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CURRENT TACTICS, DIAGNOSIS AND TREATMENT OF PERITONITIS IN NEONATES

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Abstract
Peritonitis in children is an inflammation of the peritoneum accompanied by intoxication and multi-organ disorders. It develops under the action of pathogenic bacteria in the perforation of hollow digestive organs, haematogenous spread of infection, complications of appendicitis, other acute surgical diseases. Symptoms of peritonitis include excruciating pain and abdominal muscle tension, repeated vomiting, deterioration of general condition (confusion, dehydration, hyperthermia). An ultrasound and X-ray of the abdominal organs, laparoscopy, and laboratory tests are used for diagnosis. Surgical treatment is prescribed, supplemented with antibiotics, infusion therapy.

Keywords: peritonitis, children, newborn, type of treatments.

Relevance

Peritoneal inflammation in newborn children is one of the most serious and often fatal diseases. The causes of peritoneal inflammation in this category of patients are polyetiological. A distinction is made between primary and secondary peritonitis. In primary peritonitis the infection of the abdominal cavity is haematogenous and lymphatic, or in the presence of inflammation of the umbilical vessels. The incidence of peritonitis is 3-4% of all acute surgical diseases in children. Symptoms develop more frequently in younger patients, due to the anatomical and functional features of the peritoneum and the gastrointestinal tract. Peritonitis poses a serious threat to the life and health of the child. Despite improvements in treatment techniques, the mortality rate remains high, ranging from 4.5% in early diagnosis to 50% in advanced and complicated cases. Secondary peritonitis is caused by inflammatory diseases of the abdominal or retroperitoneal organs, perforation of the intestinal wall due to ulcerative necrotic enterocolitis (NEC) or congenital intestinal obstruction, and postoperative complications. In addition, secondary peritonitis can be caused by hernias of the umbilical cord, complicated by ruptured or infected membranes, gangrene of the intestine in a pinched inguinal hernia, etc. The etiological pattern of peritonitis in children differs greatly from that in adults and is directly related to the

age of the patients. The immediate cause of the pathology is the entry of pathogenic Gram-negative or Gram-positive micro-organisms into the sterile peritoneal cavity, which causes an inflammatory process. The main etiological factors of the disease in children are:

- Perforation of the intestinal wall. Represents up to 80% of cases of peritonitis in neonates and infants. Symptoms result from perforation of the wall in necrotising enterocolitis (more common in premature infants) or from congenital abnormalities.
- Haematogenous or lymphogenous infection. Bacteria are transported through the vascular stream from distant foci or circulate in a generalized process, causing peritoneal infection. The factor is particularly significant in young children, who have underdeveloped cellular and humoral immunity and a higher propensity for sepsis.
- Appendicitis. Before the age of 5-6 years, children do not have a fully developed omentum, which should cover the inflamed appendix and prevent generalization of the process. This factor and difficulty in diagnosing appendicitis in young patients provokes complicated forms of the disease and the development of peritonitis.
- Gastrointestinal diseases. In adolescents the symptoms of peritoneal inflammation are often caused by typical "adult" causes: perforation of gastric or duodenal ulcer, exacerbation of cholecystitis or pancreatitis. These factors are very rare in young children.

Objective

To objectify the diagnosis and treatment of perforative peritonitis and organ dysfunction in neonates with JNEC and gastric perforation (GNP).

Materials and methods of investigation. A retrospective, observational, cohort study of 50 neonates, who were admitted and received intensive care in the neonatal intensive care unit at 5-14 days of life in the period 2020-2022.

Results of the Study

During the operation small intestine necrosis and perforation, secondary peritonitis occurred in 26 (56,5%) neonates, colon - in 13% of patients, gastric wall - in 12 (26,1%) cases. Lethal outcome occurred in 32 neonates of the main group. The postoperative mortality was 69.6%. Lethality predominated in neonates with multiple perforations of the small intestine.

In 4 (8.7%) examined neonates with perforative peritonitis, laparocentesis with peritoneal drainage (PD) was an indication for emergency as a temporary intra-abdominal hypertension syndrome therapy. Portal vein pneumotization and/or

pneumoperitoneum with multiple organ failure of 2 or more organs (respiratory and cardiovascular system dysfunction, gastrointestinal organs).

In PD, in neonates (n=4), mortality in the early postoperative period was 25% and in the late period 75%. In children who initially underwent median laparotomy the early postoperative mortality was 11% and the late mortality was 78.1% given a higher number of neonates (n=28) and a single more radical surgical intervention

In the postoperative period, infusion therapy (IT) aimed at correcting gastrointestinal (GI) and respiratory dysfunction and antibiotic therapy were administered. In nasogastric decompression with residual volume of gastric contents more than 6 ml/kg, administration of metoclopramide, proserine was recommended for 3±1 days after surgery, until restoration of motor and evacuatory gastrointestinal function (reduction of abdominal bloating, restoration of bowel peristalsis, gas emptying). Analgesia was managed by a one-stage caudal block with further use of analgin solution. Infusion therapy included a restrictive strategy (weight gain of no more than 1% per day), with early enteral administration of saline solutions (1 ml/kg/h), starting on the second day after surgery. Enteral nutrition was started from 10 days after surgery. The above tactics of intensive therapy made it possible to reduce the period of invasive ventilation, to resume the motor and evacuatory function of the gastrointestinal tract on the 3rd day postoperatively.

Thus, on the 3rd day after operation in comparison with preoperative period, decrease of leukocyte count to 12,0±1,1*109/l, decrease of myelocyte number to 2,1±0,4%, of bacilliform leukocytes to 5,4±0,6%, CRP to 12,0±3,3 mg/l ($P<0,05$) was registered. The pSOFA score was 1.4±0.4, i.e. there was an organ dysfunction of gastrointestinal system 1, and the probability of lethal outcome was within 22%.

On the 7th day there was a further reduction in the laboratory values under investigation, as well as stabilization of the clinical homeostasis indicators in the newborn. The neonatal pSOFA score and the likelihood of an adverse outcome were similar to the previous phase of the study.

Short-term mortality in the neonates studied was 6.3%. Long-term mortality - 56,3%, was caused by the development of late complications of JNEC - adhesive intestinal obstruction (GI organ dysfunction), which required repeated surgical interventions and determined thanatogenesis in cases of long-term mortality.

The diagnosis of NSEK, gastric perforation is based on widely known clinical and laboratory parameters with subjective interpretation due to the polyetiology of NSEK and GI, and the fact that neonates usually receive prophylactic antibiotic therapy for somatic pathology.

Conclusion

The carried out research allowed to single out diagnostic criteria with high specificity and sensitivity at perforative peritonitis, such as: intestinal pneumatosis, multiple air/fluid levels, pneumoperitoneum, as well as to state, that the main links of multiple organ failure in this contingent of patients are dysfunction of GIT and respiratory system.

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