Episode 76 – Oral Cancer: Part 1 January 13, 2023

Introduction

Oral cancer includes cancers of the lip and oral cavity (e.g., gingiva, anterior two-thirds of the tongue, hard palate, floor of the mouth, buccal mucosa, retromolar pad). Oropharyngeal cancer is cancer of the oropharynx (e.g., base of the tongue, tonsillar regions, soft palate, posterior and lateral pharyngeal walls). Oral cancer is a disease group with high morbidity and mortality. Survival rates can vary significantly across geographical location, tumour site, and stage at diagnosis. Those diagnosed with advanced disease have poorer outcomes than those with early disease. [1]

Squamous cell carcinoma accounts for >90% of oral cancer cases and mainly affects individuals 50-70 years who consume tobacco and alcohol. It may also be associated with human papillomavirus (HPV) infection, genetic susceptibility, and passive smoking exposure. [2]

Global prevalence of oral cancer

Lip and oral cavity cancers ranked 16th among all cancers (for both sexes); combined with cases of oropharyngeal cancers, the rank increased to the 13th most common cancer globally. More than 370,000 new cases of lip and oral cavity cancer are diagnosed and more than 170,000 individuals die from these cancers every year (according to 2020 data). Worldwide, 98,400 new cases and 48,100 deaths from oropharyngeal cancer were estimated in 2020. [3] [4]

Oral cancer is more common in males and older individuals, more deadly in males, and varies strongly by socioeconomic status. Treatment availability, outcomes, and survival rates are mainly better in high-income countries. [3] [4]

Tobacco, alcohol, and areca nut (betel quid) use are among the leading causes of oral cancer. In North America and Europe, HPV infections are responsible for a growing percentage of oral cancers among young individuals. [3]

Variations of oral cancer are observed relative to location. The tongue is the most common site of oral cavity cancers in Europe, the United States, and Japan, whereas the buccal and lip mucosa are more common cancer locations in some regions of Asia. These variations relate to the main risk factors (e.g., tobacco and alcohol use in Western countries; betel quid and tobacco chewing in South and Southeast Asia). [4]

Canadian statistics* [5] [6] [7]

Cancer	Females			Males			Both sexes		
	Cases	Deaths	5-year survival	Cases	Deaths	5-year survival	Cases	Deaths	5-year survival
Head & neck	2,000	560	65%	5,400	1,500	64%	7,400	2,060	64%
Trend	→	↓	-	↑	→	-	↑	→	-
All cancers	112,800	40,000	66%	121,100	45,100	62%	233,900	85,100	64%
Trend	↓	↓	-	↓	↓	-	↓	↓	-

*Projected number of cancer cases and deaths in 2022
Trend direction of most recent trend in case or death rate reflects statistically significant increase (↑) or decrease (↓) or no change (→)

Ontario statistics* [8]

Cancer	Females		Males		Both sexes	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Head & neck	860	210	2,100	610	2,960	820
All cancers	45,500	14,800	47,800	16,500	93,300	31,300

*Projected number of cancer cases and deaths in Ontario in 2022

Prevalence in youth

A systematic review and meta-analysis by Silveira de Paula et al. (2020) evaluated 42 studies to determine the prevalence of malignant oral lesions in children and adolescents around the world. Of the 64,522 biopsies, the prevalence of malignant lesions was 1.93% (n=1,100). Lymphomas and sarcomas were the most commonly diagnosed cancers. The prevalence of oral squamous cell carcinoma was 8.38%, even though risk factors such as smoking and alcohol consumption have not been strongly found in this population. Cancer in children and adolescents occur, but at an overall low frequency. Suspicious lesions at any age require evaluation. [9]

Types of oral cancers* [10]

Cancer	Description
Squamous cell carcinoma	Almost all oral cavity and oropharynx cancers are squamous cell carcinomas (>90%). Most lip cancers are squamous cell carcinomas. The earliest form of squamous cell cancer is carcinoma in situ (i.e., cancer cells are only in the epithelium). Invasive squamous cell cancer is where the cancer cells have infiltrated (spread) deeper into the tissues. [11] [12]
HPV-related cancer[1]	Infection with certain high-risk types of the human papillomavirus (HPV) causes most of the squamous cell cancers of the oropharynx (i.e., HPV-positive cancer). HPV is rarely associated with oral cavity cancer. HPV-positive cancers are seen more often in young individuals with no history of tobacco or alcohol use. These cancers tend to have a better prognosis than squamous cell cancers not related to an HPV infection (HPV-negative cancer). This is most likely because HPV-positive tumours are more responsive to chemotherapy and radiation, partly due to the biology of the tumours and partly because healthy nonsmokers tolerate treatment better. [11] [13]
Verrucous carcinoma	Verrucous carcinoma is a rare type of squamous cell cancer most often found in the gingiva and buccal mucosa. It is a low-grade, slow growing cancer with minimal metastatic potential. Verrucous carcinoma manifests as a cauliflowerlike, exophytic mass that typically develops at sites of chronic irritation and inflammation. Pathogenesis of verrucous carcinoma is not yet fully elucidated, theories include HPV infection; chemical carcinogenesis induced by smoking and chewing tobacco, alcohol consumption, and betel nut chewing; and chronic inflammation. For example, associations have been found in individuals who chewed or inhaled tobacco and betel nuts, dipped snuff, and/or consumed alcohol. Lesions developed at the sites where tobacco was habitually placed in the mouth. Chronic inflammation appears to play a role in the development. For example, verrucous carcinomas have been reported in individuals with long-standing oral ulcerative lichen planus and chronic candidiasis. It is also associated with poor oral hygiene, ill-fitting dentures, and low socioeconomic status. [11] [14]
Minor salivary gland carcinoma	Minor salivary glands are located throughout the oral cavity and throat. Tumours of minor salivary glands are rare, but are often more cancerous than benign. Cancers of the minor salivary glands most often start in the palate. There are several types of minor salivary gland cancers, including mucoepidermoid carcinoma, adenoid cystic carcinoma, and polymorphous low-grade adenocarcinoma. [11] [15]

[1] Refer to Episodes 7, 53, and 61 for additional information on HPV.

Cancer	Description
Lymphoma	Oral cancers that develop in lymph tissue are known as lymphomas. The tonsils and base of the tongue contain lymphoid tissue where lymphomas can start. [11] [16]
Oral melanoma	Melanoma develops from malignant transformation of melanocytes. Oral melanoma is a very rare and aggressive malignancy that progresses rapidly, and show a worse prognosis than cutaneous melanoma. The clinical presentation is varied. It usually presents as a black-brown patch, macule, or nodular lesion with different shades of grey, red, purple, or areas of depigmentation. Lesions are usually asymmetric, with irregular borders, and can be sometimes multiple. Satellite foci surrounding the tumour have been reported. Up to a third are ulcerated. Some may present without clinical evidence of pigmentation (also known as amelanotic melanoma). Primary oral melanomas prefer the maxilla, mainly developing in the hard palate, gingiva, or alveolar ridge. Secondary oral melanomas (even rarer than primary tumours) commonly occur in the tongue. Most oral melanomas arise de novo in apparently normal oral mucosa, but up to a third are preceded by melanosis. Etiology, risk factors, and pathogenesis of oral melanoma remain unclear. Unlike its skin counterpart, it is not related to sun exposure as oral mucosa is protected from ultraviolet (UV) light. Denture irritation, infection, and tobacco smoking have been listed as possible risk factors, but a direct relationship is not substantiated. Oral melanoma prevalence increases with age, developing between the 4th and 7th decade of life, with an average age of 60. [17] [18]
Kaposi sarcoma	Kaposi sarcoma (KS) is linked to the human herpesvirus 8 (HHV8)[2] and human immunodeficiency virus (HIV).[3] It is associated with immunosuppression and is the first malignancy thought to develop from acquired immunodeficiency syndrome (AIDS). The oral mucosa is the initial site of KS in 22% of individuals with HIV. Intraoral KS most frequently affects the palate, gingiva, and dorsal tongue. Intraorally, KS appears as brown, blue, purple, or red patches and papules. Early lesions appear as flat macules or patches on the mucosal surface, over time they become nodular, with tendency to ulcerate and bleed. KS can also affect the salivary glands and may cause head and neck lymphadenopathy. [18] [19]

[2] Refer to Episode 53 and 61 for additional information on human herpesvirus 8.
[3] Refer to Episode 61 for additional information on HIV.

Cancer	Description
Basal cell carcinoma	Basal cell carcinoma (BCC) is the most common type of skin cancer (~80%). It most often develops on the head and neck, including the lips, although it can be found anywhere on the skin. These cancers start in the basal cell layer (lower part of the epidermis). BCC typically presents as a shiny or waxy pink or flesh-coloured papule or nodule with surface telangiectasia.[4] Pigmented nodular BCCs are more common in dark-skinned individuals. The tumour may enlarge and ulcerate, giving the borders a rolled appearance. It may also present as a crusting and recurrent bleeding sore that heals and returns. BCC is mainly caused by years of UV exposure (e.g., sun, indoor tanning), or develops in individuals who received radiation therapy as children. BCC usually grows slowly and rarely metastases, but it can be highly destructive and disfigure local tissues if treatment is delayed. Prognosis for BCC is very good. If not removed completely, BCC can recur in the same location. [20] [21] [22] [23]

*Nonexhaustive list

Risk factors for oral cancer

Etiology of oral cancer is multifactorial with many risk factors linked to its development**.** Main risk factors for lip, oral cavity, and oropharyngeal cancers are tobacco use, alcohol consumption, and betel quid use, which act independently or in combination. Lip and oral cavity cancers are the third most common cancer related to tobacco use, and about one quarter of oral cancer cases are related to harmful alcohol consumption. Both risk factors combined greatly increase risk of oral cavity cancers. [4] [24]

Risk factors

Risk factor	Description
Tobacco	All forms of tobacco use are major risk factors for lip and oral cavity cancer. Tobacco smoke contains dozens of known carcinogens. Risk of oral cancer and premalignant lesions increases with the amount of tobacco consumed and duration of tobacco use. This increased risk holds for all types and uses of tobacco. Types tobacco products include cigarettes, pipes, cigars, snuff, paan, chewing tobacco, smokeless tobacco, areca nut, and betel quid.

[4] Telangiectasias are small widened blood vessels on the skin.

Risk factor	Description
	More than 350 million individuals globally are estimated to use smokeless tobacco, a cultural habit that is particularly popular in South Asia and some Pacific Island countries. Specific risk factors for lip and oral cavity cancers are smokeless tobacco (chewing and snuff) and betel quid (paan), substances long been established as strong carcinogens. Chewing betel quid without tobacco is estimated to increase oral cavity cancer risk 2.5-fold; in combination with tobacco the risk increases 7.74-fold, with a cumulative effect over the life course. Betel quid chewing with tobacco is one of the most common forms of smokeless tobacco and is used by approximately 600 million individuals worldwide, with a higher prevalence in South and South-East Asia and the Pacific islands. The effect of betel quid chewing is a general stimulation, similar to the effects of tobacco or caffeine. Betel quid is a mixture of areca nut (nut of a palm tree, Areca catechu), slaked lime (calcium hydroxide) and betel leaf (leaf of the piper betel vine), and it can have tobacco (mainly in South-Central Asia) or not (in East Asia and Melanesia). All the ingredients are wrapped inside the betel leaf and made into a bunch commonly known as a quid, which is then chewed. It stains teeth and tissues dark red and is spit out after chewing for a few hours. [4] [24] [25]
Environmental tobacco smoke	A systematic review and meta-analysis showed individuals who were exposed to secondhand smoke had a 51% higher risk of developing oral cancer. [26]
E-cigarettes[5]	Scientific evidence regarding health effects of e-cigarettes is limited. While e-cigarette aerosols may contain fewer toxicants than cigarette smoke, studies evaluating whether e-cigarettes are less harmful than cigarettes are inconclusive. Due to the typically long duration of carcinogenesis and the relatively short history of e-cigarette use (e-cigarettes were introduced approximately early 2000s), caution should be taken regarding their cancer risk. More research is needed to assess the impact of e-cigarettes and vaping nicotine products on oral cancer. [4] [27]
Cannabis[6]	Similar to tobacco, cannabis contains various carcinogens (e.g., phenols, nitrosamines, vinyl chloride, polycyclic aromatic hydrocarbons). Long-term cannabis smoking is associated with similar pathologies as tobacco use, such as leukoplakia and erythroplakia, which are

[5] Refer to Episode 19 for additional information on e-cigarettes and vaping.
[6] Refer to Episode 58 for additional information on cannabis and oral health.

Risk factor	Description
	potentially precancerous conditions. Cannabis may be considered a potential risk factor for oral cancer. However, its role is unclear since several studies have shown an association, while other studies have not. Also, the concurrent use of alcohol, tobacco, and possibly other social drugs makes it difficult to ascertain if cannabis alone is a risk factor for oral cancer. More research is required to reach a firm conclusion. [8] [28] [29]
Alcohol	Alcohol is one of the main risk factors for oral cancer. The amount of alcohol consumed and the length of time it is used increases oral cancer risk. Approximately 1.3 billion individuals over age 15 years worldwide consume alcohol at harmful levels, with the highest rates found across Europe, the Americas, and the Western Pacific region. [4] [24] [30]
Age[7]	Oral cancer can occur at any age, but individuals over the age of 45 are at higher risk. Individuals over 60 years have the highest incidence of oral cancer. [24]
HPV[8]	Infection with high-risk HPV is a well-recognized cause of about 4.5% of all cancers worldwide. HPV types 16 and 18 are now the most common cause of oropharyngeal cancers (base of the tongue, tonsils, back of throat). It is estimated 72% of oropharyngeal cancer is linked to high-risk HPV infection. Increases in cases have been reported in many countries where previously low incidence rates were seen, particularly among young male white adults. [4] [31]
Gender	Males are more susceptible than females to developing oral cancer. In the past, the ratio of incidence of oral cancer was 6 to 1 for males compared to females. However, this ratio is narrowing and is now closer to 2 to 1. [24]
Diet	Fruits and vegetables have a protective factor and are believed to reduce the risk of oral cancers. Substances like carotenoids, which are common in vegetables and fruit, are linked with a lower risk of oral cancer. [24] [30]
UV exposure	UV light exposure (e.g., exposure to sun or artificial UV rays like tanning beds) is a major risk factor of cancer on the lips. This is especially true for individuals who are in the sun for long time periods (e.g., farmers). Fair-skinned individuals also have a greater risk of developing lip cancer. Most lip cancers occur on the exposed vermillion of the lower lip due to higher level of sun exposure than the upper lip. [12] [24] [30] [32]

[7] Refer to Episode 55 for information on seniors and oral health.
[8] Refer to Episodes 7, 53, and 61 for additional information on HPV.

Risk factor	Description
Immunosuppression	Studies have reported individuals who are immunosuppressed (e.g., due to medications, bone-marrow transplants, disease) have an increased risk of oral cancer and premalignant lesions. [25]
Previous cancer	Individuals who have had oral cancer have a higher risk of developing another oral cancer, especially if they continue to use tobacco or alcohol. Having cancer of the esophagus, larynx, lung, or cervix also increases oral cancer risk. [30]
Family history of squamous cell carcinoma	There is a higher risk of developing squamous cell carcinoma in the head and neck region (including the oral cavity) if a first-degree family member (parent, sibling, or child) has been diagnosed with squamous cell carcinoma of the head and neck. [30]
Genetic syndromes	Individuals with syndromes caused by inherited mutations in certain genes have a very high risk of oral and throat cancer. **Fanconi anemia** is a rare genetic condition that affects the bone marrow. It is associated with a progressive deficiency of all bone marrow production of erythrocytes (red blood cells), leukocytes (white blood cells), and platelets. This can lead to anemia, leukopenia, neutropenia, and thrombocytopenia, often at an early age. **Dyskeratosis congenita** is a genetic syndrome that can cause aplastic anemia, skin rashes, and abnormal fingernails and toenails. Individuals with this syndrome have a very high risk of developing head and neck cancers, especially of the oral cavity and throat, at a young age. [5] [33] [34]
Poor oral health	Studies show individuals with poor oral health have an increased risk of developing oral cancer. [24] [35] An association between periodontal disease and oral squamous cell carcinoma has been confirmed in many studies. A case-control study by Shin et al. (2019) found individuals with periodontitis were 3.7 times more likely to have oral squamous cell carcinoma than those without periodontitis. A meta-analysis by Ye et al. (2016) indicated periodontal disease can increase oral cancer risk by nearly 2-fold. [36] [37] [38]

Risk factor	Description
	Research by Dr. Glogauer and colleagues investigated the association between periodontitis and oral squamous cell carcinoma. The study explored the possible role played by the inflammatory environment created by periodontal disease that favours oral cancer initiation, metastasis, and progression.[9] [39]
Oral potentially malignant disorders	Oral potentially malignant disorders (OPMDs) refer to lesions or conditions with an increased risk of developing cancers of the lip and the oral cavity. Many oral squamous cell carcinomas develop from potentially malignant disorders, including leukoplakia, erythroplakia, proliferative verrucous leukoplakia, oral lichen planus, oral submucous fibrosis, palatal lesions in reverse smokers, lupus erythematosus, epidermolysis bullosa, and dyskeratosis congenita. Individuals diagnosed with OPMDs may have an increased risk to develop cancer anywhere in the oral cavity during their lifetime, but only a minority of OPMDs progress to cancer. However, OPMDs provide an area of abnormality in which cancer development is more likely than in clinically normal mucosa, and more likely than in individuals without such disorders. [10]

[9] Refer to Episode 9 for discussion on Dr. Glogauer's research on the association between periodontitis and oral squamous cell carcinoma.

Characteristics of oral potentially malignant disorders (OPMDs)[10] [10] [40] [41] [42] [43] [44] [45]

Disorder	Symptoms and clinical presentation	Typical location(s)	Differential diagnosis	Risk of malignancy
Leukoplakia	**Homogeneous leukoplakia:** White patch or plaque that cannot be rubbed off Generally asymptomatic **Nonhomogeneous leukoplakia:** <u>Nodular leukoplakia</u>: Small polypoid or rounded outgrowths, red or white excrescences Generally asymptomatic <u>Verrucous leukoplakia</u>: Surface is raised, exophytic, wrinkled, or corrugated Generally asymptomatic <u>Erythroleukoplakia</u>: Mixed, white, and red (speckled) but retaining predominantly white character. Margins may be irregular May be uncomfortable	Buccal mucosa, gingiva, floor of mouth	Frictional keratoses Chemical injury Lip or cheek biting White sponge nevus Chronic candidal infection Leukoedema Fordyce spots/granules Skin graft Oral hairy leukoplakia Nicotinic stomatitis (smoker's palate) HPV lesions (e.g., oral condyloma/wart) Erythema migrans (geographic tongue or benign migratory glossitis) Erosive lichen planus or lichenoid lesions	15.6–39.2% Higher risk of transformation with nonhomogeneous leukoplakia
Erythroplakia	A localized red patch with well-defined margins and a matt surface. Discomfort, tingling, sensitivity to touch, hot beverages, and spicy foods May be asymptomatic	Mouth floor, tongue, retromolar pad, soft palate, buccal mucosa	Erythematous candidiasis Denture-associated stomatitis Erythema migrans Erosive and inflammatory/infective disorders Desquamative gingivitis Discoid lupus erythematosus	51%

[10] These disorders are classified OPMDs based predominantly on their clinical features, following discussions by an expert group at a workshop held by the World Health Organization (WHO) Collaborating Centre for Oral Cancer. [10]

Disorder	Symptoms and clinical presentation	Typical location(s)	Differential diagnosis	Risk of malignancy
			Erosive lichen planus Pemphigoid Pemphigus vulgaris Vascular hamartomas Vascular neoplasms	
Proliferative verrucous leukoplakia	Multiple, thick, white patches in more than two different oral sites. Majority present with a verrucous pattern. Lesions spread and coalesce during development. Recurrence in at least one area, despite treatment. May be uncomfortable	Gingiva, alveolar ridges, palate, buccal mucosa, tongue	Lichen planus (particularly in early stages)	63.3–100%
Oral lichen planus[11]	Erosive/ulcerative variety is sore Reticular: lace-like white lines (Wickham striae) Usually asymptomatic Papular: White dots (papules) Usually asymptomatic Plaque-like: white patch Usually asymptomatic Atrophic: red patches with white striae. Usually sore Erosive (ulcerative): red and ulcerated. Sore Bullous: bullae rupture causing painful ulcers	Typically bilaterally on buccal mucosa, mucobuccal fold, gingiva. Less common on tongue, palate, lips Buccal mucosa Dorsum tongue, buccal mucosa Attached gingiva, buccal mucosa Buccal mucosa, tongue Buccal mucosa, lateral borders of tongue	Oral lichenoid contact hypersensitivity reactions Oral lichenoid drug reactions Oral lichenoid lesions Lichenoid lesions from betel quid use Mucous membrane pemphigoid Chronic ulcerative stomatitis Chronic graft-versus host disease Lichen sclerosus Oral lupus erythematosus Oral proliferative multifocal leukoplakia	~1%

[11] Refer to Episode 75 for additional information on oral lichen planus.

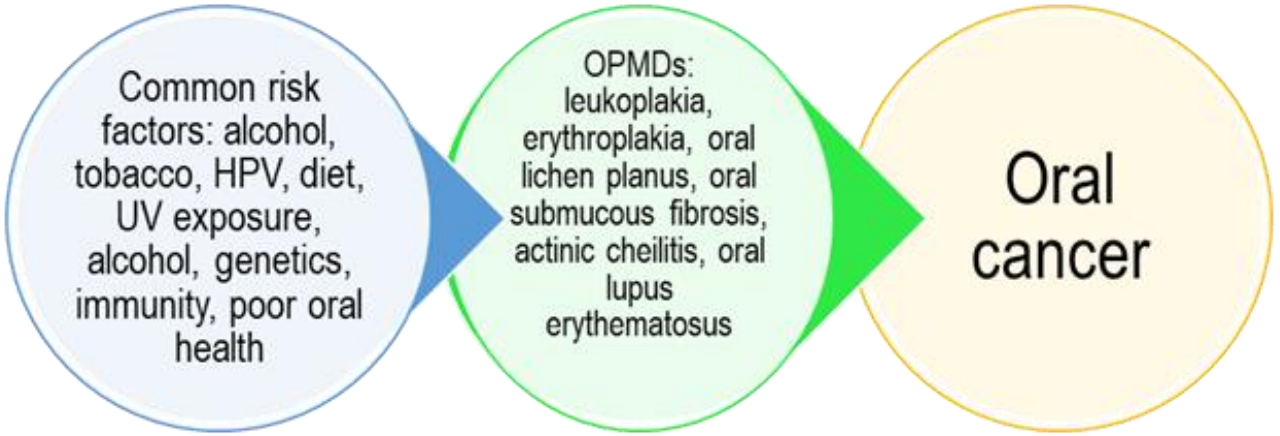
Disorder	Symptoms and clinical presentation	Typical location(s)	Differential diagnosis	Risk of malignancy
Oral submucous fibrosis[12]	Blanching/pallor of oral mucosa Loss of tongue papillae Leathery mucosa Fibrous bands develop in lips, cheek mucosa, and soft palate Limited mouth opening Limited tongue mobility (rigidity) Shrunken or deformed uvula Sunken cheeks Burning sensation, intolerance to spicy food	Buccal mucosa, retromolar area, tongue, soft palate	Scleroderma Plaque-like lichen planus	7–26%
Oral lichenoid lesion	White lines (reticular: lace-like, linear, or annular), papular, sometimes plaque-like Red and erosive with white striae Asymmetrical Asymptomatic Red and atrophic areas may be sore	Oral mucosa	Oral lichen planus	2.1%
Oral graft vs host disease	White lines (reticular: lace-like, linear, or annular), papular, sometimes plaque-like Red and erosive with white striae Asymmetrical Red and atrophic areas could be sore History of allogenic hematopoietic cell transplant (stem cell or bone marrow transplant)	Buccal mucosa, tongue, lips, buccal and labial vestibule	Oral lichen planus Oral lichenoid contact reaction Oral lichenoid drug reaction	NA
Actinic cheilitis (cheilosis)	Localized or diffuse lesions of white flaking plaques or scaly lesions with interspersed red areas. Usually painless Mild cases may simply present with dry lips	Vermillion of lower lip from chronic exposure to UV radiation	Frictional keratosis Smoking-related leukoplakia Plaque-like lichen planus	6–10%
Oral lupus erythematosus	Oral lesions of lupus exhibit similar clinical presentations as found in OLP	Buccal mucosa, palate, lips	Oral lichen planus	NA

[12] Refer to Episode 16 for additional information on oral submucous fibrosis.

Disorder	Symptoms and clinical presentation	Typical location(s)	Differential diagnosis	Risk of malignancy
Dyskeratosis congenita	Consists of a triad of oral leukoplakias (usually on the dorsal tongue but can arise in any mucous membranes within the body), hyperpigmentation of the skin (usually with a reticular pattern on the neck) and nail dystrophy. Lichenoid like lesions have also been reported	Dorsal tongue	Leukoplakia	Often poor prognosis due to either malignant change of oral lesions or bone marrow failure causing severe infection and death
Palatal lesions in reverse smokers[13]	Red, white, or mixed red and white, in a background of tobacco staining due to the burning end of a cigarette or cigar held inside the mouth	Hard palate, tongue	Leukoplakia Erythroplakia Erythroleukoplakia	83.3% dysplasia 12.5% squamous cell carcinoma

*NA: not available

OPMDs and risk factors associated with oral cancer development



[13] Reverse smoking is more common in females with low socioeconomic backgrounds. The habit of reverse smoking is seen in parts of India, the Caribbean Islands, Colombia, Panama, Venezuela, Jamaica, Sardinia, and the Philippines. [53]

Signs and symptoms of oral cancer

Oral cancers may cause one or more of the following signs or symptoms:

- Lip or mouth sore that does not heal within two weeks
- Persistent mouth pain
- Lump or thickening in the lips, cheek, or mouth
- White (leukoplakia), red (erythroplakia), or white/red patch (erythroleukoplakia) on the gingiva, tongue, tonsil, lip, palate, floor of the mouth
- Indurations
- Persistent sore throat or a feeling something is caught in the throat
- Difficult or painful chewing or swallowing
- Swollen, painless tonsil
- Constant cough
- Coughing up blood
- Trouble moving the jaw or tongue
- Numbness of the tongue, lip, or other area of the mouth
- Swelling or pain in the jaw or inability to open mouth fully
- Dentures that start to fit poorly or become uncomfortable
- Loosening of the teeth or pain around the teeth
- Voice changes, hoarseness, difficulty speaking
- Lump or mass in the neck or back of the throat
- Unexplained weight loss
- Persistent earaches, maybe unilateral
- Enlarged and/or hard lymph nodes

Note: Some individuals may not experience symptoms. [46] [47] [48]

Oral cancer prevention

Studies have demonstrated low public awareness of head and neck cancers, including risk factors (e.g., tobacco use, HPV infection) and common symptoms. [49] [50]

Research by Torabi et al. (2021) assessed public awareness of head and neck cancers and the associated role of HPV. Results indicated head and neck cancer knowledge was relatively low. Concurrently, misinformation was prevalent, suggesting unreliable information sources, such as some social media, may be responsible for a significant proportion of knowledge dissemination. This stresses the importance of healthcare providers to continually assess client knowledge and misconceptions to help individualize client education and dispel misinformation. [51]

Stull et al. (2020) found parents were comfortable having discussions about HPV and the HPV vaccine in oral healthcare settings and most parents reported an expectation that oral healthcare providers would talk to them or their children about HPV and cancer. The authors concluded oral health professionals can play a key role in increased uptake of the HPV vaccine through increased client communication. [52]

Strategies to reduce oral cancer risk include:

- Smoking cessation, including use of all tobacco products.
- Avoid heavy alcohol use. Drink within moderation.[14]
- Use proper sun protection. Apply lip balm with SPF any time outside and wear sunscreen daily to prevent other types of skin cancer. Wear a wide brimmed hat offering shade to the lips and face. Avoid use of sunbeds and tanning salons.
- Reduce risk for HPV infection by practicing safe sex and getting the HPV vaccine.
- Undergo routine oral cancer screenings and perform oral cancer self-examination on a regular basis.
- Consume a balanced and healthy diet. Increased consumption of fruits and vegetables is associated with a lower risk of all cancer. A poor diet leads to nutritional deficiencies, which compromises the immune system.
- Maintain good oral health.
- Maintain a healthy weight and be physically active to help prevent cancer in general. [12] [31]

Take home messages

- Oral cancer screening must be an integral part of an oral health clinician's routine, including a comprehensive review of client medical history and risk factors (e.g., tobacco and alcohol use, duration of use, and amount consumed).
- Oral cancer screenings need to include visual inspection and tissue palpation for all clients, including those without any apparent risk factors. Any suspicious findings are then referred to the appropriate medical or dental provider. Early detection and diagnosis are key to increasing survival rate.
- Early identification and timely treatment of potentially malignant disorders may help prevent malignant transformation in oral lesions. This would include referral to specialists.
- Clinicians should also educate clients on how to perform oral cancer self-examinations and how to reduce risk factors (e.g., smoking cessation, reducing alcohol consumption, sun safety, HPV vaccination, etc.) to decrease the burden of oral cancer.

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