IBOM MEDICAL JOURNAL
Vol.19 No.3 | July - September 2026 | Pages 793-806
www.ibommedicaljournal.org

The definition, types, risk factors, prevention, diagnosis, and treatment of oral cancer: An up-to-date literature review

Kehinde Kazeem Kanmodi[1,2,3,4], Yovanthi Anurangi Jayasinghe[4,5], Ruwan Duminda Jayasinghe[1,6], Emeka Benjamin Okeke[4,7], Misheck Julian Nkhata[1], Lawrence Achilles Nnyanzi[1]

[1]*School of Health and Life Sciences, Teesside University, Middlesbrough, UK*
[2]*Centre for Dental and Craniofacial Research, Cephas Health Research Initiative Inc., Ibadan, Nigeria*
[3]*Department of Public Health, Thomas Adewumi University, Oko, Nigeria*
[4]*Department of Research and Innovation, University of Technology and Entrepreneurship, Phnom Penh, Cambodia*
[5]*Centre for Evidence Synthesis and Implementation Research, Cephas Health Research Initiative Inc., Ibadan, Nigeria*
[6]*Department of Oral Medicine and Periodontology, Faculty of Dental Sciences, University of Peradeniya, Peradeniya, Sri Lanka*
[7]*Centre for Digital Health Research, Innovation and Practice, Cephas Health Research Initiative Inc., Ibadan, Nigeria*

Abstract

Background: The prevention, early detection, and effective management of oral cancer are critical priorities, requiring continual updates of current knowledge. This literature review aimed to provide an up-to-date overview of the definition, types, risk factors, prevention, diagnosis and treatment of oral cancer.
Methods: The evidence synthesised in this literature review were predominantly those reports published within the past five years (2020 to 2025), and they were obtained from PubMed and Google Scholar.
Results: Inconsistencies exist in the definition of oral cancer; however, only those definitions provided by few sources, such as the World Health Organization and the UK's Department of Health and Social Care, comprehensively defined oral cancer as cancer affecting the lips, oral cavity, and oropharynx. Oral cancer is commonly classified based on its anatomical site or cell type. Of all the numerous oral cancer risk factors ever reported in the literature, only four of them—tobacco, alcohol, betel quid (or areca nut), and human papillomavirus infection—are the established risk factors; all other risk factors are probable. Oral cancer prevention strategies are multi-level, and health education remains as the most widely recommended primary prevention strategy of the disease. The five most common clinical features of oral cancer are chronic non-healing ulcers, pathologic tooth mobility, oral lumps, pain, and oral bleeding. Diagnostic techniques such as integrated vital staining—toluidine blue and Lugol's iodine—support early detection with 92.5% sensitivity and 63.2% specificity, while tissue biopsy remains the gold standard. Oral cancer treatment is expensive, and its primary modalities are surgery, radiotherapy, and chemotherapy, with increasing use of targeted therapies and immunotherapy for advanced stages. Evidence consistently links early detection with improved prognosis, survival, and quality of life.
Conclusion: Oral cancer remains a largely preventable disease, with early diagnosis and timely intervention significantly improving patient outcomes.

Corresponding Author:

Kehinde Kazeem Kanmodi
School of Health and Life Sciences, Teesside University, Middlesbrough, UK.

k.kanmodi@tees.ac.uk

DOI: 10.61386/imj.v19i3.1211

Keywords: oral cancer, risk factors, prevention, diagnosis, treatment, review

1. Background

1.1. Introduction: What is Cancer?

Cancer is a term which has diverse meanings, and its specific meaning depends on its context of use. In the medical context, cancer is a disease of the human body which has been defined by multiple sources. According to the National Cancer Institute (NCI)[1], cancer is a "disease in which some of the body's cells grow uncontrollably and spread to other parts of the body". According to the United Kingdom's National Health Service (NHS)[2], cancer is a "condition where cells in a specific part of the body grow and reproduce uncontrollably. The cancerous cells can invade and destroy surrounding healthy tissue, including organs". According to the Australian Institute of Health and Welfare[3], cancer is a "large range of diseases in which some of the body's cells become defective, begin to multiply out of control, can invade and damage the area around them, and can also spread to other parts of the body to cause further damage". Based on the above-quoted definitions of cancer from multiple leading health organisations, it can be summarised, from a medical point of view, that cancer is a malignant disease that occurs from abnormal cells which grows uncontrollably and invade nearby or distant body tissues.

There are different types of cancers, and they can be broadly classified based on the tissues or organs (i.e. anatomical sites) where they occur or based on the type of body cells that formed them (Figure 1)[1,4,5]. When classified based on the type of body cells that formed them, it could be a carcinoma, sarcoma, leukaemia, lymphoma, melanoma, and multiple myeloma[1,4,5]. However, when it is classified based on the tissues or organs where they occur, it takes the name of the affected tissues or organs, and examples of such cancer include oral cancer, liver cancer, prostate cancer, brain cancer, and eye cancer, amongst others[1].

Carcinomas are those cancer types that occur from the epithelial cells of the human body; some examples of carcinomas include squamous cell carcinoma, basal cell carcinoma, adenocarcinoma, and transitional cell carcinoma[6,7]. Sarcomas are those cancer types which occurs from connective tissue cells of the human body; some examples of sarcomas include Kaposi sarcoma, liposarcoma, and leiomyosarcoma[8–10]. Leukaemia are those cancer types which occur from the blood-forming cells in the bone marrow; some examples of leukaemia include acute lymphoblastic leukaemia, acute myeloid leukaemia, and hairy cell leukaemia[1,11–13]. Melanomas are those cancer types which occurs from the cells that produces melanin; these cells are called melanocytes, and they can be found in pigment body tissues such as the skin and eyes[1]. Some examples of melanoma include ocular melanoma and cutaneous melanoma[14,15]. Lymphomas are those cancer types which occurs from the lymphocytes—the lymphocytes are the B cells and T cells, and they constitute the disease-fighting blood cells in the immune system of the human body[1,16]. The two main examples of lymphomas include the Hodgkin lymphoma and the non-Hodgkin lymphoma[16,17]. Multiple myelomas, also called Kahler disease, are those cancer types which occurs from the body immune cells called plasma cells; some examples of these cells include IgA melanoma, IgD melanoma, and IgE melanoma[1,18].

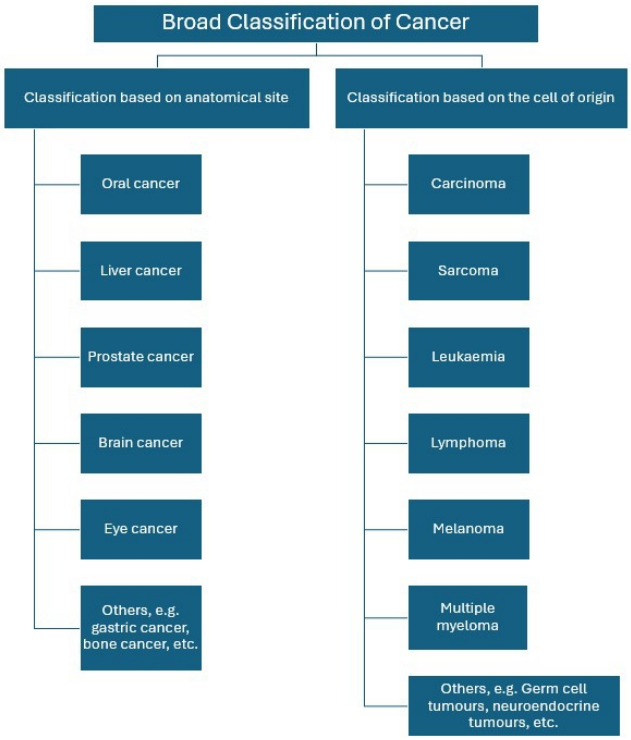

Figure 1. Schematic diagram showing the broad classifications of cancer

For those cancer types that are named based on anatomical sites, such as oral cancer, liver cancer, prostate cancer, brain cancer, and eye cancer, any of the cells within those sites can abnormally and uncontrollably proliferate to form cancer of those sites. For example, a prostate cancer can be an adenocarcinoma (which occurs from the gland cells of the prostate), a small cell carcinoma (which occurs from the flat cells of the prostate), a transitional cell carcinoma (which occurs from the cells of the urethra), or a sarcoma (which occurs from the connective tissue cells of the prostate)[1,19–22]. Overall, this indicates that any cell within an anatomical site has the potential to become cancerous.

1.2. Aim & Objectives

Cancer remains a continually evolving field in terms of its definition, classification, risk factors, diagnostic methods, and treatment, driven by ongoing advances in medicine, science, and technology. Oral cancer, likewise, is a dynamic area of research and ranks among the top 20 cancers worldwide[23]. Given its significant global impact, there is a need to consolidate and present up-to-date information on its definition, types, risk factors, diagnosis, and treatment. This literature review seeks to provide a comprehensive and current synthesis of knowledge on oral cancer, encompassing its definition, types, and associated risk factors. Additionally, it aims to identify both established and emerging determinants of the disease, review evidence-based preventive strategies at individual and population levels, and outline contemporary diagnostic approaches, including conventional and adjunctive methods. Furthermore, the review summarises available treatment modalities, highlights recent advances in clinical management, and identifies gaps in the existing literature with implications for future research and public health practice.

2. Methods/Literature Source

This literature review involved a comprehensive search of peer-reviewed articles indexed in PubMed and Google Scholar. These two databases were searched to retrieve recent and relevant literature on the definitions, types, risk factors, prevention, diagnosis, and treatment of oral cancer, using diverse set of relevant keywords. The literature obtained from the searches were used to draft this literature review.

3. Definition and Types of Oral Cancer

Oral cancer is a cancer type whose anatomical sites have been inconsistently described for several decades by clinicians and researchers; hence, its definition has varied widely in academic literature[24–26]. Among some authors, oral cancer refers to the cancer type affecting only the oral cavity[26–28]; to some, oral cancer refers to cancer type affecting only the oral cavity and/or the lip[26,29]; and to others, it refers to cancer type affecting the oropharynx, oral cavity, and/or lip[26,30]. Based on the observed heterogeneities in the definition or oral cancer in the academic literature, a universally accepted definition of oral cancer is still globally lacking, making it very confusing to describe[25].

However, in this review, the case definition of oral cancer is based on the definitions provided by two leading health organizations and authorities in the world—the World Health Organization (WHO) and the United Kingdom's Department of Health and Social Care (DHSC). These two organisations have provided definitions for oral cancer, and their definitions are consistent with the description of the anatomical site of oral cancer. In their definitions, oral cancer was referred to as a cancer type which affects the oropharynx, oral cavity, and/or lip[31,32]. Notably, their definitions were chosen based on the high reputation of these two organisations and to ensure a robust and accurate representation of the disease in this review, and henceforth, the subsequent mention of oral cancer in this review will be in reference to any cancer affecting the oropharynx, oral cavity, and/or lip of the human body.

There are different types of oral cancer, and its classification is based on the anatomical site where it occurs or based on the type of body cells that formed them, just as it was afore described for cancers in general (Figure 1). For its classification based on anatomical site, oral cancer types include cancer of the lip, cancer of the tongue, cancer of the salivary gland, cancer of the jaw, and cancer of the oropharynx[24–26,31,32]. On the other hand, if classified based on the type of body cells that formed them, oral cancer includes Kaposi sarcoma,

osteosarcoma, and adenocarcinoma[33–35].

4. Overview of the Risk/Aetiological Factors of Oral Cancer

The risk of developing oral cancer is multifactorial, and several risk or aetiological factors have been implicated or established for its pathogenesis[36–39]. These factors can be broadly classified into six factors, which are lifestyle (behavioural) factors, biological factors, microbial factors, mechanical factors, occupational and environmental factors, and demographic factors. Table 1 provides a summary of these risk/aetiological factors with examples.

Table 1. A summary of the risk/aetiological factors of oral cancer, with examples

No.	Category of Risk Factors	Examples of Risk Factors
1	Lifestyle (behavioural)	-Tobacco use (smoking or smokeless use) -Alcohol consumption -Betel quid/areca nut use -Poor oral hygiene
2	Biological	-Genetic mutations -Immunosuppression (e.g., organ transplant, acquired immunodeficiency syndrome (AIDS)) -Micronutrient deficiency (Iron deficiency anaemia, etc.) -Previous history of oral/head and neck cancer/pre-cancer
3	Microbial	-Viral pathogens (Epstein-Barr virus, Human papillomavirus (HPV) (especially type 16), human immunodeficiency virus (HIV)) -Non-viral pathogens (*Staphylococcus aureus*, *Prevotella melaninogenica, Exiguobacterium oxidotolerans, and Veillonella parvula*)
4	Mechanical	-Ill-fitting dentures -Sharp or rough dental restorations (e.g. dental crowns, dental amalgam fillings) -Sharp teeth -Chronic tongue biting -Chronic cheek biting
5	Occupational and environmental	-Exposure to carcinogenic industrial substances (asbestos, benzene, diethyl ether, tetrahydrofuran) -Exposure to ultraviolet radiation
6	Demographic	-Older age (usually >40 years of age) -Male sex -South Asian ethnicity -Socioeconomic status

4.1. Lifestyle (Behavioural) Factors

Lifestyle, or behavioural, factors refer to the category of factors related to the day-to-day activities of an individual[40]. The lifestyles of people are influenced by the religious, geographical, economic, political, and economic situations around them[40]. According to the WHO, lifestyle factors contribute about 60% of the causes of health-related and quality of life-related conditions affecting human beings[40,41]. Unhealthy lifestyles such as alcohol consumption, betel quid/areca nut use, low intake of fruits/vegetables, poor oral hygiene, and tobacco use are lifestyle or behavioural risk/aetiological factors of oral cancer[39,42–47].

Notably, most of these lifestyle risk factors—alcohol consumption, betel quid/areca nut use, and tobacco use—are, according to the International Agency for Research on Cancer (IARC), established risk factors of oral cancer while low intake of fruits/vegetables and poor oral hygiene are considered as potential risk factors[48]. These established risk factors are those factors that have been scientifically confirmed as oral cancer risk factors while the potential risk factors are those factors whose aetiopathogenesis concerning oral cancer development remains unclear[48].

4.2. Biological Factors

Biological factors are those intrinsic conditions within the human body that determines health conditions[49]. These factors are innate and are created by the interactions within the internal milieu of the human body[49,50]. Common examples of those biological factors that can cause or increase the risk of developing oral cancer include genetic mutations, immunosuppression (e.g., AIDS, organ transplant), micronutrient deficiency (e.g. iron deficiency anaemia), and previous history of oral (or head and neck) cancer (or pre-cancer)[39,51–57]. Notably, all the above-listed biological factors are not considered as established risk factors of oral cancer by the IARC but rather as potential risk factors[48].

4.3. Microbial Factors

The microbial factors are those microorganisms which affects the health conditions of the human body[58]. Microorganisms play a key role in the aetiology and pathogenesis of diseases, and these those ones that cause a disease conditions are called pathogens[58]. The popular pathogens known to cause or increase the risk of developing oral cancer can be classified into viral pathogens and non-viral pathogens[59–62]. Examples of those viral pathogens include Epstein-Barr virus, HPV (especially type 16), and HIV[39,61,62]. On the other hand, the non-viral pathogens include some bacteria such as Staphylococcus aureus, Prevotella melaninogenica, Exiguobacterium oxidotolerans, and Veillonella parvula[59]. These pathogens cause or increase the risk of developing oral cancer only when they infect the oral tissues, and not when they are ex-situ[59–61].

Notably, all these microbial risk factors, except HPV infection, were not considered as established risk factors of oral cancer by the IARC[48]. Only HPV infection was considered as an established microbial risk factor because it has been confirmed through rigorous scientific research[48].

4.4. Mechanical Factors

Mechanical factors are those conditions that harms the human body through physical trauma or irritation[63–65]. Chronic oral irritation or trauma, which occurs from persistent and repeated injury by intra-oral agents have been documented in the literature as factors that could trigger the development of oral cancer in humans[63–65]. Common examples of this chronic mechanical oral irritation or trauma includes those that occur from Ill-fitting dentures, sharp or rough dental restorations (e.g. dental crowns, dental amalgam fillings), sharp teeth, chronic tongue biting, and chronic cheek biting[63–65]. Notably, all the above-listed mechanical factors are not considered as established risk factors of oral cancer by the IARC but rather as potential risk factors[48].

4.5. Occupational and Environmental Factors

Occupational and environmental factors constitute one of the risk/aetiological factors of oral cancer. The occupational factors are those workplace-related conditions or exposures that causes or increases the risk of developing oral cancer while environmental factors are those conditions or exposures in an individual's surroundings that causes or increases the risk of developing oral cancer[66–68]. Notable examples of these occupational factors include exposures to carcinogenic industrial substances such as asbestos, benzene, diethyl ether, and tetrahydrofuran[39,68–71]. For environmental factors, a notable example is exposure to ultraviolet radiation[66]. Notably, all the above-listed occupational and environmental factors are not considered as established risk factors of oral cancer by the IARC but rather as potential risk factors[48].

4.6. Demographic Factors

Demographic factors, from a disease standpoint, refer to those personal characteristics that are associated with the risks of having a disease[72]. Common examples of demographic factors include sex, age, ethnicity/race, and socio-economic status[72–74]. Compelling evidence from rigorous research, including cohort studies and evidence synthesis research, has shown that relationships between human demographic factors and the risks of developing oral cancer[75]. Based on existing evidence, male sex, older age (usually >40 years of age), South Asian ethnicity, and low socioeconomic status are major demographic risk/aetiological factors of oral cancer[75–78]. Notably, all the above-listed demographic factors are not considered as established risk factors of oral cancer by the IARC but rather as potential risk factors[48].

4.7. The Four Major Risk/Aetiological Factors of Oral Cancer

Of all the numerous oral cancer risk/aetiological factors identified above, four of them have been widely reported in the literature, including sources from the WHO[32], the Centres for Disease Control and Prevention (CDC)[79], and the IARC Working Group on the Evaluation of Cancer-Preventive Interventions[48], as the most important factors because oral cancer causality has been established in relation to exposure to these four factors. These factors include tobacco use, alcohol use, betel quid use, and oral infection with HPV[32,48,79]. Pertinently, all these factors are preventable factors, and this shows that oral cancer is a disease that is largely preventable[39,47,55,62,80,81].

5. Overview of the Clinical Features of Oral Cancer

The clinical features of oral cancer are the obvious symptoms that a patient with oral cancer complains about, or the signs a clinician detects during clinical examination[82]. Oral cancer clinical features are diverse; however, for the ease of explanation, these features are categorised into common features and less common features[82]. The common features include chronic non-healing oral ulcer, pathologic tooth mobility, oral lump, oral pain, and oral bleeding while the less common features include enlargement of cervical lymph nodes and numbness (or paraesthesia) of the chin[39,82]. Oral cancer clinical features have been associated with serious discomforts and poor quality of life among patients afflicted with the disease, making oral cancer a severely debilitating disease[83]. Being on overview,

only the common features are discussed in this section.

5.1. Chronic non-healing Oral Ulcer

Chronic non-healing ulcer refers to a wound characterised by a persistent break in the mucosal/epithelial lining which does not heal once the cause is removed within a period of two or three weeks[84]. The presence of a solitary chronic non-healing ulcer is a common symptom of oral cancer; such ulcers typically have a raised floor, an irregular border and floor, and hard when palpated[82]. Such ulcers are often white, red, or whitish red in colour and they are typically associated with oral squamous cell carcinoma—an oral cancer type which constitute about 95% of all oral cancers[84,85]. The common sites where these chronic oral ulcers are located include the tongue, alveolar ridges, buccal mucosa, and the floor of the mouth[84].

5.2. Pathologic Tooth Mobility

Pathologic tooth mobility refers to the non-physiological axial and horizontal movement of tooth in response to normal intraoral forces such as occlusal forces; pathologic tooth mobility occurs when a portion or all its periodontium is lost[86,87]. The common causes of a pathologic tooth mobility are periodontal diseases and trauma[88,89]. The presence of pathologic tooth mobility, especially when periodontal diseases and trauma are absent, is suggestive of oral cancer[82,88,89]. Pathologic tooth mobility due to oral cancer occurs when the alveolar bone and other periodontal tissues of the teeth are destroyed through invasion by cancer cells, and notably, this mobility becomes obvious when oral cancer is at its advanced stage[82,88,89].

5.3. Oral Lump

Oral lump, also called oral swelling, refers to an elevated mass or lesion that can be seen or felt in the oral cavity[82,90]. Oral lump is often seen when oral cancer has reached its advanced stage, and it is usually solitary and characterised by progressive growth, poorly defined borders, hardness in its consistency, and warty surfaces[82]. The commonly affected sites of oral lump include the floor of the mouth, tongue, and buccal area[91].

5.4. Pain

Pain accounts for 30-40% of the symptoms of oral cancer[82]. Patient with oral cancer starts feeling pain when the cancer has grown to a remarkable size (usually at the late stage), and, unfortunately, this is the period when patients afflicted with oral cancer starts seeking medical assistance[82,92]. The early stage of oral cancer is usually free of pain, so patients rarely notice the presence of the lesion[82,92]. The severity of the pain associated with oral cancer ranges from mild discomfort to severe pain, and the tongue is the site where the pain is felt[93]. Notably, the pain could also be felt in other sites of the oral structures and even as far as the ears[82,93].

5.5. Oral Bleeding

Oral bleeding is also another common feature of oral and it refers to the loss of blood from the oral cavity. Such bleeding might be on contact (e.g. during eating or brushing) or spontaneous, and the bleeding sites are usually the oral lump(s) and the non-healing ulcer(s) associated with the oral cancer lesion[82,94–96].

6. Overview of the Prevention of Oral Cancer

Fortunately, oral cancer is a preventable disease[29,97]. Recently, several prevention strategies have been recommended by clinicians, scientists, and health organisations concerning the prevention of oral cancer[47,48]. These prevention strategies can be categorised into primary and secondary prevention[47,48] (Figure 2).

The primary prevention strategies are those strategies that focused on the prevention of the disease from occurring in the first instance, and they include tobacco cessation, discontinuation of alcohol use, betel quid cessation, vaccination against human papillomavirus infection, the use of protective oral sex barriers (such as dental dams and tongue condoms), and health education on oral cancer[47,48,80] (Figure 2).

On the other hand, the secondary prevention strategies focus on the early diagnosis and treatment of oral cancer/pre-cancer[47,48]. These strategies include mouth self-examination, oral cancer screening, regulation of healthcare, and proper organisation of healthcare services[47,48] (Figure 2).

As earlier mentioned, the major risk/aetiological factors of oral cancer—tobacco use, alcohol use,

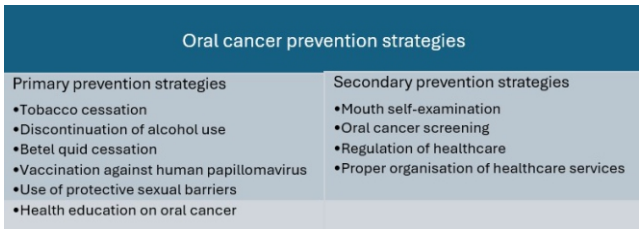

Figure 2. A chart showing the classification and examples of oral cancer prevention strategies

betel quid use, and oral infection with HPV—can be modified through healthy choices. Arguably, primary prevention strategies are the most important because they are focused on the prevention of the occurrence of oral cancer in the first instance[47]. However, among these primary prevention strategies, health education on oral cancer is arguably the most important one; this is because such health education seeks to improve public awareness and knowledge on oral cancer, and this will invariably boost the health (or oral cancer) literacy of the receivers of such health education interventions[98–101]. Also, through health education on oral cancer, the public can be made aware of the meaning of oral cancer, its risk/aetiological factors, how it could be prevented, and how it is treated if it occurs. Hence, this makes health education on oral cancer a primary prevention strategy that cuts across board.

7. Overview of the Diagnosis of Oral Cancer

The early stage of oral cancer is usually asymptomatic while its advanced stage has obvious symptoms, of which severe pain is the most reported symptom[82,102]; hence, the importance of early and accurate diagnosis of oral cancer cannot be overemphasised. Overall, the accurate diagnosis of oral cancer is largely dependent on having a detailed history of patient's symptoms, a comprehensive patient examination, and a robust diagnostic technique[47,102]. There are several diagnostic techniques that have been proposed in the literature for the diagnosis of oral cancer; however, only those two techniques—vital staining and tissue biopsy—which are commonly used in oral cancer diagnosis are critically discussed below[102–105].

7.1. Vital Staining

The vital staining technique involves the use of dyes to stain suspected oral cancer site to detect the presence of oral cancer due to a differential colour change between healthy tissues and cancerous tissues[102]. The two dyes that are commonly used for vital staining are toluidine blue and Lugol's iodine[102]. Of these two dyes, toluidine blue is the most used dye for oral cancer diagnosis, especially in community-based oral cancer screening programmes, because it is cheap and highly sensitive (92.5%) and specific (63.2%) in detecting oral cancer tissues[106]. Toluidine blue works by binding with the acidic components (e.g. nucleic acid) in human tissues[102]. Since cancerous cells containing higher nucleic acid content compared to apparently healthy cells; hence, oral cancerous tissues retain the blue colour of toluidine blue when it is used as a mouth rinse[102].

On the other hand, Lugol's iodine stains healthy oral tissues by infiltrating and reacting with the glycogen within them[102,107]. Since cancerous cells have depleted glycogen, Lugol's iodine detects oral cancer by staining normal squamous epithelial tissue only, making the cancerous oral tissues with no or light stain[102,107]. When sprayed or applied as a swab in the mouth, Lugol's iodine's actions produce a blackish brown stain on the margins of oral cancer sites while leaving the cancer site lightly stained or unstained[102,107]. Notably, Lugol's iodine has a lower sensitivity (87,5%) and a higher specificity (84.2%) when compared to toluidine blue[102].

Due to the limitations of both dyes, the combined use of both dyes—toluidine blue and Lugol's iodine—has been found to have better effect in sensitively (85.0%) and specifically (89.5%) detecting oral cancer tissues[102]. Notably, regardless of the increased sensitivity and specificity of vital staining technique, this technique is not considered as the gold standard for oral cancer diagnosis but rather a diagnostic aid and a rapid and cost-effective technique for oral cancer detection in community-based screening programmes[102–104].

7.2. Tissue Biopsy

Tissue biopsy refers to a histopathological examination of tissue removed from a living body to discover the presence, cause, or extent of a disease[103,104,108]. There are different types of tissue

biopsy techniques, and they are named after the manner which the tissues to be examined were taken from the human body[103,104,108]. Common examples of these techniques include punch biopsy, surgical biopsy, brush biopsy, and fine needle aspiration biopsy[104,108]. Of these techniques, the two most common and most reliable ones used for oral cancer diagnosis are punch biopsy and surgical biopsy[103,104,108]. Notably, these two techniques unlike vital staining techniques involve painful procedures; hence, they are done for patients under local anaesthesia[109].

The punch biopsy technique involves the use of a punch biopsy forceps to remove a cylindrical specimen of oral tissue of between 0.4–0.8 mm in diameter from a suspected oral cancer site[103,104]. The specimen obtained through this technique is smaller than that obtained through surgical biopsy; hence, it is not as reliable as surgical biopsy technique[104]. Regardless of this limitation, the punch biopsy technique is safer than the surgical biopsy technique due to the associated low rate of adverse events (or complications) from the procedure and its less invasive approach compared to the surgical biopsy technique[104,110].

The surgical biopsy technique involves the use of a scalpel to remove a tissue suspected to have oral cancer together with a bit of surrounding normal tissue[104]. Also, this technique is universally considered as the gold standard for oral cancer diagnosis[104,108]. The surgical biopsy technique of two types: incisional and excisional[103]. The incisional biopsy technique involves the removal of a fraction of the oral cancer tissue for histopathological examination while the excisional biopsy technique involves the removal of the entire cancerous tissue, often with some surrounding healthy tissue, for histopathological examination[103]. Additionally, the incisional biopsy technique is done for bigger cancer lesions while excisional biopsy technique is done for smaller cancer lesions[103,111].

8. Overview of the Treatment Modalities and Prognosis of Oral Cancer

The treatment of oral cancer is multimodal, and it often involves the use one or a combination of these modalities: surgery; chemotherapy (including immunotherapy); and radiotherapy[112–114]. Surgery is the primary treatment modality for oral cancer while chemotherapy and radiotherapy are adjuvant[115,116]. Although chemotherapy and radiotherapy are adjuvant treatment modalities for oral cancer; however, in uncommon occasions they can be deployed as the primary treatment modalities for oral cancer[115]. The occasions that warrant their deployment as the primary treatment modalities of oral cancer are: i) when the patient have severe comorbid conditions that make them high risk operative patients; ii) when the disease is at the early stage, so as to avoid the risk of aesthetic and functional defects; iii) when the disease is not resectable; iv) when the disease keeps relapsing after multiple rounds of surgeries; and v) when the patient prefers not to have surgery[112,115].

The surgical treatment of oral cancer is suitable for early-stage oral cancer as well as those in the advanced-stage that can be resected[112,114]. The main objectives of surgery in oral cancer treatment are to completely and safely remove the lesion and to preserve orofacial aesthetics and functions[112,114]. To achieve these objectives, surgical treatments such as tumour resection with/without neck dissection and reconstructive surgeries are done for oral cancer patients[112,114.]

Chemotherapy and/or radiotherapy is/are usually given to oral cancer patients after surgery has been done[112,114,115]. The main objective of these two treatment modalities is to minimise the risk of metastasis or relapse of oral cancer[116]. Notably, substantial evidence, including systematic review evidence, have shown that the use of chemotherapy or radiotherapy alone as adjuvants does not yield any significant treatment effectiveness among oral cancer patients, but rather the use of a combination of both therapies (i.e. radiochemotherapy) are more effective in oral cancer treatment, especially when they are given after surgery[116–118].

It is also noteworthy that the stage of clinical presentation of oral cancer is strongly associated with its prognosis[119]. Late- or advanced-stage presentation of oral cancer often requires the use of multiple treatment options, unlike when the presentation was done at early-stage[90]. Early presentation of oral cancer makes the treatment of the disease less complicated, more cosmetic, and with better prognosis (or clinical outcomes)[90]. However, at late presentation, the prognosis of OC is generally poor, with an average five-year survival

rate below 50%[36,120].

9. Conclusion

Oral cancer is a life-threatening disease, largely driven by preventable risk behaviours and lifestyle factors such as tobacco use, alcohol consumption, and betel quid chewing, along with certain viral infections such as human papillomavirus (HPV). Despite its high mortality, oral cancer can be prevented through behavioural modification, detected early through effective screening, and successfully treated when identified promptly. This literature review provides essential baseline information for the lay population, researchers, and healthcare professionals, helping to identify gaps that can guide the strengthening of public and global health interventions—integrating advanced diagnostic tools, and ensuring equitable access to multidisciplinary care are critical strategies for reducing its global burden.

ACKNOWLEDGEMENT

Nil.

CONFLICT OF INTEREST

Nil.

FUNDING

This study was self-funded.

TRANSPARENCY STATEMENT

All authors have read and approved the final version of the manuscript. The corresponding author and manuscript guarantor, Kehinde Kazeem Kanmodi, affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained. Also, Kehinde Kazeem Kanmodi (the corresponding author and manuscript guarantor) had full access to all of the data in this study and takes complete responsibility for the integrity of the data and the accuracy of the data analysis.

DATA AVAILABILITY STATEMENT

The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials.

ETHICAL CONSIDERATIONS

Not applicable. This study did not collect data from human or animal subjects but an open research repository.

References

1. National Cancer Institute. What is cancer? 2021. Accessed 03 May 2025. https://www.cancer.gov/about-cancer/understanding/what-is-cancer.
2. National Health Service. Cancer. 2022. Accessed 05 May 2024. https://www.nhs.uk/conditions/cancer/
3. Australian Institute of Health and Welfare. Cancer. 2024. Accessed 05 May 2025. https://www.aihw.gov.au/reports/australias-health/cancer.
4. Idikio HA. Human cancer classification: a systems biology-based model integrating morphology, cancer stem cells, proteomics, and genomics. J Cancer. 2011;2:107-15. https://doi.org/10.7150/jca.2.107
5. Serafim V, Shah A, Puiu M, Andreescu N, Coricovac D, Nosyrev A, et al. Classification of cancer cell lines using matrix-assisted laser desorption/ionization time of flight mass spectrometry and statistical analysis. Int J Mol Med. 2017;40(4):1096-104.
6. Kuhn E, Morbini P, Cancellieri A, Damiani S, Cavazza A, Comin CE. Adenocarcinoma classification: patterns and prognosis. Pathologica. 2018;110(1):5-11.
7. McDaniel, B., & Steele, R. B. Basal Cell Carcinoma. In StatPearls. StatPearls Publishing 2024. https://www.ncbi.nlm.nih.gov/books/NBK482439/
8. Lee ATJ, Thway K, Huang PH, Jones RL. Clinical and molecular spectrum of liposarcoma. J Clin Oncol. 2018;36(2):151-9.
9. Cesarman E, Damania B, Krown SE, Martin J, Bower M, Whitby D. Kaposi sarcoma. Nat Rev Dis Primers. 2019;5(1):9.
10. Menon, G., Mangla, A., Yadav, U. Leiomyosarcoma. In StatPearls. StatPearls Publishing. 2024. https://www.ncbi.nlm.nih.gov/books/NBK551667/

11. Khwaja A, Bjorkholm M, Gale RE, Levine RL, Jordan CT, Ehninger G, Bloomfield CD, Estey E, Burnett A, Cornelissen JJ, Scheinberg DA, Bouscary D, Linch DC. Acute myeloid leukaemia. Nat Rev Dis Primers. 2016;2:16010.
12. Malard F, Mohty M. Acute lymphoblastic leukaemia. Lancet. 2020;395(10230):1146-62.
13. Troussard X, Maître E, Paillassa J. Hairy cell leukemia 2024: Update on diagnosis, risk-stratification, and treatment—Annual updates in hematological malignancies. Am J Hematol. 2024;99(4):679-96.
14. Jovanovic P, Mihajlovic M, Djordjevic-Jocic J, Vlajkovic S, Cekic S, Stefanovic V. Ocular melanoma: an overview of the current status. Int J Clin Exp Pathol. 2013;6(7):1230-44.
15. Schadendorf D, van Akkooi ACJ, Berking C, Griewank KG, Gutzmer R, Hauschild A, Stang A, Roesch A, Ugurel S. Melanoma. Lancet. 2018;392(10151):971-84.
16. Sapkota S, Shaikh H. Non-Hodgkin lymphoma 2023. In StatPearls. StatPearls Publishing https://www.ncbi.nlm.nih.gov/books/NBK559328/
17. Shanbhag S, Ambinder RF. Hodgkin lymphoma: a review and update on recent progress. CA Cancer J Clin. 2018;68(2):116-32.
18. Cowan AJ, Green DJ, Kwok M, Lee S, Coffey DG, Holmberg LA, Tuazon S, Gopal AK, Libby EN. Diagnosis and management of multiple myeloma: a review. JAMA. 2022;327(5):464-77.
19. Esrig D, Freeman JA, Elmajian DA, Stein JP, Chen SC, Groshen S, Simoneau A, Skinner EC, Lieskovsky G, Boyd SD, Cote RJ, Skinner DG. Transitional cell carcinoma involving the prostate with a proposed staging classification for stromal invasion. J Urol. 1996;156(3):1071-6.
20. Chen H, Sun Y, Wu C, Magyar CE, Li X, Cheng L, Yao JL, Shen S, Osunkoya AO, Liang C, Huang J. Pathogenesis of prostatic small cell carcinoma involves the inactivation of the P53 pathway. Endocr Relat Cancer. 2012;19(3):321-31.
21. Murray, T. B. J. The Pathogenesis of Prostate Cancer. In S. R. J. Bott (Eds.) et. al., Prostate Cancer. Exon Publications 2021. 29-41.
22. Siech C, de Angelis M, Di Bello F, Rodriguez Peñaranda N, Goyal JA, Tian Z, et al. Adult prostate sarcoma: demographics, treatment patterns, and survival. Ann Surg Oncol. 2024;31(13):8993–9001.
23. Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2024;74(3):229–63.
24. Moore SR, Pierce AM, Wilson DF. 'Oral cancer'—the terminology dilemma. Oral Dis. 2000;6(3):191–3.
25. McCartan B. 'Oral cancer'--the terminology dilemma. Oral Dis 2001;7(1), 66.
26. Tapia JL, Goldberg LJ. The challenges of defining oral cancer: analysis of an ontological approach. Head Neck Pathol. 2011;5(4):376–84.
27. Speight PM, Farthing PM. The pathology of oral cancer. Br Dent J. 2018;225(9):841–7.
28. Ghanem AS, Memon HA, Nagy AC. Evolving trends in oral cancer burden in Europe: a systematic review. Front Oncol. 2024;14:1444326.
29. Sankaranarayanan R, Ramadas K, Amarasinghe H, Subramanian S, Johnson N. Oral cancer: prevention, early detection, and treatment. Cancer: disease control priorities. 3rd ed. Washington, DC: The International Bank for Reconstruction and Development/The World Bank, 2015.85-99.
30. Aminu K, Aladelusi TO, Adisa AO, Ezeagu CN, Salami AA, Nwafor JN, Uwambaye P, Amzat J, Murererehe J, Omoleke SA, Abdulaziz M, Jayasinghe RD, Kanmodi KK. Epidemiology, literacy, risk factors, and clinical status of oral cancer in East Africa: a scoping review. PLoS One. 2025;20(2):e0317217.
31. Department of Health and Social Care. Chapter 6: Oral cancer. 2021. Accessed 05 May 2025. https://www.gov.uk/government/publications/delivering-better-oral-health-an-evidence-based-toolkit-for-prevention/chapter-6-oral-cancer.
32. World Health Organization Oral health. 2025. Accessed 05 May 2025. https://www.who.int/news-room/fact-sheets/detail/oral-health
33. Vadla P, Pathipaka S, Madala J, Guttikonda VR.

Polymorphous adenocarcinoma of the oral cavity: A skeptical case mimicking lobular carcinoma of breast and gastric carcinoma. J Oral Maxillofac Pathol. 2018;22(Suppl 1):S60–S64.
34. Fatahzadeh M, Schwartz RA. Oral Kaposi's sarcoma: a review and update. Int J Dermatol. 2013;52(6):666–672.
35. Ricotta F, Bassi M, Tomasetti N, Campobassi A, Maiolo V, Bertoni F, Bacchini P, Marchetti C, Tarsitano A. Osteosarcoma of the jaws: a literature review. Curr Med Imaging. 2021;17(2):225–235.
36. Kumar M, Nanavati R, Modi TG, Dobariya C. Oral cancer: etiology and risk factors: a review. J Cancer Res Ther. 2016;12(2):458–463.
37. Irani S. New insights into oral cancer—risk factors and prevention: a review of literature. Int J Prev Med. 2020;11:202.
38. Georgaki M, Theofilou VI, Pettas E, Stoufi E, Younis RH, Kolokotronis A, et al. Understanding the complex pathogenesis of oral cancer: a comprehensive review. Oral Surg Oral Med Oral Pathol Oral Radiol. 2021;132(5):566–79.
39. Salami AA, Kanmodi KK, Nnyanzi LA. Re-emphasizing the roles of general medical and dental practitioners regarding oral cancer eradication in Nigeria. Acta Med Martiniana. 2021;21(3):90–102.
40. Farhud DD. Impact of Lifestyle on Health. Iran J Public Health. 2015;44(11):1442–1444.
41. The WHO cross-national study of health behavior in school-aged children from 35 countries: findings from 2001-2002. J Sch Health. 2004;74(6):204–206. https://doi.org/10.1111/j.1746-1561.2004.tb07933.x
42. Ogden G. R. Alcohol and oral cancer. Alcohol (Fayetteville, N.Y.), 2005;35(3):169–173.
43. Guha N, Warnakulasuriya S, Vlaanderen J, Straif K. Betel quid chewing and the risk of oral and oropharyngeal cancers: a meta-analysis with implications for cancer control. Int J Cancer. 2014;135(6):1433–43.
44. Mathur R, Singhavi HR, Malik A, Nair S, Chaturvedi P. Role of poor oral hygiene in causation of oral cancer—a review of literature. Indian J Surg Oncol. 2019;10(1):184–95.
45. Jiang X, Wu J, Wang J, Huang R. Tobacco and oral squamous cell carcinoma: A review of carcinogenic pathways. Tob Induc Dis. 2019;17:29.
46. Mello FW, Melo G, Pasetto JJ, Silva CAB, Warnakulasuriya S, Rivero ERC. The synergistic effect of tobacco and alcohol consumption on oral squamous cell carcinoma: a systematic review and meta-analysis. Clin Oral Investig. 2019;23(7):2849–59.
47. Coletta RD, Yeudall WA, Salo T. Current trends on prevalence, risk factors and prevention of oral cancer. Front Oral Health. 2024;5:1505833.
48. IARC Working Group on the Evaluation of Cancer-Preventive Interventions. Oral Cancer Prevention. International Agency for Research on Cancer. 2023.
49. Remes O, Mendes JF, Templeton P. Biological, Psychological, and Social Determinants of Depression: A Review of Recent Literature. Brain Sci. 2021;11(12):1633.
50. Farhud D, Malmir M, Khanahmadi M. Happiness & health: the biological factors – systematic review article. Iranian J Public Health. 2014;43(11):1468–77.
51. Richie JP, Kleinman W, Marina P, Abraham P, Wynder EL, Muscat JE. Blood iron, glutathione, and micronutrient levels and the risk of oral cancer. Nutr Cancer. 2008;60(4):474–482.
52. da Silva SD, Hier M, Mlynarek A, Kowalski LP, Alaoui-Jamali MA. Recurrent oral cancer: current and emerging therapeutic approaches. Front Pharmacol. 2012;3:149.
53. Karunakaran K, Muniyan, R. Genetic alterations and clinical dimensions of oral cancer: a review. Mol Biol Rep. 2020;47(11):9135–9148.
54. Rodríguez-Molinero J, Migueláñez-Medrán BDC, Puente-Gutiérrez C, Delgado-Somolinos E, Martín Carreras-Presas C, Fernández-Farhall J, López-Sánchez AF. Association between Oral Cancer and Diet: An Update. Nutrients. 2021;13(4):1299.
55. Warnakulasuriya S, Chen TH. Areca nut and oral cancer: evidence from studies conducted in humans. J Dent Res. 2022;101(10):1139-46.
56. Patini R, Cordaro M, Marchesini D, Scilla F, Gioco G, Rupe C, D'Agostino MA, Lajolo C. Is Systemic Immunosuppression a Risk Factor for

Oral Cancer? A Systematic Review and Meta-Analysis. Cancers. 2023;15(12):3077.
57. Patini R, Favetti Giaquinto E, Gioco G, Castagnola R, Perrotti V, Rupe C, Di Gennaro L, Nocca G, Lajolo C. Malnutrition as a Risk Factor in the Development of Oral Cancer: A Systematic Literature Review and Meta-Analyses. Nutrients. 2024;16(3):360.
58. Velmurugan G, Ramprasath T, Mithieux G. Editorial: Human microbiota: A key player in the etiology and pathophysiology of cardiovascular and metabolic diseases. Front Cardiovasc Med. 2022;9:1081722.
59. Chocolatewala N, Chaturvedi P, Desale R. The role of bacteria in oral cancer. Indian J Med Paediatr Oncol. 2010;31(4):126–131.
60. Speicher DJ, Ramirez-Amador V, Dittmer DP, Webster-Cyriaque J, Goodman MT, Moscicki AB. Viral infections associated with oral cancers and diseases in the context of HIV: a workshop report. Oral Dis. 2016;22 Suppl 1(Suppl 1):181–192.
61. Cancer Research UK. Mouth and oropharyngeal cancer. 2021. Accessed 01 October 2023. https://www.cancerresearchuk.org/about-cancer/mouth-cancer.
62. Gov.UK. Chapter 6: Oral cancer. 2021. Accessed 01 October 2023. https://www.gov.uk/government/publications/delivering-better-oral-health-an-evidence-based-toolkit-for-prevention/chapter-6-oral-cancer.
63. Lazos JP, Piemonte ED, Lanfranchi HE, Brunotto MN. Characterization of Chronic Mechanical Irritation in Oral Cancer. Int J Dent. 2017;2017:6784526.
64. Piemonte E, Lazos J, Belardinelli P, Secchi D, Brunotto M, Lanfranchi-Tizeira H. Oral cancer associated with chronic mechanical irritation of the oral mucosa. Med Oral Patol Oral Cir Bucal. 2018;23(2):e151–e160.
65. Pentenero M, Azzi L, Lodi G, Manfredi M, Varoni E. Chronic mechanical trauma/irritation and oral carcinoma: A systematic review showing low evidence to support an association. Oral Dis. 2022;28(8):2110–2118.
66. Agrawal A, Shindell E, Jordan F, Baeva L, Pfefer J, Godar DE. UV radiation increases carcinogenic risks for oral tissues compared to skin. Photochem Photobiol. 2013;89(5):1193–1198.
67. Awan KH, Hegde R, Cheever VJ, Carroll W, Khan S, Patil S, Warnakulasuriya S. Oral and pharyngeal cancer risk associated with occupational carcinogenic substances: Systematic review. Head Neck. 2018;40(12):2724–2732.
68. Nikkilä R, Tolonen S, Salo T, Carpén T, Pukkala E, Mäkitie, A. Occupational Etiology of Oropharyngeal Cancer: A Literature Review. Int J Environ Res Public Health. 2023;20(21):7020.
69. Paget-Bailly S, Cyr D, Luce D. Occupational exposures to asbestos, polycyclic aromatic hydrocarbons and solvents, and cancers of the oral cavity and pharynx: a quantitative literature review. Int Arch Occup Environ Health. 2012;85(4):341–351.
70. Barul C, Carton M, Radoï L, Menvielle G, Pilorget C, Woronoff AS, Stücker I, Luce D, ICARE study group. Occupational exposure to petroleum-based and oxygenated solvents and oral and oropharyngeal cancer risk in men: A population-based case-control study in France. Cancer Epidemiol. 2019;59:22–28.
71. Zhan H, Liu D, Deji Z, Liang W, Li J. Exposure to mixture particulate contaminants in the air and the risk of oral cancer: An updated systematic review and meta-analysis. Heliyon. 2024;10(19):e38568.
72. Jones SH, St Peter CC, Ruckle MM. Reporting of demographic variables in the Journal of Applied Behavior Analysis. J Appl Behav Anal. 2020;53(3):1304–1315.
73. Jalali-Farahani S, Amiri P, Bakht S, Shayeghian Z, Cheraghi L, Azizi F. Socio-Demographic Determinants of Health-Related Quality of Life in Tehran Lipid and Glucose Study (TLGS). Int J Endocrinol Metab. 2017;15(4):e14548.
74. Shao Y, Ahmed A, Liappis AP, Faselis C, Nelson SJ, Zeng-Treitler Q. Understanding Demographic Risk Factors for Adverse Outcomes in COVID-19 Patients: Explanation of a Deep Learning Model. J Healthc Inform Res. 2021;5(2):181–200.
75. Kruse AL, Bredell M, Grätz KW. Oral cancer in men and women: are there differences?. Oral Maxillofac Surg. 2011;15(1):51–55.
76. Conway DI, Petticrew M, Marlborough H,

Berthiller J, Hashibe M, Macpherson LM. Socioeconomic inequalities and oral cancer risk: a systematic review and meta-analysis of case-control studies. Int J Cancer. 2008;122(12):2811–2819.
77. Warnakulasuriya S. Significant oral cancer risk associated with low socioeconomic status. Evid Based Dent. 2009;10(1):4–5.
78. Chen S, Lin Z, Chen J, Yang A, Zhang Q, Xie C, Zhang X, Yang Z, Chen W, Song M. Older age is a risk factor associated with poor prognosis of patients with squamous cell carcinoma of the oral cavity. Eur Arch Otorhinolaryngol. 2020;277(9):2573–2580.
79. Centres for Disease Control and Prevention. About oral cancer. 2024. Accessed 07 May 2025. https://www.cdc.gov/oral-health/about/about-oral-cancer.html.
80. Kumar T, Puri G, Aravinda K, Arora N, Patil D, Gupta R. Oral sex and oral health: An enigma in itself. Indian J Sex Transm Dis AIDS. 2015;36(2):129–132.
81. Kanmodi KK, Amzat J, Nwafor JN, Nnyanzi LA. Global health trends of human papillomavirus-associated oral cancer: A review. Public Health Chall. 2023;2(4):e126.
82. Bagan J, Sarrion G, Jimenez Y. Oral cancer: clinical features. Oral Oncol. 2010;46(6):414–417.
83. Valdez JA, Brennan MT. Impact of Oral Cancer on Quality of Life. Dent Clin North Am. 2018;62(1):143–154.
84. Mortazavi H, Safi Y, Baharvand M, Rahmani S. Diagnostic Features of Common Oral Ulcerative Lesions: An Updated Decision Tree. Int J Dent. 2016; 2016:7278925.
85. Fang QG, Shi S, Li ZN, Zhang X, Liua FY, Xu ZF, Sun CF. Squamous cell carcinoma of the buccal mucosa: Analysis of clinical presentation, outcome and prognostic factors. Mol Clin Oncol. 2013;1(3):531–534.
86. Umoh A, Azodo C. Association between Periodontal Status, Oral Hygiene Status and Tooth Wear among Adult Male Population in Benin City, Nigeria. Ann Med Health Sci Res. 2013;3(2):149–154.
87. Gupta S, Mukhiya S, Kafle A, Ghimire S, Acharya UK. Tooth Mobility among Patients visiting a Tertiary Care Centre: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2023;61(257):30–35.
88. Giargia M, Lindhe J. Tooth mobility and periodontal disease. J Clin Periodontol.1997;24(11):785–795.
89. Ghods K, Alaee A, Jafari A, Rahimi A. Common etiologies of generalized tooth mobility: a review of literature. J Res Dent Maxillofac Sci. 2022;7(4):249 – 259.
90. Scully C, Porter S. Oral cancer. West J Med. 2001;174(5):348–351.
91. Montero PH, Patel SG. Cancer of the oral cavity. Surg Oncol Clin N Am. 2015;24(3):491–508.
92. Scully C, Bagan J. Oral squamous cell carcinoma overview. Oral Oncol. 2009;45(4-5):301-308.
93. Haya-Fernández M, Bagan J., Murillo-Cortés J, Poveda-Roda R, Calabuig C. The prevalence of oral leukoplakia in 138 patients with oral squamous cell carcinoma. Oral Dis. 2004;10(6):346-348.
94. Johnstone C, Rich SE. Bleeding in cancer patients and its treatment: a review. Ann. Palliat. Med. 2018;7(2):265–273.
95. Bergamini C, Ferris RL, Xie J, Mariani G, Ali M, Holmes WC, Harrington K, Psyrri A, Cavalieri S, Licitra L. Bleeding complications in patients with squamous cell carcinoma of the head and neck. Head neck. 2021;43(9):2844–2858.
96. Ghazi N, Saghravanian N, Anvari K, Saghafi Khadem, S, Hoseinzadeh, M, Barzanouni, R. Evaluation of survival rate in patients with tongue squamous cell carcinoma: a retrospective single-center study. BMC oral health, 2025;25(1):658.
97. Natarajan PM, Swamikannu B, Sivaraman NM, Stylin, AGSQ. Prevention of Oral Cancer: A Comprehensive Guide. J Pharm Bioallied Sci. 2024;16(Suppl 5):S4239–S4241.
98. Kanmodi KK, Fagbule OF. Does head and neck cancer (HNC) education have impact on adolescents' knowledge and attitudes towards HNC and HNC peer-education? An example from Nigeria. Int J Child Adolesc Health. 2018;11(3).
99. Kanmodi KK, Fagbule OF. Towards head and neck cancer prevention in Nigeria: insights from the CHANCE Programme. Head Neck. 2020;1(1):14.

100. Kanmodi KK, Kanmodi PA. A call for the inclusion of a course on basic oral healthcare practice into the Nigerian nursing and midwifery education curriculum. Pol Ann Med.2021;28(2):256–8.
101. Dodd S, Widnall E, Russell AE, Curtin EL, Simmonds R, Limmer M, Kidger J. School-based peer education interventions to improve health: a global systematic review of effectiveness. BMC Public Health. 2022;22(1):2247.
102. Chaurasia A, Alam SI, Singh N. Oral cancer diagnostics: An overview. Natl J Maxillofac Surg. 2021;12(3):324–332.
103. Oliver RJ, Sloan P, Pemberton MN. Oral biopsies: methods and applications. Br Dent J. 2004;196(6):329–362.
104. Lewis MAO. Mouth cancer: presentation, detection and referral in primary dental care. Br Dent J. 2018;225(9):833–840.
105. Mills S. How effective is toluidine blue for screening and diagnosis of oral cancer and premalignant lesions?. Evid Based Dent. 2022;23(1):34–35.
106. Vijayakumar V., Reghunathan D., Edacheriyan B., Mukundan A. Role of Toluidine Blue Staining in Suspicious Lesions of Oral Cavity and Oropharynx. Indian J Otolaryngol Head Neck Surg. 2019;71(Suppl 1):142–146.
107. Petruzzi M, Lucchese A, Baldoni E, Grassi FR, Serpico R. Use of Lugol's iodine in oral cancer diagnosis: an overview. Oral Oncol. 2010;46(11):811–813.
108. Yang G, Wei L, Thong BKS, Fu Y, Cheong IH, Kozlakidis Z, Li X, Wang H, Li X. A Systematic Review of Oral Biopsies, Sample Types, and Detection Techniques Applied in Relation to Oral Cancer Detection. Biotech. 2022;11(1):5.
109. Jeng PY, Chang MC, Chiang CP, Lee CF, Chen CF, Jeng JH. Oral soft tissue biopsy surgery: Current principles and key tissue stabilization techniques. J Dent Sci. 2024;19(1),11–20.
110. Puri A, Wuertz B, Rhodus NL, Ondrey FG. Safety of oral mucosal punch biopsy and other oral biospecimen collections in clinical research. Oral Surg Oral Med Oral Pathol Oral Radiol. 2025;139(3):344–351.
111. Martin RA. Incisional versus excisional biopsy. Semin Vet Med Surg Small Anim.1993;8(4):228–234.
112. Shah JP, Gil Z. Current concepts in management of oral cancer--surgery. Oral Oncol. 2009;45(4-5):394–401.
113. Omura K. Current status of oral cancer treatment strategies: surgical treatments for oral squamous cell carcinoma. Int J Clin Oncol. 2014;19(3):423–430.
114. Chinn SB, Myers JN. Oral Cavity Carcinoma: Current Management, Controversies, and Future Directions. J Clin Oncol. 2015;33(29):3269–3276.
115. Huang SH, O'Sullivan B. Oral cancer: Current role of radiotherapy and chemotherapy. Med Oral Patol Oral Cir Bucal. 2013;18(2):e233–e240.
116. Hartner L. Chemotherapy for Oral Cancer. Dent Clin North Am. 2018;62(1):87–97.
117. Bourhis J, Overgaard J, Audry H, Ang KK, Saunders M, Bernier J, Horiot JC, Le Maître A, Pajak TF, Poulsen MG, O'Sullivan B. Hyperfractionated or accelerated radiotherapy in head and neck cancer: a meta-analysis. Lancet. 2006;368(9538):843–854.
118. Pignon JP, Le Maitre A, Maillard E, Bourhis J, Mach-Nc Collaborative Group. Meta-analysis of chemotherapy in head and neck cancer (MACH-NC): an update on 93 randomised trials and 17,346 patients. Radiother. Oncol. 2009;92(1):4–14.
119. Van Zyl AW, Marnewick JC. Aetiology of oral cancer: Clinical review. SADJ. 2012;67(10):554–556.
120. Cheraghlou S, Schettino A, Zogg CK, Judson BL. Changing prognosis of oral cancer: An analysis of survival and treatment between 1973 and 2014. Laryngoscope. 2018;128(12):2762–2769.