Journal of Communicable Diseases
Volume 57, Issue 3 - 2025, Pg. No. 140-145
Peer Reviewed & Open Access Journal

Research Article

Microbial, Immunological, and Environmental Risk Factors for Chronic Erosive-Ulcerative Gastrointestinal Tract Lesions in Children

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DOI: https://doi.org/10.24321/0019.5138.202579

I N F O

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How to cite this article:
Kozhonazarova G, Uzakov O, Alymbaev E, Sulaymanov S, Malevannaya V, Terekhova O, Ongoyeva B, Abdurakhmanov A A A, Umashev S, Akmatova A, Chubakova B, Melisbekova A, Vityala Y. Microbial, Immunological, and Environmental Risk Factors for Chronic Erosive-Ulcerative Gastrointestinal Tract Lesions in Children. J Commun Dis. 2025;57(3):140-145.

Date of Submission: 2025-06-29
Date of Acceptance: 2025-09-15

A B S T R A C T

Introduction: Erosive-ulcerative lesions of the gastrointestinal tract (EUL-GIT) are a growing concern in paediatric populations, particularly in low-income countries.

Aim: This retrospective study aimed to identify the risk factors contributing to EUL-GIT advancement in children, measure their predictive significance, and assess therapeutic outcomes.

Methods: The study included 2,091 children aged 0–17 years with confirmed EUL-GIT, divided into three groups: oral cavity (n=648), gastroduodenal (n=1,405), and colonic (n=38). A control group of 166 children with functional GI disorders was also assessed. Clinical evaluation, endoscopic assessment, and laboratory tests were performed. A predictive coefficient (PC) was used to evaluate the predictive value of each factor.

Results: The key predictors of oral lesions were decreased *Lactobacillus spp*. (PC=101.7), increased *Staphylococcus spp*. (PC=95.4), *Clostridium spp*. (PC=88.9), and *Candida albicans* (PC=71.0). For gastroduodenal lesions, *H. pylori* infection (PC=102.0), belching (PC=129.0), epigastric pain (PC=89.7), and heartburn (PC=73.1) were high predictors. In colonic lesions, H. pylori infection (PC=72.7), SIBO (PC=66.8), and elevated IL-4 (PC=68.5) were notable.

Conclusion: The PC model effectively assessed the risk and directed treatment strategies. A multidisciplinary approach involving infection management, nutritional support, and immune modulation is essential for high-risk children.

Keywords: Erosive-ulcerative lesions, gastrointestinal tract, children, Helicobacter pylori, immune dysregulation, microbial factors

Introduction

In paediatric populations erosive-ulcerative lesions of the gastrointestinal tract (EUL-GIT) are becoming increasingly worrisome worldwide, particularly in low- and middle-income countries, where gastrointestinal infections from communicable diseases remain common. These lesions, ranging from oral aphthous ulcers to inflammation in the duodenum and colon, often have chronic or recurring patterns and are associated with significant health problems, nutritional deficiencies, and developmental delays in children.[1,2]

Microbial infections, particularly those caused by *Helicobacter (H.)* pylori, play a crucial role in gastroduodenal and colonic erosion. In developing countries, up to 70% of children are colonised by *H. pylori* by the age of 10 years, leading to chronic gastritis, peptic ulcer disease, and dyspeptic symptoms.[3,4] Similarly, small intestinal bacterial overgrowth (SIBO) and oral dysbiosis, marked by excess opportunistic microorganisms such as *Staphylococcus aureus*, *Clostridium spp.*, and *Candida albicans*, have been linked to recurrent oral ulcers and functional bowel disorders in children.[5,6]

Beyond microbial causes, the host's immune response, particularly increased cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and IL-4, is pivotal in influencing inflammation, leading to mucosal damage and lesion persistence[7,8] These immune mediators indicate disease severity and present possible therapeutic targets for high-risk paediatric populations.

Parasitic infections like *Giardia lamblia* and *Enterobius vermicularis* add complexity by causing mucosal damage, hindering nutrient absorption, and altering immune responses.[9] This issue is particularly relevant in areas with inadequate sanitation, as multiple infections can worsen gastrointestinal damage.[10,11]

The impact of EUL-GIT on children extends to public health. In Kyrgyzstan, GI issues are the second most common cause of illness in children, after respiratory diseases, with a significant proportion of chronic erosive and ulcerative conditions.[12,13] Challenges such as delayed diagnosis, limited endoscopic access, and insufficient recognition of microbial factors impede timely treatment. Although there is knowledge about specific pathogens and inflammatory agents, few studies have systematically assessed predictive risk factors, including microbial, immunological, and environmental factors, for paediatric EUL-GIT persistence. Understanding this hierarchy is crucial for risk assessment, early intervention, and minimisation of long-term GI diseases in children.

This study aimed to (1) identify high- and medium-risk factors contributing to EUL-GIT advancement in children, (2) measure their predictive significance using a coefficient model, and (3) assess outcomes from therapeutic approaches, focusing on communicable causes and immune dysregulation.

Methods

This retrospective observational study was conducted at the National Centre for Maternal and Child Health in Bishkek, Kyrgyzstan, between October 2024 and February 2025. This study identified microbial and immunological factors that predict chronic progression in children with EUL-GIT. The study complied with the Declaration of Helsinki (2013), and the patients provided informed consent. This study was approved by the Bioethics Committee of the International Higher School of Medicine (Protocol No. 15, dated September 18, 2024).

The study included 2,091 children aged 0–17 years who were hospitalised with confirmed EUL-GIT. Patients were divided into three groups: Group 1 included 648 children with oral cavity lesions, including recurrent aphthous stomatitis; Group 2 included 1,405 children with gastroduodenal erosive lesions, including gastritis, duodenitis, gastroduodenitis, and peptic ulcer disease; and Group 3 comprised 38 children with colonic erosive lesions, such as ulcerative colitis. A control group of 166 children with functional GI disorders but without structural lesions was assessed.

Participants had to be under 18 years of age, have confirmed erosive or ulcerative lesions through endoscopic and histopathological evaluations, and possess complete clinical, laboratory, and follow-up data. The exclusion criteria were patients under 18 years of age with immune deficiencies, cancers, metabolic disorders, and incomplete records.

Patients underwent a comprehensive clinical evaluation, including medical history and structured interviews regarding perinatal events, hygiene, diet, and family history of GI disorders. Pain was assessed using validated paediatric scales: the NIPS, CHEOPS, and VAS. For diagnosis, Group 2 patients underwent esophagogastroduodenoscopy, while Group 3 underwent colonoscopy. Abdominal and renal ultrasonography was performed to identify related abnormalities.

The laboratory tests included general and specialised analyses. General examinations included complete blood count, serum biochemical profiles (alanine transaminase, aspartate transaminase, and C-reactive protein), urinalysis, and stool tests for hidden blood, white blood cells, and

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202579

mucus. Specialised microbial evaluations involved a urease breath test (Helik test) for *H. pylori*, quantitative stool cultures for microbial composition (*Lactobacillus spp., Bifidobacterium spp., Escherichia coli (E. coli)*, *Staphylococcus spp., Clostridium spp., Candida albicans,* and *Bacteroides*), and oral swab cultures for dysbiosis pathogens. Parasitic infections such as *Giardia lamblia*, *Enterobius vermicularis*, and *Ascaris lumbricoides* were identified using stool microscopy and enzyme-linked immunosorbent assay (ELISA).

The lactulose hydrogen breath test was used to assess SIBO, with a hydrogen increase of 20 ppm within 90 min of lactulose consumption, indicating a positive result. Immunological profiling was performed to analyse serum cytokines using ELISA kits for IL-4, IL-6, and TNF-α, which are elevated in chronic inflammatory GI conditions.

To assess the predictive value of each factor in disease progression, a predictive coefficient (PC) was determined using the following formula: $PC = 100 \times \log(Se/Sp)$, where Se denotes sensitivity and Sp indicates specificity. The factors were classified into three categories: high risk (PC > 58), medium risk (PC 28–53), and low/minimal risk (PC < 28). Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarise the demographic and clinical data. Chi-square tests were applied to categorical variables, while t-tests or ANOVA were used for continuous variables. Correlation analyses were used to evaluate the relationships between microbial profiles, immunological markers, and chronic disease outcomes. Statistical significance was set at $p < 0.05$.

Results

This study analysed 2,091 paediatric patients with EUL-GIT. Most cases were found in the gastroduodenal area (Group 2), representing 67.2% (n = 1,405), followed by lesions in the oral cavity (Group 1); 30.9% (n = 648) and colon (Group 3); 1.8% (n = 38). The control group comprised 166 children with functional GI symptoms but without endoscopic or histological signs of ulceration.

From 2010 to 2024, there has been a steady increase in the number of children hospitalised with EUL-GIT. A linear regression analysis ($y = 14.933x + 29.253$, $R^2 = 0.9651$) forecasts that by 2028, EUL-GIT cases will increase 4.5 times compared to 2010, indicating an escalating disease burden in this paediatric group.

In Group 1, children with erosive and ulcerative oral cavity lesions showed disease recurrence and chronicity linked to intestinal and oral microbiota changes. Key predictors were decreased *Lactobacillus spp.* (PC = 101.7) and increased *Staphylococcus spp.* (PC = 95.4), *Clostridium spp.* (PC = 88.9), and *Candida albicans* (PC = 71.0) in faecal and oral cultures. Elevated *Bacteroides spp*. levels (PC = 62.3) indicated dysbiosis. Clinical factors, including poor diet (PC = 58.0) and GI issues in the first year (PC = 54.6), were significant predictors. Moderate risk factors included reduced *Bifidobacterium spp.* (PC = 42.3), *E. coli* (PC = 44.3), and SIBO (PC = 44.3) (Table 1). These findings show that oral lesion recurrence is driven by dysbiotic intestinal and oral conditions, which are worsened by poor nutrition and early GI challenges. Figure 1 shows the distribution of the predictive risk factors across the anatomical regions.

Table 1.Risk factors and predictive coefficients by patient group

S.No	Patient group	Risk factor	Predictive coefficient score	Risk category
1.	Group 1 (Oral Cavity)	Reduced Lactobacilli	101.7	High
2.	Group 1 (Oral Cavity)	Increased Staphylococcus spp.	95.4	High
3.	Group 1 (Oral Cavity)	Increased Clostridium spp.	88.9	High
4.	Group 1 (Oral Cavity)	Increased Candida albicans	71.0	High
5.	Group 1 (Oral Cavity)	Increased Bacteroides spp.	62.3	High
6.	Group 1 (Oral Cavity)	Poor diet quality	58.0	High
7.	Group 1 (Oral Cavity)	GI pathology <1 year	54.6	Medium
8.	Group 1 (Oral Cavity)	Reduced Bifidobacteria	42.3	Medium
9.	Group 1 (Oral Cavity)	Reduced Escherichia coli	44.3	Medium
10.	Group 1 (Oral Cavity)	SIBO	44.3	Medium
11.	Group 2 (Stomach and Duodenum)	Helicobacter pylori	102.0	High
12.	Group 2 (Stomach and Duodenum)	TNF-α	46.2	Medium
13.	Group 2 (Stomach and Duodenum)	IL-4	38.7	Medium

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202579

14.	Group 2 (Stomach and Duodenum)	IL-6	30.7	Medium
15.	Group 2 (Stomach and Duodenum)	Previous Hepatitis A infection	76.0	High
16.	Group 3 (Colon/Intestine)	Parasitic Infection (Giardia, Enterobius)	36.5	Medium

GI – *Gastrointestinal*, SIBO – *Small intestinal bacterial overgrowth*, IL-6 – *Interleukin*-6, TNF-α – *Tumor necrosis factor-alpha*, IL-4 – *Interleukin*-6

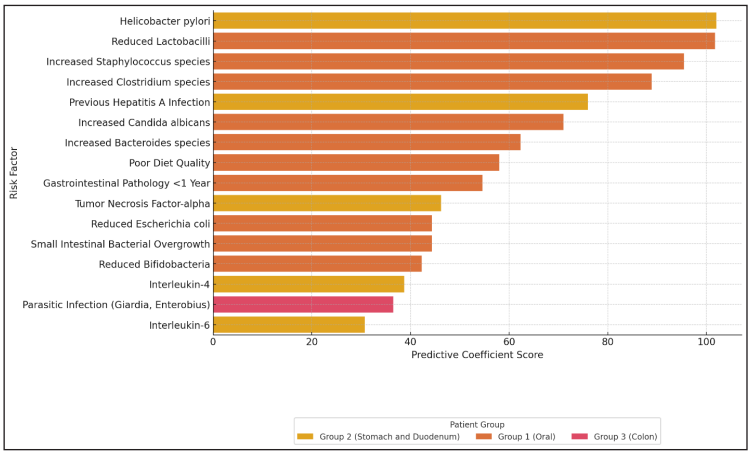

Figure 1.Predictive coefficient scores for risk factors associated with EUL-GIT in children

In Group 2, which focused on gastroduodenal erosive conditions such as gastritis, duodenitis, and peptic ulcers, infection with *H. pylori* was the key predictor of chronicity (PC = 102.0). High predictive values were found for belching (PC = 129.0), epigastric pain (PC = 89.7), and heartburn (PC = 73.1). Hepatitis A infection was significantly correlated with disease persistence (PC = 76.0). Microbiological analysis showed elevated *Staphylococcus spp*. in stool cultures (PC = 63.2), overlapping with Group 1, indicating cross-regional dysbiosis. Cytokine profiling showed moderate-risk elevations in TNF-α (PC = 46.2), IL-4 (PC = 38.7), and IL-6 (PC = 30.7), suggesting the role of systemic inflammation in gastroduodenal lesions. These findings indicate that infectious agents, proinflammatory responses, and microbial imbalance drive chronic upper GI disease in children.

In Group 3, patients with colonic conditions, such as ulcerative colitis, showed risk factors similar to those in the other groups. *H. pylori* infection (PC = 72.7) and SIBO (PC = 66.8) were notable. Immune dysregulation was significant, as indicated by high predictive coefficients for IL-4 (PC = 68.5), IL-6 (PC = 59.4), and TNF-α (PC = 48.5). Parasitic infections (PC = 36.5), especially *Giardia lamblia* and *Enterobius vermicularis*, were important, indicating susceptibility to infectious and environmental factors. Despite the small patient sample size, the strong predictive value of these factors suggests a synergistic relationship between chronic microbial colonisation and immune activation in lower GI tract inflammation.

Outcomes were categorised according to risk levels. In Groups 1 and 2, children with high and medium risk factors improved with H2-histamine blockers and proton-pump inhibitors, achieving remission in 82.3% of cases after 30 days of treatment. In Group 3, 5-aminosalicylic acid therapy led to remission in 70% of patients; however, 40% of high-risk children required corticosteroids or immunosuppressants. Probiotic therapy with *Saccharomyces boulardii* and specific antibiotics, such as metronidazole, was effective in 81% of patients with SIBO. A subgroup of 112 children with cytokine elevation required anti-relapse treatment for 30–45 d. PC stratification enabled the early detection of at-risk children with chronic EUL-GIT and allowed the implementation of treatment plans based on microbial and immunological profiles.

Discussion

This study examined the predictive microbial and immunological elements contributing to chronic EUL-GIT in children. The results highlight the role of microbial imbalance, *H. pylori* infection, cytokine-driven inflammation, and parasitic infections in these lesions, highlighting the risk profiles for the oral cavity, gastroduodenum, and colon.

The high occurrence of *H. pylori* in children with gastroduodenal lesions (PC = 102.0) supports global research showing that colonisation rates reach 80% in children from low-income areas by age 10.[3] *H. pylori* compromise the gastric mucosa through urease activity, cytotoxins, and neutrophil infiltration, increasing the risk of peptic ulcer disease and chronic gastritis.[4,15] The predictive value of epigastric symptoms, such as belching, pain, and heartburn, reflects trends in paediatric groups, where symptom severity correlates with mucosal damage.[16]

ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202579

This study identified oral and intestinal dysbiosis as significant risk factors for recurrent oral ulcers and functional bowel disorders in children. The findings showed increased *Staphylococcus spp., Clostridium spp*., and *Candida albicans* and decreased *Lactobacillus spp*. and *Bifidobacteria,* aligning with research on the altered microbiota's impact on mucosal inflammation.[17] These changes reduce the protective butyrate-producing commensals, allowing pathogenic strains to trigger inflammatory pathways.

The identification of SIBO as a moderate-to-high-risk factor aligns with evidence that bacterial overgrowth can alter gut permeability and trigger systemic inflammation, leading to mucosal damage.[18] SIBO is associated with post-infectious functional gastrointestinal disorders in children, particularly when untreated early.[19] Immune dysregulation is a key mechanism in colonic and gastroduodenal EUL-GIT. Increased cytokines (IL-4, IL-6, and TNF-α) show moderate to high predictive value, highlighting the inflammatory pathway shared with chronic paediatric conditions like Crohn's disease and ulcerative colitis.[20] IL-6 and TNF-α induce mucosal apoptosis and recruit leukocytes, contributing to prolonged lesion healing.[21]

Parasitic infections, particularly *Giardia lamblia* and *Enterobius vermicularis*, highlight the need for environmental cleanliness to address gastrointestinal issues in children. These parasites compromise the epithelial barrier, hinder nutrient uptake, and enhance immune sensitisation, causing gastrointestinal symptoms.[22] This finding aligns with a case-control study by Kotloff et al., which identified Giardia and protozoa as major contributors to persistent diarrhoea and growth retardation in developing areas.[23]

This study confirmed the success of stratified treatments, with acid suppression therapy, probiotics, and antimicrobial or anti-inflammatory agents achieving remission in over 80% of high-risk patients in Groups 1 and 2. The effectiveness of 5-aminosalicylic acid and immunosuppressive agents in Group 3 patients with colonic lesions mirrors the strategies used in juvenile idiopathic IBD.[24] In this study, the PC model provides a stratification tool, helping clinicians with early detection. Such models are increasingly important in precision paediatric gastroenterology for predicting complications and enhancing outcomes.

This study has some strengths and limitations. As a single-centre, retrospective study, there is a risk of selection bias. The small sample size of Group 3 might have reduced the statistical power. Limited information on dietary intake, socioeconomic status, and long-term follow-up may have affected the microbial and inflammatory markers.

Conclusions

The interaction between microbial imbalance, immune activation, and environmental factors plays a crucial role in the pathogenesis of paediatric EUL-GIT. The PC model effectively assesses risk, forecasts chronic conditions, and directs treatment strategies. A multidisciplinary approach involving infection management, nutritional support, and immune modulation is essential for high-risk children. Future prospective research, including metagenomic and cytokine analysis, is required to improve diagnostic and treatment accuracy.

Source of Funding: None

Conflict of Interest: None

Authors Contribution: Conceptualization, Gulsara Kozhonazarova, Orozali Uzakov, and Erkin Alymbaev; methodology, Shayirbek Sulaymanov, Valeria Malevannaya, and Oksana Terekhova; data collection, Bermet Ongoyeva; writing—original draft preparation, Almaz Abdurakhmanov AA, Samat Umashev, Altynai Akmatova, Baktygul Chubakova, Aliya Melisbekova, and Yethindra Vityala; writing—review and editing, Yethindra Vityala. All authors have read and agreed to the published version of the manuscript.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: None

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ISSN: 0019-5138
DOI: https://doi.org/10.24321/0019.5138.202579