Residência RP Pediátrica
Publicação Oficial da Sociedade Brasileira de Pediatria
ISSN-Online: 2236-6814

Submitted on: 02/18/2021
Approved on: 05/12/2021

CASE REPORT

Neonatal necrotizing enterocolitis: case report and literature review

Karla Denise Barros Ribeiro Vasques[1], Érika de Oliveira Santos[1], Neideana Ewerton Aleixo[1]

Keywords:
Enterocolitis.
Enterocolitis, Necrotizing.
Infant, Newborn,
Diseases.

Abstract

Neonatal necrotizing enterocolitis (NEC) is a syndrome characterized by inflammation and ischemic necrosis of the gastrointestinal tract that can course with variable clinical manifestations: such as abdominal distension, bilious vomiting, hematoquezia, frequent gastric waste, decreased hydro-air noises, fecal changes. The condition is usually associated with systemic signs (pallor, apnea, hypoactivity, dysthermias). NEC can manifest with partial intestinal necrosis and have complete recovery, requiring only clinical treatment. Case report: newborn (NB) of T.A.A., female, premature (33 weeks and 5 days), extremely low weight (888g), APGAR 8/9, referred to the Neonatal Intensive Care Unit (ICU NEO). On the 1st day of life, he presented significant abdominal distension and bilious debt by the orogastric tube (SOG). It eliminated meconium after rectal stimulation, with 43 hours of life. He maintained abdominal distension until the 10th day of life, progressing, with progressive improvement (reduction of abdominal distension, change in the aspect of the debt by SOG), after the institution of adequate therapy. On the 11th day of life, he started a minimal enteral diet, progressing gradually, achieving success in diet progression. Enterocolitis is one of the most frequent gastrointestinal emergencies in neonates, most frequently affecting premature newborns, especially those born with very low birth weight. Studies indicate the recent increase in the incidence of NEC in NEO ICUs, in several countries, due to the increased survival of premature newborns. Therefore, there is a need for a greater dissemination of knowledge about the pathology; mainly among health professionals who work in these units.

[1] Fundação Santa Casa de Misericórdia do Pará, Residência de Pediatria - Belém - Pará - Brazil.

Endereço para correspondência:
Karla Denise Barros Ribeiro Vasques.
Fundação Santa Casa de Misericórdia do Pará. Rua Bernal do Couto, nº 1040,Umarizal, Belém, PA, Brazil. CEP: 66055-080. E-mail: karladenisebr@hotmail.com

Residência Pediátrica; 2023: Ahead of Print.
DOI: 10.25060/residpediatr-2023.v13n3-567

Este trabalho está licenciado sob uma Creative Commons Attribution 4.0 International License.

INTRODUCTION

Neonatal necrotizing enterocolitis (NEC) is a syndrome characterized by inflammation and ischemic necrosis of the gastrointestinal tract that can present with varied manifestations, such as abdominal distension, bilious vomiting, and hematochezia, as well as frequent gastric residues (bilious or not), decreased bowel movements, fecal changes, erythema on the abdominal wall and palpable abdominal mass.[1] These symptoms are generally associated with systemic signs (such as pallor, apnea, hypoactivity, dysthermia), which can occur in variable and progressive intensity.[2]

This pathology is one of the most common gastrointestinal emergencies in newborns; it most frequently affects premature babies, especially those born weighing less than 100 grams (extreme low birth weight – ELBW). Full-term newborns (NB) account for 10% of cases of NEC; and preterm infants, small or not for their gestational age, account for 90% of the cases.[2,3]

An important aspect to be considered in this syndrome is the difficulty in establishing an early diagnosis, and thus implement premature therapeutic intervention. This is because initial signs and symptoms can be non-specific and variable, in terms of their form and period of presentation.[4]

Studies indicate a recent increase in the incidence of enterocolitis in Neonatal Intensive Care Units (NEO ICU) in several countries, due to the increase in survival of low birthweight children, demanding the need for greater dissemination of knowledge regarding the pathology, especially among healthcare professionals who work in these units. Information on appropriate management must be shared, with the aim of reducing the incidence and complications associated with NEC.[5]

CASE REPORT

A 27-year-old mother, G2P1A0, incomplete prenatal care. No complications during pregnancy. On ultrasound (33 weeks), fetal centralization, intrauterine growth restriction and oligohydramnios were detected, and a cesarean section was indicated. Antenatal corticosteroids were administered.

Newborn born to T.A.A., female, 33 weeks, EBP (888g), Apgar 8/9, developed early respiratory distress and was referred to the NEO ICU and started on antibiotic therapy.

On the 1st day of life, he developed abdominal distension, bilious output through the orogastric tube (SOG); with a flaccid abdomen on palpation. He eliminated meconium after rectal stimulation, at 43 hours of age.

On the 2nd day of life, he presented hyperemia in the abdominal wall region; umbilical catheter was removed, parenteral nutrition was started and fasting was maintained. An abdominal x-ray was requested, without significant changes.

On the 5th day of life, clear output was observed by SOG, a flaccid, painful abdomen, and fasting was maintained for 5 days. On the 7th day of life, a blood count showed leukocytosis and thrombocytopenia; and antibiotics were prescribed.

From the 5th to the 10th day of life he remained without abdominal distension, clear output due to SOG. On the 10th day of life SOG was closed. Serial abdominal radiographs showed no significant changes and abdominal ultrasound showed no changes.

On the 11th day of life, we started a minimum enteral diet, opting for colostrum in a volume of 1ml every 3 hours, progressed on the 12th day of life to 2ml, with maintenance of parenteral nutrition; when he presented episodes of apnea with central cyanosis, orotracheal intubation was performed, and he was returned to fasting.

On the 14th day of life, he presented mild abdominal distension, but with a flaccid abdomen and clear SOG output. Evacuations absent since the previous day. Antibiotics were escalated due to clinical and laboratory worsening; he was kept on fasting and SOG open for 24 hours.

On the 15th day of life, accidental extubation occurred and he tolerated circulating oxygen.

He progressed successfully in diet progression and clinical-laboratory improvement. The diet was started with 2mL of expressed human milk (LHO) via SOG and progressed to 20mL/kg/day, until reaching full volume. The patient was discharged from the ICU after clinical stability, full diet and weight required for discharge.

The diagnostic hypothesis was suspected enterocolitis, stage IA, according to Bell's classification.

LITERATURE REVIEW AND DISCUSSION

NEC has a multifactorial origin and affects the gastrointestinal tract of newborns, especially preterm infants. It is the most common cause of gastrointestinal urgency in this population, especially in newborns with ELBW.[3]

The disease has a broad spectrum, which can range from a mild case to more severe forms (characterized by intestinal necrosis, perforation and diffuse peritonitis). Those who present with partial necrosis of the intestine may recover completely, requiring only clinical treatment.[6] In the case described above, only clinical treatment was necessary and the newborn had a complete recovery from the condition. Table 1 below presents risk factors or factors strongly associated with NEC.

The main risk factors presented in this clinical case were prematurity and probable abnormal bacterial colonization, in addition to infection found in blood culture.

Prematurity is associated with NEC due to the immaturity of the intestinal barrier, altered peristalsis, changes in the intestinal microflora, overload of the enteral diet, in addition to immature intestinal immunity.[7]

About 90 to 95% of children who developed NEC had started, restarted, or were progressing on an oral formula diet. Breast milk is capable of reducing the occurrence of NEC by up to ten times when compared to enteral formulas, it contains bioactive factors, which influence the protection of the mucosal barrier and immunity, in addition to increasing intestinal bacterial diversity, it contains immunomodulatory factors.[6]

Table 1. Risk factors or factors strongly associated with NEC.

RISK FACTORS OR FACTORS STRONGLY ASSOCIATED WITH NECROTIZING ENTEROCOLITIS			
Perinatal events	**Neonatal period**	**Food factors**	**Intraluminal bacteria**
Absence or reversal of end-diastolic blood flow from the umbilical artery (chronic intestinal ischemia)	Respiratory distress syndrome	X LM Formula	Uncertain; some patients have evidence of endothermy and HMC+
Maternal eclampsia	Episodes of apnea	Milk formula with high density	Clusters
Fetal distress	Cyanotic cardiopathy (Term NB in 10% of the cases)	Early enteral feeding	Pneumatosis is made up of 30% hydrogen and methane
RUPREME	PCA (NSAID)/fetal circulation	Rapid progress of the enteral diet	Hydrogen is exclusive of bacterial metabolism
C-section delivery	Sepsis		
Perinatal asphyxia	Umbilical catheter		
Perinatal hypothermia	Exchange transfusion		

Abdominal distension is the most common initial clinical finding, occurring in 70 to 90% of cases. Other findings are: gastric stasis or vomiting, blood in the stool, abdominal wall hyperemia, abdominal tenderness. Gastric aspirate or bilious vomiting occurs in 32% of patients. Nonspecific clinical findings include lethargy, dysthermia, recurrent apnea, bradycardia, shock.[8]

Abdominal radiography can provide important information, but is not essential for diagnosis, as there may not be any radiological changes. It is useful for evaluating the presence of gas in the loop wall, presence of gas in the portal system and abdominal free gas.[8]

Clinical management should be initiated as soon as NEC is suspected, it consists of:[5]

- fasting, for intestinal rest;
- use of open SOG to decompress the gastrointestinal tract;
- total parenteral nutrition, during the fasting period and resumption of the diet, until full volume is reached;
- administration of fluids, to correct losses; cardiovascular and respiratory support;
- use of broad-spectrum antibiotics, offering coverage for pathogens that cause late-onset bacteremia (sepsis ≥72 hours of age), with coverage for anaerobes being important when intestinal perforation is suspected; 10 to 14 days of treatment are usually sufficient. If fungemia is suspected, fluconazole or amphotericin B should be started.
- perform serial physical examinations, in addition to laboratory and radiological studies, to monitor the course of the disease and response to therapies.

Surgical intervention is given to those who present evidence of pneumoperitoneum, indicating that there is intestinal perforation due to severe necrosis, being the only absolute indication.[13] Primary peritoneal drainage or exploratory laparotomy with resection of the intestinal region(s)) affected, are the main surgical procedures performed, the choice is based on the experience of the team at each center.[5]

Bell and collaborators, in 1978, developed a classification based on history findings, clinical and radiological aspects (Chart 2).

The main strategies for preventing NEC are prevention of prematurity by strengthening prenatal care, use of breast milk, prenatal corticosteroids (promotes intestinal maturation), slow advancement of the enteral diet (parenteral nutrition and minimal diet advancement); other measures such as the use of probiotics, antioxidants (vitamin E), lactoferrin supplementation, are promising, but are still under study.[9]

Currently, great importance has been given to the preventive role of enteral supplementation with probiotics, evidence shows a reduction in NEC and mortality in babies born with very low birth weight (VLBW)[10-12].Furthermore, there is evidence that the use of Probiotics cause a reduction in invasive, late-onset infections in premature babies or newborns with VLBW.[11] Its mechanism of action occurs through competition with pathogens for nutrients, limiting the growth of such pathogens.[13] Other actions described are: increasing the expression of enzymes in the intestinal digestive tracts, stimulate the differentiation and proliferation of enterocytes and improve the integrity of the intestinal mucosal barrier.[14]

Despite the benefits of its use, barriers to prophylactic use in NEO ICUs are encountered, such as: difficulty in reaching consensus on the ideal constitution of probiotics, which strains to use, number of doses, time of introduction and duration of therapy.[11] Furthermore, a risk of sepsis, bacteremia and fungemia associated with its use has been described, with the possibility of potential long-term consequences.[10]

CONCLUSION

Due to the increase in the incidence of NEC in NEO ICUs in several countries and the increase in low-weight children survival rates, greater dissemination of knowledge about this pathology is necessary, especially among healthcare professionals who work in these units, so that there is reduction in incidence, as well as adequate management, thus avoiding complications.

Table 2. Bell Stages.

BELL STAGES– CLINICAL PRESENTATION, RADIOLOGICAL FINDINGS AND TREATMENT				
Stages	**Systemic signs**	**Abdominal signs**	**Radiological signs**	**Treatment**
IA: suspicious	Thermal instability, apnea bradycardia, lethargy	Gastric residue, abdominal distension, vomiting, blood in stool (microscopic)	Normal or mild distention, similar to septic ileum	Fasting + antibiotic for three days
IB: suspicious	Same as IA	Same as IA, with enterorrhagia/melena	Same as IA	Same as IA
IIA: mild disease	Same as IA	Same as IB, No RHA, with or without abdominal distention	Distention of loops, ileum and intestinal pneumatosis	Fasting + antibiotic for 7–10 days
IIB: moderate disease	Same as IA, plus mild metabolic acidosis and low platelet count	Same as IIA + no RHA, pain upon abdominal palpation, with or without wall cellulitis or palpable mass in the right iliac fossa	Same as IIA, plus ascites	Fasting + antibiotic for 14 days
IIIA: advanced disease	Same as IIB, plus hypotension, bradycardia, severe apnea, with metabolic and respiratory acidosis, neutropenia e CIVD	Same as IIB plus signs of peritonitis, important abdominal pain and distention	Same as IIB	Fasting + antibiotic for 14 days, fluid resuscitation, inotropic, ventilatory support, paracentesis
IIIB: advanced disease with intestinal perforation	Same as IIIA	Same as IIA	Same as IIA plus pneumoperitoneum	Same as IIIA plus surgery

Interventions should be implemented such as preventing prematurity, strengthening prenatal care in the public healthcare network, early feeding of the newborn, with preference for the use of human milk to feed the newborn, in addition to early diagnostic investigation given the clinical picture presented, which leads to the suspicion of NEC.

Thus, we know that the best clinical practice is based on the prevention and early diagnosis of this pathology, and that such measures generate considerable impacts on the survival of these patients.

REFERENCES

1. Vieira MT, Lopes JMA. Fatores associados à enterocolite necrosante. J Pediatr (Rio J). 2003;79(2):159-64.
2. Santos MN, Lutti-Filho JR, Guerra P, Murbach CO, Lopes JM, Respondovesk T, et al. Análise de casos de enterocolite necrosante do Hospital Universitário São Francisco na Providência de Deus no período de janeiro 2015 a outubro 2017. Resid Pediatr. 2018;8(3):123-7.
3. Corso AL, Feldens L. Perfuração intestinal no recém-nascido pré-termo extremo: enterocolite ou perfuração isolada? In: Sociedade Brasileira de Pediatria. Procianoy RS, Leone CR, orgs. PRORN Programa de Atualização em Neonatologia: Ciclo 12. Porto Alegre: Artmed Panamericana; 2015. p. 27-63.
4. Chu A. Necrotising enterocolitis: predictive markers and preventive strategies. N Rev. 2013;14(3):113-20.
5. Kim JH. Neonatal necrotizing enterocolitis: Management. UpToDate [Internet]. 2023; [cited 2021 Mar 1]. Available from: https://www.uptodate.com/contents/neonatal-necrotizing-enterocolitis-management?search=enterocolite%20necrosante&source=search_result&selectedTitle=3~150&usage_type=default&display_rank=3.
6. Choi YY. Necrotizing enterocolitis in newborns: update in pathophysioloy and newly emerging therapeutic strategies. Korean J Pediatr. 2014;57(12):505-13.
7. Schnabl KL, Van Aerde JE, Thomson ABR, Clandinin MT. Necrotizing enterocolitis: a multifatorial disease with no cure. World J Gastroenterol. 2008;14(14):42-61.
8. Berman L, Moss RL. Necrotizing enterocolitis: an update. Semin Fetal Neonatal Med. 2011;16(3):145-50.
9. Kim JH. Neonatal necrotizing enterocolitis: Prevention. UpToDate [Internet]. 2023; [cited 2021 Mar 1]. Availabe from: https://www.uptodate.com/contents/neonatal-necrotizing-enterocolitis-prevention?search=enterocolite%20necrosante&source=search_result&selectedTitle=5~150&usage_type=default&display_rank=5.
10. Thomas JP, Raine T, Reddy S, Belteki G. Probiotics for the prevention of necrotising enterocolitis in very low-birth-weight infants: a meta-analysis and systematic review. Acta Paediatr. 2017 Nov;106(11):1729-41. DOI: https://doi.org/10.1111/apa.13902. Epub 2017 Jun 9.

11. Sharif S, Meader N, Oddie SJ, Rojas-Reyes MX, McGuire W. Probiotics to prevent necrotising enterocolitis in very preterm or very low birth weight infants. Cochrane Database Syst Rev. 2020;10(10): CD005496. DOI: https://doi.org/10.1002/14651858.CD005496.pub5.
12. Sing B, Shah PS, Afifi J, Simpson CD, Mitra S, Dow K, et al. Probiotics for preterm infants: a national retrospective cohort study. J Perinatol. 2019;39(4):533-9.
13. Embleton ND, Zalewski S, Berrington JE. Probiotics for prevention of necrotizing enterocolitis and sepsis in preterm infants. Curr Opin Infect Dis. 2016 Jun;29(3):256-61. DOI: https://doi.org/10.1097/QCO.0000000000000269. PMID: 27023404.
14. Sanders ME, Merenstein DJ, Reid G, Gibson GR, Rastall RA. Probiotics and prebiotics in intestinal health and disease: from biology to the clinic. Nat Rev Gastroenterol Hepatol. 2019;16(10):605-16.