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Association Between Alcohol Consumption and Periodontal Pocket Formation

Arina Shafia
Universitas Islam Sultan Agung Semarang, Indonesia
E-mail: drg.arinashf@unissula.ac.id

KEYWORDS

alcohol consumption; periodontal pocket formation; periodontitis; clinical attachment loss

ABSTRACT

Background: Periodontal disease is one of the most prevalent chronic diseases affecting the oral cavity worldwide. Various risk factors contribute to the development of periodontal disease, one of which is alcohol consumption. Excessive alcohol intake is widely recognized as a major public health problem and has been associated with systemic conditions such as liver disease, cardiovascular disorders, and behavioral and mental health problems. Alcohol abuse has also been reported to impair host immune responses by reducing neutrophil, macrophage, and T-cell function, thereby increasing susceptibility to infection. Consequently, alcohol consumption may contribute to the development of periodontal pockets.

Objective: This literature review aimed to summarize current evidence regarding the association between alcohol consumption and the risk of periodontal pocket formation.

Methods: A narrative literature review was conducted by searching the PubMed, Scopus, and Google Scholar databases for peer-reviewed articles published between 2008 and 2025. The search combined the terms "alcohol consumption," "periodontal pocket," "periodontitis," and "clinical attachment loss." Observational, longitudinal, cohort, and cross-sectional studies, as well as systematic reviews and meta-analyses, were included, and their findings were synthesized descriptively.

Results: Seven primary clinical studies were examined, together with supporting mechanistic and review evidence. Two studies reported no consistent association between light-to-moderate alcohol consumption and periodontal pocket development, whereas five reported that heavy or long-term alcohol consumption was associated with increased periodontal pocket depth and clinical attachment loss, most notably among heavy drinkers and smokers.

Conclusion: This review demonstrates that excessive alcohol consumption is associated with periodontitis-related clinical manifestations, including periodontal pocket formation and clinical attachment loss.

Introduction

A periodontal pocket is a gingival sulcus (a narrow V-shaped space) that has pathologically deepened. Periodontal pocket formation, defined as the pathological deepening of the gingival sulcus, represents one of the main clinical features of periodontal disease (Bosshardt, 2018). In addition to periodontal pocket formation, periodontal disease is also characterized by gingival inflammation and gingival recession (Febrina, Z., & Putri, 2014).

Periodontal disease is one of the most common chronic diseases affecting the oral cavity worldwide. Several risk factors contribute to the development of periodontal disease, including tobacco smoking, uncontrolled diabetes mellitus, and alcohol consumption (Sankaranarayanan et al., 2020).

Globally, periodontal disease represents a substantial public health burden. According to the World Health Organization Global Oral Health Status Report (2022), severe periodontal disease affects an estimated one billion people worldwide and is a leading cause of tooth loss in adults. Beyond its impact on oral function and quality of life, periodontitis has been linked to several systemic non-communicable diseases, including diabetes mellitus and cardiovascular disease, and it imposes considerable direct and indirect economic costs on health systems (Kinane et al., 2017). Because many of its principal risk factors such as tobacco use, poor oral hygiene, and alcohol consumption are modifiable, periodontal disease is regarded as a largely preventable condition.

According to the Organization, (2022), alcohol abuse is the third leading lifestyle-related risk factor globally. Alcohol acts as a predisposing factor for more than 200 diseases and is considered a primary cause of approximately 30 conditions. It is estimated that alcohol consumption contributes to around 3.3 million deaths annually worldwide (Sankaranarayanan et al., 2020). Alcohol consumption is a widespread and modifiable behaviour of particular relevance to this burden. Harmful alcohol use is common in many populations, including in South-East Asian countries where the overall burden of oral disease is among the highest globally. This makes the potential contribution of alcohol to periodontal tissue destruction a clinically and epidemiologically important question that warrants a careful synthesis of the available evidence.

Alcohol abuse is suspected to contribute to the development of periodontal disease due to its effects on immune suppression and gingival irritation. Alcohol-induced dehydration may also lead to bacterial and plaque accumulation as a result of reduced salivary flow. Lack of awareness of early gingival disease symptoms can allow disease progression to more severe conditions, ultimately leading to periodontal disease. Individuals with alcohol dependence have a higher risk of developing chronic periodontitis, which is associated with gingival inflammation, blunting of the interdental papillae, deep periodontal pockets, and accompanying alveolar bone loss (Khairnar MR, Wadgave U, 2017).

The term periodontal disease encompasses a wide range of chronic inflammatory conditions affecting the gingiva (gums), alveolar bone, and periodontal ligament (collagen fibers of connective tissue that anchor teeth to the alveolar bone). Periodontal disease begins as gingivitis, a localized inflammation of the gingiva initiated by bacteria within dental plaque, a microbial biofilm that forms on the tooth and gingival surfaces. Chronic periodontitis occurs when untreated gingivitis progresses to the destruction of gingival tissue, alveolar bone, and periodontal ligament, leading to the formation of deep periodontal pockets, which are characteristic features of the disease and may ultimately result in tooth loss (Kinane et al., 2017).

A periodontal pocket is defined as a pathologically deepened gingival sulcus surrounding a tooth at the gingival margin. Thus, the space formed between the pathologically detached gingiva and the tooth is referred to as a periodontal pocket. A gingival sulcus depth of up to 0.5 mm is considered clinically healthy (Bosshardt, 2018). When the original sulcular depth

increases concurrently with apical migration of the junctional epithelium, pathological changes have occurred. To be clinically diagnosed as a true periodontal pocket, a probing depth of 4 mm or more must be demonstrated. At this stage, most gingival fibers that initially attached the gingival tissue to the tooth are permanently destroyed. In the early stages, symptoms are usually absent and patients are often unaware of disease progression; however, as inflammation worsens, bleeding from the periodontal pocket may occur (Solanki, 2012).

Based on the location of the pocket base relative to the underlying alveolar bone, periodontal pockets can be classified into two types (Newman et al., 2018). The suprabony pocket is characterized by the base of the pocket being located coronal to the crest of the alveolar bone. In contrast, the infrabony pocket is characterized by the base of the pocket being located apical to the crest of the alveolar bone, with the lateral wall of the pocket lying between the tooth surface and the alveolar bone (Newman et al., 2018) (Figure 1).

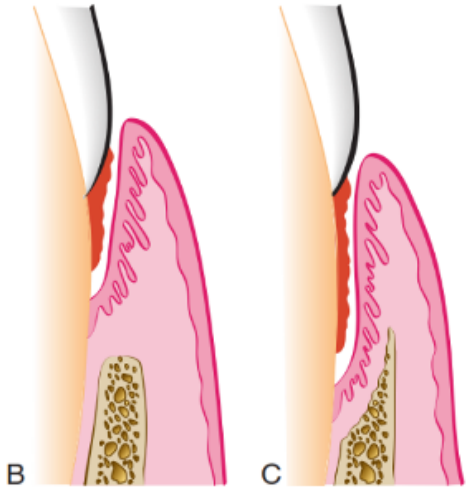

Figure 1. Types of periodontal pockets: (A) Suprabony pocket and (B) Infrabony pocket (Newman et al., 2018).

Based on tooth surface involvement, periodontal pockets are further classified into three types (Newman et al., 2018). A simple pocket involves only one surface of a tooth. A compound pocket involves two or more tooth surfaces; these pockets may have uniform or varying depths, with the base of the pocket directly connected to the marginal gingiva of each affected surface. A complex pocket is a spiral-shaped pocket originating from one tooth surface and extending around the tooth to involve one or more additional surfaces; in complex pockets, the base of the pocket does not directly communicate with the gingival margin, and this type is most commonly found in furcation areas (Newman et al., 2018) (Figure 2).

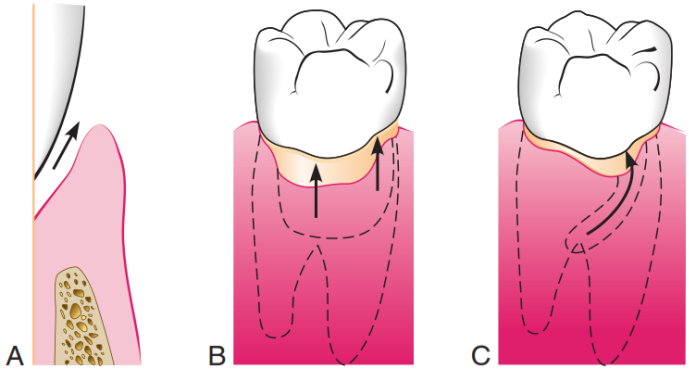

Figure 2. Classification of periodontal pockets based on involved tooth surfaces: (A) Simple pocket, (B) Compound pocket, and (C) Complex pocket (Newman et al., 2018).

Alcohol consumption is considered one of the major risk factors for numerous diseases, disabilities, and mortality. Alcohol may cause both immediate and long-term effects. Immediate effects include accidental injuries, stillbirth, intoxication leading to loss of consciousness, hypotension, hypothermia, coma, respiratory depression, or death. Long-term effects include neurological disorders, cardiovascular diseases, psychiatric problems, social issues, cancer, liver disease, and digestive disorders (Khairnar MR, Wadgave U, 2017).

In addition to its impact on general health, alcohol consumption has also been associated with oral health, particularly periodontitis. Periodontitis is a multifactorial inflammatory disease of infectious origin that results in destruction of the tooth-supporting structures. Periodontal inflammation may lead to gingival damage, clinical attachment loss (CAL), alveolar bone loss, periodontal pocket formation, tooth mobility, and pathological tooth migration (Shah et al., 2018). The biological mechanisms underlying the relationship between alcohol consumption and the risk of periodontitis remain unclear. According to a review and meta-analysis by Wang et al., (2016), several plausible biological explanations have been proposed.

First, previous studies have demonstrated that periodontal disease is associated with impaired neutrophil phagocytosis; alcohol may weaken neutrophil function, promote bacterial overgrowth, and increase bacterial penetration, thereby inducing periodontal inflammation (Wang et al., 2016). Second, alcohol intake may exert toxic effects on periodontal tissues and reduce the production of monocyte-derived inflammatory cytokines that could otherwise be beneficial in controlling microbial proliferation (Wang et al., 2016). In addition, previous research has shown that excessive alcohol consumption may affect host immune responses by reducing neutrophil, macrophage, and T-cell function, thereby increasing susceptibility to infection (Wagner et al., 2017).

Alcohol is metabolized in the liver prior to elimination from the body, producing high levels of reactive oxygen species (ROS). ROS, or free radicals, are highly reactive molecules capable of oxidizing various biomolecules essential for cellular and tissue function (Shah et al., 2018). Oxidative stress is suspected to play a role in the pathogenesis of periodontitis and associated tissue destruction. The majority of tissue damage in periodontitis is believed to result from the inflammatory and immune responses to bacterial plaque present at the gingival margin. Bacterial cell components and inflammatory cytokines subsequently lead to

recruitment and activation of hyperresponsive polymorphonuclear neutrophils (PMNs), thereby accelerating ROS production (Dahiya et al., 2013). Long-term alcohol consumption may also result in salivary gland enlargement, particularly of the parotid gland, a condition known as sialadenosis, which is associated with ethanol-induced peripheral neuropathy. This condition disrupts salivary gland metabolism and excretion. Reduced salivary secretion, combined with decreased buffering capacity and poor oral hygiene, may increase the risk of dental caries and gingival disease (Khairnar MR, Wadgave U, 2017).

Several previous studies have examined this relationship, yet their conclusions are not uniform. A meta-analysis of observational studies by Wang et al., (2016) reported a positive association between alcohol consumption and the risk of periodontitis, and a subsequent systematic review and meta-analysis by Pulikkotil et al., (2020) similarly concluded that alcohol consumption is associated with an increased risk of periodontitis. In contrast, an eleven-year follow-up study by Sankaranarayanan et al., (2019) did not detect a consistent association between alcohol-related variables and periodontal pocket development. More recently, a narrative review by Gandhi et al., (2024) concluded that alcohol is directly or indirectly associated with clinical attachment loss and pocket formation. These inconsistent findings underline the need for an updated synthesis.

Despite the existence of earlier reviews, the evidence base continues to evolve, and no consensus has been reached regarding whether, and to what extent, alcohol consumption independently promotes periodontal pocket formation. The present review is therefore timely: it consolidates both established and recent evidence (up to 2025), focuses specifically on periodontal pocket formation and clinical attachment loss rather than on periodontitis in general, and integrates the underlying biological mechanisms with the clinical findings. This focused synthesis constitutes the novelty of the review and addresses a gap left by broader prior analyses.

Accordingly, the objective of this literature review is to summarize and critically appraise the current evidence regarding the association between alcohol consumption and the risk of periodontal pocket formation, and to describe the biological mechanisms that may underlie this association. The findings are intended to benefit clinicians, who may use them to strengthen patient education and preventive counselling; researchers, who may identify remaining gaps for future investigation; and policymakers, who may consider alcohol as a modifiable risk factor within broader oral-health and non-communicable-disease prevention strategies. Clinically, recognizing alcohol consumption as a potential contributor to periodontal breakdown may support earlier risk identification and more comprehensive periodontal management.

Method

This study employed a literature review design by examining published scientific evidence regarding the association between alcohol consumption and periodontal pocket formation. The literature consisted of relevant scientific articles published in national and international journals, as well as reference books related to periodontology and alcohol consumption. The reviewed literature included observational studies, longitudinal studies, cohort studies, cross-sectional studies, systematic reviews, and meta-analyses investigating the effects of alcohol consumption on periodontal disease.

The collected literature was analyzed descriptively through the processes of identification, classification, comparison, and synthesis of the findings. The analysis focused on the relationship between alcohol consumption and the clinical manifestations of periodontal disease, particularly periodontal pocket formation, clinical attachment loss (CAL), and the biological mechanisms underlying these associations. The synthesized evidence was subsequently used to summarize the current scientific findings regarding the potential role of alcohol consumption in the development and progression of periodontal disease. More specifically, this review was conducted as a narrative literature review. A structured search of the electronic databases PubMed, Scopus, and Google Scholar was performed for articles published between 2008 and 2025. The search combined the following keywords using Boolean operators: ("alcohol consumption" OR "alcohol dependence" OR "alcohol misuse") AND ("periodontal pocket" OR "periodontitis" OR "periodontal disease" OR "clinical attachment loss"). The reference lists of relevant articles were screened manually to identify additional eligible studies.

Studies were eligible for inclusion if they were peer-reviewed articles published in English that reported original or synthesized evidence on the relationship between alcohol consumption and periodontal outcomes, particularly periodontal pocket depth and clinical attachment loss. Eligible designs included observational studies (cohort, cross-sectional, and case–control), longitudinal studies, systematic reviews, and meta-analyses. Studies were excluded if they did not report periodontal outcomes, were not available in full text, or addressed only unrelated oral conditions.

The methodological quality and relevance of the included studies were appraised narratively, giving greater weight to longitudinal designs and meta-analyses. Following screening, seven primary clinical studies were selected for detailed discussion, supplemented by additional mechanistic studies, reviews, and reference texts used to contextualize the biological pathways. The selected evidence was analysed thematically through identification, classification, comparison, and synthesis, organized according to the direction of the reported association (positive versus non-significant) and the proposed biological mechanisms.

Results and Discussion

Several studies have reported no significant correlation between alcohol consumption and periodontal pocket formation. A study conducted by Sankaranarayanan et al., (2019) reported that no consistent association was found between alcohol-related variables and periodontal pocket development in the total population or among non-smokers. Among smokers, however, a positive association was observed with the frequency of alcohol consumption. Overall, risk estimates were slightly higher in women than in men. In summary, light to moderate alcohol consumption does not appear to be consistently associated with periodontal pocket development, and adverse effects on the periodontium seem to be influenced by gender and smoking status.

Another study by Sankaranarayanan et al., (2020) also demonstrated that, overall, the amount of alcohol consumption or consumption exceeding risk thresholds was not consistently associated with periodontal pocket development. The frequency of alcohol consumption likewise showed inconsistent associations with the presence of periodontal pockets. Similar findings were observed in both smokers and non-smokers. In this study, alcohol consumption

was not consistently associated with periodontal pocket progression over an 11-year follow-up period.

In contrast, several studies have shown that alcohol consumption may increase the occurrence of periodontal pockets. A cohort study by Singh & Kaur, (2018) categorized participants into four groups based on alcohol consumption: non-drinker/low frequency, moderate frequency, high frequency, and chronic drinker. The study demonstrated a significant increase in clinical attachment loss (CAL) among high-frequency drinkers (67.67%) and chronic drinkers (87.5%) after a 4-year follow-up period.

A comparative cross-sectional epidemiological study conducted by Priyanka et al., (2017) reported similar findings. This study aimed to assess the impact of alcohol dependence on oral health status by comparing alcohol-dependent individuals with non-alcohol users. Subjects were classified as alcohol-dependent based on the diagnostic criteria of the American Psychiatric Association (DSM-5). The results showed a higher prevalence of periodontitis (89.61%) among alcohol-dependent subjects compared to controls (78.67%). Periodontal pocket depth was also significantly greater in alcohol-dependent individuals than in non-alcohol subjects.

Another study by Wagner et al., (2017) reported an increased risk of periodontal disease among alcohol consumers. In this study, alcohol consumption was assessed at baseline by asking participants about the number of drinks typically consumed per week. Alcohol consumption was categorized into four groups: non-drinkers, ≤1 drink/week, >1 drink/week and ≤1 drink/day, and >1 drink/day. Individuals presenting with ≥2 teeth showing proximal CAL progression ≥3 mm over a 5-year period were classified as having disease progression. The results demonstrated a significantly increased risk of periodontal disease progression among individuals who reported consuming >1 drink/day. This negative effect was evident in men but not in women.

According to a study by Suwama et al., (2018), a correlation was observed between alcohol consumption and clinical attachment loss (CAL). Data were collected from oral examinations of each participant, including the number of remaining teeth, mean probing pocket depth, and mean CAL, which were used for analysis. The results showed that mean individual CAL tended to increase with higher alcohol consumption among community-dwelling older adults aged 73 years. Logistic regression analysis demonstrated that alcohol consumption was significantly and positively associated with mean individual CAL among heavy drinkers. In this study, heavy drinkers were defined as individuals consuming daily amounts of alcohol reported to increase the risk of lifestyle-related diseases.

A study conducted by Hach et al., (2015) demonstrated that alcohol consumption may increase the likelihood of developing periodontitis later in life. In this study, clinical periodontal attachment loss was calculated to determine periodontitis progression. Alcohol consumption was assessed in the Copenhagen City Heart Study (CCHS) during the periods 1981–1983, 1991–1994, and 2001–2003 using standardized questionnaires. Alcohol consumption was classified into three groups: light, moderate, and heavy consumption. The results indicated that individuals who were heavy drinkers during 1981–1983 had a higher risk of developing periodontitis compared to light drinkers. This study suggests that early alcohol consumption may increase the likelihood of developing periodontitis 20 years later.

Taken together, these individual studies are broadly consistent with the pooled evidence from meta-analyses on this topic. The meta-analysis of observational studies by Wang et al., (2016) reported that alcohol consumption was associated with an increased risk of periodontitis, with the risk being more pronounced at higher levels of consumption. This finding was reinforced by the systematic review and meta-analysis of Pulikkotil et al., (2020), which likewise concluded that higher alcohol consumption is significantly associated with periodontitis compared with low or no consumption.

A more recent narrative review by Gandhi et al., (2024) reached a similar conclusion, noting that alcohol is directly or indirectly associated with clinical attachment loss, pocket formation, and an altered oral microbial balance. At the mechanistic level, a systematic review of animal studies by Martins et al., (2025) reported that chronic alcohol exposure amplifies the periapical inflammatory response and bone resorption in a dose-dependent manner, while severe clinical manifestations such as necrotising periodontal disease have also been linked to alcohol misuse (Tkacz et al., 2021). Collectively, the balance of the reviewed evidence indicates that heavy and long-term alcohol consumption is associated with periodontal pocket formation and clinical attachment loss, whereas light-to-moderate consumption shows a weaker and less consistent relationship.

Conclusion

This literature review indicates an association between alcohol consumption and the clinical manifestations of periodontitis, including periodontal pocket formation and clinical attachment loss. Although the evidence is not uniform five of the seven primary studies reported a positive association and two found none the balance of findings suggests that heavy and long-term alcohol consumption may contribute to periodontal tissue destruction, particularly among heavy drinkers and smokers. Biologically, alcohol may act through immune suppression, oxidative stress, reduced salivary flow, and increased microbial susceptibility, while variations in outcomes likely reflect differences in study design, population, consumption patterns, follow-up, and confounders. Dentists are therefore encouraged to include education and counselling on the adverse effects of alcohol within periodontal prevention and management. Further well-designed longitudinal and interventional studies using standardized alcohol measurement, consistent periodontal outcome definitions, and rigorous confounder control are needed to clarify the dose response relationship and underlying mechanisms.

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