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OPTIMIZATION OF EARLY DIAGNOSIS OF NECROTIC ENTEROCOLITIS IN NEWBORNS

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ABSTRACT — Necrotizing enterocolitis (NEC) is an inflammatory disease of newborns which causes serious damage to the gastrointestinal tract (GIT) leading to development of perforations and intestinal necrosis. In our study, we have focused on early diagnosis of NEC based on imaging and laboratory findings. Elevations in fecal calprotectin can be used as an effective biomarker for diagnosing of NEC. We found that if the level of fecal calprotectin was higher than 1.1 mg/g, NEC can be diagnosed. The level of fecal calprotectin exceeding 1,5 mg/g is considered to be an indication for surgery.

KEYWORDS — NEC, newborns, treatment, diagnosis, fecal calprotectin.

INTRODUCTION

Despite the achievements of medical science and practice there are issues in the clinical medicine that still remain relevant and this refers to necrotizing enterocolitis in newborns.

Relevance of the NEC, despite the progress achieved in diagnosis and treatment, is confirmed by the mortality among the patients after surgery, which reaches 60% and there is no tendency to its reductions [4, 5, 7].

To date, the etiology and pathogenesis of NEC has been the subject of extensive scientific discussion. The main efforts are aimed at finding potential markers to identify NEC in the early stages, to determine the nature of the course and prognosis of the disease [2, 3, 6].

Despite the progress achieved in the diagnosis, the analysis of the immediate and long-term results of treatment of NEC demonstrates that in 16–40% of children the course of the disease is complicated by intestinal perforations, the mortality in which reaches according to different authors from 40–90% [4, 7, 9]. It becomes quite obvious that the success of treatment depends not only on adequate surgical correction,

timely and rational prescription of antibiotics, but also is determined by a complex of diagnostic and therapeutic measures aimed at early detection of signs of complicated course of NEC [1, 6, 8]

It is known that in patients with NEC any parts of the gastrointestinal tract (GIT) can be damaged. The prevailing pathological process in more than 90% of cases is inflammation and coagulation necrosis of the intestinal wall. The proven risk factors in the pathogenesis of NEC are prematurity, early onset of feeding, asphyxia, intestinal ischemia, immune disorders, infection, and low gestational age [1, 5, 9, 4, 6].

Aim:

to optimize early diagnosis and treatment of necrotic enterocolitis in newborns using laboratory and instrumental diagnostic methods.

MATERIALS AND METHODS

Our study encompassed 30 newborns, 10 days old, with necrotic enterocolitis (acute and subacute forms). There were 18 boys (60%) and 12 girls (40%) in the group. All newborns underwent x-ray examination of the abdominal cavity, ultrasound of the abdominal organs. The laboratory tests included: complete blood count (CBC) every 6 hours, clinical urinalysis (UA), coagulogram (thrombin time, activated partial thromboplastin time (APTT), fibrinogen), biochemical blood analysis (C-reactive protein (CRP), glucose, total protein, albumins, triglycerides, bilirubin, creatinine, urea), tests of procalcitonin in the blood on the basis of immunochemiluminescence; bacteriological blood testing for sterility (isolation of pure culture), antimicrobial susceptibility testing of isolated cultures, fecal occult blood test.

The objective of the suggested diagnostic techniques was to identify diagnostic criteria for necrotic enterocolitis (NEC). It is very challenging for pediatric surgeons to reliably assess, whether medical management is possible or surgical intervention is required. This challenge justifies a priority of optimizing diagnosis and treatment of NEC.

RESULTS AND DISCUSSION

To assess the severity of the condition and the stage of the disease, we used the criteria of Bell's

stage. During the first stage, there was a stretching of intestinal loops on the abdominal x-ray survey, CBC: anemia, leukocytosis $+25.4 \times 10^9/l$, leukopenia, $+4.5 \times 10^9/l$, a shift of the leukocyte formula to the left, UA without changes, a biochemical blood test within normal parameters, a fecal analysis for hidden blood-positive. Ultrasound of the abdominal cavity showed the absence of infiltrates and other formations in the abdominal cavity, sluggish intestinal peristalsis. During the second stage, on an overview x-ray of the abdominal cavity, dilation of intestinal loops, an increase in the thickness of the intestinal wall due to edema and inflammation, in the CBC thrombocytopenia is lower than $+150 \times 10^9/l$, UA is unchanged, Biochemical blood test: hypoalbuminemia, hyperbilirubinemia, C-reactive protein $+10 \text{ mg/l}$, increased urea. Ultrasound of the abdominal cavity: reduced peristalsis in the affected segments, uneven, moderate dilatation of intestinal loops. Fecal calprotectin was observed in the range of $1.1-1.4 \text{ mg/g}$. During the third stage, subserous pneumatosis of the intestinal wall with its thickening, fixed intestinal loops, gas in the portal vein system, and pronounced ascites were showed on the x-ray. Ultrasound of the abdominal cavity: pronounced local thickening of the intestinal wall, infiltrates are located, between the loops of the intestine and in the pelvic cavity, CBC neutropenia $+1.5 \times 10^9/l$. Serum electrolytes: hyponatremia, hypocalcemia, hypokalemia. Coagulogram: increase in APTT, thrombin time. Biochemical blood analysis: hypoalbuminemia, hyperbilirubinemia, C-reactive protein $>10 \text{ mg/l}$, increased urea, residual nitrogen, increasing procalcitonin $+2 \text{ ng/ml}$ is a marker of sepsis; ABS of blood pH $+7.2$. The development of intestinal necrosis is indicated by the preservation of metabolic acidosis for more than 4 hours against the background of intensive therapy. In 21 (70%) children a bacteriological blood test gave a positive result. 11 (36.6%) children were operated on. The indicator of fecal calprotectin varied from $1.5-2.5 \text{ mg/g}$. This provides evidence of a deep lesion of the intestinal wall and reflects the severity of the intestinal perforation and enables to select management strategies for newborns.

CONCLUSION

1. Early diagnosis of ulcerative-necrotic enterocolitis in newborns is aimed at determining the level of fecal calprotectin in the stool.
2. If the level of fecal calprotectin is higher than 1.1 mg/g , necrotic enterocolitis is diagnosed (patent RU 2 705 379 C1).
3. If the level of fecal calprotectin is higher than 1.5 mg/g , surgery is considered to be a better option.

Therefore, we have confirmed the feasibility of using fecal calprotectin for early diagnosis of NEC.

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