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Predicting the negative effects of periodontal disease and smoking on liver functions and blood biochemical parameters: A correlational study

Abstract. This study aimed to verify the correlation between periodontal disease, liver function, and blood parameters to shed light on the potential future interactions among these factors and determine whether smoking plays a role in this relationship. **Materials and methods.** The study included 50 male, 25 smokers with periodontal disease, and 25 non-smokers with periodontal disease, aged 20—50 years. The clinical periodontal parameters pocket depth (PD) and clinical attachment level (CAL) were determined using William's periodontal probe, and 6 sites around each tooth (mesiobuccal, mid-buccal, distobuccal, mesiolingual, mid-lingual, and distolingual) were detected. **Results.** The results showed a significant increase in bleeding (p=0.004), CAL (p<0.001), and PD (p<0.001) in smokers compared with non-smokers. There were substantial differences between smokers and non-smokers with a significant increase in AST and ALT in smokers rather than in non-smokers in relation to clinical periodontal parameters. For smokers, there was a positive correlation between both CAL and PD with AST and ALT (r=0.30—0.71, p<0.05). A positive correlation between bleeding, CAL, and PD with WBCs (r=0.34, 0.57, 0.50), Neu (r=0.67, 0.40, 0.48), Lym (r=0.48, 0.37, 0.39), and Plt (r=0.55, 0.38, 0.38); there was also a significant positive correlation between CAL and bleeding with WBCs (r=0.34, 0.63, 0.30), Neu (r=0.30, 0.39), Lym (r=0.41, 0.53), and Plt (r= 0.49, 0.45) in non-smokers, respectively. **Conclusion.** The analysis revealed a significant difference in liver enzyme levels between smokers and non-smokers with periodontal disease, and hematological parameters could predict periodontitis.

Key words: AST, ALT, smoking, WBCs, RBC, periodontitis

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Прогнозирование негативного влияния заболеваний пародонта и курения на функции печени и биохимические показатели крови: корреляционное исследование

Реферат. Цель исследования — проверка корреляции между заболеваниями пародонта, функцией печени и показателями крови, чтобы прояснить потенциальное взаимодействие этих факторов и определить, играет ли курение роль в этой связи. **Материалы и методы.** В исследовании приняли участие 50 мужчин от 20 до 50 лет с заболеваниями пародонта: 25 курильщиков и 25 некурящих. Клинические параметры пародонта — глубину пародонтального кармана (ГПК) и уровень клинического прикрепления (УКП) — определяли пародонтальным зондом Уильяма в 6 участках вокруг каждого зуба (мезиально-щечный, средне-щечный, дистально-щечный, мезиолингвальный, среднеязычный и дистально-язычный). Результаты показали значительное увеличение кровоточивости (p=0,004), ГПК (p<0,001) и УКП (p<0,001) у курильщиков по сравнению с некурящими. Были выявлены существенные различия между курящими и некурящими с заболеваниями пародонта, при этом у курящих наблюдалось значительное повышение уровня ферментов АСТ и АЛТ по сравнению с некурящими. Также наблюдалась положительная корреляция показателей ГПК и УКП с АСТ и АЛТ (r=0,30—0,71), а также положительная корреляция между кровоточивостью, УКП и ГПК с лейкоцитами, нейтрофилами, липопротеинами лейкоцитов и тромбоцитами (r=0,34—0,68). У некурящих наблюдалась положительная корреляция УКП с уровнем АСТ и АЛТ (0,44 и 0,54 соответственно), значимая положительная корреляция кровоточивости и УКП с лейкоцитами, нейтрофилами, липопротеинами лейкоцитов и тромбоцитами (r=0,34—0,63). Была найдена отрицательная корреляция кровоточивости и эритроцитов, гемоглобина и гематокрита у курящих (от –0,68 до –0,54) и некурящих (от –0,71 до –0,45). **Заключение.** Анализ выявил значительную разницу уровня печеночных ферментов между курящими и некурящими с заболеваниями пародонта, а гематологические параметры могут служить предсказанием пародонтита.

Ключевые слова: АСТ, АЛТ, курение, лейкоциты, эритроциты, пародонтит

INTRODUCTION

Periodontal disease is a chronic inflammatory disease that gradually destroys the tissues surrounding the teeth. It results from the action of oral microorganisms, particularly periodontal pathogens. These pathogens are released from pockets into the bloodstream and cause local and systemic inflammatory responses in the host [1]. Periodontitis is a public health concern, and many systemic diseases are associated with periodontal disease. Many studies have shown a correlation between periodontitis and liver function, and patients with severe periodontitis are more prone to liver dysfunction [2].

Previous studies have shown that periodontitis can affect hematological parameters and the proportion and function of circulating inflammatory cells, as it can change hemoglobin levels and hematocrit (HCT) [3]. Inflammatory cells play a major role in the aggravation or resolution of periodontitis, in addition to the role of neutrophils in the innate inflammatory response and the role of lymphocytes in adaptive immunity [4].

Smoking not only affects the respiratory system but also affects oral health by changing the human microflora and human immune response, leading to damage to tooth-supporting tissues [5]. The correlation between periodontal disease and liver function is a multifaceted interaction affected by different factors, including smoking habits, and many studies have shown a higher incidence of periodontal disease in smokers than in non-smokers [6]. Smoking can also affect liver function; understanding these correlations is important for improving oral health among smokers and for devising targeted interventions to relieve the harmful effects of smoking on oral health [7].

Recent research has focused on studying the relationship between periodontal disease and systemic conditions, especially liver diseases, and has highlighted the potential impact of oral health on liver function [8]. Recent studies have indicated an interaction between periodontal health, elevated ALT levels, and other liver diseases [2].

However, studying the intricate connections between periodontal disease, liver function, and blood parameters is important for understanding the potential systemic effects of oral health, and we can promote our knowledge of how maintaining good periodontal health may influence overall well-being [8].

The null hypothesis was that there would be a significant correlation between periodontal disease, liver enzymes (AST and ALT), and blood parameters.

The aim of the present study was to understand the complex links between periodontal disease, liver function, and blood parameters. The potential future effects of periodontal diseases and smoking can contribute to liver dysfunction or damage, and changes in blood parameters. Understanding and clarifying these relationships can provide valuable insights that deepen understanding and pave the way for future studies in this area of research.

List of Abbreviations

CAL	Clinical attachment level	**Lym**	Lymphocytes
PD	pocket depth	**Plt**	Platelets
AST	Aspartate transaminase	**RBCs**	Red blood cells
ALT	Alanine transaminase	**Hb**	Hemoglobin
WBCs	White blood cells	**HCT**	Hematocrit
Neu	Neutrophils		

MATERIALS AND METHODS

The study included 50 males with periodontal disease: 25 smokers, and 25 non-smokers, aged ranged 20—50 years. Inclusion criteria: no acute infectious diseases or systemic disease. Exclusion criteria: unwell cases, loss of follow-up, uncomfortable cases.

Demographic characteristics (age, height, and weight), as well as the smoking protocol used after the screening process. The participants underwent a comprehensive periodontal examination to evaluate key parameters like clinical attachment level (CAL), pocket depth, and bleeding.

Parameters

The clinical periodontal parameters, pocket depth, and CAL were determined by William's periodontal probe, and 6 sites around each tooth (mesiobuccal, mid-buccal, distobuccal, mesiolingual, mid-lingual, and distolingual) were detected except for the third molars, where the probe was directed parallel to the long axis of the tooth. PD was measured in smokers (4—9) mm, CAL, and the distance was measured from the cementoenamel junction to beneath the pocket at any site, from 2—4 mm. Blood samples were collected from all fasting participants in the morning (between 8—9 am), as all subjects refrained from eating, drinking, or smoking for a minimum of 2 h before blood collection, to analyze liver enzymes AST (aspartate transaminase) and ALT (alanine transaminase) and blood parameters in both smokers and non-smokers [9].

Blood samples were split into two parts: one was placed in EDTA tubes and centrifuged 300 cycles per 10 min to analyze liver enzymes AST and ALT, and the other part of the blood was placed in Gel-tubes to analyze the WBCs, Lym, Neu, RBCs, Hb, HCT, and platelets.

Generally, the data collection process encompasses a thorough evaluation of oral health behaviors and clinical indicators related to periodontal disease, blood parameters, and liver function markers among smokers and non-smokers.

Statistical analysis

Pearson's χ^2-criteria was used for categorical variables. Independent and paired t-tests were used for continuous variables that followed a normal distribution. While the Mann—Whitney U-test for continuous variables did not follow a normal distribution, the $p<0.05$ was considered significant if, and was two-tailed.

RESULTS

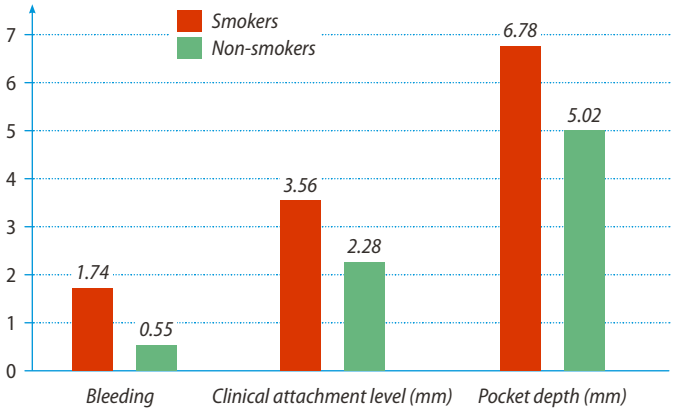
The study groups consisted of 50 males and 25 smokers with periodontal disease (bleeding, CAL, and PD), and 25 non-smokers with periodontal disease; the statistical

analysis showed a significant increase in bleeding, CAL, and PD in smokers compared with non-smokers at baseline (see the figure).

Chi-square analysis demonstrated that there were significant differences between the percentages of smokers and non-smokers with bleeding, showing that (20%) of smokers had no bleeding and (80%) had bleeding, while (72%) of the non-smokers had no bleeding, and (28%) had bleeding (table 1).

Table 1. Bleeding comparison in groups

Smokers (*n*=25)		Non-smokers (*n*=25)		χ^2	*p*
abs.	%	abs.	%		
20	80	7	28	69.67	0.004



The difference between smokers and non-smokers in terms of bleeding, clinical attachment level, and pocket depth

There's a significant difference between the percentages of smokers and non-smokers with CAL, the percentage of smokers in stage 2 was 20%, stage 3: 32%, and stage 4: 48%, while the percentage of non-smokers in stage 2 was 52%, stage 3: 16%, and stage 4: 32% (table 2).

Table 2. Clinical attachment level comparison in groups

CAL (mm)	Smokers		Non-smokers		χ^2	*p*
	abs.	%	abs.	%		
1–3	5	20	13	52		
4–5	8	32	4	16	74.08	<0.001
6–7	12	48	8	32		

For the percentage of PD in smokers 12% had 4 mm, 12% had 5 mm, 28% had 6 mm, 24% had 7 mm, 16% had 8 mm, and 8% had 9 mm whereas for the non-smokers, 20% had 4 mm, 32% had 5 mm, 12% had 6 mm, 12% had 7 mm, 20% had 7 mm, and 4% had 9 mm (table 3).

Table 3. Pocket depth (PD) comparison in groups

PD (mm)	Smokers		Non-smokers		χ^2	*p*
	abs.	%	abs.	%		
4	3	12	5	20		
5	3	12	8	32		
6	7	28	3	12	92.45	<0.001
7	6	24	3	12		
8	4	16	5	20		
9	2	8	1	4		

Spearman analysis showed a positive correlation between both CAL and PD with enzymes AST and ALT; however, there was no significant correlation between bleeding and AST and ALT enzymes in smokers. In non-smokers, there was a positive correlation between CAL and the enzymes AST and ALT, and there was no significant correlation between bleeding and PD with both AST and ALT (table 4).

Table 4. Spearman correlation (r) of AST and ALT levels with clinical periodontal parameters

Probe	Smokers		Non-smokers	
	ALT	AST	ALT	AST
Bleeding	0.28	0.06	0.12	0.22
Clinical attachment level	0.48*	0.38*	0.54*	0.44*
Pocket depth	0.71*	0.30*	0.11	0.08

*Remark. * — statistically significant difference ($p \leqslant 0.05$).*

The analysis showed In the smoker group, there was a positive correlation between bleeding, CAL, and PD with WBCs (*r*=0.34, 0.57, 0.50), Neu (*r*=0.67, 0.40, 0.48), LYM (*r*=0.48, 0.37, 0.39), and Plt (*r*=0.55, 0.38, 0.38); there was also a significant positive correlation between CAL and bleeding with WBCs (*r*=0.34, 0.63), Neu (*r*=0.30, 0.39), LYM (*r*=0.41, 0.53), and Plt (*r*=0.49, 0.45) in non-smokers, respectively. There was a negative correlation between bleeding index with RBC, Hb, and HCT in smokers and non-smokers; and there was no correlation between CAL and PD with RBC, Hb, and HCT in both smokers and non-smokers (table 5).

Table 5. Spearman correlation (r) of blood parameters with clinical periodontal parameters

Probe	Smokers							Non-smokers						
	WBCs	Neu	LYM	Plt	RBCs	Hb	HCT	WBCs	Neu	LYM	Plt	RBCs	Hb	HCT
Bleeding	0.34*	0.67*	0.48*	0.55*	−0.54*	−0.68*	−0.59*	0.34*	0.30*	0.41*	0.49*	−0.60*	−0.45*	−0.71*
CAL	0.57*	0.40*	0.37*	0.38*	0.20	0.09	0.33	0.63*	0.39*	0.53*	0.45*	0.28	0.22	0.52
PD	0.50*	0.48*	0.39*	0.38*	0.12	0.09	0.31	0.30	−0.22	0.12	0.11	0.32	0.02	0.29

*Remark. * — Statistically significant difference ($p \leqslant 0.05$).*

DISCUSSION

Indices of periodontitis, including bleeding on probing, PD, and CAL, are known as conventional methods of diagnosing periodontitis; they focus on the severity of the disease more than its action, and modern methods are predictable to help in diagnosing periodontal disease and provide information on the risk of developing a new disease [10—12].

The current results showed a significant increase in the gingival indices, bleeding on probing, CAL, and PD in smokers compared to non-smokers, which initially indicates the existence of a causal relationship between smoking and gum disease. The results of the chi-square test showed the conformity of this relationship, as the higher percentages were in the direction of the smoker's group (bleeding, PD, and CAL). These results are reinforced by the findings of a previous study, which demonstrated that smoking causes vasodilatation in some tissues, leading to increased gingival bleeding [13, 14], while this result contradicts other studies that showed decreased gingival bleeding in smokers, which may be due to vasoconstriction of gingival tissue, which is stimulated by the effect of nicotine-induced adrenaline and noradrenaline on α_1-adrenergic receptors.

The significant differences in CAL and PD between smokers and non-smokers may be due to the severity of periodontal destruction, which is compatible with [6]. Smokers are at a higher risk of clinical attachment loss $\geqslant$5 mm than non-smokers, and smoking promotes bone loss resulting from periodontitis [12]. Studies have shown that smokers tend to have more gingival recession, which contributes to the promotion of attachment loss [15—18]. Increased PD values are linked to increased inflammatory mediators and cytokines, which in turn increase inflammation compared to low values [10].

The current results study demonstrated a significant increase in AST and ALT enzymes, agreeing with [17], which indicated a probability correlation of 99% between smoking and the activity of enzymes (AST and ALT), heavy smoking leads to the higher activity of the AST and ALT enzyme. This may be because smoking impairs the liver's ability to detoxify, which can lead to inflammation and changes in the activity of liver enzymes [10].

Bleeding, CAL, and PD are biochemical tests that are important for diagnosing, observing, predicting, and checking periodontal disease, which is related to the level of enzymes involved in metabolism [11].

Cross-sectionally, we interpreted the correlation between periodontal status and AST and ALT levels. The current study demonstrated a significant positive correlation between both CAL and PD with ALT and AST enzymes among smokers, and a significant positive correlation between CAL and both ALT and AST enzymes among non-smokers, reflecting the biological activity through inflammatory responses, this result agrees [6]. Liver damage may occur due to bacterial pathogens that can destroy periodontal tissue [15].

Lipopolysaccharides (LPS) produced by bacteria that cause periodontal disease can enter the blood vessels and may destroy hepatocytes, which liberal AST and ALT, as LPS induces levels of inflammatory cytokines, especially TNF-α, which leads to damage of periodontal tissue and damage of parenchymal tissue; thus, LPS and TNF-α can be important substances that clarify the relationship between periodontal disease and elevated AST and ALT levels [15].

As CAL$\geqslant$6 mm is linked with high levels of cytokines, reflects the destruction of periodontal tissue caused by TNF-α, thus the infection in addition to smoking which causes periodontal disease may produce large quantities of LPS and TNF-α, these effects of periodontal disease can lead to inflammation in local gingiva and the other organs, including the liver [16].

The analysis showed a positive correlation between bleeding, CAL, and PD with WBCs, Neu, Lym, and Plt in smokers, and there was a positive correlation between bleeding and CAL with WBCs, Neu, Lym, and Plt in non-smokers, such as periodontitis, can significantly affect total WBCs and neutrophil counts, which is consistent with the results of [19] indicating that patients suffering from periodontitis have higher levels of WBCs compared to controls, which may be due to an increased response of the host to oral bacteria, which is manifested through an increased inflammatory response in the form of an increased WBCs and neutrophil count.

However, there was a negative correlation between bleeding and RBC, Hb, and HCT in the smoker and non-smoker groups; as gingival bleeding is considered an indicator of gingivitis and periodontitis, there is some evidence indicating that smoking may be linked with symptoms less expressive in periodontal inflammation, such as gingival bleeding, suggesting a suppressive effect in an inflammatory response. Hence, cigarette smoking affects blood flow, resulting in a significant increase in gingival blood circulation [20].

Therefore, hematological tests have proven that blood parameters such as WBCs, Neu, Lym, Plt, RBC, Hb, and HCT can provide valuable insights into systemic inflammation and overall health status, and the relationship between periodontal disease, liver function, and blood parameters has become a crucial point of interest in future studies.

CONCLUSIONS

This study focused on the necessity of understanding the complex links between periodontal disease, liver function, and blood parameters, which can contribute to liver dysfunction or damage, as well as inflammatory responses in periodontal diseases that can cause changes in blood parameters. Studying these correlations can provide valuable insights that can guide future research in this area.

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