ISSN 2692-5877
DOI: *10.46998/IJCMCR.2025.55.001363*

International Journal of
Clinical Studies & Medical Case Reports

Research Article

Cancer - Chemotherapy and Radiation Therapy: Current Advances and Future Directions: Narrative Review

Davit Mathevosyan[1], Hovhannes Karapetyan[2] and Gagik Hakobyan[3,*]

[1]Associatt Professor, Department of Oral and Maxillofacial Surgery, Yerevan State Medical University after M. Heratsi, Armenia

[2]Visiting lecturer Yerevan State Medical University after M. Heratsi,Yerevan, Armenia

3Head of Department of Oral and Maxillofacial Surgery, Yerevan State Medical University, Yerevan, Armenia

***Corresponding author:** Professor Gagik Hakobyan DMSc, PhD, Head of Department of Oral and Maxillofacial Surgery, Yerevan State Medical University, Yerevan, Armenia

Received: August 13, 2025 Published: September 22, 2025

Abstract

Background and objective: WHO emphasizes the role of dentists in the surveillance of OC, and recommends the inclusion of dentists, together with the oral health team, in the primary, secondary and tertiary prevention of OC, and the prevention of complications of radiotherapy and chemotherapy by providing preventive and ongoing oral care. At an early stage, oral cancer is often asymptomatic and mimics benign diseases, which reduces patient access to cancer care.
This study aimed to systematically review the literature to analyze proposed effective protocols for the treatment of oral complications in cancer patients.

Selection criteria: Trials were selected if they met the following criteria: design - random allocation of participants; participants - anyone with cancer receiving chemotherapy or radiotherapy treatment for cancer; interventions - agents prescribed to prevent oral mucositis; outcomes - prevention of mucositis, pain, amount of analgesia, dysphagia, systemic infection, length of hospitalisation, cost and patient quality of life.
The systematic review included articles from Google Scholar, Medline, Scopus, Web of Sciences, PubMed was conducted.

Results: 281 articles were found, and 42 full-text articles were selected of high methodological quality to synthesize methods for treating complications in the oral cavity. The review describes advances in understanding the pathobiology of mucositis, discusses as well as modern methods of treatment and prevention of pathology. There are different protocols for oral care for cancer patients, in which the main focus is on prophylaxis and pain relief.

To improve the quality of life for those with head and neck cancer, numerous agent's studies are aimed at exploring new agents, as well as reducing the radiation field and dose to maintain effective tumor control. Prevention is important, and particular attention is paid to teaching patients about oral hygiene.

Conclusion: The clinical and economic significance of oral complications in cancer patients determines the need for further trials already proposed means and methods in oncology therapy and the development of new approaches to prevention and treatment of this pathology.
The results of the review may help inform the effectiveness of safe preventive measures for oral mucositis in cancer patients

Keywords: Head and neck cancer; Oral management protocols; Oral care and prevention of complications of cancer; Oral tissue lesions manifestation in; Chemotherapy; Head and neck radiation therapy

Introduction

Currently in complex cancer treatment chemotherapy and radiation therapy are the most widely used treatment methods [1-4].

However, complications in the oral cavity remain one of the side effects of chemotherapy and radiotherapy for cancer patients [5,6].

Complications manifest themselves 7-14 days after the start of therapy and have a significant impact on the quality of life of patients may also lead to changes or interruptions in treatment, all of which affect cure rates [7].

Pathological changes appear on the mucous membrane oral cavity, on the skin, in the subcutaneous tissue, in the salivary glands and in the bone marrow of the jaw bones.

Complications in the oral cavity during cancer therapy include, dental caries, periodontitis, mucositis, fibrosis, oropharyngeal candidiasis, oral infection (viral, fungal, bacterial), salivary gland hypofunction and xerostomia, neurosensory disorders (mucosal pain and taste dysfunction) [9-16].

In patients receiving chemotherapy, approximately 40-70% are accompanied by oral mucositis (OM), a severe inflammation of the oral mucosa. Oral mucositis dramatically reduces the quality of life of patients and requires significant additional costs [17].

Clinical, morphological and biochemical changes in the oral mucosa during chemotherapy

On the 5th–10th day from the start of mucotoxic Chemotherapy (CT), the Oral Mucosa (OM) becomes clearly hyperemic and edematous. Soon, areas with pseudomembranous plaque appear on the non-keratinizing oral mucosa; in severe cases, zones of necrosis (erosion, ulcers) with an area of from 0.5 to 4 cm2. Under favorable conditions, it resolves independently (hyperemia and edema are reduced, the integrity of the epithelial tissue is restored cover, and later – the relief of the ulceration zone) in periods from several days to a month [18,19].

From a morphological point of view, pseudomembranous plaque is a fibrinous film with a relatively low neutrophil content.

Changes in the epithelium in tissues at the peak of severe are described as apoptosis, atrophy, dysplasia, parakeratosis, hyperplasia; at the level of the basement membrane, massive death of epithelial stem cells is detected [20,21].

Tissue separation and apoptosis are found in the submucosal layer cells, degeneration of collagen and glands, significant changes in blood vessels (intimal thickening, decreased lumen, destruction of elastic and muscle fibers of the vascular wall, apoptosis of endothelial cells) and damage to nerve endings. Less commonly, OM zones are described like ulcers with granulation tissue and cells of chronic inflammation.

In the OM zone, the activity of inflammatory mediators increases: transcription factor NFkB, tumor necrosis factor TNF-α, pro-inflammatory interleukins (IL-1β, IL-6, IL-8) [10], the concentration of ribosomal protein also increases in inflammatory reactions mediated by nitric oxide and p53, as well as platelet-derived growth factor IL-1122,23.

The most striking symptom of severe OM is pain in the soft tissues of the oral cavity.

With mild OM, patients note increased sensitivity to temperature, taste, consistency of drink and food, and complain of spontaneous burning [24].

Severe OM is accompanied by constant pain, which intensifies when trying to open the mouth slightly, change the position of the tongue, or make a swallowing movement. Pain, dysgeusia, dysphagia, hyposalivation in combination with nausea are the reasons why patients with OM refuse from normal eating and drinking. Lack of adequate assistance (tube or parenteral nutrition) leads to dehydration and significant loss of body weight, the prognosis sharply worsens not only for the restoration of the mucous membranes, but also for the treatment of the underlying disease [25].

There is evidence in the literature that increased production of proinflammatory cytokines in OM may be the cause of non-infectious fever accompanying “febrile” mucositis.

In accordance with the traditional thesis “OM is the entrance gate for infection” (in particular, for bacteria, herpes viruses, fungi, OM is considered as the main risk factor for infectious complications and death of patients during the period of immunosuppression.

In 25–30% of cases, severe OM forces one to delay the next course of chemotherapy and/or reduce the dose of cytostatics, which significantly reduces the patient’s chances of survival [27].

In recent years, in vivo and in vitro studies have improved knowledge about the pathogenesis of OM [27].

OM is the result of a series of complex biological changes occurring in various cells of the oral mucosa. A five-phase model of OMpathogenesis has been proposed, 5, 8 including [28]:

Pathogenesis model oral mucositis

Phase I: Initiation phase (1–2 days).

Radiation and/or chemotherapy, damaging DNA, cause immediate death of some of the clonogenic cells in basal layer of epithelium and in the submucosal layer, ulcer formation; but this alone is not sufficient for the large-scale changes observed in OM. It is assumed that the main factor damaging the DNA of replicating cells, cell membranes and connective tissue of the submucosal layer are free radicals formed in tissues under the influence of cytostatic therapy.

Phase II: Signal generation phase (2–3 days).

Free radicals not only destroy DNA, but also serve as triggers for cascading biological processes - normal responses to damage.

In the pathogenesis of OM, the main role is played by the activation of the nuclear transcription factor NF-κB in endothelial cells, epithelial cells, fibroblasts and macrophages, which regulates the expression of BCL2 family genes related to apoptosis, as well as genes involved in the production pro-inflammatory cytokines - Tumor Necrosis Factor (TNF), interleukins IL-1β and IL-6, which have a direct cytotoxic effect and stimulate apoptosis. In this phase, the synthesis of ceramides, signaling molecules, is activated apoptosis. Damage to fibroblasts stimulates the production of matrix metalloproteinases, which are

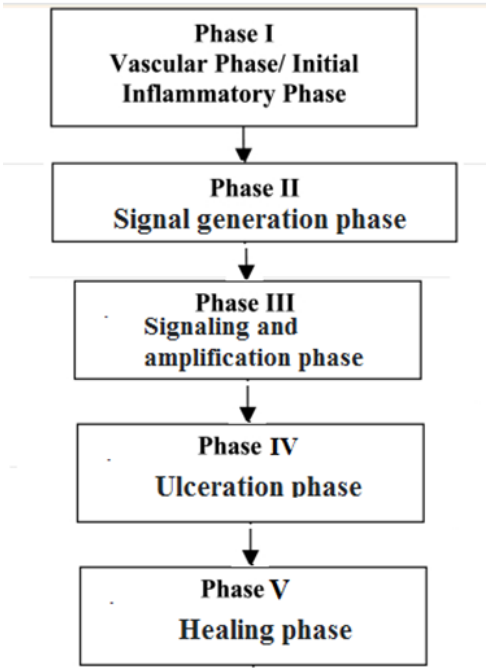

Figure 1: Chart representing the different phases of Pathogenesis model mucositis development with chemotherapy and/or radiation therapy.

involved in apoptosis and also destroy the junction of the epithelium and the submucosal layer at the level of the basement membrane. This phase results in a positive feedback loop that leads to phase III;

Phase III: Signaling and amplification phase.
The processes launched in the second phase lead to damage to the intercellular matrix and cells, which triggers the next round of signal activation, synthesis of pro-inflammatory cytokines and enzymes - cascade damage
tissue, supported by the feedback principle, expands in a spiral and therefore does not immediately stop after the end of the action of cytostatics. The phase lasts from 2 to 10 days. The main destructive events occur in the submucosal layer and remain clinically silent for some time; towards the end of the phase they may manifest as erythema (due to atrophy and thinning of the epithelium, vasodilation under the influence of IL-1β) and a burning sensation.

Phase IV: Ulceration phase.
Previous events (death of mature and stem cells, deficiency of growth factors, degradation of the intercellular matrix) lead to thinning of the oral mucosa and destruction of the connections between the epithelium and the submucosal layer.
The integrity of the epithelium is easily disrupted by mechanical microtrauma caused by speech, swallowing, chewing, pressure from dystopic teeth, etc.
Fragments of the walls of dead bacteria, entering damaged tissues, provoke an increase in the production of pro-inflammatory cytokines. Inflammation reaches its peak, which coincides with the maximum clinical manifestations of OM. The phase can last up to 10–15 days.

Phase V: Healing phase.
This stage of OM remains the least studied. It is generally accepted that a decrease in intensity damaging reactions and tissue repair is controlled by regulatory proteins (including various growth factors produced by cells and the extracellular matrix) **(Figure 1)**.
Signals reach epithelial stem cells, stimulate their migration under the pseudomembrane, proliferation and differentiation, which ensures the closure of surface defects and restoration of epithelial thickness within 1–4 weeks. Regeneration of the submucosal layer is slower, so it takes months to fully restore the architecture and function of the oral mucosa after severe OM.
The development of OM is played by a range of pro-inflammatory cytokines, including tumor necrosis factor (TNF), interleukin-6 (IL-6), IL-1β, nuclear factor kappa B (NFκB) and ceramide synthase [29,30].

The described pathogenesis model successfully used as a basis for further development of the problem of OM.
Assessment and recording of OM severity to study pathogenesis and evaluate the effectiveness of methods OM management uses systems (indices, scales) for semi-quantitative assessment of its clinical signs and symptoms.

Three systems are often used in practice:
WHO Oral Toxicity score (OT WHO), the National Cancer Institute Common Toxicity Criteria (NCI CTC) scale and Oral Mucositis Assessment Scales (OMAS).
The WHO OT Index combines anatomical, symptomatic and functional criteria in one scale:
0 – normal condition;
1 – soreness, erythema;
2 – there are ulcers, the patient takes solid food;
3 – there are ulcers, the patient takes only liquid food;
4 – eating by mouth is impossible.

The NCI CTC Index consists of two scores: clinical and functional.
According to clinical scale
1 point; erythema, isolated areas of ulceration and/or pseudomembranes
2 points; fused lesions and bleeding during minimal injury
3 points; tissue necrosis, spontaneous bleeding, condition
4 points; life-threatening,
5 points; death

On the functional scale, minimal symptoms that do not change breathing and nutrition,
1 point; the presence of complaints, changes in the nature of food with the ability to eat and swallow, with altered but independent breathing
2 points; pain, inability to receive adequate oral intake of food and liquids,
3 points; significant respiratory difficulties
4 points; life-threatening symptoms
5 points; death

The OMAS scale involves assessing the clinical condition of each of 8 areas of the mucous membrane of the upper and lower lips, left and right cheeks, the ventral surface of the tongue on the right and left, the floor of the mouth along with the frenulum of the tongue, the soft palate and pharynx) according to the severity of erythema (0 – normal, 1 – mild/moderate erythema, 2 – severe erythema) and ulceration of pseudomembranous plaque (0 – no signs; 1 – total area of the affected area S≤1 cm2; 2 – 1 cm2 <S≤3 cm2; 3 – S>3 cm2).

Each zone can receive a total score from 0 to 5 points; the final OMAS value is calculated as the average for all eight zones. In head and neck cancer, oral cancer treatment may require un-

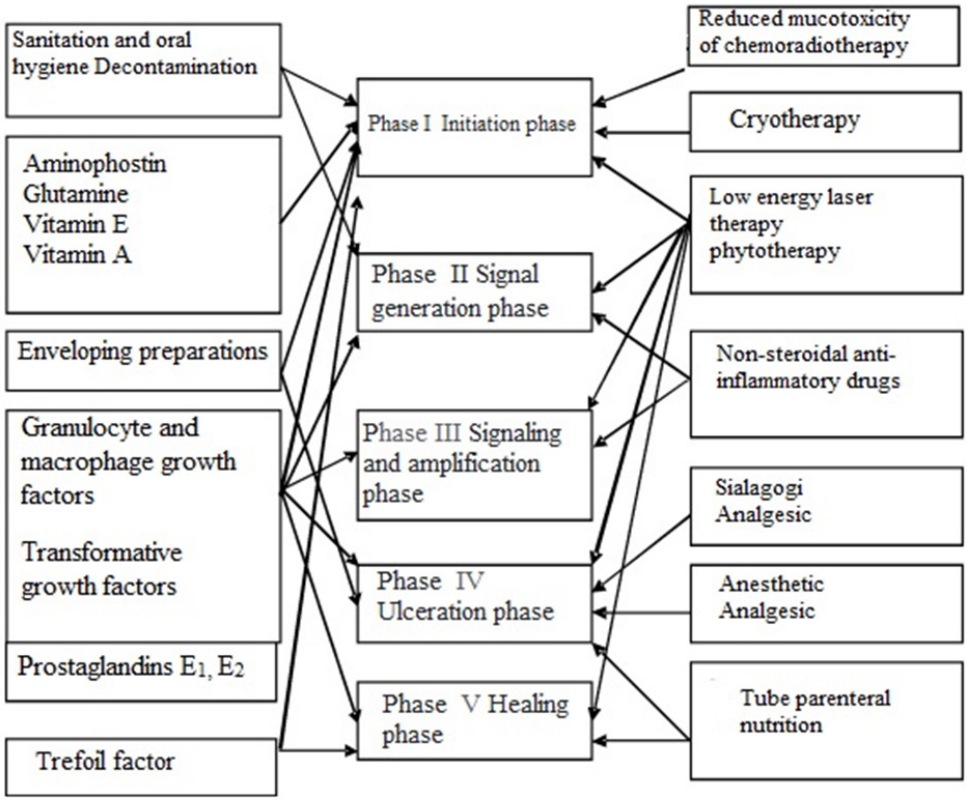

Figure 2: Algorithm for the prevention and treatment of chemotherapeutic oral mucositis.

planned interruptions in treatment, which will reduce the intensity of radiation therapy and, as a result, the effectiveness of chemoradiation therapy may decrease, this may lead to regrowth of the tumor or lead to a deterioration in quality of life and reduced survival [31]. OM can significantly worsen the clinical condition of cancer patients and increase the risk of infection. Resulting from radiation and chemoradiation therapy can lead to long-term changes in swallowing function, due to pain and swelling, leading to wasting and weight loss, aspiration of nutrients; insufficient hydration.

In patients receiving only chemotherapy, mucositis occurs in 60% of patients; in patients receiving chemotherapy and radiation therapy in combination, mucositis occurs in 100% of patients [5,33-36].

Treatment Options and Preventative treatment for Oral Mucositis

There are now various treatment options available to prevent and treat oral mucositis.

Oral care protocols for clinical practice associated with cancer therapy, developed by the Multinational Association for the Supportive Care of Cancer (MASCC) and the International Society of Oral Oncology (ISOO), contain recommendations for prevention aimed at reducing the severity of oral cancer.

The recommendations emphasize the need to improve educational programs for health care workers and patients receiving radiochemotherapy [37,38].

An effective program to reduce the risk of mucositis: oral hygiene, constant rinsing of the mouth to reduce the level of oral-oropharyngeal flora. For mouth rinses, the electrolyte mouth rinse Kaposol is recommended, containing high concentrations of calcium and phosphate, which successfully helps eliminate pain in patients with OM and reduces the severity of inflammation Cryotherapy has been proposed as a method to reduce the burden of OM in high-risk cancer patients [39].

In recent decades, approximately three hundred studies of more than three dozen means/methods of prevention and treatment of OM **(Figure 2)**, of which a few are assessed by experts as proven effective, dozens - as promising, corresponding to modern paradigm of OM pathogenesis, but requiring conducting more thorough and extensive clinical trials [40].

There are different protocols for oral care for cancer patients, in which the main focus is on prophylaxis and pain relief.

Oral mucositis is one of the extremely serious and complex complications of chemotherapy and radiation therapy in cancer patients [41-43]. To minimize the acute and chronic oral and systemic consequences of antitumor and radiation therapy, evaluation of oral pathology is necessary. Pre-treatment is aimed at reducing systemic infection, but currently none of the drugs taken has been able to achieve a full clinical effect. The literature does not pay much attention to the general treatment of oral complications in cancer patients. Prevention is important, and particular attention is paid to teaching patients about oral hygiene.

The clinical and economic significance of OM determines the need for further trials already proposed means and methods in oncology therapy and the development of new approaches to prevention and treatment of this pathology.

Conclusions: The clinical and economic significance of oral complications in cancer patients determines the need for further trials already proposed means and methods in oncology therapy and the development of new approaches to prevention and treatment of this pathology. The results of the review may help inform the effectiveness of safe preventive measures for oral mucositis in cancer patients.

Conflict of interest and financial disclosure: The author declares that he has no conflict of interest and there was no external source of funding for the present study. None of the authors have any relevant financial relationship(s) with a commercial interest.

Acknowledgements: "Not applicable"
DM: contributed to design, analysis and interpretation, and drafted the manuscript. critically revised the manuscript, final wrote.

References

1. Baskar R, Lee KA, Yeo R, Yeoh KW. Cancer and radiation therapy: current advances and future directions. Int J Med Sci, 2012; 9(3): 193-199. doi: 10.7150/ijms.3635.
2. Bayat Mokhtari R, Homayouni TS, Baluch N, et al. Combination therapy in combating cancer. Oncotarget, 2017; 8: 38022–38043.
3. El-Hussein A, Manoto SL, Ombinda-Lemboumba S, et al. A review of chemotherapy and photodynamic therapy for lung cancer treatment. Anticancer Agents Med Chem, 2021; 21(2): 149–161.
4. American Cancer Society Cancer Facts and Figures, 2023.
5. Lalla RV, Brennan MT, Schubert MM. Oral complications of cancer therapy. In: Yagiela JA, Dowd FJ, Johnson BS, et al., eds.: Pharmacology and Therapeutics for Dentistry. 6th ed. Mosby Elsevier, 2011; pp 782-798.
6. Vidya Sankar,Yuanming Xu,Yuanming Xu. Oral Complications from Oropharyngeal Cancer Therapy. Cancers, 2023; 15(18): 4548; https://doi.org/10.3390/cancers15184548
7. Brook I. Late side effects of radiation treatment for head and neck cancer. Radiat Oncol J, 2020; 38(2): 84-92. doi: 10.3857/roj.2020.00213.
8. Gupta N, Pal M, Rawat S, et al. Radiation-induced dental caries, prevention and treatment: a systematic review. Natl J Maxillofac Surg, 2015; 6: 160–166.
9. Elad S, Zadik Y. Chronic oral mucositis after radiotherapy to the head and neck: A new insight. Support. Care Cancer, 2016; 24: 4825–4830.
10. Bonar-Alvarez P, Padin-Iruegas E, Chamorro-Petronacci C, Gandara-Vila P, Lorenzo-Pouso AI, Somoza-Martin M, et al. Assessment of saliva and oral candidiasis levels 12, 24 and 36 months after radiotherapy in patients with head and neck cancer. J. Stomatol. Oral Maxillofac. Surg, 2021; 122: 566–572.
11. Nishii M, Soutome S, Kawakita A, Yutori H, Iwata E, Akashi M, et al. Factors associated with severe oral mucositis and candidiasis in patients undergoing radiotherapy for oral and oropharyngeal carcinomas: A retrospective multicenter study of 326 patients. Support. Care Cancer, 2020; 28: 1069–1075.
12. Hyer S, Kong A, Pratt B, et al. Salivary gland toxicity after radioiodine therapy for thyroid cancer. Clin Oncol (R Coll Radiol), 2007; 19(1): 83-86.
13. Ortigara GB, Schulz RE, Soldera EB, et al. Association between trismus and dysphagia-related quality of life in survivors of head and neck cancer in Brazil. Oral Surg Oral Med Oral Pathol Oral Radiol, 2019; 128: 235–242.
14. Sroussi HY, Epstein JB, Bensadoun RJ, et al. Common oral complications of head and neck cancer radiation therapy: mucositis, infections, saliva change, fibrosis, sensory dysfunctions, dental caries, periodontal disease, and osteoradionecrosis. Cancer Med, 2017; 6: 2918–2931.
15. Kojima Y, Yanamoto S, Umeda M, et al. Relationship between dental status and development of osteoradionecrosis of the jaw: a multicenter retrospective study. Oral Surg Oral Med Oral Pathol Oral Radiol, 2017; 124: 139–145.
16. Al Barmawi M, Al Hadid LA, Alqudah HN, Al Hadid WA, Shamoun SA. Measuring the quality of life among head-and/or-neck cancer patients with oral mucositis using the functional assessment of cancer therapy-general in Jordan. Asia Pac J Oncol Nurs, 2018; 5: 320–326.
17. Ramezani V, Ghadirian S, Shabani M, et al. Efficacy of curcumin for amelioration of radiotherapy-induced oral mucositis: a preliminary randomized controlled clinical trial. BMC Cancer, 2023; 23: 354. https://doi.org/10.1186/s12885-023-10730-8.
18. Fiwek P, Irga-Jaworska N, Wojtylak S, Biernat W, Emerich K, Pomiecko D. Assessment of Cytological Changes in the Oral Mucosa in Young Hematological Patients Treated with Systemic Chemotherapy. J Clin Med, 2023; 12(7): 2665. doi: 10.3390/jcm12072665.
19. Hong BY, Sobue T, Choquette L, et al. Chemotherapy-induced oral mucositis is associated with detrimental bacterial dysbiosis. Microbiome, 2019; 7: 66. https://doi.org/10.1186/s40168-019-0679-5.
20. Kumari P, Debta P, Dixit A. Oral Potentially Malignant Disorders: Etiology, Pathogenesis, and Transformation into Oral Cancer. Front Pharmacol, 2022; 13: 825266. doi: 10.3389/fphar.2022.825266.
21. Zhang T, Ma C, Zhang Z, Zhang H, Hu H. NF-κB signaling in inflammation and cancer. MedComm, 2021; 2(4): 618-653. doi: 10.1002/mco2.104.
22. Taniguchi K, Karin M. NF-κB, inflammation, immunity and cancer: coming of age. Nat Rev Immunol, 2018; 18(5): 309-324.
23. Raber-Durlacher JE, Brennan MT, Verdonck-de Leeuw IM, Gibson RJ, Eilers JG, et al. Dysphagia Section, Oral Care Study Group, Multinational Association of Supportive Care in Cancer (MASCC)/International Society of Oral Oncology (ISOOSwallowing dysfunction in cancer patients. Support Care Cancer, 2012; 20(3): 433-443. doi: 10.1007/s00520-011-1342-2.
24. Raber-Durlacher JE, Brennan MT, Verdonck-de Leeuw IM, Gibson RJ, Eilers JG, Waltimo T, et al. Oral mucositis. Oral Oncol, 2010; 46: 452–456.
25. Colella G, Boschetti CE, Vitagliano R, Colella C, Jiao L, King-Smith N, et al. Interventions for the Prevention of Oral Mucositis in Patients Receiving Cancer Treatment: Evidence from Randomised Controlled Trials. Curr Oncol, 2023; 30(1): 967-980. doi: 10.3390/curroncol30010074.
26. Satoshi Watanabe, Alexander V. Misharin, G.R. Scott Budinger the role of macrophages in the resolution of inflammation J Clin Invest, 2019; 129(7): 2619-2628. https://doi.org/10.1172/JCI124615.
27. Pulito C, Cristaudo A, Porta C, Zapperi S, Blandino G, Morrone A, et al. Oral mucositis: the hidden side of cancer therapy. J Exp Clin Cancer Res, 2020; 39(1): 210. doi: 10.1186/s13046-020-01715-7.
28. Logan RM, Stringer AM, Bowen JM, Gibson RJ, Sonis ST, Keefe DM. Serum levels of NFkappaB and pro-inflammatory cytokines following administration of mucotoxic drugs. Cancer Biol Ther, 2008; 7: 1139–1145. doi: 10.4161/cbt.7.7.6207.
29. Villa A, Sonis ST. Pharmacotherapy for the management of cancer regimen-related oral mucositis. Expert Opin Pharmacother, 2016; 17: 1801–1807. doi: 10.1080/14656566.2016.1217993.
30. Anderson G, Ebadi M, Vo K, Novak J, Govindarajan A, Amini A. An Updated Review on Head and Neck Cancer Treatment with Radiation Therapy. Cancers (Basel), 2021; 13(19): 4912. doi: 10.3390/cancers13194912.
31. Harris DJ, Eilers J, Harriman A, et al. Putting evidence into practice: evidence-based interventions for the management of oral mucositis. Clin J Oncol Nurs, 2008; 12: 141-152.
32. WHO toxiticy criteria, 2003.
33. Chaveli-López B, Bagán-Sebastián JV. Treatment of oral mucositis due to chemotherapy. J Clin Exp Dent, 2016; 8: e201-209.
34. PDQ Supportive and Palliative Care Editorial Board. Oral Complications of Chemotherapy and Head/Neck Radiation (PDQ®): Patient Version. 2023 Dec 6. In: PDQ Cancer Information Summaries. Bethesda (MD): National Cancer Institute (US), 2002.
35. National Cancer Institute, Oral complications of chemotherapy and head/neck radiation (PDQ®), 2008.
36. Carneiro-Neto JN, de-Menezes JD, Moura LB, Massucato EM, de-Andrade CR. Protocols for management of oral complications of chemotherapy and/or radiotherapy for

oral cancer: Systematic review and meta-analysis current. Med Oral Patol Oral Cir Bucal, 2017; 22(1): e15-e23. doi: 10.4317/medoral.21314.
37. Keefe DM, Schubert MM, Elting LS, et al., Cancer, 2007; 109(5): 820–831.
38. Naidu MU, Ramana GV, Rani PU, Mohan IK, Suman A, Roy P. Chemotherapy-induced and/or radiation therapy-induced oral mucositis--complicating the treatment of cancer. Neoplasia, 2004; 6(5): 423-431. doi: 10.1593/neo.04169.
39. Alterio D, Jereczek-Fossa BA, Fiore MR, Piperno G, Ansarin M, Orecchia R. Cancer treatment-induced oral mucositis. Anticancer Res, 2007.
40. Pulito C, Cristaudo A, Porta CL, et al. Oral mucositis: the hidden side of cancer therapy. J Exp Clin Cancer Res, 2020; 39; 210. https://doi.org/10.1186/s13046-020-01715-7.
41. Pai RR, Ongole R, Banerjee S, Prasad K, George LS, George A, et al. Oral Care Protocol for Chemotherapy- and Radiation Therapy-Induced Oral Complications in Cancer Patients: Study Protocol. Asia Pac J Oncol Nurs, 2019; 6(4): 417-423. doi: 10.4103/apjon.apjon_30_19.