STUDIES REGARDING THE BIDIRECTIONAL RELATIONSHIP BETWEEN THE PERIODONTAL DISEASE AND HYPERLIPIDAEMIA

Amelia Macovei-Surdu*, Ioana Rudnic, Ioana Mârțu, Sorina Solomon, Liliana Păsărin, Silvia Mârțu

Odontology, Periodontology and Fixed Prosthodontics Department
Faculty of Dentistry, "Gr.T. Popa" University of Medicine and Pharmacy - Iasi, Romania

[*]*Corresponding author:*   *Amelia Macovei-Surdu, DMD, PhD student*
     Department of Implantology, Removable Prosthodontics, and Dental Technology
     Faculty of Dental Medicine
     University of Medicine and Pharmacy "Grigore T. Popa" - Iasi, Romania
     16, Universității Street, 700115
     E-mail: macovei.amelia.surdu@dentesse.com

ABSTRACT

Introduction Hyperlipidaemia is influenced by the oral diseases, especially by the periodontal disease. The systemic exposure to infection challenges may have as a result the release of inflammatory cytokines, modifying the lipid metabolism, promoting hyperlipidaemia and atherosclerosis. **Aim of the study** The present study was conducted in order to assess the levels of cholesterol, low density lipoproteins (LDL), high density lipoproteins (HDL) and triglycerides on subjects with and without periodontal disease. **Materials and methods** We selected for the study a group of 36 male subjects and 24 female subjects, with the age between 30 and 50 years. We analysed separately the biochemical markers evolution, for a more efficient scientific presentation of the date obtained. **Results and discussions** The obtained values indicated a strong relation between the lowering of the lipid profile and the periodontal health; we also observed the diminishing of the plasmatic lipidc concentrations. The pathological levels of the triglycerides were around 6.5 times more frequent in periodontitis patients than in control group subjects, while no HDL difference was observed. **Conclusions** Pro-arteriogenic modifications of plasmatic lipids and blood glucose levels observed in periodontal disease patients may provide further evidence of a tight association between the periodontal disease and cardiovascular disease, with a possible role of hyperlipidaemia in periodontal disease.

Keywords hyperlipidaemia, risk factor, periodontal status

INTRODUCTION

Recent studies have demonstrated the influence of oral diseases on hyperlipidaemia, especially of periodontal disease [1, 2]. There is evidence of the tight relationship between the periodontal lesions, high blood lipid levels and *Porphyromonas gingivalis* antibodies presence. Hyperlipidaemia determines a high activity of the white corpuscles in blood (a high production of oxygen radicals) that might be associated to periodontitis onset in adult population [3]. Chronic bacterial infections, including periodontitis, have been associated to a high risk for arteriosclerosis and coronary disease. The systemic exposure to infection challenges, like bacterial lipopolysaccharides, as in periodontitis, may have as a result the release of inflammatory cytokines, such as IL-1β and TNF-α, modifying the lipid metabolism, promoting hyperlipidaemia and atherosclerosis. Bacterial translocation in periodontal pockets

determines the release of systemic inflammatory mediators, with monocyte activation and lipoprotein modification to an atheriogenic profile [4, 5, 6].

Aim of the study

The present study was conducted in order to assess the levels of cholesterol, low density lipoproteins (LDL), high density lipoproteins (HDL) and triglycerides on subjects with and without periodontal disease. Additionally, we assessed the association between high lipid profiles and periodontal disease in order to allow prophylactic measures against cardiovascular diseases (CVD) by periodontal management.

MATERIALS AND METHODS

We selected for the study a group of 36 male subjects and 24 female subjects, with the age between 30 and 50 years, presented in the Periodontology Department and in the private dental office during September 2011-July 2012.

We divided the subjects in two groups: a test group and a control group, with 30 subjects each.

The control group (64.10% female subjects and 35.90% male subjects) included patients with generalized chronic periodontitis who received only oral hygiene maintenance measures (professional brushing, CHX 0.09%).

The study group (51.28% female subjects and 48.72% male subjects) included chronic periodontitis patients who received initial etiologic therapy.

All the participants received detailed information regarding the applied assessment and treatment measures; the informed consent was obtained from each of them. Both groups were sequentially recruited within a period of 6 months from the patients who came for a usual control visit in the personal private dental office.

The exclusion criteria were as follows: no dental treatment in the last 6 months, diabetes mellitus or any other endocrine disease, myocardial infarction, stroke, tumoral pathology. Smoking subjects (one subject in the control group and 7 from the study group) were also excluded. No subject received any treatment for the hypercholesterolemia.

According to the questionnaire, there weren't any differences of the social status between the two groups.

All the patients with more than 3 periodontal pockets>4mm were included in the test group.

We analysed separately the biochemical markers' evolution, for a more efficient scientific presentation of the date obtained.

The blood samples were obtained after at least 12 hours of fasting for biochemical analysis of lipid levels. The 5ml blood samples were obtained with red vacutainer collection tubes or simple dry test tubes and centrifuged after 45 minutes. The separated serum was collected in a clean tube. We analysed the lipid levels by homogenous enzymatic calometry using COBAS6000 for the test instrument.

The measurements were conducted in the laboratory of the Infectious Diseases Clinical Hospital and at Synevo Laboratory Iaşi using enzymatic spectrophotometry methods.

We present the mean values and the standard deviation. The differences between average values were proven to be significant by the Student's t-test for unpaired samples.

RESULTS

The obtained values indicated a tight relation between the lowering of the lipid profile and the periodontal health status. We observed decreased plasmatic lipid concentrations on patients who received periodontal treatment, with an improved periodontal status.

According to the depth of the periodontal pocket, most of the patients suffered from

moderate periodontal disease; the probing depth of 4-5mm was present on 47.2 ±24.6% of the probing sites and more than 5mm on 8.6 ± 16.3% of the probing sites.

A few control cases presented moderate probing depths (8.7 ± 8.5% of sites) but no profound periodontal pockets.

As we expected, the bleeding on probing after the treatment was lower for the test subjects (5.1 ± 26.3%) than for the control group (35.0 ± 6.9%, p = 0.0001) (Fig. 1).

A qualitative estimation of plaque accumulation did not reveal a significant difference between the test and control group (50± 19% and 48 ± 23%, respectively).

Total cholesterol, LDL cholesterol and triglyceride levels were significantly higher on test group subjects with approximately 8% (p=0.03), 13% (p=0.003) and 39% (p=0.001), respectively, comparing to the control group subjects at a month after treatment.

The mean age values were higher in the control group subjects (40.3667 / 38.7). The mean values of the body mass index were higher for the test group participants (22.8120 / 24.0230).

The values for the total cholesterol at a month after treatment were higher for the control group subjects (180,113 / 171.4883) (Fig. 2).

On control group patients the triglyceride levels were higher than those from the test group (136.3640 / 103.0487) (Fig. 3).

On the test group patients HDL values were higher than in the control group (45.8873 / 48.6653) (Fig. 4).

On the control subjects LDL values were higher than test subjects values (103.0667 / 88.7400) (Fig. 5).

On test group patients VLDL values were higher than the control group patients (33.000/ 37.7433) (Fig. 6).

On test group subjects the glucose levels were higher than the control group values (81.7667 / 82.1000) (Fig. 7).

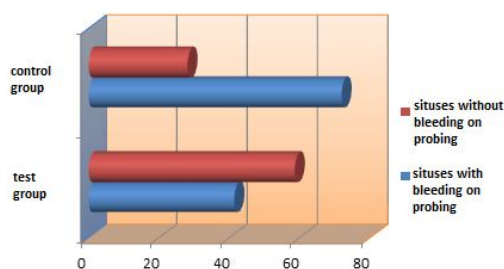On control group subjects Chol_HDL values were higher than the values in the test group (4.09 / 3.66) (Fig. 8).
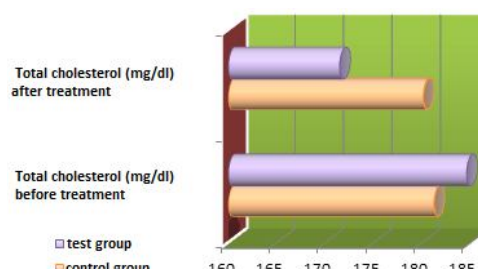

Fig. 1. Bleeding on probing after treatment

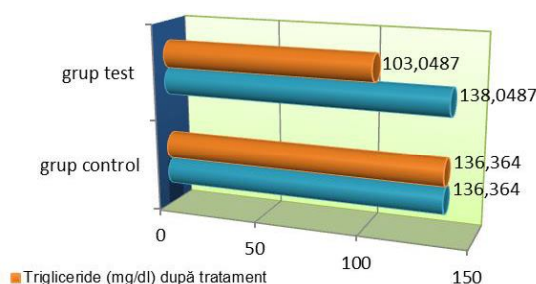**Fig. 2. Total cholesterol values distribution before and at a month after treatment**

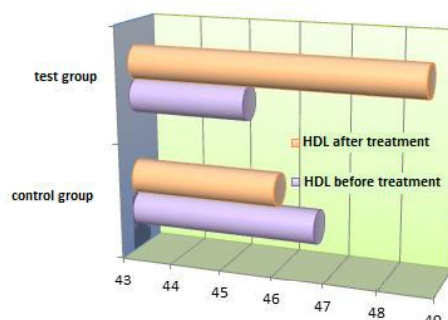

Fig. 3. Trigliceride values distribution on both groups
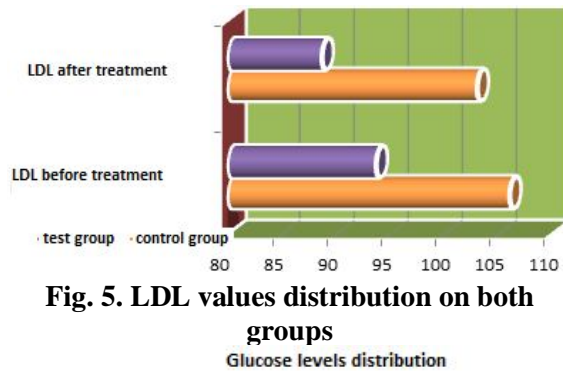

Fig. 4. HDL values distribution on both groups

Fig. 5. LDL values distribution on both groups

Fig. 6. VLDL values distribution on both groups

Fig. 7. Glucose values distribution on both groups

Fig. 8. Chol_HDL values distribution on both groups
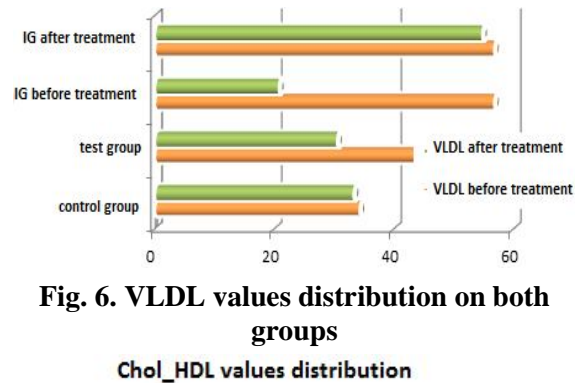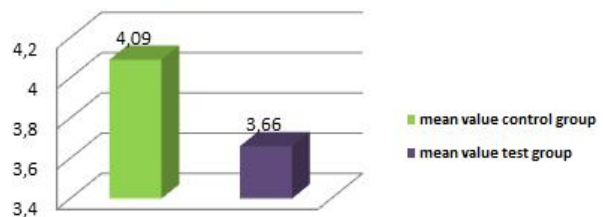
DISCUSSIONS

In our study the degree of periodontal lesions was positively correlated to the cholesterol plasmatic levels. The obtained values revealed a tight connection between the lowering of the lipid profile and the periodontal health; we also observed the diminishing of the plasmatic lipid concentrations (Fig. 9).
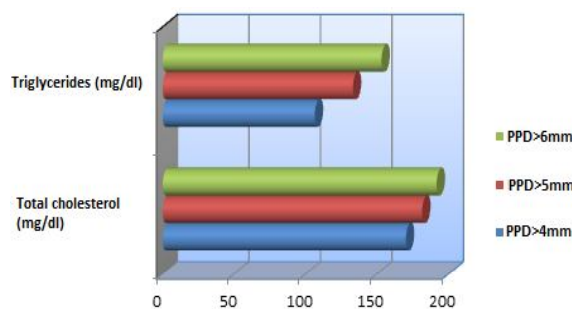

Fig. 9. Lipids profile and periodontal health status

Therefore, we can conclude that the lipidic markers represent a risk factor for the cardiovascular disease and may influence indirectly also the periodontal health status. The pathological levels of the triglycerides were around 6.5 times more frequent in periodontitis patients than in control group

subjects, while no HDL difference was observed.

Hypercholesterolemia (especially high levels of LDL cholesterol), hypertriglyceridemia and diabetes mellitus are major risk factors for cardiovascular disease. In opposition, high levels of HDL cholesterol have been proven to associate to a low risk for cardiovascular disease.

Our results demonstrate that hypercholesterolemia patients present a poor periodontal status.

We observed extremely significant differences in the assessment of plasmatic pathological lipid concentrations between the periodontitis patients and the control subjects.

More and more epidemiology studies confirm the association between periodontal disease and atherosclerotic coronary disease. These two diseases (chronic periodontitis and atherosclerosis) have complex ethology, genetic predisposition and many common risk factors, smoking being the most significant one. Chronic periodontal infection may contribute to atherogenesis process, to the evolution of this process on arterial levels. Periodontal disease, as cardiovascular disease,

has inflammation as a common aspect. Local periodontal inflammation stimulates the systemic inflammation. In our study we measured the plasmatic lipids levels on patients with periodontitis who received etiologic treatment and who didn't receive such treatment. Furthermore, we determined de periodontal modification degree, correlated to the plasmatic levels of cholesterol. Comparative to the control group, the periodontitis patients had higher levels of total cholesterol, LDL and triglycerides; the frequency of pathological lipid profiles was higher than those in the control group.

According to the assessments in our study regarding the periodontal pocket depth, most of the patients presented moderate periodontal disease; 47.2 ±24.6% of the situses presented a PPD between 4 and 5mm and 8.6 ± 16.3%-PPD>5mm. A few control cases presented moderate probing depths (8.7 ± 8.5% of sites) but no profound periodontal pockets.

As we expected, the bleeding on probing was more frequent in the control group subjects than the test subjects (25.1 ± 26.3% and 5.0 ± 6.9%, p = 0.0001, respectively).

A qualitative estimation of the bacterial plaque didn't reveal a significant difference between the test and control groups (50 ± 19% and 48 ± 23%, respectively).

As we have shown, the mean values for cholesterol and LDL on periodontitis patients from the control group were significantly higher with 8% and 13% than the test subjects. Besides, plasmatic triglyceride levels were higher in patients than the control subjects (+39%); no difference was observed for HDL cholesterol [6, 7].

We observed significant differences between the periodontitis and control subjects in estimating the frequency of plasmatic pathological lipids concentrations. Hypercholesterolemia frequency in the test group was approximately double than the control group [8, 9]. This aspect is similar to

total cholesterol and LDL cholesterol.

The pathological triglyceride levels were 6.5 times higher for the periodontitis patients; no difference was noticed for HDL cholesterol.

Although diabetes mellitus represented one of the exclusion criteria in our study, we determined the blood glucose levels à jeun too. We didn't notice pathological values of this variable in none of the groups [11, 12, 13, 14]. Nevertheless, there was a significant difference between the mean values in the groups. The mean value for the glucose level was 15% higher in the test patients [15].

We demonstrated in this study a possible role of hyperlipidaemia in the periodontal disease. The pro-atherogenic modifications of plasmatic lipids and blood glucose observed in periodontal disease patients may offer further evidence of a tight association between the periodontal disease and cardiovascular disease. However, it isn't clear that the observed modification in the glucose and lipids metabolism represent a cause or an effect for the periodontal disease.

Further studies are necessary to clarify the mechanism of the association between the cardiovascular disease and periodontitis.

CONCLUSIONS

The pro-atherogenic modifications of plasmatic lipids and blood glucose observed in periodontal disease patients may offer further evidence of a tight association between the periodontal disease and cardiovascular disease.

We demonstrated in the present study a possible role of hyperlipidaemia in the periodontal disease by assessment of the periodontal destruction degree correlated to the cholesterol plasmatic levels. Still, it isn't clear that the observed modification in the glucose and lipids metabolism represent a cause or an effect for the periodontal disease.

REFERENCES

1. Cutler CW, Shinedling EA, Nunn M, Jotwani R, Kim B, Nares S, Iacopino AM. Association between periodontitis and hyperlipidemia: cause or effect? J Periodontol 1999b;70:1429-1434
2. Izumi A, Yoshihara A, Hirotomi T, Miyazaki H. The relationship between serum lipids and periodontitis in elderly non-smokers. J Periodontol 2009;80(5):740–748
3. Jain A, Batista EL, Serhan C, Stahl GL, Van Dyke TE. Role for periodontitis in the progression of lipid deposition in an animal model. Infect Immun 2003;71(10):6012–6018
4. Katz J, Flugelman MY, Golberg A, Heft M. Association between periodontal pockets and elevated cholesterol and low density lipoprotein cholesterol levels. J Periodontol 2002;73:494-500
5. D'Aiuto F, Nibali L, Parkar M, Suvan J, Tonetti MS. Short-term effects of intensive periodontal therapy on serum inflammatory markers and cholesterol. J Dent Res 2005;84:269-273
6. Mârţu S, Pasarin L, Solomon S, Rudnic I. Studies regarding the interrelation and interdependence between periodontal disease and sistemic diseases/Studii privind interrelatia si interdependenta boala parodontala boli sistemice. Medicina Stomatologica / Romanian Dentistry J., 2006,10,4:62-69
7. Saxlin T, Suominen-Taipale L, Kattainen A, Marniemi J, Knuuttila M, Ylostalo P. Association between serum lipid levels and periodontal infection. J Clin Periodontol 2008;35(12):1040–1047
8. D'Aiuto F, Parkar M, Nibali L, Suvan J, Lessem J, Tonetti MS. Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomized controlled clinical trial. Am Heart. J 2006;151(5):977–984
9. D'Aiuto F, Parkar M, Tonetti MS. Acute effects of periodontal therapy on bio-markers of vascular health. J Clin Periodontol 2007;34(2):124–129
10. Surdu Macovei AE, Pasarin L, Potârnichie O, Balan A. On the modifications of the lipidic biochemical parameters and extent of periodontal affection. Int. J. of Medical Dentistry, 2012; 2(3): 209-21
11. Higashi Y, Goto C, Hidaka T, et al. Oral infection-inflammatory pathway, periodontitis, is a risk factor for endothelial dysfunction in patients with coronary artery disease. Atherosclerosis 2009;206 (2):604–610
12. Scannapieco FA, Bush RB, Paju S. Associations between periodontal disease and risk for atherosclerosis, cardiovascular disease, and stroke. A systemic review. Ann Periodontol 2003;8:38–53
13. Sridhar R, Byakod G, Pudakalkatti P, Patil R. A study to evaluate the relationship between periodontitis, cardiovascular disease and serum lipid levels. Int J Dent Hygiene 2009;7:114-50
14. Wu T, Trevisan M, Genco RJ, Falkner KL, Dorn JP, Sempos CT. Examination of the relation between periodontal health and cardiovascular risk factors: Serum total and high density lipoprotein cholesterol, C-reactive protein, and plasma fibrinogen. Am J Epidemiol 2000a;121:273-282
15. Loesche W, Karapetow F, Pohl A, Pohl C, Kocher T. Plasma lipid and blood glucose levels in patients with destructive periodontal disease. J Clin Periodontol 2000;27:537-41