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Perspective

Factors Involved in NEC (Necrotizing Enterocolitis) Pathogenesis

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DESCRIPTION

The destruction of intestinal tissue is known as Necrotizing Enterocolitis (NEC). Intestinal inflammation and necrosis are the characteristics of necrotizing enterocolitis, which can develop to systemic infection, multiple organ failure, death, and long-term neurological complications. The primary risk factor is being premature, meconium aspiration syndrome, postnatal hypoxia, congenital heart disease, and abnormal microbial colonisation and infections.

Gram-negative enteric bacteria such as *Escherichia coli*, *Klebsiella spp.*, and *Pseudomonas aeruginosa*, Gram-positive bacteria such as *Enterococcus*, *Staphylococcus aureus*, and *S. epidermidis*, viruses such as rotavirus, norovirus, astrovirus, cytomegalovirus, and echovirus, and fungi are some of the microorganisms (*Candida spp.*). The mediation of multifactorial dysbiosis and an altered intestinal barrier, as well as the immunological response, have been demonstrated to be important factors in NEC pathogenesis in a variety of organisms. Increased AMP and mucin production, decreased AMP synthesis, and impaired production of certain growth factors and cytokines are all characteristics of this mutation. Intestinal bacterial translocation, a targeted and systemic pro-inflammatory response, destruction and necrosis of the gut mucosa are the causes. When compared to infants, human prematurity itself is associated with a distinct BGM (Bacterial Gut Microbiota) development. Other prematurity-related variables, such as gastrointestinal and immune system immaturity, early antibiotic therapy, prolonged hospitalisation, mechanical ventilation, and parenteral nutrition, have an impact on this abnormal growth. Affected infants exhibit an evolutionary pattern distinct, with the class *Bacilli* initially leading and followed by the class Gammaproteobacteria, and then the class *Clostridia*. On a minor taxonomic level, the development is divided into four phases, with *Staphylococcus*, *Enterococcus*, *Enterobacter*, and finally *Bifidobacterium genera*, respectively.

When compared to healthy controls, preterm infants with NEC (Necrotizing Enterocolitis) have different patterns of BGM development, early-onset NEC have a higher abundance of *Clostridia*, while those with late-onset NEC have a higher abundance of Gammaproteobacteria (*E. coli and Shigella*). These modifications show the absence of a certain gut bacterial pattern that has been linked to NEC and varies with the age of infants that develop NEC. Metagenomic analysis indicates that identifying uropathogenic *E. coli* was a risk factor for both NEC and mortality. BGM in preterm infants developing NEC tended to be less heterogeneous from days 17 to 22 postpartum, with a higher abundance of *E. coli*.

Changes in BGM composition between children with NEC and controls were observed in cohorts of low birth weight infants (1500 gm), with higher proportions of Gammaproteobacteria and lower proportions of Negativicutes and the class *Clostridia*. Lower xenobiotic biodegradation and metabolic activity, indicating not only a modified composition but also a functionally modified microbiota.

Lactobacillus, *Phascolarctobacterium*, and *Streptococcus salivarius* were more prevalent in control mechanisms. Reduced xenobiotic detoxification by BGM in these newborns is either linked to an inflammatory response in the gut, similar to that which occurs in inflammatory bowel disease. Variations in the microbiota composition depending on the number of days of life when compared to preterm controls.

CONCLUSION

BGM composition of preterm infants who develop NEC is different, and is primarily characterised by a less diverse microbiota, enrichment in some components particularly Gammaproteobacteria, and a potential role for certain bacterial components (uropathogenic *E. coli*) in prognosis. Gram-negative bacteria are linked to early histological damage in mice models of hypoxia-induced NEC, whereas *Clostridia* and *Bifidobacteria* group produce SCFA (Short Chain Fatty Acids), which, in physiological proportions, protects against harm to enterocytes. NEC development is characterised by the identified BGM components in preterm infants.

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