Alcohol and Oral Health

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Abstract: According to the World Health Organization, there are almost two billion people worldwide who consume alcohol, which is the most common drug of abuse, and almost 80 million with diagnosable alcohol abuse disorders (WHO 2002, 2004). Excessive alcohol consumption is causally related to more than 60 different medical conditions, some of them, such as suicide, homicide and different forms of accidents, are acute, while other conditions, such as liver cirrhosis, chronic pancreatitis, haemorrhagic stroke and various forms of cancer, are chronic consequences of alcohol use (40,41). Dental erosion is also associated with frequent alcohol consumption, because of precipitation of salivary protein-rich proteins caused by polyphenols present in most alcoholic drinks, the high concentration of organic and inorganic acids and the habit of keeping the alcoholic drink in the mouth.

Keywords: alcohol, alcoholism, oral disease, public health

1. Introduction

The term “alcohol” as used in medicine and by the lay public typically applies to ethanol, rather than other alcohols. Ethanol (CH₃CH₂OH) is therefore the active ingredient in alcoholic beverages. For consumption purposes it is produced by fermentation of carbohydrates, such as simple carbohydrates in fruit and starch (which is previously hydrolysed during a process called malting) in grains, by yeasts. Spirits, such as whisky, brandy and vodka are produced by distillation of fermented products. Carbohydrate fermentation is incomplete in beer and complete in wine, with resulting alcohol contents between 3–8% and 7–18% by volume, respectively. Distilled products, such as liqueurs and spirits, are 30% or greater alcohol by volume (4).

All alcohols are toxic, but ethanol is less toxic because the body can metabolize it rapidly. The various alcoholic drinks also contain substances that interact synergistically or antagonistically with ethanol. The limit discriminating between moderate and heavy alcohol drinking has been generally set at 40 g/day and is defined as tolerable upper alcohol intake level (TUAL) (6). Nevertheless, national drinking guidelines vary from country to country. For example, in the UK the recommended alcohol consumption level is of ≤2–3 AU/day for women (the number of AU in alcohol, alcoholism, oral disease, public health

2. Systemic effects of alcohol, influencing orofacial health: Caries and Periodontal disease

Chronic alcoholics may neglect themselves as they are preoccupied with their addiction and they may have very poor dental hygiene. Alcoholics have a high incidence of decayed missing filled teeth (DMF) and also more missing teeth compared to non-alcoholics(45). Alcoholics have an increased rate of chronic, advanced generalized periodontitis with inflamed gingivae, blunting of the interdental papillae and deep pocketing with associated bone loss. Alcoholics have more absent, traumatized, major cavitated and extracted teeth. Addicts had a worse severity index and dental index (based on both the number and severity of decayed, missing and filled teeth; 13.13 versus 4.74) (3,35) Significantly fewer teeth and more caries are found in alcoholics and there is a tendency for alcoholics to have more endodontically treated teeth than controls, but no difference in the number of periapical lesions in endodontically treated teeth. Horizontal bone loss and the presence of calculus is more frequent in alcoholic men than in alcoholic women (20,21). Significantly more horizontal bone loss was observed in alcoholic non-smokers than in non-alcoholic non-smokers. In the non-smoking groups, alcoholics had significantly more periodontal destruction than did non-smoking controls (15). Age, low income, low education level, smoking and alcohol abuse seemed to be risk markers for periodontal destruction (30, 49,50). In another study, measures of oral hygiene, dental care and periodontal parameters were significantly worse and the number of teeth requiring treatment was higher in alcoholics with or without cirrhosis than in healthy subjects and in nonalcoholic patients with cirrhosis. Alcoholics had fewer teeth and more cavities than patients without alcohol abuse and healthy controls. The dental and periodontal status of patients with non-alcoholic cirrhosis did not differ from the control group (2).

Most ethanol, rapidly absorbed through the gastric and duodenal mucosa, is metabolized by the liver, with a small fraction also metabolized by oral and other mucousa of the upper digestive tract (20). The enzyme acetaldehyde dehydrogenase (ADH) catalyses ethanol oxidation to cetaldehyde, which is oxidized into non-toxic acetate by the enzyme acetaldehyde dehydrogenase (ALDH). In turn, acetate is oxidized into fatty acids, carbon dioxide and water. Ethanol increases membrane permeability of the oral mucosa epithelial cells, thus promoting the penetration of other tobacco carcinogens such as nitrosornicotine (23). Alcohol drinking is also strongly associated with the risk of development of liver cirrhosis, which may result in impaired metabolism of carcinogens, and in impaired immunity. A diet poor in fruits and vegetables is also typical of heavy drinkers (47).
3. Direct effects of alcohol influencing orofacial health: Enamel erosion and Dry mouth

Several intrinsic factors are involved in the development of dental erosion, such as salivary flow rate, buffering capacity and composition, pellicle formation, tooth composition, and extrinsic factors, such as chemical (pH, titratable acidity, phosphate and calcium concentration, fluoride content of the material in contact with the tooth) and behavioural (eating and drinking habits, lifestyle, excessive consumption of acids) factors (44,50). Although low pH plays a crucial role in determining tooth wear and the evidence that acidic foodstuffs and beverages play a crucial role in the development of erosion is convincing, the density of acids in the food/beverage, that is, the titratable acidity, is more important than the pH itself, as well as other behavioural factors, such as the contact time between the acidic food/beverage and the dental surfaces (25,49). The pH of most alcoholic beverages is acidic, with values around 4, and the concentration of organic and inorganic acids is high. Indeed, it is important that the pH of incompletely fermented drinks, such as beer and wine, is acidic because it helps prevent contamination by other microorganisms, while the concentration of carbonic acid in alcopops (and in cocktails) is high because of the presence of soft drinks or fruit juices in the beverage (20). Most alcoholic beverages are involved in determining dental erosion, but the major role is for alcopops (32,37,38,39) and cocktails (10,34), which combine the erosive potentials of primary alcoholic beverages and of soft drinks and/or fruit juices, at the same time. Among primary drinks, white wines are more erosive than red wines, because of higher concentration of titratable acids (36), while beers (22,31) and ciders (36) have a moderate erosive potential. The astringency of alcoholic beverages is likely to be another factor promoting tooth wear. Indeed, this taste which is typical of some alcoholic beverages is due to presence of high levels of polyphenols, mostly tannins, which bind salivary proteins, such as proteins and mucopolysaccharides, causing their precipitation, with consequent sensation of astringency owing to loss of lubrication of the oral mucosa and teeth and simultaneous decreased protection of teeth from acids (12).

Discomfort of the teeth is acknowledged to be a problem for those people making or tasting wines regularly which, because of wine’s acidity, may have a deleterious erosive effect on teeth (8,9,16,17,27,28,29,46). In fact, dental erosions are so frequent among wine merchants (10), winetasters (48) and winemakers in general (9), who keep wine in their mouth for long time, as to be considered an occupational hazard. The erosive effects vary between wines (8). Riesling style wine is more erosive than champagne style and both are more than claret (29). Most white wines tested were at least as erosive as orange juice, while some wines, notably the cava, were significantly more erosive than orange juice. Red wines may also be erosive (7,24) and all ciders tested are acidic and had considerable erosive potential in vitro which was broadly similar to that of orange juice (36). Many commercially available designer drinks also have considerable erosive potential (35) as do alcoholic soft drinks (32) such as Hooch alcoholic lemonade (37). Fluoride gels or varnishes may significantly reduce such enamel erosion (19). Dental erosion is strongly associated with drinking behaviour. In fact, it is more frequent and severe among those whose alcohol consumption is continuous rather than in the form of episodic binges (10,33,38) and it is affected by the time the drink is kept into mouth before swallowing. Several epidemiologic studies report a time-dependent association between chronic alcoholism and dental erosion, independently of socioeconomic status, with prevalence values as high as 50% (3,18,42). The typical dental erosions of heavy alcohol drinkers generally affect the palatal surfaces of the upper anterior teeth (43). Summarizing, the evidence of the association between alcoholic beverage intake and dental erosion is convincing, with secondary drinks showing greater erosive potential than primary drinks and with an important role of drinking behaviour, particularly those habits characterized by frequent intake and long retention of the drink in the oral cavity.

Dental erosion was related to duration of alcoholism irrespective of confounding control of dental health behaviour and social situation (18). Tooth erosion may arise from gastritis and reflux (48) and oral malodour may be exacerbated . The teeth in alcoholic patients have significantly more wear than age- and sex-matched controls. Tooth wear is most marked in males and in those whose alcohol consumption was continuous rather than in the form of episodic binges. Wear appeared to be erosive in nature and in 40% of the sample it affected the palatal surfaces of the upper anterior teeth. (38).

4. Dry Mouth

The effects of alcohol on salivation have not been clearly defined (5). The submandibular gland showed a proportional increase in adiposity and reduction in fibrovascular tissues but no noticeable reduction in its acinar proportional volume (39). The salivary findings of increased flow rate, protein and amylase levels indicate that hypertrophy and increased acinar function may be a component of the parotid enlargement and that, furthermore, a fatty replacement of functional gland tissue is probably not involved. (1,13). Reduction in parotid saliva flow rate was associated with a statistically significant decrease in total protein and amylase secretion in this group of patients. (14).

5. Conclusion

High alcohol consumption has a deep impact on oral health. Some systemic alcohol-related conditions may indirectly affect oral health (40,41). These are the cases of missing teeth caused by caries or periodontal disease among chronic alcoholics, who generally neglect themselves and have poor oral hygiene; of tooth erosion arising from gastric reflux; of stomatitis caused by several micronutrient deficiencies; of sialosis and dry mouth with impact on caries development.

In addition, high alcohol consumption has a direct impact on oral health. The principal effect of heavy drinking is the high risk of cancer of the oral mucosa, which could also be increased by the use of alcohol-containing mouthwashes.
References

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