

Effect of Ferocity of Alcohol on Robustness of Periodontium: A Case Control Study

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Abstract

Background: Alcohol consumption is the world’s third largest risk factor for diseases and disabilities. The knowledge regarding effect of intensity of alcohol on periodontal health is limited. So this study was carried out to determine the possible correlation between the frequency of alcohol intake and periodontal diseases.

Materials & methods: A total of 120 subjects were included in the study between age group of 35-75 years. 60 patients were screened for alcohol consumption or alcohol dependence, assessed by using Alcohol Use

Disorder Identification Test (AUDIT) and Cut down, Annoyed, Guilty, Eye-opener (CAGE) questionnaire. Subjects were evaluated for blood levels of Gamma Glutamyl Transpeptidase (GGT), a liver enzyme indicator of alcohol drinking. The periodontal parameters such as plaque index, gingival index, probing depth and clinical attachment loss were assessed by ANOVA test.

Result: when periodontal variables i.e. plaque index, gingival index, missing teeth, probing depth and clinical attachment loss were compared. Statistically significant difference was found between test and control groups.

Among the alcoholics plaque index, gingival index, probing depth and clinical attachment loss was statistically significant ($p=0.000$) and increased as the intensity of alcohol consumption increased i.e. no or occasional drinkers (NA) < moderate drinkers (MA) < intense drinkers (IA) < alcohol dependence (DA). Within the alcoholics there was no significant difference ($p=0.008$) when missing teeth variable was considered.

Conclusion: Alcoholics has been found to have an increased prevalence of periodontal diseases as compared to healthy individuals and can be considered as a risk indicator for periodontal diseases.

Keywords: GGT, AUDIT, CAGE questionnaire, DA, IA, MA, NA.

Introduction

The existence of the relationship of alcohol on periodontal diseases and its effects has been previously explained through different biological mechanisms. Severe infections, altered immune responses, negative effect on clotting mechanism and nutritional disorders like protein and vitamin deficiency are few frequently seen consequences in alcoholic individuals.¹

Alcohol is a risk factor in 60 types of diseases. About 4% of all deaths worldwide are attributed to alcohol consumption, which is greater than the deaths caused by HIV/AIDS, domestic violence and tuberculosis. Alcohol has both local and systemic adverse effects. The local action of alcohol attributes to bone resorption and suppresses bone turnover. It may have a direct toxic effect on periodontal tissues. Excessive and constant alcohol consumption may affect the response of host to bacterial infections, thus increasing the host vulnerability.^{2, 3} The investigations on the association between intensity of alcoholism and periodontal diseases are limited.^{4, 5, 6} However, this is one of the few studies to assess the relationship between intensity of

alcohol consumption and its effect on periodontal health.

Materials and methods

A total of 200 male subjects from the outpatient department were screened with the institutional ethical approval between the age group of 35 to 75 years and 120 systemically healthy subjects were included in the study. 60 subjects were selected for alcohol consumption habit and 60 systemically healthy subjects without alcohol consumption habit were included in the control group. The controls and the test group were matched for age and gender. Complete periodontal examination including plaque index, gingival index, probing depth and clinical attachment loss was recorded.

Patient Selection

For test group 1: Patients were screened for alcohol consumption or alcohol dependence, by using Alcohol Use Disorder Identification Test (AUDIT) and Cut down, Annoyed, Guilty, Eye-opener (CAGE) questionnaire system. Subjects were evaluated for blood levels of Gamma Glutamyl Transpeptidase (GGT), a liver enzyme indicator of alcohol drinking.

For control group: The individuals with no history of alcohol consumption, subjects with ≥ 5 teeth present in the oral cavity and non-smokers were excluded in this study as there is a direct correlation of smoking and periodontal diseases. Individuals with other debilitating diseases that compromise the immune system like HIV, neoplasia, auto-immune diseases, illicit drug users and subjects with history of periodontal treatment in the last 6 months were enlisted in the exclusion criteria. All the study participants were well-versed about the procedures to be carried out during the study and a written informed consent was taken.

A general examination following the age, gender, body mass index, education level, income grade, medical status,

smoking patterns and past dental history was recorded in the form of questionnaires. A complete intraoral examination was performed using a UNC-15 probe and periodontal examination included plaque index, gingival index, probing depth, clinical attachment loss and number of missing teeth were documented on case sheets. Alcohol consumption was noted with the help of Alcohol Use Disorder Identification Test (AUDIT) and CAGE questionnaire. Collection of blood samples was done to estimate Gamma Glutamyl Transpeptidase levels by auto-analyzer.

Result

The periodontal parameters such as plaque index, gingival index, probing depth and clinical attachment loss were higher in test group as compared to the control group, the difference was statistically significant ($p = 0.000$). No statistically significant difference was found in the missing teeth among alcoholics as compared to systemically healthy controls. Within alcoholic group it was found that the frequency of alcohol consumption has effect on the progression of periodontal diseases. The mean plaque index of the alcoholic group was 2.02 ± 0.53 as compared to the 0.899 ± 0.42 for the control group which was found statistically significant in alcoholic groups ($p=0.000$) compared to systemically healthy controls. The mean plaque index within alcoholic groups was significantly correlated to the frequency of alcohol consumption i.e. no or occasional drinkers < moderate drinker < intense drinker < alcohol dependent. The mean gingival index in alcoholic group was 2.09 ± 0.54 as compared to systemically healthy controls which was 0.69 ± 0.31 . The gingival index was statistically significant among alcoholic group ($p=0.000$) as compared to systemically healthy controls. The mean gingival index within alcoholics was no or occasional drinkers (0.70 ± 0.11) < moderate drinkers (1.87 ± 0.40) < intense drinkers

(2.44 ± 0.24) = alcohol dependent (2.32 ± 0.33) group. The mean number of missing teeth in alcoholic group was 2.45 ± 2.67 as compared to systemically healthy controls which was 1.50 ± 1.77 . There was statistically significant difference found in missing teeth parameter among alcoholic as compared to systemically healthy controls. When the mean number of missing teeth within alcoholic groups was compared, it was found that there was no correlation between the frequency of alcohol consumption and missing teeth. The mean number of missing teeth among alcoholic group was 2.25 ± 1.70 for no or occasional drinkers, 1.05 ± 1.07 for moderate drinkers, 1.90 ± 1.72 for intense drinkers and 3.67 ± 3.30 for alcohol dependence group. The mean probing depth in alcoholic group was 4.23 ± 1.07 as compared to systemically healthy controls 2.12 ± 0.31 which was statistically significant. The mean probing depth among alcoholics was no or occasional drinkers (2.63 ± 0.33) < moderate drinkers (3.44 ± 0.93) < intense drinkers (4.59 ± 0.33) < alcohol dependence (4.88 ± 0.79). There was statistically significant difference ($p=0.000$) among alcoholic groups. The mean clinical attachment loss in alcoholic group was 4.32 ± 1.15 as compared to systemically healthy controls 2.12 ± 0.32 . The mean clinical attachment loss among alcoholics was 2.63 ± 0.33 for no or occasional drinkers, 3.44 ± 0.93 for moderate drinkers, 4.70 ± 0.46 for intense drinkers and 5.05 ± 0.85 for alcohol dependence group. The mean gamma Glutamyl Transpeptidase levels in alcoholic groups was found to be significant.

| Parameters | Alcoholic n (%) | Control n (%) |
|-----------------|-----------------|---------------|
| Age (mean + SD) | 45.92 ± 9.24 | 44 ± 8.04 |
| BMI | 21.15 ± 3.006 | 22.53 ± 2.97 |
| Income | 19422 ± 1059 | 13041 ± 9840 |
| Education | 7.083 ± 4.38 | 5.76 ± 4.75 |

| Periodontal parameter | Group | N | Mean | SD | f | significance |
|---------------------------|----------------------|----|-------|-------|--------|--------------|
| Plaque index | Alcoholic group | 60 | 2.02 | 0.53 | 107.48 | 0.000 |
| | Systemically healthy | 60 | 0.899 | 0.42 | | |
| Gingival index | Alcoholic group | 60 | 2.09 | 0.54 | 108.8 | 0.000 |
| | Systemically healthy | 60 | 0.699 | 0.31 | | |
| Missing teeth | Alcoholic group | 60 | 2.45 | 2.67 | 3.15 | 0.045 |
| | Systemically healthy | 60 | 1.50 | 1.77 | | |
| Probing depth | Alcoholic group | 60 | 4.23 | 1.07 | 167.06 | 0.000 |
| | Systemically health | 60 | 2.12 | 0.311 | | |
| Clinical attachment level | Alcoholic group | 60 | 4.32 | 1.158 | 160.60 | 0.000 |
| | Systemically healthy | 60 | 2.12 | 0.329 | | |

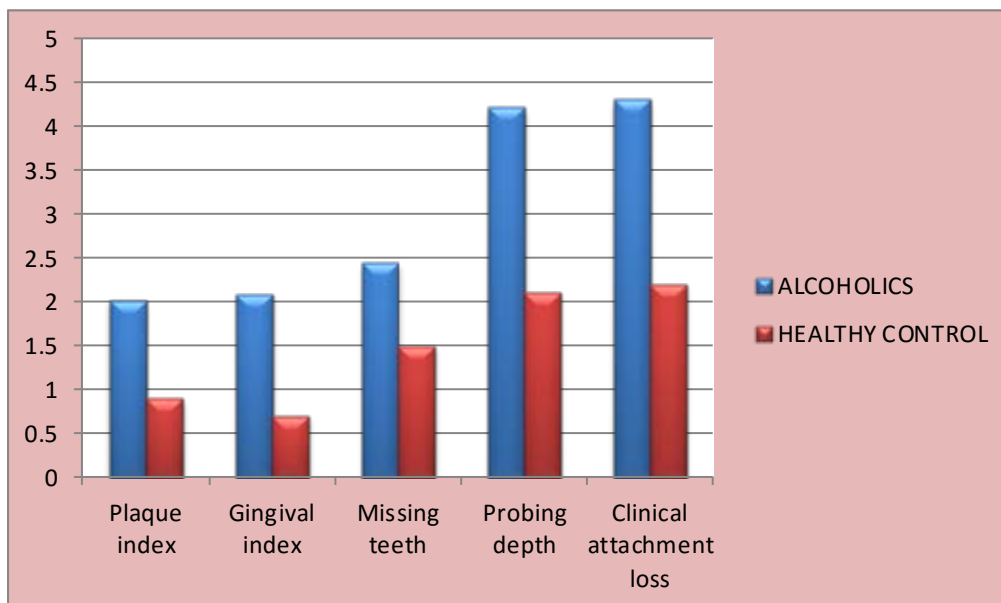


Figure 1: mean and standard deviation of periodontal parameters among alcoholic and healthy controls

Table 3: Mean and standard deviation of plaque index, gingival index, missing teeth, probing depth and clinical attachment loss among alcoholic group

| Periodontal parameters | Group | N | Mean | SD | f | Significance |
|--------------------------|-----------------------|----|-------|--------|--------|--------------|
| Plaque index | No/occasional drinker | 4 | 0.77 | 0.094 | 28.060 | 0.000 |
| | Moderate drinker | 19 | 1.76 | 0.418 | | |
| | Intense drinker | 10 | 2.27 | 0.255 | | |
| | Alcohol dependent | 27 | 2.33 | 0.345 | | |
| Gingival index | No/occasional drinker | 4 | 0.700 | 0.1154 | 32.436 | 0.000 |
| | Moderate drinker | 19 | 1.872 | 0.404 | | |
| | Intense drinker | 10 | 2.441 | 0.246 | | |
| | Alcohol dependent | 27 | 2.322 | 0.338 | | |
| Missing teeth | No/occasional drinker | 4 | 2.25 | 1.708 | 43.98 | 0.008 |
| | Moderate drinker | 19 | 1.05 | 1.079 | | |
| | Intense drinker | 10 | 1.90 | 1.729 | | |
| | Alcohol dependent | 27 | 3.67 | 3.305 | | |
| Probing depth | No/occasional drinker | 4 | 2.630 | 0.338 | 19.406 | 0.000 |
| | Moderate drinker | 19 | 3.444 | 0.938 | | |
| | Intense drinker | 10 | 4.594 | 0.333 | | |
| | Alcohol dependent | 27 | 4.887 | 0.794 | | |
| Clinical attachment loss | No/occasional drinker | 4 | 2.630 | 0.338 | 20.911 | 0.000 |
| | Moderate drinker | 19 | 3.444 | 0.938 | | |
| | Intense drinker | 10 | 4.703 | 0.462 | | |
| | Alcohol dependent | 27 | 5.05 | 0.859 | | |

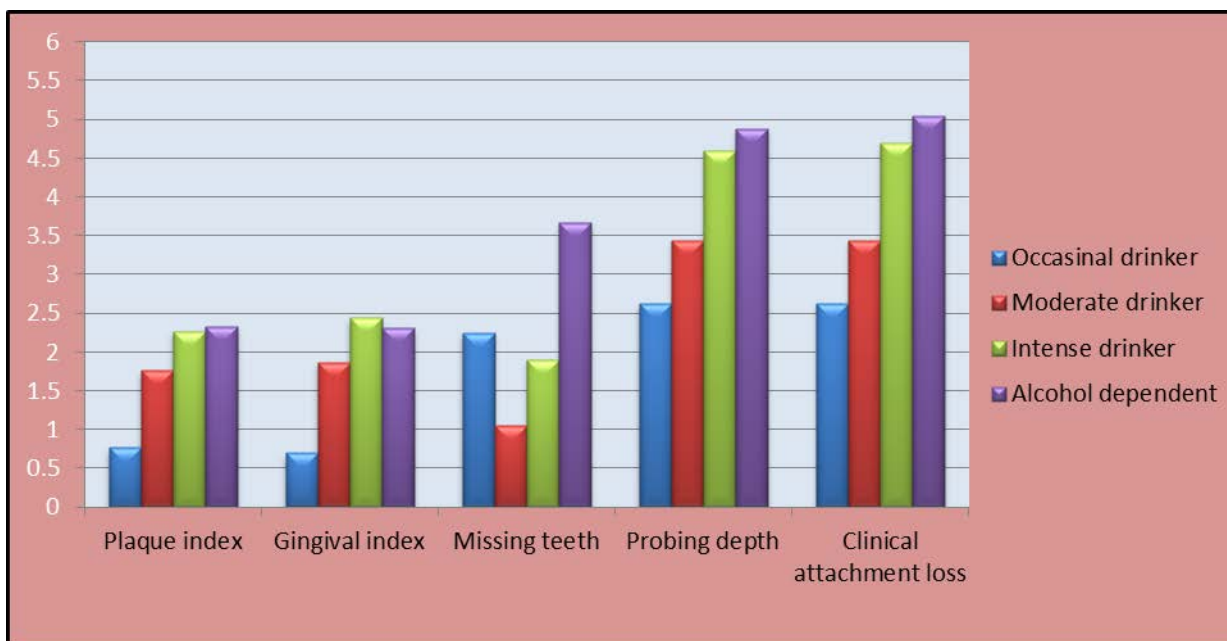


Figure 2: Mean and standard deviation of periodontal parameters among alcoholic group

| Periodontal parameters | Comparison | Sum of squares | df | Mean squares | f | Significance |
|--------------------------|----------------|----------------|----|--------------|--------|--------------|
| Plaque index | Between groups | 10.318 | 3 | 3.439 | 28.060 | 0.000 |
| | Within groups | 6.864 | 56 | 0.123 | | |
| | Total | 17.181 | 59 | | | |
| Gingival index | Between groups | 11.3139 | 3 | 3.77 | 32.436 | 0.000 |
| | Within groups | 6.514 | 56 | 116 | | |
| | Total | 17.832 | 59 | | | |
| Missing teeth | Between groups | 80.253 | 3 | 26.751 | 43.98 | 0.008 |
| | Within groups | 340.59 | 56 | 6.082 | | |
| | Total | 420.85 | 59 | | | |
| Probing depth | Between groups | 34.966 | 3 | 11.655 | 19.406 | 0.000 |
| | Within groups | 33.634 | 56 | 601 | | |
| | Total | 68.60 | 59 | | | |
| Clinical attachment loss | Between groups | 41.852 | 3 | 13.951 | 20.911 | 0.000 |
| | Within groups | 37.361 | 56 | 667 | | |
| | Total | 79.214 | 59 | | | |

Table 5 :Mean and standard deviation for gamma Glutamyl Transpeptidase in alcoholic groups

| Parameter | N | Min. | Max. | Mean± SD |
|-------------------------------|----|-------|-------|---------------|
| Gamma Glutamyl Transpeptidase | 60 | 32.00 | 59.00 | 51.300± 5.139 |

Discussion

Alcohol dependence is considered as one of the most serious medical problems. Excessive ingestion of alcohol is the third most common cause of death in the world after cancers and cardiovascular complications.⁸There are direct toxic damages related to alcohol consumption such as liver cirrhosis, cerebral atrophy, cardio-myopathy, gastrointestinal bleeding and pancreatitis.⁹Alcohol dependence also causes indirect oral problems including caries, tooth loss, periodontal diseases and cancers.^{10,11}According to Lang et.al (1999) cases of aggressive periodontitis are more common in age groups of less than 30 years in otherwise healthy individuals.¹²

In the present study all subjects were male because even today, in India, females with alcohol addiction are very few and they are not open about it due to social stigma. To avoid the bias in sample distribution only male subjects were included in both the test groups. The conjunction of smoking and drinking can occur at several levels. In the first place, in industrial societies, smokers are more likely to be drinkers.¹³Hence they were excluded precluding their harmful effect on the periodontium.

The alcohol consumption subjects were those patients with previous history of alcohol intake but without any systemic conditions. The study subjects were classified into no or occasional drinkers (NA), moderate drinkers (MA), intense drinkers (IA), and alcohol dependence (DA) based on CAGE and AUDIT scores. Subjects were also evaluated for blood levels of Gamma Glutamyl Transpeptidase (GGT), a liver enzyme indicator of alcohol intake.

Plaque index is an indicator of an individual’s oral hygiene. In the present study, the mean Plaque index is higher and statistically significant in alcoholic group (p=0.000) when compared to systemically healthy controls. When compared between the alcoholics, the mean plaque index was statistically significant (p=0.000) in no or occasional drinkers (NA) < moderate drinkers (MA) < intense drinkers (IA) < alcohol dependence (DA). This finding is in concurrence with a study conducted by Lages et.al 2012.¹⁴They noticed that subjects from MA, IA and DA had shown a higher Plaque index than NA. These findings are not in concurrence with a study conducted by Amaral et.al 2008¹⁵ and Jansson et.al in 2008.¹⁶They found that there was no statistically significant difference between alcoholic and non-alcoholic subjects.

Bleeding on probing is an indicator of acute response of periodontal tissues to bacterial challenge as a part of innate immunity. This inflammatory response of gingiva may be due to impaired neutrophil, macrophage and T-cell functions which increases host vulnerability. Alcohol has local as well as systemic effect on individuals consuming alcohol. Locally, it acts by toxic effect on periodontal tissue. It is also expressed as exaggerated gingival response and bleeding with slight provocation in alcoholics. The gingival index was higher and statistically significant (p=0.000) in the alcohol consumption when compared to the systemically healthy controls. Among the alcoholics’ gingival index was statistically significant (p=0.000) and increased as the intensity of alcohol consumption increased i.e. no or occasional drinkers (NA) < moderate drinkers (MA) < intense drinkers (IA) <

alcohol dependence (DA). This finding is in concurrence with that of a study conducted by Bouchard et al 2006¹⁷ had shown the higher gingival index in regular drinkers when compared to non-drinkers or occasional drinkers. In contrary to above studies, a study by Kranzler et.al in 1990¹⁸ found that gingival index was not statistically significant.

The number of missing teeth is considered as strong risk indicator for periodontitis. The major cause of missing teeth is caries. The micro-organisms responsible for caries and periodontal diseases act synergistically i.e. plaque micro-organisms causes 'demineralization of tooth surface. Due to the antimicrobial action of alcohol, growth of these micro-organisms is prevented. The prevention of microbial growth resists the biofilm formation and progression of both caries and periodontal diseases which are responsible for tooth loss.

The number of missing teeth was similar in all groups. There was no statistically significant difference ($p=0.045$) between the alcohol consumption and systemically healthy control groups. Within the alcoholics there was no significant difference ($p=0.008$) between occasional drinkers, moderate drinkers, intense drinkers and alcohol dependence subjects.

While the study conducted by Lages et al 2012¹⁴, done the comparison within alcoholics. There was no statistically significant difference found between frequencies of alcohol consumption and missing teeth.

These results are not in concurrence with the results of the study conducted by Hornecker et. al 2003¹⁹

and Jansson et.al 2008¹⁶, in 100 alcohol addicted patients. They concluded that missing teeth in alcoholics was twice as high as in the healthy population.

Alcohol is considered as an important risk factor for various bone-related disorders, such as reduced bone mass and fractures. Chronic alcohol abuse is a major risk factor for osteoporosis.^{20, 21} If alcohol exacerbates alveolar bone resorption, the observed effect of drinking on increasing periodontal pocket depth may lead to extensive periodontal destruction. It is also supported by vitro studies which suggest that ethanol stimulates bone resorption and blocks the stimulation of bone formation.²²

The mean probing depth was higher and statistically significant ($p=0.000$) in alcohol consumption when compared to systemically healthy controls. The mean probing depth among alcoholics was statistically significant ($p=0.000$) and increased as the intensity of alcohol consumption increases i.e. no or occasional drinkers (NA) < moderate drinkers (MA) < intense drinkers (IA) < alcohol dependence (DA). Lages et.al in 2012¹⁴ observed that subjects from MA, IA and DA had higher probing depth as compared to NA group. Shimazaki et.al 2005²³ found that, more alcohol consumed subjects had shown the greater proportion of their teeth with $PD \geq 4\text{mm}$. Alcohol consumption did not show any significant association with mild or moderate PD.

The increased clinical attachment loss is due to periodontal inflammatory responses are triggered by bacteria. These responses lead to most of the tissue destruction along with direct destructive effect by the bacteria. It has been recognized that the neutrophils

as well as antibody/complement system is critical for protection against periodontal bacteria. Abnormalities in this system often lead to increased susceptibility to periodontal disease.

Clinical attachment loss was statistically significant ($p=0.000$) in alcoholic group as compared to systemically healthy controls. Among the alcoholics, the clinical attachment loss was statistically significant ($p=0.000$) and increased as the intensity of alcohol consumption increased i.e. no or occasional drinkers (NA) < moderate drinkers (MA) < intense drinkers (IA) < alcohol dependence (DA). Similar results were seen in the study conducted by Lages et.al in 2012¹⁴. The mean clinical attachment level increases as the intensity of alcohol consumption increases i.e. NA < MA < IA < DA. Whereas study conducted by Khocht et.al in 2003²⁴, both alcohol and non-alcoholics had shown increased gingival margin recession with increasing age, but the extent of this recession was greater in alcoholics. In contrary to above study, Shimazaki et.al 2005²³ found that there was no significant relationship between drinking and clinical attachment loss in multivariate analysis.

A significant correlation was found between increased levels of Gamma Glutamyl Transpeptidase when compared with the intensity of alcoholism. Gamma Glutamyl Transpeptidase is a component of antioxidant defense systems as well as liver enzyme which play a major role in the local as well as systemic inflammatory conditions.

Conclusion

The present study concludes that due to neglected oral hygiene, an alcoholic has increase an in occurrence and

progression of periodontal diseases. Alcoholic subjects have an increased prevalence of periodontitis as compared to healthy individuals which indicates that alcohol consumption can be considered as a risk indicator for periodontal diseases. GGT as a liver enzyme took part in the diagnosis of periodontal diseases hence, it won't be completely wrong to consider GGT as a possible biomarker for periodontal diseases which will help in quick and early diagnosis. And thus, it is further recommended to carry more clinical studies to establish the role of GGT and periodontal health.

Limitations

The type of alcohol-containing drinks was not considered due to variation in alcohol percentage in different alcohol preparations. Also the brushing technique, frequency and method were not taken into account due to variation in the socioeconomic status, physical and psychological status in test groups. Microbiological parameters were not considered during clinical examination to differentiate between active and inactive sites of periodontal disease. Therefore, furthermore studies can be designed and are needed to evaluate the correlation of periodontal health and other factors in relation with alcohol consumption.

References

1. Tezal M, Grossi SG, Ho AW, Genco RJ. The effect of alcohol consumption on periodontal disease. J Periodontol 2001; 72:183-189.
2. Pitiphat W, Merchant AT, Rimm EB, Joshipura KJ. Alcohol consumption increases periodontitis risk. J Dent Res 2003; 82:509-513.
3. Szabo G. Consequences of alcohol consumption on host defense. Alcohol Alcohol 1999; 34(6):830-841.
4. Thurman RG. Mechanism of hepatic toxicity: II Alcoholic liver injury involves activation of Kupffer

- cells by endotoxin. *Am J Physiol* 1998; 275: G605–11.
5. Novacek G, Plachetzky U, Potzi R. Dental and periodontal disease in patients with cirrhosis – Role of etiology of liver disease. *J Hepatol* 1995; 22:576-582.
 6. Enberg N, Wolf J, Ainamo A. Dental diseases and loss of teeth in a group of Finnish alcoholics: A radiological study. *Acta Odontol Scand* 2001; 59:341-347.
 7. Anton RF, Lieber C, Tabakoff B; CD Tect Study Group. Carbohydrate-deficient transferrin and gamma-glutamyl transferase for the detection and monitoring of alcohol use: results from a multisite study. *Alcohol Clin Exp Res.* 2002; 26(8):1215-1222.
 8. Kranzler HR, Babor TF, Goldstein L, Gold J. Dental pathology and alcohol related indicators in an outpatient clinic sample. *Community Dent Oral Epidemiol* 1990; 18: 204-7.
 9. Chester J. Summers and Albert Oberman. Association of oral disease with 12 selected variables I. Periodontal Disease. *J Dent Res* 1968; May-June, 457-462.
 10. Vladimir Panov, Assay Krasteva. Oral health in patients with liver diseases. *Journal of IMAB*; issue 2011:17, book 2.
 11. L. Lins, P.L. Bittencourt, M.A. Evangelista, R. Lins, L. Codes, A.R. Cavalcanti, R. Paraná, J. Bastos. Oral Health Profile of Cirrhotic Patients Awaiting Liver Transplantation in the Brazilian Northeast. *Transplantation Proc.* May 2011; 43(4):1319–1321.
 12. Seyedali Banihash, Abbas Shirdel, Samira Pakro. Evaluation of the relationship between periodontal parameters and liver cirrhosis. *Journal of Periodontology and implant dentistry* 2009; 1(1):28-30.
 13. Susanne Movin. Relationship between periodontal disease and cirrhosis of the liver in humans. *Journal of Clinical Periodontology* 1981; 8: 450-458.
 14. Eugenio J.P Lages, Fernando O. Costa, Elizabeth M. B. Lages, Luis O.M. Cota, Sheila C. Cortelli, Gilson C. Nobre- Franco, Renata M. Cyrino, Jose R. Cortelli. Risk variables in the association between frequency of alcohol consumption and periodontitis. *Journal of clinical Periodontology* 2012; 39:115-122.
 15. Cristine da Silva Furtado Amaral, Ronir Raggio Luiz and Anna Thereza Thome Leao. The relationship between alcohol dependence and periodontal disease. *Journal of clinical Periodontology* 2008; 79:993-998.
 16. Leif Jansson. Association between alcohol consumption and dental health. *Journal of clinical Periodontology* 2008; 35:379-384.
 17. Philippe Bouchard, Pierre Boutouyrie, Catherine Mattout, Denis Bourgeois. Risk assessment for severe clinical attachment loss in an adult population. *J Periodontol* 2006; 77:479-489.
 18. Kranzler HR, Babor TF, Goldstein L, Gold J. Dental pathology and alcohol related indicators in an outpatient clinic sample. *Community Dent Oral Epidemiol* 1990; 18:204-7.
 19. Hornecker E, Muuß T, Ehrenreich H. A Pilot Study on the oral conditions of severely alcohol addicted persons. *J Contemp Dent Pract* 2003 May; (4) 2:051-059.
 20. Klein RF. Alcohol-induced bone disease: Impact of ethanol on osteoblast proliferation. *Alcohol Clin Exp Res* 1997; 21:392-399.
 21. Rico H. Alcohol and bone disease. *Alcohol Alcohol* 1990; 25:345-352.
 22. Farley JR, Fitzsimmons R, Taylor AK, Jorch UM, Lau KH. Direct effect of ethanol on bone resorption and

formation in vitro. Arch Biochem Biophys 1985; 238:305-314.

23. Yoshihiro Shimazaki, Toshiyuki Saito, Yutaka Kiyohara, Isao Kato, Michiaki Kubo, Mitsuo Iida, and Yoshihisa Yamashita. Relationship between Drinking and Periodontitis: The Hisayama Study J Periodontol 2005; 76:1534-1541
24. Ahmed Khocht, Malvin Janal, Steven Schleifer, Steven Keller. The influence of gingival margin recession on loss of clinical attachment in alcohol dependent patients without medical disorders. Journal of Periodontology 2003; 74:485-493.