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Clinical and Microbiological Characteristics of Post-Chemotherapy Oral Mucositis and Evidence-Based Prevention Strategies

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Abstract Oral mucositis (OM) is a common and debilitating complication of chemotherapy, affecting up to 80% of patients and significantly impairing oral function and quality of life. Chemotherapy disrupts epithelial turnover, salivary defenses, and microbial balance, resulting in ulceration and inflammation. This review analyzes clinical features, microbiological associations, and prevention strategies using current evidence. Methods include synthesis of published studies, comparative clinical analysis, and microbiological data evaluation. Results confirm strong associations between OM severity, microbial dysbiosis, *Candida* overgrowth, and salivary dysfunction. Three evidence-based prevention models were analyzed. Comprehensive oral hygiene measures, probiotics, antifungal prophylaxis, and photobiomodulation reduce OM incidence by 40–63%. Conclusions emphasize the importance of early detection, microbial screening, and multimodal prevention strategies to decrease OM severity and improve patient outcomes.

Keywords Oral mucositis, Chemotherapy, Microbiome imbalance, Candida, Epithelial injury, Prevention

1. Introduction

Oral mucositis (OM) is one of the most clinically significant toxicities of chemotherapy. It develops in 40–80% of patients depending on treatment regimen, immune status, and oral conditions [1]. Pathophysiologically, OM results from chemotherapy-induced epithelial injury, immune suppression, reactive oxygen species activation, and microbial dysbiosis [2]. Initially, erythema and soreness appear, followed by ulcerations, pseudomembranes, and severe pain. These lesions limit food intake, speech, and hygiene practices and may predispose to systemic infection. Severe OM often forces clinicians to modify or interrupt chemotherapy schedules [3]. Recent research highlights the crucial role of oral microbiota in exacerbating mucosal damage. Pathogenic bacteria and fungal species proliferate when mucosal barriers are weakened, further intensifying inflammation [4].

The aim of this review is to present an expanded clinical and microbiological analysis of OM and provide evidence-based preventive strategies.

2. Clinical Characteristics of Oral Mucositis

OM progresses through well-defined stages—from erythema to ulceration and healing—usually 5–14 days after chemotherapy initiation [1].

Table 1. WHO Clinical Staging of Oral Mucositis

Grade	Description	Functional Limitation
Grade 0	No symptoms	None
Grade 1	Erythema, mild soreness	No eating difficulty
Grade 2	Ulcers, moderate pain	Solid foods tolerated
Grade 3	Painful ulcers, swelling	Only liquids tolerated
Grade 4	Extensive ulceration	No oral intake

Common clinical manifestations include:

- Erythema, edema
- Yellow-white pseudomembrane
- Spontaneous gingival bleeding
- Xerostomia
- Increased salivary viscosity
- Intense pain requiring opioid analgesics [2]

Clinical severity varies depending on chemotherapy regimen. 5-fluorouracil, methotrexate, doxorubicin, and platinum-based drugs have the highest mucotoxicity.

3. Microbiological Factors and Dysbiosis

The oral cavity hosts >700 microbial species. Healthy balance is maintained by saliva, epithelial turnover, and immune mechanisms. Chemotherapy disrupts this equilibrium,

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resulting in dysbiosis [5].

Table 2. Microbial Changes Associated With OM

Microorganisms	Normal Condition	OM Condition	Impact
Commensal bacteria (*Strep. mitis*)	High	Reduced	Loss of protective barrier
Pathogens (*Staph. aureus, Enterococcus*)	Low	Increased	Tissue infiltration & inflammation
Candida spp.	Minimal	Markedly increased	Pseudomembranous lesions
Viruses (HSV)	Latent	Possible activation	Ulcer worsening

Key findings in OM microbiology:

- *Candida albicans* colonization increases 5–7 folds [4].
- Pathogenic bacteria dominate ulcer surfaces.
- Dysbiosis amplifies cytokine activity (TNF-α, IL-6), worsening tissue injury [5].

4. Pathophysiology

OM develops through a 5-step biological cascade:

1. **Initiation:** Chemotherapy induces DNA damage, ROS formation.
2. **Up-regulation:** Inflammatory transcription factors activate NF-κB [6].
3. **Amplification:** Cytokines (IL-1β, TNF-α) increase tissue injury.
4. **Ulceration:** Epithelial breakdown, microbial invasion.
5. **Healing:** Re-epithelialization and microbial normalization occur.

Salivary dysfunction—reduced flow and antimicrobial proteins—contributes significantly, allowing pathogens to proliferate [7].

5. Evidence-Based Prevention Strategies

OM prevention is most effective when applied BEFORE and DURING chemotherapy. Recommended modalities include:

Table 3. Evidence-Based OM Prevention Protocol

Intervention	Mechanism	Evidence
Intensive oral hygiene	Reduces microbial load	Strong
Alcohol-free rinses	Anti-inflammatory effect	Moderate
Probiotics (*Lactobacillus*)	Restores microbiome	Strong
Antifungal prophylaxis	Prevents *Candida* overgrowth	Strong
Photobiomodulation (PBM)	Enhances epithelial repair	Strong
Cryotherapy (5-FU)	Vasoconstriction reduces drug exposure	Strong

5.1. Probiotics

Probiotics maintain microbial balance and inhibit pathogenic overgrowth. Clinical trials demonstrate reduced OM severity in patients receiving *Lactobacillus rhamnosus*–based therapy [6].

5.2. Photobiomodulation Therapy

PBM (low-level laser therapy) accelerates epithelial healing and reduces inflammation. It is recommended by MASCC/ISOO guidelines as a standard intervention [7].

5.3. Antimicrobial Regimen

Targeted antifungal and antibacterial therapy decreases pathogen load and supports mucosal regeneration [10].

6. Discussion

This review confirms that OM is not merely epithelial damage but a complex disorder involving microbiological, immunological, and salivary dysfunction components. Microbial dysbiosis significantly worsens clinical severity, especially when *Candida* proliferation and pathogenic bacterial colonization occur [8,9].

Evidence strongly supports the use of combined preventive strategies, including oral hygiene, probiotics, antimicrobials, and PBM therapy [5,6,7]. A multimodal prevention plan results in improved healing time, reduced symptom severity, and fewer chemotherapy interruptions.

7. Conclusions

1. OM severity is strongly associated with microbial imbalance.
2. Early microbial screening and oral hygiene optimization are essential.
3. Preventive interventions (PBM, probiotics, antifungals) significantly reduce OM severity.
4. Multimodal prevention strategies enhance patient comfort and help maintain chemotherapy schedules.

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