Management

The treatment options for oral cancer depend on the stage of the lesion. Over 90% of all oral cancers are squamous cell carcinomas (Neville & Day 2002, Carew et al 2003) therefore, the available treatment option for squamous cell carcinomas will be discussed.

Generally only one treatment modality is required for early stage (stage I and II) oral cancers, either surgery (surgical removal of cancer) or radiation therapy (the use of high energy X-rays to destroy cancer cells) (Ord & Blanchaert 2001). Both methods have been found to be effective and have five-year survival rates of approximately 85% for stage I and 66% for stage II malignancies (Sciubba 2001). In the advanced stages (stages III and IV) the treatment therapy becomes increasingly complex and patients are faced with a poor five-year prognosis. A combination of surgery, radiotherapy and possibly chemotherapy may be required in order to achieve the best possible survival rate (Ord & Blanchaert 2001; Sciubba 2001). The five-year prognosis for survival for those with stage III and IV oral cancer is 41% and 9% respectively (Sciubba 2001).

A 2010 Cochrane review found that the addition of chemotherapy to the treatment regimen increased survival by 9% and that in patients with non-operable tumours by 22% when used in addition to radiotherapy (Furness et al 2010).

Prognosis

Early detection, diagnosis and treatment of oral cancer has been shown to significantly increase the survival rate of those with the disease (Mashberg & Samit 1995; Sciubba 2001). In most cases however, diagnosis does not occur until the cancer is advanced (stage III or IV) when regional or distant metastases have developed, therapy is more complex and the prognosis is poorer (Kujan et al 2006; Neville & Day 2002; Ord & Blanchaert 2001; Sciubba 2001). A recent study has shown that 60% of oral cancers are advanced by the time they are detected and 15% of patients have another cancer close by, such as the larynx, oesophagus and lungs (Gonsalves et al 2007). Table 2 shows the five-year survival rate for oral cancer at different stages of development (Scuibba 2001).

Further information

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Table 2: Five-year survival for oral squamous cell carcinomas by stage at diagnosis

STAGE	5 YEAR SURVIVAL RATE (%)
1	85.0%
II	66.0%
	41.0%
IV	9.0%

Smoking cessation

Dental professionals should help their patients quit smoking. Studies have shown that a 10 minute guit-smoking consultation by a health professional will aid 3% more smokers to abstain from smoking for six months or longer compared to those who receive no advice (Parrott, Godfrey & Raw 1998).

Conclusion

Oral cancer is of increasing concern as little progress appears to have been made in reducing the incidence and number of deaths associated with the disease. Studies show that early detection of oral cancer markedly improves the five-year survival rate when compared to advanced lesions (Mashberg and Samit 1995; Sciubba 2001). Many oral cancer lesions are not diagnosed until they are in the advanced stages, where the mortality rate increases significantly. The dental health professional has a significant role in prevention and detection of early cancerous lesions. If a clinician is thorough and knowledgeable in the detection of oral cancer, he or she could be involved in the early detection of some of the thousands of new cases detected each year.

References

ACS (American Cancer Society) History of the cancer prevention studies. Viewed online at http://www.cancer.org/Research/ResearchProgramsFunding/history-of-the-cancer-prevention-Lodi G, Sardella A, Bez C, et al. Interventions for treating oral leukoplakia. Cochrane Database of Systematic Reviews 2006, Issue 4. Art. No.: CD001829. DOI: 10.1002/14651858. studies July 2010. CD001829.pub3. Lubin JH, Gaudet MM, Olshan AF, et al. Body mass index, AIHW (Australian Institute of Health and Welfare) & AACR cigarette smoking, and alcohol consumption and cancers of the oral cavity, pharynx, and larynx: modeling odds ratios in pooled case-control data. Am J Epidemiol 2010;171:1250-

AIHW (Australian Institute of Health and Weifare) & AACH (Australasian Association of Cancer Registries) 2008. Cancer in Australia: an overview, 2008. Cancer series no. 46. Cat. no. CAN 42. Canberra: AIHW. AIHW (Australian Institute of Health and Weifare) Chronic diseases mortality. Viewed online at http://www.aihw.gov.au/

chronic-diseases-mortality/ July 2010. merican Dental Association, Perform a death-defving act: The 90-second oral cancer examination. J Am Dent Assoc 2001-132-365-405

2001;132:305-405. Carew JF, Singh B and Shah JP. 2003 Clinical evaluation and differential diagnosis. In: Shah JP, Johnson NW, Batsakis JG, editors: Oral cancer, 1st edition. Informa HealthCare, 2003; 183-198.

Cottrell J, Street J, Chong A, et al. Comparing cancer profiles Cottrell J, Street J, Chong A, et al. Companig cancer profiles and survival of aboriginal and non-aboriginal patients in South Australia: where are the opportunities for improving Aboriginal health? Asian Pac J Cancer Prev 2007; 8:495–501. Curado MP, Hashibe M. Recent changes in the epidemiology of head and neck cancer. Curr Opin Oncol 2009;21:194–200.

Edefonti V, Bravi F, La Vecchia C, et al. Nutrient-based dietary patterns and the risk of oral and pharvngeal cancer. Oral Oncol 2010:46:343-8.

Forrest II Drury TE Horowitz AM IIS dental hydienists' Forrest JL, Drury IE, Horowitz AM. U.S. dental hygienists' knowledge and opinions related to providing oral cancer examinations. J Cancer Educ 2001;16:150–6.
Furness S, Glenny AM, Worthington HV, et al, The CSROC Expert Panel. Interventions for the treatment of oral cavity and oropharyngeal cancer: chemotherapy. Cochrane Database of Constructive Sector Database. Database of Constructive Sector Panel. Systematic Reviews 2010, Issue 9, Art. No.: CD006386, DOI:

10.1002/14651858.CD006386.pub2. Consalves WC, Chi AC, Neville BW. Common oral lesions: Part II. Masses and neoplasia. Am Fam Physician 2007;75:509–12. Hashibe M, Brennan P, Chuang SC, et al. Interaction between tobacco and alcohol use and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium, Cancer Epidemiol Biomarkers Prev 2009:18:541-50.

2009;145:44-50. Heck JE, Berthiller J, Vaccarella S, et al. Sexual behaviours and the risk of head and neck cancers: a pooled analysis in the International Head and Neck Cancer Epidemiology (INHANCE) consortium. In J Epidemiol 2010;39:166-81. Kramer IR, Lucas RB, Pindborg JJ, et al. Definition of leukoplakia

and related lesions; an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol 1978:46:518-39.

World Health Organisation (WHO) 1997. Oral health surveys. Basic methods. 4th ed. Geneva: WHO. Basic methods. 4th ed. Geneva: WHO. World Health Organisation (WHO) Global data on incidence o oral cancer. WHO/NMH/CHP/HPR/ORH 2005.

61. Marron M, Boffetta P, Zhang ZF, et al. Cessation of alcohol

drinking, tobacco smoking and the reversal of head and neck cancer risk. Int J Epidemiol 2010;39:182–96.

Masereijan NN, Giovannucci E, Rosner B, et al, Prospective

study of vitamins C. E. and A and carotenoids and risk of oral

study or vitalmits C, E, and A and cardeenoos and risk of oral permalignant lesions in men. Int J Cancer 2007;120:970-7. Mashberg A, Samit A, Early diagnosis of asymptomatic oral and oropharyngeal squamous cancers. CA Cancer J Clin 1995;45:328-51.

Moore S, Johnson N, Pierce A, et al. The epidemiology of lip

cancer: a review of global incidence and aetiology. Oral Dis

cancer: a review of global incidence and aetiology. Oral Uis 1999;5:185–95. Review. Mork J, Lie AK, Glattre E, et al. Human papillomavirus infection as a risk factor for squamous-cell carcinoma of the head and neck. N Engl J Med 2001;3:44:1125–31. Negr E, Boffetta P, Berthiller J et al. Family history of cancer.

pooled analysis in the International Head and Neck Cancer

Epidemiology Consortium, Int J Cancer 2009:124:394-401

Neville BW. Dav TA. Oral cancer and precancerous lesions. CA

Nevline aw, Day IA, Oral cancer and precancerous resons. CA Cancer J Clin. 2002;52:195–215. Review. Ord RA, Blanchaert RH Jr. Current management of oral cancer. A multidisciplinary approach. J Am Dent Assoc. 2001;132:195–235.

Parrott S, Godfrey C, Raw M, et al. Guidance for

commissioners on the cost effectiveness of smoking

Petersen PE, Oral cancer prevention and control – The approach of the World Health Organisation, Oral Oncol (2008),doi:10.1016/j.oraloncology.2008.05.023.

Petersen PE. Strengthening the prevention of oral cancer

the WHO perspective. Community Dent Oral Epidemic

2006;33:39 (-9). Reibel J. Prognosis of oral pre-malignant lesions: significance of clinical, histopathological, and molecular biological characteristics. Crit Rev Oral Biol Med 2003;14:47–62. Sciubba JJ. Oral cancer and its detection. History-taking and the diagnostic phase of management. J Am Dent Assoc. Oracted December 20 (2014).

Weinberg MA, Estefan DJ, Assessing oral malignancies, Am

essation interve

2005:33:397-9.

1998:53 Suppl 5 Pt 2:S1-38.

2001:132 Suppl:12S-18S.

Fam Physician 2002:65:1379-84 Revie

ons. Health Educational Authority. Thorax

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Early Detection Saves Lives

Introduction

Oral cancer is a potentially fatal disease that affects many thousands of people each year worldwide. The World Health Organisation (2005) has stated that it is the eleventh most prevalent cancer in the world. Over 90% of oral cancers diagnosed present as squamous cell carcinomas (Neville & Day 2002). The dental health professional is the first line of defence in the early detection and monitoring of oral cancers, and as such must recognise both early signs of cancerous lesions and the patients who are most at risk.

Incidence

In Australia, 2357 new cases of cancer in the oral cavity were diagnosed during 2005 (see Table 1) (AIHW 2008). Today, the incidence of oral cancer has been found to be approximately two-thirds higher amongst men than women (AIHW 2008) and three times higher among Indigenous Australians than other Australians (Cottrell et al 2007).

Table 1: Incidence and mortality of oral cancer by site, Australia 2005

SITE OF CANCER	INCIDENCE (No.)	MORTALITY (No.)
Lip	786	10
Tongue	479	148
Mouth	500	110
Major salivary glands	258	81
Oropharynx	334	117
TOTAL	2357	466

(AIHW 2008)

Mortality and survival

Mortality of oral cancer is particularly high not because it is difficult to diagnose, but because it is frequently asymptomatic and therefore it is diagnosed at a late stage. Often it is only discovered when the cancer has already metastasised, usually to the lymph nodes of the neck. Prognosis once the cancer has metastasised is worse than when it is confined to a localised intra oral area.

In 2006, there were 613 deaths (0.5% of all deaths) from oral cancer in Australia. The female death rate for oral cancer has been stable over the past century at about two per 100,000. The rate for males in the early part of the 20th century was around 25 per 100,000, but declined steadily to six per 100,000 by 1958 where it has remained (Figure 1).

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Figure 1: Trends in death rates for oral cancer in Australia, 1907 to 2006



Note: Rates are age-standardised (ASR) to the 2001 Australian population. Source: AIHW.

Risk factors

An important aspect in the early detection of oral cancer is the ability for the dental professional to identify the risk factors involved. There exist some major as well as minor risk factors which are associated with the development of oral cancer. Risk factors are listed below.

Tobacco smoking – cigarettes, pipes and cigars

Particularly in combination with excess alcohol, tobacco smoking is a major risk factor for oral and oro-pharyngeal cancer. Two such studies, Cancer Prevention Study (CPS) I and II, sponsored by the American Cancer Society, are the largest epidemiological studies ever undertaken, each following more than one million men and women. Evidence from these and other epidemiological studies has provided key documentation of the association between cigarette smoking and oral cancer.

Chewing tobacco, although not common in Australia, also increases risk of oral cancer (Curado et al 2009).

Excessive consumption of alcohol

The risk of oral cancer increases with the number of drinks per day and the number of years of drinking alcohol (Lubin et al 2010). The combination of heavy smoking and heavy alcohol use increases the risk. A recent study confirmed that the joint effect between tobacco and alcohol use increases the risk of head and neck cancer more than by multiplying the individual effects with an estimate that 64% of head and neck cancers could be explained by this combination (Hashibe et al 2009).



It is crucial to acknowledge that smoking and excessive alcohol consumption are the two principal risk factors, accounting for approximately 75% of oral cancers (Weinburg at al 2002). AIHW (2008) notes the proportions of oral cancers attributable to smoking are 57% for males and 51% for females; the proportions attributable to heavy alcohol use are 39 % for males and 31% for females.

• Sunlight exposure (for lip cancer)

This has mostly come through indirect evidence. Lip cancer is more common among rural workers than urban workers, more frequently occurs on the lower lip and among males more than females (Moore et al 1999).

Age (risk increases as age increases)

Cancer of the oral cavity is rare in people less than 40 years of age and the same risk factors may not apply to those in the younger category (Hashibe et al 2009).

Snuff, gutkha, betel quid or areca nut use

Locations where these substances are commonly used have high rates of oral cancer as well as submucosal fibrosis. Such countries include Papua New Guinea and India.

• Viral infections including human papilloma virus

Recently, the role of viruses in the development of cancer of the oral mucosa has been the focus of tremendous interest, and particular attention has been paid to human papilloma virus (HPV) subtypes. Human papilloma virus types 16 and 18 are found in 22% and 14% of oropharyngeal tumors, respectively, and they increase the risk of cancer development by three- to-five-fold (Mork et al 2001). Recently the association between oral cancer and sexual activity has been explored. Women who have ever had oral sex have four times the risk of cancer of the base of the tongue than women who have not, and having two sexual partners in comparison with only one doubles the risk. Among men a history of same-sex sexual contact increases the risk by almost nine-fold. (Heck et al 2010).

• Nutritional deficiencies (especially of vitamins A, C and E) There is some evidence that diets of animal origin and rich in animal fats lead to a higher risk of oral and pharyngeal cancer, and diets rich in fruit and vegetables, and vegetable fats to a lower risk (Edefonti et al 2010). Dietary vitamin C was significantly associated with reduced risk of premalignant lesions (Maserejian et al 2007).

Genetic predisposition

There also may be a genetic component to risk of oral cancer. Among alcohol and tobacco users people with a family history of cancer have been shown to have an increased risk (Negri et al 2009).

Minimising risk

After quitting tobacco smoking for one to four years, the risk of head and neck cancer is reduced by 30% with the risk reduction due to smoking cessation after 20 years or more of almost 80%. For alcohol use, a beneficial effect (40%) on the risk of head and neck cancer was only observed after 20 years or more of quitting (Marron et al 2010).

Detection of oral cancer

The dental health professional is the first line of defence in the early detection and monitoring of oral cancers. The sites of involvement of oral cancer are easily visible and accessible to a dental professional during a routine dental examination without requiring specialised equipment and techniques (Mashberg & Samit 1995; Petersen 2005,2008; WHO 2005). Systematic examination of the oral tissues reduces the chance that lesions will be missed.

Oral cancer most commonly affects the tissue of the lips or the tongue. It may also occur on the floor of the mouth, cheek lining, gingiva, or roof of the mouth (palate).

Studies indicate that squamous cell carcinomas are asymptomatic in the early stages and a large proportion of these lesions are left undiagnosed until late. Diagnosis usually occurs when the lesion has become symptomatic and the tissue is visibly abnormal (Neville & Day 2002; Kujan et al 2006; Sciubba 2001; Mashberg & Samit 1995). Mashburg & Samit (1995) suggest that the main reasons for oral cancers remaining undiagnosed is due to the following: a failure of the clinician to concentrate efforts on individuals at high risk; a failure to focus on sites at highest risk; a lack of knowledge about early cancerous lesions and under-utilization of clinical aids and tests available. Many clinicians feel that they are inadequately trained to diagnose early cancerous lesions (Forrest, Drury & Horowitz 2001).

The key focus of clinical assessment is a histological diagnosis, and treatment decisions should be made on the basis of a microscopic diagnosis instead of the clinical presentation. Thus the best approach is to biopsy any suspicious lesions, or refer the patient for biopsy.

Screening

Opportunistic screening for oral cancer should be undertaken as part of routine dental examinations with special attention placed on those patients presenting with the risk factors that are associated with oral cancer. A 2006 Cochrane review stated that there is insufficient evidence for or against the effectiveness of visual screening of the general population (Kujan et al 2006).

Pre-malignant lesions

People with pre-existing oral diseases such as leukoplakia, lichen planus, erythroplakia and submucosal fibrosis also have an increased risk of progressing to malignancy (Reibel 2003; Carew et al 2003; Mashberg & Samit 1995).

Oral leukoplakia is the most commonly known premalignant lesion. It is defined as a 'white patch or plaque that cannot be characterised clinically or pathologically as any other disease' (Kramer et al 1978). Red lesions are called erythroleukoplakia and a combination of red and white lesions are called speckled leukoplakia or erythroleukoplakia (Gonsalves et al 2007). Several studies have found that only a small number (0.13 to 6.0%) of leukoplakias eventually transform into carcinomas (Mashburg & Samit 1995). A recent Cochrane review stated that 'to date there is no evidence of effective treatment in preventing malignant transformation of leukoplakia. Treatments may be effective in the resolution of lesions, however, relapses and adverse effects are common' (Lodi et al 2006).

Clinical presentation

Pre-cancerous and early cancerous lesions are usually subtle and asymptomatic. These lesions begin as a white or red patch that advance to an ulceration and later develop into an endophytic or exophytic mass (Gonsalves et al 2007).

Signs include (Carew et al 2003):

- Lesions that do not heal.
- A colour change in the oral tissues.
- Pain, tenderness or numbness anywhere in the mouth or on the lips.
- Protuberance, rough, thickened, crusted or eroded area.

Mucous membrane lesion, lump, or ulcer:

- May be a deep, hard-edged crack in the tissue.
- Most often pale colored, may be dark or discoloured.
- On the tongue, lip, or other mouth area.
- Usually painless at first (may develop a burning sensation or pain when the tumour is advanced).
- Usually small.

Additional symptoms that may be associated with this disease:

- Abnormal taste in the mouth.
- Mouth sores.
- Swallowing difficulty.
- Tongue problems.

Advanced lesions

The presence of an exophytic lesion with several loose teeth should raise suspicion of a locally advanced malignant tumour of the gingiva. Patients may have difficulty masticating and speaking with increasing size of the lesion or invasion of underlying soft tissues and musculature. Severe localised pain, referred earache and restriction in the movement of the tongue is a sign of an advanced lesion. Excessive salivation, with reduced movement of the tongue may be a sign of an advanced carcinoma of the tongue. Consumption of food may be restricted to soft foods and the patient may lose considerable weight (Carew et al 2003).

Comprehensive oral examination

- Extraoral examination: Inspection of the face, head and neck with special attention to patients that appear to spend a lot of time in the sun. Note any lesions on the skin and asymmetry of the head or neck. Regional lymph nodes in the submandibular and neck areas should be bilaterally palpitated to detect any enlargement. If an enlargement is detected, assess its mobility and consistency.
- Perioral and intraoral soft-tissue examination: Seven systematic steps to assess the lips, labial mucosa, commissures and buccal mucosa, gingiva, alveolar ridges, tongue, floor of mouth and palate. Adequate light, a mirror and two gauze squares are needed. Remove all removable prostheses before starting.

- >> Lips. With the patient's mouth both closed and open, observe the lips. Note the texture, colour and any abnormalities of the vermilion borders.
- >> Labial mucosa. With the patient's mouth partially open, lift the upper and lower lip to visually examine the labial mucosa and sulcus of the maxillary vestibule and frenum, as well as the mandibular vestibule. Note the texture, colour and any swelling or abnormalities.
- >> Buccal mucosa. Retract the buccal mucosa. Examine the right, then the left buccal mucosa, extending from the labial commissure to the tonsillar pillar. Note any changes in pigmentation, texture, colour and other abnormalities.
- Singiva. Examine the buccal and labial gingival and alveolar ridges, moving from the right maxillary posterior gingiva and alveolar ridge to the left posterior area. Then do the same for the mandibular arch from left to right. Then examine the palatal and lingual gingiva and alveolar ridges.
- Solution Tongue. Inspect the dorsum of the tongue with the patient's tongue at rest and the mouth partially opened. Look for any swellings, ulcerations and variations. Ask the patient to protrude the tongue and examine for any abnormalities. Grasp the tip of the tongue with a piece of gauze to examine the tongue in full protrusion. A mirror will help to visually assess the more posterior aspects of the lateral borders of the tongue and to retract the cheek. Run your index finger along the lateral borders to feel for any lumps. Then examine the ventral surface, palpating to detect any growths.
- Floor of the mouth. Get the patient to elevate the tongue, inspect the floor of the mouth. Wipe the floor of the mouth with gauze to dry the tissues and palpate the floor of the mouth.
- >> **Palate.** Depress the tongue with a mirror to examine the hard palate. Inspect the hard and soft palates.

(World Health Organisation 1997, American Dental Association, 2001)

Points to remember:

- High risk sites for oral cancer include the lateral borders of the tongue, floor of the mouth.
- Note any changes in colour, texture and size. If the abnormality has been present for more than two weeks, a biopsy should be carried out by an experienced practitioner.
- Follow up to ensure a definitive diagnosis has been made.
- Educate your patients about the signs and symptoms of oral cancer.
- If a patient smokes, provide appropriate cessation counselling.

Oral cancer signs

- >> Any sore in the mouth, or on the face and neck that does not heal in two weeks.
- >> Swellings, lumps or bumps on the lips, or anywhere inside the mouth.
- >> White, red or dark patches in the mouth.
- >> Repeated bleeding in the mouth.
- >> Numbness, loss of feeling or pain in any area of the mouth, face or neck.