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Non Contrast Roentgenology Diagnostic of Necrotizing Enterocolitis (NEC) in Newborns

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Abstract: This is a non-specific inflammatory disease caused by infectious agents against a background of immature local defense mechanisms and hypoxia. Ischemic - this is a non-specific inflammatory disease that occurs against a background of ischemic intestinal mucosal damage. The mortality rate is 23-38%. Necrotizing enterocolitis (NEC) in newborns (necrotizing enterocolitis, necrotizing enterocolitis, ulcerative enterocolitis) necrotizing enterocolitis, necrotizing enterocolitis ulcerosa, "premature survival") is a non-specific inflammatory disease of the newborn. Inflammatory diseases of the newborn (necrotizing enterocolitis, ulcerative necrotizing enterocolitis, "premature survival") are non-specific inflammatory diseases caused by infectious agents against the background of immature local defense mechanisms. It is a non-specific inflammatory disease caused by infectious agents against a background of immature local defense mechanisms and hypoxic-ischemic enteritis.

Keywords: GIT - Gastrointestinal tract, NN - Premature newborn, NEC - Necrotizing Enterocolitis, OBP - Abdominal Organs, OPN - Department of Neonatal Pathology.

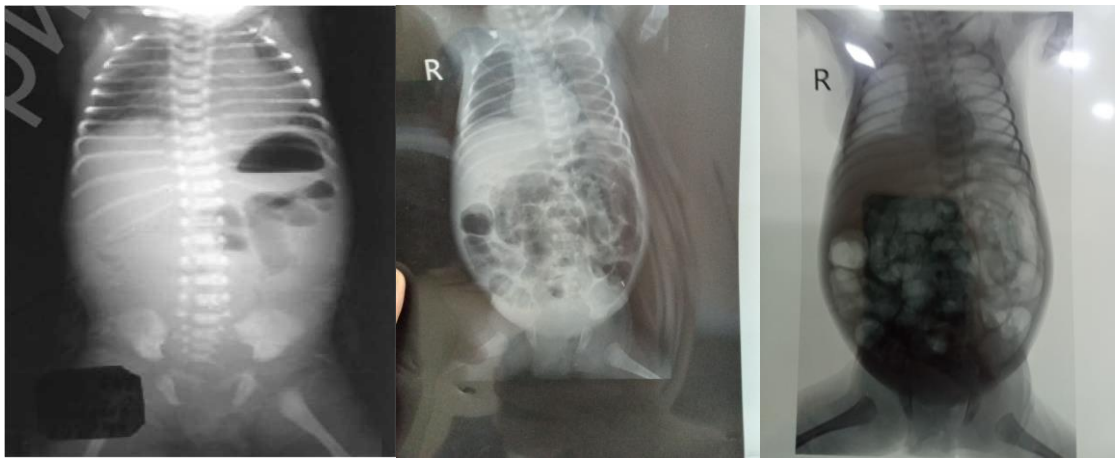
Non-specific inflammatory disease caused by damage to the intestinal mucosa, with a tendency to generalize symptoms due to the development of a systemic inflammatory response. Necrotizing enterocolitis (NEC) in neonates is a nonspecific inflammatory disease caused by infectious agents against the background of immature local defense function. NEC is a nonspecific inflammatory disease caused by infectious agents against the background of hypoxic-ischemic damage to the intestinal mucosa. Mortality is 23-38. Early diagnosis is based on the clinical picture, radiography Based on abdominal radiography and laboratory data, the general classification of M. Bell (1979), modified by M. Walsh and R. R., is as follows The general classification according to M. Bell (1979) modified by Kliegman (1986) is as follows.

Necrotizing enterocolitis (NEC) (necrotizing enterocolitis, necrotizing enterocolitis, necrotizing enterocolitis, ulcerative necrotizing enterocolitis, 'premature infant survival disease') in neonates is a non-specific inflammatory disease caused by infectious agents against a background of immature local defense mechanisms and hypoxic-ischemic damage to the intestinal mucosa, and the systemic process tends to generalize due to the development of an inflammatory response; ICD code P77. In the neonatal period, NEC occurs at an average rate of 4-6 per 1000 live births. Approximately 84-91% of these cases occur in premature infants with low body weight. As a rule, NEC develops in the first 5-10 days of life, but if the baby weighs less than 1000 g, it develops from 3 months. The lower the birth weight, the more often and later NEC occurs. In 1944 W. Heinrich (Switzerland) reported 62 cases of necrotizing enterocolitis in neonates, all of whom died; however, in 1969 J. Lloyd (8) from Detroit suggested that the onset of this condition was associated with neonatal hypoxia. Currently, the mortality rate for this condition is 23-38%. Etiology: NEC occurs in newborns with ischemic damage to the intestinal wall. It occurs in

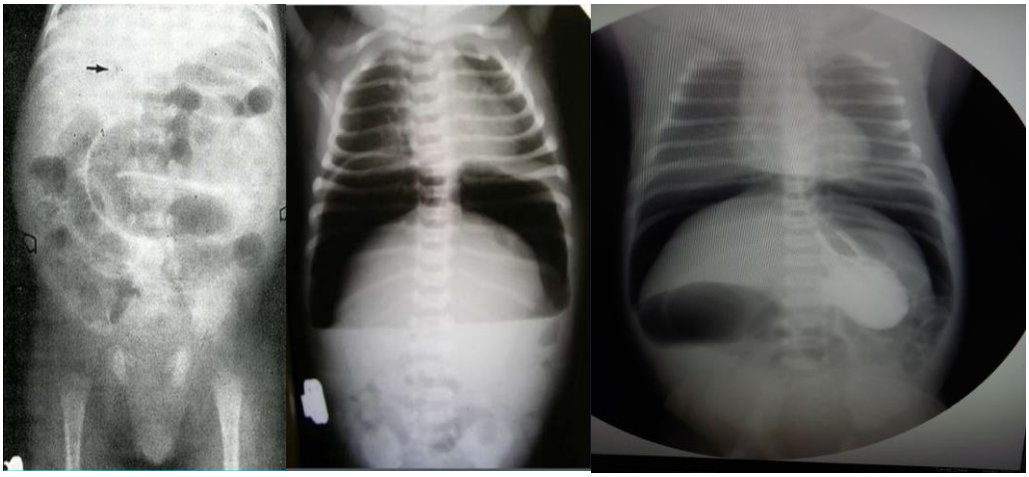
neonates with ischemic damage to the intestinal wall, dysbiosis or hyperosmotic feeding. Predisposing factors. Hypotension, hyperthermia, right-to-left shunts and congenital heart disease or Poly-anemia, anemia. Umbilical vein catheterization and replacement transfusion, e.g. factors that cause reduced intestinal perfusion have specific effects.

The combination of prematurity, immature immune response and impaired gastrointestinal function is important. Gram-negative microorganisms (e.g. Escherichia coli, Pseudomonas aeruginosa, Proteus, Klebsiella and anaerobes) are found in the ascitic fluid of children affected by NEC. In 24-36% of patients, the microflora spreads from the blood. No specific pathogen has been identified. Etiology. Mesenteric vascular spasm, microembolism or thrombosis of small arteries causes localized hypo-oxygenation and damage to the intestinal wall, leading to disruption of its integrity. Hypoperfusion rapidly reduces mucosal resistance and ischemic areas are more susceptible to microflora. However, ischemia of the intestinal wall is not the only etiology of NEC. The permeability of the intestinal wall increases and anaerobic and (or) water-producing bacteria invade the intestinal wall and secrete gas (Clostridium, Bacteroides). This submucosal or subplasma air accumulation produces symptoms of necrotizing enterocolitis in the form of cystic intestinal emphysema. Increased permeability facilitates the passage of bacteria through the intestinal wall into the abdominal cavity and systemic circulation. In most patients shows a high degree of inflammation. Local and systemic inflammatory reactions and increased blood cytokine levels. The first histopathologic changes are edema and detachment of the intestinal villous epithelium. Then destruction of the villi, signs of submucosal swelling, followed by microbleeding, the appearance of microthrombi and capillary stasis are characteristic. In severe cases, the villous structures may disappear completely and the mucosa may ulcerate. Submucosa and serosa (blisters) are present. Serosa is edematous. Due to edema. Degenerative changes in the submucosal and intermuscular plexus are also detected. Detected The plexus loses glial cells and neurons. Ganglia look like "empty baskets". Lesions The lesions appear as follows. Production of neurotransmitters that inhibit muscle tone. Decreased production of neurotransmitters that inhibit muscle tone (vasoactive intestinal peptides and nitric oxide). Changes in the submucosal plexus Changes in the submucosal plexus predominate and are more pronounced the more severe the mucosal damage. The more severe the mucosal damage, the more prominent these changes are. Therefore, degenerative changes in the enteric plexus are observed in NEC. Observed. Reduced blood flow to tissues leads to a decrease in the amount of adenosine. (ATP), the oxygen supply required for ATP decreases and the cellular potential drops. Loss of ability to maintain the ionic gradient that allows transport across the cell membrane. a Redistribution of calcium ions takes place. As the concentration of calcium ions increases, proteases are activated. Xanthine dehydrogenase is converted to xanthine oxidase. Intracellular. As intracellular ATP stores are depleted, the concentration of other adenine nucleotides increases. Adenosine is catabolized to inosine, hypoxanthine and xanthine. They are catabolized to xanthine. These reactions result in a 'burst' of superoxide, hydrogen peroxide and other toxic oxygen anion radicals. Other toxic oxygen anion radicals are produced and cause cell destruction. Another mechanism that suppresses superoxide production is the inhibition of xanthine oxidase by the inhibitor allopurinol. Clinical picture. Initial symptoms are usually non-specific: dehydration, low-grade fever, persistently distended abdomen, often bloody diarrhea, vomiting with bile, apnea attacks, bradycardia. In parallel, there are gastrointestinal symptoms such as persistently increasing abdominal distension and foul-smelling stools with blood. After various periods, the patient's condition deteriorates rapidly. The child becomes sleepy and the skin is pale gray, marble-like and cold to the touch. The abdomen is tense and shiny, with a prominent vascular pattern. Soon, abdominal sputum (edema and hyperemia), sepsis, metabolic acidosis, intravascular coagulation syndrome and respiratory failure appear, which indicate the progressive course of the disease. In observational dynamics, the following stages are distinguished: The classification according to the stage of NEC progression proposed by M. Bell et al. (1979) and modified by M. Walsh and R. Kliegman (1986) is common and often used: Stage 1 - 'Suspicion of necrotizing enterocolitis' or first symptoms 1A - Temperature instability,

apnea, bradycardia, In children, lethargy, mild abdominal distension, blood in the stool; 2A - "reversible phase", appearance of stage 1 symptoms + intestinal paralysis + radiologic intestinal emphysema. Radiologic intestinal emphysema; 2B - 'irreversible phase', same symptoms + X-radiographic portal system gas; Phase 3 - 'onset of complications': 3A - symptoms of peritonitis, dilatation of intestinal loops, horizontal multilevel, pneumothorax, necrosis of the intestinal wall; 3B - diffuse peritonitis with complications (diffuse endovasculitis) Disseminated peritonitis with complications (disseminated intravascular coagulation syndrome, multiorgan failure syndrome, sepsis). Disseminated intravascular coagulation syndrome, multiorgan failure syndrome, sepsis), pneumoperitoneum. To suspect the development of this disease, a dynamic radiographic examination is necessary. The examination is carried out. X-ray examination of the abdomen is performed; X-ray signs of NEC: Irregular swelling of the intestinal loops, emphysema of the intestinal wall (cystic or linear), gas in the portal system Irregular swelling of the intestinal loops, emphysema of the intestinal wall (cystic or linear), gas in the hepatic portal system, presence of low loops in the intestine Presence of low intestinal loops, pneumoperitoneum, presence of fluid in the abdominal cavity. Intestinal emphysema and gas in the hepatic portal system. Radiographic findings at various stages of NEC by M. Bell (1979) and M. Walsh and R. Kliegman (1986) (1979) and M. Walsh and R. Kliegman (1986): Stage 1A: Dilatation of the intestinal loop is the most common sign and is found in 65% of newborns. It is found in 65% of newborns in the early stages of the disease. Prominent. A marked dilatation of the intestinal loops and the appearance of horizontal levels indicates the severity and progressive course of the disease. Severity and progressive course of the disease. 1B - blood in the stool with the above. Stage 2 - 'marked necrotizing enterocolitis': Stage 1B - intestinal loops are fixed (stationary) and do not change position on repeated imaging (intestinal loops are flattened and rigid). This sign indicates bowel ischemia and is present in 15% of cases. These loops may be localized, solitary or multiple. By comparing radiographs taken at regular time intervals (after 6-8 hours), it is possible to distinguish between normal dilatation of the intestinal loops and fixed and immobile areas (Figure 3). Stage 2A: Gastric dilatation is a symptom of NEC and is caused by both gastric atony due to exposure to bacterial toxins and pseudo-obstruction of the pylorus as a result of mucosal edema and occurs in 7-10% of children with NEC.



Ist A Stage II B (Image 1).



IInd A stage III A (image II)

Stage 2 A Stage 2 B (Figure 1). Stage 3 A (Figure 2) Stage 2B: Insufflation of intestinal loops and heterogeneous gas filling is present in 30-40% of patients. It is present in 30-40% of patients. This symptom is the result of active peristalsis Active peristalsis results in the movement of intestinal contents from the viable to the ischemic areas of the intestine. This is because intestinal contents move to ischemic sites where peristalsis is reduced or completely absent. The hypoperistalsis of the intestine is shown in Figure 5. Stage 3A: Sudden appearance of signs of ascites on radiographs, present in 11% of patients, an unfavorable factor indicating peritoneal reaction to bacterial peritonitis and possible perforation of the bowel covering. Stage 3B: emphysema of the intestinal wall (intramural gas retention). This symptom is characteristic of Stage 3. Emphysema is present in about 18%. Emphysema does not always precede bowel perforation. It does not always precede perforation. Emphysema of the bowel wall is present in 90% of patients. Pneumatosis of the bowel wall is a definite sign of NEC and is seen against a background of swelling during surgery. Recognized during surgery or radiographic examination against a background of swelling of the bowel loops. It is recognized on examination. Gas in the portal vein is a reliable sign of an advanced course and determines a poor prognosis. This symptom is present in 3% of cases. Gas in the form of emboli may enter the portal system through the necrotic bowel wall (with emphysema) or form directly in the vascular capillaries as a result of invasion of gas-producing bacteria (anaerobes) into the vascular capillaries. The presence of gas in the portal system is present in 61% of children with total intestinal necrosis and determines a poor prognosis, so the presence of this manifestation is an absolute indication for surgical treatment. Pneumoperitoneum is the accumulation of free gas in the abdominal cavity, often under the dome of the diaphragm, with unquestionable perforation of hollow organs, which is characteristic only in stage 3B of the disease and is detected in 98-99% of newborns with NEC.

Absence of free gas in the abdominal cavity on X-ray examination. Absence of free gas in the abdominal cavity on radiographic examination is seen in cases of 'covered' perforation when the location of the hole in the omentum is atypical. Pneumoperitoneum can occur in many patients ventilated for lung lesions (pressure trauma ruptures the alveoli, allowing air to enter the abdominal cavity through the mediastinum) and in debilitated patients even in the absence of perforation of hollow organs. This occurs in 10-15% of cases. This should be considered in the differential diagnosis of necrotizing enterocolitis. Recently, increased intra-abdominal pressure and the development of intra-abdominal hypertension syndrome associated with the progression of necrotizing enterocolitis have attracted attention. In 2004, WSACS (World Society of Abdominal Compartment Syndromes) In 2004, WSACS (World Society for Abdominal Compartment Syndromes) defined intra-abdominal hypertension as a persistent increase in intra-abdominal pressure. It is defined as defined in the above article. intra-abdominal hypertension is defined as a persistent increase in intra-abdominal pressure of greater than or equal to Intra-abdominal hypertension is a precursor to the development of abdominal inflammatory syndrome.

It is a precursor stage of abdominal inflammatory syndrome. Intra-abdominal hypertension syndrome is a multi-organ failure caused by increased intra-abdominal pressure in a confined space. It leads to organ and tissue circulatory disorders, hypoxia and ischemia. It causes ischemia of organs and tissues and a marked decrease in their functional activity. It significantly reduces their functional activity until they come to a complete standstill. A useful diagnostic method is ultrasonography. Dilatation of the intestinal loops, stratification and thickening of the intestinal wall, free fluid and gas in the abdominal cavity Free fluid and gas in the abdominal cavity can be detected. Doppler ultrasonography can Assess blood flow in the vessels of the superior mesenteric artery and in some cases allow a clear progressive staging of intestinal necrosis. In some cases the stages of necrotizing enterocolitis can be clearly classified. This is the transitional stage to necrosis of the intestinal wall and requires aggressive surgical treatment. The most common findings on clinical examination for diagnosis are changes in the hematogram (increased or decreased leukemia, left shift of the leukemic formula, thrombocytopenia), increased C-reactive protein, acidosis and electrolyte imbalance. Unfortunately, all these are not specific to this condition. According to foreign authors, the role of inflammatory markers in the pathogenesis of necrotizing enterocolitis is being actively studied. Among many potential molecular prognostic factors, the matrix metalloproteinase system, lactoferrin, calprotectin and fatty acid binding proteins occupy a special place. Surgical intervention is necessary if there is free fluid in the abdominal cavity and signs of perforated peritonitis. This is because the chances of recovery are significantly increased. Surgery consists of transverse laparotomy, abdominal lavage, resection of the necrotic bowel and creation of one or more enterostomies. Resection should be done sparingly to avoid a short bowel. 1. NEC usually occurs in low birth weight neonates weighing 500 to 1550 g. In newborns up to 32 weeks of age with ischemic damage to the intestinal wall, Newborns with ischemic damage to the intestinal wall, dysbiosis and hyperosmotic feeding. In this case, increased permeability of the intestinal wall was observed and In this case, increased permeability of the intestinal wall was observed and anaerobic bacteria penetrated the intestinal wall and released gas (intestinal emphysema pneumatosisintestinalis) and colonization by pathogenic bacteria leads to enteritis and peritonitis. 2. 2. Three stages can be distinguished during the course of the disease.

Stage II 'suspected necrotizing enterocolitis' or initial symptoms, Stage II 'overt necrotizing enterocolitis'. Stage III 'development of complications'. The clinical picture of each stage and The clinical and radiographic picture of each stage is different and requires appropriate treatment tactics by a physician. Appropriate treatment by a physician is necessary.

3. Surgical treatment is indicated in stage III. To comes down to transverse laparotomy, sanitation of the abdominal cavity, resection of the affected intestine, and the application of an enterostomy. Restoration of intestinal patency is planned in 6-8 weeks.

REFERENCES:

1. Golovko, O. K., Levitskaya E. M., Linchevsky G. L. Features of the management of newborns with necrotizing enterocolitis // Archives of Clinical and Experimental Medicine. - 2002. - T. 11, No. 2. - P. 241-244.
2. Ivanov, V.V., Chevzhik V.P., Cherpalyuk E.A. Surgical tactics in the treatment of enterocolitis in newborns // Pediatric surgery. - 2003. - No. 6. - P. 25-27.
3. Karavaeva, S. A. Diagnosis and features of the clinical course of necrotic enterocolitis in children // Bulletin of surgery. - 2002. - T. 161, No. 4. - pp. 41-46
4. Krasovskaya, T.V., Kobzeva T.N. Diagnostics and intensive care in neonatal surgery. - M.: Publisher Mokeev, 2001. - P. 23-28. 18.
5. Krasovskaya, T.V. Surgery of newborns: diagnosis and intensive care: Methodological recommendations. - 2nd ed., revised. and additional / T. V. Krasovskaya, T. N. Kobzeva. - M.: Moscow, 2003. - 80 p.

6. Amoury, R. A. In the book: “Pediatric surgery / K. W. Ashcraft, T. M. Holder. - St. Petersburg, 1996. - T. 2. - P. 10.
7. Bell, M. J., Shackelford P., Feigin R. D. et all. Epidemiologic and bacteriologic evaluation of neonatal necrotizing enterocolitis. Pediatric Surgery, 1979, 14, 1-4.
8. Lioyd, J. R. The etiology of gastrointestinal perforations in the newborn // J. Pediatr.Surg., 1969, 4, 77-84.
9. McCord. J. M. Oxygen-derived free radicals in postischemic 1 3 tissue injury // N. Engl. J. Med., 1985, 312, 159-163.
10. Walsh, M. C. Necrotizing enterocolitis: treatment based on staging criteria / M. C. Walsh, R. M. Kliegman // Pediatric clinic of North America. 1986, 33, 1, 179- 197