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The Contribution Of Opportunistic Microflora To The Development Of Angular Cheilitis

Takhirova Kamola Abrarovna

DSc (Doctor of Medical Sciences), Associate Professor of the Department of Hospital Therapeutic Dentistry, Tashkent State Medical University, Tashkent, Uzbekistan

Mirzakhodjaeva Nigina Firdavsovna

Basic Doctoral Student of the Department of Hospital Therapeutic Dentistry, Tashkent State Medical University, Tashkent, Uzbekistan

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Abstract: Angular cheilitis, or inflammation at the corners of the mouth, often becomes chronic and prone to recurrence, significantly impairing quality of life. Although its causes are numerous, microorganisms play a key role, primarily the fungus Candida albicans and the bacterium Staphylococcus aureus. These pathogens often act in concert to form persistent microbial colonies. This review summarizes current knowledge on how these microorganisms initiate the disease and why symptoms persist even after treatment. Particular attention is paid to biofilms—structures that allow microbes to establish themselves, evade treatment, and sustain the inflammatory process. Based on this, the practical conclusion is that the treatment of angular cheilitis requires consideration of the microbial factor, appropriate diagnostics, and the use of comprehensive treatment methods, rather than simply addressing symptoms.

Keywords: Angular cheilitis, Candida albicans, Staphylococcus aureus, biofilm, mixed microflora.

Introduction: Angular cheilitis is an inflammatory lesion of the skin and mucous membranes at the corners of the mouth. Typical manifestations include redness, softening of the skin (maceration), painful cracks, crusting, and severe discomfort. In clinical practice, this condition often becomes protracted and prone to repeated exacerbations, indicating the inadequacy of symptomatic treatment alone and the need for a comprehensive approach aimed at identifying and eliminating the underlying causes (1,7).

Factors contributing to the development of angular cheilitis include mechanical irritation (e.g., from maceration or microtrauma at the corners of the mouth), malocclusion, wearing dentures, increased salivation, vitamin deficiencies, and immune system disorders (1,7). All of these conditions make the tissues more vulnerable and contribute to the persistence of inflammation. However, in many cases, microorganisms play a decisive role, initiating symptoms and triggering relapses, as evidenced by the high frequency of pathogen detection in affected areas (2-4). The most frequently detected are yeast-like fungi of the genus Candida (primarily C. albicans) and the bacterium Staphylococcus aureus, which can occur both individually and in combination, forming stable microbial associations (2-4).

METHODS

A narrative review of the literature was conducted, focusing on three main areas:

1. Clinical data on the microbial composition of angular cheilitis and the frequency of pathogen detection;

2. Publications analyzing the causes of symptom recurrence after antimicrobial therapy and the role of predisposing conditions;

3. Experimental studies and reviews devoted to Candida-Staphylococcus dual biofilms and the mechanisms of their resistance.

The analysis included articles on clinical dentistry, dermatology, and microbiology, as well as current

reviews and clinical guidelines (1–12).

RESULTS

According to bacteriological studies, pathogenic microorganisms are detected in the affected area in most patients with angular cheilitis, often not singly, but in combination. One key study showed that Staphylococcus aureus and Candida albicans were isolated in a significant proportion of patients, and microbial contamination was detected in all patients with this diagnosis (3).

Later observations only reinforced these findings. Various microorganisms—including S. aureus, Candida spp., and streptococci—were detected in more than 80% of cases. S. aureus and representatives of the Candida genus remained the most common pathogens (4). Similar data are presented in current reviews and practice guidelines: the frequency of detection of Candida spp. Often exceeds 50%, and bacterial-fungal communities are directly associated with persistent inflammation (1,7).

If Candida spp. and/or S. aureus are detected in the corners of the mouth in a patient with characteristic symptoms, especially in cases of frequent relapses and a poor response to conventional treatment, this is a serious diagnostic signal (1,7).

The ability of microbes to form biofilms is increasingly being cited as one of the causes of chronic inflammation. Laboratory experiments have shown that Candida albicans not only actively attaches to tissues but also helps S. aureus colonize the same areas, forming stable mixed communities (8). These so-called dual-species biofilms are quite intricately structured: staphylococci are closely associated with Candida hyphae, which increases resistance to therapy and complicates the elimination of the infection (11).

Of particular interest is the phenomenon of "commensal protection": the Candida biofilm matrix literally shields S. aureus from the effects of antibiotics (10). At the same time, the fungal colonies themselves are resistant: the polysaccharide matrix (especially β-1,3-glucan) reduces sensitivity to antifungal agents (9).

As a result, treatment may only provide external improvement. Internally, the inflammatory focus persists. Additional irritation (maceration, excess salivation, minor trauma) can cause symptoms to return (5-7).

Microorganism/microbial association	**Frequency of detection in patients with AC, %**	**Sources**
Candida albicans	50–85	[1–4,7]
Candida spp. (no-*albicans*)	5–20	[1,4,7]
Staphylococcus aureus	30–60	[3,4,6]
Streptococcus spp.	15–35	[3,4]
Mixed infection (*Candida* + *S. aureus*)	25–50	[3–6]
Absence of clinically significant microflora	<10	[1,7]

Table 1. Frequency of microorganism detection in angular cheilitis (based on literature data)

Note: The range of results is due to differences in study design, microbiological diagnostic methods, and clinical sample composition.

Clinical data confirm that even after an effective course of treatment, angular cheilitis can recur. This is associated with residual microbial colonization and the effects of unfavorable conditions (5). A number of studies emphasize the need not only for therapy but also for a comprehensive diagnosis—including microbiological analysis and identification of factors that reduce the effectiveness of treatment, such as dentures, maceration, excess saliva, and unhealthy habits (6). Current clinical guidelines also emphasize

that sustained remission can only be achieved with a comprehensive approach, considering both the microbial and mechanical causes of the disease (1,7).

Microbiological Diagnosis

From a practical standpoint, microbiological testing (culture and/or microscopy) is warranted in a number of situations:

– if angular cheilitis persists for a long time or regularly recurs;

– if standard treatment has failed;

– if a combined fungal-bacterial infection is suspected;

– if the patient has factors that increase the risk of candidiasis or staphylococcal colonization (1, 6, 7).

In routine clinical practice, it is important to collect a swab from the lesion and examine it for at least the presence of Candida spp. and bacterial flora. In recurrent cases, antimicrobial susceptibility testing is also advisable, especially given the increasing resistance of microorganisms and the role of biofilms (6, 9).

Pathogenetic mechanism	Microorganisms involved	Clinical significance	References
Colonization of macerated skin at the oral commissures	*Candida albicans*, *S. aureus*	Initiation of inflammation and formation of fissures	[1,3,7]
Formation of stable biofilms	*Candida* spp., *S. aureus*	Prolonged disease course and reduced treatment efficacy	[8–11]
Synergistic interaction of microorganisms	*Candida* + *S. aureus*	Persistent infection and more pronounced tissue inflammation	[8,10,11]
Increased resistance to therapy	Biofilm-forming *Candida* species	Frequent relapses after treatment	[9–11]
Reactivation in the presence of local predisposing factors	Opportunistic microflora	Recurrent exacerbations associated with salivation and prosthetic appliances	[5–7]

Table 2. Pathogenetic role of microbial factor in angular cheilitis

DISCUSSION

Clinical and laboratory data increasingly confirm that the microbial factor is not a minor detail, but a crucial element in the development of angular cheilitis. Damaged skin at the corners of the mouth—from maceration, friction, and microcracks—becomes a convenient entry point for pathogens. The inflammation is then sustained not by single microbes, but by complex communities, most often combinations of Candida and Staphylococcus (1, 3, 5, 8, 10, 11).

This model helps explain why treatment is sometimes ineffective. Typical errors include:

– using only one drug, without considering the possible fungal-bacterial nature of the infection;

– too short courses of therapy, without eliminating persistent irritants;

– failure to culture and analyze patients with chronic or recurrent cheilitis (1, 6, 7).

For treatment to be truly effective, it is important to take a comprehensive approach. This means:

– confirming or ruling out the involvement of Candida spp. and S. aureus;

- eliminate local causes such as salivation, malocclusion, problems with dentures or their care;

- select the correct medications based on the expected composition of the microbial association, and use combination regimens if necessary;

- implement measures to reduce the risk of relapse (1,

6, 7).

CONCLUSIONS

1. Most patients with angular cheilitis have significant pathogens—most commonly Candida spp. and Staphylococcus aureus, often as part of mixed microbial associations (3, 4).

2. Such mixed biofilms demonstrate pronounced interactions: C. albicans promotes the attachment and colonization of S. aureus, which increases resistance to therapy and provokes relapses (8, 10, 11).

3. Effective patient care is impossible without microbiological verification (especially in protracted cases), selection of therapy taking into account the possible mixed nature of the infection, and mandatory correction of local conditions that support inflammation (1.5–7).

REFERENCES

1. Federico J.R., Basehore B.M., Zito P.M. Angular Cheilitis. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.
2. Devani A. Can you identify this condition? (обзор по АХ, клинические и этиологические аспекты). 2007.
3. Öhman S.C., Jontell M., Dahlen G. Angular cheilitis: a clinical and microbial study. (Scand J Dent Res / related dental journal record).
4. Oza N., Doshi J. Angular cheilitis: A clinical and microbial study. Indian J Dent Res. 2017.
5. Öhman S.C., Jontell M., Dahlen G. Recurrence of angular cheilitis. Scand J Dent Res. 1988;96(4):360–365.
6. Öhman S.C., Jontell M. Treatment of angular cheilitis: The significance of microbial analysis, antimicrobial treatment, and interfering factors. (Acta Odontol Scand). 1988
7. DermNet NZ. Angular cheilitis: Symptoms, causes, and management.
8. Kean R. et al. Candida albicans Mycofilms Support Staphylococcus aureus Colonization and Enhances S. aureus Biofilm Formation. Front Microbiol. 2017.
9. Mitchell K.F. et al. Role of Matrix β-1,3 Glucan in Antifungal Resistance of Biofilms. Antimicrob Agents Chemother. 2013.
10. Kong E.F. et al. Commensal Protection of Staphylococcus aureus against Antimicrobials by Candida albicans Biofilm Matrix. mBio. 2016.
11. Peters B.M. et al. Candida albicans–Staphylococcus aureus dual-species biofilms: architecture and interactions. 2010.
12. Baxter K.J. et al. Time-lapse mesoscopy of Candida albicans and Staphylococcus aureus dual-species biofilms reveals structural role for hyphae. Microbiology. 2024.