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Article

Polymicrobial Composition of Oral Microbiota in Ulceronecrotic Gingivostomatitis: Structure of Microbial Associations and the Role of Key Microorganisms

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Abstract: The aim of this study was to investigate the species composition of the oral mucosal microbiota and to characterize the structure of microbial associations in patients with ulceronecrotic gingivostomatitis. A clinical and microbiological examination was conducted, with samples collected from pathological lesions of the oral mucosa. Microorganisms were identified using standard bacteriological methods, determining their species affiliation and the nature of microbial associations. Results showed that all patients harbored representatives of both resident and transient microbiota, forming complex polymicrobial associations. In 24.3% of cases, associations included aerobic, anaerobic, and fungal microorganisms, while in 75.7% of cases, the complexes consisted of aerobic and anaerobic flora without fungal components. The average number of species in one association was 7–9, confirming the polymicrobial nature of the inflammatory process. Conclusion: Ulceronecrotic gingivostomatitis is characterized by the formation of complex microbial complexes, in which Fusobacterium nucleatum and Treponema vincentii play a leading role, interacting with other representatives of aerobic, anaerobic, and in some cases, fungal flora.

Keywords: ulceronecrotic gingivostomatitis, microbiocenosis, microbial associations, anaerobic flora, dysbiosis.

Introduction

Ulceronecrotic gingivostomatitis remains one of the most common forms of inflammatory and destructive lesions of the oral mucosa. According to the literature, it accounts for up to 70% of cases of oral mucosal integrity disorders encountered in dental practice.

The disease is an infectious-inflammatory process that primarily develops against the background of reduced systemic and local resistance of the body. In scientific sources, this pathology is described under various terms: ulcerative gingivitis, ulceronecrotic stomatitis, ulceromembranous stomatitis, fusospirochetal stomatitis, Plaut–Vincent stomatitis, and others.

Historical evidence indicates the long-standing existence of this nosological form. Presumably, as early as 401 BCE, during military campaigns, cases of ulcerative lesions of the oral mucosa were observed, accompanied by a pronounced fetid odor. Some of the first detailed medical descriptions of the disease date back to the late 18th century, when French military physicians provided a clinical characterization of the pathology observed among soldiers during military campaigns [1], [2].

In 1889, Frunwald first distinguished this pathology as an independent nosological entity, naming it "ulcerative stomatitis" and providing a detailed clinical description of the disease. In subsequent years, significant contributions to the study of its etiology were made by the German physician Plaut (1894) and the French bacteriologist Vincent (1895), who independently discovered spindle-shaped rods and spirochetes in the ulcerative lesions [3], [4], [5].

During the First and Second World Wars, the disease was widely recorded among military personnel, earning the popular name "trench mouth," reflecting its association with poor sanitary-hygienic conditions and reduced systemic resistance.

According to the prevailing view, the pathogenesis of ulceronecrotic gingivostomatitis is based on the symbiotic interaction of opportunistic microorganisms — Fusobacterium nucleatum and Treponema vincentii. Under normal conditions, these microorganisms are part of the oral microbiocenosis, predominantly located in the gingival sulcus, interdental spaces, periodontal pockets, carious cavities, root canals, and tonsillar crypts [6].

Under unfavorable conditions, their quantitative ratio changes: fusobacteria and spirochetes actively proliferate, beginning to dominate the microbial community. Triggering factors contributing to disease development include hypothermia, stress, trauma to the oral mucosa, surgical interventions, somatic diseases, and significant immune suppression.

Local defense factors also play an important role. Poor oral hygiene, sharp tooth edges, substandard prosthetic constructions, and chronic trauma to the oral mucosa significantly reduce its barrier function. Disruption of epithelial integrity facilitates microbial invasion into underlying tissues. This explains why ulceronecrotic gingivostomatitis more often develops in patients with unsanitary oral conditions and inadequate hygiene [7], [8].

Microbiological studies show that in this disease, smear samples from ulcer surfaces contain large amounts of spindle-shaped rods and spirochetes, which sharply predominate over other microbial species. Fusobacteria and spirochetes are obligate anaerobes. They localize not only on the ulcer surface and within dental plaque but are also capable of penetrating tissues. Spirochetes, in particular, penetrate deeper than other microorganisms, positioning themselves between epithelial cells and infiltrating underlying layers, emphasizing their crucial role in the progression of the inflammatory-necrotic process [9], [10].

However, despite the consistent detection of Fusobacterium nucleatum and Treponema vincentii in ulceronecrotic gingivostomatitis, the leading role of the fusospirochetal symbiosis in the etiology of the disease is not recognized by all researchers. Some authors consider this pathology to result from complex polymicrobial interactions against a background of immunological disturbances and pronounced oral dysbiosis [11].

Despite the introduction of modern antibacterial agents and improvements in local therapy methods, the treatment of ulceronecrotic gingivostomatitis remains a relevant problem in dentistry. In recent years, there has been a trend toward an increase in the incidence of this pathology, which may be associated with changes in the oral microbiocenosis structure, the growth of microbial antibiotic resistance, and insufficient study of certain pathogenetic links of the disease [12], [13].

The insufficient understanding of specific etiological aspects of ulceronecrotic gingivostomatitis necessitates a more in-depth analysis of the microbial composition of lesion sites. Therefore, the aim of this study was to investigate the species composition of the oral mucosal microbiota in patients with ulceronecrotic gingivostomatitis to support the rationale for the selection of local etiotropic therapy [14], [15].

Materials and Methods

Microbiological studies were conducted in the laboratory of medical microbiology at the Institute of Epidemiology and Infectious Diseases.

The study included 12 patients with a clinically confirmed diagnosis of ulceronecrotic gingivostomatitis. Samples for analysis were taken from the pathological lesions of the oral mucosa.

Sample collection was performed using sterile instruments, followed by placement in Amies transport medium with charcoal (Transport Medium w/Charcoal, HIMEDIA, India). Delivery to the laboratory was carried out as quickly as possible, following the rules for transporting anaerobic cultures.

Primary inoculation for the isolation of anaerobic microorganisms was performed on the following media:

1. Wilkinson–Chaldgren medium,
2. 5% blood agar,
3. Thioglycolate medium.

Quantitative assessment of microbial colonization was performed using the Gold sector streaking method. Anaerobic conditions were created using the GENbox anaer system (bioMérieux, France).

Plates with 5% blood agar were incubated at 37°C for 24 hours under aerobic conditions. Colony growth was evaluated according to:

1. Morphology,
2. Color,
3. Size,
4. Consistency,
5. Type of hemolysis.

Gram staining was performed. From each morphotype, a well-isolated colony was selected and subcultured:

1. On Wilkinson–Chaldgren medium under anaerobic conditions,
2. On 5% blood agar under aerobic conditions.

Microorganisms were considered obligate anaerobes if growth occurred under anaerobic conditions and no growth was observed under aerobic conditions. The growth of anaerobic cultures was assessed after 48 hours of incubation.

Primary identification of anaerobic microorganisms was performed using An-ident Discs (OXOID, UK): erythromycin (60 μg), rifampicin (15 μg), colistin (10 μg), penicillin (2 IU), and kanamycin (1000 μg). For final identification, commercial test systems API 20 A (bioMérieux, France) were applied. Taxonomic interpretation of results was carried out according to Bergey's classification.

Result and Discussions

During the microbiological analysis, representatives of both resident (permanent) and transient (temporary) oral microbiota were isolated and identified in all examined patients.

The data confirmed the pronounced polymicrobial nature of the inflammatory-necrotic process and the presence of complex microbial associations within the lesion sites. In total, 110 strains belonging to 21 microbial species from various taxonomic groups were isolated, confirming the pronounced polymicrobial character of the inflammatory-necrotic process.

In all patients, microorganisms were found exclusively within microbial associations of varying complexity. In 24.3% of cases, the associations included representatives of aerobic, anaerobic, and fungal microbiota, whereas in 75.7% of observations, microbial complexes consisted of a combination

of aerobic and anaerobic bacteria without a fungal component. On average, each association included 7–9 microbial species, indicating a significant disruption of the oral mucosal microbiocenosis.

Analysis of species composition revealed that lesion sites were dominated by representatives of resident microbiota, primarily facultative anaerobic oral streptococci (S. salivarius, S. sanguis, S. mitis, S. mutans), as well as anaerobic cocci (Peptococcus spp.) and anaerobic streptococci (Peptostreptococcus spp.).

The frequency of Staphylococcus aureus isolation was 41.7%. Representatives of the Enterobacteriaceae family were detected in 33.3% of patients.

Particular attention was given to the obligate anaerobes Fusobacterium nucleatum and Treponema vincentii, whose symbiosis is traditionally considered a leading etiological factor in ulceronecrotic gingivostomatitis. F. nucleatum was detected in 66.7% of cases, and T. vincentii in 58.3% of observations. These findings indicate their high prevalence but do not support a monocausal role, as they were found within complex polymicrobial associations.

Conclusion

1. Ulceronecrotic gingivostomatitis is characterized by a pronounced polymicrobial nature of the inflammatory process, with the formation of stable associations including 7–9 microbial species.
2. Lesion sites contain both resident and transient microbiota, indicating the development of a pronounced dysbiotic state of the oral mucosa.
3. Obligate anaerobes Fusobacterium nucleatum and Treponema vincentii are frequently detected in this disease; however, their role is realized within complex microbial communities that include aerobic, anaerobic, and, in some cases, fungal flora.
4. The findings expand current understanding of the etiology of infectious erosive-ulcerative lesions of the oral mucosa and justify the need for a comprehensive approach in selecting local etiotropic therapy.

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