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Chemotherapy-Induced Oral Mucosal Changes: Pathogenesis, Clinical Manifestations, and Management Strategies

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Abstract

Chemotherapy remains a cornerstone in cancer management; however, its non-selective cytotoxic effects frequently result in significant oral mucosal complications that adversely affect patient wellbeing and treatment outcomes. The oral mucosa is particularly susceptible to injury because of its rapid epithelial turnover, rich vascularity, and constant exposure to mechanical and microbial insults. Chemotherapy-induced oral changes commonly include oral mucositis, xerostomia, opportunistic infections, dysgeusia, ulcerative lesions, mucosal pigmentation, and bleeding manifestations. Among these, oral mucositis is the most common and debilitating complication, often leading to pain, dysphagia, impaired nutrition, reduced quality of life, and interruption of cancer therapy. The pathogenesis involves direct epithelial toxicity, oxidative stress, inflammatory cytokine activation, and breakdown of the mucosal barrier, predisposing patients to secondary infections and delayed healing. Early diagnosis, preventive oral care, and multidisciplinary management are essential for minimizing morbidity and improving therapeutic outcomes. This review summarizes the pathogenesis, clinical manifestations, diagnosis, prevention, and management of chemotherapy-induced oral mucosal changes.

Keywords: Chemotherapy, Oral mucositis, Oral complications, Xerostomia, Supportive care.

INTRODUCTION

Cancer is one of the major global health challenges, accounting for nearly 20 million new cases and approximately 10 million deaths worldwide in 2022. The growing incidence of malignancies, along with improved survival rates, has led to an increased use of chemotherapy as a primary or adjunctive treatment modality in cancer management. Despite its therapeutic benefits, chemotherapy is associated with several adverse effects, particularly within the oral cavity, due to its non-selective action on rapidly dividing normal cells.[1]

The oral mucosa is highly susceptible to chemotherapeutic injury because of its high epithelial turnover rate, rich vascularity, and constant exposure to mechanical and microbial insults. Studies have reported that nearly 40–80% of patients receiving chemotherapy develop oral complications, with the incidence rising to almost 90% in patients undergoing intensive chemotherapy for hematological malignancies. Chemotherapy-induced oral mucosal changes commonly include oral mucositis, ulceration, erythema, xerostomia, opportunistic infections, taste alterations, spontaneous bleeding, and mucosal atrophy. Among these, oral mucositis is considered one of the most painful and debilitating complications, affecting approximately 20–40% of patients receiving conventional chemotherapy and up to 80% of high-risk patients. [2,3]

These oral complications significantly affect patients' daily functioning and overall well-being. Painful mucosal lesions may impair mastication, swallowing, and speech, leading to nutritional deficiencies, weight loss, dehydration, and poor oral intake. In immunocompromised patients, disruption of the mucosal barrier also increases the risk of secondary bacterial, viral, and fungal infections. Importantly, severe oral toxicities may necessitate chemotherapy dose reduction, treatment delay, or temporary discontinuation, thereby compromising the effectiveness of cancer therapy and negatively influencing prognosis. [4]

Therefore, early recognition and multidisciplinary management of oral mucosal complications are essential to improve patient comfort, maintain nutritional status, and ensure uninterrupted cancer treatment. This review aims to summarize the pathogenesis, clinical manifestations, diagnosis, prevention, and management of oral mucosal changes occurring during chemotherapy.

Normal Structure and Turnover of Oral Mucosa

The oral mucosa forms a specialized protective lining of the oral cavity and serves as an important barrier against mechanical trauma, microorganisms, toxins, and chemical irritants. Histologically, it consists of a stratified squamous epithelium supported by an underlying connective tissue layer known as the lamina propria. Depending on the functional demand, the oral mucosa may be keratinized, as seen in the gingiva and hard palate, or non-keratinized, as observed in the buccal mucosa, floor of the mouth, soft palate, and ventral surface of the tongue. The epithelium is organized into distinct layers including the basal layer, spinous layer, granular layer, and superficial layer. Among these, the basal cell layer contains actively proliferating cells responsible for continuous epithelial renewal. [5]

One of the most important biological characteristics of the oral mucosa is its rapid cellular turnover. Under normal physiological conditions, oral epithelial cells are renewed approximately every 7–14 days. Basal cells undergo continuous mitosis, gradually migrate toward the surface, and are eventually shed. This high proliferative activity helps maintain mucosal integrity and facilitates rapid healing following minor trauma. However, it also makes the oral mucosa particularly vulnerable to chemotherapeutic agents, which primarily target rapidly dividing cells by interfering with DNA synthesis and cellular replication. [6]

Saliva plays a vital protective role in maintaining oral mucosal health. It provides lubrication, buffering action, tissue hydration, and antimicrobial defense through components such as lysozyme, lactoferrin, immunoglobulin A, and peroxidases. Saliva also contributes to tissue repair and helps regulate the oral microbial environment. Reduction in salivary flow during chemotherapy weakens these protective mechanisms and predisposes patients to mucosal injury and infection. [7]

The oral cavity normally harbors a complex and balanced microbiome consisting of bacteria, fungi, and viruses that coexist in harmony with the host. Chemotherapy-induced immunosuppression and mucosal barrier disruption can alter this microbial balance, allowing opportunistic organisms such as *Candida albicans* and herpes simplex virus to proliferate. Consequently, the combination of rapid epithelial turnover, salivary dysfunction, and microbial imbalance contributes significantly to the development of oral mucosal complications during chemotherapy. [8]

Pathophysiology of Chemotherapy-Induced Oral Mucosal Injury

Chemotherapy-induced oral mucosal injury is a complex biological process resulting from direct cytotoxic effects of antineoplastic agents on the oral epithelium and the subsequent activation of inflammatory pathways. The oral mucosa is particularly susceptible because of its high cellular turnover rate and the continuous proliferation of basal epithelial cells. Chemotherapeutic agents primarily target rapidly dividing cells by interfering with DNA replication and cellular mitosis, thereby affecting both malignant and normal tissues. As a result, the basal epithelial layer of the oral mucosa undergoes significant cellular damage, leading to impairment of epithelial renewal and mucosal integrity. [9]

The initial stage of mucosal injury begins with direct DNA damage caused by cytotoxic drugs such as methotrexate, 5-fluorouracil, and doxorubicin. These agents induce apoptosis and inhibit normal epithelial regeneration. Simultaneously, chemotherapy promotes the formation of reactive oxygen species (ROS), which generate oxidative stress within the mucosal tissues. Excessive ROS production causes lipid peroxidation, mitochondrial dysfunction, and further cellular injury, amplifying tissue destruction beyond the initial chemotherapeutic insult. [10]

Oxidative stress also activates several intracellular signaling pathways that stimulate the release of proinflammatory cytokines, particularly tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6). These inflammatory mediators play a central role in the progression of mucosal injury by enhancing epithelial apoptosis, increasing vascular permeability, and recruiting inflammatory cells into the damaged tissues. TNF-α further amplifies tissue destruction by activating nuclear factorkappa B (NF-κB), thereby creating a self-propagating inflammatory cycle that intensifies mucosal breakdown. [11]

As epithelial destruction progresses, the protective mucosal barrier becomes disrupted, resulting in erythema, ulceration, and exposure of underlying connective tissue. The loss of mucosal integrity facilitates colonization by oral microorganisms and predisposes immunocompromised patients to secondary bacterial, fungal, and viral infections. Opportunistic pathogens such as *Candida albicans* and herpes simplex virus commonly complicate chemotherapy-induced oral lesions, leading to increased pain, delayed healing, and risk of systemic infection. [12]

The pathogenesis of oral mucositis has been extensively explained by the five-stage model proposed

by Sonis, which remains one of the most accepted frameworks for understanding chemotherapy-induced mucosal injury. The first stage, initiation, involves direct tissue injury and generation of reactive oxygen species following chemotherapy exposure. The second stage, primary damage response or signaling, is characterized by activation of transcription factors and inflammatory mediators. In the third stage, signal amplification, cytokines such as TNF-α intensify tissue injury through positive feedback mechanisms. The fourth stage, ulceration, represents the clinically evident phase in which painful ulcerative lesions develop and become colonized by microorganisms. Finally, during the healing stage, epithelial proliferation and tissue differentiation restore mucosal integrity once chemotherapy-induced suppression decreases. [13]

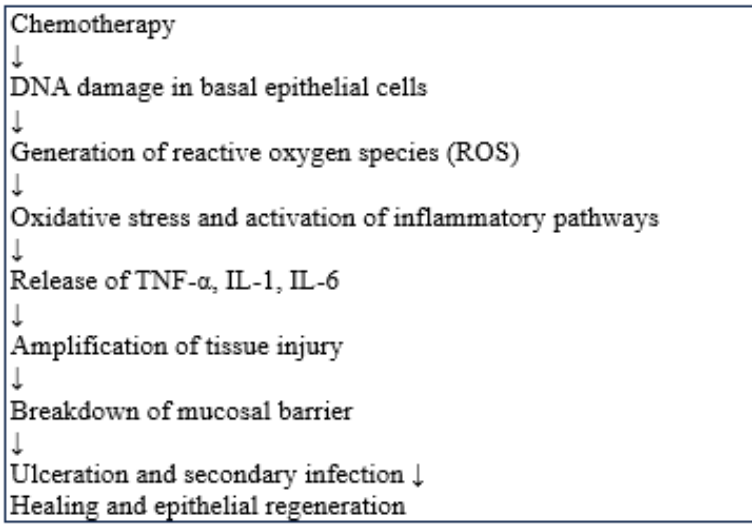

Figure 1: Pathophysiology of Chemotherapy-Induced Oral Mucositis

Risk Factors

The severity and incidence of chemotherapy-induced oral mucosal changes are influenced by multiple interacting factors related to the patient, cancer therapy, and underlying malignancy. Identification of these risk factors is important for early prevention, risk stratification, and supportive oral care planning.

Table 1: Risk Factors Associated with Chemotherapy-Induced Oral Mucosal Changes [14–16]

Category	Risk Factor	Mechanism/Clinical Significance
Patient-related factors	Advanced age or younger age	Altered mucosal regenerative capacity and immune response
	Poor oral hygiene	Increased microbial colonization and secondary infection
	Nutritional deficiencies	Impaired epithelial repair and healing
	Smoking and alcohol consumption	Mucosal irritation and delayed healing
	Immunosuppression/neutropenia	Increased susceptibility to opportunistic infections
	Pre-existing dental disease	Local inflammatory burden and infection risk
	Salivary dysfunction/xerostomia	Reduced mucosal lubrication and antimicrobial protection
Treatmentrelated factors	Type of chemotherapeutic agent	Certain drugs such as methotrexate and 5-FU are highly mucotoxic
	High-dose chemotherapy	Greater epithelial toxicity and tissue injury
	Combination chemotherapy	Synergistic increase in mucosal damage
	Prolonged treatment duration	Cumulative mucosal toxicity
	Concurrent radiotherapy	Enhanced epithelial destruction and delayed healing
Tumor-related factors	Hematological malignancies	Severe immunosuppression and intensive chemotherapy protocols
	Head and neck cancers	Direct involvement of oral tissues and combined chemoradiotherapy

"Among these factors, treatment-related variables such as high-dose chemotherapy and concurrent chemoradiotherapy are considered the strongest predictors of severe oral mucosal injury."

Types of Oral Mucosal Changes During Chemotherapy

Chemotherapy-induced oral mucosal changes represent a broad spectrum of inflammatory, infectious, degenerative, and functional alterations affecting the oral cavity. These manifestations vary according to the type of chemotherapeutic agent, dosage, duration of treatment, and the patient's immune status.

Oral complications may range from mild discomfort to severe debilitating lesions that interfere with nutrition, speech, swallowing, and continuation of cancer therapy.

A. Oral Mucositis

Oral mucositis is the most common and clinically significant oral complication associated with chemotherapy. It is characterized by inflammation and ulceration of the oral mucosa resulting from cytotoxic injury to rapidly dividing epithelial cells. The incidence of oral mucositis ranges from 20– 40% in patients receiving conventional chemotherapy and may exceed 80% in patients undergoing high dose chemotherapy or hematopoietic stem cell transplantation. [17]

Clinically, oral mucositis initially presents as diffuse erythema and mucosal soreness, which progressively develops into painful ulcerative lesions covered by pseudomembranous slough. Nonkeratinized mucosal surfaces such as the buccal mucosa, ventral tongue, soft palate, and floor of the mouth are most commonly affected. Patients frequently complain of severe pain, burning sensation, dysphagia, odynophagia, and difficulty in mastication and speech. Severe mucositis may result in dehydration, nutritional compromise, and interruption of chemotherapy. [18]

The World Health Organization (WHO) grading system is commonly used to assess severity [19]:

Grade	Clinical Features
Grade 0	No mucositis
Grade 1	Erythema and soreness
Grade 2	Ulcers present; able to eat solids
Grade 3	Extensive ulcers; liquid diet only
Grade 4	Severe ulceration; alimentation impossible

Several chemotherapeutic agents are strongly associated with oral mucositis, particularly methotrexate, 5-fluorouracil (5-FU), doxorubicin, and cisplatin due to their potent effects on DNA synthesis and epithelial turnover.

B. Xerostomia and Salivary Changes

Chemotherapy may impair salivary gland function, leading to xerostomia or subjective oral dryness. Damage to salivary acinar cells and altered autonomic regulation contribute to reduced salivary secretion and changes in salivary composition. Patients commonly experience dry mouth, thick or viscous saliva, difficulty in swallowing dry food, altered speech, and oral discomfort. [20]

Reduced salivary flow compromises the protective functions of saliva, including lubrication, buffering capacity, antimicrobial activity, and tissue repair. Consequently, xerostomia predisposes patients to mucosal trauma, opportunistic infections, halitosis, and increased dental caries susceptibility. Persistent salivary dysfunction may also negatively affect taste perception and oral quality of life. [21]

C. Opportunistic Infections

Chemotherapy-induced immunosuppression and neutropenia significantly increase the risk of opportunistic oral infections. Disruption of the mucosal barrier further facilitates microbial invasion and colonization. Fungal infections, particularly oral candidiasis caused by *Candida albicans*, are among the most frequently encountered complications. Clinically, candidiasis may present as pseudomembranous plaques, erythematous patches, angular cheilitis, or burning sensation. [22]

Viral infections such as herpes simplex virus (HSV) and cytomegalovirus (CMV) may reactivate during periods of immunosuppression, resulting in painful ulcerative lesions and delayed mucosal healing. Secondary bacterial infections may also occur, contributing to increased inflammation, ulceration, and risk of systemic dissemination in severely neutropenic patients. [23]

D. Taste Alterations (Dysgeusia)

Taste disturbances are common during chemotherapy and may manifest as metallic taste, bitter sensation, reduced taste acuity, or complete loss of taste perception. Dysgeusia occurs due to direct toxic effects of chemotherapeutic agents on taste buds, salivary alterations, mucosal injury, and neural dysfunction. [24]

Drugs such as cisplatin and 5-fluorouracil are frequently associated with taste abnormalities. These alterations can significantly affect appetite and food intake, leading to nutritional deficiencies, weight loss, and reduced quality of life. In many patients, persistent unpleasant taste sensations contribute to aversion toward food and impaired nutritional rehabilitation. [25]

E. Mucosal Pigmentation

Chemotherapy-associated mucosal pigmentation is an uncommon but recognized complication characterized by diffuse or localized melanotic discoloration of the oral mucosa. Drug-induced melanosis may result from increased melanin production, direct drug deposition, or stimulation of melanocytes. [26]

Commonly implicated agents include cyclophosphamide, doxorubicin, and busulfan. Clinically, pigmentation may involve the tongue, buccal mucosa, gingiva, or palate and usually appears as brown, black, or bluish discoloration. Differential diagnosis should include physiologic pigmentation,

Addison's disease, smoker's melanosis, amalgam tattoo, and malignant melanoma. [27]

F. Ulcerative and Erosive Lesions

Apart from classical mucositis, chemotherapy may produce aphthous-like ulcers, diffuse erosions, and non-healing ulcerative lesions. These lesions often arise secondary to epithelial thinning, immune suppression, and impaired tissue regeneration. [10]

Patients may also exhibit increased susceptibility to traumatic ulcers due to mucosal fragility and reduced healing capacity. Persistent ulcerative lesions can become secondarily infected and may significantly interfere with oral intake and speech. Careful clinical evaluation is necessary to differentiate chemotherapy-induced ulcers from infectious or malignant lesions. [19]

G. Bleeding and Petechiae

Chemotherapy frequently causes thrombocytopenia due to bone marrow suppression, resulting in increased bleeding tendency within the oral cavity. Patients may present with petechiae, ecchymosis, spontaneous gingival bleeding, or hemorrhagic bullae. [28]

Minor trauma during tooth brushing or mastication may precipitate excessive bleeding in thrombocytopenic individuals. Severe hemorrhagic lesions may interfere with oral hygiene maintenance and increase the risk of secondary infection. Gingival tissues are particularly vulnerable because of their rich vascularity and susceptibility to local irritation. [29]

H. Atrophic and Desquamative Changes

Chemotherapy-induced epithelial damage may lead to mucosal atrophy and desquamation. Clinically, the mucosa appears thin, smooth, erythematous, and fragile. Patients commonly complain of burning sensation, increased sensitivity to spicy food, and discomfort during eating or speaking.[2] Desquamative changes are often associated with loss of epithelial integrity and reduced regenerative capacity. The atrophic mucosa becomes highly susceptible to trauma, ulceration, and secondary microbial infection, thereby contributing to persistent oral discomfort throughout the course of chemotherapy. [30]

7. Chemotherapeutic Agents Commonly Associated with Oral Lesions

The severity and type of oral mucosal lesions during chemotherapy largely depend on the stomatotoxic potential of the chemotherapeutic agent, treatment dosage, duration, and concomitant therapies. Certain drugs exhibit a higher affinity for rapidly dividing oral epithelial cells and are therefore more frequently associated with mucosal injury, ulceration, salivary dysfunction, and opportunistic infections.

Antimetabolites and alkylating agents are particularly recognized for their significant oral toxicity.[31]

Table 2: Common Chemotherapeutic Agents and Associated Oral Manifestations [32–34]

Chemotherapeutic Agent	Common Oral Manifestations	Mechanism/Clinical Significance
Methotrexate	Severe oral mucositis, ulceration, erythema	Inhibits DNA synthesis and rapidly affects basal epithelial cells
5-Fluorouracil (5-FU)	Painful mucositis, diffuse ulceration, burning sensation	High stomatotoxic potential due to interference with epithelial cell replication
Doxorubicin (Adriamycin)	Erythema, mucosal inflammation, ulcerative lesions	Direct cytotoxic injury and oxidative stress-mediated tissue damage
Cisplatin	Xerostomia, dysgeusia, mucosal ulceration	Salivary gland dysfunction and epithelial toxicity
Cyclophosphamide	Mucosal ulceration, hemorrhagic lesions	Myelosuppression and impaired mucosal healing
Cytarabine	Severe mucositis, diffuse erythema	High affinity for rapidly proliferating mucosal cells
Melphalan	Extensive ulcerative mucositis	Commonly associated with high-dose chemotherapy regimens
Busulfan	Mucosal pigmentation, ulceration	Cytotoxic injury with melanocytic stimulation
Vincristine	Neurogenic oral pain, taste disturbances	Neurotoxicity affecting oral sensory pathways

Chemotherapeutic Agent	Common Oral Manifestations	Mechanism/Clinical Significance
Bleomycin	Mucosal erythema, hyperpigmentation	Oxidative tissue injury and epithelial toxicity
Docetaxel/Paclitaxel	Mucositis, xerostomia, dysgeusia	Microtubule inhibition affecting mucosal regeneration
Etoposide	Oral ulceration and secondary infections	Bone marrow suppression with impaired mucosal repair

Certain combination chemotherapy regimens have been shown to produce synergistic stomatotoxic effects, especially when administered concurrently with radiotherapy. High-dose chemotherapy protocols used in hematological malignancies and hematopoietic stem cell transplantation are associated with the highest incidence of severe oral mucosal injury. Early recognition of drug-specific oral manifestations is essential for prompt preventive and therapeutic interventions.

8. Clinical Diagnosis and Assessment

Diagnosis of chemotherapy-induced oral mucosal changes is mainly based on detailed history and careful oral examination. Common symptoms include pain, burning sensation, xerostomia, dysphagia, altered taste, and bleeding, while clinical findings may include erythema, ulceration, pseudomembrane formation, candidal plaques, petechiae, and mucosal atrophy. Non-keratinized areas such as the buccal mucosa, ventral tongue, floor of the mouth, and soft palate are most commonly affected. The WHO mucositis grading system and Oral Assessment Guide (OAG) are widely used to assess severity and monitor progression. Differential diagnosis is important to exclude conditions such as candidiasis, herpes simplex infection, aphthous ulcers, autoimmune disorders, nutritional deficiencies, and oral malignancies. Laboratory investigations including complete blood count, microbial culture, fungal smear, viral testing, or biopsy may be required in selected cases. [35]

9. Impact on Quality of Life and Cancer Therapy

Chemotherapy-induced oral mucosal complications significantly affect the quality of life of cancer patients by interfering with eating, swallowing, speech, and routine oral functions. Painful lesions may lead to poor oral intake, dehydration, nutritional deficiencies, and weight loss. Breakdown of the mucosal barrier also increases the risk of secondary bacterial, fungal, and viral infections, particularly in immunocompromised individuals. Severe mucositis may require hospitalization, supportive care, and opioid analgesics, thereby increasing treatment burden and healthcare costs. Importantly, severe oral complications can result in chemotherapy dose reduction or treatment interruption, negatively affecting cancer therapy outcomes and patient prognosis. [36]

10. Prevention Strategies

Prevention of chemotherapy-induced oral mucosal injury mainly focuses on maintaining good oral hygiene, reducing microbial load, and minimizing mucosal trauma. Pre-chemotherapy dental evaluation helps eliminate potential sources of infection and local irritants. Supportive measures such as soft toothbrushes, non-irritating mouth rinses, adequate hydration, nutritional support, and avoidance of tobacco or spicy foods are beneficial in reducing mucosal irritation. Oral cryotherapy, benzydamine mouthwash, and low-level laser therapy have shown effectiveness in reducing the severity and duration of oral mucositis, especially in high-risk patients. [19]

11. Management of Oral Mucosal Changes

Management of chemotherapy-induced oral lesions aims to relieve symptoms, control infection, promote healing, and maintain oral function. Pain is managed using topical anesthetics, analgesics, and anti-inflammatory agents, while opportunistic infections may require antifungal, antiviral, or antibiotic therapy. Xerostomia can be managed with hydration, saliva substitutes, and salivary stimulants.

Supportive measures such as bland mouth rinses, soft diet, nutritional supplementation, and maintenance of oral hygiene are essential for improving patient comfort. In severe cases, therapies such as photo biomodulation and growth factors like palifermin may help accelerate mucosal healing. [19]

12. Role of Dental Professionals

Dental professionals play an important role in the prevention and management of chemotherapy-related oral complications. Comprehensive oral examination before initiation of cancer therapy helps identify and treat potential infection sources and local irritants. Regular follow-up during chemotherapy allows early detection and timely management of mucosal lesions. Patient education regarding oral hygiene, hydration, dietary care, and early symptom recognition is equally important. A multidisciplinary approach involving oncologists, dentists, nurses, and nutritionists helps improve treatment compliance and overall patient outcomes.

13. Recent Advances and Emerging Therapies

Recent advances in supportive oncology have improved the management of chemotherapy-induced oral mucosal injury. Growth factors such as palifermin have shown promising results in promoting epithelial regeneration and reducing mucositis severity. Cytokine modulators, probiotics, and natural agents including

honey, aloe vera, curcumin, and glutamine are being explored for their anti-inflammatory and wound-healing properties. Photo biomodulation therapy has also gained importance as a non-invasive method for prevention and treatment of oral mucositis. Emerging approaches such as stem cell therapy and nanotechnology-based drug delivery systems may further improve targeted tissue repair and supportive care in the future.

14. Future Perspectives

Future research on chemotherapy-induced oral mucosal injury is expected to focus on personalized supportive care and early prediction of high-risk patients. Identification of genetic and molecular biomarkers may help predict susceptibility to severe mucositis and guide individualized preventive strategies. Development of standardized evidence-based protocols and integration of newer technologies such as artificial intelligence may improve early diagnosis, treatment monitoring, and patient outcomes. Continued interdisciplinary research remains essential for reducing the burden of oral complications during cancer therapy.

15. CONCLUSION

Chemotherapy-induced oral mucosal changes are common complications that can significantly affect oral function, nutrition, quality of life, and continuation of cancer therapy. Manifestations may range from mild erythema and xerostomia to severe ulcerative mucositis and opportunistic infections. Early diagnosis, preventive oral care, maintenance of oral hygiene, and timely management are essential for reducing treatment-related morbidity and improving patient comfort. Advances in supportive therapies and a multidisciplinary approach involving oncologists and dental professionals have further improved the prevention and management of these complications.

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