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Oral and Intestinal Microbiocenosis in Children with Erosive and Ulcerative Oral Diseases

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Abstract: Erosive and ulcerative oral diseases, such as recurrent aphthous stomatitis and herpetiform ulcers, are common in children and significantly affect their quality of life. This observational study investigated the relationship between oral and intestinal microbiocenosis and the onset of these conditions in 648 children aged 1–16 years. Microbiological examination revealed dysbiotic changes in the oral and gut microbiota of affected children compared to controls, with a significant decrease in beneficial commensal bacteria, particularly *Bifidobacterium* and *Lactobacillus* species, and an increase in opportunistic pathogens, such as *Staphylococcus* and *Candida* spp. The reduction in *Bifidobacteria* and *Lactobacilli* in both oral and stool samples supports the concept of a gut-oral microbial axis in the manifestation of these diseases. These findings suggest that systemic factors, such as intestinal microbial imbalance, may contribute to the development of recurrent oral lesions in children. Incorporating microbiota analysis into the diagnostic process and considering targeted treatments, such as probiotics and dietary adjustments, could improve clinical outcomes. This study emphasizes the importance of a multidisciplinary approach involving collaboration among ENT specialists, pediatric dentists, gastroenterologists, and microbiologists to comprehensively evaluate and manage these conditions in children. Further research is needed to elucidate the causal relationships between microbial shifts and disease flares and to develop personalized microbiota-based interventions for pediatric patients with erosive and ulcerative oral diseases.

Keywords: Recurrent Aphthous Stomatitis; Herpetiform Ulcers; Oral Microbiota; Gut Microbiota; Pediatric; Dysbiosis

1. Introduction

Erosive and ulcerative oral diseases in children, such as aphthous stomatitis and herpetiform ulcers, are clinically significant because of their frequency and impact on pediatric health. These conditions manifest as painful lesions on the oral mucosa, causing difficulties in eating, speaking, and maintaining oral hygiene, which are essential for children's growth and development [1,2].

Recurrent aphthous stomatitis (RAS) is the most common ulcerative condition affecting the oral mucosa in children worldwide. It presents as recurring round or oval sores with red halos and pseudomembrane-covered

centers. Different forms include minor, major, and herpetiform types, with the latter showing clusters of small ulcers resembling herpetic lesions without a viral cause [3,4]. These self-resolving ulcers often recur and cause pain, leading to reduced nutritional intake in young patients [5]. Treatment remains challenging as the cause is unclear, although genetic predisposition, immune dysfunction, nutritional deficiencies (in iron, vitamin B12, and folate), and systemic diseases may trigger it [3–5].

Herpetiform ulcers, despite their name, differ from herpes simplex virus infections. These ulcers are more numerous and painful, often merging into larger erosive areas and recurring frequently. Accurate differentiation from conditions such as primary herpetic gingivostomatitis is essential for proper treatment and prognosis [2,3].

In clinical practice, erosive and ulcerative lesions in the mouth may indicate systemic diseases. Children with autoimmune conditions such as Behçet's disease, celiac disease, Crohn's disease, and periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA) often experience recurrent ulcers resembling aphthous ulcers [4,6–8]. While oral ulcers in patients with celiac disease may not differ from those in healthy individuals, they can indicate the effectiveness of a gluten-free diet, highlighting the need for a systemic assessment [8]. Severe ulcerations may indicate immune system issues or nutritional deficiencies, necessitating comprehensive diagnosis and treatment [4,5].

From a therapeutic standpoint, there is no cure for RAS or herpetiform ulcers; treatments focus on alleviating pain, speeding up healing, and reducing recurrence. Topical corticosteroids and antimicrobial mouth rinses are the primary treatments, whereas systemic medications are used in severe cases. New approaches, such as immunomodulators and probiotics, are being explored but require validation [9,10].

The global impact of erosive and ulcerative oral conditions, such as RAS and herpetiform ulcers, is significant among children, although epidemiological data from Kyrgyzstan remain unavailable. Worldwide, RAS affects 10% to 20% of the population, with prevalence rates varying by region, age, and diagnostic criteria [4,10]. A U.S. survey of children aged 2 to 17 years found a RAS prevalence of 1.64%, making it one of the most common oral mucosal lesions [11]. These findings highlight RAS as an idiopathic condition that causes considerable morbidity in the pediatric population.

The oral–gut axis describes bidirectional interactions between the mouth and gastrointestinal (GI) tract through their microbiomes and host immune system. The oral cavity and GI tract contain site-specific microbial populations that affect immune responses, mucosal barriers, and metabolism. Disruptions at either site can impact the other through systemic immune communication, shared mucosal immunity, and circulating microbial metabolites, creating a connection between oral and gut health [12–14].

Intestinal microbiocenosis refers to the community of microorganisms in the gut, primarily comprising the *Bacteroidetes* and *Firmicutes* phyla. The composition of these microbes shifts with age and environmental factors [14,15]. These commensal organisms shape both innate and adaptive immune responses through various mechanisms. The gut microbiota aids in the differentiation of regulatory T cells and tolerogenic antigen-presenting cells, which help suppress inappropriate immune responses and maintain self-tolerance [12,13].

Microbial dysbiosis, characterized by an imbalance between commensal and opportunistic microbial communities, affects both systemic and local immune responses. This concept is essential for understanding mucosal health, particularly in the gut and oral cavities, which contain diverse microbiota that are vital for immune homeostasis. Under normal conditions, commensal microbiota in these regions educate and regulate the host immune system by balancing inflammatory pathways. These microbes enhance epithelial barrier integrity, stimulate anti-inflammatory metabolite production, and trigger tolerance mechanisms that prevent inappropriate immune activation [14–16]. These interactions shape mucosal immunity by promoting the production of secretory IgA and the differentiation of regulatory T cells, thereby contributing to immune balance [14,17].

Dysbiosis is marked by decreased microbial diversity, reduced beneficial commensals, and overgrowth of opportunistic species, such as Fusobacterium nucleatum or Prevotella intermedia, in the mouth. In the GI tract, dysbiosis causes increased intestinal permeability ("leaky gut"), allowing microbial products such as lipopolysaccharides to enter the systemic circulation, triggering immune activation and chronic inflammation [14,18]. Oral dysbiosis intensifies inflammatory responses, disrupts innate immune signaling, and weakens the mucosal epithelium, leading to erosive and ulcerative lesions in RAS and herpetiform ulcers [18,19].

Recent findings have highlighted a reciprocal gut-oral axis, where disruptions in the gut microbiota affect oral mucosal immunity and vice versa. Patients with inflammatory bowel disease (IBD) experience intestinal dysbiosis linked to aphthous-like oral ulcers, indicating a systemic immune imbalance [9]. Oral microbial imbalances can

reflect or contribute to systemic inflammation, influencing diseases in distant mucosal regions through immune cells and cytokine signaling [20]. Research has shown altered oral microbial patterns in Behçet's syndrome and RAS, where mucosal immune responses and microbial compositions indicate changes that may trigger mucosal diseases [18,19].

In IBD, up to half of pediatric patients experience oral symptoms, such as aphthous-like ulcers and angular cheilitis, associated with disease activity and gut microbial imbalance [9, 18]. Research has begun to connect IB syndrome and small intestinal bacterial overgrowth with oral conditions such as bad breath and ulcers, although the mechanisms require further study [21,22].

Awareness of the gut-oral axis and its role in mucosal immunity is growing; however, research on oral microbiocenosis changes during erosive oral diseases in children remains limited. Microbiome studies have primarily focused on adults, leaving gaps in our understanding of pediatric oral microbial communities in conditions such as RAS, herpetiform ulcers, and ulcerations linked to systemic diseases such as IBD and celiac disease.

Scientific understanding is limited because ear, nose, and throat (ENT) approaches rarely include GI health or microbiological assessments of chronic oral mucosal lesions. ENT specialists focus on infections, injuries, and allergies, prioritizing symptom relief with topical pain relievers or antimicrobials. Microbiome-related causes of chronic erosive lesions, especially those linked to gut dysbiosis, are rarely considered during ENT consultations. Evidence shows that gut microbiota imbalances impact oral mucosal immunity, contributing to the development of persistent lesions [9, 22, 23]. Incorporating gut microbiome analysis with oral microbial studies can help identify dysbiosis pathways affecting oral diseases and reveal new biomarkers and treatments. Altering gut flora with probiotics has shown promise in treating oral aphthous-like ulcers in patients with IBD [19]. Understanding microbial changes during disease cycles in children will help create personalized treatments and improve prognosis.

This study aimed to investigate changes in oral and intestinal microbiocenosis in children with erosive and ulcerative oral diseases and examine the relationship between GI dysbiosis and recurrent stomatitis onset. By comparing the microbial profiles of these children with those of children with functional GI disorders but no oral lesions, this study sought to identify dysbiotic patterns that could contribute to mucosal damage and inform diagnostic and therapeutic strategies relevant to ENT practice.

2. Methods

This observational study used prospective and retrospective elements to investigate the influence of gastrointestinal and oral microbiocenosis in children with erosive and ulcerative oral conditions. Conducted from 2012 to 2024 at the National Center for Maternal and Child Health in Bishkek, Kyrgyzstan, the study included 648 children aged 1–16 years.

Group I comprised pediatric patients clinically diagnosed with erosive and ulcerative oral diseases affecting the oral cavity. This included conditions such as minor and major aphthous stomatitis, herpetiform stomatitis, recurrent aphthous ulcers, and recurrent muconecrotic periadenitis, which are categorized under ICD-10 code K12.0. The children experienced recurrent mucosal ulceration and were assessed prospectively through direct microbiological sampling or retrospectively through chart review. This group served as the main comparison cohort to assess microbial differences in children without oral mucosal disease.

In this study, 100 children underwent clinical assessments in an outpatient environment, and biological samples were collected for testing. An additional 548 children underwent retrospective analysis, in which medical records were examined to determine the clinical features, diagnostic classifications, and patterns related to erosive and ulcerative oral conditions. For comparison, a control group of 50 children was formed, matched by age and sex, who had been diagnosed with functional gastrointestinal disorders such as irritable bowel syndrome and functional dyspepsia. All individuals in the control group were verified to have no oral mucosal lesions during the examination.

To evaluate the local and systemic microbial profiles, samples were collected from the mouth and digestive tract. Sterile cotton applicators were used to collect oral swabs from the buccal mucosa and gingivolabial sulcus, while stool samples were gathered in sterile containers within two hours after defecation. All samples were transported to the microbiology laboratory under controlled temperature conditions and analyzed using standard aerobic and anaerobic culturing techniques.

Quantitative cultures were used to assess the colony-forming units (CFUs) of various microorganisms. Among

the identified commensal organisms, *bifidobacteria*, *lactobacilli*, and common strains of *Escherichia (E.) coli* were identified. Opportunistic and conditionally pathogenic organisms, including *Staphylococcus* species (*S. aureus*, *S. epidermidis*, *S. saprophyticus*, and *S. haemolyticus*), *Candida* species, *Clostridia*, and *Bacteroides*, were isolated and measured. Microbial counts were evaluated based on the pediatric reference values. Reductions were characterized as less than 10^9 CFU/mL for *bifidobacteria*, less than 10^7 CFU/mL for *lactobacilli*, and deviations from 10^7 to 10^8 CFU/mL for *E. coli*. Overgrowth of opportunistic flora was defined as a concentration exceeding 10^4 CFU/mL.

Data analysis was performed using SPSS software (version 25.0, IBM Corp., Armonk, NY). Descriptive statistics were used to summarize the prevalence and reduction rates of microbes in the experimental and control groups. Statistical significance was set at $p < 0.05$. This analysis assessed whether dysbiotic patterns in the oral and intestinal flora were significantly associated with erosive and ulcerative lesions in pediatric patients.

3. Results

Among 648 children diagnosed with erosive and ulcerative oral diseases, aphthous stomatitis was the most common, affecting 47.8% of the cases. Herpetiform stomatitis and recurrent aphthous ulcers accounted for 31.5% and 15.4% of all cases, respectively. A smaller proportion (5.2 %) had recurrent muconecrotic periadenitis. This distribution highlights a clinical pattern primarily characterized by recurrent aphthous ulcers.

Microbiological examination revealed dysbiotic changes in the oral and gut microbiota of children in the erosive and ulcerative oral disease group compared to the controls. All children (100%) with erosive and ulcerative oral diseases showed decreased *Bifidobacteria* in oral and fecal samples, whereas in the controls, reductions occurred in 36% of oral samples and 18% of stool samples. These differences were statistically significant ($p < 0.001$ for both sites), indicating a severe disruption of essential commensal populations among affected children.

In the erosive and ulcerative oral disease group, *lactobacilli* decreased in 82% of oral samples and 73% of stool samples, whereas the controls showed reductions of 4% and 52%, respectively. The oral microbiota decrease was highly significant ($p < 0.001$), whereas the stool reduction was moderately significant ($p = 0.0021$). These results indicate that *lactobacilli*-mediated mucosal immune support is weakened in children with oral ulcerative lesions.

In contrast, *E. coli* showed only moderate changes. The decrease in *E. coli* was more pronounced in patients with erosive and ulcerative oral diseases (32% oral, 48% stool) than in controls (20% and 40%, respectively), although these variations were not statistically significant ($p = 0.0543$ for oral; $p = 0.3187$ for stool). This suggests that *E. coli* may play a less definitive role in the observed dysbiosis.

No statistically significant differences were observed between the groups for *Bacteroides* and *Clostridia*. For erosive and ulcerative oral diseases, *Bacteroides* were reduced in 26% of oral samples versus 16% in controls ($p = 0.0862$), while *Clostridia* showed slight differences (21% versus 26%, $p = 0.4676$). In stool samples, both taxa remained absent or showed no change in reduction rates across groups, with non-significant p-values ($p = 1.0$) (**Table 1**).

Table 1. Comparison of microbial reductions in oral and intestinal samples from children with erosive and ulcerative oral diseases versus controls.

No.	Microorganisms	Oral			Stool		
		Group I (%)	Control (%)	*p*-Value	Group I (%)	Control (%)	*p*-Value
1.	*Bifidobacteria*	100	36	0.0001	100	18	0.0001
2.	*Lactobacilli*	82	4	0.0001	73	52	0.0021
3.	*Escherichia coli*	32	20	0.0543	48	40	0.2654
4.	*Bacteroides*	26	16	0.0862	0	0	1.0
5.	*Clostridia*	21	26	0.4676	0	0	1.0

Although *Staphylococcus* and *Candida* showed significant increases in the group with erosive and ulcerative oral diseases based on absolute overgrowth percentages, statistical tests for "reduced counts" were not applicable, as they represent opportunistic overgrowths rather than deficiencies. The statistically validated findings confirm that substantial decreases in protective commensals, especially bifidobacteria and lactobacilli, are closely linked to erosive and ulcerative oral disease onset in children, reinforcing a microbial pathogenic hypothesis of mucosal compromise (**Table 2**).

Table 2. Comparison of microbial overgrowth in oral and intestinal samples from children with erosive and ulcerative oral conditions versus controls.

No.	Microorganisms	Oral		Stool	
		Group I (%)	Control (%)	Group I (%)	Control (%)
1.	*Staphylococcus* spp.	72	8	89	8
2.	*Candida* spp.	88	8	96	11

Opportunistic overgrowth shows a pathogenic transformation in the microbiota of children with erosive and ulcerative oral conditions. *Staphylococcus* spp. were detected at high levels in 72% and 89% of oral and stool samples from Group I, respectively, versus 8% in both sample types in the control group. *Candida* spp. showed significant overgrowth in 88% of oral samples and 96% of stool samples in affected children, whereas it remained low in controls at 8% and 11%, respectively. These differences indicate that dysbiosis in children with oral mucosal diseases involves reduced beneficial bacteria and colonization by opportunistic pathogens, which may worsen mucosal inflammation and hinder epithelial recovery.

Although this observational study cannot determine causation, the changes observed in the oral and gut ecosystems prompt inquiries about the direction and systemic nature of dysbiosis. The reduction of *Bifidobacteria* and *Lactobacilli* in both areas, coupled with an increase in *Staphylococcus* and *Candida*, supports the idea of a gut-oral microbial axis affecting the manifestation of the disease. This could have vital consequences for ENT specialists, as oral ulcers in children may indicate an underlying gastrointestinal imbalance rather than a localized infection.

The current findings underscore the potential avenues for diagnosis and treatment. The recurring dysbiotic patterns in Group I indicate that microbial screening, especially for *bifidobacteria*, *lactobacilli*, and opportunistic overgrowth, could supplement the assessment of chronic oral lesions in children. Moreover, strategies aimed at restoring the microbiota, such as customized probiotics or dietary changes, may be a valuable addition to standard treatments.

4. Discussion

This study showed that children with erosive and ulcerative oral diseases, such as RAS, herpetiform stomatitis, and recurrent muconecrotic periadenitis, exhibited imbalances in oral and gut microbiomes compared to healthy individuals. The findings revealed a decrease in beneficial commensal bacteria, especially *bifidobacteria* and *lactobacilli*, along with an increase in opportunistic pathogens, such as *Staphylococcus* spp. and *Candida* spp. This supports the theory that a systemic connection between the gut and oral microbiomes plays a role in the development of mucosal diseases.

The absence of *bifidobacteria* in oral and stool samples of affected children aligns with research identifying these bacteria as crucial for regulating mucosal immunity and maintaining epithelial barrier integrity [24–26]. *Bifidobacteria* block pro-inflammatory pathways and aid in the differentiation of regulatory T cells, which is essential for reducing mucosal inflammation [27]. The decrease in *lactobacilli*, which are important for producing lactic acid and antimicrobial peptides, indicates compromised mucosal immune defense in children with erosive and ulcerative oral conditions [28].

The gut-oral axis theory posits that gut microbiota imbalances can affect distant mucosal sites through systemic immune signals and metabolites [29]. Our study supports this theory, as children with erosive and ulcerative oral diseases showed similar reductions in beneficial commensals in both the oral and intestinal regions. Short-chain fatty acids, such as butyrate and propionate, produced by gut *lactobacilli* and *bifidobacteria*, are known for their anti-inflammatory properties and epithelial repair; their reduction is likely to increase mucosal vulnerability and oral lesion development [30,31].

Opportunistic pathogens, such as *Candida* and *Staphylococcus*, support the concept of dysbiosis-driven disease development. Fungal colonization links to disrupted competition among commensal organisms and mucosal inflammation. *Candida albicans* has been associated with RAS flare-ups in children and may worsen epithelial damage through biofilm formation and Th17-mediated responses [32]. High colonization rates in the oral and gut areas indicate a common pathogenic environment, facilitated by weakened immunity or damaged epithelial barriers.

The results of this study suggest that the gut serves as a mirror of immune health, contributing to oral diseases

through immune activation and microbial movement [33]. Microbial movement from the gut due to increased intestinal permeability occurs in IBD and celiac disease, both of which are associated with frequent oral ulcers in children [34,35].

Gut dysbiosis-induced systemic inflammation can increase cytokines, such as interleukin-6, interleukin-17, and tumor necrosis factor alpha, which are linked to oral cavity mucosal lesions. Research on pediatric Crohn's disease has shown elevated pro-inflammatory cytokines associated with aphthous-like oral ulcers before gastrointestinal symptoms appear [36]. This suggests that ENT and pediatric dental professionals should consider the systemic microbial environment when treating recurrent oral ulcers.

While there was a statistically significant reduction in *bifidobacteria* and *lactobacilli*, changes in *E. coli*, *Bacteroides*, and *Clostridia* were less marked. These bacteria play a crucial role in gut balance, but their function in the oral-gut microbial axis may be more variable or dependent on specific strains and the local immune environment [37]. The lack of significant differences in *Bacteroides* and *Clostridia* abundance might indicate the limitations of conventional culturing methods in identifying fastidious or less prevalent microbial populations.

Although this study used traditional culture-based microbiological techniques, we recognize that molecular methods, such as 16S rRNA gene sequencing, metagenomic shotgun sequencing, or quantitative Polymerase Chain Reaction, could provide a more detailed analysis of oral and intestinal microbiota. These techniques would enable the identification of hard-to-culture organisms, provide greater taxonomic detail, and reveal microbial functional pathways, deepening our understanding of the gut–oral microbial connection in pediatric erosive and ulcerative oral conditions.

This study has significant implications for clinical practice in several ways. Children with frequent oral ulcers unresponsive to standard topical treatments may benefit from comprehensive evaluations, including gut microbiota assessments. Incorporating probiotics targeting *bifidobacteria* and *lactobacilli*, along with dietary adjustments, could help reestablish microbial balance and reduce ulcer recurrence [38]. Probiotic treatments have shown improvements in gut-related symptoms and oral ulcers in conditions such as IBD and celiac disease [39]. Although experimental, personalized microbial therapies show potential as supplementary treatments for persistent oral mucosal conditions in pediatric patients.

This study highlights the importance of interdisciplinary collaboration. ENT and pediatric dental experts who identify oral mucosal lesions rarely include systemic microbial evaluations. Involving gastroenterologists, immunologists, and microbiome experts could enable the early identification of systemic factors, thereby improving treatment outcomes [40]. This study shows a connection between oral and intestinal dysbiosis in pediatric erosive oral conditions. The decrease in protective commensals and the growth of opportunistic organisms indicate a compromised mucosal immune environment. These findings support the need to shift ENT and pediatric dental practices from lesion management to a comprehensive approach considering the microbial ecosystem.

5. Limitations

This study provides insights into oral conditions and dysbiosis in children, although several limitations exist. As the study was observational, the design restricted the ability to infer causality. Although associations have been identified between dysbiotic patterns and mucosal diseases, the direction remains uncertain. It is not possible to determine whether microbiota changes lead to erosive lesions or whether mucosal inflammation alters microbial communities. The reliance of the study on conventional culturing methods may have underestimated the microbial diversity. Advanced techniques such as 16S rRNA sequencing could provide a more thorough characterization of microbial communities. The study did not evaluate immunological profiles, limiting our understanding of how the microbiota modulates the immune system in children with oral ulcers. The retrospective aspect introduces potential information bias, with data subject to variability in documentation. The study lacked longitudinal follow-up of microbial changes during the active and remission phases, leaving the temporal relationships between microbial shifts and disease flares speculative. Factors such as dietary intake, oral hygiene, antibiotic use, and socioeconomic status were not systematically controlled, potentially influencing microbial variability. Although the study population came from a single pediatric referral center in Kyrgyzstan, the absence of comparative data from other regions limits the generalizability of the findings across ethnic and geographic backgrounds. To enhance the understanding of microbial diversity and function, future studies should integrate molecular microbiome methods, such as 16S rRNA sequencing or metagenomic analysis, with traditional culture-based approaches. This combination could en-

hance diagnostic accuracy and support targeted microbiota-based treatment.

6. Clinical Implications

The results have significant clinical implications for pediatricians, otorhinolaryngologists, pediatric dentists, and gastroenterologists who treat recurrent oral mucosal diseases in children. When children exhibit recurrent erosive or ulcerative oral lesions, clinicians should consider underlying dysbiosis rather than viewing it as a localized mucosal problem. Examining only the oral cavity may overlook systemic factors, such as intestinal microbial imbalance. Stool microbiota screening or salivary microbial analysis could improve the diagnostic precision. This study shows that dysbiosis, marked by reduced *bifidobacteria* and *lactobacilli* and increased *Candida* and *Staphylococcus* spp., is linked to pediatric erosive oral diseases. Screening of gut and oral flora may be valuable in chronic stomatitis cases. Identifying microbial changes may help identify at-risk children and monitor treatment responses, particularly in those with IBD or celiac disease. ENT and dental specialists, who are often the first to assess children with oral symptoms, should understand the gut-oral microbiome connection to expand their differential diagnoses. This promotes multidisciplinary approaches, including referrals, when systemic symptoms occur. Integrating microbial perspectives could reveal underlying disorders such as PFAPA syndrome or Crohn's disease. While corticosteroids and antimicrobial mouth rinses help in the short term, long-term management may benefit from microbiota-targeted interventions, such as probiotics or dietary adjustments. Understanding that recurrent aphthous ulcers may stem from systemic dysbiosis helps prevent the overuse of antibiotics or corticosteroids. These findings emphasize the need for nutritional screening and counseling, addressing deficiencies, and promoting microbiota-supportive habits.

7. Conclusion

This study presents evidence that children with erosive and ulcerative oral conditions exhibit notable dysbiotic alterations in both the oral and intestinal microbiomes. There is a reduction in beneficial commensal bacteria, particularly *Bifidobacterium* and *Lactobacillus* species, alongside an increase in opportunistic organisms, such as *Staphylococcus* and *Candida* spp. This imbalance indicates that a disturbed microbial environment contributes to mucosal barrier dysfunction and inflammation. These results support a gut–oral microbial connection, wherein changes in the gut microbiota may affect oral mucosal immunity through inflammatory mediators, microbial metabolites, and compromised immune tolerance. Similar dysbiotic patterns observed in both areas indicate a significant bidirectional relationship in the pediatric population. The decrease in commensal bacteria among affected children compared to the minimal changes in the control group suggests that these microbial imbalances may play a causal role in the development of recurrent oral lesions. The lack of differences in conventional pathogens between the groups implies that crucial changes involve protective species rather than traditionally pathogenic bacteria.

The results of this study have significant implications for ENT and pediatric dental care. When children experience persistent oral ulcers unresponsive to treatment, providers should consider systemic microbial imbalances beyond the localized causes. Incorporating microbiota analysis can improve diagnostic precision and inform targeted treatments. These findings support a microbiota-based pathogenic model for erosive oral conditions in children. This approach highlights the importance of systemic assessment, probiotic treatment, and collaboration among specialists. Addressing oral and gut dysbiosis may be crucial for improving the clinical outcomes of children with oral mucosal diseases.

8. Recommendations

Pediatric patients with frequent erosive oral ulcers should be evaluated for microbiota imbalances using oral swabs and stool cultures to identify decreases in beneficial commensals and overgrowth of opportunistic organisms. Microbiota assessments should be incorporated into ENT and pediatric dental practices. ENT specialists, pediatric dentists, and pediatricians should collaborate with gastroenterologists, microbiologists, and immunologists to evaluate children with recurrent oral lesions. Integrated care pathways will help detect systemic factors such as IBD, celiac disease, and immunodeficiency syndromes. Clinical guidelines should include microbiome restoration strategies using age-appropriate probiotics and prebiotic-rich dietary counseling. Targeted probiotic supplementation may help restore immune tolerance in children with gut dysbiosis. Professional development for ENT and dental

clinicians should emphasize the role of the gut microbiota in oral immunity. Understanding the gut-oral axis will improve the identification of systemic triggers and reduce misdiagnosis. Affordable microbial testing tools are needed, particularly in resource-limited settings. Future research should include longitudinal studies that track changes in oral and gut microbiota in relation to ulcer flare-ups and treatment responses. Studies on probiotics, dietary interventions, and immunomodulatory agents should be given priority. Public health strategies should support early life microbiome development through breastfeeding, appropriate food introduction, and reduced exposure to antibiotics. Dietary counseling focusing on fiber-rich and anti-inflammatory foods, along with addressing nutrient deficiencies, is essential for the affected children.

Author Contributions

Conceptualization, G.K. and O.U.; methodology, E.A.; software, S.S.; validation, V.M.; formal analysis, O.T.; investigation, B.O., A.A.A.A., S.U., and A.A.; data curation, B.C.; writing—original draft preparation, A.M., and Y.V.; writing—review and editing, Y.V. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

The study complied with the Declaration of Helsinki, and this study was approved by the Bioethics Committee of the International Higher School of Medicine (Protocol No. 15, dated September 18, 2024).

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

Data Availability Statement

Data are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that there is no conflict of interest.

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