Alcoholism and its implications for the dental team, an update and review of the literature

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Abstract

Precis: This Literature review is intended to provide dental practitioners with an update of the implications that Alcoholism poses to them.

Statement of the problem: Alcoholism raises several distinct problems for dental professionals.

Purpose of the study: The focus of this review has been to primarily identify and critically appraise the associations between alcoholism and the various difficulties that have been derived from the literature.

Materials and methods: A structured review of the literature was undertaken using PubMed, Google Scholar and the Cochrane library, additional searching of reference lists was also undertaken. A number of articles were critically analyzed which included Cochrane and systematic reviews, meta-analyses and a number of cross-sectional studies. The data was compared under several headings and tabulated in certain instances.

Results: Alcoholism raises various implications that dental professionals should be aware of including increased incidences of caries, periodontal disease, pathological tooth wear & oral cancer. Chronic Alcohol intake interacts with the pharmacodynamics and kinetics of many routinely prescribed medications in routine dental practice. In addition, there are problems related to access to care and during acute episodes of intoxication abusers may participate in antisocial behavior in the healthcare environment.

Conclusion: Alcoholism has several implications for the dental team, however, dentists who are familiar with the manifestations of the illness, as well as the challenges raised in dental practice can confidently offer these patients a full range of dental treatment.

Introduction

The American Medical Association defines alcoholism as an illness characterised by significant impairment (a type of drug dependence) that is directly associated with persistent and excessive use of alcohol [1]. Alcoholism is a broad term that envelopes a variety of Alcohol Use Disorders (AUD). The World Health Organization (WHO) estimates that approximately 3.3 million deaths globally are a result of harmful drinking and lists alcohol consumption as the third largest risk factor for disease and disability [2].

Alcoholism raises several distinct difficulties for dental patients. Alcoholics may exhibit greater levels of caries, periodontal disease and Pathological Tooth Wear (PTW) [3-5]. Management of these conditions is more difficult due to a lower level of compliance observed in alcoholics [6]. The alcoholic dental patient has an increased risk of developing oral cancer [7]. In alcoholic liver disease, there is an increased risk of prolonged bleeding after invasive dental procedures, because of a reduction in production of clotting factors by the liver [8]. Difficulties exist when prescribing medications for these patients, as alcohol interacts with most of the frequently prescribed pharmaceuticals in dentistry. In addition, chronic alcoholism can alter the pharmacodynamics of some of these medications [9]. During episodes of acute intoxication, abusers may participate in antisocial and sometimes violent behaviour, which can be challenging for the dentist to manage, all the while obstructing the fruition of a positive and healthy relationship between dentist and patient [9].

The focus of this article has been to primarily identify and
critically appraise the associations between alcoholism and the various difficulties that have been described above.

**Alcoholism and saliva/salivary glands**

Sialadenosis (asymptomatic enlargement of the salivary glands) is observed frequently in alcoholics [10-12]. Depending on the study incidence varied from 30%-80% [13-17]. The precise mechanism is unknown but it is thought to be due to adipose infiltration (abnormal fat metabolism due to altered liver function) and acinar hypertrophy [18-22]. The morphological and histological change found could account for the reduction in Salivary Flow Rate (SFR) observed in alcoholic patients [18]. The reduction in SFR appears to be more prevalent in cirrhotic alcoholics but a reduction is also seen in non-cirrhotic alcoholics [23]. There is also an altered mineral, electrolyte and enzyme composition of saliva observed in these patients and such differences in composition may be in part contributory to the increased incidence of dental diseases in alcoholic patients [24-27].

**Alcoholism and caries**

Globally, reliable epidemiological data on dental caries and alcohol abusers are scarce, however a link between regular consumption of alcohol and caries has been established [3,28-30].

A common treatment for liver cirrhosis is diuretic drugs which can cause reduced SFR, also patients may suffer from Sjögren's syndrome as a result of primary biliary cirrhosis which may contribute to the development of caries [31,32]. An in vivo study on rats ingesting only an alcoholic diet showed a higher count of Streptococcus mutans. The precise mechanism is unknown but may be related to an increased rate of production of acetaldehyde [33].

Interestingly, a considerable number of studies found lower or comparable caries experience when compared to control groups, national averages and other substance abusers [34-40]. Summaries of the main findings of these studies are included in table 1. Possible explanations for the decreased caries rate observed are explored in figure 1.
A fundamental flaw in many of the studies cited in table 1 is that information on participants’ alcohol consumption was based on self-reporting which cannot be validated independently (However, a detailed study comparing self-reporting to dietary analysis interview found very little difference in the rates of consumption [41]). Clinical findings were also subjective in nature and no appropriate radiographs were used in the diagnosis of caries. Cross-sectional studies also fundamentally do not allow temporal relationships to be formed between two variables.

**Alcoholism and periodontal disease**

Early associations between alcoholism and periodontal disease have been made but most of these studies attributed the higher incidence of periodontal disease due to poor oral hygiene (OH) practices [31,46]. More recent research may indicate that alcoholism is an independent risk factor for periodontal disease.

Alcoholics are more likely to have poor OH, this may be related to impaired motor activity as a result of alcoholism, use of a hard toothbrush, the alcoholic lifestyle or limited knowledge and access to dental care. Alcoholics may use a hard toothbrush to mask the alcoholic odor after consumption [6].

A systematic review including twelve studies on alcohol consumption and four studies on alcohol dependence concluded that there is insufficient evidence to support that there is a relationship between alcoholism and periodontal disease, however sufficient evidence exists to suggest alcohol consumption is a risk indicator for periodontitis. Meta-analysis could not be performed as each of the studies used different measures to clinically assess alcohol consumption/dependence and periodontal disease [47]. However, a recent meta-analysis concluded that there was a linear dose-response relationship between alcohol consumption and risk of periodontal disease (Figure 2). Eleven of the Eighteen studies showed a statistically significant correlation between alcohol consumption and periodontitis. This risk, when stratified was doubled in females compared to a 25% increase in men. The analysis was based on a large number of studies (18) and the studies were adjusted for confounding variables which did not seem to have a major effect on the results. However, differences between the studies contributed to a large amount of heterogeneity, this high level of heterogeneity may have not been completely explained by meta-regression of the sub-group analysis. The combination of data from both cross-sectional and cohort studies could mean an overestimation of Relative Risk (RR), as the cohort studies showed lower risk. There was little information included on type of beverage and associated risk. A number of the studies did not adjust for OH [4,48].

Few studies have found that no relationship exists between the two variables [49,50]. Kongstad, et al. [51], suggested a possible antimicrobial effect of ethanol similar to that of alcohol containing mouthrinses and the possible beneficial effect of wine as men consuming wine and spirits had lower odds-ratios for bleeding on probing [51]. Chronic alcoholism as indicated by Gamma-Glutamyl Transpeptidase (GGTP) levels was positively associated with increased plaque levels in one study, suggesting that for alcohol to exert its antibacterial effect it requires more time than the mere act of swallowing and drinking. This study used GGTP as a biological marker for alcohol abuse in order to alleviate the biases involved with self-reporting [52].

Tezal, et al. [53], found that alcoholics harbored high levels of Bacteroides forsythus and Porphyromonas gingivalis. In an attempt to quantify periodontal pathogens and cytokines in alcoholics a study found that alcohol dependents with periodontal disease had a higher frequency of some periodontal pathogens namely Prevotella intermedia, Eikenella corrodens and Fusobacterium nucleatum [54]. An increased production of cytokines has been observed in these patients suggesting that cytokines may be regulated as a result of alcohol induced damage to the periodontium [52,54,55]. When smokers were excluded from this group the microbiological and immunological results were similar suggesting an independent effect of alcohol. Novacek, et al., highlighted that dental aggregates of bacteria could be a potential source of liver transplantation failure in patients with advanced cirrhosis [31].

Shimazaki, et al. [56], postulated that alcoholism increases the risk of periodontitis when drinking causes a buildup of acetaldehyde, the precise mechanism was not explored.

**Alcoholism and dental implants**

Alcoholism is not considered a risk indicator for peri-implantitis [57,58]. However, alcohol consumption chronic or otherwise has been shown in vitro and in animal studies to negatively impact osteointegration and osteoinduction of dental implants [59-64].
Alcoholism and oral cancer

Ethanol is a well-established carcinogen [65-69] and table 2 identifies the plausible biological mechanisms involved.

A recent meta-analysis reviewing 43 case-control and two cohort studies (17000 cases) provided more definite quantification of Oral and Pharyngeal Cancer (OPC) risk for heavy alcohol drinkers (≥ to 4 drinks/day), the overall Relative Risk (RR) for heavy drinking was 5.24 (95% Confidence Interval, 4.36-6.30) [90]. Figure 3 demonstrates the dose-response relationship observed. Further analysis was performed using the same set of studies in an attempt to find particular subsites more at risk, the authors concluding that the RR was greater for pharyngeal cancer when compared to oral cancer [91]. Bagnardi, et al. [92], used previous analyses to create site specific dose-response relationships for all types of cancer, finding that OPC had the highest relative risk when compared to cancer of other parts of the body. An additional 5 publications were included in a more recent meta-analysis attempted to quantify risk by sex, smoking status and other potential confounders. RR was similar between men and women, risk was present in the absence of tobacco smoking (however the association was weaker in non-smokers than in smokers, particularly in those who consumed heavy doses of alcohol, suggesting smoking increases risk in a multiplicative fashion) [7]. Many of the studies included in the analysis demonstrate that the interaction between the two risk factors could be more than multiplicative [93-96]. Recent evidence from the International Head and Neck Cancer Epidemiology Consortium supports this finding [97]. There was little variation in geographic pattern of risk and type of alcoholic beverage consumed suggesting that ethanol and its metabolites are the primary carcinogens conveying this increased risk. The most frequently consumed alcoholic beverages tend to be associated with the highest risk of OPC [65,98].

The above analyses suffer from many drawbacks, those studies included relating to heavy drinking were prone to heterogeneity and the use of random-effect models may account for only part of this heterogeneity, meaning the dose heterogeneity and the use of random-effect models may not be more than multiplicative [93-96]. Recent evidence from the International Head and Neck Cancer Epidemiology Consortium supports this finding [97]. There was little variation in geographic pattern of risk and type of alcoholic beverage consumed suggesting that ethanol and its metabolites are the primary carcinogens conveying this increased risk. The most frequently consumed alcoholic beverages tend to be associated with the highest risk of OPC [65,98].

Whether or not alcohol cessation has an effect on OPC risk requires further investigation as it is difficult to assess without the influence of potential confounders such as smoking, studies with larger sample sizes are required [109]. Larger studies are also required to investigate the impact of alcohol consumption on OPC survival. A systematic review is currently ongoing to investigate the effect of alcohol cessation on oral dysplasia and head and neck cancer [110].

Alcoholism and pathological tooth wear (PTW):

Alcoholism has also been implicated as a risk factor for PTW. Evidence and possible mechanisms are summarized in figure 4.

Pharmacological management of the alcoholic dental patient

There are several recognized adverse interactions
### Table 3: Adverse Interactions between Alcohol/Chronic alcoholism and medications used in dentistry. Adapted from Friedlander, et al. [9].

<table>
<thead>
<tr>
<th>Medication</th>
<th>Adverse interaction with alcohol</th>
<th>Chronic Alcoholism</th>
<th>Dentists' Actions</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracetamol</td>
<td>Hepatotoxicity may occur because of toxic acetaminophen metabolites and glutathione depletion [9].</td>
<td>Increased risk of hepatotoxicity in alcoholic liver disease [9].</td>
<td>Counsel patient about the risk of long term alcohol use and acetaminophen toxicity [9].</td>
<td>A systematic review [130] and meta-analysis [131] conclude that therapeutic dosing of paracetamol is not associated with liver damage in alcohol users. This conclusion is supported by several other studies [132,133]. However there have been cases of acute liver toxicity even at therapeutic doses [134] and been reports of acute interstitial nephritis in acetaminophen overdose in patients with acute/chronic alcohol toxicity [135,136].</td>
</tr>
<tr>
<td>Nonsteroidal Anti-inflammatory drugs (NSAIDs)</td>
<td>Excessive bleeding may occur because of aspirin-induced prolongation of bleeding time. Increased risk of mucosal ulceration. Renal toxicity with binge drinking with ibuprofen [9].</td>
<td>Possible increased risk of Gastrointestinal Bleeding (GIB).</td>
<td>Counsel patient to discontinue alcohol use during analgesic therapy [9].</td>
<td>It is unclear from the literature whether chronic alcohol consumption affects risk estimation of GIB [137-139]. Many studies have found increased risk and incidence of GIB with greater amounts of alcohol consumption [140-142] this is further supported by the fact that alcohol has been shown to cause damage to the gastric epithelium [143,144] and alcohol induced gastric damage is enhanced by the presence of NSAIDs in dogs [145].</td>
</tr>
<tr>
<td>Cephalosporins (some)</td>
<td>Cephalosporin and alcohol may interact to produce a cephalosporin induced disulfiram reaction (CIDLR) presenting as facial flushing, nausea or vomiting and in severe reactions angioedema, hypotension, shock, or death [9].</td>
<td>Not explored in the literature.</td>
<td>Avoid use of cefoperazone and cefotetan [9].</td>
<td>Evidence based mainly on case reports [146-149].</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Decreased absorption of erythromycin, with consequent decrease in effectiveness possibly due to an increase in gastric emptying [9].</td>
<td>Not explored in the literature.</td>
<td>Counsel patient to discontinue alcohol use during erythromycin therapy [9].</td>
<td>Evidence based from in vivo studies [150-152].</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>A disulfiram effect may occur, permitting the accumulation of acetaldehyde, leading to facial flushing, headache, palpitation and nausea [9].</td>
<td>Not explored in the literature.</td>
<td>Counsel patient to discontinue alcohol use during metronidazole therapy [9].</td>
<td>Evidence based from case reports, animal and experimental studies [153-158].</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Increased absorption and increased plasma concentration in healthy subjects after acute ingestion of ethanol [9].</td>
<td>Diminished effectiveness in long term alcoholics because of induction of metabolizing enzymes. Preexisting liver disease such as alcoholic liver disease has been associated with increased risk of developing tetracycline induced hepatotoxicity [159].</td>
<td>Counsel patient to discontinue alcohol use during tetracycline therapy [9].</td>
<td>Evidence based from case reports and analytic studies [159,160].</td>
</tr>
<tr>
<td>Ketoconazole</td>
<td>A disulfiram effect may occur, permitting the accumulation of acetaldehyde, leading to facial flushing, headache, palpitation and nausea [9].</td>
<td>May increase risk of liver damage [9].</td>
<td>Counsel patient to discontinue alcohol use during ketoconazole therapy [9].</td>
<td>Only 1 case report found in the literature [161].</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Fatal poisoning can occur with concomitant use of benzodiazepines and alcohol as well as increased sedative effects [162].</td>
<td>Diminished effectiveness in long-term alcoholics because of cellular tolerance to CNS depression [9], increased induction of CYP2E1 enzyme [165], or both.</td>
<td>Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol during treatment.</td>
<td>An extensive review [162] as well as case reports/animal and experimental studies confirm a toxicological interaction [163-167]. Few studies (case reports) evaluate the effect of chronic alcoholism on benzodiazepine administration and most report diminished effectiveness [168-171].</td>
</tr>
<tr>
<td>Chlora Hydrate</td>
<td>Concurrent use may significantly increase CNS depressant effects [9].</td>
<td>Not explored in the literature.</td>
<td>Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol during treatment [9].</td>
<td>Evidence based on early case reports [172-174].</td>
</tr>
<tr>
<td>Opioids</td>
<td>Sedative effects are markedly increased. Increased respiratory depression [9].</td>
<td>Not explored in the literature.</td>
<td>Initially decrease the usual dosage of medication and observe patient for CNS depression. Counsel patient to discontinue alcohol during treatment [9].</td>
<td>Evidence based on case reports, an in vivo and experimental human study [175-177].</td>
</tr>
<tr>
<td>Warfarin</td>
<td>There is only one case report evaluating possible interaction between warfarin and ethanol suggesting alcohol consumption daily may decrease the effectiveness of warfarin reducing the International Normalised Ratio (INR) [178]. A recent study suggests that alcoholism is risk factor for major bleeding in patients on warfarin therapy [179].</td>
<td>Evidence based from case reports, animal and experimental studies [153-158].</td>
<td></td>
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<tr>
<td>Other Beta lactams</td>
<td>There does not appear to be a clinical significant interaction between alcohol and other B-lactams but the rate of absorption appears to be increased [180].</td>
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</tr>
</tbody>
</table>
between alcohol and medications used in dentistry. In addition, chronic alcohol abuse alters the pharmacodynamics of certain pharmaceuticals [9]. These interactions and specific recommendations are summarized in table 3.

Many of the pharmacotherapies used for alcohol dependence have oral side effects for example Naltrexone treatment has been associated with xerostomia, cold sores, headaches and sinusitis raising additional difficulties for these patients [181]. Patients using disulfiram should not use alcohol containing mouth rinses because of potentially adverse reactions [9].

**Alcoholism and bleeding**

Thrombocytopenia as a result of alcohol consumption is usually transient and platelet counts generally return to reference ranges within 4-8 weeks [182].

There is reduced synthesis of coagulation proteins in alcoholic liver disease. As a result, Prothrombin-time (PT) and Activated-Partial-Thromboplastin time (APTT) may be prolonged [183,184]. Hematological investigations are not required in alcoholics without a positive bleeding history. A Full Blood Count (FBC) and coagulation screening may be indicated in patients where there is a positive bleeding history prior to invasive dental/oral surgical procedures. If prolonged bleeding occurs it can be successfully managed using local measures, an emphasis should be placed on gathering an accurate bleeding history from the alcoholic patient. These guidelines are based on an audit completed in the UK, evidence-based guidelines may be required on the hematological management of the alcoholic dental patient [8,185,186].

**Alcoholism and access to dental care**

Few studies have evaluated access to dental care in alcoholics, however a low level of access would be expected because of depression and psychiatric disorders coexisting in these patients. Interestingly, two studies found access to dental care which was comparable to national levels, this high level of access may be because participants from both studies were selected from alcohol treatment centres [6,40].

Dentists have a professional duty to enquire about alcohol intake. It is unlikely that an alcoholic will disclose their alcohol consumption because of the stigma associated with alcoholism. To overcome this, there have been various questionnaires that can be used by dental and medical professionals in practice that identify alcohol dependence and dentists should refer patients with suspected alcohol dependence to their general medical practitioner [209-211]. An Example of one of these questionnaires (AUDIT (Alcohol Use Disorder Identification Test)) that may be used in primary care can be seen in figure 5 [214,215]. In addition, it has been shown that brief interventions and motivational interviewing are effective in reducing alcohol consumption in primary care settings, [212,213] whether such interventions are feasible and practical in dental practice remains to be seen.

During episodes of intoxication, an abuser may act inappropriately, frequently get into arguments and violent behavior may ensue, a dentist will have to refuse treatment in
such situations. Dentists should appreciate that alcoholics are unreliable attenders as a result of the aberrant lifestyle many lead, mainly attending in acute pain. In addition, atrophy of several regions of the brain clinically correlate with deficits in judgement and decision making, in these situations the alcoholic dental patient is of questionable competence to consent for treatment [1,9].

Conclusion

There are many considerations to take into account when treating the alcoholic dental patient. These patients are at a greater risk of developing oral/dental diseases, namely PTW, periodontal disease and OPC. Poor compliance and limited access to dental care confounds issues, allowing conditions to deteriorate further and making management more challenging. Medical management of these patients is more difficult, as alterations in coagulation, drug metabolism, liver function and bone remodeling may be encountered as well as gastrointestinal and central nervous system disturbances. Dentists are professionally obligated to enquire about alcohol consumption and are well positioned to offer appropriate referrals to primary care physicians.

Larger scale epidemiological and interventional studies are needed to explore the effect of alcoholism on caries and sleep bruxism, as well as supportive experimental studies to explore the mechanisms involved.

To conclude, alcoholism among patients raises significant difficulties among dental patients, however, dentists who are familiar with the manifestations of the illness, as well as the challenges raised in dental practice can confidently offer these patients a full range of dental treatment.

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