concentration of inflammatory markers. The aim of this investigation was to explore if periodontitis influences over plasma antioxidant profile in men.
Material and methods: A total of 27 male subjects (periodontitis = 19; control = 8) were included in this study. Established periodontitis was diagnosed as the presence of ≥ 2 teeth with clinical loss attachment ≥ 6 mm and ≥ 1 sites with probing depth ≥ 5 mm. Those participants not fulfilling these criteria were included in the control group. Blood samples were collected and oxidized coenzyme Q, reduced coenzyme Q, total coenzyme Q total and vitamin E were analyzed by HPLC-EC. Mann–Whitney test was used to analyze differences between groups.
Results: Plasma vitamin E levels did not show differences between both groups, but significant differences were found for oxidized coenzyme Q, being the highest values found for the periodontitis group.
Conclusion: Periodontitis is related to an increased oxidized coenzyme Q level in men, which might be considered as a net increase of the oxidation process.

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The influence of periodontal therapy on plasma rantes, PMN elastase/ (α1-PI) and IL-10 levels in diabetes mellitus patients with gingivitis

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Background: The aim of this study was to investigate whether periodontal therapy could affect the glycemic control and plasma inflammatory markers of type 2 diabetes mellitus (DM) patients with gingivitis.
Methods: Blood samples were collected at baseline, 1st and 3st months after periodontal therapy from 12 DM patients with gingival inflammation and poor oral hygiene (mean age: 45.5 ± 4.2). Patients received full mouth scaling with ultrasonic and manual instruments and oral hygiene motivation. Plasma levels of RANTES, interleukin (IL)-10, polymorphonuclear (PMN) elastase/α1-proteinase inhibitor (α1-PI) were measured by enzyme-linked immunosorbent assay at the same time periods.
Results: α1-PI plasma levels decreased significantly ($P < 0.01$). Mean plasma levels were 100.54 ng/ml before treatment and 54.183 ng/ml 3 months after periodontal therapy. Subjects had significant reduction in mean hemoglobin A1c (HbA1c) from 9.29% to 8%. RANTES and IL-10 plasma levels reduced after the therapy but the significant difference was not observed.
Conclusions: α1-PI levels in plasma can show the activity of granulocytes during inflammatory response. Therefore, eliminating the gingival inflammation might result with reduction in the plasma PMN elastase/(α1-PI) levels and improvement in glycemic control. The effect of periodontal therapy on plasma inflammatory markers of DM patients with gingival inflammation will require further studies that will have to include much larger sample sizes.

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Periodontal disease as a risk factor of carotid artery atherosclerosis

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Aims: The carotid plaque severity has been associated with increased risk of cardiovascular disease. Our aims were to investigate the association between carotid artery atherosclerosis and periodontal status as well as to estimate the role of inflammatory markers and dyslipidemia in periodontal disease and carotid artery atherosclerosis.
Materials and methods: A total of 60 non-smoker participants (32 females in 28 males), aged 58 ± 8 years, were included in the study.Periodontal clinical examination included probing depths, recessions, attachment levels, plaque record and bleeding on probing score. The common carotid arteries and bulbi were evaluated ultrasonographically in all participants to determine the IMT, site of plaques, and stenosis grade. The hs-CRP, total cholesterol, LDL, HDL and trigliceride levels were determined in blood samples from every participant.
Results: Positive correlation between probing depth and IMT was found at common carotid artery and bulbi sites. In addition, hs-CRP and atherogenic index correlated positively with probing depth in all plaque groups. Groups of calcified and lipid plaques exhibited deeper pockets in comparison to control group ($P < 0.05$). The deepest periodontal pockets were recorded in calcified plaque group.
Conclusions: The results of our study suggest periodontal disease as a possible risk factor of carotid artery atherosclerosis which might predict the cerebrovascular ischemic events.