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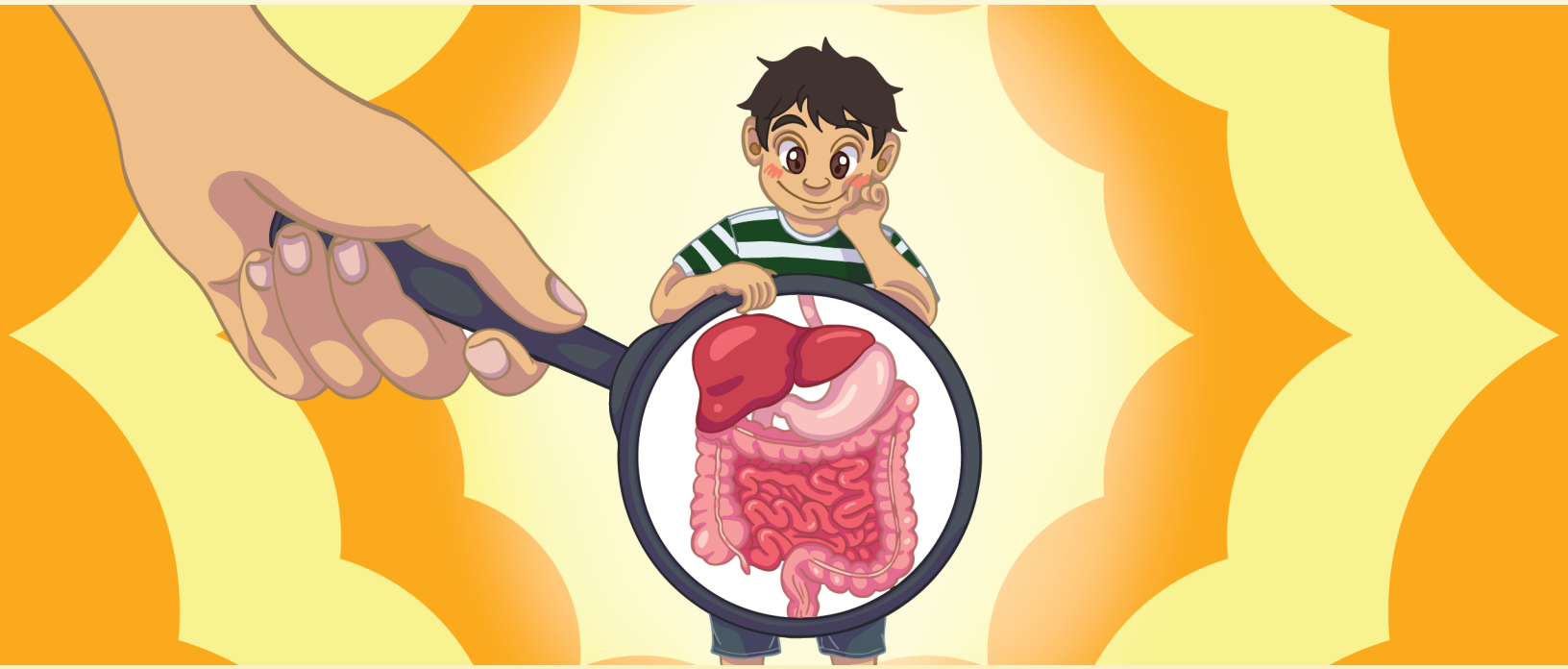

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APGHN Archives of Pediatric Gastroenterology, Hepatology, and Nutrition

Original Article

Pediatrics Upper Gastrointestinal Endoscopy Profile in Riau in 2020 and 2021

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Putra DS. Pediatrics upper gastrointestinal endoscopy profile in riau in 2020 and 2021. *Arch Pediatr Gastr Hepatol Nutr*. 2025;4(1).1-7

Abstract:

Background: Children's limited communication skills when expressing complaints often limit healthcare practitioners in making diagnoses and treatment decisions, particularly regarding gastrointestinal issues. Endoscopy is a valuable tool for determining the appropriate course of action in these cases. This study aims to describe the characteristics of pediatric patients who underwent upper gastrointestinal endoscopic procedures in Riau between 2020 and 2021.

***Methods*:** This retrospective study used secondary data from endoscopy registries and patient medical records at two tertiary hospitals in Riau between Januari 2020 to December 2021.

***Results*:** A total of 114 patients underwent upper gastrointestinal endoscopy from 2020 to 2021. It was found that the adolescent age group (42%) was the most frequently encountered group. The female sex (65%) was more prevalent than male. Meanwhile, the most common problem encountered was abdominal pain (57%). Gastroscopy alone was the most frequently performed procedure in this study (65%), with the most common endoscopic finding being hyperemic gastritis (54%).

Conclusion: Upper gastrointestinal endoscopy in pediatric patients is a safe and very helpful procedure in diagnostics and therapeutics.

Keywords: abdominal pain, endoscopy, gastritis, gastroscopy, upper gastrointestinal tract

Introduction

Gastrointestinal endoscopy has become an essential component of the diagnosis and treatment of pediatrics gastroenterology. Upper gastrointestinal endoscopy is one of the procedures that can be performed in establishing the diagnosis of gastrointestinal diseases in pediatric cases. Gastrointestinal endoscopy is a recommended procedure for diagnosing gastrointestinal (GI) disorders in children.[1] Advances in pediatric endoscopy have enabled more accurate diagnoses of GI diseases. The diagnostic indications for pediatric endoscopy differ from those in adults, with chronic abdominal pain is the most common indication for gastrointestinal endoscopy in pediatric patients.[1, 2]

While certain situations, such as substantial upper GI bleeding, are considered clear indications for GI endoscopy, the appropriate application of GI endoscopy for various clinical situations has not been formally established, particularly in Indonesia. The absence of clear guidelines for determining when to perform endoscopy has led to procedures being performed at inappropriate times, potentially causing discomfort for patients, especially children.[2]

There are several considerations when performing an upper GI endoscopy during the COVID pandemic. First, pediatric endoscopic procedures are considered to carry a high risk of COVID-19 transmission. Furthermore, parents are often hesitant and anxious about bringing their children to hospitals, let alone to do GI endoscopy that frequently requires sedation. Therefore, careful consideration of whether to proceed with a pediatric GI endoscopy is essential.[3]

The purpose of this study is to describe the characteristics of pediatric patients undergoing upper gastrointestinal endoscopic procedures during the COVID-19 pandemic in Riau (2020 to 2021). We also aim to evaluate the indications for and findings from the upper GI endoscopic procedures we performed. Determining the diagnostic indications of endoscopy will help clinicians make more informed decisions about which patients will benefit most from this procedure.

Methods

Study Population and Study Design

Between January 2020 and December 2021, a total of 114 patients underwent upper gastrointestinal endoscopy. These procedures were performed at Syarifa Hospital and Arifin Achmad General Hospital in Pekanbaru, with various indications and types of endoscopic interventions. These two hospitals are the only tertiary care facilities in Riau province that have endoscopy facilities for pediatric patients. Endoscopy registries and patient medical records were reviewed, and the following variables were recorded: age, gender, type of procedure, indication for the procedure, and diagnosis based on endoscopic findings. Patients younger than 1 year and older than 18 years

were excluded. Ethical clearance was obtained from the health research ethics committees at both hospitals.

Data Collection

All upper gastrointestinal procedures were performed by a pediatric gastroenterologist with the assistance of anesthesiologist, given the use of deep sedation during the procedure. All endoscopic findings were recorded, including incidental findings even if unrelated to the presenting symptoms. These findings were then evaluated by the pediatric gastroenterologist who performed the procedure. Endoscopic findings were considered significant or positive if they had diagnostic or prognostic value. This was defined as a reasonable explanation for the reported symptoms and/or findings that change in management. If no endoscopic findings were found and/or no intervention was required, the findings were classified as negative or normal.

Furthermore, non-specific minor endoscopic findings, such as minor erythema, minor increased or decreased vascularity, or mild pallor were considered normal if not accompanied by significant histological changes. Similarly, minor non-specific histological abnormalities not related to the symptoms were also accepted as normal.

Results

Table 1 shows the characteristics of the patients undergoing GI endoscopy. We divided the age groups into three categories. The adolescent age group (12 – 18 years) was the most common age group in this study, representing 42% of the cases. Of the one hundred and fourteen patients, 65% of them were female (n=74).

Table 1. Characteristics of pediatric patients undergoing upper GI endoscopy.

Characteristics of Patients	Frequency (n) n = 114	Percentage (%) n = 100
Age groups		
1 – 5 years old	19	17
6 – 11 years old	47	41
12 – 18 years old	48	42
Gender		
Male	40	35
Female	74	65

Table 2 presents the characteristics of endoscopy, including indications for upper GI endoscopy in pediatric patients, the corresponding types of procedures performed, and resulting diagnoses. Abdominal pain was the most common indication for upper GI endoscopy (57%), with gastroscopy alone being the most frequent procedure performed (65%). A variety of diagnoses were observed during upper gastrointestinal

endoscopy. Hyperemic gastritis (54%) was the most common diagnosis, followed by esophageal varices (16%), foreign body (25%), peptic ulcer (6%), and gastric tumor (1%).

Table 2. Characteristics of upper GI endoscopy performed.

Characteristics of Endoscopy	Frequency (n) n = 114	Percentage (%) n = 100
Endoscopy Indication		
Abdominal pain	65	57
Hematemesis (bloody vomit)	24	21
Foreign object ingestion	25	22
Types of Endoscopy		
Gastroscopy	74	65
Gastroscopy with extraction	25	22
Gastroscopy with ligation	15	13
Endoscopy Findings		
Normal	4	4
Hyperemic gastritis	62	54
Foreign body	25	22
Esophageal varices	16	14
Peptic ulcer	6	5
Gastric tumor	1	1

Discussion

The adolescent age group (42%) and the female group (65%) were the most common groups in this study. This may be because in this age group, children have already begun to express their physical complaints, including complaints of the digestive tract. Whereas younger children, such as toddlers, may have difficulty expressing the discomfort they feel related to their stomach or digestive system in general.

Gastroscopy (65%) was the most common type of procedure performed in this study. This is in line with the most frequent indication, which is abdominal pain (57%). Recurrent abdominal pain is one of the most common complaints in children, and persistent abdominal pain despite various medical treatments is a strong indication for GI endoscopy.[4, 5] These findings are similar to a study by Fachler et al., which found that abdominal pain was the main indication for GI endoscopy at Shaare Zedek Medical Center, Jerusalem.[4]

Diagnosing the cause of abdominal pain in children is difficult for clinicians because children, especially young children, are usually difficult to communicate the pain they feel and the characteristics that accompany it. Therefore, diagnostic tools such as endoscopy are needed to determine the etiology or cause of recurrent abdominal pain

in children. However, endoscopists should be aware that chronic abdominal pain does not have an organic cause in more than half of cases. A pediatric endoscopist should refrain from performing unnecessary endoscopies in children.

In our study, hyperemic gastritis (54%) was the most frequently diagnosed condition. This is consistent with the most common indication for endoscopy in our study, which is abdominal pain. These results are also in line with a study conducted by Isa HM et al., which found that gastritis was the most common organic lesion found in children undergoing GI endoscopy in tertiary care in Bahrain.[6]

The role of endoscopy as a diagnostic tool goes beyond the detection of organic lesions. Negative results or normal findings are also important to reassure parents, confirm a functional etiology, or indicate further investigation. Normal or negative endoscopy results in this study were 4%, quite low compared to the findings reported by Isa HM et al. which revealed 21.3% negative results.[6] This low number suggests that most GI endoscopy procedures performed in our two institutions were selective and performed according to indications.

Twenty-two percent of cases (n=25) in our institution were cases of foreign body ingestion, for which we performed foreign body extraction gastroscopy. In children, foreign bodies in the esophagus are an absolute emergency, and endoscopy should be performed immediately, regardless of whether X-rays are available as confirmation. Common symptoms often found include difficulty swallowing, hypersalivation, and even coughing.[7]

Endoscopy is a high-risk procedure for COVID-19 transmission.[8] This study was conducted during the COVID-19 pandemic, leading to stricter indications for performing endoscopic procedures. A multicentre study in the Asia Pacific region found that for COVID-19 confirmed patient, 63% of institutions performed endoscopic procedures only in emergency cases, while 34.9% were either postponed or not performed at all.[9] Our study demonstrated that 43% of cases involved urgent indications, including hematemesis and foreign body ingestion. Other studies have reported a decline in endoscopic procedures during the pandemic; however, as this study did not include a comparison with pre-pandemic data and no prior data before COVID-19 was reported, we cannot evaluate the impact of the pandemic in this study.[1]

The limitation of this study is the lack of more complete data to support the diagnosis, such as biopsy results. The diagnosis is determined only from the findings on endoscopy, without further intervention such as tissue biopsy for examination by a pathologist. Further studies with biopsies are recommended to determine the exact

pathology of certain endoscopic findings, such as masses or tumors. Determining the exact etiology of a disease entity will result in more appropriate treatment.

Upper gastrointestinal endoscopy in pediatric patients is a very helpful and safe procedure for diagnostic and therapeutic purposes. Patient selection is very important in the indications for performing upper gastrointestinal endoscopy procedures in children. Further research may be needed to determine the algorithm for upper gastrointestinal endoscopy in children.

Conclusion

Upper gastrointestinal endoscopy in pediatric patients is a helpful and safe procedure for diagnostic and therapeutic purposes. Appropriate patient selection is essential for determining the indications for upper gastrointestinal endoscopy in children. Further research is needed to establish an algorithm for patient selection for this procedure.

Conflict of Interest

None declared

Funding Statement

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Original Article

Impact of Probiotics or Synbiotics Supplementation on Weight Gain and Diarrhea in Children with Severe Acute Malnutrition: A Systematic Review and Meta-Analysis

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Sabrina H, Tsania NM, Prasadajudio M. Impact of probiotics or synbiotics supplementation on weight gain and diarrhea in children with severe acute malnutrition: a systematic review and meta-analysis. *Arch Pediatr Gastr Hepatol Nutr.* 2023;4(1).8-22

Abstract:

Background: Malnutrition remains a critical global health concern, with both short- and long-term consequences. Children suffering from malnutrition frequently exhibit gut dysfunction, which leads to growth retardation, impaired absorption of essential nutrients and vitamins, and immune dysfunction. Diarrhea is one of the most common conditions in children with malnutrition and can further worsen their condition. Probiotics have been proposed as a potential adjunctive therapy in malnutrition due to their role in modulating gut microbiota. This study aims to evaluate the effects of probiotics on weight gain and diarrhea specifically in children with severe acute malnutrition (SAM).

Methods: A systematic literature search was conducted across six databases (PubMed, Cochrane Library, ProQuest, EBSCOhost, ScienceDirect, Google Scholar) using relevant keywords. Data were extracted and analyzed using Review Manager for meta-analysis.

Result: Four studies, encompassing a total 1662 patient met the inclusion criteria. Probiotics are proven to reduce significantly the duration of diarrhea and improve the recovery (SMD -0.70; 95% CI -0.89 to -0.50; $I^2 = 0\%$, $p < 0.00001$). However, they are not diminishing the incidence of diarrhea. Moreover, this study concluded that the use of probiotics or synbiotics did not significantly impact weight gain.

Conclusion: Probiotics demonstrated efficacy in reducing the duration of diarrhea, but not its incidence, potentially contributing to improved recovery outcomes. However, their impact on weight gain in children with SAM remains inconclusive. Further research with larger studies is warranted to identify factors influencing probiotic efficacy and to explore their potential role in the comprehensive management of SAM.

Keyword: diarrhea, probiotics, severe acute malnutrition, synbiotics, weight gain

Introduction

Severe acute malnutrition (SAM), as defined by the World Health Organization, is characterized by one or more of the following: a weight-for-height z-score (WHZ) below -3 standard deviations, a mid-upper arm circumference (MUAC) less than 11.5 mm in children aged 6-59 months, or the presence of bilateral pitting edema.[1] SAM arises from a complex interplay of factors, including inadequate food consumption and chronic infections.[2] This condition significantly increases morbidity and mortality in children, with long-term consequences such as impaired cognitive development, metabolic disorders, and diminished adult potential.[3]

Globally, SAM affects an estimated 18.7 million children, with a disproportionate burden in low- and middle-income countries (LMICs).[1] Malnutrition remains a significant public health challenge in Indonesia. Based on World Health Organization (WHO) criteria and Indonesian population data from 2017, the estimated number of children under five with SAM was approximately 805,000 in that year.[3] According to the 2022 Indonesian Nutritional Status Survey, the prevalence of wasting (low weight-for-height) in Indonesia was 7.7%, an increase from 7.1% in 2021.[4]

The management of SAM consist of emergency stabilization, correction of electrolyte imbalances, infections management if present, and the provision of therapeutic feeding to promote catch-up growth.[3] However, therapeutic food interventions have limitations, such as the potential for relapse and incomplete recovery. Notably, evidence shows that the gut microbiome plays a crucial role in the recovery process.[5]

Probiotic administration has been observed to advance weight gain through the modulation of gut microbiota.[6] When administered in sufficient amounts, probiotics may provide health benefits to the host.[7] The most utilized microorganism include *Lactobacillus* and *Bifidobacterium* in probiotic formulations. Probiotics benefit the host by modulating the host's immune system, preventing pathogen adhesion to the intestinal epithelium, and improving nutrient absorption.[8]

Prebiotics are a group of nutrients that are fermented by gut microorganisms but are not digested. These indigestible compounds produce beneficial physiological effects for the host, such as promoting the growth of native bacteria and encouraging the production of short-chain fatty acids. These short-chain fatty acids, in turn, inhibit the growth of pathogenic microorganisms, thereby strengthening the body's defenses.[9]

Furthermore, the combined use of probiotics and prebiotics, known as symbiotic, result in a synergistic effect that, amplifies their positive effect. A systematic review by Mugambi et al. demonstrated that synbiotic supplementation has a positive impact on child developmental anthropometric indicators. [10]

Diarrhea is a prevalent condition among children with malnutrition, potentially delaying recovery and increasing the risk of mortality. Therefore, effective diarrhea management is essential. Probiotics have been shown to be safe and demonstrate clear benefits in reducing the duration and severity of diarrhea in pediatric.[11, 12]

Previous evidence suggests that probiotic, prebiotic, and synbiotic interventions can promote weight gain and diarrhea in malnourished children.[13, 14] However, the effects of probiotic, prebiotic, or synbiotic administration specifically in children with SAM remain understudied. Therefore, this systematic review and meta-analysis synthesizes existing evidence to assess their potential on weight gain and diarrhea in SAM children.

Method

Data Source and Search Strategy

A comprehensive literature search was conducted according to Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) flowchart. Literature search was initiated across six databases (PubMed, Cochrane Library, ProQuest, EBSCOhost, ScienceDirect, and Google Scholar). The search strategy utilized a combination of keywords such as "Severe Acute Malnutrition," "Children," "Probiotic," and "Weight Gain," combined with Boolean operators (AND, OR) to refine the search results. **Table 1** details the specific search queries for each database. Articles were independently screened by authors to remove duplicates and identify relevant studies, which were then compiled into a spreadsheet for further review.

Data Extraction

A standard data extraction was created using Microsoft Sheets with multiple reviewers independently pulling information from four randomized clinical trials (RCTs). For each study, key details were recorded, including the first author, publication year, study design, participant demographics, setting, age range, intervention specifics (e.g., probiotic strain, dosage), control group (e.g., placebo, standard care), intervention duration, follow-up period, and outcomes like diarrhea duration, weight gain, and hospitalization rates. Additional data on breastfeeding, antibiotic use, and HIV status were collected to account for potential confounders.

Studies were included if they met specific criteria: had to be randomized clinical trials involving severe acute malnutrition participants, clearly describe probiotic interventions, and report relevant outcomes such as diarrhea duration or weight gain. Studies were excluded if they were not randomized, lacked a control group, involved animal models, full text did not accessible, or did not provide sufficient data on the outcomes of interest. Additionally, studies with unclear methods or those not published in peer-reviewed sources were left out. This thorough and structured approach helped ensure the accuracy and reliability of the data for further analysis.

Data Synthesis

A meta-analysis was performed to synthesize the results of the included studies, focusing on comparable outcomes such as weight gain, and diarrhea duration. This statistical approach enabled a quantitative summary of the overall effect size and assessed heterogeneity across the studies. A random-effects model was employed for continuous meta-analysis, using standard mean difference as the effect size to account for variability in study designs and populations. The findings were visualized through forest plots, providing a clear representation of the pooled effect estimates and the degree of consistency among the studies. This synthesis aimed to evaluate the efficacy of prebiotic, probiotic, and synbiotic interventions in improving outcomes for children with severe acute malnutrition (SAM), while accounting for potential cofounding factors such as antibiotic use, HIV status, and breastfeeding practices.

Table 1. Search queries and first-hit results of each database

Database	Keyword	First-Hits Articles
PubMed	("Severe Acute Malnutrition" OR "SAM") AND ("Children" OR "Infant" OR "Child") AND ("Probiotics" OR "Probiotic Supplementation") AND ("Growth" OR "Weight Gain" OR "Anthropometry" OR "Growth Indices")	7
Cochrane Library	(("Severe Acute Malnutrition" OR "SAM") AND ("Children" OR "Infant" OR "Child")) AND (("Probiotics" OR "Probiotic Supplementation") AND ("Growth" OR "Weight Gain" OR "Anthropometry" OR "Growth Indices"))	18
ProQuest	(("Severe Acute Malnutrition" OR "SAM") AND ("Children" OR "Infant" OR "Child")) AND (("Probiotics" OR "Probiotic Supplementation") AND ("Growth" OR "Weight Gain" OR "Anthropometry" OR "Growth Indices"))	4660
EBSCOhost	("Severe Acute Malnutrition" OR "SAM" OR "Malnutrition, Severe Acute") AND ("Probiotic Supplementation" OR "Probiotics" OR "Probiotic Therapy") AND ("Growth" OR "Weight Gain" OR "Anthropometry" OR "Growth Indices") AND ("Children" OR "Pediatric" OR "Child" OR "Infant")	9

ScienceDirect	("Severe Acute Malnutrition" OR "SAM") AND "Probiotics" AND ("Growth" OR "Weight Gain") AND "Children"	142
Google Scholar	"prebiotics" "severe acute malnutrition" "children" "randomized controlled trial"	323

Quality Assessment

The quality of the included studies was assessed using the Risk of Bias for RCT tool. The quality assessment was conducted independently by all reviewers, with disagreements resolved through consensus deliberation.

Result

Population and Study Characteristic

A total of 5159 studies were identified on initial search from 6 databases. Nine duplicate records were removed, and 3521 articles were disqualified as they are ineligible records from automation tools. Other 1612 titles/abstracts were excluded because they did not match the study questions. Six articles were excluded due to the unavailability of articles and additional 7 articles were further excluded after full text reading because the outcome is not relevant to this study. At last, 4 studies were included and analyzed in this study (**Figure 1**). The quality of each study was assessed using Risk of Bias. Based on the result, we found that all studies exhibited low risk of bias (**Figure 2).**

The characteristics of studies included are summarized in **Table 2**. Among the total of 1662 participants, 820 patients received probiotics, 23 received synbiotic, and 819 received placebo. Three studies were conducted in both inpatient and outpatient settings, while 1 study was limited to outpatient participants. Multiple strains of probiotics were also utilized, with *Bifidobacterium* and *Lactobacillus* being the most frequently used genera in this study.

Two of the studies also reported the breastfeeding status of the participants in their study. Nuzhat et al. reported that the breastmilk intake for each study was 6.42% in the probiotic group, 4.92% in the placebo group, and 0% in the synbiotic group. Kambale et al. reported higher prevalence of breastfeeding, with 142 patients in probiotics (71.0%) and 46 patients in placebo (73.0%) being breastfed. [15, 16]

Furthermore, three of the studies administered probiotics during antibiotic treatment. Grenov et al. administered antibiotics as part of standard treatment for a minimum 5 days, with ampicillin and gentamicin as first-line antibiotics, and chloramphenicol,

ceftriaxone, cloxacillin, and ciprofloxacin were used as second- and third- line antibiotics. All patients in Kerac et al. received cotrimoxazole, and 50% of the participants had other types of parenteral antibiotics. Kambale et al used amoxicillin as a 5-days course of treatment in their study. Meanwhile, Nuzhat et al. administered the probiotics after completion of antibiotic treatment. [15-18]

Three of the studies also included HIV status in their studies. Kambale et al. reported that all the children enrolled in their study were HIV negative. Meanwhile, Grenov et al. and Kerac et al. revealed that the HIV positivity rates between their study participants were 14% and 95%, respectively.[16-18]

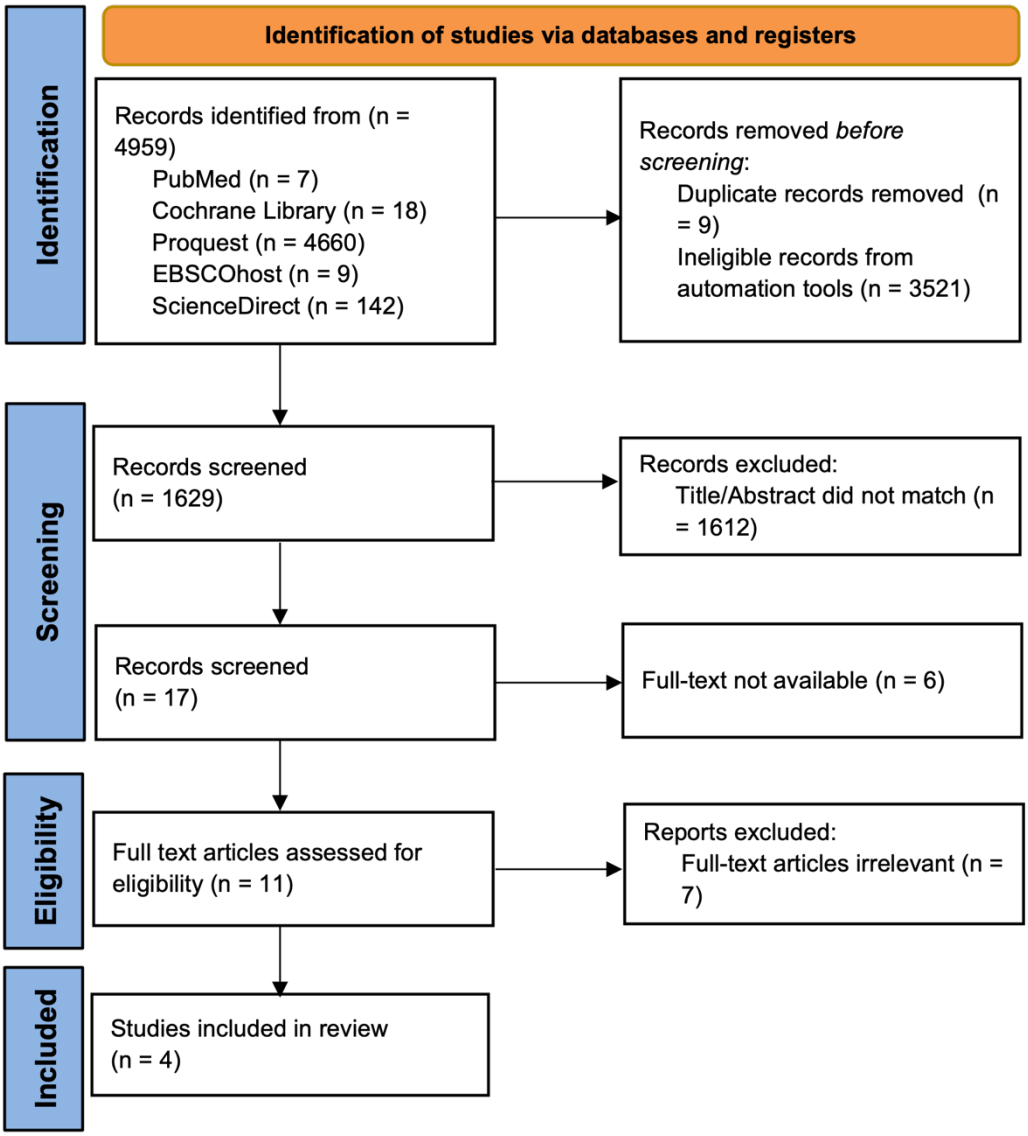

Figure 1. PRISMA Diagram

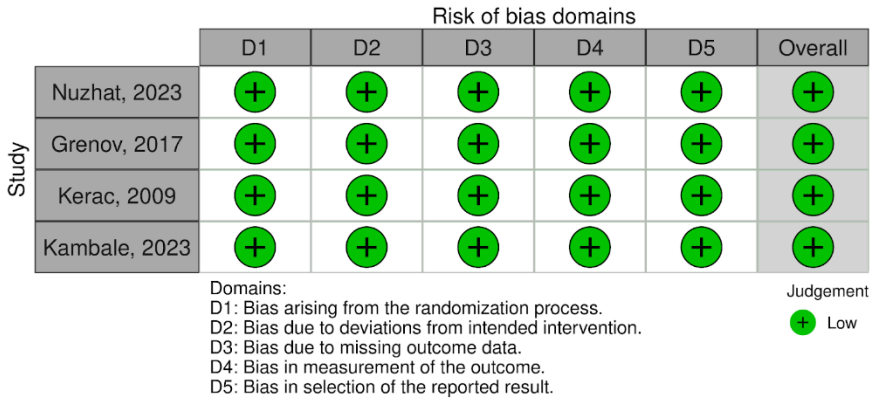

Figure 2. Risk of Bias Results

Table 2. Characteristics of included studies

Author, year	Study Design	Study Population	Age (mo)	Intervention	Control	Duration	Follow up	Outcomes
Nuzhat et al., 2023[15]	Single-blind RCT	Probiotic (21) Synbiotic (23) Placebo (23)	2-6	Probiotic: B. infantis EVC001 Synbiotic: (B. infantis EVC001, 8 billion CFU/day) + Prebiotic (Lacto-N-neotetraose - LNnT)	Lactose	28 days	4 weeks post supplementation	1.Hospitalization 2.Rate weight gain 3.Duration of diarrhea
Grenov et al., 2017[17]	Double-blind RCT	Probiotic (200) Placebo (200)	6-59	Bifidobacterium animalis subsp lactis Lactobacillus rhamnosus	Maltodextrin	During hospitalization followed by an 8- to 12-weeks outpatient treatment period	Depend on patients recovery rate	1.Incidence of diarrhea 2. Pneumonia 3.Weight gain 4.Recovery 5.Hospitalization 6.Fever 7.Vomit
Kerac et al., 2009[18]	Double-blind RCT	Probiotic (399)	5-168	Pediococcus pentosaceus	No placebo (RUTF	Depend on patients	Until nutritional	1.Nutritional cure 2.Death

		Placebo (396)		16:1 LMG P-20608, Leuconostoc mesenteroides 23-77:1 LMG P-20607, Lactobacillus paracaseissp paracasei F-19 LMG P-17806, Lactobacillus plantarum 2362 LMG P-20606) and 4 prebiotic-fermentable bioactive fibres (2·5 g of each per 10^{11} bacteria) (oat bran [rich in β-glucans], inulin, pectin, and resistant starch).	without any placebo supple-menta-tion	reco-very rate (median 33 days)	reco-very was achi-eved	3.Weight gain 4.Time to cure 5.Incidence of diarrhea
Kambale et al., 2023[16]	Double -blind RCT	Probiotic (200) Placebo (200)	6-24	Lacticase-bacillus rhamnosus GG Limosi-lactobacillus reuteri DSM 17938	Coco-nut oil	1 month	Until nutri-tional reco-very or the end of 12-week period	1.Duration of diarrhea 2.Risk of diarrhea 3.Nutri-tional recovery 4.Weight gain 5.Frequen-cy of pneumonia 6.Transfer to inpatient care rate

Weight Gain

Four studies reported weight gain after interventions. Our study showed that the overall analysis [0.30 (95% CI -0.11 to 0.71); I^2 = 92%, p = 0.15] as well as the subgroup analysis on probiotic-only [0.45 (95% CI -0.21 to 1.10); I^2 = 93%, p = 0.18] and synbiotic-only [0.02 (95%CI: -0.12 to 0.15); I^2 = 0%, p = 0.79] did not exhibit any statistical significance compared to the placebo. Furthermore, the overall and probiotic subgroup analysis exhibited high level of heterogenicity (**Figure 3**).

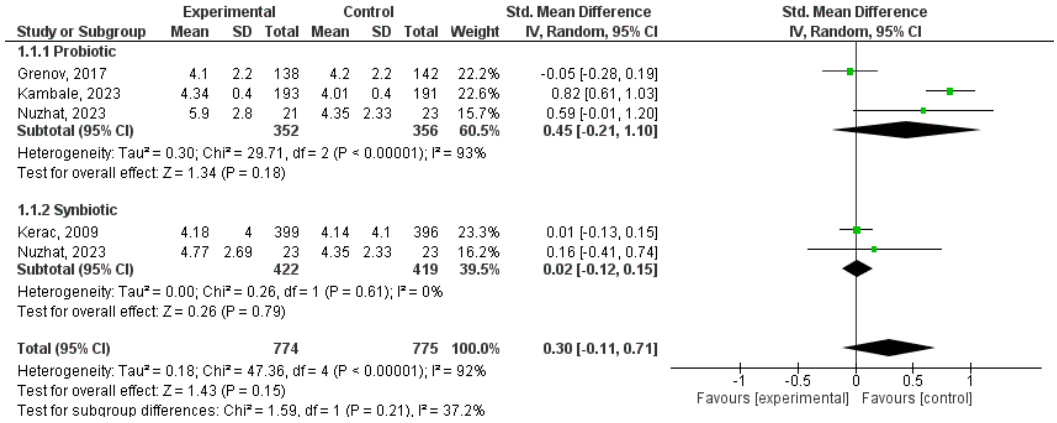

Figure 3. Forest plots for weight gain outcomes. SD: standard deviation, CI: confidence interval

Incidence of Diarrhea

Two studies reported the incidence of diarrhea after probiotic (experimental) and placebo (control) interventions, categorized by inpatient and outpatient settings. Among inpatient participants, there was no significant difference between the two groups [72.8% vs 64.2%; RR = 1.11 (95% CI 0.85 to 1.47); I^2 = 94%, p = 0.44). Interestingly, probiotics slightly lowered diarrhea incidence, despite being not statistically significant [27.1% vs 30.9%; RR = 0.90 (95% CI 0.80-1.01); I^2 = 0%, p = 0.08]. Overall, the studies showed no significant difference in diarrhea incidence between probiotics and placebo [50.8% vs 48.1%; RR = 1.02 (95% CI 0.89-1.17); I^2 = 79%, p = 0.81] (**Figure 4**).

Duration of Diarrhea

Two studies reported the duration of diarrhea after probiotic (experimental) and placebo (control) interventions. Kambale et al. found a shorter duration in the probiotic group, while Nuzhat et al. observed a smaller, non-significant decrease. Overall, probiotics significantly shortened diarrhea duration, with a mean difference of -0.70 (95% CI -0.89 to -0.50); I^2 = 0%, $p < 0.00001$ (**Figure 5**).

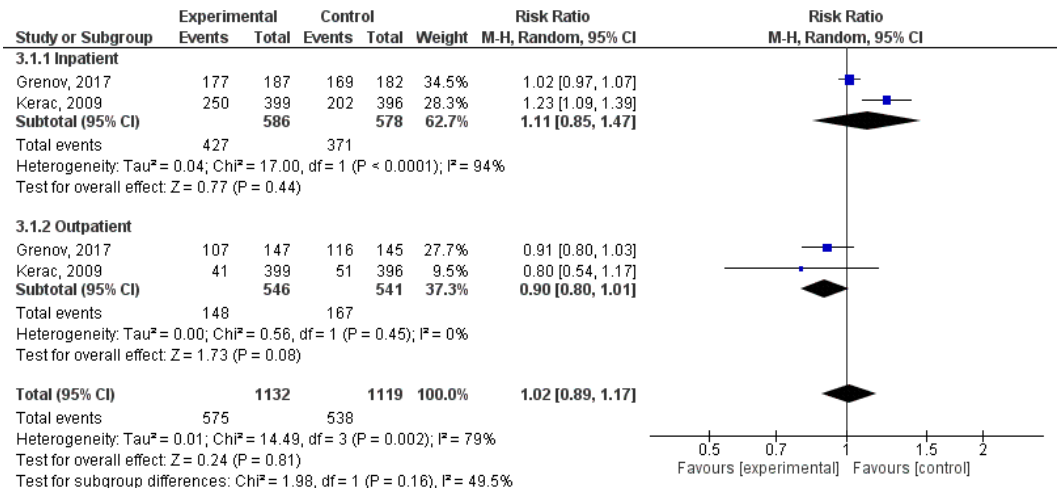

Figure 4. Forest plots for incidence of diarrhea outcomes. CI: confidence interval.

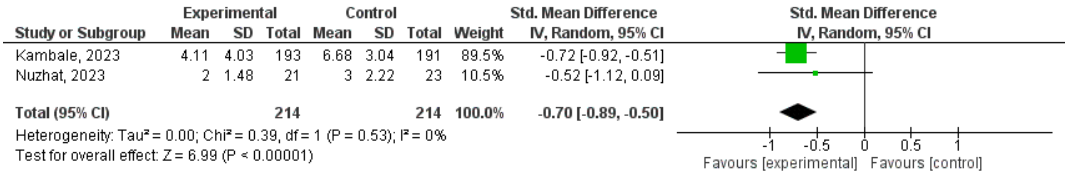

Figure 5. Forest plots for duration of diarrhea outcomes. SD: standard deviation, CI: confidence interval.

Discussion

The four studies included in this study reveal differences in design, population, settings, and interventions. Most studies used double-blind designs with sample sizes ranging from 21 to 399 participants, ages from 2 months to 168 months. Settings included both inpatient and outpatient treatments, with some studies transitioning from hospital care to community-based follow-up (e.g., Grenov et al., 2017; Kerac et al., 2009).[17, 18] Interventions involved probiotic strains like *Bifidobacterium* and *Lactobacillus*, often combined with prebiotics such as galacto-oligosaccharides, compared to placebos like lactose or maltodextrin. Study durations varied from 28 days to 12 weeks, with follow-up until nutritional recovery or 4–12 weeks post-intervention. All studies have a low risk of bias.

In children with severe acute malnutrition (SAM), gut function is significantly compromised due to gut junction impairment and increased permeability, leading to both intestinal and systemic inflammation.[19] This dysfunction can manifest as diarrhea, poor nutrient absorption, bacterial overgrowth in the small intestine, intestinal damage, and weakened immune system. Research indicates that SAM is also linked to an unbalanced gut microbiome (dysbiosis), which restricts children's growth and worsens malnutrition.[20] Probiotics are expected to support child growth by preventing

infections and enhancing nutrient and vitamin absorption through modulation of the gut microbiota.[21] Previous meta-analyses suggest that malnourished children receiving prebiotics and probiotics experience significantly greater weight gain compared to those in the control group.[13] Conversely, our meta-analysis indicated that weight gain was greater in the control group compared to the probiotic group, although this difference was not statistically significant (SMD = 0.3; 95% CI -0.11 to 0.71; p = 0.15). These differences in findings may be attributed to several factors. The study population in our analysis exhibited more severe clinical conditions compared to previous meta-analyses, which primarily included children with underweight status without complication. This can be seen from the high heterogeneity exhibited in the analysis. Additionally, one randomized controlled trial by Batool et al. reported a significant difference in weight between the probiotic and control groups after the intervention.[22] There is a distinguishing factor in this study, as the Batool et al. study included children with uncomplicated SAM undergoing outpatient treatment, whereas in majority of our studies focused on hospitalized children with more severe forms of SAM requiring inpatient care.[17, 18] This finding underscores the importance of illness severity in determining the efficacy of probiotics.

Among the four included studies, three studies (Grenov et al., Kerac et al., Kambale et al.) reported a greater weight gain in the probiotic group compared to the control group, however, the difference was not statistically significant.[16-18] Conversely, one study by Nuzhat et al. demonstrated a significant difference in weight gain between the probiotic, symbiotic, and placebo groups.[15] A key distinction in the Nuzhat et al. study was that the intervention was administered after the completion of antibiotic treatment, which may have influenced the observed effects. Antibiotics may decrease gut colonization and viability of the probiotics, thereby reducing the effectiveness of the treatment.[16, 18]

According to Suez et al., antibiotics disrupt the natural balance of the gut microbiome, causing dysbiosis and reducing microbial diversity. It also impairs the microbiome's ability to recolonize, which leads to prolonged dysbiosis. After antibiotics disrupt the gut microbiome, probiotics rapidly occupy the vacant spots, thereby outcompeting the native commensal bacteria for adhesion sites and nutrients. This competitive exclusion impedes the regrowth of the host's original microbiota. Furthermore, the probiotics themselves can release soluble factors, particularly from *Lactobacillus* species, that inhibit the growth of native bacteria, further impairing microbiome recovery. This combination of disrupted colonization by antibiotics and probiotics-induced inhibition reduces the effectiveness of probiotics in restoring a healthy gut microbiome after antibiotic use.[23]

Our meta-analysis indicates that there was a slightly higher risk of diarrhea in the probiotic group in inpatients, but the difference was not statistically significant (RR = 1.1 [95% CI: 0.85, 1.47]). In contrast, in the outpatient group, there was a possible reduction in diarrhea incidence with probiotics, although it is not statistically significant (RR = 1.02 [95% CI: 0.89, 1.17]). These results suggest that probiotics did not significantly impact the incidence of diarrhea compared to the control group, despite the observed trend towards lower diarrhea incidence in the outpatient participants. These results may be attributed to severe illness and antibiotic use in both studies.[17, 18]

Among the two included studies which analyzed diarrhea duration, only one study reported a significant difference between the probiotic and control groups. A key difference between these studies was that among the study population reported by Nuzhat et al., greater severity of illness was observed compared to the other included study.[15] Additionally, in the Kambale et al. study, all children were HIV-negative, which may have influenced the outcome.[16] In HIV patients, there are changes in gut microbial composition, significant loss of CD4+ T cells in the gastrointestinal tract, inflammation and immune activation, and the formation of viral reservoirs.[24] However, the overall meta-analysis revealed that probiotics significantly reduced diarrhea duration compared to the control (SMD = -0.70 [95% CI: -0.89, -0.50]; $p < 0.00001$). This finding suggests that while probiotics may not significantly prevent diarrhea in SAM children, they could be effective in shortening its duration, potentially improving recovery outcomes.

HIV status has become an important factor to investigate, there had been concerns that, due to the high prevalence of HIV among subjects, probiotic administration might cause sepsis due to the immunocompromised status.[21] HIV patients often undergo prolonged antibiotic treatment, which can result in lasting changes to the gut microbiota. Extended antibiotic use may significantly reduce gut bacterial concentrations, and in some cases, lead to the complete loss of specific bacterial communities.[25]

The type of strain used may also influence efficacy of the treatment. Among the four studies, the study that demonstrated significant weight gain was using *Bifidobacterium infantis* as the probiotics.[15] Human milk oligosaccharides (HMOs) are sugar present in breast milk that function as selective growth promoters for beneficial gut bacteria, especially Bifidobacteria.[26] In the probiotic group, breast milk intake was the highest, which may have contributed to the significant weight gain observed in this group. *B. infantis* is a key gut bacterium in infancy that is depleted in severely malnourished infants, leading to immature gut microbiota. The SYNERGIE trial found that B. infantis EVC001 supplementation improves weight gain and reduces intestinal

inflammation in malnourished infants.[15, 27] This strain may also explain why significant weight gain and reduced diarrhea duration were observed in studies with a population under two years old. This could be due to the fact that many children in this age group are still consuming breast milk, where human milk oligosaccharides (HMOs) enhance the effectiveness of probiotics.

We acknowledge that our study has limitations, including variations in probiotic strains, dosages, and treatment durations, which make comparisons challenging. We recognize that the concurrent use of antibiotics likely reduced probiotic efficacy, and the inclusion of HIV-positive children may have introduced confounding factors. Additionally, the lack of long-term follow-up limits our understanding of the sustained effects on weight gain and overall health outcomes in SAM children. Thus, further research is required to analyze these factors and their role in determining probiotic effectiveness. Investigating specific strains like *Bifidobacterium infantis* could provide more effective treatment options.

Conclusion

This systematic review and meta-analysis revealed that probiotics and synbiotics did not noticeably enhance weight gain in children with severe acute malnutrition when compared to placebo. Nonetheless, probiotics were linked to shorter durations of diarrhea, which could aid in recovery. The effectiveness of probiotics seems to be affected by various factors, including the severity of illness, antibiotic use, HIV status, breastfeeding, and age. These observations underline the importance of further studies to better understand the interactions between probiotics and these influencing factors, ultimately aiming to improve treatment strategies for malnourished children.

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Conflict of Interest

None declared

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APGHN Archives of Pediatric Gastroenterology, Hepatology, and Nutrition

Case Report

Multiple Thrombi in Portal Vein with Protein C and Protein S Deficiency: A Case Report

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Abstract:

Background: Portal vein thrombosis (PVT) is a rare but significant cause of the gastrointestinal bleeding, often leading to delayed diagnosis due to its subtle presentation. PVT can result in portal hypertension, which cause the formation of portosystemic collaterals as a compensatory mechanism. Deficiencies in protein C and protein S are recognized as risk factor for PVT. This study aims to describe a rare underlying condition of PVT in children.

***Case*:** A 14-year-old boy presented with recurrent hematemesis and melena over the past eight years. Physical examination revealed no epigastric pain but showed splenomegaly. Laboratory findings showed pancytopenia, mildly elevated AST, prolonged plasma prothrombin time and partial thromboplastin time, increased INR, and low protein C and S. Endoscopic evaluation revealed esophageal and fundal varices, with portal hypertensive gastropathy. Multi-slice CT (MSCT) confirmed thrombosis in the main, right, and left portal veins, supporting the diagnosis of PVT. The patient was also diagnosed with an underlying myeloproliferative disorder, further contributing to his condition.

***Discussion*:** Protein C and S deficiency is a prominent risk factor of PVT. Identification of risk factors is essential to accurately treat the condition and prevent worse outcomes. Imaging modalities remain essential for diagnosing PVT. Management focuses on treating complication, particularly those related to portal hypertension, with endoscopic variceal ligation as the preferred treatment.

Conclusion: Comprehensive investigation on etiology and risk factors, close monitoring and individualized treatment are essential in PVT management, especially in pediatric patients.

Keywords: children, portal vein thrombosis, protein C, protein S

Introduction

Portal vein thrombosis (PVT) is an uncommon condition, occurring in approximately 1 in 100,000 live births or between 1 and 36 per 1000 newborns.[1-3] PVT is characterized by the formation of a thrombus in the main trunk of the portal vein, along with its right and/or left intrahepatic branches.[4] The main clinical manifestations of PVT are upper gastrointestinal bleeding and splenomegaly.[1, 2, 5] Splenomegaly results from portal hypertension.[3, 6] The cause of PVT in children is unknown, but several factors were found to predispose this pathology. These can be divided into three groups: local factors that may lead to portal vein injury (such as abdominal infections, surgical procedures in the abdomen, or the use of umbilical catheters), general factors (such as a procoagulant state), and, occasionally, vascular abnormalities. The most common cause is umbilical vein catheterization (UVC), with prevalence ranging from 20% in low-income countries to 60% in developing countries. The general factors predisposing to venous thrombosis are thrombophilia, sepsis, and dehydration. Deficiency or qualitative abnormalities of anti-coagulation factors (antithrombin III, protein C, protein S, and activated protein C resistance) and chronic myelodysplasia syndrome often predispose to thrombotic events, including extrahepatic PVT (EHPVT).[1, 4, 6]

The fundamental pathophysiology of portal hypertension resulting from a prehepatic obstruction of portal blood circulation, while liver function remains well-preserved without any cirrhotic alterations in the liver. This increase in blood volume within the venous portal system causes an elevation in hepatic venous pressure. As a compensatory mechanism, portosystemic collaterals are formed, resulting in increased flow in splenorenal, paraumbilical, or gastric and esophageal veins. Additionally, cavernomatous transformation of the portal vein (CTPV) arises from the formation of hepatopetal collateral vessels surrounding the obstructed portal vein.[3, 6]

Currently, studies on pediatric portal vein thrombosis are still limited. Thus, this study aims to report the rare occurrence of portal vein thrombosis on a 14-year-old with protein C and protein S deficiency.

Case

A 14-year-old boy was admitted to the hospital with a history of recurrent hematemesis and melena. These episodes occurred 2-3 times yearly over the past 8 years. The last time hematemesis and melena occurred was on Mei 2024. He had a history of polyps and neonatal infection. Figure 1 shows colonic polyps observed during a colonoscopy performed in 2017.

During his hospital visit, physical examination revealed normal vital signs, no epigastric pain, and splenomegaly classified as Schuffner stage 2. He also had a history of pancytopenia and slightly elevated SGOT and normal SGPT, with an increase in plasma prothrombin time (1.58x), partial thromboplastin time (1.42x), and INR (2.04x). Endoscopy in 2020 identified grade 2-3 esophageal varices, grade 1-2 fundal varices, and portal hypertensive gastropathy **(Figure 2)**.

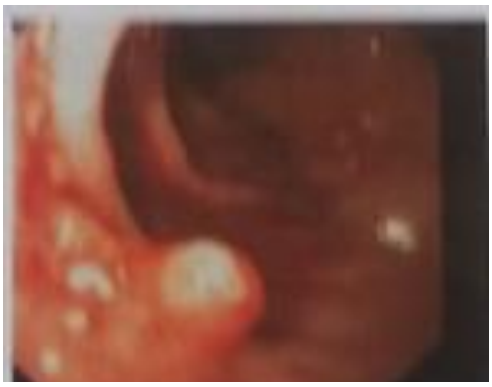

Figure 1. Colonoscopy performed in 2017 revealed hyperemic mucosa with extensive ulcerative lesions in the rectum, sigmoid colon, ascending colon, transverse colon, descending colon, and cecum. Additionally, three polyps measuring 2–3 mm in diameter were identified 10 cm from the anal verge.

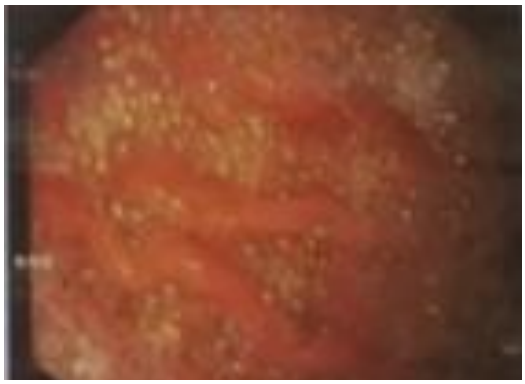

Figure 2. Gastroscopy revealed grade 2-3 esophageal varices, snake-skin appearance with erythema in gaster, erythema and petechiae in the antrum, normal bulbous duodenum, and grade 1-2 varices in the fundus.

Abdominal MSCT with contrast was conducted in December 2023 and revealed thrombi in the main, right, and left portal veins, with the largest thrombus measuring 1.1 cm in thickness and 5.7 cm in length **(Figure 3).**

A subsequent bone marrow puncture confirmed the diagnosis of hypoplastic myelodysplastic syndrome (hMDS) **(Figure 4)**. The patient also exhibited low levels of protein C (27 IU/dL) and protein S (36 IU/dL).

The patient was initiated on maintenance doses of propranolol, a non-selective beta blocker, to reduce portal pressure and received treatment for portal vein thrombosis. Varices ligation was also planned.

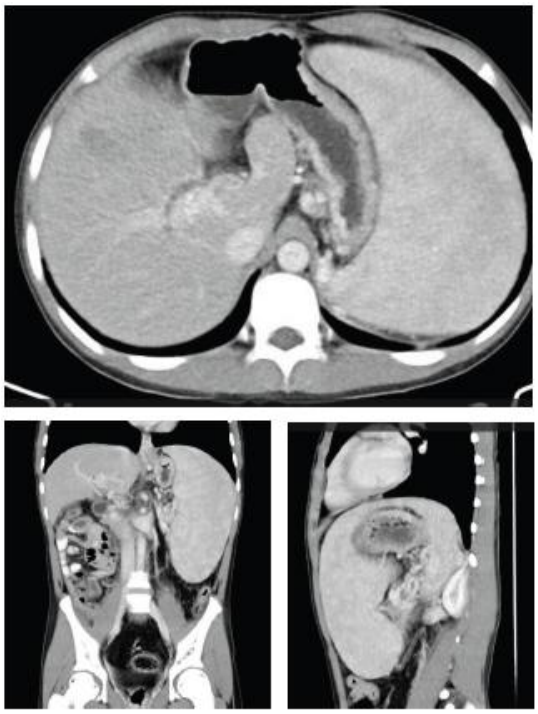

Figure 3. MSCT scan exhibited thrombi in the main, right, and left portal veins, with the largest thrombus measuring 1.1 cm in thickness and 5.7 cm in length. Splenomegaly was observed, with splenic widening and tortuosity of splenic vein, oesophagal vein, left gastric vein, right gastric vein, gastroduodenal vein, and gastroepiploic vein. Furthermore, multiple lymphadenopathies were found in paraaorta, interaortocava, and left and right inguinal region, with the largest measuring around 1.7 × 0.7 cm in the interaortocaval region.

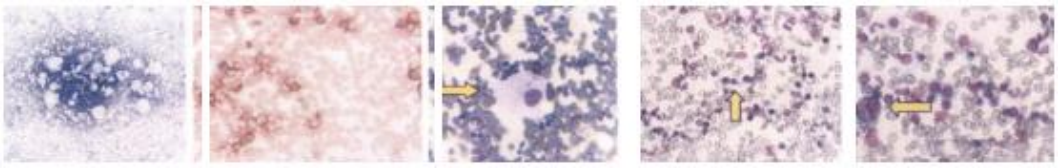

Figure 4. Bone marrow puncture showed hypocellular bone marrow with trilineage dysplasia and mild erythroid hyperplasia, suggesting a myelodysplastic syndrome (hypoplastic MDS)

Discussion

In this case, a 14-year-old boy presented with recurrent hematemesis and melena for 8 years, along with a history of polyps in the colon and splenomegaly. The patient also exhibited pancytopenia, as well as esophageal and gastric varices. Esophageal varices (EV) are present in 90% to 95% of patients with portal vein thrombosis, and gastric varices (GV) in 35% to 40%, which extrahepatic thrombi is one of the rare causes of

esophageal varices. Initial obstruction of the PV by thrombus formation is followed by compensatory vasodilation of the hepatic artery and the formation of collateral vessels that bypass the thrombosed PV and constitute the "cavernous transformation" or "portal cavernoma" which leads to varices formation.[2] The endoscopic result showed gastropathy portal hypertension in this patient, which worsens the prognosis of this patient.[2, 7] This patient then underwent bone marrow puncture due to its pancytopenia, and the result suggests a myelodysplastic syndrome. Myeloproliferative disorders account for 30% to 40% of PVT in adults but are uncommon in children.

Interestingly, the patient exhibited low protein C and protein S. These proteins are natural vitamin K-dependent anticoagulants, which may result from either hereditary or acquired causes.[1, 8] Deficiency in these proteins has been associated with the occurrence of thrombosis at multiple sites, as well as childhood stroke.[8, 9] Notably, a study by Grama et al. reported a high prevalence of protein C and S deficiency in children with extrahepatic portal vein thrombosis. The decreased level of proteins S and C can be secondary to the thrombosis or as the result from their consumption in portosystemic shunts. Neovascular formations (cavernoma) will generate hepatopetal flow that is insufficient to reduce the pressure, which may cause the formation of spontaneous natural portosystemic shunts. These shunts function as "release valves" to relieve the pressure in the portal space. However, this compensatory mechanism is insufficient and does not allow adequate reduction of portal pressure, thus causing the consumption of these anticoagulant proteins. Another cause of protein C or S deficiency is the hepatic injury caused by reduced flow through the portal vein.[1] In this patient, the occurrence of bleeding due to the portal hypertension had occurred for several years. This highlights the importance of extensive investigation for the etiology and risk factors, which should be conducted thoroughly to prevent prolonged manifestation which may worsen the prognosis of the patient.

In patient with PVT, history taking and physical examination, imaging studies are the mainstay for diagnosis. Ultrasonography is the initial modality due to its noninvasive technique. The results expected for PVT are detection of hyperechoic thrombi in the PV lumen, collateral vessels, and portal cavernous. Portal cavernoma constitutes a tangle of tortuous vessels in the porta hepatis. Contrast-enhanced computed tomography and magnetic resonance angiography are valuable tools for evaluating the degree of thrombosis.[2] MSCT abdomen was performed in this patient and showed multiple thrombi in the portal vein.

Treatment includes pharmacologic, endoscopic, and surgical modalities. Nonselective b-blockers reduce hepatic venous pressure gradient by decreasing cardiac output (b-1 receptor antagonism) and inducing splanchnic vasoconstriction (b-2 receptor antagonism).[2] Endoscopic techniques, including sclerotherapy and endoscopic variceal ligation (EVL), are very effective for managing acute variceal hemorrhage and

eliminating varices. EVL is preferred because it has limited complications and requires fewer endoscopic treatments. EVL facilitates portal decompression either by forming collateral vessels or by surgical portosystemic shunting, when vessels grow to the proper diameter for anastomosis.

The medical management of PVT involves the use of beta-blockers for initial prevention.[10, 11] Sclerotherapy and variceal band ligation are essential treatment methods both in severe life-threatening bleeding and in preventing possible bleeding in those with high-grade varices. Children who have significant varices should be evaluated for primary prevention using endoscopic treatment. Children with recurrent bleeding, despite these measures, will be suitable for surgical treatment (shunts or bypass). PVT can arise following a transplant, but the best approach to management remains unclear.[12]

Surgical portosystemic shunts are typically utilized only in severe cases due to considerable risks and technical challenges. Transjugular portosystemic shunts are gaining importance in treating portal hypertension resulting from PVT.[13] Portosystemic shunts can either alleviate pressure throughout the entire splanchnic area (including portacaval, mesocaval, or proximal splenorenal shunts) or specifically relieve pressure in the gastroesophageal veins while maintaining a steady flow from the superior mesentery (as seen in distal splenorenal shunts). The role of portosystemic shunts is well known in controlling variceal bleeding and symptoms related to hypersplenism.

The primary reasons for considering surgical intervention include uncontrollable severe bleeding despite endoscopic treatment, ongoing high-grade varices, significant splenomegaly accompanied by notable thrombocytopenia, or hindered growth.[12] Various factors, such as the overall health of the child, the presence of other medical conditions, the vascular structure, and the capabilities and experience of the surgical team influence the selection of the type of shunt. Only a small number of children experienced complications, including complete or partial narrowing of the shunt.

Another surgical option that shows excellent outcomes is the mesenteric-left portal bypass (Meso-Rex bypass).[14] This procedure seeks to restore blood flow in the portal vein by establishing an anastomosis between the superior mesenteric vein and the left portal vein. This technique was first employed for portal vein thrombosis following liver transplant surgery. It is utilized in children with extrahepatic portal vein thrombosis (EHPVT) who have a weight-to-portal vein diameter ratio exceeding 10. The meso-Rex shunt reduces the portal vein pressure, the degree of esophageal and gastric varices, or the splenomegaly and significantly improves the prognosis in children with EHPVT.[14, 15] Doppler ultrasound has demonstrated a remarkable intrahepatic portal flow following the Rex-bypass shunt.

The transjugular intrahepatic portosystemic shunt (TIPSS) is a non-surgical method by which a metallic stent is inserted between the hepatic and intrahepatic portal veins.[16, 17] The experience with TIPSS in pediatric patients is restricted to a few case reports or small series that highlight complications, which may hinder the viability of alternative surgical interventions.[18] TIPSS may be used when surgical shunting is contraindicated or as a bridge to liver transplantation.[16, 17]

Conclusion

Portal vein thrombosis should be considered a potential cause of gastrointestinal bleeding, with a thorough investigation into its underlying etiology and risk factors. Treatment strategies must be individualized to address the patient's underlying condition and any contributing factors.

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Conflict of Interest

None declared

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Case Report

Acute Intestinal Obstruction Due to Ascariasis in a Child: A Case from a Resource Limited Setting

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Abstract:

Background: Ascariasis remains a widespread and frequently undiagnosed condition, with most cases being asymptomatic. However, severe manifestations can result in total or partial intestinal obstruction. In resource-limited settings, diagnosis primarily depends on clinical presentation, stool examination, and basic radiographic imaging.

***Case*:** A three-year-old child from rural Southwest Sumba presented with vomiting, fever, abdominal pain, and constipation. Physical examination, faecal examination, and imaging revealed partial small bowel obstruction due to an *Ascaris* bolus, complicated by mild to moderate dehydration. Following conservative management including fluid resuscitation, NPO status, NGT insertion, and single dose albendazole, the patient passed numerous worms, improved clinically, and was discharged on day five.

***Discussion*:** This case report highlights the diagnostic and management challenges of a severe form of ascariasis that caused partial small bowel obstruction in a resource-limited setting. The patient's differential diagnoses included intussusception, mesenteric cyst, and abdominal tuberculosis. The patient presented with risk factors including young age, frequent barefoot contact with soil, poverty, residence in an area with limited access to water and healthcare, and a lack of participation in deworming programs. Through clinical assessment, the patient was successfully managed with conservative treatment and antihelminthic therapy.

Conclusion: While small bowel obstruction presents a diagnostic challenge with a wide range of differential diagnoses, ascariasis offers distinct risk factors and can be readily confirmed with basic diagnostic tools. Ascariasis remains a significant public health issue, highlighting the urgent need for intensified community education on hygiene and improved public health infrastructure in Southwest Sumba.

Keywords: ascariasis, deworming program, resource-limited setting, small bowel obstruction

Introduction

Ascariasis, classified as a neglected tropical disease, is an infection of the small intestine caused by the roundworm Ascaris lumbricoides.[1] Ascariasis is the most common helminth infection in humans worldwide, with an estimated one billion people infected globally.[2] While specific prevalence data for ascariasis in Indonesia are lacking, the overall prevalence of soil-transmitted helminthiasis, of which ascariasis is the most prevalent, ranges from 2.5% to 62%.[3] Infection occurs via the fecal-oral route, typically through the ingestion of food or water contaminated with Ascaris eggs from feces-contaminated soil.[4]

Ascariasis is prevalent in tropical and subtropical regions within resource-limited settings, including Indonesia.[5] This disparity in prevalence is attributable to factors common in these settings, such as poor sanitation, limited access to clean water, a lack of deworming programs, and poverty.[6]

Ascariasis is often asymptomatic, leading to underdetection, particularly in adults.[7] However, in children, ascariasis has significant impacts on health, including malnutrition and impaired growth, cognitive development issues, and in severe cases, intestinal obstruction, as observed in the present case.[8]

This case is of particular interest as it directly reports a case of ascariasis presenting with a severe manifestation of ascariasis in a 3-year-old child from Southwest Sumba. The Southwest Sumba region is an area with a relatively high poverty rate. In 2024, the percentage of the poor population reached 102.000 people.[9] This condition is worsened by the geographical landscape, which makes the distance between residential areas and healthcare facilities far and difficult to access. In these resource-limited settings, diagnosis relied on clinical presentation, stool examination, and basic radiographic imaging, such as plain radiography. We also describe the diagnostic approach to ascariasis, emphasizing the importance of recognizing clinical presentation and effectively using available resources.

Case

A three-year-old boy from Waimahaka, Kodi Bangedo District, a remote rural area in Southwest Sumba, presented to the emergency department of Karitas Hospital in December 2024 with a three-day history of vomiting. He had four to five vomiting episodes in a day that were nonbilious, containing food contents. He presented with a two-day history of fever, refused to eat, and mild abdominal pain. He also presented with a one-week history of constipation. According to the parents, the patient's abdomen had appeared distended for the past week. The patient had no previous similar complaints and was otherwise healthy.

He frequently had unprotected contact with soil (i.e., without footwear), infrequent handwashing, and consumption of boiled water from a dug well. The patient had no history of prior deworming medication. There was no family history of tuberculosis. Parental report indicated normal growth and developmental milestones according to his age.

Upon physical examination, the patient presented with signs of mild to moderate dehydration, with a heart rate of 102 beats per minute, a respiration rate of 22 breaths per minute, and a temperature of 37.7 °C. Abdominal examination revealed distension and tenderness to palpation in the right hypochondriac and left lumbar regions. Additionally, a mobile, firm, and elastic mass approximately 2 x 2 cm in size was palpable in the same regions. Bowel sounds were increased and shifting dullness was absent. The remaining head-to-toe examination was unremarkable. The child had good nutritional status.

A complete blood count was normal except for an elevated platelet count of 430,000/µL. Stool analysis was positive for *Ascaris lumbricoides* ova (1 – 5 ova per high power field). Plain abdominal radiography revealed partial small bowel obstruction with a "cigar bundle" appearance in the ascending and descending colon, suggestive of an *Ascaris* bolus (**Figure 1**).

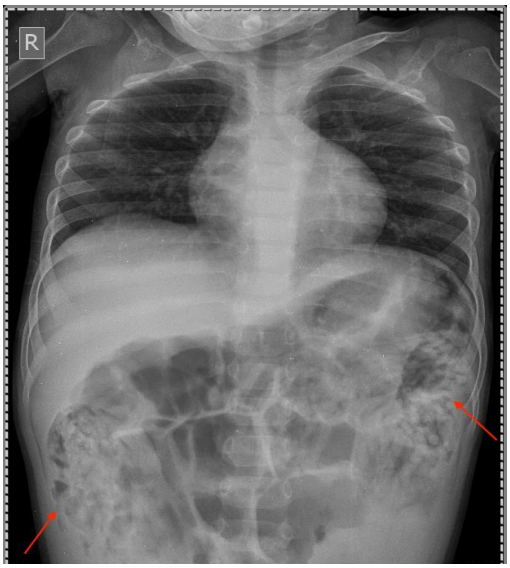

Figure 1. A plain abdominal radiograph of the patient revealed a "cigar bundle" appearance (red arrows).

The patient's signs and symptoms, along with radiographic findings, were consistent with partial bowel obstruction due to an *Ascaris* bolus, complicated by mild to moderate dehydration.

Following diagnosis, the patient was placed on nil per oral (NPO) status, a nasogastric tube (NGT) was inserted, and fluid resuscitation was initiated per guidelines for mild to moderate dehydration. During hospitalization, a shift in the palpable abdominal mass to the umbilical and right iliac regions was noted on day two. Between days two and four, numerous adult *Ascaris lumbricoides* worms were passed in the patient's stool (**Figure 2)**. Oral feeding was subsequently resumed, and a single-dose anthelmintic therapy of albendazole 400 mg was administered for three consecutive days.

Following cessation of worm passage and administration of anthelmintic therapy, the patient exhibited increased activity, resolution of emesis, return of bowel sounds to normal, and passage of stool. Surgical intervention was deemed unnecessary, and the patient was discharged on the fifth day.

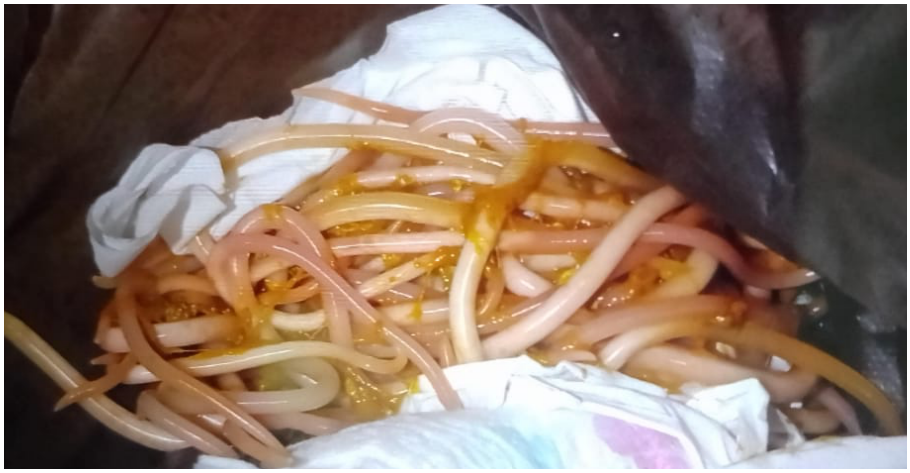

Figure 2. *Ascaris lumbricoides* worms passed by the patient.

Discussion

Soil-transmitted Helminth (STH) infections are among the most widespread infections globally, affecting approximately 1.5 billion people, or 24% of the world's population. These infections primarily impact developing countries and underserved communities in tropical and subtropical regions, where access to clean drinking water, adequate sanitation, and proper hygiene is limited. The highest prevalence has been recorded in sub-Saharan Africa, China, South America, and Asia.[10]

This finding aligns with a case observed in our study, involving a preschool-aged boy from a low socioeconomic background residing in a rural community in Indonesia. These established risk factors are particularly pertinent in the context of West Sumba, where access to clean drinking water, adequate sanitation, and proper hygiene remains limited.[11]

The life cycle of *Ascaris lumbricoides* starts when infected eggs are consumed through fecal-oral transmission, a comp (Figure 3). Once inside the body, the eggs hatch into larvae, which penetrate the intestinal mucosa and travel through the portal and systemic circulation to the lungs. Over a period of 10 to 14 days, the larvae continue to mature in the lungs, break through the alveolar walls, move up the bronchial tree to the throat and are then swallowed. Upon returning to the small intestine, they develop into adult worms and are capable of reproducing. Female worms release unfertilized or fertilized eggs, which are then excreted in the feces. In the external environment, under suitable conditions of warmth, moisture, and oxygen, the fertilized eggs embryonate, developing into infective larvae within the eggshell. These embryonated eggs are then capable of infecting a new host, completing the life cycle.[12]

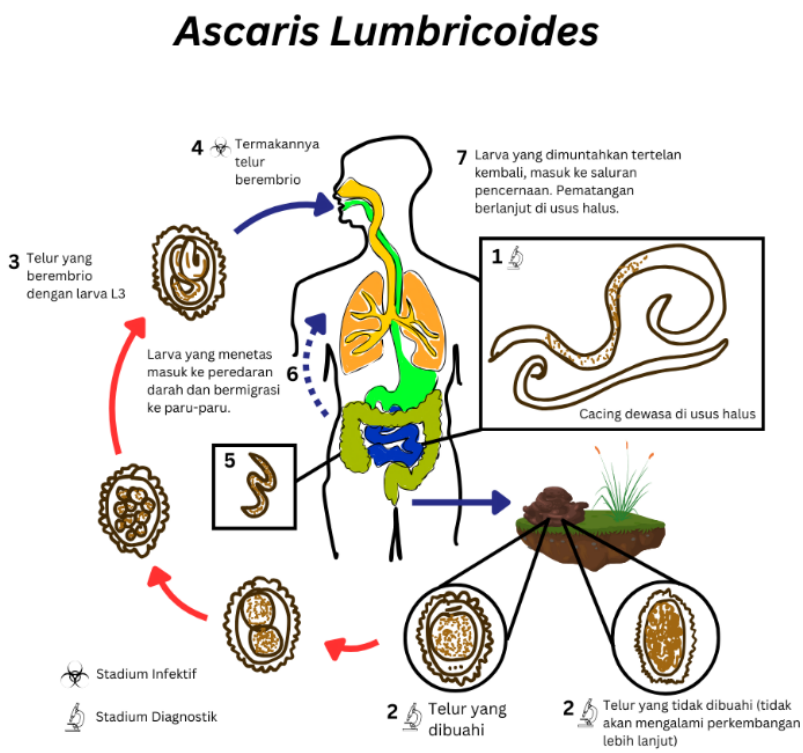

Figure 3. Life cycle of *Ascaris lumbridocides*.[12]

This patient's history included frequent barefoot contact with soil, infrequent handwashing, and consumption of boiled water from a dug well, all of which are recognized risk factors for soil-transmitted helminth infections. Although infection can occur at any age, preschool and early school age children have the highest infection rates, due to low levels of acquired immunity and increased exposure to contaminated soil.[13]

Furthermore, considering the severe clinical presentation of the patient, it is likely that this patient has experienced recurrent infections, likely occurring for more than at least 2 to 4 weeks. This highlights the public health significance of ascariasis, emphasizing the existing barriers between society and healthcare.[12]

Most *Ascaris lumbricoides* infestations are asymptomatic. However, due to the widespread prevalence of the infection, the overall burden of symptomatic cases remains significant. Individuals with a heavy worm burden are more likely to develop symptoms, which may include diarrhea, loss of appetite, weakness, abdominal pain, altered bowel habits, weight loss, and in rare cases, the expulsion of worms through the mouth or rectum. Severe infections can lead to intestinal obstruction, presenting with abdominal distension, the presence of a palpable mass, tenderness, or pain. The likelihood of developing symptoms is related to the worm load. Intestinal blockage caused by roundworms are more commonly observed in children due to their smaller intestinal lumen and higher parasite burden.[8, 14]

This patient presented with a three-day history of vomiting and a week-long absence of bowel movements, accompanied by abdominal distension of one week's duration. Physical examination revealed palpable masses in the right hypochondriac and left lumbar regions. These findings strongly suggested a mechanical bowel obstruction. The initial differential diagnoses encompassed intra-abdominal masses, intussusception, mesenteric cyst, and abdominal tuberculosis.

Intussusception commonly manifests with sudden wax and wane abdominal pain.[15] However, in this case, the patient presented continuous mild intensity of pain. Furthermore, while the classic presentation of intussusception includes the passage of 'red currant jelly' stools, indicative of blood and mucus, this patient did not exhibit hematochezia or pass any stool.[16]

Mesenteric cysts may also manifest with abdominal distension, nonspecific abdominal pain, and obstructive symptoms. However, these cysts typically present as soft, fluid-filled intra-abdominal masses.[17] In contrast, the mass palpated in this patient was a mobile, firm, and elastic mass, suggestive of a solid mass.

While abdominal tuberculosis remains a diagnostic consideration due to its ability to mimic other conditions and the presence of constitutional symptoms, it is deemed less likely in this case. This is based on the absence of typical TB symptoms such as chronic cough, fever for two weeks or more, weight loss, and malaise.[18] Additionally, the patient is not immunocompromised, has no history of contact with TB patients, and physical examination does not reveal ascites or enlarged lymph nodes. Although the patient does present with fever, it is of recent onset. There were no abnormalities detected in the chest x-ray.

In children with severe worm infestations, large clusters of worms may appear as "cigar bundles" – radiolucent areas visible on plain abdominal radiography. In some cases, the contrast between the worm mass and intestinal gas creates a "whirlpool" effect. Abdominal ultrasonography is also a useful tool for detecting suspected intestinal worm infestations. Roundworms can appear in various sonographic patterns, such as a thick echogenic strip with a central anechoic tube or multiple long, linear, parallel echogenic strips without acoustic shadowing.[14] To confirm the diagnosis, abdominal ultrasound was initially planned. However, due to limited hospital resources, the procedure was only available twice weekly, and the patient had already passed the worms the following day of hospitalization. Given these constraints, the available tests included routine stool examination and plain radiography was performed in this case.

Ascaris-induced intestinal obstruction is a common complication of ascariasis, with a reported prevalence ranging from 38% to 87.5% of all complications.[19] Intestinal obstruction in ascariasis can arise through several mechanisms: Due to the accumulation of large worm clusters that physically block the intestine, the worms acting as lead points for intussusception, or the release of neurotoxins that induce intestinal contractions and inflammation, ultimately leading to obstruction. Furthermore, adult *Ascaris* worms can contribute to various acute abdominal conditions, including small bowel obstruction, upper gastrointestinal bleeding, intussusception, volvulus, and intestinal perforation with peritonitis. The hepatobiliary system can also be affected, resulting in conditions such as acute cholecystitis, acute cholangitis, biliary colic, liver abscess, and acute pancreatitis.[20]

Other frequent complications include pulmonary eosinophilia, or Loeffler's syndrome, resulting from the migration of adult *A. lumbricoides* to the lungs, causing respiratory symptoms.[21] In this patient, this complication was absent, as there were no respiratory symptoms, and both thoracic radiology and laboratory findings were normal, with no evidence of eosinophilia.

Ascariasis can negatively impact growth and development, strongly correlating with undernutrition. In young children, ascariasis can obstruct the small intestine and occasionally migrate into and obstruct the pancreatic and bile ducts, leading to malabsorption of vitamin A and reduced lactose digestion. These consequences can result in growth retardation, undernutrition, impaired cognitive function, low educational achievements, and ultimately, loss of productive years.[22]

The treatment of uncomplicated ascariasis generally conservative and involves the use of antiparasitic medication such as albendazole, mebendazole, or pyrantel pamoate. Partial intestinal obstruction caused by ascariasis can sometimes resolve on its own with measures such as bowel rest, nasogastric decompression, anthelmintic therapy, and fluid-electrolyte replacement. However, in patient with complete obstruction, perforation or peritonitis, surgical intervention may be necessary.[23]

The patient was managed with conservative treatment, including management for dehydration, nil per oral status, and nasogastric tube insertion. During this period, the patient reported the spontaneous passage of multiple worms and as a result the patient's symptoms and clinical condition improved. The patient also received a single dose of 400 mg of albendazole for three consecutive days, considering its potential to improve the cure rate in this case. A randomized controlled trial (RCT) conducted in an endemic area demonstrated that a three-dose regimen of albendazole significantly enhanced cure rates compared to one- or two-dose regimens.[24] Adult *Ascaris lumbricoides* worms do not multiply within the host; therefore, the infection in this patient should resolve following treatment if reinfection by ingestion of fecally contaminated food does not occur. Consequently, the first line of management emphasizes education regarding good hygiene and public health measures.[2]

One program aimed at addressing ascariasis is the deworming program. The deworming program in Southwest Sumba aligns with Indonesian Ministry of Health regulations, encompassing health promotion, helminthiasis surveillance, risk factor control, patient management, and mass drug administration for helminthiasis, specifically albendazole 200 mg for children aged 1 – 2 years and 400 mg for children older than 2 years.[3] In this case, the patient missed the routine deworming program provided at the community health center, and it is unknown whether a mass drug administration program was conducted at his school. This emphasizes that ascariasis remains a problem and that the public health sector in Southwest Sumba must intensify efforts to educate the community about hygiene and improve public health.

Conclusion

Ascariasis, a widespread soil-transmitted helminth infection, disproportionately affects children with poor access to sanitation and hygiene, leading to significant long-term health consequences. This case of a preschool-aged boy underscores the challenges of diagnosing and managing partial bowel obstruction due to ascariasis in a resource-limited setting. Intestinal obstruction is a serious complication of ascariasis that can be identified using basic diagnostic tools, and an appropriate approach can help prevent life-threatening outcomes. Furthermore, this case highlights the need to strengthen preventive measures such as deworming programs, while also emphasizing the importance of improving sanitation, hygiene, and access to healthcare, particularly in endemic regions like Southwest Sumba. Enhancing public health initiatives, including consistent deworming campaigns and community education, is crucial for reducing the burden of ascariasis and its associated complications.

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Conflict of Interest

None declared

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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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