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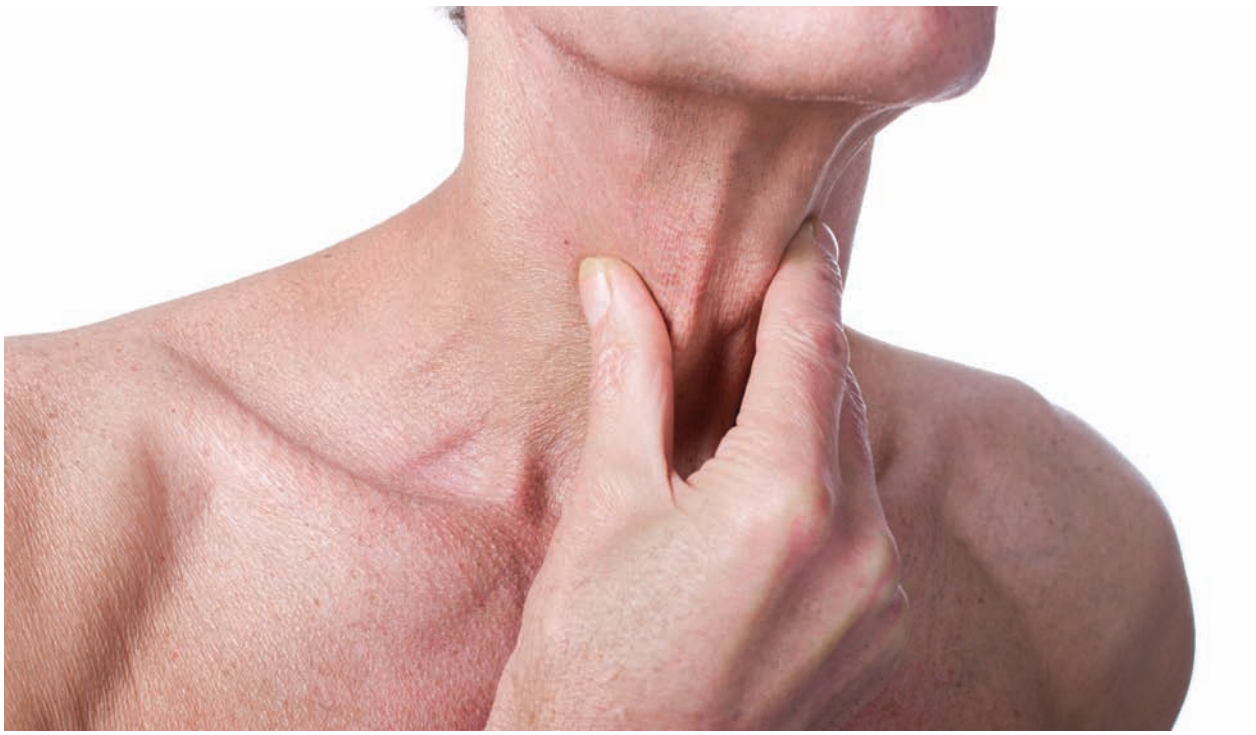

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Oral and oropharyngeal cancer: An overview for nurses

BY RICHARD L. PULLEN, JR., EdD, MSN, RN, CNE, CNE-CL, ANEF, FAAN

Abstract: Approximately 1 in 60 men and 1 in 141 women will develop oral and oropharyngeal cancer in the US. This article presents the risk factors, pathogenesis, clinical presentation, and management of patients with oral and oropharyngeal squamous cell carcinoma.

Keywords: cancer, oral cancer, oropharyngeal cancer, oral surgery

An estimated 476,125 cases of oral and oropharyngeal squamous cell carcinoma (SCC) were reported worldwide in 2020.[1] About 2.5 per 100,000 people died from oral and oropharyngeal cancer between 2016 and 2020.[2] Approximately 1 in 60 men and 1 in 141 women will develop oral and oropharyngeal cancer in the US.[3] About 54,540 new cases and 11,580 deaths from oral and oropharyngeal SCC are projected in the US in 2023.[1]

Oral and oropharyngeal cancer are two types of head and neck cancer, specifically SCC. The other cancer types are basal cell carcinoma, adenocarcinoma, and lymphoma. About 90% of head and neck cancers are SCC, originating from the stratiform squamous epithelial cells of the mucosa.[4-6] Oral SCC affects the lips, the front two-thirds of the tongue, buccal mucosa, gingiva, hard palate, and retromolar trigone, a small region behind the last molar, the bottom row of teeth. Oropharyngeal SCC affects the back one-third of the tongue, tonsils, soft palate, nasopharynx, oropharynx, and hypopharynx.[4-6] Most cases occur between the fifth and seventh decades of life.[7] This article presents the risk factors, pathogenesis, clinical presentation, and management of patients with oral and oropharyngeal SCC.

Risk factors

Most researchers consider SCC of the oral mucosa a different disease than

SCC of oropharyngeal mucosa because most oral SCC cases occur secondary to tobacco and alcohol consumption. In contrast, most oropharyngeal SCC is related to human papillomavirus (HPV).[8-11]

Tobacco is smoked, chewed, sniffed, or sucked by 1.3 billion people worldwide.[8,12] Tobacco users are 5-10 times more likely to develop oral SCC than nontobacco users.[8] Tobacco contains more than 70 carcinogenic chemicals, including polycyclic aromatic hydrocarbons and N-nitrosamines, damaging and altering the DNA.[8] Alcohol irritates the oral and oropharyngeal tissues, metabolizes to carcinogenic acetaldehyde, and generates reactive oxygen species that damage and alter DNA.[8] Tobacco and alcohol consumption also increases the risk of oropharyngeal SCC, but they are not the cause in most cases.[8]

HPV infection is the predominant cause of oropharyngeal SCC.[2,8,10,11] There are more than 100 HPV subtypes, 40 of which are transmitted through oral, anal, and vaginal sex, including the highly oncogenic HPV-16.[2,8,10,11] HPV-driven oral and oropharyngeal cancer has steadily increased since 2008.[2,8,10,11] This rise is partly due to the increasing practice of unprotected sex, including oral sex, with multiple partners.[2,8,10,11]

Once infected with HPV, oncoproteins cause rapid cell growth and DNA mutation.[13,14] Approximately 70% of oropharyngeal SCCs are caused by HPV-16.[2,10] An estimated 42 million Americans are infected with oncogenic subtypes of HPV, including HPV-16.[15]

Other risk factors for developing oral and oropharyngeal cancer include immunosuppression; oral bacterial infections; chronic inflammation of the oral and oropharyngeal mucosa; poor oral hygiene; poorly fitted dentures; poor nutrition; poverty; obesity; environmental pollutants, such as dust, pollen, and asbestos; exposure to heavy metals, such as nickel and arsenic; and UV light.[1-3,8] Men are twice as likely to have oral and oropharyngeal cancer than women.[8]

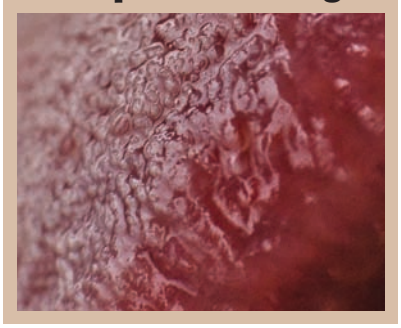
The epithelium and evolution of SCC

The oral cavity and oropharynx have two types of epithelium: squamous keratinized and stratified squamous nonkeratinized.[13,14] The former protects the mucous membranes from mechanical stress, microbial penetration, and radiation and provides a barrier to prevent fluid loss; the latter protects the moist internal membranes, such as the oral cavity, esophagus, upper gastrointestinal tract, cornea, and lining of the vagina.

Beneath the epithelium is the basement membrane.[13,14] The basement membrane consists of collagen, supports the epithelial and endothelial cells, regulates cell behavior, and acts as a barrier to tumor invasion. Beneath the basement membrane is the lamina propria—connective tissue supporting the mucosal endothelium and regulating immune response—which contains blood vessels, lymphatics, and nerves.[13,14]

Oral potentially malignant disorders (OPMD) occur when oral and oropharyngeal dysplastic lesions carry a high risk for malignant transformation in the epithelium.[16,17] Dysplasia is abnormal tissue growth from damage to the epithelium. It is graded as mild, moderate, or severe and is a reliable indicator of malignant transformation.[13,14,16,17]

Leukoplakia on tongue



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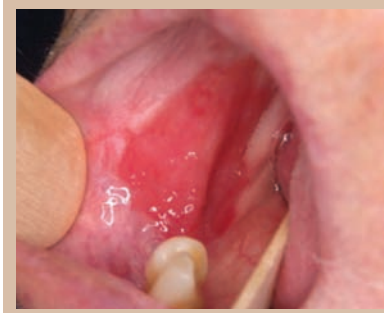
Erythroplakia on buccal mucosa[41]



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Dysplastic lesions can transform into several pathologic premalignant conditions.[13,14,16,17] Leukoplakia is a potentially malignant white patch or plaque in the mucosa strongly associated with tobacco use (see *Leukoplakia on tongue*). Microscopically, leukoplakia has a thick keratin layer and appears white when wet, given keratin is impervious to water.[13,14,16,17] Leukoplakia is generally painless and can be scraped away. Lesions may become thick, bumpy, or nodular. Wartlike leukoplakia is called verrucous leukoplakia. Proliferative verrucous leukoplakia presents as multiple white patches and plaques likely to grow, spread, and develop into SCC.[13,14,16,17]

Lesions may evolve into erythroleukoplakia, a red and white plaque with a high potential for malignancy.[13,14,16,17] The red areas reflect immature cells and DNA damage in the epithelium. As immature cells proliferate, the epithelium becomes thin, allowing easy visibility of blood vessels and making the lesion(s) completely red (see *Erythroplakia on buccal mucosa*). Erythroplakia reflects severe dysplasia and is the earliest form of SCC called carcinoma in situ (CIS). Most CISs become invasive SCC if not treated aggressively. SCCs begin from squamous cell mutations activated by oncogenic genes or deactivated tumor suppressor genes (see *SCC on tongue*).

SCC on tongue

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Cancerous cell division occurs when oncogenic genes, such as epidermal growth factor, are activated.[13,14,16,17] Tumor suppressor genes encode proteins, such as protein 16 (p16), protein 53 (p53), and retinoblastoma protein (pRb), to regulate cell division.[13,14] Uncontrolled cell division results in tumor development; invasion of the basement membrane, blood vessels, and lymphatics; and metastasis throughout the body.

Signs and symptoms

Most oral cancers are visible and palpable.[16-18] Crusting, bleeding, ulceration, induration, and color changes in lesions may be visible. Oropharyngeal lesions may manifest as pharyngitis, dysphagia, or coughing. Masses under the jaw and neck may indicate lymph node invasion. Patients may have lung, liver, and bone metastasis symptoms (see *Signs and symptoms of oral and oropharyngeal cancer*).

Diagnosis and staging

Diagnosing and staging oral and oropharyngeal cancer require a history and physical examination; evaluation of risk factors; endoscopy, such as nasopharyngoscopy, laryngoscopy, or pharyngoscopy; or imaging studies, such as computed tomography scan, MRI, positron emission tomography, bone scan, and tissue biopsy (the most conclusive evidence).[19]

The pathologist uses the American Joint Committee on Cancer's (AJCC) Tumor, Node, and Metastasis (TNM) Classification System to evaluate tumor(s).[5,19,20] **T** is the size of a primary tumor. **N** occurs when cancer cells from a primary tumor invade the lymph nodes. **M** occurs when cancer cells invade body organs. Numbers or letters following T, N, or M comprise stage groupings.

The AJCC has two TNM classifications for oral and oropharyngeal cancers. The first TNM classification is for lip, oral cavity, and HPV-16-negative oropharyngeal cancer and includes the following stages. Stage 0 is confined to the epithelium (in situ). Stage I is less than or equal to 2 cm without invasion of lymph nodes or body organs. Stage II is greater than 2 cm but not larger than 4 cm without invasion of lymph nodes or body organs. Stage III is greater than 4 cm without lymph nodes or body organ invasion. Stage III can also include cancer of any size with regional lymph node involvement. Stage IV, cancer of any size, may involve the jaw, chin, mouth, tongue, lymph nodes, or body organs.

The TNM Classification for HPV-16 positive oropharyngeal cancer includes five stages. Stage I is less than or equal to 4 cm without invasion of lymph nodes or body organs. Stage II is not larger than 4 cm and involves lymph nodes or is larger than 4 cm involving lymph nodes and may have spread to the tongue, epiglottis, and larynx, but not to distant organs. Stage III is greater than 4 cm, invading the epiglottis, tongue muscle, larynx, pterygoid plate, hard palate, or jaw. Stage III may include invasion of lymph nodes, but not body organs. Stage IV involves any sized tumor invading lymph nodes and body organs. The evidence suggests that patients with HPV-16-positive oral and oropharyngeal cancer have a better prognosis than patients with HPV-negative disease because of the sensitivity to treatment.[10,21]

The pathologist also grades cancer on a 1 to 4 scale according to the appearance of cells under the microscope.[13,14] Grade 1 tumors (Low grade) are well-differentiated, meaning most cells look normal, with a few immature cells indicating premalignant status. Grade 2 tumors are moderately differentiated (Intermediate stage). Grade 3 tumors are poorly differentiated (High grade). Grade 4 tumors are undifferentiated, look disorganized under the microscope, and metastasize rapidly (High grade).

Inactivation of the tumor suppressor gene p16 is associated with HPV

Signs and symptoms of oral and oropharyngeal cancer

- Lesions on the lip or mouth that do not heal.
- Edema of the lips or mouth.
- Oral or pharyngeal leukoplakia or erythroplakia
- Persistent oral bleeding.
- Pharyngitis.
- Globus sensation (feeling like something is stuck in the throat).
- Difficulty chewing, dysphagia, or odynophagia .
- Cervical lymphadenopathy
- Otalgia
- Difficulty moving the mandible or tongue.
- Numbness of the lip, tongue, mouth, or pharynx .
- Pain and discomfort wearing dentures.
- Loose teeth.
- Hoarseness
- Lesion or mass on the lip, mouth, or pharynx.
- Unexplained weight loss.
- Persistent halitosis.

infection. The presence of p16 in tissue is a hallmark of oncogenic HPV-16. HPVs also encode E6 and E7 proteins directly responsible for HPV-driven oncogenesis. Additional tumor markers in SCC include carcinoembryonic antigen, ferritin, SCC antigen, alpha-fetoprotein, p53, and pRb.

The 5-year survival rate is an estimate relative to the cancer stage and the presence of comorbidities.[22] The American Cancer Society uses information from the Surveillance, Epidemiology, and End Results (SEER) database to report survival rates.[22] SEER groups cancers into localized, regional, and distant, but not according to the AJCC TNM Classification System: (1) Localized: The cancer is confined to the tissue where it originated; (2) Regional: Cancer invades nearby structures or lymph nodes; (3) Distant: Cancer invades body organs. Pleural effusion, shortness of breath, reduction in oxygen saturation, abdominal distension from ascites, bone pain, and pathologic fractures may indicate lung, liver, and bone metastasis.

The 5-year survival rates for oral cancers vary, with the survival rate for oropharyngeal cancer slightly higher for regional disease than localized disease (see *5-year survival rates*).

SEER 5-year survival rates[22]

Cancer	Localized	Regional	Distant
Lip	94%	63%	38%
Tongue	84%	70%	41%
Floor of the mouth	73%	42%	23%
Oropharyngeal	59%	62%	29%

Management

Oral and oropharyngeal SCCs require an interprofessional team.[18,21] Treatment methods are used alone or in combination, depending on the tumor's stage, grade, and location.[21,23,24] Surgery is usually the initial method in oral SCC. It may be combined with radiation, chemotherapy, targeted therapy, and immunotherapy. Oropharyngeal SCC is treated with radiation, chemotherapy, targeted therapy, and immunotherapy.

Surgery[21,25]

Surgery involves the removal of the entire primary tumor and its margins with normal tissue. Surgery is usually the initial treatment for premalignant lesions and early-stage SCC that have not invaded other tissues. Surgical excision is recommended for severe dysplastic lesions and SCC in situ. Moderate dysplastic lesions may be removed by carbon-dioxide laser to minimize bleeding and difficulty in articulating, chewing, and swallowing.

Microscopic surgery (Moh's procedure) is often necessary for lip SCC. Oral cavity SCCs are easily visible, accessible, and removable. Typically, SCC is located in the front part of the tongue.

A partial, hemi-, or total glossectomy may be required. A total glossectomy prevents talking or swallowing foods or fluids without aspirating unless reconstructive surgery is performed. Mandiblectomy (removal of the jaw) and maxillectomy (removal of the hard palate) may be necessary for extensive SCC, requiring reconstructive surgery. Oropharyngeal SCC may require a mandibulotomy (an incision in the neck allowing access to all of the tongue and pharynx).

Trans-oral robotic surgery (TORS) has become a standard approach to accessing oropharyngeal tissues. In TORS, a surgeon uses a special endoscope, camera, and technology to remove SCC while preserving swallowing and speech. Large SCCs in the larynx may require a laryngectomy to prevent aspiration. Oral and oropharyngeal SCCs often invade lymph nodes in the head and neck.[18,21,25] Removing these lymph nodes, called lymph node dissection (LND), helps contain the SCC while removing the primary tumor.[18,21,25] A combination of chemotherapy and radiation may follow LND to ensure no cancer cells remain in the lymph nodes. Tracheostomy and enteral nutrition are supportive treatments resulting from surgical intervention, such as LND.

Radiation therapy[18,21,26]

Radiation therapy (RT) uses high-energy X-rays to damage and destroy DNA in cancer cells. The most common type of RT in oral and oropharyngeal cancer is external beam radiation therapy (EBRT). EBRT is administered like an X-ray. RT may be used for small cancers; individuals not candidates for surgery because of advanced disease or comorbidities; and in conjunction with surgery, chemotherapy, immunotherapy, and targeted therapies. RT is a palliative measure in advanced cancer to control pain, decrease bleeding, and promote swallowing. RT can cause inflammation, edema, pain, and infection in the oral and oropharyngeal tissues.

Medication[21,27]

Chemotherapy prevents cells from dividing quickly, decreasing the spread of cancer and shrinking the primary tumor. Adjuvant chemotherapy is used with surgery or radiation at different times during treatment. Chemotherapy with or without radiation is used when surgery cannot remove extensive tumors, such as Cisplatin, a prototype to manage oral and oropharyngeal cancer (see *Chemotherapy: Cisplatin*).[21,27] Immunotherapy boosts a person's immune system to identify, attack, and destroy malignant

cells.[21,27] Immunotherapeutic agents called immune checkpoint inhibitors (ICIs) block checkpoint proteins to destroy malignant cells. Checkpoints are a normal part of a person's immune system. Nivolumab is one ICI prototype for treating oral and oropharyngeal cancer (see *Immunotherapy: Nivolumab*).[29] Targeted therapies, a form of immunotherapy, target genes, proteins, and tissues of a specific malignancy to block its growth and spread. Cetuximab is one targeted therapy prototype (see *Targeted therapy: Cetuximab*).[30]

Nursing interventions

The nurse should conduct a health history interview and head-to-toe physical assessment focusing on inspecting the lips, buccal mucosa, tongue, floor of the mouth, hard and soft palate, and pharynx.[8,16,18] The nurse should assess for the presence of comorbidities; medication history; history of tobacco or alcohol consumption; and unprotected oral, vaginal, or anal sex.[8,16,18] The mucosa should be assessed for moisture, dryness, color, lesions, edema, nodules, or malformation.[8,16,18] Assessment findings should be correlated with signs, symptoms, surgical procedures, results of diagnostic studies, and treatment regimens to develop a patient teaching plan.[1-3,10-12,18,25-38]

Postoperative care depends on the type and extent of a surgical procedure.[18,21,24,25]

Managing the airway, preventing hemorrhage, preventing infection, ensuring optimal nutrition, and promoting comfort are common priority goals in patients undergoing excision of a tumor, Mohs procedure, glossectomy, mandibulectomy, mandibulotomy, laryngectomy, and LND.[18,21,24,25]

For example, most patients after LND will be hospitalized for at least a few days.[18,21,24,25]

Tracheostomy and percutaneous endoscopic gastrostomy tube, usually temporary, may be necessary to ensure optimal gas exchange and nutrition while the surgical sites heal.[18,21,24,25] The head of the patient's bed should be elevated 30 degrees or higher to promote oxygenation and prevent aspiration.[18,25] Patients may have multiple incisions and a variety of absorbent foam mould dressings and vacuum suction tubes in the head and neck region.[18,25] Wound drainage and dressings should be monitored for excessive bleeding.[18,25] The patient should avoid the Valsalva maneuver to prevent tension on the carotid artery and a well-vascularized graft to avoid hemorrhage.[18,25] Numbness in the neck, shoulder, and ear may occur following LND from damage to the spinal accessory and facial nerves.[18,25] Shoulder dysfunction usually requires extensive physical therapy.[18,25] Patients may have difficulty speaking and swallowing food and liquids re-

Chemotherapy: Cisplatin[21, 27]

Action, Side Effects, and Adverse Effects	Priority Assessment
Action: Binds to N7 reactive center on purine residue to cause DNA damage to cancer cells, blocks cell division, and results in apoptotic cell death. **Side Effects and Adverse Effects:** Anorexia, nausea, vomiting, diarrhea, alopecia, infection, dry skin, loss of ability to taste food, dehydration, electrolyte imbalances, neurotoxicity, neuropathy, ototoxicity, elevated liver enzymes, nephrotoxicity, leukopenia, thrombocytopenia, hemolytic anemia, impaired fertility, eye disorders, such as retinal toxicity	Assess complete blood count (CBC) for anemia, thrombocytopenia, and leukopenia and leukocytosis. Assess cardiac rate and rhythm. Assess renal function, including blood urea nitrogen (BUN), serum creatinine, estimated glomerular filtration rate, and creatinine clearance. Assess serum liver enzymes, bilirubin, prothrombin time, albumin, and proteins for hepatic function. Assess serum electrolytes, especially sodium, potassium, calcium, and magnesium. These electrolytes tend to decrease during therapy. Assess for symptomatic hypomagnesemia or hypocalcemia, including muscle irritability or cramps, clonus, tremor, carpopedal spasm, and/or tetany. Assess for changes in mental status and the presence of paresthesias, indicating possible neurotoxicity. Assess for signs and symptoms of infection, including fever, increasing fatigue or malaise, or pharyngitis. Assess hearing before, during, and after therapy. Periodic audiometric testing should be performed. Assess vision. Periodic eye exams should be instituted. Assess hydration status, including BUN. Ensure the patient is well-hydrated through oral and I.V. intake during therapy. Hydration is essential to prevent nephrotoxicity.

Immunotherapy: Nivolumab[22, 27]

Action, Side Effects, and Adverse Effects	Priority Assessment
Action: A monoclonal antibody and an immune checkpoint inhibitor (ICI). Immune checkpoints are pathways in the immune system that regulate identifying and destroying malignant cells. **Side Effects and Adverse Effects:** Arthralgia, anorexia, diarrhea, constipation, headache, fatigue, malaise, dry skin, pruritis, Stevens-Johnson Syndrome or toxic epidermal necrolysis, encephalitis, insomnia, pneumonia, myocarditis, thrombocytopenia, endocrine disorders, hepatotoxicity, nephrotoxicity, electrolyte imbalances, colitis, and anaphylaxis, impaired fertility.	Assess CBC and correlate with anemia and bleeding. Assess heart rate and rhythm, ECG, respiratory rate and rhythm, oxygen saturation, and breath sounds. Assess mental status changes possibly indicating encephalitis. Assess for signs and symptoms of immune-mediated colitis, including blood or mucous in the stool, abdominal cramping, and diarrhea. Assess serum electrolytes, especially sodium, potassium, calcium, and magnesium. Assess serum liver enzymes, bilirubin, prothrombin time, albumin, and proteins for hepatic function. Assess renal function, including BUN, serum creatinine, estimated glomerular filtration rate, and creatinine clearance. Assess for skin dryness, lesions, rashes, or blisters. Blistering may be a sign of Stevens-Johnson Syndrome. Assess for changes in weight, energy, and libido indicating endocrine dysfunction. Assess for abnormal thyroid studies and blood glucose levels indicating endocrine dysfunction.

quiring speech therapy, including swallowing studies.[18,25]

The patient should be assessed for a chyle leak.[18,25] Chyle is a by-product of digestion and absorption of fat and appears as a milky-white substance in drainage systems and dressings.[18,25] It occurs from injury to the thoracic duct during surgery and can lead to dehydration, electrolyte imbalances, protein deficiency, tissue loss, poor wound healing, and immunodeficiency.[18,25] A very low-fat diet is implemented.[18,25]

Other interventions include elevating the head of the bed, stool softeners to prevent straining, pressure dressings, and fluid and electrolyte replacement.[18,25] Speech, nutritional, physical, and respiratory therapy collaborate with the nurse and physician in the patient's recovery and discharge planning.[18,25] Postoperative discharge teaching includes reporting bleeding from the wounds, redness or swelling of wounds, difficulty breathing, a new cough, and difficulty eating or drinking.[18,25] Patients should engage in activities as tolerated to prevent pneumonia and thrombosis.[18,25] Mobility, sufficient fluids, and dietary fiber are necessary to avoid constipation, causing stool strain and tension on the surgical sites.[18,25]

Complications of chemotherapy include inflammation and ulcers in mucous membranes, neuropathy, infection, and dry skin.[21,27,28,35-37] Drinking plenty of water, using a saliva substitute, sucking on ice chips, mouth rinses with baking soda and salt, and using hard candy or sugarless gum is soothing to the mucous membranes.[21,27,28,35-37] The patient should report numbness, tingling, bruising, and fever.[21,27,28,35-37] Medications for neuropathic pain and infection may be prescribed.[35-37] Patients should maintain good body hygiene and avoid groups of people to prevent infection.[35-37] Complications of immunotherapy and targeted therapies include cardiopulmonary, endocrine, and cutaneous pathologies.[29,30,35-37] Patients should report chest pain, dyspnea, increased fatigue, mood changes, and the onset of new lesions, rashes, or blisters.[29,30] Patients receiving radiation therapy should report a sunburn-like rash in the treated area; redness, pain, and soreness in the mouth or throat; hoarseness; trouble swallowing; and severe fatigue.[18,21,26] Meticulous skin care is crucial in patients receiving chemotherapy, immunotherapy, targeted therapy, and radiation therapy.[26,29,30,35-37] Cutaneous adverse reactions are a significant source of discomfort for the patient.[35-37] Skincare is paramount in patients receiving chemotherapy, immunotherapy, targeted therapies, and radiation therapy (*see General skin care measures*).[37]

Nurses must teach the patient about the impact of tobacco and alcohol consumption, sexual practices, and nutrition in developing and preventing oral and oropharyngeal cancer.[10-14,34,38,39] These risk factors influence how the patient responds to treatment.[10-14,34,38]

Abstaining from sexual practices, using condoms and dental dams properly during sex, and HPV vaccination effectively reduce the incidence of oropharyngeal cancer.[7,8,10,31-34] A

Targeted Therapy: Cetuximab[22,27]

Action, Side Effects, and Adverse Effects	Priority Assessment
Action: A monoclonal antibody targeting and blocking the epidermal growth factor receptor protein on cells to slow or stop malignant cells from growing.	Assess respiratory rate, depth, rhythm, breath sounds, and oxygen saturation.
	Assess cardiac rate and rhythm.
	Assess CBC for leukopenia indicating infection.
Side Effects and Adverse Effects: Nausea, vomiting, diarrhea, constipation, stomatitis, insomnia, depression, fatigue, malaise, pulmonary embolism, cough, dyspnea, endocrine disorders, rash, dermatitis, sunburn feeling of the skin, acne, pruritis, hand-foot skin reactions, photosensitivity, kidney failure, leukopenia, fever, sepsis, anaphylaxis, electrolyte imbalances, and impaired fertility.	Assess renal function, including BUN, serum creatinine, estimated glomerular filtration rate, and creatinine clearance.
	Assess serum electrolytes, especially sodium, potassium, calcium, and magnesium. These electrolytes tend to decrease during therapy.
	Assess for signs and symptoms of hypomagnesemia, such as severe fatigue, paresthesias, muscle twitching, and cardiac dysrhythmias.
	Assess for skin dryness, peeling, lesions, rashes, and sensitivity to light.
	Assess for hand-foot skin reactions, including thickening and tenderness of the palms and soles with blisters.

well-balanced diet helps prevent oral and oropharyngeal cancer and is necessary during cancer treatment consisting of carbohydrates, protein, fat, and plenty of fruits and vegetables.[7,8,34] Oily fish, such as tuna, salmon, and sardines; chicken; pork; milk; and yogurt should be consumed.[7,8,34] Hot, spicy, and highly acidic fruits, juices, and rough food such as potato chips should be avoided to prevent irritation to sensitive oral and oropharyngeal mucosa.[34] Dry foods, toast, and unsalted crackers are beneficial when the patient has anorexia or nausea, secondary to medication and radiation therapies.[40] Soothing and nutritional drinks with milk-like consistency and various flavors are available.[40] An oncology dietitian can develop a plan to meet dietary needs, especially when a patient is not hungry, and their ability to communicate, taste, chew, and swallow is impaired.[40] Daily oral hygiene is preventive and necessary during treatment.[40] Patients should brush their teeth with a soft bristle toothbrush and gently floss twice daily to prevent infection and dental decay.[40]

General skin care measures[26, 35-37]

- Drink 8 to 10 glasses of water daily to maintain hydration.
- Avoid tobacco and alcohol consumption.
- Take a short bath using lukewarm water and mild soap. Gently cleanse the area treated with radiation using one's hands; avoid abrasive washcloths and sponges.
- Take soothing baths with oatmeal-containing products.
- Apply gentle lotions or creams after bathing.
- Avoid alcohol-containing products. A soothing skin protectant may be used with lotions or creams. It can be used on an area treated with radiation.
- Use an electric razor to limit skin irritation and bleeding when shaving. Avoid shaving an area treated with radiation.
- Avoid deodorant, talcum powder, colognes, and perfumes.
- Wear a wide-brimmed hat to protect the head and neck from light sources. Seek a shaded area when outdoors.
- Use only soft fabrics like cotton for clothing and bed linen. Avoid irritating fabrics such as wool.
- Wear loose-fitting clothes.
- Wear a sun protection factor of 30 or above.

Teach patients to avoid large groups of people, wear masks, and perform optimal hand hygiene.

Conclusion

Oral and oropharyngeal SCC is preventable. Avoiding tobacco consumption, limiting alcohol intake, engaging in safe sex practices, having good nutrition, maintaining good oral hygiene, and having regular dental checkups are effective ways to reduce risk. Early recognition and treatment of premalignant lesions and aggressive treatment of SCC are crucial to reducing morbidity and mortality. ■

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Richard Pullen is a professor and the RN-to-BSN program director at the Texas Tech University Health Sciences School of Nursing. He is also a member of the *Nursing2023 editorial board.*

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