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INTEGRATED CLINICAL AND LABORATORY EVALUATION OF PATIENTS WITH CHRONIC RECURRENT APHTHOUS STOMATITIS

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Abstract:
Chronic recurrent aphthous stomatitis (CRAS) is a common oral condition marked by periodic ulcerations, discomfort of varying intensity, and unpredictable healing durations. Clinical assessment reveals inconsistencies in the local immune system, such as altered lysozyme activity and deviations in immunoglobulin content within the saliva. Microbial studies identify diverse populations of aerobic and anaerobic organisms inhabiting ulcerated areas, potentially driving ongoing inflammation and secondary infections. Laboratory evaluations suggest that weakened mucosal immunity plays a role in the persistence and depth of tissue involvement. These findings emphasize the relevance of combining clinical, immunologic, and microbial data to develop more effective, personalized treatment strategies

Keywords: Chronic aphthous ulcers, oral immunity, lysozyme, salivary immunoglobulins, oral microbiota, diagnostic assessment

Materials and Methods:
The investigation included a total of 123 individuals clinically diagnosed with CRAS. Of these, 61 participants were from areas classified as environmentally at-risk, while the remaining 62 resided in regions with favorable ecological conditions. Selection criteria required a documented history of recurring aphthous lesions confirmed through both clinical observation and patient history.

Conclusion:

The results indicate substantial variations in clinical and immunological features between patients from ecologically disadvantaged zones and those from healthier environments. Subjects in the former group experienced more frequent ulcer recurrences, extended healing times, and intensified clinical symptoms. These were associated with significantly reduced levels of lysozyme and secretory IgA in their oral fluids. Moreover, a higher prevalence of anaerobic bacteria and opportunistic fungi was observed, underscoring the link between impaired immune response and microbial colonization in the pathogenesis of CRAS.

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