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Literature Review

Current Evidence of Probiotics in Pediatrics with Short Bowel Syndrome, Is It Safe and Beneficial? A Literature Review

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Abstract:

Background: Short Bowel Syndrome (SBS) presents significant challenges in pediatric care, particularly due to its high incidence in neonates and the associated health burdens, including elevated mortality rates primarily from hepatic failure and sepsis. SBS in infants and young children primarily arises from congenital defects or acquired conditions that necessitate significant bowel resection. The predominant cause of SBS during the neonatal period is necrotizing enterocolitis (NEC), accounting for 35% to 50% of cases. In older children, SBS is frequently associated with midgut volvulus or traumatic injuries.

***Discussion*:** Managing pediatric SBS requires a multidisciplinary approach that involves evaluating dietary, pharmacology, and surgical factors. Key strategies focus on improving absorptive capacity, promoting intestinal adaptation, and regulating bowel motility. In infants and young children, SBS often leads to a range of complications, including nutrient deficiencies, fluid imbalances, and growth delays. The condition necessitates careful medical management to address these challenges and promote optimal health outcomes. Recent studies have investigated probiotics as an adjuvant treatment for SBS, demonstrating enhanced growth, nutritional status, and inhibition of harmful microbes in afflicted children

Conclusion: The use of probiotics in children with SBS has shown both safety and beneficial effects, making it a feasible alternative therapy in routine medical practice. However, the lack of significant clinical data highlights the need for more study to better understand the efficacy of probiotics in the treatment of SBS.

Keywords: children, intestinal failure, probiotics, short bowel syndrome

Introduction

Short bowel syndrome (SBS) is a condition characterized by malabsorption that arises after significant intestinal resection. In infants, SBS is the leading cause of Intestinal Failure. Intestinal failure (IF) is characterized by a severe reduction in functional intestinal mass, falling below the threshold required for sufficient digestion and absorption to meet the body's nutritional and fluid needs in adults or to support growth in children.[1, 2]

In infants and young children, SBS typically results from congenital defects or acquired conditions necessitating extensive bowel removal. Necrotizing enterocolitis (NEC) is the leading cause of SBS in the neonatal period, responsible for approximately 35%–50% of cases.[3] In older children, it most often develops due to midgut volvulus or traumatic injury. Other contributing factors include congenital abdominal wall defects such as gastroschisis and omphalocele, intestinal atresia, meconium ileus, Hirschsprung's disease, and abnormalities of the superior mesenteric artery.[4, 5]

Previous study from developed countries estimated the incidence at 22.1 per 1,000 neonatal intensive care unit (NICU) admissions in a tertiary care center, while the population-based incidence was 24.5 per 100,000 live births. Notably, only three out of 40 infants with SBS were born at term.[4] Approximately 80% of SBS cases in the pediatric population occur during the neonatal period.[5]

SBS imposes a substantial health burden. Four retrospective studies have reported a case fatality rate ranging from 27.5% to 37.5% over follow-up periods of 1.5 to 5 years.[4, 6-8] Hepatic failure was the leading cause of mortality, accounting for 60% of deaths, while sepsis contributed to 10%–20%. The incidence of sepsis is notably high and represents the primary reason for hospital readmission in SBS patients, leading to prolonged hospitalization and increased healthcare costs. [2, 4]

Lately, the use of probiotics has been widely used as an additional supplementation for several diseases including SBS. Several previous studies reported good clinical outcomes in children with SBS after being given probiotics in the form of improved growth and nutritional status, suppressing pathogenic bacteria and even two studies proved the success of therapy where conventional therapy failed.[9, 10]

This literature review will provide a detailed explanation of the efficacy and safety of probiotics as an adjunctive therapy in children with SBS. By examining several studies that evaluate these outcomes, this review aims to explore the potential of probiotics as an alternative therapeutic approach in routine clinical practice.

Short Bowel Syndrome

Definition

Short bowel syndrome (SBS) is the condition characterized by a shortened length of the small intestine. In pediatrics, the definition of SBS is more precise, requiring less than 25% of the remaining small bowel for the gestational age.[11]

Complications Related to SBS

The severity of malabsorption in SBS is influenced by the reduction of the intestinal absorptive surface. Malabsorption can lead to deficiencies of essential nutrients that are crucial for children, particularly during the first two years of their life. Consequently, children with SBS are at risk of growth failure.[12] After extensive bowel loss, the gastrointestinal tract undergoes a process of adaptation that can begin as soon as 48 hours post-surgery and may persist for as long as 18 months. This process aims to enhance the absorption of essential nutrients and fluids, thereby restoring some of the residual functionality.[13, 14]

SBS is associated with higher morbidity and mortality in infants. Casaccia et al. indicated that infants with SBS are more susceptible to sepsis and motor developmental delays. The mortality rate in newborns with SBS was shown to be greater than in infants without SBS (16% vs 4%).[15] Numerous patients with short bowel SBS undergo their first small bowel resection during the neonatal period, a phase characterized by considerable intestinal microbial transition and susceptibility.[12, 16]

Gut bacteria perform multiple roles, including the process of digestion, vitamin production, the control of the immune system through metabolites, the breakdown of lipids, and defense against pathogens. However, children with SBS exhibit changes in the normal intestinal microbiota, leading to diverse clinical outcomes.[16] Analyses of the fecal microbiome of SBS patients show an increased of *Proctobacteria*, which are recognized as pro-inflammatory.[17]

Small-intestinal bacterial overgrowth (SIBO) frequently occurs in individuals with SBS and is linked to major health issues. Clinical manifestations of bacterial overgrowth include abdominal pain, loss of appetite, vomiting, diarrhea, cramps, and metabolic acidosis.[18, 19] Regardless of the cause, SIBO can result in classic problems known as D-lactic acidosis.[16] This condition arises from the body's inability to digest carbohydrates, leading to their delivery to intestinal bacteria and the production of lactic acid L and D. However, only the L-lactic acid was absorbed and metabolized. Consequently, the D-lactic acid accumulates and can lead to altered mental status.[13]

Another complications in SBS related to parenteral nutrition (PN). While PN is a critical and life-saving method for nutritional management, its prolonged use can result in a range of hepatic complications. These may include cholestasis, steatosis, fibrosis, and cirrhosis, which can further lead to portal hypertension and coagulopathy.[20] The liver of neonates is particularly susceptible to pathophysiological challenges caused by factors such as infections, disturbances in the gut-liver axis, and the abnormal administration of parenteral nutrients. These vulnerabilities increase the risk of developing intestinal failure-associated liver disease (IFALD).[21] To prevent IFALD, various management strategies can be applied, including reducing lipid intake or modifying the lipid composition, avoiding continuous PN and overfeeding, also implementing measures to prevent infections.[13, 20]

Children with SBS often require long-term PN, which is associated with a significant risk of catheter-related bloodstream infections (CRBSIs). This complication can arise from several factors, including contamination and inadequate care of the catheter, contamination of the hub, or infections at the exit site that allow bacteria to migrate into the catheter. However, these infections are predominantly linked to the use of parenteral nutrition PN during the initial month of life.[22]

Management

The management of pediatric short bowel syndrome is complex, requiring multidisciplinary collaboration for optimal support.[14] The management include a comprehensive assessment of nutritional, medical, and surgical factors.[23] Pharmacological and nutritional treatments are categorized based on their efficacy in enhancing absorptive capacity, facilitating intestinal adaptation, and regulating bowel motility, which are the three primary techniques utilized in the management of SBS.[24]

The acute phase of SBS begins right after surgical resection and typically lasts for three to four weeks. During this period, patients experience significant enteric fluid losses, leading to a metabolic imbalance. Consequently, it is crucial to closely monitor the patient's total output.[16] The initial management during the first few months following resection focuses on sustaining volume status, avoiding electrolyte imbalances, and ensuring nutritional support through PN.[17]

PN may be administered for extended periods, sometimes spanning several years, and in certain cases, children may require PN for their entire lives. The specific fluid requirements for each patient can differ significantly based on various factors, including the patient's age, anatomical considerations, the volume of intestinal output, and other relevant conditions.[14] However, even with comprehensive PN support, achieving optimal linear growth remains challenging.[15]

Whenever feasible, nutrients should be administered orally to encourage oral motor function and prevent the development of feeding aversion behaviors. For initial feeding, either breast milk or a standard polymeric formula is recommended. Feeding volumes should be incrementally increased based on the patient's tolerance. Tolerance is assessed by monitoring the frequency and volume of stools, as well as observing for signs of vomiting, irritability, and both abdominal and intestinal distension.[13]

Probiotic

Definition

Probiotics are defined as "live microorganisms that, when administered in adequate amounts, provide health benefits to the host." The term "probiotic" originated in the 1960s and is derived from a Greek word meaning "for life." Although the terminology is relatively recent, the health benefits associated with consuming foods containing live bacteria have been acknowledged for centuries.[23]

Mechanism Probiotics may Benefit Children with SBS

1. Role in Intestinal Maturation and Adaptation

The importance of gut commensal organisms in intestinal maturation has been well established through studies on germ-free animals, which exhibited reduced mucosal cell turnover, enzyme activity, local cytokine production, mucosa-associated lymphoid tissue development, lamina propria cellularity, vascularization, muscle wall thickness, and motility.[16] The intestinal microbiota play a crucial role in regulating the expression of genes associated with various intestinal functions, including nutrient absorption, mucosal barrier integrity, metabolism, angiogenesis, and overall intestinal maturation. Probiotics may contribute to these processes by promoting intestinal adaptation in children with SBS.[24, 25]

Previous studies in animals indicate that the reestablishment of a healthy microbiota occurs rapidly following antibiotic therapy when supplemented with probiotics. By promoting the colonization of normal commensal bacteria, probiotics may facilitate gut maturation in infants with SBS, who are frequently exposed to antibiotics.[26]

Short-chain fatty acids (SCFAs), produced through the fermentation of carbohydrates and soluble fiber by probiotics which play a trophic role in intestinal adaptation. They mitigate ileal mucosal atrophy associated with total parenteral nutrition (TPN), enhance mucosal epithelial cell proliferation, and reduce apoptosis. Additionally, *Lactobacillus rhamnosus GG (*LGG) has been shown to secrete soluble proteins that stimulate the growth of intestinal epithelial cells and protect against cytokine-induced apoptosis.[24, 27]

2. Enhancement of Intestinal Barrier Function

Pathogenic bacteria can compromise intestinal permeability by disrupting tight junctions, which, when combined with impaired mucosal immunity, may facilitate bacterial translocation and increase the risk of sepsis. Multiple studies have demonstrated that probiotics contribute to strengthening the mucosal barrier through various mechanisms. These include adherence to the intestinal lining, competitive inhibition of pathogenic bacteria by preventing their attachment, and secretion of factors that enhance barrier integrity.[24, 27, 28]

Additionally, probiotics exert immunomodulatory effects, support gut epithelial tight junctions by improving the expression of occludin and claudin, and promote the production of mucin, zona occludens, and cytoprotective heat shock proteins, all of which contribute to maintaining gut barrier function.[28]

3. Suppression of Pathogens

Probiotics contribute to colonization resistance by competing with pathogenic bacteria for both nutrients and adhesion sites while also producing antimicrobial compounds. Their antibacterial properties play a crucial role in managing SIBO.[12,19,26] Probiotics or their components stimulate the secretion of antibacterial peptides, such as defensins, from intestinal epithelial and Paneth cells, which exhibit broad-spectrum antimicrobial activity against bacteria, fungi, and viruses. Certain probiotic strains, including *Lactobacilli* and *Bifidobacterium*, can inhibit or directly eliminate pathogenic bacteria by producing antimicrobial substances such as SCFA, acetate, and lactate. These compounds lower luminal pH, creating an environment that suppresses pathogen growth.[29, 30]

Additionally, probiotics produce bacteriocins that target bacterial cell membranes, and *Bifidobacterium* has been shown to secrete an unidentified antimicrobial molecule effective against *Escherichia coli, Klebsiella pneumoniae, Yersinia pseudotuberculosis, Staphylococcus aureus,* and *Salmonella typhimurium.*[31, 32] Furthermore, probiotics help restore microbial balance following antibiotic use, thereby reducing the incidence of antibiotic-associated diarrhea, which is often caused by the overgrowth of pathogenic bacteria such as *Clostridium difficile.*[28]

4. Immune Modulating Effects

Lactobacilli and *Bifidobacteria* stimulate the production of both total and pathogen-specific IgA in the intestinal mucosa without inducing probiotic-specific IgA.[28] *Lactobacillus casei Shirota* has been reported to enhance the activity of natural killer cells. In neonatal rat models, treatment with LGG has been shown to suppress the

production of proinflammatory cytokines in response to bacterial lipopolysaccharide (LPS) across multiple sites, including the intestine, liver, plasma, and lungs.[24]

Additionally, LGG administration partially mitigated LPS-induced pre-necrotic alterations in the intestinal mucosa. This probiotic-mediated effect is regulated via the TLR9 receptor, which suppresses the activation of inflammatory genes. By modulating gut inflammation associated with SIBO in SBS, probiotics may contribute to improved feed tolerance and provide hepatoprotective benefits.[18, 33]

Efficacy of Probiotics in SBS from Case Reports Perspective

Only a limited number of clinical studies have been identified that specifically address the use of probiotics in the context of SBS. Vanderhoof et al. reported 2 patient with SBS undergoing probiotic treatment. The first patient was diagnosed with SIBO using *Lactobacillus plantarum 299V* once a day. After 2 to 3 weeks of probiotic treatment, there was a noticeable improvement in stool consistency, particularly characterized by a reduction in water content. The second patient, probiotic therapy was initiated with *Lactobacillus plantarum 299V*. After several weeks of treatment, both antibiotic therapy and intravenous nutrition were discontinued, along with the medication regimen for arthritis.[34]

Candy et al. reported the administration of *L. casei Shirota* to a child with SBS resulted in a significant improvement in sodium balance.[35] Other study involving four pediatric patients with SBS receiving probiotics therapy. Three months after starting synbiotics therapy, there was a significant improvement in the bacterial flora, with increased counts of *Bifidobacterium*, facultative anaerobic bacteria, *Enterobacteriaceae*, and *Lactobacillus* in all patients, along with notable growth of other *Bifidobacteria* and *Lactobacilli* species.[9]

Kanamori et al. described the treatment of a two-year-old patient with SBS using *Bifidobacterium breve* Yakult, *Lactobacillus casei* Shirota, and galactooligosaccharides over a two-year duration. This treatment resulted in a marked improvement in both intestinal motility and absorptive capacity. Furthermore, there was a reduction in the levels of *E. coli* and *Candida*, along with a decrease in the ratio of facultative anaerobic bacteria to total bacteria.[36]

Kanamori et al. also reported the long-term use of synbiotic therapy composed of *Bifidobacterium breve, Lactobacillus casei,* and galactooligosaccharides in 7 SBS patients with refractory enterocolitis. The probiotics enhanced the intestinal bacterial flora by promoting the dominance of anaerobic bacteria and suppressing the presence of pathogenic bacteria, while also increasing the levels of short-chain fatty acids in the feces, rising from 27.8 to 65.09 μmol/g of wet feces.[10]

Safety Profile of Probiotics in SBS

Kunz et al. reported two cases of sepsis related to LGG supplementation is SBS patient. While a primary intravenous line sepsis caused by Lactobacillus cannot be definitively ruled out, this study hypothesize that the gastrointestinal tract was the most probable portal of entry. This speculation is based on the fact that proper line care protocols were being followed, and the Lactobacillus was administered at a considerable distance from the intravenous insertion sites. This case was similar from a case reported by De Groote et al. that described a case of bacteremia after ingestion of LGG.[37, 38] Another complication associated with probiotic supplementation is D-lactic acidosis. This issue has been documented in two studies following the use of *Lactobacillus acidophilus* and *Bifidobacterium infantis*, with symptoms resolving after the probiotics were discontinued.[39, 40]

Conclusion

Probiotics present significant benefits for children with SSBS, including enhancing intestinal maturation, improving barrier function, and modulating immune responses, which aid in nutrient absorption and infection prevention. Although generally safe, with rare complications like sepsis and D-lactic acidosis, the evidence supporting their use is limited, primarily comprising small studies and case reports. This lack of data emphasizes the necessity for more extensive clinical studies to thoroughly evaluate the efficacy and safety of probiotics in pediatric patients with SBS.

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Conflict of Interest

None declared.

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