Risk factors

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Tobacco, alcohol and betel quid (areca nut, catechu, slaked lime wrapped in a piper betel leaf) are long-established risk factors for oral cavity squamous cell carcinoma (OCSCC). There is a dose–response relationship between the use of Guido Fanconi , 1892–1979, Swiss paediatrician, named several conditions, including Fanconi anaemia, a rare genetic disorder of DNA repair leading to bone marrow failure and the development of haematological and solid malignancies typically within early life. Frederick Pei Li , 1940–2015, Boston, MA, USA, and Joseph F Fraumeni Jr syndrome of soft-tissue sarcomas, breast cancer and other malignancies in 1969. tobacco, alcohol and betel quid and the development of oral cancer. Transcriptionally active human papillomavirus (HPV) accounts for only a small percentage (approximately 5%) of OCSCCs, which is in stark contrast to oropharyngeal squa - mous cell cancers (OPSCCs), where 50–70% are caused by HPV . Although HPV-positive SCC has prognostic signifi cance in the oropharynx (see Chapter 52), this survival advantage does not appear to be conferred within the oral cavity . Other risk factors include pr evious exposure to radiation, chronic infection, immunosuppression and hereditary conditions such as Fanconi anaemia and Li–Fraumeni syndrome. , b. 1933, National Institutes of Health, Bethesda, MD, USA, described a familial

Gingiva (gum) Hard palate Soft palate Retromolar trigone Buccal mucosa (lip and cheek lining) Tongue Nasal cavity Oral cavity Larynx Hyoid bone Trachea

Risk factors for oral cavity malignancy /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Premalignant lesions The majority of oral cancers do not originate from a pre existing lesion. However, there are a group of oral premalignant lesions, or more accurately described potentially malignant lesions, that are mucosal abnormalities from which oral cancer can arise. These lesions include leukoplakia, erythroplakia, erythroleukoplakia, prolifera tive verrucous leukoplakia (PVL), oral submucous fibrosis, oral lichen planus and lupus erythematosus, as well as inherited conditions such as epidermolysis bullosa and dyskeratosis congenita. A leuko plakia is a white patch or plaque that cannot be rubbed o ff , while an erythroplakia is a bright red velvety plaque, neither of which can be characterised clinically or pathologically as any other recognisab le condition. A speckled leukoplakia or erythroleukoplakia is essentially a combination of both; it carries the greatest risk for malignant change. The management of premalignant lesions is challenging, not least because of an inconsistency with nomenclature inter nationally but also because the natural history of these lesions remains unclear. The reported rates of malignant transformation v widely between studies and countries. A systematic review of observa tional studies in 2016 reported that malignant transfor mation in oral leukoplakia could vary from 0.13% to 34.0%. Risk assessment forms the cornerstone of the management of these lesions. Among these lesions, erythroleukoplakia, PVL and dyskeratosis congenita carry the highest risk for malignant transformation. Clinical factors to be considered include size, location and lifestyle exposure to known carcinogens. Biopsy of lesions is advocated for accurate pathological diagnosis

as well as to ascertain the degree of dysplasia (mild, moderate, severe), or indeed the presence of malignancy in a lesion. Summary box 53.2 Factors associated with increased risk for malignant change in pre-existing (dysplastic) lesions /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF malignant or dysplastic lesion does not completely remove the risk of transformation and as such appropriate surveillance regimes are necessary .

Smoking Alcohol Betel quid HPV Hereditary conditions Immunosuppression Chronic infection Potentially/premalignant lesions Female sex 2 Size >200 /uni00A0 mm Non-homogeneous lesion Non-smoker Presence of multiple lesions Location (e.g. lateral border of tongue/ /f_l oor of mouth)

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