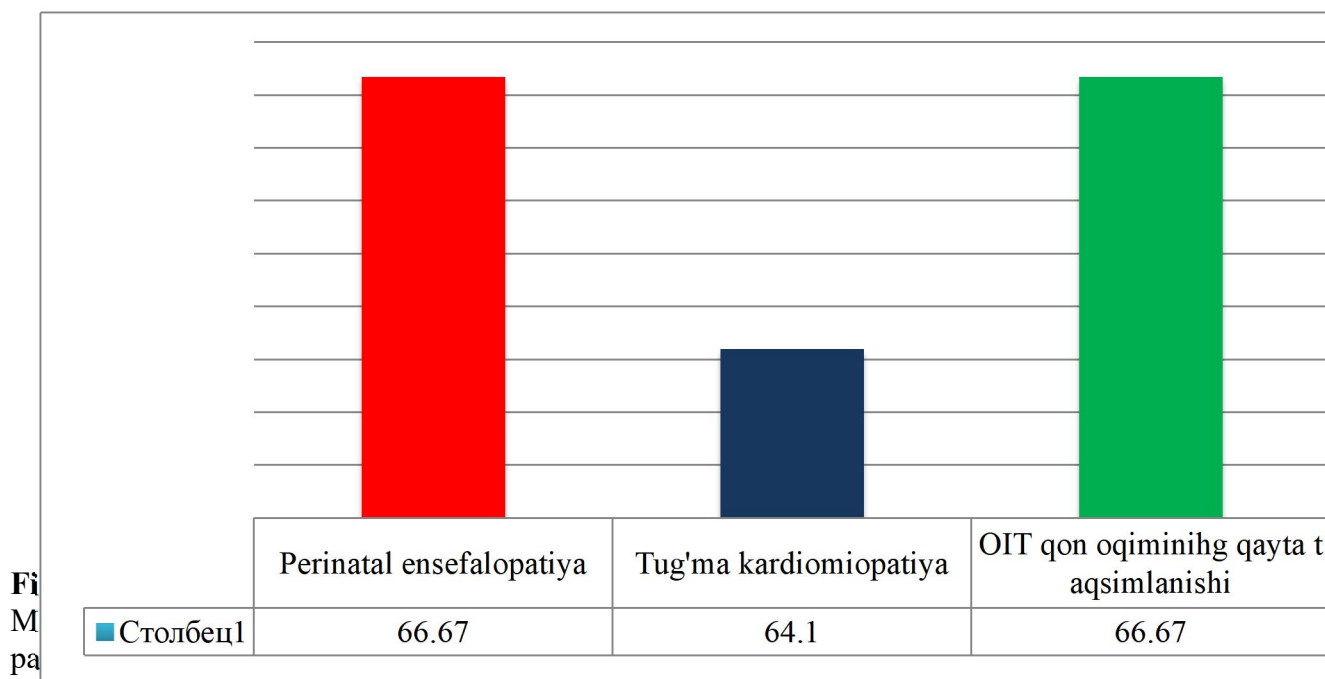
In 8 clinical cases, intraoperative perforation of the ileum and colon with a typical clinic of neonatal peritonitis was diagnosed. For histological examination, the material was fixed in 10% neutral formalin solution and embedded in paraffin after standard transfer. Subsequently, paraffin sections with a thickness of 5-7 μm were stained with hematoxylin, eosin, and picrofuchsin according to Van Gieson.

Results of the study and their discussion. In all studied cases, macroscopic changes characteristic of congenital malformations were not noted, in which secondary perforations could be observed.

In both groups of children, perforation of the gastrointestinal tract occurred mainly in premature infants with low birth weight. However, in the group of infants born with perforating NEC, all 5 infants born at term and with spontaneous perforation were born prematurely and had an average birth weight of 880 g. In infants born with spontaneous perforation, intestinal pneumatization was usually not observed due to pseudoobstruction of the pyloric part of the stomach and significant edema of the perifocal tissues.

The risk factors for the occurrence and development of intestinal perforation in NEC in newborns were as follows (Figure 1):

- 1) chronic fetal hypoxia against the background of perinatal encephalopathy (66.67%) and congenital cardiomyopathy (64.10%);
- 2) intensive enteral feeding (66.67%), which led to ischemic and reperfusion injury of the intestinal mucosa with the appearance of significant intestinal pneumatosis due to the redistribution of systemic and regional blood flow in the gastrointestinal tract of the newborn (66.67%).

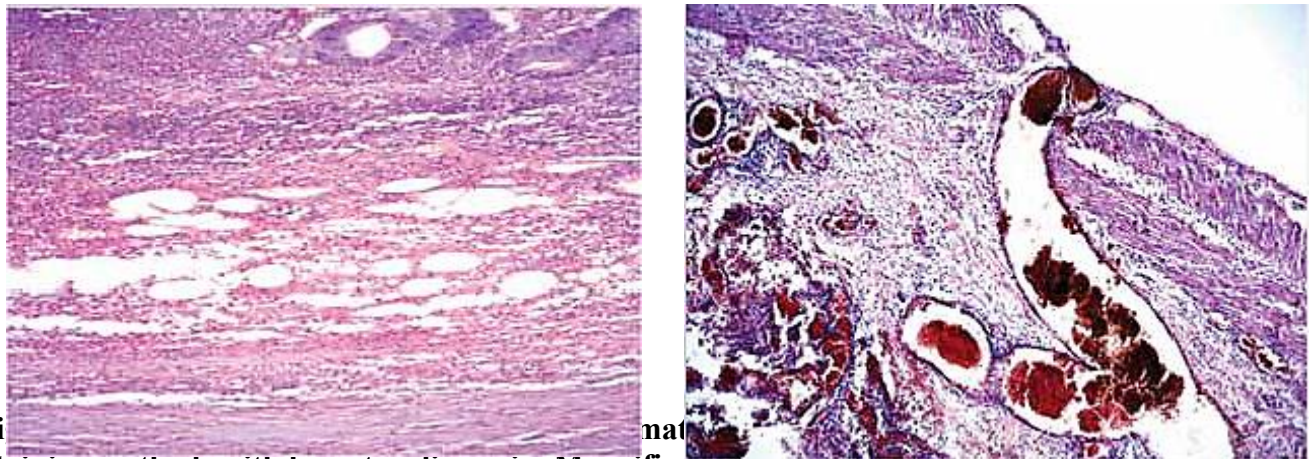


the ileum, in 13 cases multisegmental damage of the jejunum and ileum, in 5 cases in the colon, and in 1 case in pannecrosis.

In 7 infants with NEC, pre-excision perforation was detected in the necrotizing wall of the ileum and colon against the background of a typical clinical picture of acute peritonitis. The most reliable risk factors for spontaneous perforation were acute asphyxia during childbirth and pathology of the respiratory organs, which required tracheal intubation in all infants. Respiratory distress syndrome was the leading risk factor for spontaneous intestinal perforation. Perforations in NEC were numerous, rarely single.

Histological examination revealed ischemia of the intestinal wall, signs of chronic and acute inflammation in almost all cases. In more than half of the presented cases, pneumatosis was detected, which was linear (when gas bubbles are as if elongated and are located in the form of stripes in the muscular and subserous layers) and cystic (in the form of bubbles in the submucosal layer), and in extensive lesions, pneumatosis had a mixed character (Fig. 2).

Perforations occurred in the zone of necrosis of all layers of the intestinal wall. It should be noted that in most cases of spontaneous intestinal perforation in newborns, vascular-muscular hypoplasia or vascular dysplasia of the stomach or intestinal wall (Fig. 3), ectopia of pancreatic tissue fragments to the gastric wall, detected only in histological examination and considered by us as one of the leading risk factors for spontaneous intestinal perforation in newborns.



Fi
mal
Staining method: with hematoxylin-eosin. Magnification x 10, ob. 40.
Fig. № 3. Vaso-muscular dysplasia and vascular malformations are detected in the wall of the small intestine in spontaneous perforation. Staining method: with hematoxylin-eosin. Magnification x 10, ob. 40

Postnatal perforations were always observed with disseminated fibrinous purulent and fecal peritonitis (in 46 children or 77.97% of cases).

Such dysplasias manifested themselves in the form of a circular, focal or segmental defect of the wall of the hollow organ, without involving a large area of the stomach or intestinal wall in the pathological process.

Thus, in intestinal perforation in newborns, damage to the upper parts of the gastrointestinal tract was observed, in our observations an additional risk factor for perforation was tissue defects (vascular dysplasia, less often ectopia), which occurred in almost 2/3 cases of intestinal perforation in infants born with NEC. All newborns with intestinal perforation underwent a surgical procedure. The mortality rate after the visit was 38.5% in perforated NEC (15 infants died), and 10.5% in spontaneous perforation (2 patients died). The main cause of death in intestinal perforation in newborns was premature birth and severe cardiopulmonary diseases (13 newborns), as well as a very severe condition associated with severe neonatal sepsis with multiorgan failure in 4 patients.

Conclusions

1. Gastrointestinal perforations in newborns have clinical and morphological differences in NEC and SP, which allows them to be considered as separate nosological units.
2. The main links in the etiopathogenesis of perforating NEC include a decrease in intestinal blood supply in conditions of prolonged intrauterine hypoxia of the fetus and intensive enteral nutrition, leading to ischemic diseases, and reperfusion damage to the intestinal mucosa.
3. The leading risk factor for spontaneous perforation is distress syndrome, additional tissue defects (vascular dysplasia, less often ectopia), which arise under the influence of adverse factors in the embryonic or early fetal period and manifest as pathology of the labor and early postnatal period.

4. The prognosis for the treatment of children with stenosis of the gastrointestinal tract is more favorable than for perforating NEC, which is associated with the specific features of the morphogenesis of this pathology and local damage to the wall of the hollow organ.

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