Effects of Smoking and Lipid Profile of the Patient on the Onset and Maintenance of Periodontal Disease

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The purpose of our study is to determine the correlation between the effects of smoking, triglycerides and cholesterol (total, HDL, LDL) and periodontal disease. 90 patients, smokers diagnosed with periodontal disease and 30 nonsmoker patients (control group), in the age of 30-60 years, were studied. Patients were divided into 4 groups, each of 30 patients as follows: group I - smokers up to 10 cigarettes a day, group II- smokers between 10-20 cigarettes a day, group III-over 20 cigarettes a day, group IV- control group, nonsmokers. The values of the Silness-Loe dental plaque index (DPI), the gingival bleeding index (GI), the Periodontal Disease Index (PDI) -Ramfjord were noted in both the examined and control groups. The values of total cholesterol and HDL cholesterol showed statistically significant difference between the mean values †in the examined groups, while mean values †of HDL cholesterol in the second and third examined subgroups and control group were in higher range than the normal one. We concluded also that there is a relationship between smoking, the number of cigarettes smoked and the periodontal disease.

Keywords: smoking, cholesterol, periodontal disease

Smoking in increased and long-term amounts leads to an increased concentration of total serum cholesterol, triglycerides, LDL cholesterol, but also to lowering anti-atherogenic HDL cholesterol [1], which plays a key role in the atherosclerosis process. The combination of nicotine and lipopolysaccharide (LPS) leads to the release of decitokines such as IL-1β and TNF-α, which influence lipid metabolism and promotes hyperlipidemia [2]. With regard to the potential association between smoking, hyperlipidemia and periodontal disease, it is now believed that smoking can induce changes in cellular immune function, resulting in low metabolic regulation of lipids by mechanisms involving proinflammatory cytokines [3].

In the oral cavity there is a balance between the condition of the oral mucosa and the marginal periodontium and the factors that tend to destabilize this equilibrium. The periodontitis of the periodontal disease has been much discussed in the literature. Lastly, the bacterial plaque was considered the sole responsible for triggering the infection in the cavity. At the present, local factors are also taken into account by the general factors [4].

Roughly half of the periodontitis or inflammation around the teeth cases is attributed to current or former smoking. Smokeless tobacco causes gingival recession and white mucosal lesions. Up to 90% of patients with periodontitis who are not helped by common modes of treatment are smokers. Smokers have significantly higher bone loss than nonsmokers, and the trend can be extended to pipe smokers to have more bone loss than nonsmokers. Smoking has been proven to be an important factor in the staining of teeth [5,6]. Halitosis or bad breath is common among tobacco smokers. Tooth loss has been shown to be 2 to 3 times higher in smokers than in non-smokers [7]

Smoking can be involved in the pathogenesis of periodontal disease through the release of proinflammatory cytokines and inflammatory mediators, which can initiate a cascade of biochemical reactions and cause periodontal and endothelial damage [8]. Thus, smokers can be systemically affected even in the absence of clear clinical symptoms of the disease [9]. Cholesterol is affected by tobacco smoking leading to various conditions associated with cardiovascular disease, cerebrovascular accident and coronary artery disease. This condition arises as carbon monoxide in the blood of cigarette smokers can damage the endothelium and accelerate the entry of cholesterol from plasma into the walls of the coronary artery, which accumulation of cholesterol ultimately leads to blockage of the coronary artery resulting in arthrosclerosis [10,11]. In addition, due to tobacco use, nicotine can cause or act as a vasoconstrictor there by decreasing the blood supply to the heart muscle, apart from increasing fatty acid deposition on the walls of the inner walls of the arteries of the heart [12]. Smoking tends to increase blood cholesterol levels. Furthermore, the ratio of high-density lipoprotein (low-density lipoprotein) to low-density lipoprotein tends to be lower in smokers than non-smokers. [13]. Smoking also raises the levels of fibrinogen and increases platelet production (both involved in blood clotting) which makes the blood viscous. Carbon monoxide binds to the hemoglobin (the oxygen-carrying component in the red blood cells), resulting in a much more stable complex than hemoglobin bound with oxygen or carbon dioxide - the result is permanent loss of blood cell function. [14,15]. Blood cells are naturally recycled after a certain period of time, allowing for the creation of new, functional erythrocytes. [16]. However, if carbon monoxide exposure reaches a certain point before it can be recycled, hypoxia (and later death) occurs. All these factors make smokers more at risk of developing various forms of arteriosclerosis [17]. As arteriosclerosis progresses, blood flows less easily through rigid and narrowed blood vessels, making the blood more likely to form a thrombosis (clot). Sudden blockage of a blood vessel may lead to an infarction [18].

Taking into account the research on the effects of smoking and especially those related to the onset and

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maintenance of periodontal disease, as well as those related to the connection between cardiovascular and smoking disorders, we considered it to be a concern to both effects.

Experimental part

Material and methods

120 patients were selected from a group of 840 subjects based on the age criterion: 30-60 years, smokers with periodontal disease at different stages. We studied a sample of 120 people who presented themselves for analyzes in the period 15.08.201-13.02.2017, among which triglycerides, total cholesterol, HDL and LDL. In the study we also took a number of 30 subjects who present periodontal diseases but are non-smokers. All patients signed an informed consent and agreed that the collected data and materials would only be used for scientific and research purposes. The researches were conducted in the Dental Medicine Faculties of Oradea and Targu Mures.

Periodontal disease was diagnosed after a dental consultation later. Triglycerides, cholesterol, HDL and LDL cholesterol were determined on the biochemistry machine. The BS 600 Biochemistry Automated Analyzer has a fully automated system for wet chemistry reagents, random access with the principle of photometric absorbance, turbidimetry, potentiometry. The serum used is harvested in the morning 8-10, after a 12-hour fasting.

Standard values are [19,20]:

-Total cholesterol: Normal <200 mg/dL, High at limit 200-239 mg/dL, High ≥ 240 mg/dL;

-LDL Cholesterol: Normal <100 mg/dL, High at limit 100-159 mg/dL, High160-189 mg/dL, Very high ≥190 mg/dL;

-HDL Cholesterol: Normal 40-60 mg/dL,

-Triglycerides: Normal <150 mg/dL, High at limit 150-199 mg/dL, High 200-499 mg/dL, Very high ≥ 500 mg/dL.

The periodontal disease that was present was classified according to the American Association of Periodontology from 1999 [21]. The diagnosis was based on anamnesis, clinical evaluation and x-ray findings. The indices taken into account were Silness-Loe Plaque Index, Gingival bleeding index (GI) and Periodontal Disease Index (PDI) – Ramfjord. The Silness-Loe plate index measures the thickness of the bacterial plaque along the gumline. To visualize the plate the teeth are air-dried and the plate does not stain. It is noteworthy: 0=plaque absence; 1= plaque is observed only after scraping with the probe; 2=plaque is visible with the naked eye in the fine deposit; 3=plaque is visible in thick layer from the free gingival edge to the dental surface [22,23]. Gingival bleeding index (GI) determines the presence or absence of interproximal gingival bleeding and on the free faces of all teeth. The importance of this index lies in the fact that bleeding at the survey is a much more objective indicator of gingival inflammation than the incipient changes in gingival color [24]. Divide the circumference of the marginal gingival into four areas (B,M,L,D), gently massage the periodontal prostate gland and score for each face values †from 0 to 3 as follows: 0=normal gum; 1=slight inflammation, discreet change in color and texture (slight edema) without touch-probe bleeding, 2=moderate inflammation, erythema, edema that gives glossy appearance to the gum, bleeding at probe pressure, 3=severe inflammation, redness and marked edema, tendency to spontaneous hemorrhage, some ulceration. Periodontal Disease Index (PDI) - Ramfjord was used to analyze the loss of gingival attachment to the following degrees [25]: 0=no attachment loss; 1=Attachment loss up to 3 mm, measured from the enamel-cement junction, 2=Attachment loss of 3-6 mm, 3=Attachment loss over 6mm.

The 90 smoker patients were divided into three study groups, depending on the number of smoked cigarettes/day, in three groups (up to 10 cigarettes a day; 10-20 cigarettes a day; over 20 cigarettes a day). Control group (30 patients) of non-smoking subjects had the same age category. The blood supply used for this work was collected through the venous puncture of the antecubital vein of the forearm using fresh disposable hypodermic syringes and needles, after sterilization of the forearm with cotton wool soaked in 70% alcohol.

Results and discussion

Table 1 shows the mean values of total cholesterol in examined groups with its sub-groups and control group which are in normal ranges in the first, and fourth sub-group, while the mean values in second and third sub-group is higher than the referent values. The results regarding to the total cholesterol show statistically significant differences between the mean values of the groups.

Table 1

TOTAL CHOLESTEROL VALUE

Group	Number	Mean value
I	30	145.5
II	30	210.6
III	30	257.4
IV	30	147.3

Table 2 shows the mean values of LDL cholesterol in examined group and control group. We underline that the mean value in smokers group are higher than normal and are higher than in nonsmokers and in light smokers group.

Table 2

LDL CHOLESTEROL VALUE

Group	Number	Mean value
I	30	124.3
II	30	175.2
III	30	190.1
IV	30	122.5

Table 3 shows the mean values of HDL cholesterol in examined groups. The results shows normal ranges (40-60 mg/dL) and there are no significant differences.

Table 3

HDL CHOLESTEROL VALUE

Group	Number	Mean value
I	30	45.6
II	30	41.2
III	30	40.1
IV	30	44.5

Table 4 present the mean values of triglycerides in the examined groups, which are higher than the referent one (<150 mg/dL), but the differences are not statistically significant.

Table 4

TRIGLYCERIDES VALUE

Group	Number	Mean value
I	**30**	**300.4**
II	**30**	**310.5**
III	**30**	**370.4**
IV	**30**	**290.3**

Analysis of figure 1 revealed that the Silness-Loe dental plaque index has the highest value in the patients of *group III* of smokers (over 20 cigarettes per day). There are no significant differences between of Silness-Loe plaque index in any of the groups (p=0.000) correlated with the degree of periodontal disease.

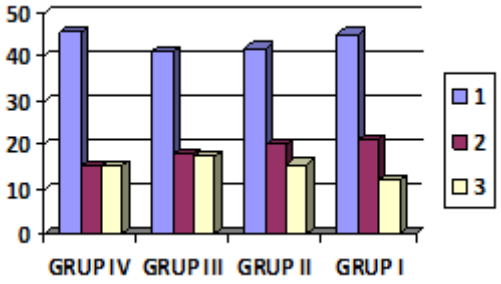

Fig.1 Distribution of Silness-Loe dental plaque index in groups

Analysis of figure 2 showed that the bleeding index presented higher values in the patients of III group. This group experienced gingival bleeding in 100% of patients in varying degrees, most of the grade 3. The differences are not significant for the group of non-smokers and the group of occasional smokers, but are significant for smokers.

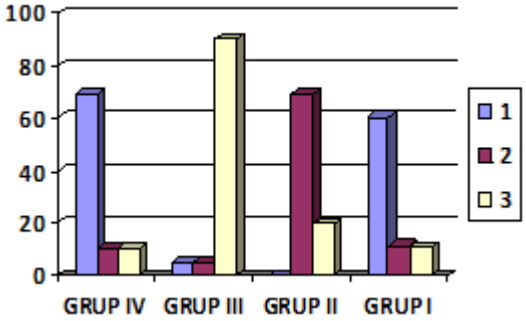

Fig.2 Distribution of gingival bleeding index

Analysis of the figure 3, which studied the Periodontal Disease Index (PDI), demonstrated that the periodontitis degree increases with the number of smoked cigarettes/ day. Group III (35%), presented a greater reduction of gingival attachment was (between 3-6 mm in 41.3% and over 6 mm in 35% of patients). For the other of studied groups, periodontal disease does not differed significantly.

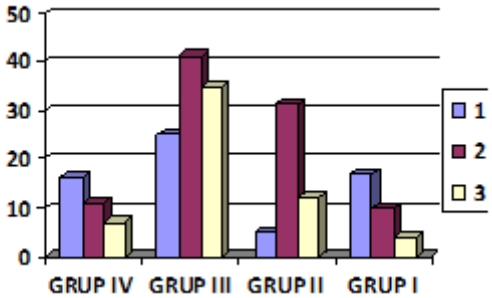

Fig.3 Distribution of the Periodontal Disease Index (PDI)

Some studies may contradict our findings [3], that the bacterial plaque is influenced by the smoker-non-smoking status by depositing the nicotinic deposit on the dental enamel.

Conclusions

Analyzing the obtained results of our study, we have drawn the following conclusions:

The mean of total cholesterol and LDL level are significantly elevated (by over 70-80%) in smokers, both in group III and IV vs. control and occasional smokers group, results which clearly give a link between smoking and elevation of the total cholesterol level.

HDL cholesterol and triglycerides levels are not significantly different, so that we cannot conclude that exist relationships between smoking and these parameters.

It is clear that there is a relationship between smoking, the number of cigarettes smoked and the bleeding index. There are no significant differences between group IV (non-smokers) and group I (occasional smokers). The increasing number of smoked cigarettes per day increases the degree of periodontal affection.

We cannot say the same thing about the bacterial plaque degree, because there are no significant differences between the studied groups.

Gingival attachment loss in smokers is increased, and the loss rate depend on the nicotine dose taken by smoking and the effect shown years later.

The study would be beneficial to be extended over a longer period of time in order to follow the evolution of the patients (both if they continue smoking and if they give up this habit), and to monitored if the medication and diet will normalize the values determined in laboratory.

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