EFFECTS OF TOBACCO ON ORAL HEALTH

Definition

Tobacco is consumed in a variety of different ways, though smoking of manufactured cigarettes is the most prevalent form of its use. The emergence of widespread cigar use particularly among adolescents of both sexes has been reported in the past decade in the US. Cigars have higher total nicotine content than cigarettes do and can deliver nicotine both through smoke and through direct oral contact with the tobacco wrapper. Cheroots are small cigars made of heavy bodied tobacco. Bidi smoking is a popular form of tobacco use in south Asia, accounting for one-third of the tobacco produced in India for smoking. Bidis and kreteks are gaining popularity among young people in North America, and more than 15% of adolescent smokers use these tobacco products. Pipe smoking is one of the oldest methods of smoking and was brought to Europe by sailors from Americas. Water pipes include special receptacles through which smoke has to pass, ostensibly to reduce its harmful effects. Hookah is an Indian water pipe. The habit of reverse smoking by holding the glowing end of cigarettes or cigars within the oral cavity is described in parts of India, south America and the Philippines. The habit is practiced extensively by older women living in rural areas.

Smoked tobacco

Tobacco smoke is made up of “side-stream smoke” from the burning tip of the cigarette and “main-stream smoke” from the filter or mouth end.

Tobacco smoke contains thousands of different chemicals which are released as particles and gases. Many toxins are present in tobacco smoke. The particulate phase includes nicotine, "tar" (itself composed of many chemicals), benzene and benzo(a)pyrene. The gas phase includes carbon monoxide, ammonia, dimethylnitrosamine, formaldehyde, hydrogen cyanide and acrolein. Some of these have marked irritant properties and some 60, including benzo(a)pyrene and dimethylnitrosamine, have been shown to cause cancer.

The tar yield of different brands of cigarettes range from 0.5 mg to 26 mg (averaging 12.5 mg.), with the most popular brands containing 15-17 mg of tar. In the European Union (EU) cigarettes have to contain less than 12 mg of tar from 1998. Nicotine yields range from 0.05 mg to 1.7 mg, with the most popular brands yielding 1.0 mg of nicotine. In developed countries over 95% of manufactured cigarettes consumed are filter-tipped.

About 10% consume tobacco as roll-your-own cigarettes mostly among lower socioeconomic groups.

Smokeless (chewing) tobacco (ST)

There are two main types of smokeless tobacco - chewing tobacco and snuff. The most written about is smokeless tobacco use among Asians taken with betel/areca quid. Over 90 percent of Indians add tobacco to the betel quid mixture. Commercially prepared betel quid products that contain mostly areca nut and flakes of tobacco are called gutka. Other ST products which carry significant mutagenicity are toombak used in the Sudan, shamma in the Jizan province in Saudi Arabia, powdered tobacco and alkali mixtures such as nass/naswar used in northern and central Asia and in Pakistan, khaini (a mixture of ST and lime) used in Bihar state of India and Nepal, and boiled/sweetened ST called zarda mostly used by people from Bangladesh. All these forms of tobacco use are associated with an increased risk of oral cancer.

Second-hand or environmental tobacco smoke (ETS)

ETS is carcinogenic to human beings. Meta-analyses show that there is a significant association between lung cancer and smoke exposure from a spouse and also between lung cancer and exposure at work. Risks for other cancer types are inconclusive. There is at present insufficient evidence that children exposed to parental smoke have an altered risk of developing any cancer.

Epidemiology

Tobacco, which annually kills 4.9 million people worldwide at present, is estimated to take 10 million lives every year by 2020. The more depressing part is that half of them will die in their middle age. Global estimates of smoking prevalence by each country is given in a WHO data base for reference. These are based on adult and youth smoking behaviours collected from population-based, cross sectional surveys at a given point of time. China is the largest producer of tobacco in the world as well as the largest consumer. A national prevalence survey in 1996 among adults (age 15+) found that 63% of males smoked.

National surveys of persons aged 18 and older from 1970 onwards report a decline in prevalence in USA and most western European countries. The rate of decline among women is less than for men, and the quit index for men is substantially higher than for women. Fig 1 illustrates trends in the USA, UK and Japan over five decades. Both in UK and USA there are now twice as many former cigarette smokers as current smokers. In

general the prevalence of smoking in most population groups is lowest among those with the highest educational level. By race, smoking among adult blacks is similar to whites in most countries. Denmark has the highest rates of smoking in the EU.

Clinical presentation

General effects

Smoking-attributed diseases include cancer of trachea, lung, bronchus, lip, mouth and pharynx, ischemic heart disease, stroke, hypertension, bronchitis, chronic obstructive pulmonary disease, emphysema and asthma. Due to substantial decrease in smoking in countries such as the UK male lung cancer rates have decreased rapidly in the last decade. On current trends, the annual number of smoking-attributable deaths among women should exceed that for men shortly after the year 2000.

Effects of tobacco on teeth and oral health

The damaging and harmful effects of tobacco usage on oral health are now well recognized, in particular a higher prevalence and severity of periodontal diseases among smokers and the association of tobacco use with candidosis, and with oral malignancies. Several recent documents have reviewed the scientific evidence relating to the oral disease burden attributable to tobacco use and have highlighted the role and the need for the dental profession to get involved with tobacco intervention.

Smoking causes discolouration of teeth and some argue that tobacco in fact might increase dental decay as it lowers salivary pH and the buffering power. Smoking is likely to cause halitosis and may affect smell and taste. Smokers may present with generalised melanosis of the oral mucosa (Fig 2) that often necessitate investigations to exclude other systemic disorders. Wound healing is impaired in tobacco smokers possibly due to local vasoconstriction and poor neutrophil function. There is fair evidence that tobacco use is a major factor in the progression of periodontal disease. Smokers have an increased prevalence of periodontitis, and their disease severity is higher with greater alveolar bone loss resulting in deeper pockets compared with non smokers. Acute necrotising ulcerative gingivitis (ANUG) (Fig 3) has been shown to be associated with heavy smoking. Periodontal therapy often fails among smokers and it is difficult to halt attachment loss. Possibly for similar reasons dental implant failure is more common in smoking subjects compared with non smokers.

Oral cancer

Oral squamous cell carcinoma presents in a variety of ways such as white and red patches, non-healing ulcers or exophytic growths. Most early lesions are asymptomatic. Persistent ulceration with rolled margins and fixation to underlying tissues are pathognomonic signs of oral malignancy (Fig 4). In late stages disease spreads to adjacent structures notably involving regional lymph nodes, and can cause mobile teeth and loss of teeth or even pathological mandibular fractures. These stages may be associated with pain, numbness or paraesthesia. The clinical features of oral cancer are described elsewhere. Currently diagnosing oral cancer relies on pathological examination by biopsy and use of imaging techniques to estimate the spread of the disease. Over 80 percent of oral cancers are associated with tobacco use.

Oral leukoplakia

Oral leukoplakia is the most common potentially malignant lesion defined as a predominantly white lesion of oral mucosa that cannot be characterised as any other definable lesion. The appearance of leukoplakia varies from uniformly white homogeneous lesions (Fig 5) to non-homogeneous speckled lesions with red and/or nodular features (Fig 6). Leukoplakia is often associated with tobacco use though idiopathic forms of leukoplakia are recognised. The site of the oral cavity affected by leukoplakia is often said to be associated with the type of tobacco habit practiced; lateral tongue and floor of mouth in cigarette smokers, palate in pipe smokers and reverse smokers, commissures in bidi smokers, buccal groves in tobacco chewers where they park the quid and lower or upper labial mucosa in snuff dippers. In a recent study in the Netherlands 64% of men and 60% of women with oral leukoplakia were smokers. Tobacco use in men was significantly associated with leukoplakia of buccal mucosa and with all leukoplakia of floor of mouth in both sexes (6). Oral leukoplakia in smokers need to be investigated by biopsy to assess any dysplasia. Moderate to severe oral epithelial dysplasia (Fig 7) when present necessitate surgical intervention. Intervention studies have demonstrated that leukoplakia present in smokers might be reversible when smoking habit was reduced or given up.

Erythroplakia

Tobacco may underlie some cases of erythroplakia.

Smoker's palate (Leukokeratosis nicotina palati)

A greyish white discolouration of the palate with multiple red elevated dots (inflamed minor salivary gland openings) is often encountered in chronic smokers. This is

considered as a benign lesion as cancer is not known to arise from this benign keratosis (Fig 8).

Reverse smoker's keratosis

This is serious potentially malignant lesion encountered in people who place the glowing end of the cigar or cigarette inside the mouth. The clinical appearance is often a mixture of red and white plaques. Excrescences are found within the lesion corresponding to inflamed minor salivary glands. Whereas aforementioned smoker's palate is not considered as a precancerous lesion palatal changes in reverse smokers is a high risk lesion that is associated with cancer development.

Aetiopathogenesis

Tobacco smoking (i.e., cigarette, pipe or cigar smoking) particularly when combined with heavy alcohol consumption has been identified as the primary risk factor for approximately 80% of oral malignancies. The risk of oral and pharyngeal cancers is similar for cigarette and cigar smokers, with an overall risk seven to ten times higher than for never smokers. This is not surprising as the oral cavity is exposed to the carcinogens in smoke whether the smoke is inhaled or not. When the frequency of daily tobacco use is computed there is strong dose response relationship between smoking rates and risk of mouth cancer. Addition of ST to the areca quid raises the relative risk of the product by nearly 15 times.

Case-control studies from Europe have reported adjusted odds ratios (ORs) of 11.1 for oral cavity and 12.9 for pharyngeal cancer. In particular, smoking frequency and duration, and age at start have significant associations. After giving up tobacco for a decade or so the risk of oral cancer of a past smoker drops significantly to levels almost comparable to never smokers.

Smoking patients show reduction of inflammatory clinical signs that might be associated with local vasoconstriction from nicotine, influence on vasculature and cellular metabolism. This may suppress symptoms of gingival inflammation. Pathogenesis of periodontitis in smokers could be linked to defects in neutrophil function, impaired serum antibody responses to periodontal pathogens and potentially diminished gingival fibroblast function suggesting altered host response and susceptibility. It is claimed that among smokers, more patients remain culture positive for periodontal pathogens after therapy. This may contribute to the often observed unfavourable treatment results among non-compliant smokers.

Diagnosis

Detection of tobacco consumption is mostly based on taking a social history. This should include questions on type of tobacco habit, daily frequency and duration of use. Age of commencement is also an important risk factor for many disorders and should be recorded. Current smokers could be regular or occasional smokers, regular being daily smokers. Some have the habit of binge smoking when consuming alcohol only but are unlikely to be addicted to tobacco.

Tobacco handling can usually be seen on heavily smoking patients' fingers and the tobacco stains on the oral mucosa and teeth. Dorsal tongue is often stained in many smokers. A bad breath can also highlight a smoker.

Validation of smoking can be done using the carbon monoxide breath test (piCO, Bedfont) or by measuring salivary, urine or serum cotinine which is a metabolite of nicotine. Cut off concentration of salivary cotinine is taken as 14 ng/ml to detect a regular smoker.

Level of dependence to tobacco can be assessed using the Fagerström test.

Treatment and interventions

Tobacco dependence shows many features of a chronic disease. Regular smokers are addicted to the habit as tobacco use results in true drug dependence. A minority is able to quit in one attempt but the majority may need some assistance to cease tobacco use. Numerous effective treatments are now available, and the dentists, oral physicians and their team members should become actively involved in efforts to reduce smoking. Smoking cessation advice delivered by dentists have shown to be effective. Brief advice given by a clinician lasting about 3 minutes can yield a cessation rate up to 5%. With additional support such as recommended use of nicotine replacement therapy the quit rates achieved could be doubled. In treating a smoker (willing to quit) the 5A's, designed as a brief counseling intervention, is helpful:

1. **A**sk about tobacco use - every patient/every visit
2. **A**ssess willingness to make a quit attempt
3. **A**dvice (those willing) to quit tobacco use. Those unwilling will need motivation to return to the topic at a later time

4. **A**ssist in quit attempt - set a quit date, emphasize total abstinence, prompt support seeking, provide supplementary material and recommend pharmacotherapy (see below)
5. **A**rrange follow up and refer to a specialist clinic if the quit attempt has failed

Pharmacotherapy

As with other chronic diseases, the most effective treatment of smoking requires multiple approaches in addition to clinician's advice. Pharmacotherapy is proven to be effective, and several products are available; nicotine patches, nicotine gum, nicotine lozenge, nicotine inhaler and nicotine nasal spray.

Non-nicotine medication approved for smoking cessation is bupropion (Zyban) therapy starting one week before the quit date and continued up to 12 weeks after quitting. There are several medical contra-indications particularly those with a predisposition to seizures and/or a history of epilepsy. Pregnant and breast-feeding mothers should not be prescribed this drug. Bupropion therapy increases the chances of quitting considerably up to 30% giving up smoking.

Prevention

Prevention of tobacco use is a key element in public health. As tobacco use and experimentation starts in early life preventive approaches should be appropriately targeted to young people. Paedodontists, orthodontists, school dentists and family practitioners can take steps to initiate advice to young children never to start smoking. Banning tobacco smoking in public places, legislation on tobacco advertising and taxation are known to effect tobacco sales. Primary prevention, the helping of people not to use tobacco in the first place and assisting current smokers to quit, is an effective way to reduce morbidity and mortality from oral cancer.

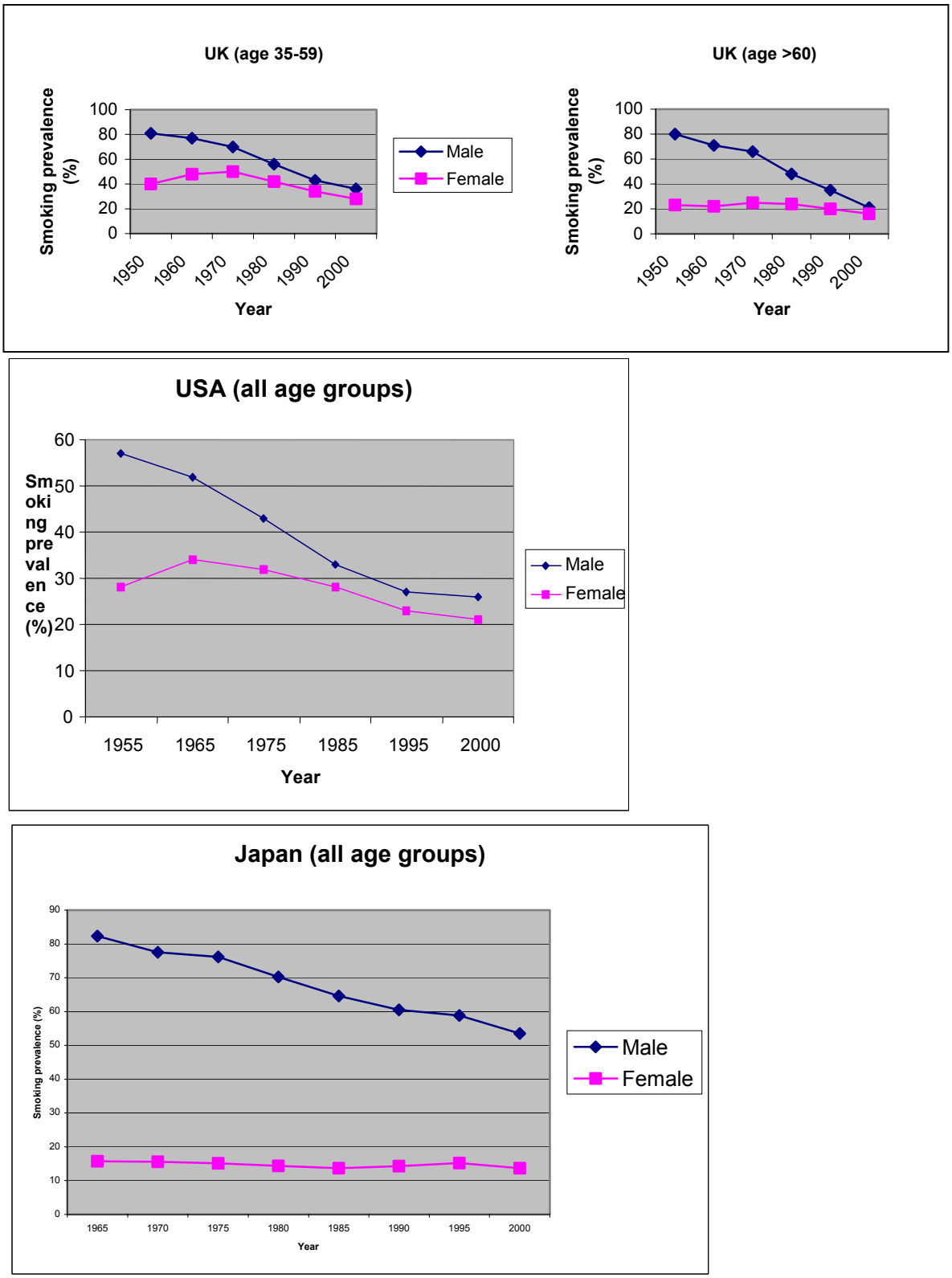

Figure 1. Trends of smoking in USA, UK and Japan by sex.

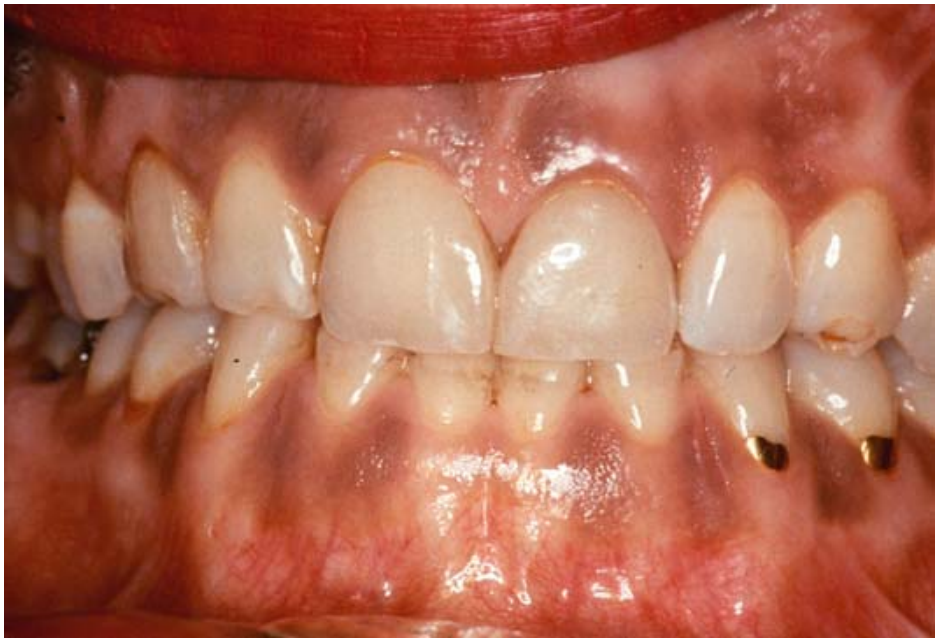

Figure 2. Diffuse melanin pigmentation on the gingiva in a Danish heavy smoker.

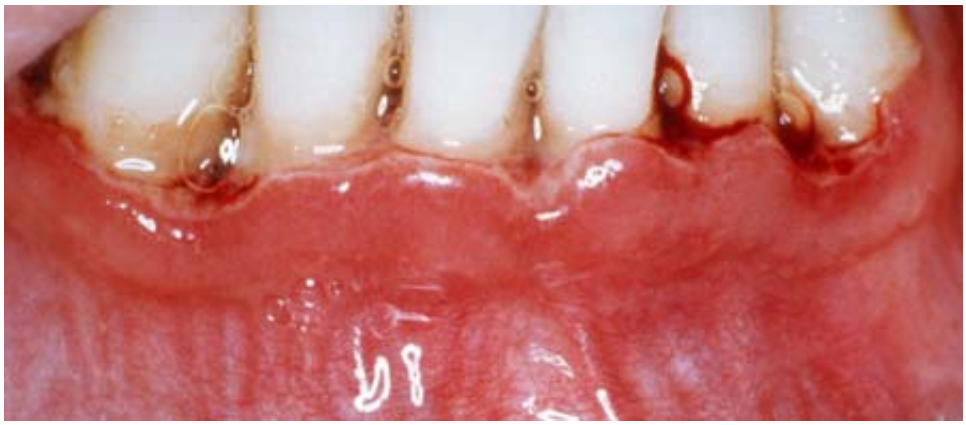

Figure 3. Acute necrotising ulcerative gingivitis showing destruction of the interdental papillae, pseudomembrane, and spontaneous bleeding.

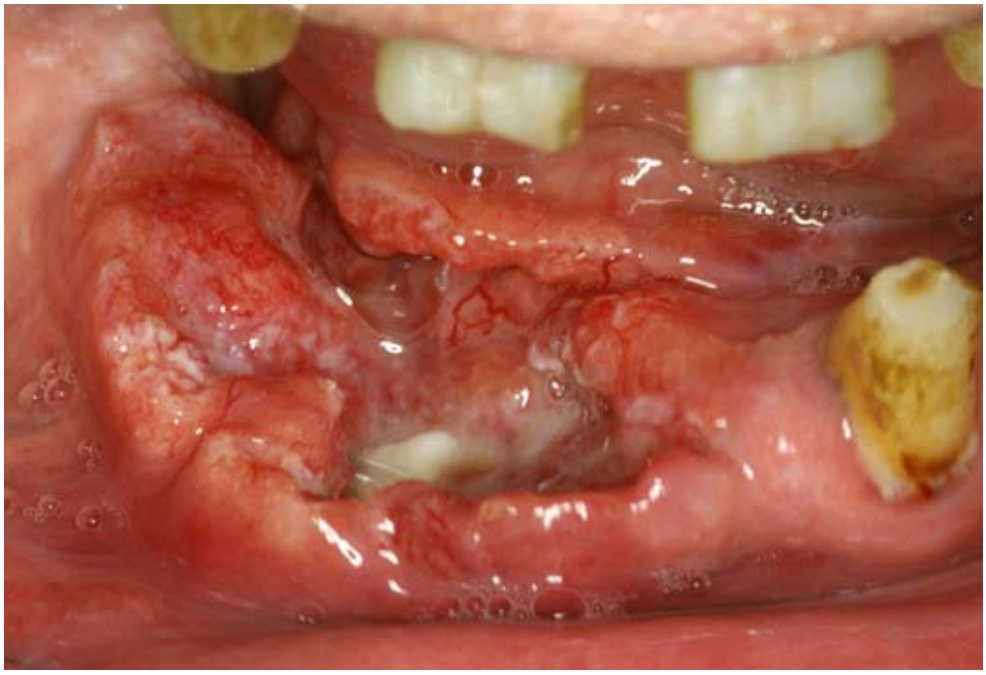

Figure 4. Cancer (squamous cell carcinoma) of the floor of the mouth/alveolar process in a heavy smoker.

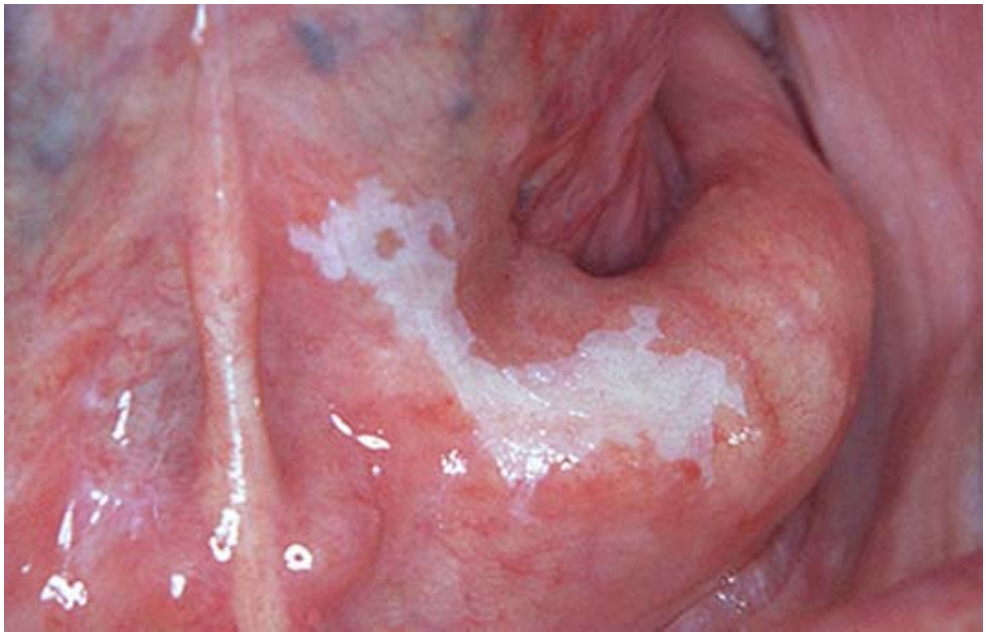

Figure 5. Homogeneous leukoplakia in the floor of the mouth.

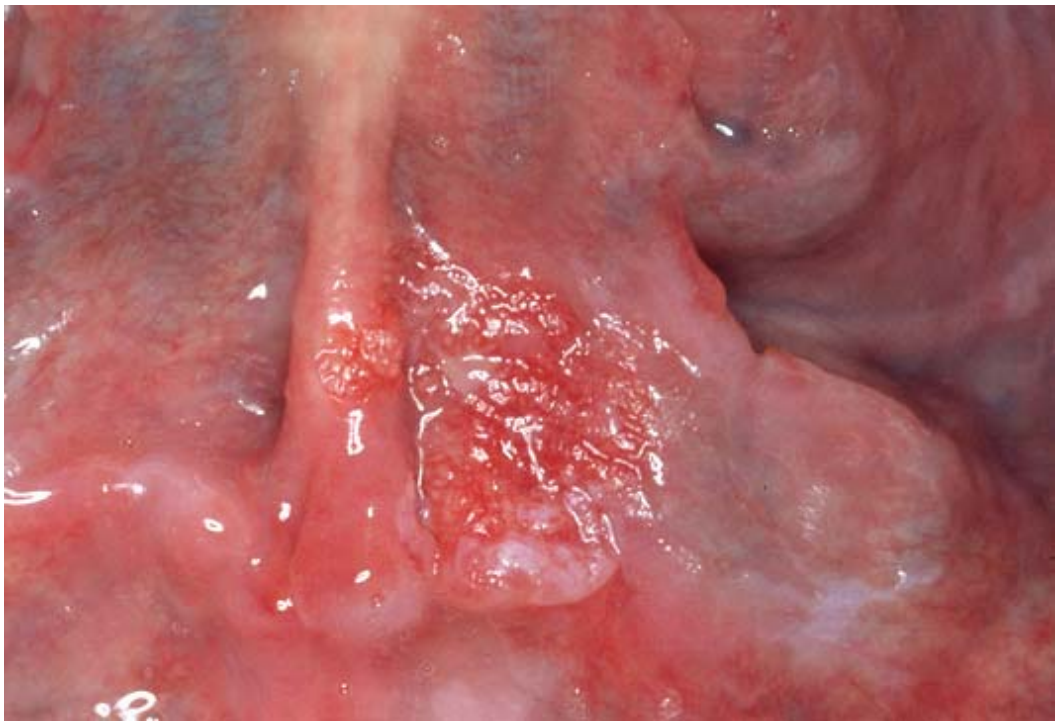

Figure 6. Non homogeneous leukoplakia in the floor of the mouth presenting with whitish, reddish, and nodular features.

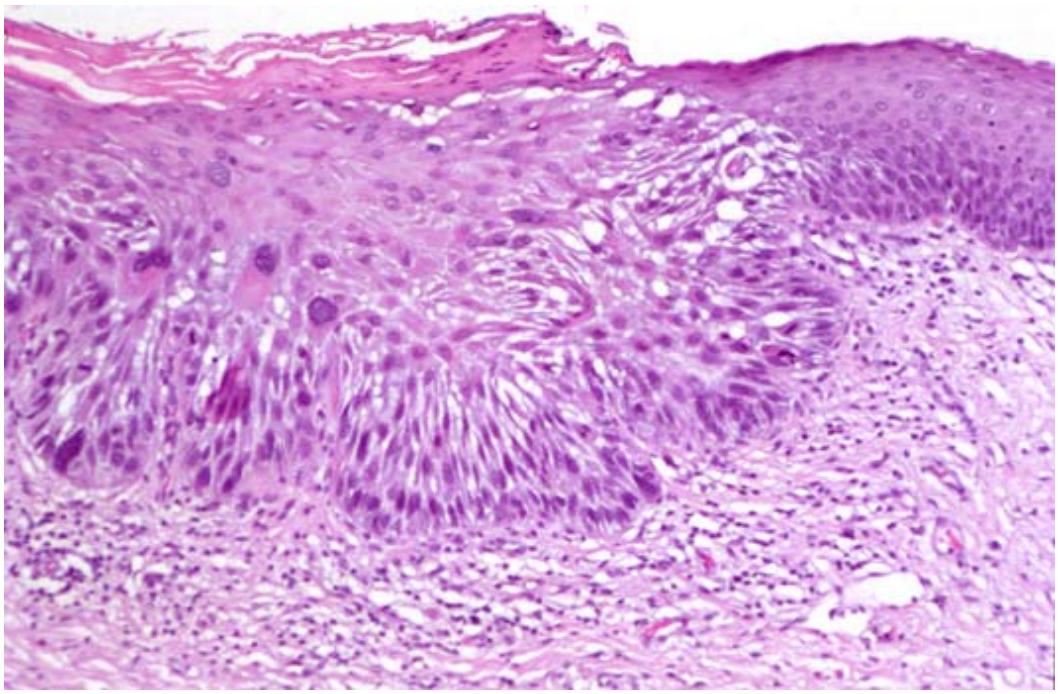

Figure 7. Biopsy of non homogeneous leukoplakia of the floor of the mouth showing severe epithelial dysplasia. Normal epithelium in right part.

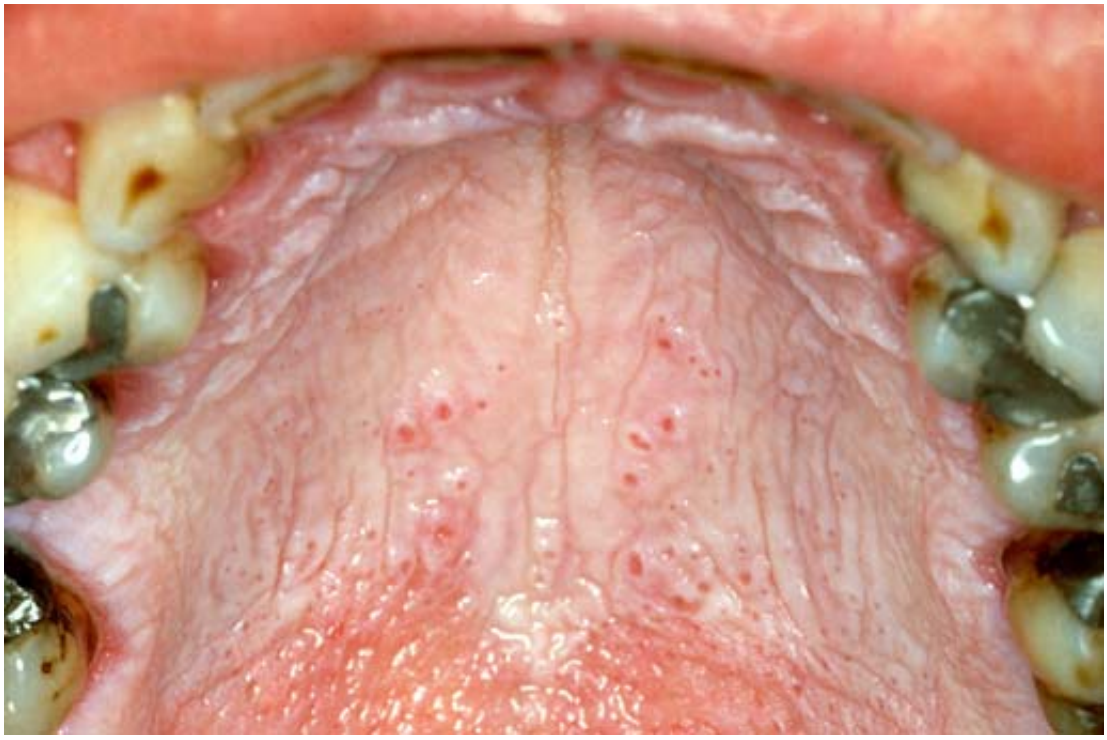

Figure 8. Smokers palate in a pipe smoker.

Further reading

1 World Health Organization. Addressing the Worldwide Tobacco Epidemic through Effective Evidence-Based Treatment. Expert Meeting March1999, Rochester, Minnesota, USA. Tobacco Free Initiative, WHO 2000.

2 Tomar SL, Asma S. Smoking-attributable periodontitis in the United States; findings from NHANES III. J Periodontol 2000; 71: 743-751.

3 EU Working Group on Tobacco and Oral Health. Meeting Report. Oral Dis 1998; 4: 464-467.

4 Warnakulasuriya S. Effectiveness of tobacco councelling in the dental office. J Dent Education 2002; 66: 1079-1087.

5 Schepman KP, Bezemer PD, van der Meij EH, Smeele LE, van der Waal I. Tobacco usage in relation to the anatomical site of oral leukoplakia. Oral Diseases 2001; 7: 25-27.

6 Smith SE, Warnakulasuriya KAAS, Feyeraband C, Belcher M, Cooper DJ, Johnson NW. A smoking cessation programme conducted through dental practices in the UK. Br Dent J 1998; 185: 299-303.

Links

1. Tobacco or health: a global status report
http://www.cdc.gov/tobacco/who/

2. Tobacco Cessation Guideline
http://www.surgeongeneral.gov/tobacco/